RonanRx
PERSONALIZED MEDICINE

CLINICAL MONOGRAPH · METABOLIC & LONGEVITY (UNDER FDA REVIEW)

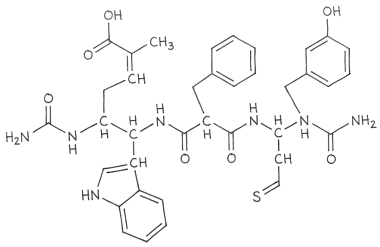
MOTS-C

Mitochondrial peptide research ingredient with case-by-case review

MOTS-c is a very small protein, only 16 amino acids long, that is encoded inside the mitochondrial genome rather than the cell nucleus. It was identified by Changhan Lee and Pinchas Cohen at the University of Southern California in 2015. Almost everything we know about what MOTS-c does comes from cell-culture experiments and mouse studies; there are no controlled clinical trials supporting any human use.

In mouse studies, MOTS-c activates a cellular energy-sensing enzyme called AMPK, improves insulin sensitivity on a high-fat diet, increases with exercise, and translocates to the cell nucleus under metabolic stress where it appears to influence which stress-response genes are expressed [lee2015; kim2018]. These are interesting basic-science findings; they are not evidence that MOTS-c is safe or effective in humans.

MOTS-C has no FDA approval in the United States. This ingredient is part of an evolving FDA review process. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case, and availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance.



EVIDENCE POSTURE PRECLINICAL

REVIEWED **2026-05-11**

State-licensed 503A | Pharmacist reviewed | Doctor led | Cold-chain ready | Patient choice preserved

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FOR CLINICIANS

MOTS-c is a 16-amino-acid mitochondrial-derived peptide (MDP) encoded within an open reading frame internal to the mitochondrial 12S ribosomal RNA gene. It was identified and named by Lee and colleagues in the Cohen laboratory at USC in 2015 [lee2015]. The proposed mechanism centers on activation of AMP-activated protein kinase (AMPK), modulation of the folate, methionine, AICAR pathway in metabolic stress, and, in the metabolic-stress state, translocation of MOTS-c to the cell nucleus where it engages stress-responsive transcription factors and regulates nuclear gene expression [kim2018].

Published evidence is almost entirely preclinical. The discovery paper [lee2015] established the metabolic phenotype in diet-induced-obese mice, with intraperitoneal MOTS-c reducing weight gain and improving insulin sensitivity on a high-fat diet. Subsequent mouse work characterized exercise-induced upregulation of MOTS-c, age-dependent physical decline that is reversed by exogenous MOTS-c [reynolds2021], protection against ovariectomy-induced bone loss via AMPK activation [ming2016], improvement of insulin resistance in a D-galactose accelerated-aging model [li2019], suppression of autoimmune-diabetes islet destruction [kong2021], and reduction of hyperglycemia in a gestational diabetes model [yin2022]. Human studies are limited to observational measurements of MOTS-c in plasma and skeletal muscle [dsouza2020, liu2019] and a single-subject acute-exercise report [hyatt2022]; no randomized placebo-controlled efficacy or safety trials have been published.

MOTS-C has no FDA approval in the United States. This ingredient is part of an evolving FDA review process. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case, and availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance.

Why Personalized MOTS-C

The evidence base for MOTS-C is early. Most published work comes from cellular and mouse models of metabolism, insulin resistance, exercise signaling, and aging biology; the related CohBar analog program did not become an FDA-approved product.

Physicians may submit patient-specific prescription requests for MOTS-C for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, supported by patient-specific documentation, and approved by the dispensing pharmacy. Availability is determined case by case. This is not a consumer access promise; it is a clinical, sourcing, formulation, and regulatory review process. FDA has scheduled MOTS-C-related bulk drug substances for discussion at the 23-24 Jul 2026 Pharmacy Compounding Advisory Committee meeting.

The patient-specific pharmacy pathway is the opposite of a wellness peptide menu. A prescriber must document why the request fits the patient, and the pharmacy must decide whether the preparation can be sourced, formulated, and released responsibly.

Quick Facts About MOTS-C

Category: Mitochondrial-derived peptide (MDP); 16-amino-acid bioactive peptide encoded within the mitochondrial 12S rRNA gene

Active ingredient: MOTS-c (mitochondrial open reading frame of the twelve S rRNA-c), a 16-residue peptide identified by computational analysis of the human mitochondrial genome and characterized by the Cohen lab at the University of Southern California

FDA-approval status: Category 2, evolving FDA review process. Valid patient-specific prescription required; supporting clinical rationale may be requested.

503A bulk substances status: Category 2, evolving FDA review process. Valid patient-specific prescription required; supporting clinical rationale may be requested.

Evidence posture: Almost entirely preclinical (cell culture and mouse models). The discovery paper [lee2015] and the principal mechanism, glucose-homeostasis, exercise, and aging studies are mouse work. Limited human PK and safety data are publicly available; no randomized placebo-controlled efficacy trials have been published.

RonanRx availability: Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

> **SPECIALS: PATIENT-SPECIFIC PRESCRIPTION ONLY**
>
> Physicians may submit patient-specific prescription requests for MOTS-C for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case.
>
> - **Made to order, not off a shelf.** No batch sits in a warehouse waiting for buyers. Your prescription triggers the prep.
> - **Named-patient label.** The bottle carries one patient's name. The batch records carry one prescription.
> - **Dose, strength, and route chosen for the patient.** A prescriber decides what gets compounded, not a manufacturer who set the strength for a trial population.
> - **Licensed pharmacist on the hook.** A real person, with a license that can be pulled, signs off on every prep. State inspectors check the facility.
> - **Compounded drugs are not FDA-approved.** They should not be evaluated using branded-drug trial data alone. Availability varies by state and prescribed medication.

How This Differs from a Research-Use-Only Website

A research-use-only website ships a vial from a warehouse. There is no prescription, no pharmacist, no facility inspection, and no way to recall the product if something is wrong with it. If the vial is mislabeled, contaminated, or under-potent, there is nobody whose license is at stake.

A 503A compounding pharmacy is the other thing. The doctor writes the prescription. A licensed pharmacist, whose name is on the label, prepares the medicine in a facility the state inspects. If something goes wrong, there is a person and a license on the hook, and a documented chain of custody on every lot. That accountability is what makes it safe.

What is MOTS-C?

MOTS-c (mitochondrial open reading frame of the twelve S rRNA-c) is a 16-amino-acid peptide encoded within a short open reading frame internal to the human mitochondrial 12S ribosomal RNA gene [kim2018]. The Cohen laboratory at the University of Southern California identified and characterized MOTS-c in 2015 using computational analysis of the mitochondrial genome to screen for previously unannotated bioactive peptides [lee2015]. MOTS-c belongs to the broader category of mitochondrial-derived peptides (MDPs), which also includes humanin (encoded within the mitochondrial 16S rRNA) and the SHLP family.

MOTS-c is detectable in human plasma, skeletal muscle, and a range of other tissues. The peptide has been measured by enzyme-linked immunosorbent assay and by targeted mass spectrometry; assay standardization across the published literature remains limited. Circulating MOTS-c levels appear to decline with age in cross-sectional human studies, although the relationship between plasma and intracellular MOTS-c is not straightforward, D'Souza and colleagues reported that skeletal muscle MOTS-c expression in healthy aging men increases with age in parallel with a shift toward slower-contracting myofibers, while circulating levels decline [dsouza2020] [kim2018].

There is no FDA-approved MOTS-c product. CohBar Inc. (Menlo Park, CA / Pasadena, CA), founded in part by the original MOTS-c discoverers, developed several modified mitochondrial-derived peptide analogs in the late 2010s and early 2020s, including the MOTS-c-derived analog CB4211 (subsequently re-designated MBT-2 and MBT-3), through phase 1 clinical evaluation in obesity, nonalcoholic steatohepatitis (NASH), and idiopathic pulmonary fibrosis [kim2018]. CohBar discontinued its development programs and dissolved in 2023. No CohBar product reached FDA approval, and no peer-reviewed phase 2 efficacy data on a MOTS-c analog have been published.

How MOTS-C Works

MOTS-c is proposed to act primarily through activation of AMP-activated protein kinase (AMPK), a central cellular energy sensor. In cultured cells and in mouse skeletal muscle, exogenous MOTS-c increases the AMP:ATP ratio and the phosphorylation of AMPK at Thr172, driving downstream activation of catabolic, glucose-uptake, and mitochondrial-biogenesis programs while suppressing anabolic ATP-consuming pathways [lee2015, ming2016].

A second proposed mechanism is mitochondrial-to-nuclear retrograde signaling. Kim and colleagues demonstrated that under metabolic stress (glucose restriction, oxidative challenge), MOTS-c translocates

from the mitochondria to the cell nucleus, where it associates with stress-responsive transcription factors and modulates expression of antioxidant and metabolic-adaptation genes [kim2018]. This nuclear translocation is regulated and reversible and is one of the first described examples of a mitochondrial-encoded peptide acting directly on the nuclear genome.

A third proposed mechanism, more biochemical, links MOTS-c to one-carbon metabolism. The discovery paper reported that MOTS-c modulates de novo purine biosynthesis via inhibition of the folate cycle, raising intracellular AICAR (5-aminoimidazole-4-carboxamide ribonucleotide), which is itself a direct AMPK activator [lee2015]. This 'folate, methionine, AICAR, AMPK' axis is the proposed molecular link between MOTS-c, cellular nutrient status, and the downstream metabolic phenotype.

Biological Role of MOTS-C

The discovery of MOTS-c expanded the proteomic boundary of the mitochondrial genome. Until the 2000s, the human mitochondrial genome (16,569 bp) was considered to encode 13 proteins, 22 tRNAs, and 2 rRNAs. The Cohen-lab work on humanin (encoded within the 16S rRNA) and then MOTS-c (encoded within the 12S rRNA) established that small open reading frames internal to mitochondrial rRNA genes encode bioactive peptides, the mitochondrial-derived peptide (MDP) family. MDPs are now understood as one form of mitochondrial-to-nuclear retrograde signaling, allowing the mitochondrion to communicate its metabolic state to the rest of the cell beyond classical ATP/AMP, ROS, and Ca^{2+} signaling [lee2015, kim2018, merry2020].

MOTS-c specifically has been positioned in the preclinical literature as a candidate 'exercise-mimetic' molecule on the basis of the Reynolds 2021 finding that exercise raises endogenous MOTS-c and that exogenous MOTS-c partially recapitulates exercise-associated improvements in age-related physical decline in mice [reynolds2021]. The framing should be read with caution: 'exercise mimetic' is a mechanistic hypothesis in mouse models, not a demonstrated human clinical effect. Aging and longevity marketing claims that extrapolate directly from these preclinical findings to human healthspan are not supported by controlled trial evidence.

Detailed Mechanism of MOTS-C

The Cohen-lab discovery paper [lee2015] established the central mechanistic frame. In a mouse diet-induced-obesity (DIO) model, daily intraperitoneal MOTS-c (5 mg/kg) prevented high-fat-diet-induced weight gain and improved glucose tolerance and insulin sensitivity. In cultured myotubes and adipocytes, MOTS-c increased 2-deoxyglucose uptake in an AMPK-dependent manner. Metabolomic analysis identified accumulation of AICAR, the direct allosteric AMPK activator, downstream of MOTS-c treatment, with concomitant alterations in folate-cycle intermediates. This led to the working model in which MOTS-c inhibits a step in the folate/methionine cycle, raises AICAR, and thereby activates AMPK [lee2015].

Kim and colleagues (2018) extended the mechanism into mitochondrial-to-nuclear retrograde signaling [kim2018]. Using cellular fractionation, microscopy, and mass spectrometry, they showed that MOTS-c translocates from the mitochondria to the nucleus under metabolic stress (glucose restriction, oxidative challenge). Once nuclear, MOTS-c interacts with stress-responsive transcription factors, including NRF2, and contributes to the transcriptional adaptation to metabolic stress. This finding established MOTS-c as one of the first mitochondrial-encoded peptides shown to act directly on the nuclear transcriptome.

Reynolds and colleagues (2021) demonstrated that MOTS-c is exercise-induced: acute and chronic exercise raise plasma and skeletal-muscle MOTS-c in mice and humans, and exogenous MOTS-c administration improves age-dependent physical performance in older mice [reynolds2021]. The same group documented age-dependent decline in plasma MOTS-c in mice and characterized MOTS-c as a regulator of muscle homeostasis with relevance to age-related physical decline. Hyatt (2022) reported that long-term voluntary physical activity in rodents was associated with increased skeletal-muscle MOTS-c, and that a single dose of MOTS-c improved acute exercise performance after prolonged inactivity [hyatt2022].

Disease-model preclinical work has elaborated the AMPK-pathway mechanism in tissue-specific contexts. Ming and colleagues (2016) showed that MOTS-c suppresses ovariectomy-induced bone loss in mice by inhibiting osteoclast differentiation through AMPK activation [ming2016]. Li and colleagues (2019) reported that MOTS-c improves insulin resistance and oxidative-stress markers in a D-galactose-induced accelerated-aging mouse model in an AMPK-dependent fashion [li2019]. Kong and colleagues (2021) showed that MOTS-c attenuates pancreatic islet destruction in non-obese diabetic (NOD) mice, a preclinical model of autoimmune type 1 diabetes, through a mechanism involving regulation of beta-cell function and apoptosis [kong2021]. Yin and colleagues (2022) reported that MOTS-c relieves hyperglycemia and insulin resistance in a gestational-diabetes-mellitus mouse model [yin2022]. A 2023 review consolidated the AMPK-centered mechanism across stress, metabolism, and aging contexts [wan2023], and a mitochondrial-derived-peptide review summarized the broader MDP family pharmacology [merry2020].

Human observational evidence remains thin. D'Souza and colleagues (2020) measured skeletal-muscle MOTS-c by mRNA and immunoassay in young, middle-aged, and older healthy men and found that muscle MOTS-c expression rises with age, paradoxically alongside reported declines in circulating MOTS-c in other cross-sectional studies [dsouza2020]. Liu and colleagues (2019) reported reduced skeletal-muscle expression of MOTS-c and humanin alongside altered NRF2 signaling in adults with chronic kidney disease compared with controls [liu2019]. Yu and colleagues (2021) showed that MOTS-c promotes homeostasis in aged human placenta-derived mesenchymal stem cells in vitro [yu2021]. None of these studies are randomized interventional trials, and assay methodology varies substantially across reports.

MOTS-C Research History

MOTS-c was identified by computational scanning of the human mitochondrial genome for previously unannotated small open reading frames, building on the earlier Cohen-lab discovery of humanin within the mitochondrial 16S rRNA. Lee and colleagues reported the discovery and initial metabolic characterization in Cell Metabolism in March 2015 [lee2015], establishing the AMPK-pathway mechanism and the diet-induced-obesity mouse phenotype. A 2016 review by Lee, Kim, and Cohen consolidated the early evidence and introduced MOTS-c to a broader metabolic-research audience [lee2016].

Mechanistic work expanded over the following years. Ming and colleagues (BBRC 2016) demonstrated that MOTS-c suppresses ovariectomy-induced bone loss via AMPK activation [ming2016]. Kim and colleagues (Cell Metabolism 2018) demonstrated mitochondrial-to-nuclear retrograde signaling and characterized the metabolic-stress-induced nuclear translocation of MOTS-c [kim2018]. Li and colleagues (BBRC 2019) reported improvement of insulin resistance in a D-galactose accelerated-aging model [li2019]. Reynolds and colleagues (Nature Communications 2021) established MOTS-c as an exercise-induced regulator of muscle homeostasis and age-dependent physical decline [reynolds2021]. Kong and colleagues (Cell Reports 2021) demonstrated protection against autoimmune diabetes in non-obese diabetic mice [kong2021]. Yin and colleagues (Pharmacological Research 2022) reported amelioration of gestational diabetes in mice [yin2022]. Hyatt (Physiological Reports 2022) characterized acute and chronic exercise effects on MOTS-c in skeletal muscle [hyatt2022]. A 2023 comprehensive review by Wan and colleagues consolidated the mechanism across stress, metabolism, and aging [wan2023].

Human observational work has been comparatively sparse. D'Souza and colleagues (Aging 2020) reported age-related changes in skeletal-muscle MOTS-c expression and association with myofiber composition in healthy men [dsouza2020]. Liu and colleagues (Am J Physiol Renal Physiol 2019) reported reduced muscle MDP expression in chronic kidney disease [liu2019]. Yu and colleagues (Mitochondrion 2021) reported in-vitro effects of MOTS-c on aged placental mesenchymal stem cells [yu2021]. A meal-test review by Merry and colleagues (Am J Physiol Endocrinol Metab 2020) summarized MDP biology in human energy metabolism [merry2020].

On the translational side, CohBar Inc., founded in 2007 with Cohen-lab IP, developed several MOTS-c-derived analogs (CB4211, subsequently re-designated MBT-2 and MBT-3) and other MDP-based candidates for indications including obesity, NASH, and idiopathic pulmonary fibrosis. CB4211 advanced through phase 1 clinical evaluation. CohBar discontinued its programs and wound down operations through 2022, 2023, and the company dissolved in 2023. No CohBar product received FDA marketing authorization, and no peer-reviewed phase 2 efficacy results for a MOTS-c analog have been published. The molecule MOTS-c itself remains a research compound with no FDA-approved form. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

MOTS-C Timeline

2015 • Lee et al [lee2015]. (Cell Metabolism), discovery and naming of MOTS-c; characterization of AMPK-pathway mechanism and prevention of diet-induced obesity and insulin resistance in mice

2016 • Lee, Kim, and Cohen (Free Radic Biol Med), review article introducing MOTS-c to a broader metabolic-research audience and consolidating early evidence [lee2016]

2016 • Ming et al [ming2016]. (BBRC), MOTS-c suppresses ovariectomy-induced bone loss in mice via AMPK activation

2018 • Kim et al [kim2018]. (Cell Metabolism), MOTS-c translocates to the nucleus to regulate nuclear gene expression in response to metabolic stress, first demonstration of mitochondrial-to-nuclear retrograde signaling for an MDP

2019 • Li et al [li2019]. (BBRC), MOTS-c improves insulin resistance and oxidative-stress markers in a D-galactose accelerated-aging mouse model in an AMPK-dependent manner

2019 • Liu et al [liu2019]. (Am J Physiol Renal Physiol), reduced skeletal-muscle expression of MOTS-c and humanin in adults with chronic kidney disease vs controls

2020 • D'Souza et al [dsouza2020]. (Aging), skeletal-muscle MOTS-c expression increases with age in healthy men and correlates with myofiber composition; circulating MOTS-c declines with age in cross-sectional comparisons

2020 • Merry et al [merry2020]. (Am J Physiol Endocrinol Metab), review of mitochondrial-derived peptides in human energy metabolism

2021 • Reynolds et al [reynolds2021]. (Nature Communications), MOTS-c is an exercise-induced mitochondrial-encoded regulator of age-dependent physical decline and muscle homeostasis in mice

2021 • Kong et al [kong2021]. (Cell Reports), MOTS-c prevents pancreatic islet destruction in the non-obese diabetic (NOD) mouse model of autoimmune type 1 diabetes

2021 • Yu et al [yu2021]. (Mitochondrion), MOTS-c promotes homeostasis in aged human placenta-derived mesenchymal stem cells in vitro

2022 • Yin et al [yin2022]. (Pharmacological Research), MOTS-c relieves hyperglycemia and insulin resistance in a gestational-diabetes-mellitus mouse model

2022 • Hyatt (Physiological Reports), MOTS-c increases in rodent skeletal muscle following long-term physical activity; single-dose MOTS-c improves acute exercise performance after prolonged inactivity [hyatt2022]

2023 • CohBar Inc., the principal corporate developer of MOTS-c-derived analogs (CB4211 / MBT-2 / MBT-3), discontinues its programs and dissolves; no MOTS-c product received FDA marketing authorization [fda_503a_lists]

2023 • Wan et al [wan2023]. (J Transl Med), comprehensive review of MOTS-c effects and mechanisms related to stress, metabolism, and aging; consolidates the AMPK-centered mechanistic model

Clinical Contexts for MOTS-C

Type 2 diabetes / obesity / insulin resistance PRECLINICAL

Evidence should be interpreted in context for MOTS-C. Any patient-specific request requires prescriber rationale and pharmacy review; availability is determined case by case.

The discovery paper [lee2015] established that intraperitoneal MOTS-c (5 mg/kg/day) in mice on a high-fat diet prevented weight gain, improved glucose tolerance, and improved insulin sensitivity, with the proposed mechanism mediated through AMPK activation. Li and colleagues [li2019] reported improvement of insulin resistance and oxidative-stress markers in a D-galactose accelerated-aging model in an AMPK-dependent fashion. Yin and colleagues [yin2022] reported amelioration of hyperglycemia and insulin resistance in a gestational-diabetes-mellitus mouse model. Cross-sectional human observational data report that circulating MOTS-c levels are lower in adults with obesity-related metabolic dysfunction, but causal direction has not been established. No randomized controlled trial of exogenous MOTS-c in adults with type 2 diabetes, obesity, or metabolic syndrome has been published.

Aging-related physical decline / sarcopenia / 'longevity' PRECLINICAL

Preclinical only, mouse model evidence on exercise-induced MOTS-c and age-related physical decline. Marketing claims in the consumer wellness market for human longevity outpace the evidence.

Reynolds and colleagues [reynolds2021] reported that MOTS-c is exercise-induced in mice and humans and that exogenous MOTS-c partially reversed age-related decline in physical performance and muscle homeostasis in older mice. Hyatt [hyatt2022] reported that long-term voluntary physical activity in rodents was associated with increased skeletal-muscle MOTS-c and that single-dose MOTS-c improved acute exercise performance after prolonged inactivity. Human observational work [dsouza2020, liu2019] shows age- and disease-related changes in muscle and circulating MOTS-c but does not establish a clinical-outcome link. The 'exercise-mimetic' and 'longevity-peptide' framing in the consumer market should be read as a preclinical hypothesis, not a demonstrated human effect.

Autoimmune type 1 diabetes PRECLINICAL

Preclinical only, non-obese diabetic (NOD) mouse model.

Kong and colleagues [kong2021] reported that MOTS-c attenuates pancreatic islet destruction in the NOD mouse model of autoimmune type 1 diabetes through effects on beta-cell function and apoptosis. No human clinical trial in type 1 diabetes has been conducted.

Postmenopausal bone loss / osteoporosis PRECLINICAL

Preclinical only, ovariectomized-mouse model.

Ming and colleagues [ming2016] reported that MOTS-c suppresses ovariectomy-induced bone loss in mice by inhibiting osteoclast differentiation through AMPK activation. No human clinical trial in postmenopausal osteoporosis or any other bone indication has been published.

Off-Label Uses of MOTS-C

Gray-market 'longevity' and 'mitochondrial health' use outside the regulated pharmacy framework PRECLINICAL

Evidence should be interpreted in context for MOTS-C. Any patient-specific request requires prescriber rationale and pharmacy review; availability is determined case by case.

Direct-to-consumer 'research peptide' retailers, anti-aging clinics, and longevity-focused wellness vendors market MOTS-c for 'mitochondrial health,' 'longevity,' 'metabolic optimization,' and exercise mimicry. These claims extrapolate directly from mouse-model preclinical findings to human outcomes; they are not supported by randomized controlled trial evidence. The supply chain for these products falls outside the regulated 503A framework and does not satisfy section 503A(b)(1)(A) of the Federal Food, Drug, and Cosmetic Act [fda_503a_statute, fda_503a_lists]. RonanRx does not participate in this market.

FDA-Approved Uses of MOTS-C

There are no FDA-approved MOTS-c products. No New Drug Application has been filed for MOTS-c or any of its analogs. CohBar Inc., founded around the Cohen-lab MDP intellectual property, developed several MOTS-c-derived analogs (CB4211, subsequently re-designated MBT-2 and MBT-3) and advanced CB4211 through phase 1 clinical evaluation in obesity / NASH and pulmonary indications. CohBar discontinued its development programs and dissolved in 2023. No CohBar product received FDA marketing authorization.

MOTS-C has no FDA approval in the United States. This ingredient is part of an evolving FDA review process. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case, and availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance.

Compounded MOTS-C (503A)

Physicians may submit patient-specific prescription requests for pharmacy review. For MOTS-C, certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case and may depend on patient-specific documentation, ingredient status, source qualification, formulation feasibility, state requirements, and pharmacist judgment. The review starts with the evidence constraint: The evidence base for MOTS-C is early. Most published work comes from cellular and mouse models of metabolism, insulin resistance, exercise signaling, and aging biology; the related CohBar analog program did not become an FDA-approved product.

This ingredient is part of an evolving FDA review process. RonanRx is monitoring FDA's PCAC process and any subsequent agency action. FDA has scheduled MOTS-C-related bulk drug substances for discussion at the 23-24 Jul 2026 Pharmacy Compounding Advisory Committee meeting. Availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance. For MOTS-C, RonanRx ties that monitoring to the evidence limits described above and to any patient-specific documentation submitted by the prescriber.

Valid patient-specific prescription required. Supporting clinical rationale may be requested. Compounded medications are not FDA-approved. No consumer self-ordering, no office stock, no bulk dispensing. Requests for MOTS-C are reviewed before any preparation is made or released. The patient-specific pharmacy pathway is the opposite of a wellness peptide menu. A prescriber must document why the request fits the patient, and the pharmacy must decide whether the preparation can be sourced, formulated, and released responsibly.

MOTS-C Formulations and Routes

Form	Concentration	Description
Research-grade lyophilized peptide (preclinical use only)	—	MOTS-c used in published preclinical work is supplied as a synthetic lyophilized 16-amino-acid peptide for reconstitution in saline or sterile water and parenteral administration to laboratory animals. There is no FDA-approved or pharmacy-compounded human formulation. RonanRx does not source, hold, or dispense MOTS-c API.
Gray-market 'research peptide' lyophilized vial (not pharmacy-compounded)	—	Lyophilized peptide sold outside the regulated pharmacy framework by 'research chemical' retailers. Content, purity, sterility, identity, and beyond-use dating are not verifiable to USP standards. RonanRx does not endorse or dispense these products.

Routes used in published literature: subcutaneous, intramuscular, intravenous.

MOTS-C Dosing

Route	Population	Range	Duration	Study type
Intraperitoneal (mouse model)	Mice, diet-induced obesity preclinical model	Approximately 5 mg/kg/day intraperitoneally in the original Lee et al. discovery paper [lee2015]	7-day to several-week regimens reported in preclinical studies	Preclinical mouse experiment (not a human dose)
Intraperitoneal or intramuscular (mouse model)	Mice, aging / exercise-mimetic preclinical model	Doses on the order of 0.5, 15 mg/kg reported across preclinical exercise and aging studies [reynolds2021, hyatt2022]	Single-dose and multi-week regimens reported	Preclinical rodent experiment (not a human dose)

There is no FDA-labeled dose for MOTS-c. No phase 2 or phase 3 human dose-finding has been published. The doses cited above are derived from preclinical mouse experiments and are listed only to document the literature; they should not be interpreted as human dosing guidance. RonanRx does not prescribe or compound MOTS-c [lee2015; reynolds2021].

Gray-market dosing protocols for human use circulating in longevity and anti-aging communities (typically 5, 10 mg subcutaneously two to three times per week) have no published controlled-trial evidence base and no published human safety characterization. RonanRx does not endorse these protocols.

MOTS-C Safety

Honest gap. No peer-reviewed systematic safety characterization of exogenous MOTS-c administration in humans has been published. Preclinical mouse studies [lee2015, reynolds2021, kim2018] report no overt drug-related toxicity at the doses tested, but the assessments are not designed as regulatory-grade safety evaluations and do not characterize long-term, reproductive, carcinogenicity, or immunogenicity risk. CohBar Inc. advanced the MOTS-c-derived analog CB4211 (subsequently re-designated MBT-2/MBT-3) through phase 1 clinical evaluation in obesity / NASH and pulmonary indications; CohBar discontinued its development programs and dissolved in 2023, and no peer-reviewed phase 1 safety report on a MOTS-c analog has been published. Theoretical safety considerations of sustained AMPK activation, including mild hypoglycemia, gastrointestinal effects, and uncharacterized long-term metabolic and immunologic consequences, are extrapolations from the broader AMPK-pathway pharmacology and have not been characterized for MOTS-c specifically. Gray-market preparations introduce additional quality, identity,

sterility, and dose-accuracy risks that RonanRx cannot verify. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

Searched: PubMed, FDA bulk substances 503A lists, ClinicalTrials.gov on 2026-05-11 · terms *MOTS-c safety; MOTS-c adverse events; MOTS-c clinical trial; CB4211 safety; MBT-2 MBT-3 CohBar*.

Contraindications

Honest gap. No FDA-labeled contraindications exist because MOTS-c has never been FDA-approved. No regulator-defined contraindication list has been published. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

Searched: PubMed, FDA bulk substances 503A lists on 2026-05-11 · terms *MOTS-c contraindications; MOTS-c warnings*.

Drug interactions

Honest gap. No MOTS-c-specific drug-interaction data have been published. Theoretical interaction concerns based on the AMPK-activation mechanism would include co-administration with other AMPK activators (metformin, AICAR-class agents) on grounds of additive pharmacology, but no controlled drug-interaction studies have been conducted. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

Searched: PubMed on 2026-05-11 · terms *MOTS-c drug interactions; MOTS-c metformin; mitochondrial-derived peptide pharmacokinetic interaction*.

Adverse events

Honest gap. No peer-reviewed systematic adverse-event characterization of exogenous MOTS-c administration in humans has been published. Preclinical mouse studies [lee2015, reynolds2021, kong2021, yin2022] report no overt drug-related adverse findings at the doses tested but were not designed as regulatory-grade safety assessments. Gray-market adverse-event reporting for MOTS-c is unsystematic and does not constitute regulated pharmacovigilance. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

Searched: PubMed, FDA Adverse Event Reporting System (FAERS), public dashboard on 2026-05-11 · terms *MOTS-c adverse events; CB4211 adverse events; mitochondrial peptide adverse events*.

↗ Monitoring MOTS-C Therapy

No RonanRx-specific monitoring protocol has been established for MOTS-C. If a patient-specific prescription is submitted, supporting clinical rationale may be requested, and monitoring expectations

would be reviewed case by case against the published evidence, route, sterile or nonsterile status, concomitant therapies, and patient risk factors.

MOTS-C in Special Populations

MOTS-C Evidence Quality

The MOTS-c evidence base is dominated by preclinical mouse and cell-culture work. The discovery paper [lee2015] and the principal mechanistic, glucose-homeostasis, exercise, aging, bone, and autoimmune-diabetes studies are mouse experiments. Human evidence is limited to small observational studies of circulating and skeletal-muscle MOTS-c levels in healthy adults and selected disease populations [dsouza2020, liu2019, yu2021] and to narrative reviews [lee2016, merry2020, wan2023]. No randomized placebo-controlled efficacy trials of exogenous MOTS-c administration in humans for any clinical indication have been published [kim2018; reynolds2021; ming2016].

Clinical translation has been limited. CohBar Inc. advanced the MOTS-c-derived analog CB4211 (MBT-2/MBT-3) through phase 1 clinical evaluation in obesity/NASH and pulmonary indications and wound down operations in 2022, 2023; no peer-reviewed phase 2 efficacy report on a MOTS-c analog has been published. Aging and longevity marketing claims in the consumer wellness market for native MOTS-c outpace the evidence base, which remains preclinical [li2019; kong2021; yin2022].

From a 503A perspective, MOTS-c is not on FDA's Category 1 bulk substances list and is part of an evolving FDA review process for 503A patient-specific compounding pending further FDA review [fda_503a_lists]. Section 503A(b)(1)(A) of the FD&C Act limits 503A bulk substances to those that are USP/NF monograph subjects, components of an FDA-approved drug, or on the Category 1 list [fda_503a_statute]; MOTS-c satisfies none of these criteria [hyatt2022]. RonanRx's position is that the current evidence posture does not support 503A compounding and that the appropriate response is informational documentation rather than dispensing.

Major MOTS-C Clinical Studies

Study	Design	Participants	Duration	Finding
Lee et al. (2015, Cell Metabolism), Discovery and metabolic phenotype	Preclinical discovery study; computational mitochondrial-genome screen for novel ORFs; in-vitro characterization in myotubes and adipocytes; in-vivo diet-	—	—	Identified MOTS-c as a 16-amino-acid mitochondrial-encoded peptide; demonstrated AMPK-pathway activation, AICAR

Study	Design	Participants	Duration	Finding
	induced-obesity mouse experiment with intraperitoneal MOTS-c			accumulation, and prevention of high-fat-diet-induced weight gain and insulin resistance in mice, the foundational MOTS-c paper [lee2015]
Kim et al. (2018, Cell Metabolism), Mito-nuclear retrograde signaling	Preclinical mechanistic study in cultured cells using subcellular fractionation, immunofluorescence, mass spectrometry, and metabolic-stress challenges	—	—	Demonstrated that MOTS-c translocates from mitochondria to the nucleus under metabolic stress and modulates nuclear gene expression, first demonstration of mitochondrial-to-nuclear retrograde signaling by an MDP [kim2018]
Reynolds et al. (2021, Nature Communications), Exercise-induced regulator of physical decline	Preclinical study in mice with translational human cohort component; exercise-induced MOTS-c measurements; exogenous MOTS-c administration in older mice with physical-performance endpoints	—	—	MOTS-c is exercise-induced in mice and humans; exogenous MOTS-c partially rescued age-dependent physical decline and muscle homeostasis deficits in older mice, primary preclinical 'exercise-mimetic' evidence [reynolds2021]
Lee, Kim, and Cohen (2016, Free Radic Biol Med), Review	Narrative review of MOTS-c discovery and early metabolic characterization	—	—	Consolidated the 2015 discovery and subsequent mechanistic work for a broader free-radical-biology and metabolism audience [lee2016]
Ming et al. (2016, Biochem Biophys Res Commun), Ovariectomy bone loss	Preclinical mouse study, ovariectomized C57BL/6 mice receiving MOTS-c with bone-density and histomorphometric endpoints	—	—	MOTS-c suppressed ovariectomy-induced bone loss through inhibition of osteoclast differentiation in an

Study	Design	Participants	Duration	Finding
				AMPK-dependent manner [ming2016]
Li et al. (2019, Biochem Biophys Res Commun), D-galactose accelerated aging	Preclinical mouse study, D-galactose-induced accelerated-aging model with MOTS-c intervention; insulin resistance and oxidative-stress endpoints	—	—	MOTS-c improved insulin resistance and oxidative-stress markers in D-galactose-treated mice in an AMPK-dependent manner [li2019]
Kong et al. (2021, Cell Reports), Autoimmune diabetes (NOD mouse)	Preclinical study in the non-obese diabetic (NOD) mouse model of autoimmune type 1 diabetes; MOTS-c intervention with islet-histology and glucose-tolerance endpoints	—	—	MOTS-c prevented pancreatic islet destruction in NOD mice through effects on beta-cell function and apoptosis, preclinical proof-of-concept in autoimmune diabetes [kong2021]
Yin et al. (2022, Pharmacological Research), Gestational diabetes	Preclinical mouse study, gestational-diabetes-mellitus model with MOTS-c intervention; hyperglycemia and insulin-resistance endpoints	—	—	MOTS-c relieved hyperglycemia and insulin resistance in a gestational-diabetes-mellitus mouse model [yin2022]
Hyatt (2022, Physiological Reports), Long-term activity and acute performance	Preclinical rodent study, long-term voluntary physical activity with MOTS-c skeletal-muscle measurements; single-dose acute exercise performance after prolonged inactivity	—	—	MOTS-c increased in skeletal muscle following long-term physical activity; a single dose improved acute exercise performance after prolonged inactivity [hyatt2022]
D'Souza et al. (2020, Aging), Healthy aging skeletal muscle	Human cross-sectional observational study, skeletal-muscle MOTS-c expression in young, middle-aged, and older healthy men with myofiber-composition correlates	—	—	Skeletal-muscle MOTS-c expression increased with age and correlated with myofiber composition; circulating MOTS-c showed the opposite age-related trend [dsouza2020]

Study	Design	Participants	Duration	Finding
Liu et al. (2019, Am J Physiol Renal Physiol), Chronic kidney disease	Human observational study, skeletal-muscle MOTS-c, humanin, and Nrf2 expression in adults with chronic kidney disease vs controls	—	—	Reduced muscle MOTS-c and humanin expression in chronic kidney disease vs controls; positions MDPs as disease-state biomarkers in CKD [liu2019]
Yu et al. (2021, Mitochondrion), Aged placental MSCs in vitro	In-vitro human-tissue study, MOTS-c effects on aged human placenta-derived mesenchymal stem cells	—	—	MOTS-c promoted homeostasis in aged human placenta-derived mesenchymal stem cells in vitro, translational signal in primary human cells (not a clinical trial) [yu2021]
Merry et al. (2020, Am J Physiol Endocrinol Metab), Mitochondrial-derived peptide review	Review article on mitochondrial-derived peptides in human energy metabolism	—	—	Consolidated MDP biology including MOTS-c, humanin, and SHLPs in the context of human energy metabolism and metabolic disease [merry2020]
Wan et al. (2023, J Transl Med), Stress, metabolism, and aging review	Comprehensive review article on MOTS-c effects and mechanisms	—	—	Consolidated the AMPK-centered mechanism across stress, metabolism, and aging contexts; representative of the current state of the preclinical evidence base [wan2023]

MOTS-C Pharmacokinetics & Pharmacodynamics

Pharmacokinetics

There are no publicly available regulatory-grade pharmacokinetic data on exogenous MOTS-c administration in humans. Preclinical work in mice describes parenteral administration (typically intraperitoneal) with rapid clearance characteristic of a small unmodified peptide; specific half-life and

bioavailability values vary across studies and have not been standardized [lee2015, reynolds2021]. Endogenous MOTS-c is detectable in human plasma and tissue by ELISA and targeted mass spectrometry, with limited cross-assay standardization across published reports.

Modified analogs developed by CohBar (CB4211 / MBT-2 / MBT-3) were engineered with stability modifications intended to improve pharmacokinetic profile relative to the native 16-amino-acid peptide; no peer-reviewed human PK data on these analogs are publicly available. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

Pharmacodynamics

Pharmacodynamic effects of MOTS-c in preclinical models include AMPK Thr172 phosphorylation, increased glucose uptake in myotubes and adipocytes, AICAR accumulation via folate-cycle modulation, mitochondrial-to-nuclear translocation under metabolic stress, and modulation of stress-responsive nuclear gene expression [lee2015, kim2018] [reynolds2021]. Downstream phenotypes in mouse models include reduced weight gain on a high-fat diet, improved insulin sensitivity, increased exercise performance in older animals, suppression of ovariectomy-induced bone loss, and reduced beta-cell destruction in autoimmune diabetes [ming2016; kong2021; yin2022].

No controlled clinical pharmacodynamic endpoint has been characterized for exogenous MOTS-c administration in humans [li2019]. The mouse-model findings should not be assumed to translate to human clinical effect.

↓↑ Comparing MOTS-C Formulations

MOTS-c is one of the better-characterized members of the mitochondrial-derived peptide (MDP) family, which also includes humanin (encoded within the mitochondrial 16S rRNA, ~24 amino acids) and the SHLP1, 6 family [merry2020]. Across the MDP family, humanin has the longest history and the broadest preclinical literature; MOTS-c has the most published evidence for AMPK-pathway metabolic effects. None of the MDPs has an FDA-approved human product, and none currently appears on FDA's Category 1 list for 503A compounding [fda_503a_lists, fda_503a_statute].

If a MOTS-C preparation is approved after pharmacy review, RonanRx applies source documentation, formulation records, lot traceability, release checks, and storage controls appropriate to the actual dosage form. Research-use vial storage practices do not substitute for pharmacy-assigned storage, beyond-use dating, sterility controls when applicable, or recallable batch records.

MOTS-C Storage and Handling

MOTS-c is not commercially manufactured for human use and is not compounded by RonanRx; there is therefore no RonanRx-specific storage guidance. Research-grade lyophilized peptide used in preclinical

work is typically stored at −20 °C or −80 °C in sealed vials with desiccant and reconstituted with sterile saline or water immediately prior to use. Gray-market lyophilized peptide preparations sold outside the regulated supply chain have variable stability and identity and should not be assumed to behave like a regulated drug product.

MOTS-C Compounding & Operations

503A compounding

Physicians may submit patient-specific prescription requests for pharmacy review. For MOTS-C, certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case and may depend on patient-specific documentation, ingredient status, source qualification, formulation feasibility, state requirements, and pharmacist judgment. The review starts with the evidence constraint: The evidence base for MOTS-C is early. Most published work comes from cellular and mouse models of metabolism, insulin resistance, exercise signaling, and aging biology; the related CohBar analog program did not become an FDA-approved product.

This ingredient is part of an evolving FDA review process. RonanRx is monitoring FDA's PCAC process and any subsequent agency action. FDA has scheduled MOTS-C-related bulk drug substances for discussion at the 23-24 Jul 2026 Pharmacy Compounding Advisory Committee meeting. Availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance. For MOTS-C, RonanRx ties that monitoring to the evidence limits described above and to any patient-specific documentation submitted by the prescriber.

Valid patient-specific prescription required. Supporting clinical rationale may be requested. Compounded medications are not FDA-approved. No consumer self-ordering, no office stock, no bulk dispensing. Requests for MOTS-C are reviewed before any preparation is made or released. The patient-specific pharmacy pathway is the opposite of a wellness peptide menu. A prescriber must document why the request fits the patient, and the pharmacy must decide whether the preparation can be sourced, formulated, and released responsibly.

Pharmacist review

For MOTS-C, the pharmacist review starts before any preparation is made. Valid patient-specific prescription required. Supporting clinical rationale may be requested. The pharmacist reviews ingredient status, sourcing, formulation feasibility, state requirements, patient-specific documentation, and whether dispensing is appropriate case by case.

Quality and traceability

If a MOTS-C preparation is approved after pharmacy review, RonanRx applies source documentation, formulation records, lot traceability, release checks, and storage controls appropriate to the actual dosage form. Research-use vial storage practices do not substitute for pharmacy-assigned storage, beyond-use dating, sterility controls when applicable, or recallable batch records. The patient-specific framework and quality controls are documented in the cited compounding references [usp_797].

Cold chain

If a MOTS-C preparation is approved after pharmacy review, RonanRx applies source documentation, formulation records, lot traceability, release checks, and storage controls appropriate to the actual dosage form. Research-use vial storage practices do not substitute for pharmacy-assigned storage, beyond-use dating, sterility controls when applicable, or recallable batch records.

Frequently Asked Questions About MOTS-C

Can physicians request MOTS-C through RonanRx?

Physicians may submit patient-specific prescription requests for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case. Compounded medications are not FDA-approved, and no consumer self-ordering, office stock, or bulk dispensing is offered.

What is MOTS-c?

MOTS-c is a 16-amino-acid peptide encoded within the human mitochondrial 12S ribosomal RNA gene. It was identified by the Cohen laboratory at the University of Southern California and reported by Lee and colleagues in Cell Metabolism in 2015. In mouse models, MOTS-c activates AMP-activated protein kinase (AMPK), improves insulin sensitivity on a high-fat diet, and translocates to the cell nucleus under metabolic stress [lee2015; kim2018].

What evidence exists for MOTS-c in humans?

Almost none. The published evidence base is dominated by mouse and cell-culture work. Human evidence is limited to small cross-sectional observational studies of MOTS-c levels in plasma and skeletal muscle in healthy adults and in chronic kidney disease, and in-vitro studies on human placental mesenchymal stem cells [reynolds2021; dsouza2020; liu2019]. No randomized placebo-controlled efficacy or safety trials of exogenous MOTS-c administration in humans for any clinical indication have been published [lee2015] [yu2021].

Is MOTS-c FDA-approved?

No. There is no FDA-approved MOTS-c product. CohBar Inc. developed several MOTS-c-derived analogs (CB4211, subsequently re-designated MBT-2 and MBT-3) through phase 1 clinical evaluation in obesity / NASH and pulmonary indications, but discontinued its programs and dissolved in 2023. No CohBar product received FDA marketing authorization [fda_503a_lists].

Why do longevity clinics market MOTS-c if there's no human evidence?

The marketing extrapolates directly from mouse-model findings on AMPK activation, exercise-induced upregulation, and age-related decline in circulating peptide levels, presenting these as a human longevity story [reynolds2021; dsouza2020]. The extrapolation is not supported by controlled clinical evidence and is well outside the regulated 503A pharmacy framework. RonanRx flags this honestly and does not participate in this market [fda_503a_lists].

What is the relationship between MOTS-c, AMPK, and metformin?

Both MOTS-c (in preclinical models) and metformin (FDA-approved) act in part through AMPK activation. The mechanisms differ, metformin's effects on hepatic glucose production and AMPK activation are well-characterized in human clinical use, while MOTS-c's AMPK-pathway effects are characterized in mouse and cell-culture work. The two molecules are not pharmacologically interchangeable, and the human evidence base for metformin is incomparable in size to the human evidence base for MOTS-c [lee2015; li2019].

Could MOTS-c become 503A-eligible in the future?

FDA reviews 503A bulk substances on an ongoing basis as new safety and efficacy data become available [fda_503a_lists]. A move from current under-review status to Category 1 placement would require a substantially expanded human safety database, which does not exist today and is unlikely to be generated outside a formal regulatory pathway such as an IND-supported clinical trial. RonanRx will reassess MOTS-c if and when FDA changes its determination [fda_503a_statute].

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How to Access MOTS-C

Compounded MOTS-C is dispensed under 503A on a patient-specific prescription. Depending on your role, the next step looks different.

FOR PRESCRIBING CLINICIANS

Offer this medication

A pharmacist will follow up within two business days. We'll cover state availability, supported formulations, and what integration looks like for your clinic.

ronanrx.com/request-partnership-call

PATIENT WITH A DOCTOR

Receive your prescription

If your doctor has prescribed MOTS-C, sign up so we can prepare and ship your medication. The signup wizard collects intake and connects you to the prescribing workflow.

ronanrx.com/patients

PATIENT WITHOUT A DOCTOR

Find a partner clinic

RonanRx prescribes through partner clinics — we don't initiate prescriptions on this site. Read how the referral process works and how to find a partner clinic in your state.

ronanrx.com/find-clinic

Other compounds RonanRx makes

This monograph is one of many in the RonanRx formulary. Every compound below is prepared under 503A on a patient-specific prescription. Browse the full catalog at ronanrx.com/medications and ronanrx.com/peptides, or scan the codes at right for each index.

Medications

Peptides

MEDICATIONS (40)

Alpha-Lipoic Acid (ALA) — Antioxidant & mitochondrial
Coenzyme Q10 (CoQ10) — Antioxidant & mitochondrial
Glutathione — Antioxidant & mitochondrial
NAD+ / NMN — Antioxidant & mitochondrial
Compounded Topical Anesthetics (BLT, LET) — Dermatology
Topical Minoxidil — Dermatology
Topical Tretinoin — Dermatology
Compounded Magnesium — Energy & nutritional
Cyanocobalamin — Energy & nutritional
High-Dose Vitamin D — Energy & nutritional
Hydroxocobalamin — Energy & nutritional
Iron (Compounded) — Energy & nutritional
L-Carnitine — Energy & nutritional
Methylcobalamin (B12) — Energy & nutritional
Methylfolate — Energy & nutritional
Anastrozole — Hormone optimization
Clomiphene & Enclomiphene — Hormone optimization
DHEA — Hormone optimization
Estradiol — Hormone optimization
Estriol — Hormone optimization
Human Chorionic Gonadotropin (HCG) — Hormone optimization
Pregnenolone — Hormone optimization
Progesterone — Hormone optimization
Testosterone — Hormone optimization
Compounded Metformin — Metabolic & weight
Compounded Semaglutide — Metabolic & weight
Compounded Tirzepatide — Metabolic & weight
Lipotropic Injection (MIC, MICC) — Metabolic & weight
Low-Dose Naltrexone (LDN) — Metabolic & weight
Naltrexone-Bupropion Combination — Metabolic & weight
Topiramate — Metabolic & weight
Bremelanotide / PT-141 — Sexual health
Compounded Sildenafil — Sexual health
Compounded Tadalafil — Sexual health
Trimix Injection — Sexual health
Compounded Gabapentin — Sleep & recovery
Compounded Melatonin — Sleep & recovery
Compounded T3 (Liothyronine) — Thyroid
Compounded T3/T4 Combinations — Thyroid
Compounded T4 (Levothyroxine) — Thyroid

PEPTIDES (21)

Sermorelin — Available now

Tesamorelin — Available now

AOD-9604 — Growth-hormone axis (under FDA review)

CJC-1295 — Growth-hormone axis (under FDA review)

GHRP-2 / GHRP-6 — Growth-hormone axis (under FDA review)

Hexarelin — Growth-hormone axis (under FDA review)

Ipamorelin — Growth-hormone axis (under FDA review)

MK-677 / Ibutamoren — Growth-hormone axis (under FDA review)

5-Amino 1MQ — Metabolic & longevity (under FDA review)

Epitalon / Epithalon — Metabolic & longevity (under FDA review)

MOTS-C — Metabolic & longevity (under FDA review)

Thymosin Alpha-1 / Thymalin — Metabolic & longevity (under FDA review)

DSIP, Delta Sleep-Inducing Peptide — Neuro & cognitive (under FDA review)

Selank — Neuro & cognitive (under FDA review)

Semax — Neuro & cognitive (under FDA review)

Vasoactive Intestinal Peptide (VIP) — Neuro & cognitive (under FDA review)

BPC-157 — Tissue repair (under FDA review)

KPV — Tissue repair (under FDA review)

LL-37 — Tissue repair (under FDA review)

Pentadeca Arginate (PDA) — Tissue repair (under FDA review)

TB-500 / Thymosin Beta-4 — Tissue repair (under FDA review)