



PEPTIDE CATALOG

TAILOR MADE COMPOUNDING

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OUR STORY



Tailor Made Compounding was launched in the U.S. in January of 2016. We have since become a major contributor to peptide medicines in the integrative health space.

Through our compounding expertise, knowledge and experience, we have quickly grown as one of the top compounding pharmacies in the nation. Together, with our sister pharmacies around the world, we have established a reputation for working closely with our physicians and are well known for obtaining difficult to source and hard to compound pharmaceuticals. Our sister facility and first establishment, Como compounding in Melbourne, has been working with peptide therapies for over a decade. After witnessing the success these products were having for both doctors and patients in Australia, we saw the demand to bring our compounding expertise to the

United States. Since the inception of TMC, we have sourced and compounded over 40 unique peptides. We are proud to be the first in the United States to offer compounds like CJC 1295, Bremelanotide, PT 141, BPC-157, and Epitalon and continue to introduce new and exciting compounds to our formulary.

At Tailor Made, we pride ourselves in being partners with our practitioners and their staff. That's why a major part of what we do involves educating our prescribers on the products we develop, including their clinical indications, and dosing. Through building these relationships and creating cutting-edge peptide therapies, we promise to maintain our status as leaders in the industry. Collectively, all of our locations work to support the same mission; To positively disrupt medicine using innovative technologies through education and partnership.

*Positively disrupting
medicine using
innovative technology*



PEPTIDE

FACT SHEETS

At Tailor Made Compounding, we want you to have all the required information to understand the products we offer. Our peptide fact sheets contain a description, protocol, and clinical research for each product.

If you have any further questions please reach out to us:

Phone: 1 859 887 0013

Email: admin@tailormadecompounding.com

3-Desoxy DHEA

Purity: >98% (HPLC on request) | Molecular Formula: R1=H, R2=CH3
Molecular Weight: 288.43 g/mol | Sequence: Non-Peptide

DESCRIPTION:

3-Desoxy DHEA is a competitive aromatase inhibitor for use in controlling estrogen and increasing endogenous testosterone production. This compound is shown to potently reduce aromatase activity through binding to the enzyme and blocking access to endogenous estrogen precursors (androstenedione, testosterone). The competitive inhibition offers a more short term solution to estrogen control, as

aromatase inhibition only occurs while 3-Desoxy DHEA is present in the body. 3-Desoxy DHEA is a relatively quickly metabolized compound and has an excellent potency with IC50 and Ki values. 3-Desoxy DHEA offers you a new option for estrogen control and natural testosterone elevation along with a high potency coupled with better dose control.

PROTOCOL:

Content & Potency: 100mg capsules provided in quantity of 30.
Suggested dosage: Take 1 capsule by mouth once daily.

CLINICAL RESEARCH:

What is desoxy-DHEA and How Does it Work?

Although Desoxy DHEA is an effective competitive inhibitor of aromatase it is not nearly as potent as SERMs or other steroidal and non-steroidal suicide inhibitors. The structure- activity relationships mentioned are not comprehensive as there undoubtedly exists additional structural manipulations that occur to further enhance aromatase activity. Therefore, it is NOT an appropriate replacement to prescription compounds when heavy aromatase inhibition is required. It does however, offer a cost-effective, safe, and legal method of estrogen management as validated by independent lab tests and

selected in vitro data. Competitive aromatase inhibition works differently than suicide substrate inhibition, which is the mode of action through which other over the counter aromatase inhibitors such as 6-OXO and ATD work. Competitive inhibition offers a more short term solution to estrogen control, as aromatase inhibition only occurs while Desoxy DHEA is present in the body. Desoxy DHEA is a relatively quickly metabolized compound. So with Desoxy DHEA Tailor Made offers you a new option for estrogen control.

Ben Esgro, BS,CSCS,CISS

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



5-amino-1MQ

Purity: >98% (HPLC on request) | Molecular Formula: C10H11N2
Molecular Weight: 159.21 g/mol | Sequence: Non-Peptide

DESCRIPTION:

5-amino-1MQ is a small, selective, membrane permeable molecule that is an inhibitor of nicotinamide N-methyltransferase (NNMT), a cytosolic enzyme that plays a role in cellular metabolism and energy homeostasis. NNMT has been found to be up-regulated in white adipose tissue of mice compared to other tissues, thus, NNMT is an ideal target for anti-obesity medication. 5-amino-1MQ is a derivative of methylquinolinium (MQ) which has exhibited a high efficacy in NNMT inhibition, cell viability, and membrane permeability. It did not have any effect on the activity of any other enzymes in the relevant metabolic cycles, therefore reducing the risks of potential side effects. This NNMT inhibitor could be used

to prevent adipogenesis and type II diabetes and reverse diet-induced obesity as a result of increased intracellular NAD+ and SAM. In a study using diet-induced obese mice fed a high fat diet, the effects of 5-amino-1MQ on obesity measures and plasma lipid were evaluated. After 11 days, mice treated with 5-amino-1MQ lost weight, exhibited a decrease in white adipose mass and cholesterol levels, and displayed reduced lipogenesis. The results of this study validate NNMT as a practical target to treat obesity and related metabolic conditions and support the development of an NNMT inhibitor therapeutics to reverse diet-induced obesity.

PROTOCOL:

Content & Potency: 10ml at 300mg/ml
Suggested dosage: 150mcg (.5ml) daily for 20 days 30 minutes after oral administration of NAD+ precursor

CLINICAL RESEARCH:

Selective and membrane-permeable small molecule inhibitors of nicotinamide N-methyltransferase reverse high fat diet-induced obesity in mice

There is a critical need for new mechanism-of-action drugs that reduce the burden of obesity and associated chronic metabolic comorbidities. A potentially novel target to treat obesity and type 2 diabetes is nicotinamide-N-methyltransferase (NNMT), a cytosolic enzyme with newly identified roles in cellular metabolism and energy homeostasis. To validate NNMT as an anti-obesity drug target, we investigated the permeability, selectivity, mechanistic, and physiological properties of a series of small molecule NNMT inhibitors. Membrane permeability of NNMT inhibitors was characterized using parallel artificial membrane permeability and Caco-2 cell assays. Selectivity was tested against structurally-related methyltransferases and nicotinamide adenine dinucleotide (NAD+) salvage pathway enzymes. Effects of NNMT inhibitors on lipogenesis and intracellular levels of metabolites, including NNMT reaction product 1-methylnicotinamide (1-MNA) were evaluated in cultured adipocytes. Effects of a potent NNMT inhibitor on obesity measures and plasma lipid were assessed in diet-induced obese mice fed a high-fat diet. Methylquinolinium scaffolds with

primary amine substitutions displayed high permeability from passive and active transport across membranes. Importantly, methylquinolinium analogues displayed high selectivity, not inhibiting related SAM-dependent methyltransferases or enzymes in the NAD+ salvage pathway. NNMT inhibitors reduced intracellular 1-MNA, increased intracellular NAD+ and S-(5'-adenosyl)-L-methionine (SAM), and suppressed lipogenesis in adipocytes. Treatment of diet-induced obese mice systemically with a potent NNMT inhibitor significantly reduced body weight and white adipose mass, decreased adipocyte size, and lowered plasma total cholesterol levels. Notably, administration of NNMT inhibitors did not impact total food intake nor produce any observable adverse effects. These results support development of small molecule NNMT inhibitors as therapeutics to reverse diet-induced obesity and validate NNMT as a viable target to treat obesity and related metabolic conditions. Increased flux of key cellular energy regulators, including NAD+ and SAM, may potentially define the therapeutic mechanism-of-action of NNMT inhibitors.

Harshini Neelakantan, Virginia Vance, Michael D. Wetzel, Hua-Yu Leo Wang, Stanton F. McHardy, Celeste C. Finnerty, Jonathan D. Hommel, Stanley J. Watowich

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Amlexanox

Purity: >98% (HPLC on request) | Molecular Formula: C₁₆H₁₄N₂O₄
Molecular Weight: 298.3 g/mol | Sequence: Non-Peptide

DESCRIPTION:

Amlexanox is an anti-inflammatory and anti-allergic compound which has traditionally been used to treat ulcers by reducing healing time and pain. It has multiple mechanisms of action such as inhibiting inflammation by inhibiting the release of histamine and leukotrienes. It has been

shown to selectively inhibit TBK1 and IKK-ε, producing reversible weight loss and improved insulin sensitivity. It is through this mechanism that it has produced substantial results in terms of reducing HbA1C levels and increase insulin sensitivity.

PROTOCOL:

Content & Potency: 40mg capsules provided in a quantity of 90.
Suggested dosage: Take one capsule by mouth 3 times daily.

CLINICAL RESEARCH:

Inhibition of IKKε and TBK1 improves glucose control in a subset of patients with type 2 diabetes.

Numerous studies indicate an inflammatory link between obesity and type 2 diabetes. The inflammatory kinases IKKε and TBK1 are elevated in obesity; their inhibition in obese mice reduces weight, insulin resistance, fatty liver and inflammation. Here we studied amlexanox, an inhibitor of IKKε/TBK1, in a proof-of-concept randomized, double blind, placebo-controlled study of 42 obese patients with type 2 diabetes and nonalcoholic fatty liver disease. Treatment of patients with amlexanox produced a statistically significant reduction in Hemoglobin A1c and

fructosamine. Interestingly, a subset of drug responders also exhibited improvements in insulin sensitivity and hepatic steatosis. This subgroup was characterized by a distinct inflammatory gene expression signature from biopsied subcutaneous fat at baseline. They also exhibited a unique pattern of gene expression changes in response to amlexanox, consistent with increased energy expenditure. Together, these data suggest that IKKε/TBK1 inhibitors may be effective therapies for metabolic disease in an identifiable subset of patients.

Oral, Elif A et al. "Inhibition of IKKε and TBK1 Improves Glucose Control in a Subset of Patients with Type 2 Diabetes." Cell metabolism 26 1 (2017): 157-170.e7 .

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Ammonium Tetrathiomolybdate (TBM)

Purity: >98% (HPLC on request) | Molecular Formula: H₈N₂M₀S₄
Molecular Weight: 260.28 g/mol | Sequence: Non-Peptide

DESCRIPTION:

Ammonium tetrathiomolybdate (TM) was developed as a non-toxic treatment for Wilson's disease, which is a condition that results in copper buildup in the body. Tetrathiomolybdate binds both food copper and endogenously produced copper and prevents their absorption when taken with food. When taken without food it enters the blood and binds with available copper to prevent it being used by cells. Tetrathiomolybdate has also shown to be a

promising treatment for cancer. Copper is involved in turning on the growth of new blood vessels that tumors depend on for growth. By depriving the tumors of the copper supply that is needed for new blood vessels, the growth may be slowed or stabilized. It has also been shown to target the copper transporter ATP7A and enhance the sensitivity of breast cancer to Cisplatin treatment, as well as, decreasing the development of resistance to cisplatin.

PROTOCOL:

Content & Potency: 40mg capsules provided in a quantity of 90.
Suggested dosage: Take one capsule by mouth three times daily between meals.

CLINICAL RESEARCH:

Ammonium Tetrathiomolybdate treatment targets the copper transporter ATP7A and enhances sensitivity of breast cancer to cisplatin.

Cisplatin is an effective breast cancer drug but resistance often develops over prolonged chemotherapy. Therefore, we performed a candidate approach RNAi screen in combination with cisplatin treatment to identify molecular pathways conferring survival advantages. The screen identified ATP7A as a therapeutic target. ATP7A is a copper ATPase transporter responsible for intercellular movement and sequestering of cisplatin. Pharmaceutical replacement for ATP7A by ammonium tetrathiomolybdate (TM) enhanced cisplatin treatment in breast cancer cells. Allograft and xenograft models in athymic nude mice treated with cisplatin/TM exhibited retarded tumor growth, reduced accumulation of cancer stem cells and

decreased cell proliferation as compared to mono-treatment with cisplatin or TM. Cisplatin/TM treatment of cisplatin-resistant tumors reduced ATP7A protein levels, attenuated cisplatin sequestering by ATP7A, increased nuclear availability of cisplatin, and subsequently enhanced DNA damage and apoptosis. Microarray analysis of gene ontology pathways that responded uniquely to cisplatin/TM double treatment depicted changes in cell cycle regulation, specifically in the G1/S transition. These findings offer the potential to combat platinum-resistant tumors and sensitize patients to conventional breast cancer treatment by identifying and targeting the resistant tumors' unique molecular adaptations.

Chisholm, Cristine & Wang, Haitao & Hang-Heng Wong, Ada & Vazquez-Ortiz, Guelaguetza & Chen, Weiping & Xu, Xiaoling & Deng, Chu-Xia. (2016). Ammonium tetrathiomolybdate treatment targets the copper transporter ATP7A and enhances sensitivity of breast cancer to cisplatin. Oncotarget.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Aniracetam

Purity: >98% (HPLC on request) | Molecular Formula: C₁₂H₁₃NO₃
Molecular Weight: 219.237 g/mol | Sequence: Non-Peptide

DESCRIPTION:

Aniracetam employs a similar method of action to other racetams. It has been shown to specifically stimulate the AMPA receptor site. The AMPA receptor is the most common glutamate-activated receptor associated with the Central Nervous System and its functions. AMPA receptors play a role in learning and memory formation.

Aniracetam seems to have a higher affinity with the AMPA receptors than other racemic compounds. Another interesting action of Aniracetam is the observed anxiety- reducing

effects. It completes this action without causing sedation and the anxiolytic benefit of the substance has been extensively studied in animal models. This anxiolytic response is believed to be caused in part, by activation of the D2 and D3 dopamine receptors. Nicotinic Ach receptor activation is also believed to contribute to anxiolytic effects and nootropic effects as well. Additionally, Aniracetam seems to enact on the 5-HTP(2a) receptor which helps to process Serotonin and may further advance anxiolytic/ antidepressant functions.

PROTOCOL:

Content & Potency: 375mg capsules provided in a quantity of 60.
Suggested dosage: Take two capsules by mouth with food.

CLINICAL RESEARCH:

Clinical Efficacy of Aniracetam, Either as Monotherapy or Combined with Cholinesterase Inhibitors, in Patients with Cognitive Impairment: A Comparative Open Study

In the present study, we aimed to evaluate the efficacy of aniracetam, either as monotherapy or combined with cholinesterase inhibitors (ChEIs), in terms of several neuropsychological parameters, in a considerable number of patients with dementia. In our prospective, open-label study, we enrolled a total of 276 patients (mean age 71 ± 8 years, 95 males) with cognitive disorders. Our study population comprised four groups: no treatment group (n = 75), aniracetam monotherapy group (n = 58), ChEIs monotherapy group (n = 68), and group of combined treatment (n = 68). Patients were examined with validated neuropsychological tests at baseline, 3, 6, and 12 months of treatment. In patients treated with aniracetam, all studied parameters were adequately maintained at 6 and 12 months, while emotional state was significantly improved at 3 months. In patients treated with ChEIs, we observed a significant cognitive deterioration at 12 months. The

comparison between aniracetam and ChEIs in patients with relatively mild dementia (15 ≤ MMSE ≤ 25) revealed a significantly better cognitive performance with aniracetam at 6 months and improved functionality at 3 months. Comparing aniracetam monotherapy with combined treatment in the same population, aniracetam performed better in the cognitive scale at 6 months, and displayed a notable tendency for enhanced mood at 12 months and improved functionality at 6 months. **Conclusions:** Our findings indicate that aniracetam (a nootropic compound with glutamatergic activity and neuroprotective potential) is a promising option for patients with cognitive deficit of mild severity. It preserved all neuropsychological parameters for at least 12 months, and seemed to exert a favorable effect on emotional stability of demented patients.

Koliaki, C. C., Messini, C. and Tsolaki, M. (2012), Clinical Efficacy of Aniracetam, Either as Monotherapy or Combined with Cholinesterase Inhibitors, in Patients with Cognitive Impairment: A Comparative Open Study*. CNS Neuroscience & Therapeutics, 18: 302-312.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

AOD 9604

Purity: >98% (HPLC on request) | Molecular Formula: C₇₈H₁₂₃N₂₃O₂₃S₂
Molar Weight: 1815.1 | Sequence: Tyr-Leu-Arg-Ile-Val-Gln-Cys-Arg-Ser-Val-Glu-Gly-Ser-Cys-Gly-Phe

DESCRIPTION:

AOD 9604 is a modified form of amino acids 176-191 of the GH proteins. Investigators at Monash University discovered that the fat-reducing effects of GH appear to be controlled by a small region near one end of the GH molecule. This region, which consists of amino acids 176-191, is less than 10% of the total size of the GH molecule and appears to have no effect on growth or insulin resistance. This hypothesis was proven in animals to a tremendous degree with specimen losing a significant

amount of fat mass. However, in phase three clinical trials the peptide didn't mean its confidence interval. Instead, it is now being studied for its effect on bone and cartilage. AOD 9604 possesses many other regenerative properties associated with growth hormone. Currently, trials are underway to show the application of AOD 9604 in osteoarthritis, Hypercholesterolemia, bone and cartilage repair & pain. AOD 9604 has an excellent safety profile, recently obtaining Human GRAS status in the USA.

PROTOCOL:

Content & Potency: 1200mcg/ml subcutaneous injectable provided in a 5ml vial.
Suggested dosage: Inject 0.25ml subcutaneously once daily for 20 days.

CLINICAL RESEARCH:

Safety and Tolerability of the Hexadecapeptide AOD 9604 in humans

Background: The human growth hormone (hGH) has fat loss properties making it a potential candidate to treat obesity. AOD 9604 is a peptide fragment of the C-terminus of hGH (Tyr-hGH177-191), which harbors the fat reducing activity of hGH, without its negative effects. In this paper the safety data of AOD 9604 obtained in clinical trials are summarized.

Methods: Six randomized, double-blind, placebo-controlled trials were performed with AOD 9604. Special focus was given to undesired effects associated with hGH treatment: increases in IGF-1 levels, insulin resistance, and impaired glucose tolerance. Blood samples were analyzed for presence of antiAOD 9604 antibodies to exclude immunogenicity.

Results: AOD 9604 had no effect on serum IGF-1 levels, which confirms the hypothesis that AOD 9604 does not act via IGF-1. Results of oral glucose tolerance test demonstrated that, in contrast with hGH, AOD 9604 has no negative effect on carbohydrate metabolism. There were no anti-AOD 9604 antibodies detected in any of the patients selected for antibody assay. In none of the studies did a withdrawal or serious adverse event occur related to intake of AOD 9604.

Conclusion: AOD 9604 displayed a very good safety and tolerability profile indistinguishable from placebo. AOD 9604 did not result in any of the adverse effects associated with full-length hGH treatment

Stiera, Heike et al. "Safety and Tolerability of the Hexadecapeptide AOD 9604 in Humans." (2013).

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

AOD 9604 + HA

Purity: >98% (HPLC on request) | Molecular Formula: C₇₈H₁₂₃N₂₃O₂₃S₂
Molecular Weight: 1815.1 g/mol | Sequence: Ac-Ser-Asp-Lys-Pro-Asp-Met-Ala-Glu-Ile-Glu-Lys-Phe-Asp-Lys-Ser-Lys-Leu-Lys-Lys-Thr-Glu-Thr-Gin-Glu-Lys-Asn-Pro-Leu-Pro-Ser-Lys-Glu-Thy-IleGlu-Gin-Glu-Lys-Gin-Ala-Gly-Glu-Ser

DESCRIPTION:

AOD9604 is a GH fragment which comprised the last 16 amino acids of the larger growth hormone molecule. Although originally studied for fat loss, further studies have transitioned its application for regenerative medicine. In combination with hyaluronic acid (HA), it is now being used to help regenerate hyaline cartilage and is showing strong efficacy in the treatment of osteoarthritis. The combination acts to enhance the differentiation of adipose mesenchymal stem cells into bone, promote proteoglycan and collagen production in chondrocytes, and promote

differentiation of myoblasts into C2C12 cells; all of which are essential for bone, cartilage, and muscle repair. These studies indicate that it has stronger therapeutic benefits compared to Bone Marrow Aspirate Concentrate (BMAC) and Platelet Rich Plasma (PRP) therapy, which have also been emerging as candidates for osteoarthritis medications. AOD9604 + HA has proceeded to human WOMAC trials which allow the combination to be investigated for on an osteoarthritis index which considers pain, stiffness, and functionality on a variety of scores.

PROTOCOL:

Content & Potency: AOD 1000mcg/ml + HA 10mg/ml intra-articular injectable provided in a 5ml vial.
Suggested dosage: 0.5-0.75ml injected intra-articularly by a medical professional once a week for 4 weeks, then once a month for 5 months.

CLINICAL RESEARCH:

Effect of Intra-articular Injection of AOD9604 with or without Hyaluronic Acid in Rabbit Osteoarthritis Model

Objective: To investigate the effects of AOD9604 intra-articular injections with or without hyaluronic acid (HA) in a collagenase-induced knee osteoarthritis (OA) rabbit model.

Design: Mature New Zealand white rabbits (n=32) were randomly administered 2 mg collagenase type II twice in each knee joint. Weekly injections of 0.6 mL saline (Group 1), 6 mg HA (Group 2), 0.25 mg AOD9604 (Group 3), and 0.25 mg AOD9604 with 6 mg HA (Group 4) were administered for 4-7 weeks after the first intra-articular collagenase injection. The degree of cartilage degeneration was assessed using morphological and histopathological findings, and the degree of lameness was observed at 8 weeks after the first collagenase injection.

Results: Mean gross morphological and histopathological scores were significantly higher in Group 1 than in Groups 2, 3, and 4, and the scores were significantly lower in Group 4 than in Groups 2 and 3. The lameness period in Group 4 was significantly shorter than those in Groups 1, 2, and 3. The lameness period in Group 1 was significantly longer than those in Groups 2 and 3.

Conclusion: Intra-articular AOD9604 injections using ultrasound guidance enhanced cartilage regeneration, and combined AOD9604 and HA injections were more effective than HA or AOD9604 injections alone in the collagenase-induced knee OA rabbit model.

Beom Kim, Sang & Kwon, Dong & Kwak, Hyun & Shin, Yong & Han, Hyun-Jung & Hwa Lee, Jong & Choi, Seok Hwa. (2010). Additive Effects of Intra-articular Injection of Growth Hormone and Hyaluronic Acid in Rabbit Model of Collagenase-induced Osteoarthritis. Journal of Korean medical science. 25.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



BPC-157

Purity: >98% (HPLC on request) | Molecular Formula: C₆₂H₉₈N₁₆O₂₂
Molecular Weight: 1419.5355 | Sequence: L-Valine,glycyl-L-alpha-glutamyl-L-prolyl-L-prolyl-L-prolyl-glycyl-L-lysyl-L-prolyl-L-alanyl-L-alpha-aspartyl-L-alpha-aspartyl-L-alanylglycyl-L-leucyl-glycyl-L-alpha-glutamyl-L-prolyl-L-prolyl-L-prolyl-glycyl-L-lysyl-L-prolyl-L-alanyl-L-alpha-aspartyl-L-alpha-aspartyl-L-alanylglycyl-L-leucyl-L-valine

DESCRIPTION:

Pentadecapeptide BPC 157, composed of 15 amino acids, is a partial sequence of body protection compound (BPC) that is discovered in and isolated from human gastric juice. Experimentally it has been demonstrated to accelerate the healing of many different wounds, including tendon-to-bone healing and superior healing of damaged ligaments. Additionally, BPC 157 has shown to protect organs and aids in the prevention of gastric

ulcers. BPC-157 acts systemically in the digestive tract to combat leaky gut, IBS, gastrointestinal cramps, and Crohn's disease. This peptide has been known to exhibit analgesic characteristics as well. Those who suffer from discomfort due to muscle sprains, tears and damage may benefit from treatment with this peptide. It can also help to aid skin burns at a faster rate by increasing blood flow to damaged tissues.

PROTOCOL:

Content & Potency:

- Injectable: 2000mcg/ml subcutaneous injectable provided in a 5ml vial.
- Oral: 500mcg capsule provided in a quantity of 30.
- Nasal: 2mg/mL nasal spray in a 3mL bottle
- Cream: 100mcg/mL transdermal 30g

BPC/HOD/GHK-<0 2mg/1mg/2mg/mL 30mL

Suggested dosage:

- Injectable: 0.15ml injected subcutaneously every day for 30 days.
- Oral: 30 capsules at 500mcg. Take one capsule by mouth once daily for 30 days.

CLINICAL RESEARCH:

The Promoting effect of Pentadecapeptide BPC 157 on tendon healing involves tendon outgrowth, cell survival, and cell migration

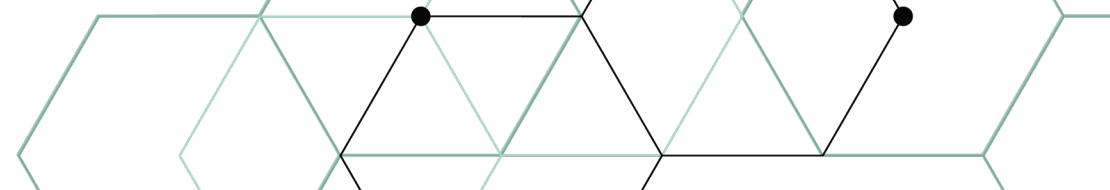
Many growth factors such as epidermal growth factor (EGF), transforming growth factor-(TGF-), and bone morphogenetic proteins (BMPs) have been used to improve the healing of torn tendon in the lab. However, the short duration of these easily digested growth factors hampers their clinical usage. Gastric pentadecapeptide BPC 157 is a partial sequence of human gastric protein BPC, which has been discovered in and isolated from gastric juice. It is highly stable and resistant to hydrolysis or enzyme digestion, even in the gastric juice. Besides, it is

easily dissolved in water and needs no carrier for its application. Experimentally it was demonstrated to enhance the healing of different wounds, such as gastric ulcer, skin, cornea, muscle, colon-colon anastomosis, colocolutaneous fistula, and segmental bone defect. It was also found to accelerate the healing of transected rat Achilles tendon (12, 29) and medial collateral ligament of knee. Currently it is in clinical trials for treating inflammatory bowel disease.

Chung-Hsun Chang, Wen-Chung, Tsai Miao-Sui Lin, Ya-Hui Hsu, and Jong-Hwei Su Pang

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

TAILOR MADE COMPOUNDING



Cerebrolysin

CJC-1295

Purity: >99% (HPLC on request)

DESCRIPTION:

Cerebrolysin (synonym FPE 1070) is a nootropic drug which consists of low-molecular peptides which possesses neuroprotective and neurotrophic repair properties. The active fragment of Cerebrolysin is made of proteins with very low molecular masses that do not exceed 10.000 daltons. This means they can penetrate the blood-brain (or blood-SCF) barrier and reach neurons directly, making the drug able to show organo-specific combined effects towards the brain. Cerebrolysin has been proven to have

neurotrophic action similar to nerve growth factors, which cause peripheral and central neuronal stimulation. It improves efficiency within the brain's aerobic metabolic processes and improves intracellular peptide synthesis. The neuroprotective properties of this nootropic agent help to shield neurons from lactocidosis, to prevent the formation of free radicals, and have been studied in Parkinson's, Alzheimer's, MS, ALS, TBI, and stroke.

PROTOCOL:

Content & Potency: 215.2mg/mL x 10mL x 4 vials : ready-to-inject subcutaneously
Suggested dosage: Inject 1ml daily for over a 40 week course.

CLINICAL RESEARCH:

Cerebrolysin in Alzheimer's disease: a randomized, double-blind, placebo-controlled trial with a neurotrophic agent

Cerebrolysin (Cere) is a compound with neurotrophic activity. It has been shown to be effective in the treatment of Alzheimer's disease (AD) in earlier trials. In this multicenter, randomized, double-blind, placebo-controlled, parallel-group study, patients were injected intravenously with placebo or 30mL Cere five days per week for four weeks. Effects on cognition and global function were evaluated with the Alzheimer Disease Assessment Scale – Cognitive Subscale (ADAS-Cog) and the Clinician Interview based Impression of Change with Caregiver Input scale (CIBIC+) 4, 12, 24 weeks after the beginning of the injections. 192 patients were enrolled, 95 were randomized to placebo, and 97 to Cere. At baseline, there was a significant difference between groups for age of

onset of dementia, and the number of patients with hallucinations. At week 12 there was a significant difference on the CIBIC + (p=0.033) in favor of Cere. The number of CIBIC+ responders (score < 4), was significantly higher (p=0.007), with 68 (76%) in the Cere group and 51 (57%) in the placebo group. Trends were noted in the Disability Assessment in Dementia scale and the Cornell Depression Scale. Adverse events were recorded in 73% of placebo and 64% of Cere patients. Most common adverse events were headaches, dizziness, weight loss and anxiety. Conclusions: Cere treatment was well tolerated and resulted in significant improvements in the global score two months after the end of active treatment.

M. Panisset, S. Gauthier, H. Moessler, M. Windisch

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Purity: >98% (HPLC on request) | Molecular Formula: C165H269N47O46
Molecular Weight: 3647.15 | Sequence: Tyr-D-Ala-Asp-Ala-Ile-Phe-Thr-Gln-Ser-Tyr-Arg-Lys-Val-Leu-Ala-Gln-Leu-Ser-Ala-Arg-Lys-Leu-Leu-Gln-Asp-Ile-Leu-Ser-Arg-NH2

DESCRIPTION:

CJC 1295 stimulates growth hormone release via binding to the pituitary. This peptide directly mimics the endogenous Growth Hormone Releasing Hormone (GHRH) typically secreted by the hypothalamus. CJC 1295 comes in 2 chemical forms. One includes a binding group called DAC, or Drug Activity Complex. The DAC bind to serum albumin and significantly increases half life. However, for many reasons, this is not the preferred product for many patients clinically.

Instead, the tetrasubstituted 29 amino acid modified growth releasing factor often called Mod-GRF is the

version of CJC most use clinically. This version of the CJC 1295 outperforms the older and outdated GHRHs such as Sermorelin. The half-life of Sermorelin ranges from 8-12 minutes, whereas the half-life of CJC 1295 extends to 30 minutes.

As a result of better stimulation and release you see many clinical results including fat loss, increases lean muscle mass, better lipid profiles, better deep wave sleep and increased repair and recovery. Use in combination with Ipamorelin, the CJC is one of the most widely prescribed products.

PROTOCOL:

Content & Potency: 2000mcg/ml subcutaneous injection provided in a 2ml vial.
Suggested dosage: Inject 0.10ml subcutaneously 5 out of 7 nights of the week before bedtime on an empty stomach.

***We suggest using the CJC 1295 in combination with Ipamorelin as it provides a synergistic effect, generating five times the benefits of using the CJC 1295 or Ipamorelin alone. The combination allows for a maximized release of GH because CJC 1295 and Ipamorelin have different mechanisms of action and work on different receptors (GHRH-R & Ghrelin-R).

CLINICAL RESEARCH:

Factor I Secretion by CJC 1295, a Long-Acting Analog of GH-Releasing Hormone, in Healthy Adults

Context: Therapeutic use of GHRH to enhance GH secretion is limited by its short duration of action.

Objective: The objective of this study was to examine the pharmacokinetic profile, pharmacodynamic effects, and safety of CJC 1295, a long-acting GHRH analog.

Design: The study design was two randomized, placebo-controlled, double blind, ascending dose trials with durations of 28 and 49 d.

Setting: The study was performed at two investigational sites.

Participants: Healthy subjects, ages 21–61 yr, were studied.

Interventions: CJC 1295 or placebo was administered sc in one of four ascending single doses in the first study and in two or three weekly or biweekly doses in the second study.

Main Outcome Measures: The main outcome measures were peak concentrations and area under the curve of GH and IGF-I;

standard pharmacokinetic parameters were used for CJC 1295.

Results: After a single injection of CJC 1295, there were dose dependent increase in mean plasma GH concentrations by 2- to 10-fold for 6 d or more and in mean plasma IGF-I concentrations by 1.5- to 3-fold for 9-11 d. The estimated half-life of CJC 1295 was 5.8-8.1 d. After multiple CJC 1295 doses, mean IGF-I levels remained above baseline for up to 28 d. No serious adverse reactions were reported.

Conclusions: Subcutaneous administration of CJC 1295 resulted in sustained, dose-dependent increases in GH and IGF-I levels in healthy adults and was safe and relatively well tolerated, particularly at doses of 30 or 60 ug/kg. There was evidence of a cumulative effect after multiple doses. These data support the potential utility of CJC 1295 as a therapeutic agent.

Sam L. Teichman, Ann Neale, Betty Lawrence, Catherine Gagnon, Jean-Paul Castaigne, Lawrence A. Frohman, Prolonged Stimulation of Growth Hormone (GH) and Insulin-Like Growth Factor I Secretion by CJC-1295, a Long-Acting Analog of GH-Releasing Hormone, in Healthy Adults, The Journal of Clinical Endocrinology & Metabolism, Volume 91, Issue 3, 1 March 2006, Pages 799–805, <https://doi.org/10.1210/jc.2005-1536>

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

DHH-B

Purity: >98% (HPLC on request) | Molecular Formula: C₂₄H₃₁NO₄
Molecular Weight: 397.51 g/mol | Sequence: Non- peptide

DESCRIPTION:

DHH-B, dihydrohonokiol-B, is a natural supplement which has anxiolytic-effects. Treatment with DHH-B does not cause any significant changes in motor activity or muscle relaxation. Benzodiazepines are some of the most commonly prescribed medications in the United

States. These anxiolytics have many well-known side effects including motor function and more. That being said, this product has the potential to help people transition from benzodiazepines to DHH-B or act as an alternative to benzodiazepines.

PROTOCOL:

Content & Potency: 30, 7.5mg capsules
Suggested dosage: Take 1-2 capsules as needed

CLINICAL RESEARCH:

Comparative assessment of the anxiolytic-like activities of honokiol and derivatives

A Department of Neuropsychopharmacology (Tsumura), Gunma University School of Medicine, 3-39-22 Showa-machi, Maebashi, Gunma 371-8511, Japan Research Laboratories, Tsumura and Co., Ami-machi, Inashiki-gun, Ibaraki 300-1192, Japan Department of Biochemistry, The University of Texas Health Science Center at San Antonio, San Antonio, TX, USA

Honokiol has previously been shown to be an effective anxiolytic-like agent in mice when administered for 7 days at 0.2 mg/kg/day prior to evaluation in an elevated plus-maze, while 20 mg/kg is required for efficacy as a single oral dose. The aim of this study was to find analogs of honokiol that are more effective for acute administration. Among the eight analogs evaluated, one partially reduced

derivative of honokiol [30-(2-propenyl)-5-propyl-(1,10-biphenyl)-2,40-diol] exhibited significant anxiolytic-like activity at 0.04 mg/kg. Following oral administration of 1 mg/kg of this analog, anxiolytic-like activity was clearly evident at 1 h, peaked at 3 h, and remained significant for longer than 4 h after treatment. Combined administration of the derivative with diazepam led to enhanced anxiolytic-like efficacy. Moreover, as with diazepam, the anxiolytic-like effect of the analog was reduced by flumazenil. In contrast, bicuculline, a GABA_A antagonist, had no effect on the activity of the derivative. Taken together, these results suggest that this analog of honokiol acts at the benzodiazepine recognition site of the GABA_A ± benzodiazepine receptor complex.

Hisashi Kuribara, Eiko Kishia, Masayuki Kimurab, Susan T. Weintraubc, Yuji Maruyamaa,*

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Dihexa

Purity: >98% (HPLC on request) | Molecular Formula: C₂₇H₄₄N₄O₅
Molecular Weight: 504.672 g/mol | Sequence: Non-peptide

DESCRIPTION:

Dihexa is a peptide variant derived from angiotensin IV which has been found to potently improve cognitive function in animal models of disease such as Alzheimer's. Angiotensin IV is a derivative of the potent vasoconstrictor angiotensin II and has been shown to enhance acquisition, consolidation, and recall of learning

and memory in animal models when administered centrally or peripherally. In an assay of neurotrophic activity, Dihexa was found to be seven orders of magnitude more potent than BDNF. It could possibly help in the repair of the brain and nerves in neurological disease.

PROTOCOL:

- Content & Potency:**
- Cream: 20mg/ml transdermal cream provided in a 30ml transdermal applicator.
 - Oral: 1mg or 2mg capsules
- Suggested dosage:**
- Cream: Apply 0.5-1.0ml (2-4 clicks) to inner forearms once daily, rub in until absorbed.
 - Oral: Taken once daily.

CLINICAL RESEARCH:

The Procognitive and Synaptogenic Effects of Angiotensin IV– Derived Peptides Are Dependent on Activation of the Hepatocyte Growth Factor / c-Met System

A subset of angiotensin IV (AngIV)–related molecules are known to possess procognitive/antidementia properties and have been considered as templates for potential therapeutics. However, this potential has not been realized because of two factors: 1) a lack of blood- brain barrier– penetrant analogs, and 2) the absence of a validated mechanism of action. The pharmacokinetic barrier has recently been overcome with the synthesis of the orally active, blood-brain barrier–permeable analog N-hexanoic-tyrosine-isoleucine-(6) aminohexanoic amide (dihexa). Therefore, the goal of this study was to elucidate the mechanism that underlies dihexa's procognitive activity. Here, we demonstrate that dihexa binds with high affinity

to hepatocyte growth factor (HGF) and both dihexa and its parent compound Norleucine 1-AngIV (Nle1-AngIV) induce c-Met phosphorylation in the presence of subthreshold concentrations of HGF and augment HGF-dependent cell scattering. Further, dihexa and Nle1-AngIV induce hippocampal spinogenesis and synaptogenesis similar to HGF itself. These actions were inhibited by an HGF antagonist and a short hairpin RNA directed at c-Met. Most importantly, the procognitive/antidementia capacity of orally delivered dihexa was blocked by an HGF antagonist delivered intracerebroventricularly as measured using the Morris water maze task of spatial learning.

Benoist, Caroline C et al. "The procognitive and synaptogenic effects of angiotensin IV-derived peptides are dependent on activation of the hepatocyte growth factor/c-met system." The Journal of pharmacology and experimental therapeutics vol. 351,2 (2014): 390-402.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

DSIP

Purity: >98% (HPLC on request) | Molecular Formula: C₃₅H₄₈N₁₀O₁₅
Molecular Weight: 848.81 | Sequence: Trp-Ala-Gly-Gly-Asp-Ala-Ser-Gly-Glu

DESCRIPTION:

DSIP is a well-known neuromodulator and natural somnogenic nonapeptide with many other physiological functions. It is typically found in the brain and easily passes the blood-brain barrier. It is mainly prescribed for the treatment of pain conditions, alcohol and opioid withdrawal, CRH and stress-related symptoms, low testosterone (via stimulation of LH), and even sometimes as an antioxidant and anti-oncogenic protein. It has been discovered and heavily studied for over 40 years, yet, the mechanism of action is still complex and not well understood. The results of studies of DSIP and its analogues over a period of 30 years since its discovery enable one to state with confidence that DSIP is a unique member of the family of peptide neuromodulators. It exhibits a

pronounced stress protective action and decreases stress-induced metabolic and functional disorders in human and animal organisms exposed to a variety of stresses. Some of the effects of the peptide are accomplished through the modulating action on central regulatory processes, owing to the systemic antioxidant action, the modulating influence on the activity of GABAergic, glutamatergic, and other neuronal systems. It also works on the expression of early response genes in brain structures, and on the activity of biosynthetic and proteolytic processes. DSIP has traditionally been dosed as an IV infusion, however, it can be given subcutaneously as well. Traditional doses have been 100mcg.

PROTOCOL:

Content & Potency: 1000mcg/ml subcutaneous injectable provided in a 3ml vial.
Suggested dosage: Inject 0.1ml subcutaneously once daily 2-3 times per week.

CLINICAL RESEARCH:

DSIP-more than a sleep peptide?

In several species DSIP at low doses has been shown to promote sleep. Although its physiological role remains to be clarified, DSIP illustrates several concepts applicable to other brain peptides. These include the bell-shaped dose-response curve, central effects after peripheral administration, a delayed and prolonged time course, and

some penetration of the blood-brain barrier in essentially intact form. Concepts applicable to one neuropeptide, therefore, appear to be applicable to others. In this article Abba Kastin and colleagues review the known effects of DSIP and argue that more work needs to be carried out before it can be labelled functionally.

J. Kastin, Abba & A. Olson, Gayle & V. Schally, Andrew & H. Coy, David. (1980). DSIP — more than a sleep peptide?. Trends in Neurosciences - TRENDS NEUROSCI. 3. 163-165.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Enclomiphene

Purity: >98% (HPLC on request) | Molecular Formula: C₂₆H₂₈ClNO
Molecular Weight: 405.966 g/mol | Sequence: Non-peptide

DESCRIPTION:

Enclomiphene is used in the process of treating male hypogonadism (lower function of the reproductive organs) and is a single isomer with pure estrogen antagonism. Enclomiphene is a non-steroidal selective estrogen receptor modulator (SERM) and acts by increasing

gonadotropin secretion and gonadal production of testosterone. Enclomiphene has the potential to help the reproductive status in men and improve metabolic profiles. It is a single isoform of the more abundant clomiphene and has much fewer side effects.

PROTOCOL:

Content & Potency: 25mg capsules provided in a quantity of 30.
Suggested dosage: Take one capsule by mouth once daily.

CLINICAL RESEARCH:

Enclomiphene citrate stimulates testosterone production while preventing oligospermia: a randomized phase II clinical trial comparing topical testosterone.

Objective: To determine the effect of enclomiphene citrate in men with secondary hypogonadism.

Methods: iws randomized phase IIB study enrolled 124 men with a morning serum T level of <250 ng/dL on 2 occasions. e subjects were randomized to one of two doses of enclomiphene citrate (12.5-mg or 25-mg), 1% topical testosterone, or placebo. Hormone levels of LH, FSH, and T and seman level were measured before, during and aer 3 months of treatment.

Results: A total of 113 men received 3 months of treatment, and 73 completed the study and provided both baseline and at least 1 seman sample at the end of the study. All 3 active treatment groups showed significantly increased in total testosterone level from baseline compared with placebo, with no statistically significant difference in testosterone levels found between the active treatment groups compared with placebo.

Wiehle RD, Fontenot GK, Wike J, et al. ZA-203 Clinical Study Group. Fertil Steril. 2014; 102(3): 720-727.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Epitalon

Purity: >98% (HPLC on request) | Molecular Formula: C₁₆H₂₄N₄O₁₀
Molecular Weight: 432.4 g/mol | Sequence: H-Ala-Glu-Asp-Gly-O

DESCRIPTION:

Epithalon (also known as Epitalon or Epithalone) is the synthetic version of the polypeptide Epithalamin which is naturally produced in humans. The pineal peptide preparation is secreted in the epithalamium-epiphysal region of the brain. Its more prominent tasks are: to regulate metabolism in the epiphysis, increase the sensitivity of hypothalamus to its natural hormonal influences, normalize the function of the anterior pituitary, regulate the levels of gonadotropins and melatonin in the body. Epithalamin increases a person's resistance to emotional stress and also acts as an antioxidant.

It is a bio-regulator for the endocrine system, especially for the pineal gland, and has been shown to lengthen telomeres in human cells. The mechanisms in Epitalon are a lot more complex than just activating telomerase. It reduces lipid oxidation and ROS, along with normalizing T cell function. It seems to normalize cholesterol and uric acid, along with prolactin levels. It has shown promise in restoring pancreatic hormone function. Additionally, it restored and normalized melatonin levels in older patients who have lost some pineal function due to aging.

PROTOCOL:

Content & Potency: 10mg/ml subcutaneous injection provided in a 5ml vial.
Suggested dosage: Inject 10mg every 3 days for 15 days - repeat twice yearly.

CLINICAL RESEARCH:

Peptide Geroprotector from the Pituitary Gland Inhibits Rapid Aging of Elderly People: Results of 15-Year Follow-Up

The paper presents the results of randomized comparative study of the efficiency of peptide geroprotector from the pituitary gland in elderly patients with rapidly aging cardiovascular system. Over three years 39 coronary patients received, in addition to basic therapy, regular courses of epithalamin (peptide drug), while 40 coronary patients (control group) received basic therapy alone. Long-term treatment with epithalamin (6 courses over 3

years) decelerated aging of the cardiovascular system, prevented age-associated impairment of physical endurance, normalized circadian rhythm of melatonin production and carbohydrate and lipid metabolism. A significantly lower mortality in the group of patients treated with epithalamin in parallel with basic therapy also indicated a geroprotective effect of the peptide preparation from the pineal gland.

O. V. Korkushko, V. Kh. Khavinson*, V. B. Shatilo, and I. A. Antonyk-Sheglova Translated from Byulleten' Eksperimental'noi Biologii i Meditsiny, Vol. 151, No. 3, pp. 343-347, March, 2011.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



FGL(L)

Purity: >98% (HPLC on request) | Molecular Formula: C₇₁H₁₁₆N₂₀O₂₅
Molecular Weight: 3432.62 g·mol⁻¹ | Sequence: Glu-Val-Tyr-Val-Val-Ala-Glu-Asn-Gln-Gln-Gly-Lys-Ser-Lys-Ala

DESCRIPTION:

FGL(L) is a peptide with neurotrophic and memory enhancing properties. FGL peptide is a variant of the natural neural cell adhesion molecule. Neural cell adhesion molecule (NCAM) is a membrane-bound glycoprotein expressed on the surface of neuronal and glial cells. FGL(L) was directly created as a fibroblast growth factor receptor agonist. FG loop (FGL) peptide, that is derived from the second F3 module of NCAM has been found to activate FGFR1. Activating the NCAM-FGFR signaling pathways result in increased neurite outgrowth and survival and leads to its effects in memory. In addition to its effects on memory, FGL was found to have a positive impact on the healing of neuronal tissues subjected to ischemia

by decreasing oxidative stress-induced neuronal cell death. FGL was also demonstrated to affect neuropathological symptoms related to Alzheimer's disease by inhibiting neuronal degeneration and death.

Its potential effect on memory and neurodegenerative disease progression have excited many people and it was awarded a 60 million dollar grant in 2016 for further exploration. In addition to its effects on neurodegenerative disease, it also has been viewed as a targeted treatment for TBI, stroke, depression, and general improvement of cognitive function.

PROTOCOL:

Content & Potency: 10mg/ml solution provided in a 3ml vial.
Suggested dosage: Inject 0.1ml subcutaneously daily into the abdomen.

CLINICAL RESEARCH:

A Neural Cell Adhesion Molecule-Derived Fibroblast Growth Factor Receptor Agonist, the FGL-Peptide, Promotes Early Postnatal Sensorimotor Development and Enhances Social Memory Retention

Abstract—The neural cell adhesion molecule (NCAM) belongs to the immunoglobulin (Ig) superfamily and is composed extracellularly of five Ig-like and two fibronectin type III (F3) modules. It plays a pivotal role in neuronal development and synaptic plasticity. NCAM signals via a direct interaction with the fibroblast growth factor receptor (FGFR). A 15-amino-acid long peptide, the FG loop (FGL) peptide, that is derived from the second F3 module of NCAM has been found to activate FGFR1.

We here report that the FGL peptide, when administered intranasally to newborn rats, accelerated early postnatal development of coordination skills. In adult animals s.c. administration of FGL resulted in a prolonged retention of social memory. We found that FGL rapidly penetrated into the blood and cerebrospinal fluid after both intranasal and s.c. administration and remained detectable in the fluids for up to 5 hours.

Echer, T & Novitskaia, V & Berezin, Vladimir & Bock, Elisabeth & Glenthøj, B & Klementiev, B. (2006). A neural cell adhesion molecule-derived fibroblast growth factor receptor agonist, the FGL-peptide, promotes early postnatal sensorimotor development and enhances social memory retention. Neuroscience. 141. 1289-99. 10.1016/j.neuroscience.2006.04.059.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

GHK-Cu

Purity: >98% (HPLC on request) | Molecular Formula: C₂₈H₅₂CuN₁₂O₈
Molecular Weight: 748.346 g/mol | Sequence: Non-Peptide

DESCRIPTION:

GHK-Cu is a naturally occurring copper complex that was first identified in human plasma, but has hence been found in multiple locations such as saliva and urine. Copper peptides are small, naturally occurring protein fragments that have high affinity for copper ions, which are critical to normal body function. GHK-Cu has a variety of roles in the human body including, but not limited to, promoting activation of wound healing, attracting immune cells, antioxidant and anti-inflammatory effects, stimulating collagen and glycosaminoglycan synthesis in skin fibroblasts, and promoting blood vessel growth. There has been evidence that has shown that it acts as a

feedback signal that is generated after tissue injury. First, it seems to act as a potent protector of tissue and anti-inflammatory agent that controls the oxidative damage that occurs post-tissue injury. Further, it then plays a big role in signaling tissue remodeling which removes damaged/scarred tissue and generates new, healthy tissue. However, these positive effects decline with age because the concentration of GHK-Cu in the body decreases with age. Thus, there is an increase in inflammation, cancerous activity, and tissue destruction. Clinically, it is mostly used to decrease fine lines and wrinkles and to improve hair regrowth.

PROTOCOL:

- Content & Potency:**
- Injectable: 10mg/ml subcutaneous injection provided in a 5ml vial.
 - Topical: 5mg/ml (5%) topical foam provided in a 50ml foaming applicator.
- Suggested dosage:**
- Injectable: Inject 0.2ml subcutaneously once daily.
 - Topical: Apply 1ml (2 pumps) to scalp once daily at night.

CLINICAL RESEARCH:

GHK Peptide as a Natural Modulator of Multiple Cellular Pathways in Skin Regeneration

GHK (glycyl-L-histidyl-L-lysine) is present in human plasma, saliva, and urine but declines with age. It is proposed that GHK functions as a complex with copper 2+ which accelerates wound healing and skin repair. GHK stimulates both synthesis and breakdown of collagen and glycosaminoglycans and modulates the activity of both metalloproteinases and their inhibitors. It stimulates collagen, dermatan sulfate, chondroitin sulfate, and the small proteoglycan, decorin. It also restores replicative vitality to fibroblasts after radiation therapy. The molecule attracts immune and endothelial cells to the site of an injury. It accelerates wound-healing of the skin, hair follicles, gastrointestinal tract, boney tissue, and foot pads

of dogs. It also induces systemic wound healing in rats, mice, and pigs. In cosmetic products, it has been found to tighten loose skin and improve elasticity, skin density, and firmness, reduce fine lines and wrinkles, reduce photodamage, and hyperpigmentation, and increase keratinocyte proliferation. GHK has been proposed as a therapeutic agent for skin inflammation, chronic obstructive pulmonary disease, and metastatic colon cancer. It is capable of up and down regulating at least 4,000 human genes, essentially resetting DNA to a healthier state. The present review revisits GHK's role in skin regeneration in the light of recent discoveries.

GHK Peptide as a Natural Modulator of Multiple Cellular Pathways in Skin Regeneration

Pickart, Loren & Vasquez-Soltero, Jessica & Margolina, Anna. (2015). GHK Peptide as a Natural Modulator of Multiple Cellular Pathways in Skin Regeneration. BioMed Research International.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Glycyrrhetic Acid & Aminophylline Transdermal Fat Loss Cream

Glycyrrhetic Acid- Purity: >98% | Molecular Formula: C₃₀H₄₆O₄
Molecular Weight: 470.69 g/mol | Sequence: Non-Peptide
Aminophylline- Purity: >98% | Molecular Formula: C₁₆H₂₄N₁₀O₄
Molecular Weight: 420.43 g/mol | Sequence: Non-Peptide

DESCRIPTION:

Aminophylline and glycyrrhetic acid transdermal cream is used for fat loss. Aminophylline and glycyrrhetic prevent cAMP breakdown. Cyclic AMP (cAMP) functions in several biochemical processes including the regulation of glycogen, sugar, and lipid metabolism. cAMP works by activating protein kinase A (PKA) which assists in glycogen, sugar, and lipid metabolism. Aminophylline has displayed topical fat reduction from the waist. In a study examining aminophylline cream application, the reduction in waist circumference was significant for both men and women. Over a period of 12 weeks, participants in the study lost 11cm in waist circumference. In differentiated adipocytes,

18β-glycyrrhetic acid increases the level of glycerol release and up-regulates the mRNA of hormone-sensitive lipase, adipose triglyceride lipase, and perilipin, as well as the phosphorylation of hormone-sensitive lipase. 18 β-glycyrrhetic acid alters fat mass by directly affecting adipogenesis in maturing preadipocytes and lipolysis in mature adipocytes. Therefore, aminophylline and 18β-glycyrrhetic acid may be useful for treating obesity. Both aminophylline and glycyrrhetic acid combined effectively combat fat loss. Aminophylline and glycyrrhetic acid fat loss cream yields the best results through topical application.

PROTOCOL:

Content & Potency: Aminophylline/Glycyrrhetic Acid 0.5%/2.5% transdermal cream provided in a 60ml Topiclick cream applicator.
Suggested dosage: Apply 1ml twice daily transdermally.

CLINICAL RESEARCH:

18β-Glycyrrhetic acid inhibits adipogenic differentiation and stimulates lipolysis

18β-Glycyrrhetic acid (18β-GA) obtained from the herb liquorice has various pharmacological properties including anti-inflammatory and anti-bacterial activities. However, potential biological anti-obesity activities are unclear. In this study, novel biological activities of 18β-GA in the adipogenesis of 3T3-L1 preadipocytes and in lipolysis of differentiated adipocytes were identified. Mouse 3T3-L1 cells were used as an in vitro model of adipogenesis and lipolysis, using a mixture of insulin/dexamethsone/3-isobutyl-1-methylxanthine (IBMX) to induce differentiation. The amount of lipid droplet accumulation was determined by an AdipoRed assay. The expression of several adipogenic transcription factors and enzymes was investigated using real-time reverse transcriptase-polymerase chain reaction (RT-PCR) and Western

blotting. 18β-GA dose-dependently (1–40 IM) significantly decreased lipid accumulation in maturing preadipocytes. In 3T3-L1 preadipocytes, 10 IM of 18β-GA down-regulated the transcriptional levels of peroxisome proliferator-activated receptor c, CCAAT/enhancer-binding protein and adiponectin, which are markers of adipogenic differentiation via Akt phosphorylation. Also, in differentiated adipocytes, 18β-GA increased the level of glycerol release and up-regulated the mRNA of hormone-sensitive lipase, adipose TG lipase and perilipin, as well as the phosphorylation of hormone-sensitive lipase at Serine 563. The results indicate that 18β-GA alters fat mass by directly affecting adipogenesis in maturing preadipocytes and lipolysis in matured adipocytes. Thus, 18β-GA may be useful for the treatment of obesity.

Moon, Myung-Hee et al. "18β-Glycyrrhetic acid inhibits adipogenic differentiation and stimulates lipolysis." Biochemical and biophysical research communications 420 4 (2012): 805-10 .

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

IGF-1

Purity: >98% (HPLC on request) | **Molecular Formula:** C71H119N17O19S
Molecular Weight: 1546.893 g/mol | **Sequence:** Gly-Pro-Glu-Thr-Leu-Cys-Gly-Ala-Glu-Leu-Val-Asp-Ala-Leu-Gln-Phe-Val-Cys-Gly-Asp-Arg-Gly-Phe-Tyr-Phe-Asn-Lys-Pro-Thr-Gly-Tyr-Gly-Ser-Ser-Ser-Arg-Arg-Ala-Pro-Gln-Thr-Gly-Ile-Val-Asp-Glu-Cys-Cys-Phe-Arg-Ser-Cys-Asp-Leu-Arg-Arg-Leu-Glu-Met-Tyr-Cys-Ala-Pro-Leu-Lys-Pro-Ala-Lys-Ser-Ala

DESCRIPTION:

IGF-1 is a peptide consisting of 70 amino acids with a molecular weight of 7649 Da. IGF-1 has an A and B chain connected by disulphide bonds, like insulin, which is how it gets its name. The structural similarity to insulin explains the ability of IGF-1 to bind (with low affinity) to the insulin receptor. IGF-1 is secreted by many tissues and the secretory site seems to determine its actions. Most IGF-1 is secreted by the liver and is transported to other tissues, acting as an endocrine hormone. IGF-1 is also secreted by other tissues, including cartilaginous cells, and acts locally as a paracrine hormone. Most of the IGF-1 produced by the liver is secreted for its proliferative and growth effects. Lower IGF-1 and growth hormone are often associated with excess body fat. IGF-1 and other proteins in the IGF family are growth factors that stimulate the proliferation and survival of various cell types

including muscle, bone, and cartilage tissue. IGF-1 plays an important role in childhood growth and continues to have anabolic effects in adults. A synthetic analog of IGF-1, mecasermin is commercially available and is used for the treatment of growth failure. Therapeutic administration of IGF-1 is associated with reversing insulin sensitivity, reducing weight and increasing metabolic expenditure as well potential reversal of degeneration of spinalcord motor neuron axons in certain peripheral neuropathies. IGF-1 LR3 has a modified amino acid sequence compared to biological IGF-1. It has an additionally arginine at amino acid position 2. By making this change, it gives the molecule higher potency and a much longer half-life. For this reason it is commonly used as long acting version for the same therapeutic reasons as the IGF-1.

PROTOCOL:

Content & Potency: 620mcg/ml subcutaneous injectable provided in two 6.2ml vials.
Suggested dosage: Inject 0.4ml subcutaneously once daily.

CLINICAL RESEARCH:

Effects of human growth hormone, insulin-like growth factor I, and diet and exercise on body composition of obese postmenopausal women.

To determine the effects of GH and insulin-like growth factor I (IGF-I) administration, diet, and exercise on weight loss, body composition, basal metabolic rate (BMR), muscle strength, and psychological status, 33 moderately obese postmenopausal women (67.1 +/- 5.2 yr) participated in a 12-week randomized, double blind study. Participants were placed on a diet that provided 500 Cal/day less than that needed for weight maintenance, and they walked 3 days and strength trained 2 days each week. Subjects also self-injected GH (0.025 mg/kg BW.day), IGF-I (0.015 mg/kg BW.day), a combination of these doses of GH and IGF-I, or placebo (P). Twenty-eight women completed the study, as ve subjects dropped out due to

intolerable side-effects (e.g. edema). Weight loss occurred in all groups, with the largest decrease occurring in the GH plus IGF-I group (5.6 +/- 1.4 kg). Fat mass significantly decreased in all groups, with the largest losses observed in GH and GH plus IGF-I groups (6.3 +/- 1.8 and 8.4 +/- 2.8 kg, respectively). Despite weight loss, BMR was maintained in all groups. Muscle strength increased with training for all groups, and depression and anxiety scores decreased in groups receiving IGF-I. These data show that obese postmenopausal women can lose weight and fat without compromising fat free mass, BMR, or gains in muscle strength, and that GH and IGF-I given together may enhance fat loss over either given alone.

L. Thompson, J & E Butterfield, G & K Gylfadottir, U & Yesavage, J & Marcus, Robert & L Hintz, R & Pearman, Ann & Hoffman, AR. (1998). Effects of Human Growth Hormone, Insulin-Like Growth Factor I, and Diet and Exercise on Body Composition of Obese Postmenopausal Women 1. The Journal of clinical endocrinology and metabolism. 83.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Ipamorelin

Purity: >98% (HPLC on request) | **Molecular Formula:** C38H49N9O5
Molecular Weight: 711.863 | **Sequence:** Aib-His-D-2-Nal-D-Phe-Lys-NH

DESCRIPTION:

Ipamorelin is a selective GH-Secretagogue and ghrelin receptor agonist. The potency of ghrelin stimulation can be compared to GHRP6 with less appetite stimulation properties. However, unlike other GH-Secretagogues this pentapeptide

doesn't release the same volumes of cortisol, acetylcholine, prolactin and aldosterone. It is for this reason Ipamorelin has been considered the first selective, and best, GH Secretagogue.

PROTOCOL:

Content & Potency: 2000mcg/ml subcutaneous injectable provided in a 5ml vial.
Suggested dosage: Inject 0.10ml subcutaneously once daily 5 out of 7 days of the week.

***We suggest using the Ipamorelin in combination with CJC 1295 as it provides a synergistic effect, generating five times the benefits of using the CJC 1295 or Ipamorelin alone. The combination allows for maximized release of GH because the CJC 1295 and Ipamorelin have different mechanisms of action and work on different receptors (GHRH-R & Ghrelin-R).

CLINICAL RESEARCH:

Pharmacokinetic-Pharmacodynamic Modeling of Ipamorelin, a Growth Hormone Releasing Peptide, in Human Volunteers

To examine the pharmacokinetics (PK) and pharmacodynamics (PD) of ipamorelin, a growth hormone (GH) releasing peptide, in healthy volunteers. A trial was conducted with a dose escalation design comprising 5 different infusion rates (4.21, 14.02, 42.13, 84.27 and 140.45 nmol/kg over 15 minutes) with eight healthy male subjects at each dose level. Concentrations of ipamorelin and growth hormone were measured. The PK parameters showed dose-proportionality, with a short terminal half-life of 2 hours, a clearance of 0.078 L/h/kg and a volume of distribution at steady-state of 0.22 L/kg. The time course of GH stimulation by ipamorelin showed a single episode of GH release with a peak at 0.67 hours and an exponential

decline to negligible GH concentration at all doses. The ipamorelin-GH concentration relationship was characterized using an indirect response model and population fitting. The model employed a zero-order GH release rate over a finite duration of time to describe the episodic release of GH. Ipamorelin induces the release of GH at all dose levels with the concentration (SC50) required for half-maximal GH stimulation of 214 nmol/L and a maximal GH production rate of 694 mIU/L/h. The inter-individual variability of the PD parameters was larger than that of the PK parameters. The proposed PK/PD model provides a useful characterization of ipamorelin disposition and GH responses across a range of doses.

Gobburu, Jogarao & Agerso, Henrik & J. Jusko, William & Ynddal, Lars. (1999). Pharmacokinetic-Pharmacodynamic Modeling of Ipamorelin, a Growth Hormone Releasing Peptide, in Human Volunteers. Pharmaceutical research. 16. 1412-6.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

iRGD

Purity: >98% (HPLC on request) | Molecular Formula: C₃₅H₅₈N₁₄O₁₃S₂
Molecular Weight: 947.07g/mol | Sequence: Cys-Arg-Gly-Asp-Lys-Gly-Pro-Asp-Cys

DESCRIPTION:

iRGD is a cyclic peptide that binds to integrins that are expressed on tumor endothelial cells. Upon binding, a protease cleavage event is activated. When this event is activated the peptide is then able to bind neuropilin-1, activating an endocytotic/exocytotic transport pathway. As a result of this, it is able to hone to tumor cells and make them permeable to transport of many types of cancer therapies. This makes traditional cancer therapies target cells

better and makes the therapy less toxic. One study showed that doxorubicin, liposomal doxorubicin, Herceptin trastuzumab or Abraxane nab-paclitaxel had greater drug accumulation in the tumor by up to 40-fold than mice injected with one of the drugs alone. They equaled greater reductions in tumor growth. In all, the drug-peptide combination was as effective as threefold higher doses of drug alone.

PROTOCOL:

Content & Potency: 2.5mg/ml subcutaneous injection provided in a 10ml vial.
Suggested dosage: 40mcg/kg subcutaneously once daily in combination with other Cancer treatment.

CLINICAL RESEARCH:

Co-administration of a Tumor-Penetrating Peptide Enhances the Efficacy of Cancer Drugs

Poor penetration of anti-cancer drugs into tumors can be an important factor limiting their efficacy. Studying mouse tumor models, we show that a previously characterized tumor-penetrating peptide, iRGD (CRGDK/RGPD/EC), increased vascular and tissue permeability in a tumor-specific and neuropilin-1-dependent manner, allowing co-administered drugs to penetrate into extravascular tumor tissue. Importantly, this effect did not require the drugs to be chemically conjugated to the peptide. Systemic

injection with iRGD improved the therapeutic index of drugs of various compositions including a small molecule (doxorubicin), nanoparticles (nab-paclitaxel and doxorubicin liposomes), and a monoclonal antibody (trastuzumab). Thus, co-administration of iRGD may be a valuable way to enhance the efficacy of anti-cancer drugs while reducing their side effects, a primary goal of cancer therapy research.

Sugahara, Kazuki N et al. "Coadministration of a tumor-penetrating peptide enhances the efficacy of cancer drugs." Science (New York, N.Y.) vol. 328,5981 (2010)

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Kisspeptin-10

Purity: >98% (HPLC on request) | Molecular Formula: C₆₃H₈₃N₁₇O₁₄
Molecular Weight: 1302.462 g/mol | Sequence: Tyr-Asn-Trp-Asn-Ser-Phe-Gly-Leu-Arg-Phe-NH

DESCRIPTION:

Kisspeptins are a group of neuroendocrine peptides that stimulate the release of Gonadotropin Releasing Hormone (GnRH) and is involved in the regulation of developmental sex hormones at the beginning stages of puberty. There have been problems in maturation centered around receptor mutations for kisspeptin. Kisspeptins are encoded by the KISS1 gene, which was originally identified as a human metastasis suppressor gene for melanoma and breast cancer. Kisspeptins have shown therapeutic benefits regarding the upregulation of the endogenous production of Luteinizing Hormone (LH) and Follicular Stimulating Hormone (FSH) through the HPA axis. Thus, it can stimulate

Leydig cells to produce testosterone without the result of hypogonadism shown with exogenous testosterone usage. The expression of Kiss1 has also been altered in other situations of energy imbalance such as obesity and diabetes. It has also been shown to reverse the effects of hypogonadotropic hypogonadism. It also shows other physiologic effects such as helping with egg implantation and maturation in reproduction, as well as the prevention of ectopic pregnancy. Further, in the kidneys it has been shown to increase aldosterone production as well as pregnenolone breakdown and kisspeptin – angiotensin2 production.

PROTOCOL:

Content & Potency: 100mcg/ml subcutaneous injection provided in a 5ml vial.
Suggested dosage: Inject 0.10ml subcutaneously once daily.

CLINICAL RESEARCH:

Kisspeptin-10 Is a Potent Stimulator of LH and Increases Pulse Frequency in Men

Intravenous bolus kisspeptin-10 resulted in a rapid and dose-dependent rise in serum LH concentration, with maximal stimulation at 1 µg/kg (4.1 ± 0.4 to 12.4 ± 1.7 IU/liter at 30 min, P < 0.001, n = 6). Administration of 3 µg/kg elicited a reduced response vs. 1 µg/kg (P < 0.05). Infusion of kisspeptin-10 at 4 µg/kg · h for 22.5 h elicited an increase in LH from a mean of 5.4 ± 0.7 to 20.8 ± 4.9 IU/liter (n = 4; P < 0.05) and serum testosterone increased from 16.6 ± 2.4 to 24.0 ± 2.5 nmol/liter (P < 0.001). LH pulses were obscured at this high rate of

secretion, but a lower dose infusion of kisspeptin-10 (1.5 µg/kg · h) increased mean LH from 5.2 ± 0.8 to 14.1 ± 1.7 IU/liter (n = 4; P < 0.01) and increased LH pulse frequency from 0.7 ± 0.1 to 1.0 ± 0.2 pulses/h (P < 0.05) and secretory burst mass from 3.9 ± 0.4 to 12.8 ± 2.6 IU/liter (P < 0.05). Kisspeptin-10 boluses potently evoke LH secretion in men, and continuous infusion increases testosterone, LH pulse frequency, and pulse size. Kisspeptin analogues have therapeutic potential as regulators of LH and thus testosterone secretion.

George, J T et al. "Kisspeptin-10 is a potent stimulator of LH and increases pulse frequency in men." The Journal of clinical endocrinology and metabolism vol. 96,8 (2011): E1228-36. doi:10.1210/jc.2011-0089

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

KPV

Purity: >99% (confirmed by HPLC) | Molecular Formula: Ac-C16H32N4O6-NH2
Molecular Weight: 378.47 g/mol | Sequence: Lys-Pro-Val

DESCRIPTION:

KPV (Lysine-Proline-Valine) is a C-terminal tripeptide fragment of α -melanocyte stimulating hormone (α -MSH). α -MSH stimulates the production and release of melanin by melanocytes in skin and hair, acting through melanocortin 1 receptor. Several studies have previously shown that KPV has decreased inflammation and tumorigenesis in the body. The KPV anti-inflammatory effect is PepT1-mediated in intestinal epithelial and immune cells. PepT1 is an oligopeptide transporter that is overexpressed in the in the colonic epithelial cells of chronic ulcerative colitis, which can deliver KPV into cytosol in the intestine. The tripeptide, KPV has significant antimicrobial and

anti-inflammatory properties especially in those with psoriasis. Psoriasis is a chronic autoimmune condition which causes the rapid build-up of skin cells and is usually treated using hydrocortisone. In people with psoriasis, KPV has shown to limit symptoms of the condition including itchiness, dryness, redness, peeling, and more. Therefore, KPV could be used for an extended period of time without risking the unwanted complications of long term steroid therapy. KPV is a promising, therapeutic treatment for inflammatory bowel disease (IBD), colon cancer, and inflammatory skin disorders, in particular, psoriasis.

PROTOCOL:

Content & Potency: 30ml TopiClick at 15mg/ml
Suggested dosage: 0.5ml applied to area twice daily

CLINICAL RESEARCH:

Alpha-Melanocyte-Stimulating Hormone and Related Tripeptides: Biochemistry, Anti Inflammatory and Protective Effects in Vitro and in Vivo, and Future Perspectives for the Treatment of Immune-Mediated Inflammatory Diseases

Alpha-MSH is a tridecapeptide derived from proopiomelanocortin. Many studies over the last few years have provided evidence That Alpha -MSH has potent protective and antiinflammatory effects. These effects can be elicited via centrally expressed melanocortin receptors that orchestrate descending neuro-genic antiinflammatory pathways. Alpha-MSH can also exert antiinflammatory and protective effects on cells of the immune system and on peripheral nonimmune cell types expressing melanocortin receptors. At the molecular level, Alpha-MSH affects various pathways implicated in regulation of inflammation and protection, i.e., nuclear factor-B activation, expression of adhesion molecules and chemokine receptors, production of proinflammatory cytokines and mediators, IL-10 synthesis, T cell proliferation and activity, inflammatory cell migration, expression of antioxidative enzymes, and apoptosis. The antiinflammatory effects of -MSH have

been validated in animal models of experimentally induced fever; irritant and allergic contact dermatitis, vasculitis, and fibrosis; ocular, gastrointestinal, brain, and allergic airway inflammation; and arthritis, but also in models of organ injury. One obstacle limiting the use of alpha-MSH in inflammatory disorders is its pigmentary effect. Due to its preserved antiinflammatory effect but lack of pigmentary action, the C-terminal tripeptide of -MSH, KPV, has been delineated as an alternative for antiinflammatory therapy. KdPT, a derivative of KPV corresponding to amino acids 193–195 of IL-1, is also emerging as a tripeptide with antiinflammatory effects. The physicochemical properties and expected low costs of production render both agents suitable for the future treatment of immune-mediated inflammatory skin and bowel disease, fibrosis, allergic and inflammatory lung disease, ocular inflammation, and arthritis.

Endocr Rev. 2008 Aug;29(5):581-602. doi: 10.1210/er.2007-0027. Epub 2008 Jul 8. Brzoska T1, Luger TA, Maaser C, Abels C, Böhm M.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Leuphasyl

Purity: >99% (HPLC on request) | Molecular Formula: C29H39N5O7
Molecular Weight: 569.659 g/mol | Sequence: Tyr-D-Ala-Gly-Phe-Leu

DESCRIPTION:

Leuphasyl, pentapeptide-18, is a five amino acid peptide that reduces the depth of wrinkles by contraction of facial muscles. It is an enkephalin modified for enhanced stability that modulates acetylcholine activity in neuron cells and catecholamine release. Leuphasyl targets the wrinkle-formation mechanism of the expression of wrinkles in a unique way, offering an alternative to other cosmetic peptides like argireline and SNAP-8.

It is important to note that leuphasyl and argireline have a synergistic effect. For 28 days, a cream containing 5% argireline solution and a 5% leuphasyl solution was applied twice daily to the eyes of 43 volunteers. 24.62% wrinkle reduction was observed in the participants using the solution. Due to their complementary mechanisms, both peptides show additive effects. Leuphasyl indicates a proven efficacy for improving firmness and skin tone, reducing fine lines and wrinkles, and moisturizing the skin.

PROTOCOL:

Content & Potency: 5%/5% cream (with Argireline) in 30ml TopiClick
Suggested dosage: 1ml applied topically to the face daily

CLINICAL RESEARCH:

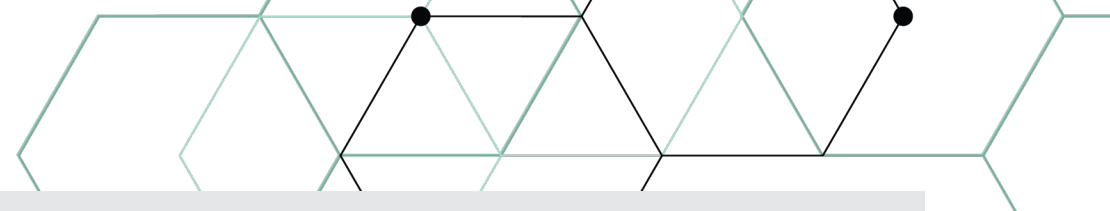
The Efficiency and Safety of Leuphasyl—A Botox-Like Peptide

Peptides of synthesis are a very new strategy in cosmetic science and technology for at least two reasons: (1) they are small molecules, easily penetrable in the skin and (2) they are able to induce a very specific action, because all skin cells (keratinocytes, fibroblasts, nervous cells) have membrane receptors for peptides. This group of cosmeceutics includes the botox-like peptides, represented by acetyl hexapeptide 3 (Argireline) and pentapeptid-3 (Leuphasyl). The latter is less known and has been less studied. This substance inhibits the neuromuscular synapses in the mimic muscles, acting as enkephalins. It links the enkephalin receptor to nervous cells, thereby modulating the release of acetylcholine in synaptic space. This cellular activity will be translated in vivo in a relaxation of the

muscle and a reduction of expression wrinkles. The aim of our study is to evaluate the optimal concentration of Leuphasyl for skin application at the mimic muscle level, the efficiency and the safety of this peptide. We formulated three emulsions of different concentrations (0.5%, 1%, 2%) which were applied to the skin, at the level of mimic muscles (1) at the eyebrows zone (above the corrugator supercilii muscle) and (2) at the periorbital zone (above the orbicularis oculi muscle). We evaluated the regression of the wrinkles between the eyebrows using an imagistic method: pro-derm Analyser. The study is of interest to discussions concerning how to apply these kinds of cosmetic products at the mimic muscle skin level and not at the level of the wrinkles.

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LL-37

Melanotan II

Purity: >98% (HPLC on request) | Molecular Formula: C205H340N60O53
Molecular Weight: 4493.33 g/mol | Sequence: Leu-Leu-Gly-Asp-Phe-Phe-Arg-Lys-Ser-Lys-Glu-Lys-Ile-Gly-Lys-Glu-Phe-Lys-Arg-Ile-Val-Gln-Arg-Ile-Lys-Asp-Phe-Leu-Arg-Asn-Leu-Val-Pro-Arg-Thr-Glu-Ser

DESCRIPTION:

LL-37 is an antimicrobial peptide which belongs to the cathelicidin family of AMPs (antimicrobial peptides). LL-37, like cathelicidins, are stored in neutrophil granules as inactive precursors and are released as mature peptides when neutrophils are stimulated. LL-37 is expressed in various cells and tissues such as circulating neutrophils and myeloid bone marrow cells, epithelial cells of the skin, and is also expressed in the gastrointestinal tract, as well as in the epididymis and lungs. Moreover, production of LL-37 in macrophages is stimulated by vitamin D released by sunlight

through the skin. LL-37 plays an important role in the first line of defense against infection and systemic invasion of pathogens at sites of inflammation and wound. It is cytotoxic to both bacterial and normal eukaryotic cells and is significantly resistant to proteolytic degradation in solution. LL-37 shows a broad spectrum of antimicrobial activity against bacteria, enveloped viruses, and fungi. It has also demonstrated success in helping promote wound healing but it may play a negative role in atopic dermatitis and psoriasis.

PROTOCOL:

Content & Potency: 2000mcg/ml subcutaneous injection provided in a 5ml vial.
Suggested dosage: Varies with indication and patient.

CLINICAL RESEARCH:

Membrane Core-Specific Antimicrobial Action of Cathelicidin LL-37 Peptide Switches Between Pore and Nanofibre Formation

Membrane-disrupting antimicrobial peptides provide broad-spectrum defence against localized bacterial invasion in a range of hosts including humans. The most generally held consensus is that targeting to pathogens is based on interactions with the head groups of membrane lipids. Here we show that the action of LL-37, a human antimicrobial peptide switches the mode of action based on the structure of the alkyl chains, and not the head groups of

the membrane forming lipids. We demonstrate that LL-37 exhibits two distinct interaction pathways: pore formation in bilayers of unsaturated phospholipids and membrane modulation with saturated phospholipids. Uniquely, the membrane modulation yields helical-rich fibrous peptide-lipid superstructures. Our results point at alternative design strategies for peptide antimicrobials.

Shahmiri, Mahdi & Enciso, Marta & Adda, Christopher & Smith, Brian & Perugini, Matthew & Mechler, Adam. (2016). Membrane Core-Specific Antimicrobial Action of Cathelicidin LL-37 Peptide Switches Between Pore and Nanofibre Formation. Scientific Reports.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Purity: >98% (HPLC on request) | Molecular Formula: C50H69N15O9
Molecular Weight: 1024.2 | Sequence: Ac-Nle-Asp-His-D-Phe-Arg-Trp-Lys-NH₂(cyclic 2-7)

DESCRIPTION:

Melanotan I and Melanotan II are both analogs of the peptide hormone alpha-melanocyte-stimulating hormone (α -MSH) that induces skin tanning. Like its predecessor, Melanotan I, MT 2 plays a role in stimulating melanogenesis and thus provides a protective mechanism against UV rays since under its actions melanocytes are able to increase production and secretion of the

hormone melanin. Scientists have also noticed that MT 2 had a positive effect on libido due to its aphrodisiac properties. Additionally, MT 2 exhibits a mild positive fat-mobilizing effect. Melanotan I is an FDA approved drug under the brand name Scenesse. Scenesse is most commonly used to treat patients that have an intolerance to light.

PROTOCOL:

Content & Potency: 2000mcg/ml subcutaneous injection provided in a 5ml vial.
Suggested dosage: Inject 0.15ml once daily for 1 - 2 weeks then 0.25mL twice weekly thereafter for maintenance.
For CIRS: Inject 100mcg every other day for 40 days.

CLINICAL RESEARCH:

Evaluation of melanotan-II, a super potent cyclic melanotropic peptide in a pilot phase-I clinical study.

A pilot phase I study was conducted with a cyclic heptapeptide analog of alpha-melanocyte stimulating hormone (alpha-MSH). The lactam-bridged molecule, called Melanotan-II (MT-II), has the structure Ac-Nle4-Asp5-His6-D-Phe7-Arg8-Trp9-Lys10 alpha-MSH4-10-NH₂ (MT-II) and has superpotent melanotropic activity in vitro. A single-blind, alternating day (saline or MT-II), placebo-controlled trial was conducted in 3 normal male volunteers at the starting dose of 0.01 mg/kg of MT-II. Subcutaneous injections of MT-II or saline were given daily (Monday-Friday) for 2 consecutive weeks. Two subjects were escalated by 0.005 mg/kg increments to 0.03 mg/kg and one to 0.025 mg/kg. The 0.03 mg/kg dose produced Grade

II somnolence and fatigue in one of two subjects (WHO standards). Mild nausea, not requiring antiemetic treatment, was reported at most MT-II dose levels. A stretching and yawning complex appeared to correlate with the onset of spontaneous, Penile erections which were intermittently experienced for 1-5 hours after MT-II dosing, depending on the MT-II dose. Two subjects had increased pigmentation in the face, upper body and buttock, as measured by quantitative reflectance and by visual perception 1 week after MT-II dosing ended. These results demonstrate that MT-II has tanning activity in humans given only 5 low dose every other day by subcutaneous injection. The recommended single MTII dose for future Phase I studies is 0.025 mg/kg/day.

T. Dorr, Robert & Lines, Ruskin & Levine, Norman & Brooks, Chris & Xiang, Li & Hruby, Victor & E. Hadley, Mac. (1996). Evaluation of Melanotan-II, a superpotent cyclic melanotropic peptide in a pilot phase-I clinical study. Life sciences. 58. 1777-84.

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Met-Enkephalin

Purity: >95% (confirmed by HPLC) | Molecular Formula: C₂₇H₃₅N₅O₇S
Molecular Weight: 573.66 g/mol | Sequence: H-Tyr-Gly-Gly-Phe-Met-OH

DESCRIPTION:

In 1975, Met-Enkephalin (ME) was first isolated from porcine brain matter. ME is an endogenous pentapeptide with antagonist activity at the μ and δ opioid receptors. It is one of two forms of enkephalin, the other form being leu-enkephalin. Essentially, this peptide functions as a neurotransmitter or neuromodulator in the central nervous system (CNS). Opioid receptors play a role in numerous physiological processes in the body including pain mediation, opiate dependence, and euphoria. ME acts as a cytokine, a small secreted protein released by cells which have a specific effect on the interactions and communications between cells, and has demonstrated to increase immune functions at low concentration while suppressing at high concentration. In addition, ME behaves as an opioid growth factor (OGF) on many cell types as a

receptor that is distinct from the neural opioid receptors. OGF activates a specific receptor called the opioid growth factor receptor (OGFr or ζ -opioid receptor). The OGF and OGFr axis regulates cell growth in normal and abnormal cells. It has been concluded that ME could potentially be used as a drug to treat cancer and work as a strong immune booster. Modulation of the OFG-OGFr receptor axis represents a promising and therapeutic avenue for effective treatment such as cancer (hepatoblastoma, breast, colon, renal, ovarian, pancreatic, melanoma and many others), autoimmune encephalomyelitis, and multiple sclerosis. Low-dose naltrexone (LDN) actually functions by increasing endogenous levels of enkephalins. As a result, it is postulated that Met-enkephalin can also be used as an alternative to LDN.

PROTOCOL:

Content & Potency: 10mg Lyophilized Vial
Suggested dosage: 10mg IV once weekly

CLINICAL RESEARCH:

Opioid growth factor improves clinical benefit and survival in patients with advanced pancreatic Cancer

Background: Advanced pancreatic cancer carries the poorest prognosis of all gastrointestinal malignancies. Once the tumor has spread beyond the margins of the pancreas, chemotherapy is the major treatment modality offered to patients; however, chemotherapy does not significantly improve survival.

Objective: Opioid growth factor (OGF; [Met5]-enkephalin) is a natural peptide that has been shown to inhibit the growth of pancreatic cancer in cell culture and in nude mice. The purpose of this study was to evaluate the effects of OGF biotherapy on subjects with advanced pancreatic cancer who failed chemotherapy.

Methods: In a prospective phase II open-labeled clinical trial, 24 subjects who failed standard chemotherapy for advanced pancreatic cancer were treated weekly with OGF

250 μ g/kg intravenously. Outcomes measured included clinical benefit, tumor response by radiographic imaging, quality of life, and survival.

Results: Clinical benefit response was experienced by 53% of OGF-treated patients compared to historical controls of 23.8% and 4.8% for gemcitabine and 5-fluorouracil 5-FU), respectively. Of the subjects surviving more than eight weeks, 62% showed either a decrease or stabilization in tumor size by computed tomography. The median survival time for OGF-treated patients was three times that of untreated patients (65.5 versus 21 days, p 0.001). No adverse effects on hematologic or chemistry parameters were noted, and quality of life surveys suggested improvement with OGF.

Jill P Smith, Sandra I Bingaman, David T Mauger, Harold H Harvey, Laurence M Demers, Ian S Zagon
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MOTS-c

Purity: >98% (HPLC on request) | Molecular Formula: C₁₀₁H₁₅₂N₂₈O₂₂S₂. C₂H₃F₃O₂.
Molecular Weight: 2288.6 g/mol | Sequence: MRWQEMGYIFYPKLR

DESCRIPTION:

Human mitochondrial DNA (mtDNA) encodes 37 known genes, including 2rRNAs, 22 tRNAs and 13 polypeptide subunits of the electron transport chain (ETC) complexes. Recent work has revealed that the rRNA loci contain small open reading frames (ORFs) that can be transcribed and translated into short peptides called mitochondrial-derived peptides (MDPs), which have biological activity. MOTS-c is a mitochondrial-encoded peptide with 16-aa's encoded within the 12S rRNA locus of mtDNA in human cells.

MOTS-c can translocate into the nucleus in response to metabolic stress and regulation of adaptive nuclear gene expression. This allows the peptide to promote resistance of metabolic stress by upregulating the mitochondrial genome. Upregulating these genes encourages mitochondrial biogenesis. MOTS-c inhibits the methionine-folate cycle resulting in purine synthesis, increase in PCG-1 α (a key regulator

of energy metabolism), and AICAR (5-Aminoimidazole-4-carboxamide ribonucleotide) accumulation which activates AMPK (5'- adenosine monophosphate-activated protein kinase). This acts as an energy sensor by monitoring the ratio of AMP and ATP. AMPK restores homeostasis by initiating catabolic processes for ATP production in case of energy deficits. In addition, literature suggest that MOTS-c decreases insulin resistance and increases GLUT4 uptake in muscle.

The peptide is mainly used for weight loss (regulating muscle and fat metabolism) and energy (cell survival in toxic conditions). MOTS-c is consistently used by sports performance athletes to enhance one's performance. It also displays a promising effect in longevity. The Japanese long-lived people (population with the longest lifespan in the world) have demonstrated a phenotypic expression and biological link between MOTS-c and an extended lifespan.

PROTOCOL:

Content & Potency: 10mg/ml solution provided in a 4ml vial.
Suggested dosage: Inject 10mg subcutaneously weekly into the abdomen.

CLINICAL RESEARCH:

The Mitochondrial-Derived Peptide MOTS-c Promotes Metabolic Homeostasis and Reduces Obesity and Insulin Resistance

Mitochondria are known to be functional organelles, but their role as a signaling unit is increasingly being appreciated. The identification of a short open reading frame (sORF) in the mitochondrial DNA (mtDNA) that encodes a signaling peptide, humanin, suggests the possible existence of additional sORFs in the mtDNA. Here we report a sORF within the mitochondrial 12S rRNA encoding a 16-amino-acid peptide named MOTS-c (mitochondrial open reading frame of the 12S rRNA-c) that regulates insulin sensitivity and metabolic

homeostasis. Its primary target organ appears to be the skeletal muscle, and its cellular actions inhibit the folate cycle and its tethered de novo purine biosynthesis, leading to AMPK activation. MOTS-c treatment in mice prevented age-dependent and high-fat-diet-induced insulin resistance, as well as diet-induced obesity. These results suggest that 1 mitochondria may actively regulate metabolic homeostasis at the cellular and organismal level via peptides encoded within their genome.

Lee, Changhan & Zeng, Jennifer & G. Drew, Brian & Sallam, Tamer & Martin-Montalvo, Alejandro & Wan, Junxiang & Kim, Su-Jeong & Mehta, Hemal & Hevener, Andrea & Cabo, Rafael & Cohen, Pinchas. (2015). The Mitochondrial-Derived Peptide MOTS-c Promotes Metabolic Homeostasis and Reduces Obesity and Insulin Resistance. Cell metabolism. 21. 443-454.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

MK-677

Purity: >98% (HPLC on request) | Molecular Formula: C₂₇H₃₆N₄O₅S
Molecular Weight: 528.668 g/mol | Sequence: Non-peptide

DESCRIPTION:

MK-677 is a long active orally bioactive agonist of the GHS-R1a. As such, binds to the same receptor that GHRP2, GHRP6, and Ipamorelin also stimulate. Also called Ibutamoren, it has shown to cause a predictable rise in IGF-1 but unlike other GH secretagogues doesn't help to

decrease adipose tissue. It has been shown to increase lean muscle mass and might be a good candidate for sarcopenic patients with low bone mineral density. In order to reduce the negative effect of somatostatin, MK-677 is best taken on an empty stomach with no insulin in the system.

PROTOCOL:

Content & Potency: 25mg capsules provided in quantities of 30.
Suggested dosage: Take one capsule by mouth once daily on an empty stomach.

CLINICAL RESEARCH:

Effects of an Oral Ghrelin Mimetic on Body Composition and Clinical Outcomes in Healthy Older Adults A Randomized Trial

Daily administration of MK-677 significantly increased growth hormone and insulin-like growth factor I levels to those of healthy young adults without serious adverse effects. Mean fat-free mass decreased in the placebo group but increased in the MK-677 group (change, 0.5 kg [95% CI, 1.1 to 0.2 kg] vs. 1.1 kg [CI, 0.7 to 1.5 kg], respectively; P 0.001), as did body cell mass, as reflected by intracellular water (change, 1.0 kg [CI, 2.1 to 0.2kg] vs. 0.8 kg [CI, 0.1 to 1.6 kg], respectively; P 0.021). No significant differences were observed in abdominal visceral fat or total fat mass; however, the average increase in limb fat was greater in the MK-677 group than the placebo group (1.1 kg vs. 0.24 kg; P 0.001). Body weight increased 0.8 kg (CI, 0.3 to 1.8 kg) in the placebo group and 2.7 kg (CI, 2.0 to 3.5 kg) in the MK-677 group (P 0.003). Fasting blood glucose level increased an average of 0.3

mmol/L (5 mg/dL) in the MK-677 group (P 0.015), and insulin sensitivity decreased. The most frequent side effects were an increase in appetite that subsided in a few months and transient, mild lower-extremity edema and muscle pain. Low-density lipoprotein cholesterol levels decreased in the MK-677 group relative to baseline values (change, 0.14 mmol/L [CI, 0.27 to 0.01 mmol/L]; 5.4 mg/dL [CI, 10.4 to 0.4 mg/dL]; P 0.026); no differences between the groups were observed in total or high-density lipoprotein cholesterol levels. Cortisol levels increased 47 nmol/L (CI, 28 to 71 nmol/L (1.7 g/dL [CI, 1.0 to 2.6 g/dL]) in MK-677 recipients (P 0.020). Changes in bone mineral density consistent with increased bone remodeling occurred in MK-677 recipients. Increased fat-free mass did not result in changes in strength or function. Two-year exploratory analyses confirmed the 1-year results.

Nass, Ralf et al. "Effects of an oral ghrelin mimetic on body composition and clinical outcomes in healthy older adults: a randomized trial." *Annals of internal medicine* vol. 149,9 (2008): 601-11.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Myristyl

Purity: >98% (HPLC on request) | Molecular Formula: CH₃(CH₂)₁₂CO-hE18A-NH₂
Molecular Weight: 3805.67 g/mol | Sequence: Myr-Leu-Arg-Arg-Leu-Arg-Arg-Arg-Leu-Leu-Arg-Asp-Trp-Leu-Lys-Ala-Phe-Tyr-Asp-Lys-Val-Ala-Glu-Lys-Leu-Lys-Glu-Ala-Phe-NH₂

DESCRIPTION:

The hepatic uptake of LDL is highly regulated. The LDL receptor (LDL-R) present on hepatocytes mediates the endocytosis of LDL and thus regulates the plasma level of the lipoprotein and cholesterol. In the acidic environment of the endosome, the LDL-R dissociates from its ligand and recycles back to the cell surface for further uptake of LDL. Apo E is an alternate ligand for the LDL-R and mediates the clearance of triglyceride-rich lipoproteins such as chylomicron remnants and VLDL (2). ApoE also binds to additional hepatic receptors such as LDL-R related protein (LRP) and heparan sulfate proteoglycans

(HSPG). Like apoE, Ac-hE18A-NH₂ and its variants can reduce plasma cholesterol and displays anti-inflammatory properties in model animals. Ac-hE18A-NH₂ has already undergone phase 1 clinical trials as a lipid-lowering agent. In a recent study, the myristyl peptide variant of ApoE has been shown to reduce total and LDL cholesterol (even under severe dyslipidemic conditions) in apoE-null mice. As a result, it is though myristyl can act as an alternative to statins and HMG-CoA reductase inhibitors. Additionally, because of its enhanced potency at lower doses, myristyl has great therapeutic potential to lower cholesterol.

PROTOCOL:

Content & Potency: 5ml vial at 6mg/ml
Suggested dosage: 100mcg/kg once weekly intravenously

CLINICAL RESEARCH:

Novel Fatty Acyl ApoE Mimetic Peptides Have Increased Potency To Reduce Plasma Cholesterol In Mice And Macaques

Ac-hE18A-NH₂ is a dual-domain apoE mimetic peptide that possesses the putative receptor binding domain from apoE (LRKLRKLLR, denoted hE, residues 141 to 150) covalently attached to lipid-associating peptide 18A. Like apoE, Ac-hE18A-NH₂ reduces plasma cholesterol in animal models and exhibits anti-inflammatory properties independent of its cholesterol-reducing effect. Ac-hE18A-NH₂ has already undergone phase I clinical trials as a lipid-lowering agent. To explore the therapeutic potential, we designed and synthesized new analogs by linking α -aminohexanoic acid, octanoic acid, or myristic acid to LRRLRRLLR-18A-NH₂ ([R]hE18A-NH₂) and examined the cholesterol-lowering potency in animals. The modified peptides effectively reduced plasma cholesterol in

apoE-null mice fed standard chow or a Western diet; the myristyl analog was the most effective. A single administration of the myristyl analog reduced plasma total and LDL cholesterol in a dose-dependent manner in hypercholesterolemic cynomolgus macaques for up to 1 week despite continuation of a cholesterol-supplemented diet. The myristyl peptide (7.4 mg/kg) reduced total and LDL cholesterol at 24 hours by 64% and 74%, respectively; plasma HDL levels were modestly reduced and returned to baseline by the seventh day. These new analogs should exhibit enhanced potency at lower doses than Ac-hE18A-NH₂, which may make them attractive therapeutic candidates for clinical trials.

Gattadahalli M, Anantharamaiah, David W. Garbera, Dennis Goldberg, Eric Morrel, Geeta Datta, Mayakonda N. Palgunacharia, Thomas C. Register, Susan E. Appt, and C. Roger Whitea* Department of Medicine, University of Alabama at Birmingham Medical Center, Birmingham, Alabama 35294, Lipimetix Development, Inc. 5 Commonwealth Rd. Suite 2A, Natick, MA 01760, Wake Forest School of Medicine, Winston-Salem, NC 27157

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

NMN

Purity: >97% (HPLC on request) | Molecular Formula: C₃₂H₄₂N₉O₂₂P₃
Molecular Weight: 997.65 g/mol | Sequence: Non-peptide

DESCRIPTION:

Nicotinamide adenine dinucleotide (NAD⁺) and its reduced form NADH are essential cofactors for multiple cellular metabolic reactions and has a central role in energy production. Nicotinamide mononucleotide (NMN) is a key NAD⁺ intermediate that has been shown to enhance NAD⁺ biosynthesis. NMN has suppressed age-associated body weight gain, enhanced energy metabolism, promoted physical activity, improved insulin sensitivity and plasma lipid

profile, enhanced eye function and other pathophysiologies. It has also prevented age-associated gene expression changes in key metabolic organs and enhanced mitochondrial oxidative metabolism and mitochondrial protein imbalance in skeletal muscle. NAD⁺ with the addition of NMN enhances physical performance and serves as an effective anti-aging intervention in humans.

PROTOCOL:

Content & Potency: 10ml vial at 200mg/ml
Suggested dosage: 200mg injected subcutaneously twice daily.

CLINICAL RESEARCH:

Nicotinamide Mononucleotide: Exploration of Diverse Therapeutic Applications of a Potential Molecule

Nicotinamide mononucleotide (NMN) is a nucleotide that is most recognized for its role as an intermediate of nicotinamide adenine dinucleotide (NAD⁺) biosynthesis. Although the biosynthetic pathway of NMN varies between eukaryote and prokaryote, two pathways are mainly followed in case of eukaryotic human—one is through the salvage pathway using nicotinamide while the other follows phosphorylation of nicotinamide riboside. Due to the unavailability of a suitable transporter, NMN enters inside the mammalian cell in the form of nicotinamide riboside followed by its subsequent conversion to NMN and NAD⁺. This particular molecule has demonstrated several beneficial

pharmacological activities in preclinical studies, which suggest its potential therapeutic use. Mostly mediated by its involvement in NAD⁺ biosynthesis, the pharmacological activities of NMN include its role in cellular biochemical functions, cardioprotection, diabetes, Alzheimer's disease, and complications associated with obesity. The recent groundbreaking discovery of anti-aging activities of this chemical moiety has added a valuable essence in the research involving this molecule. This review focuses on the biosynthesis of NMN in mammalian and prokaryotic cells and mechanism of, absorption along with the reported pharmacological activities in murine model.

Saikat Kumar Poddar,1,* Ali Ehsan Sifat,1 Sanjana Haque,1 Noor Ahmed Nahid,1 Sabiha Chowdhury,1 and Imtias Mehedi2

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



PEG-MGF

Purity: >98% (HPLC on request) | Molecular Formula: C₁₂₁H₂₀₀N₄₂O₃₉
Molecular Weight: 2888.16 | Sequence: Tyr-Gln-Pro-Pro-Ser-Thr-Asn-Lys-Asn-Thr-Lys-Ser-Gln-Arg-Arg-Lys-Gly-Ser-Thr-Phe-Glu-Glu-Arg-Lys

DESCRIPTION:

MGF is a split variant of IGF-1 but its sequence differs from the systemic IGF-1 produced by the liver. IGF-1 is spliced towards MGF which initiates hypertrophy and repair of local muscle damage. MGF is expressed by mechanically overloaded muscle and is involved in tissue repair and adaptation. It is expressed as a pulse following muscle damage and is involved in the activation of muscle satellite (stem) cells. These donate nuclei to the muscle fibers that are

required for repair and for the hypertrophy process, which may have similar regulatory mechanisms. MGF is essential for repair and therefore growth of new cells, similar to IGF-1. If MGF is not PEGylated, the half-life lasts only minutes therefore PEGylated MGF must be considered during the compounding process to ensure an appropriate half-life, thereby increasing duration of action.

PROTOCOL:

Content & Potency: 2000mcg/ml subcutaneous injection provided in a 5ml vial.
Suggested dosage: Inject 0.10ml to 0.20ml once daily for 5 out of 7 days of the week.

CLINICAL RESEARCH:

Expression of IGF-1 Isoforms after Exercise-induced Muscle Damage in Humans: Characterization of the MGF E Peptide Actions In Vitro

Different insulin-like growth factor-1 (IGF-1) isoforms, namely IGF-1Eb and IGF-1Ec (MGF), have been proposed to have various functions in muscle repair and growth. To gain insight into the potentially differential actions of IGF-1 isoforms in the regulation of muscle regeneration, we assessed the time course of their expressions at both mRNA and protein levels after exercise-induced muscle damage in humans. In addition, we characterized mature IGF-1 and synthetic MGF E peptide signalling in C2C12 myoblast-like cells in vitro. Ten healthy male volunteers were subjected to exercise-induced muscle damage and biopsy samples were taken from the exercised muscles before and 6 h, 2,5 and 16 days post exercise. Muscle damage was documented by specific functional and biochemical responses post exercise. PCR-based analyses of muscle biopsy samples revealed a rapid and transient

up-regulation of MGF mRNA expression which was followed by a prolonged increase of IGF-1Ea and IGF-1Eb mRNA expression (p<0.05). Patterns similar to those for mRNA expression were detected for MGF and IGF-1Ea expression at the protein level. The action of synthetic MGF E peptide differed from that of mature IGF1 since its proliferative effect on C2C12 myoblast-like cells was not blocked by an anti-IGF-1 receptor neutralizing antibody and it did not phosphorylate Akt. Therefore, we conclude that the differential expression profile of IGF-1 isoforms in vivo and the possible IGF-1R-independent MGF E peptide signalling in skeletal muscle-like cells in vitro support the notion that tissue-specific mRNA expression of MGF isoform produces mature IGF-1 and MGF E peptides which possibly act as distinct mitogens in skeletal muscle regeneration.

Philippou, Anastassios & Papageorgiou, E & Bogdanis, Gregory & Halapas, Antonios & Sourla, A & Maridaki, Maria & Pissimissis, Nikolas & Koutsilieris, Michael. (2009). Expression of IGF-1 Isoforms after Exercise-induced Muscle Damage in Humans: Characterization of the MGF E Peptide Actions In Vitro. In vivo (Athens, Greece). 23. 567-75.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Pentosan Polysulfate

PNC-27

Purity: >98% (HPLC on request) | Molecular Formula: (C5H6Na2O10S2)n
Molecular Weight: 602.473 g/mol | Sequence: Non- peptide

DESCRIPTION:

Pentosan polysulfate is a semi-synthetic polysulfated xylan used for the relief of Osteoarthritis. The mechanism of PPS action in osteoarthritis is multifactorial, with both stimulation of cartilage matrix synthesis and prevention of cartilage breakdown. There are also systemic effects on blood lipid and fibrinolysis

that may help clear the subchondral circulation. In one study, after a series of four to six intra- articular PPS injections into knees of human volunteers, there was a significant increase in the size of the synovial fluid hyaluronan without causing any inflammation or bleeding into the joint cavity.

PROTOCOL:

Content & Potency: 250mg/ml solution provided in a 10ml vial.
Suggested dosage: Inject 2mg/kg subcutaneously twice weekly for 6 weeks.

CLINICAL RESEARCH:

Intra-articular injection pentosanpolysulphate results in increased hyaluronan molecular weight in joint fluid.

The influence of an oversulphated glycosaminoglycan, pentosanpolysulphate, on hyaluronan metabolism of the synovial lining cell was studied in vivo in human volunteers. Significant increases in the mean degree of polymerisation of the hyaluronan chains were observed

after a series of four to six intra-articular injections of this glycosaminoglycan. No increases in hyaluronan synthesis rates were observed. Repeated administration of the drug did not cause any inflammation or bleeding in the joint cavity.

Clin Exp Rheumatol. 1992 May-Jun;10(3):249-54.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Purity: >98% (HPLC on request) | Molecular Formula: C188H293N53O44S
Molecular Weight: 4031.7 g/mol | Sequence: PPLSQETFSDLWKLL

DESCRIPTION:

PNC-27 is a membrane active anticancer peptide that has been found to kill cancer cells by inducing membranoylsis via cellular necrosis. It has been designed to bind tightly to the p53-binding pocket on the mdm2 protein, a negative regulator of the P53 tumor suppressor. Almost all cancers have a mechanism to decrease the functionality of P53 which can stop cellular replication. P53 is usually not expressed in high degrees in normal cells. Through blocking its inhibition via mdm2 protein modulators, we can make sure P53 is expressed. Thus, cancer cells can

be selectively targeted for necrosis and death. This complex works in cancer cell membranes. Together, PNC-27 and Mdm2 result in trans-membrane pore formation which results in cancer cell death. This is evident in literature including studies on P53-null K562 in leukemia cells, melanoma, pancreatic cancer, breast cancer, epithelial ovarian cancer, and additional cancers. Essentially, the peptide has been found to be cytotoxic to human cancer cells while having no effect on healthy cells and is functional almost across all cancer cell types.

PROTOCOL:

Content & Potency: 5mg/ml solution provided in a 5mL vial.
Suggested dosage: Inject 0.2ml to 0.4mL subcutaneously three times daily.

CLINICAL RESEARCH:

Anticancer peptide PNC-27 adopts an HDM-2-binding conformation and kills cancer cells by binding to HDM-2 in their membranes.

The anticancer peptide PNC-27, which contains an HDM-2-binding domain corresponding to residues 12-26 of p53 and a transmembrane-penetrating domain, has been found to kill cancer cells (but not normal cells) by inducing membranolysis. We find that our previously determined 3D structure of the p53 residues of PNC-27 is directly superimposable on the structure for the same residues bound to HDM-2, suggesting that the peptide may target HDM-2 in the membranes of cancer cells. We now find significant levels of HDM-2 in the membranes of a variety of cancer cells but not in the membranes of several

untransformed cell lines. In colocalization experiments, we find that PNC-27 binds to cell membrane-bound HDM-2. We further transfected a plasmid expressing full-length HDM-2 with a membrane-localization signal into untransformed MCF-10-2A cells not susceptible to PNC-27 and found that these cells expressing full-length HDM-2 on their cell surface became susceptible to PNC-27. We conclude that PNC-27 targets HDM-2 in the membranes of cancer cells, allowing it to induce membranolysis of these cells selectively.

Sarafraz-Yazdi, Ehsan et al. "Anticancer peptide PNC-27 adopts an HDM-2-binding conformation and kills cancer cells by binding to HDM-2 in their membranes." Proceedings of the National Academy of Sciences of the United States of America vol. 107,5 (2010): 1918-23.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

PT-141

Purity: >98% (HPLC on request) | Molecular Formula: C₅₀H₆₈N₁₄O₁₀
Molecular Weight: 1025.2 | Sequence: Ac-Nle-cyclo[Asp-His-D-Phe-Arg-Trp-Lys]-OH

DESCRIPTION:

Bremelanotide(PT-141) was developed from the peptide hormone Melanotan II. In initial testing, Melanotan II induced darkening of skin pigment, but additionally caused sexual arousal and spontaneous erections as unexpected side effects in nine out of the ten original male volunteer test subjects. Further testing in animals showed Bremelanotide to induce lordosis (a sexual mating behavior) and subsequently tested for its effect in humans. Although, most of the research has been targeted to women with female sexual dysfunction an it is effective medication in treating sexual dysfunction in both men (erectile dysfunction or impotence) and women (sexual

arousal disorder). Unlike Viagra and other related medications, it does not act upon the vascular system, but directly increases sexual desire via the nervous system. It is estimated that 43% of women (30 million in the US) suffer from sexual dysfunction and 30 million men suffer from ED, with incidence increasing 2-3 fold between ages 40-70. Bremelanotide currently has no contraindications and is 80% effective in men don't respond to Viagra or Cialis. For women, it causes a 50% increase in sexually satisfying experiences. If FDA approved, it will have the trade name Ayleesi.

PROTOCOL:

Content & Potency: 10mg/ml subcutaneous injection provided in a 2ml vial.
Suggested dosage: Inject 0.2mL subcutaneously as needed, 30 minutes to 6 hours prior to sexual activity. The initial dose will establish a time frame for response. Men should start at 0.1ml and titrate up to and not exceed 0.2ml. Women should start at 0.2ml dosing protocol.

CLINICAL RESEARCH:

Melanocortins in the treatment of male and female sexual dysfunction.

Melanocortinergic agents are currently being investigated for a possible therapeutic role in male and female sexual dysfunction. These investigations were sparked by findings that systemic administration of a synthetic analog of alpha-MSH, MT-II, causes penile erections in a variety of species, including humans. Several other melanocortinergic agents including HP-228, THIQ, and bremelanotide (PT 141) have since been shown to have erectogenic properties thought to be due to binding to melanocortin receptors in the central nervous system, particularly the hypothalamus. Bremelanotide, a nasally administered synthetic peptide, is the only melanocortinergic agent that has been clinically studied in both males and females. Data from Phase II clinical trials of bremelanotide support the use of melanocortin based

therapy for erectile dysfunction. Studies using animal models have demonstrated that pre-copulatory behaviors in female rats analogous to sexual arousal are evoked, and preliminary clinical data also suggest a role in promoting sexual desire and arousal in women. Based on bremelanotide clinical experience, administration of a melanocortin agonist is well tolerated and not associated with the hypotension observed with phosphodiesterase-5 inhibitors currently used to treat erectile dysfunction. This review discusses investigations of melanocortin agonists for the treatment of sexual dysfunction with emphasis on proposed sites and mechanisms of action in the central nervous system that appear to be involved in melanocortinergic modulation of sexual function.

Shadiack, Annette & D Sharma, Shubh & C Earle, Dennis & Spana, Carl & J Hallam, Trevor. (2007). Melanocortins in the Treatment of Male and Female Sexual Dysfunction. Current topics in medicinal chemistry. 7. 1137-44.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



PTD-DBM

Purity: >98% | Molecular Formula: C₁₂₄H₂₂N₆₁O₂₈S₂
Molecular Weight: 3082.65 | Sequence: RRRRRRRRGGGGRKTGHQICKFRKC

DESCRIPTION:

PTD.-DBM is a topical hair product which helps activate the Wnt-Beta-catenin pathway via inhibition of CXXC5. This articular pathway has been proven to help rescue DHT induced hair follicle miniaturization. CXXC-type zinc finger protein 5 (CXXC5) is a negative regulator of the Wnt/ β -catenin pathway which has been associated with hair restoration and wound healing. Follicle development and formation can be impeded by CXXC5 binding with the protein Dishevelled. PTD-DBM is a very

recently created peptide which interferes with the binding process of CXXC5 and Dishevelled. Studies have proven that PTD-DBM is significantly more effective at inducing hair neogenesis when combined with Valproic Acid, which stimulates the Wnt/ β -catenin pathway when applied topically. This combination should be applied post-microneedling to take advantage of the follicle development that is induced naturally by the wound healing process.

PROTOCOL:

Content & Potency: 0.5% solution in a 20ml spray bottle.
Suggested dosage: Apply topically to area of intended hair regrowth once daily.

CLINICAL RESEARCH:

CXXC5 is a negative-feedback regulator of the Wnt/ β -catenin pathway involved in osteoblast differentiation

The positive roles of the Wnt/ β -catenin pathway in osteoblast differentiation and bone mineral density (BMD) maintenance have been clearly demonstrated in both animal experiments and clinical investigations. CXXC finger protein 5 (CXXC5), a recently identified negative regulator of the Wnt/ β -catenin pathway, showed altered cellular localization and function, which were dependent on the cell type in previous studies. However, the in vivo function of CXXC5 has not been clearly investigated yet. Here, we characterized CXXC5 as a negative regulator of osteoblast differentiation and bone formation. Deficiency of CXXC5 resulted in elevated BMD in mice without any

severe gross developmental abnormalities. CXXC5 exerted a negative-feedback effect on the Wnt/ β -catenin pathway via Wnt-dependent binding to Dishevelled (Dvl) during osteoblast differentiation. Suppression of the Dvl-CXXC5 interaction using a competitor peptide resulted in the activation of the Wnt/ β -catenin pathway and osteoblast differentiation, and accelerated thickness growth of ex vivo-cultured calvariae. Overall, CXXC5 is a negative-feedback regulator induced by Wnt/ β -catenin signaling that inhibits osteoblast differentiation and bone formation via interaction with Dvl.

CXXC5 is a negative-feedback regulator of the Wnt/ β -catenin pathway involved in osteoblast differentiation

Kim, Hyun-Yi & Yoon, Juyong & Yun, J-H & Cho, K-W & Lee, S-H & Rhee, Yumie & Jung, H-S & Lim, Hwan Jung & Lee, Hyuk & Choi, J & Heo, J-N & Lee, W & No, Kyoung Tai & Min, D & Choi, K-Y. (2015). CXXC5 is a negative-feedback regulator of the Wnt/ β -catenin pathway involved in osteoblast differentiation. Cell death and differentiation. 22.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



RG3, Methylcobalamin, NAD+

RG3- Purity: >99.5% | Molecular Formula: C₄₂H₇₂O₁₃
 Molecular Weight: 85.025 g/mol | Sequence: Non-Peptide
 Methylcobalamin- Molecular Formula: C₆₃H₉₁CoN₁₃O₁₄P
 Molecular Weight: 1344.4 g/mol | Sequence: Non-Peptide
 NAD+ Molecular Formula: C₂₁H₂₇N₇O₁₄P₂
 Molecular Weight: 663.43 g/mol | Sequence: Non-Peptide

DESCRIPTION:

RG3 is a Panax ginseng that has been used in oriental countries for its pharmacologic effects, such as antidiabetic, neurological, and anti-inflammatory activities. Neuroinflammation is associated with activation of the central nervous system (CNS) glia with significant cytokine and chemokine production, infiltration of immune cells, edema, increased blood-brain barrier (BBB) permeability and breakdown. Ginsenoside 20(S) RG3 is one of the many active ingredients of ginseng saponins. RG3 is a ginseng known for aiding chronic inflammation. Specifically,

RG3 has been shown to reduce chronic neurodegenerative inflammation, the proinflammatory cytokine, interleukin-6 (IL-6) and interleukin-1 β (IL-1 β), and tumor necrosis factor- α (TNF- α). Methylcobalamin and NAD+ are combined with RG3 to enhance its effects. Target treatments for RG3/Methylcobalamin/NAD+ include aging, traumatic brain injury (TBI), Alzheimer's, diabetes, atherosclerosis, and hypertrophic scar formation. RG3 also has promising views for treatment in ovarian cancer, prostate cancer, and other cancers as well.

PROTOCOL:

Content & Potency: 2/2/50mg/mL provided in both a 15ml and 30 ml nasal spray applicator.
Suggested dosage: Instill one spray intranasally 2-4 times daily.

CLINICAL RESEARCH:

Suppressive Effect of Ginsenoside Rg3 against Lipopolysaccharide- Induced Depression-Like Behavior and Neuroinflammation in Mice

Ginsenoside Rg3 (Rg3), a major active ingredient enriched in red ginseng, possess well-confirmed immunoregulatory effects. Immune disturbance is a common trigger and aggravating factor in depression. The aim of this study was to explore the effects on Rg3 on lipopolysaccharides (LPS)-induced depression-like behavior in mice and the involvement of immune regulation. Pretreatment with Rg3 (i.g., 20 and 40 mg/kg) effectively ameliorated LPS (i.p., 0.83 mg/kg) induced body weight loss, anorexia, and immobility time in both the tail suspension test and the forced swimming test. Rg3 attenuated the disturbed turnover of tryptophan and serotonin in the hippocampus, accompanied by decreased

mRNA expression of cytokines and indoleamine-2, 3-dioxygenase (IDO). These central benefits were partially linked to the regulation of microglia activation and nuclear factor kappa B (NF- κ B) pathway. In addition, Rg3 significantly reduces LPS-induced elevation of interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α) in plasma, and restored the systemic balance of tryptophan-ky-nurenine metabolism. Taken together, our results demonstrated the Rg3 was effective in ameliorating depressive-like behavior induced by immune activation, adding new evidence to support its health benefits by immunoregulation.

Kang, An & Xie, Tong & Zhu, Dong & Shan, Jinjun & di, liuqing & Zheng, Xiao. (2017). Suppressive Effect of Ginsenoside Rg3 against Lipopolysaccharide-Induced Depression-Like Behavior and Neuroinflammation in Mice. Journal of Agricultural and Food Chemistry. 65.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Sarms LGD-4033

Purity: >98% | Molecular Formula: C₁₄H₁₂F₆N₂O
 Molecular Weight: 338.25/mol | Sequence: Non-Peptide

DESCRIPTION:

Selective Androgen Receptor Modulators (SARMs) provide the benefits of traditional anabolic/androgenic agents such as testosterone (including increased muscle mass, fat loss, and bone density), while having lower unwanted side effects characteristic of oral anabolics (aromatization / increased DHT). By stimulating the androgen receptor, SARMs can provide a similar therapeutic outcome to androgen therapy without any increase in androgen levels. SARMs have the potential to take the place of androgens, and therefore exert

many of the same positive effects on muscle tissue. SARMs can be administered in an injectable dosage form and are absorbed orally with no liver toxicity as with most oral steroids. The anabolic effect has been measured to be roughly the same or greater than testosterone. It has also been shown to produce dose-dependent increases in bone mineral density and mechanical strength, decrease body fat and increase lean body mass. LGD-4033 is a relatively new SARM on the market. It can be dosed orally at low doses and has a very strong anabolic effect.

PROTOCOL:

Content & Potency: 0.5mg capsule provided in a quantity of 32.
Suggested dosage: Take one capsule once daily for 32 days should be cycled (one month on, one month off).

CLINICAL RESEARCH:

The Safety, Pharmacokinetics, and Effects of LGD-4033, a Novel Nonsteroidal Oral, Selective Androgen Receptor Modulator, in Healthy Young Men

LGD-4033 was well tolerated. There were no drug-related serious adverse events. Frequency of adverse events was similar between active and placebo groups. Hemoglobin, prostate-specific antigen, aspartate aminotransferase, alanine aminotransferase, or QT intervals did not change significantly at any dose. LGD-4033 had a long elimination half-life and dose-proportional accumulation upon multiple dosing. LGD-4033 administration was associated with dose-dependent suppression of total testosterone, sex hormone – binding globulin, high density lipoprotein cholesterol,

and triglyceride levels. Follicle-stimulating hormone and free testosterone showed significant suppression at 1.0-mg dose only. Lean bodymass increased dose dependently, but fat mass did not change significantly. Hormone levels and lipids returned to baseline after treatment discontinuation. LGD-4033 was safe, had favorable pharmacokinetic profile, and increased lean body mass even during this short period without change in prostate-specific antigen. Longer randomized trials should evaluate its efficacy in improving physical function and health outcomes in select populations.

Basaria, Shehzad & Collins, Lauren & Dillon, Edgar & Orwoll, Katie & Storer, Thomas & Micek, Renee & Ulloor, Jagadish & Zhang, Anqi & Eder, Richard & Zientek, Heather & Gordon, Gilad & Kazmi, Syed & Sheffield-Moore, Melinda & Bhasin, Shalender. (2012). The Safety, Pharmacokinetics, and Effects of LGD-4033, a Novel Nonsteroidal Oral, Selective Androgen Receptor Modulator, in Healthy Young Men. The journals of gerontology. Series A, Biological sciences and medical sciences. 68.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Selank

Purity: >98% (HPLC on request) | Molecular Formula : C33H57N11O9
Molecular Weight: 751.89 g-mol-1 | Sequence: Thr-Lys-Pro-Arg-Pro-Gly-Pro

DESCRIPTION:

Selank is another ACTH/MSH-like peptide of the melanocortin class most closely related to the analog tufstin. While traditionally prescribed for anxiety and depression, it has been known to be effective in many other treatments related to immune modulation, anticoagulation, PTSD, ADHD, and metabolic syndromes. Selank has pronounced anxiolytic activity and acts as a stable neuropsychotropic, antidepressant, and anti-stress drug that relieves aggression and fear reaction in different animal species. Selank also has a nootropic action, which positively influences the formation of memory and learning processes, and marked immunomodulatory activity. Clinical studies have shown that the effect of selank is similar to that of tranquilizers at low doses, but is not accompanied by the unwanted side effects of benzodiazepine tranquilizers such as amnesia, withdrawal, or

dependence. Experiments have also demonstrated the effectiveness of Selank in preventing the accumulation of body fat (i.e., weight gain) with simultaneous activation of the functional state of the anticoagulation system in development of the metabolic syndrome.

Furthermore, decreased blood glucose levels have been observed with chronic treatment of this peptide. The peptide Selank, like the drug Semax, induces anticoagulant and hyperglycemia effects possibly due to the presence of the same amino acid sequence, Pro-Gly-Pro, in its structure. Thus, Selank can be used as a broad-spectrum therapeutic agent for the treatment of metabolic syndrome.

Often prescribed for: Anxiolytic, Immune improvement, gastric protection, as a preventative weight gain/metabolic syndrome, and with opioid and alcohol withdrawal/ dependence.

PROTOCOL:

Content & Potency: 7500mcg/ml provided in a 3ml nasal spray applicator.
Suggested dosage: Spray 1 to 2 sprays intranasally once daily.

CLINICAL RESEARCH:

P-1114 - Rapid and Slow Response During Treatment of Generalized Anxiety Disorder with Peptide Anxiolytic Selank

Results: 40% of patients were rapid responders (RR) and characterized by abrupt reduction of whole set of symptoms in first 1-3 Days. At the Day 3 Hamilton Anxiety Rating Scale (HARS) mean total score [SD] reduced from 20.3[11.9] to 7.0[2.9] ($p < 0.01$). 60% of patients responded gradually (conventional responders - CR). Clinically significant changes of mean HARS total

score from 16.1[7.2] to 6.2[4.7] were achieved at Day 14, $p < 0.01$. In contrast to CR, RR demonstrated obvious EEG-reaction after single dose (900 μg) with increase of beta-rhythm, decrease of theta- and low frequencies of alpha-rhythm (all $p < 0.05$). Initially RR and CR significantly differed by the score of asthenic and cognitive symptoms ($p < 0.05$).

Syunyakov, Timur & S Teleshova, E & G Neznamov, G & Bochkarev, Vitaly. (2012). P-1114-Rapid and slow response during treatment of generalized anxiety disorder with peptide anxiolytic selank. *European Psychiatry*. 27.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Semax

Purity: >98% (HPLC on request) | Molecular Formula: C37H51N9O10S
Molecular Weight: 813.92g/mol | Sequence: Met-Glu-His-Phe-Pro-Gly-Pro

DESCRIPTION:

It is well known that ACTH/MSH-like peptides (melanocortins) exert pleiotropic non-hormonal actions among their larger activities.

Melanocortins affect learning processes and exploratory behavior, regeneration and development, nociceptive and inflammatory processes, accelerate nerve regeneration and improve neuromuscular performance. Together these classes of peptides control many behaviors such as regulating attention, processes of learning, and memory formation as a result of their pronounced effect on CNS functions. Heptapeptide SEMAX (MEHFPGP) is the analogue of ACTH (4-10) that has prolonged neurotropic activity and thus is a good candidate for medical therapy. Currently this peptide is successfully used in treatment of patients with pathologies related to brain circulation dysfunction and with different intellectual-amnesic problems of the CNS. Doctors have

prescribed it for many conditions like anxiety, memory improvement, ischemic events, stroke, nerve regeneration, ADHD, opioid withdrawal, and even chronic diseases such as ALS, Parkinson's, and Alzheimer's. Some doctors use it as a preventative measure to protect against chronic disease and to acutely help improve memory and learning processes. It also has a marked antithrombotic and fibrinolytic effect and a gastric protective effect. It has also been suggested in literature that due to its effect on carboxypeptidase it can also increase physical performance and adaptation capacities in exposure to high intensity exercise. At its higher doses, .5mg/kg can even be analgesic.

Often prescribed for: Anti-Thrombosis, ADHD/ Learning, Gastric protection, Physical exertion improvement pain, Metal toxicities.

PROTOCOL:

Content & Potency: 7500mcg/ml provided in a 3ml nasal spray applicator.
Suggested dosage: Spray two sprays intranasally once daily.

CLINICAL RESEARCH:

The Nootropic and Analgesic Effects of Semax Given via Different Routes

The heptapeptide Semax (MEHFPGP) is an analog of the fragment ACTH(4-10) with long-lasting actions. The aim of the present work was to study the effects of Semax on learning ability and pain sensitivity in white rats given different doses via the intraperitoneal and intranasal routes. The nootropic effects of Semax were studied in a test based on the acquisition of a conditioned passive avoidance reaction to pain stimulation. Pain sensitivity was assessed

in a hindpaw compression test. The results showed that i.p. Semax had nootropic and analgesic actions. Dose-response characteristics were different for these different effects. Intranasal Semax was more effective in improving learning in animals than i.p. Semax but had no effect on pain sensitivity. Our results provide evidence that different mechanisms and brain structures are involved in mediating the nootropic and analgesic effects of Semax.

D.M, Manchenko & N.Yu, Glazova & Levitskaya, Natalia & L.A, Andreeva & , Kamenskii.A.A & N.F, Myasoedov. (2012). The Nootropic and Analgesic Effects of Semax Given via Different Routes. *Neuroscience and Behavioral Physiology*. 42. 264-270.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Tesamorelin

Purity: >98% (HPLC on request) | Molecular Formula : C221H366N72O67S
Molecular Weight: 5135.77 | Sequence: trans-hexenoyl-acid-Tyr-Ala-Asp-Ala-Ile-Phe-Thr-Asn-Ser-Tyr-Arg-Lys-Val-Leu-Gly-Gln-Leu-Ser-Ala-Arg-Lys-Leu-LeuGln-Asp-Ile-Met-Ser-Arg-Gln-Gln-Gly-Glu-Ser-Asn-Gln-Glu-Arg-Gly-Ala-Arg-Ala-Arg-Leu-NH2

DESCRIPTION:

Tesamorelin is a growth hormone releasing hormone analog that increases IGF-1 levels in men and women, by an average of 181 micrograms/liter. It binds to and stimulates GHRH receptors with similar potency as endogenous GHRH. It has a host of other benefits including nootropic effects and reducing triglycerides. Tesamorelin has subsequently been shown to decrease carotid intima-media

thickness (cIMT), visceral adipose tissue (VAT), and c-reactive protein (CRP). It has not been linked to significantly affect other pituitary hormones and their respective mechanisms in the body. Additionally, it can improve cognitive function for healthy seniors and patients with an increased risk of Alzheimer's disease, due to mild cognitive impairment.

PROTOCOL:

Content & Potency: 1mg lyophilized subcutaneous injectables presented in a quantity of 24 vials with 10ml of sterile water for injection for reconstitution.

Suggested dosage: Reconstitute each vial prior to injection with 0.6ml sterile water for injection, inject 0.5ml subcutaneously before bed 6 out of 7 days 90 minutes after last food intake.

CLINICAL RESEARCH:

Long-term safety and effects of tesamorelin, a growth hormone-releasing factor analogue, in HIV patients with abdominal fat accumulation

Treatment of HIV patients with daily tesamorelin, a growth hormone-releasing factor analogue, for 26 weeks resulted in a significant decrease in visceral adipose tissue (VAT) and improvement in lipids. The objective of the 26-week extension phase was to evaluate long-term safety and effects of tesamorelin. HIV patients with central fat accumulation in the context of antiretroviral therapy were randomized to tesamorelin 2 mg (n = 273) or placebo (n = 137) s.c. daily for 26 weeks. At week 26, patients originally on tesamorelin were rerandomized to 2 mg tesamorelin (T-T group, n = 154) or placebo (T-P group, n = 50), whereas patients originally on placebo were switched to tesamorelin (P-T group, n = 111). Safety included adverse events and glucose parameters. Tesamorelin was generally well tolerated. The prevalence of adverse events and serious

adverse events during the extension phase was comparable with the initial phase. Changes in glucose parameters over 52 weeks were not clinically significant and similar to those after 26 weeks. The change in VAT was sustained at -18% over 52 weeks of treatment (P < 0.001 versus baseline) as was the change in triglycerides (-51 mg/dl, P < 0.001 versus baseline). Similar sustained beneficial effects were seen for total cholesterol, but high-density lipoprotein decreased minimally over 52 weeks. Upon discontinuation of tesamorelin, VAT reaccumulated. Treatment with tesamorelin was generally well tolerated and resulted in sustained decreases in VAT and triglycerides over 52 weeks without aggravating glucose. Though effects on VAT are sustained during treatment for 52 weeks, these effects do not last beyond the duration of treatment.

Falutz, Julian & Allas, Soraya & Mamputu, Jean-Claude & Potvin, Diane & Kotler, Donald & Somero, Michael & Berger, Daniel & Brown, Stephen & Richmond, Gary & Fessel, Jeffrey & Turner, Ralph & Grinspoon, Steven. (2008). Long-term safety and effects of tesamorelin, a growth hormone-releasing factor analogue, in HIV patients with abdominal fat accumulation. AIDS (London, England). 22. 1719-28.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Tesofensine

Purity: >98% (HPLC on request) | Molecular Formula: C17H23Cl2NO
Molecular Weight: 3.277 g/mol | Sequence: Non-Peptide

DESCRIPTION:

Tesofensine is serotonin-noradrenaline-dopamine reuptake inhibitor which was originally studied for its effect on Parkinson's and Alzheimer's. Unfortunately, its exploration for these indications were limited because the patients starting losing too much weight. Since then, the tesofensine has been studied as a way to treat obesity via its ability to reduce appetite. This medication indirectly stimulates the cholinergic system and showed to be more successful than average weight loss medication.

Tesofensine (TE) increases near transmission of 3 monoaminergic neurotransmitters in the brain. These neurotransmitters are serotonin, norepinephrine, and dopamine which help regulate energy balance and are linked to obesity and depression. On average, its 6 month weight loss results from a phase 2b clinical trial resulted in a weight loss of around 25 pounds.

PROTOCOL:

Content & Potency: 500mcg capsule provided in a quantity of 30 capsules.

Suggested dosage: Take 1 capsule by mouth once daily in the morning.

CLINICAL RESEARCH:

Effects of Tesofensine on bodyweight loss, body composition, and quality of life in obese patients: a randomised, double-blind, placebo-controlled trial

Weight-loss drugs produce an additional mean weight loss of only 3-5 kg above that of diet and placebo over 6 months, and more effective pharmacotherapy of obesity is needed. We assessed the efficacy and safety of tesofensine-an inhibitor of the presynaptic uptake of noradrenaline, dopamine, and serotonin in patients with obesity. We undertook a phase II, randomised, double-blind, placebo-controlled trial in five Danish obesity management centres. After a 2 week run-in phase, 203 obese patients (body-mass index 30-40 kg/m²) were prescribed an energy restricted diet and randomly assigned with a list of randomisation numbers to treatment with tesofensine 0.25 mg (n=52), 0.5 mg (n=50), or 1.0 mg (n=49), or placebo (n=52) once daily for 24 weeks. The primary outcome was percentage change in bodyweight. Analysis was by modified intention to treat (all randomised patients with measurement after at least one dose of study drug or placebo). The study is registered with

ClinicalTrials.gov, number NCT00394667. 161 (79%) participants completed the study. After 24 weeks, the mean weight loss produced by diet and placebo was 2.0% (SE 0.60). Tesofensine 0.25 mg, 0.5 mg, and 1.0 mg and diet induced a mean weight loss of 4.5% (0.87), 9.2% (0.91), and 10.6% (0.84), respectively, greater than diet and placebo (p<0.0001). The most common adverse events caused by tesofensine were dry mouth, nausea, constipation, hard stools, diarrhoea, and insomnia. After 24 weeks, tesofensine 0.25 mg and 0.5 mg showed no significant increases in systolic or diastolic blood pressure compared with placebo, whereas heart rate was increased by 7.4 beats per min in the tesofensine 0.5 mg group (p=0.0001). Our results suggest that tesofensine 0.5 mg might have the potential to produce a weight loss twice that of currently approved drugs. However, these findings of efficacy and safety need confirmation in phase III trials.

Astrup, Arne & Madsbad, Sten & Breum, Leif & Jensen, Thomas & Peter Kroustrup, Jens & Meinert Larsen, Thomas. (2008). Effect of tesofensine on bodyweight loss, body composition, and quality of life in obese patients: a randomised, double-blind, placebo-controlled trial. Lancet. 372.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Tetradecylthioacetic Acid (TTA)

Purity: >98% (HPLC on request) | Molecular Formula : C₁₆H₃₂O₂S
Molecular Weight: 288.49 g/mol | Sequence: Non-Peptide

DESCRIPTION:

Tetradecylthioacetic Acid, otherwise known as TTA, is a PPAR-alpha activator. Although similar in structure to an omega-3 fatty acid, it cannot be utilized for energy and thus has no relevant caloric value to humans. PPAR-alpha is a transcription factor and a major regulator of lipid metabolism in the liver. PPAR-alpha is activated under conditions of energy deprivation and is necessary for the process of ketogenesis, a key adaptive response to prolonged fasting.

Activation of PPAR-alpha promotes uptake, utilization, and catabolism of fatty acids by upregulation of genes involved in fatty acid transport, fatty acid binding and activation, and peroxisomal and mitochondrial fatty acid β -oxidation. The clearing of fat from the blood causes a drop in lipoproteins and a lowering of LDL cholesterol. TTA has also been shown to decrease blood pressure and exert a mild anti-oxidant effect.

PROTOCOL:

Content & Potency: 200mg capsules provided in quantities of 90 capsules.
Suggested dosage: Take 1 capsule by mouth 3 times daily.

CLINICAL RESEARCH:

Dietary supplementation of tetradecylthioacetic acid increases feed intake but reduces body weight gain and adipose depot sizes in rats fed on high-fat diets

Despite higher feed intake during the final 2 weeks of the study, rats fed on TTA gained less body weight than lard-fed rats and had markedly decreased subcutaneous, epididymal, perirenal and mesenteric adipose depots. The effects of TTA feeding with reduced body weight gain and energy efficiency (weight gain/feed intake) started between day 10 and 13. Body contents of fat, protein and water were reduced after feeding lard plus TTA, with a stronger decrease in fat relative to protein. Plasma lipids, including Non-Esterified Fatty Acids (NEFA), were significantly reduced, whereas fatty acid β -oxidation in liver and heart

was enhanced in lard plus TTA-fed rats. Hepatic UCP3 was expressed ectopically both at protein and mRNA level (>1900-fold), whereas Ucp1 mRNA was increased 30-fold in epididymal and 90-fold in mesenteric fat after lard plus TTA feeding.
Conclusion: Our data support the hypothesis that TTA feeding may increase hepatic fatty acid β -oxidation, and thereby reduce the size of adipose tissues. The functional importance of ectopic hepatic UCP3 is unknown but might be associated with enhanced energy expenditure and thus the reduced feed efficiency.

Wensaas AJ, Rustan AC, Rokling-Andersen MH, Caesar R, Jensen J, Kaalhus O, Graff BA, Gudbrandsen OA, Berge RK, Drevon CA.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Thymosin Alpha-1

Purity: >98% (HPLC on request) | Molecular Formula: C₁₂₉H₂₁₅N₃₃O₅₅
Molecular Weight: 3108.28 | Sequence: Ac-Ser-Asp-Ala-Ala-Val-Asp-Thr-Ser-Ser-Glu-Ile-Thr-Thr-Lys-Asp-Leu-Lys-Glu-Lys-Lys-Glu-Val-Val-Glu-Glu-Ala-Glu-Asn-OH

DESCRIPTION:

Thymosin α -1 is a major component of Thymosin Fraction 5 and is responsible for restoring and modulating immune function, particularly cell mediated immune function. Recent studies showed that Thymosin Alpha-1 molecule increased major histocompatibility complex (MHC) class-1 and Toll-like receptor expression as well as cytokine production, suggesting its immunoregulatory role.

It is an FDA approved medication under the trade name zadaxin after it received orphan drug

approval status. It is widely used and studied in multiple types of cancer and viral illnesses. Some physicians are using thymosin for chronic fatigue and Lyme disease as well as autoimmune function as well.

TA 1 is thought to modulate the immune system by augmenting T-cell function. TA1 may affect thymocytes by stimulating their differentiation or by converting them to active T cells. TA1 is rapidly absorbed, achieving peak serum concentrations within two hours.

PROTOCOL:

Content & Potency: 1 x 5mL at 3000 mcg/mL ready-to-inject subcutaneous.
Suggested dosage: Inject .15mL subcutaneously once daily until vial is empty (1 month supply) or inject 1.6mg 2 x 1 week.

CLINICAL RESEARCH:

Thymosin Alpha 1: Biological activities, applications and engineering production

Thymosin alpha 1 (T α 1), a 28-amino acid peptide, was first described and characterized from calf thymuses in 1977. This peptide can enhance T-cell, dendritic cell (DC) and antibody responses, modulate cytokines and chemokines production and block steroid-induced apoptosis of thymocytes. Due to its pleiotropic biological activities, T α 1 has gained increasing interest in recent years and has been used for the treatment of various diseases in clinic. Accordingly, there is an increasing need for the

production of this peptide. So far, T α 1 used in clinic is synthesized using solid phase peptide synthesis. Here, we summarize the genetic engineering methods to produce T α 1 using prokaryotic or eukaryotic expression systems. The effectiveness of these biological products in increasing the secretion of cytokines and in promoting lymphocyte proliferation were investigated in vitro studies. This opens the possibility for biotechnological production of T α 1 for the research and clinical applications.

Li, Juan Jenny et al. "Thymosin alpha 1: Biological activities, applications and genetic engineering production." Peptides 31 (2010): 2151-2158.

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Thymosin Beta

VIP

Purity: >98% (HPLC on request) | Molecular Formula : C212H350N56O78S
Molecular Weight: 4963.506 g/mol | Sequence: Ac-Ser-Asp-Lys-Pro-Asp-Met-Ala-Glu-Ile-Glu-Lys-Phe-Asp-Lys-Ser-Lys-Leu-Lys-Lys-Thr-Glu-Thr-Gin-Glu-Lys-Asn-Pro-Leu-Pro-Ser-Lys-Glu-Thy-IleGlu-Gin-Glu-Lys-Gin-Ala-Gly-Glu-Ser

DESCRIPTION:

Thymosin is a hormone secreted from the thymus. Its primary function is to stimulate the production of T cells, which are an important part of the immune system. Thymosin also assists in the development of B cells to plasma cells to produce antibodies. The predominant form of Thymosin, Thymosin Beta 4, is a member of a highly conserved family of actin monomer-sequestering proteins. In addition to its role as a major actin-sequestering molecule, Thymosin Beta 4 plays a role in tissue repair. Tβ4 has been

found to play an important role in protection, regeneration and remodeling of injured or damaged tissues. The gene for Tβ4 has also been found to be one of the first to be upregulated after injuries. Thymosin Beta 4 is currently being trialed as a potential therapy for HIV, AIDS, and Influenza. Thymosin Beta 4 is most often prescribed for acute injury, surgical repair and for senior athletes. It has most recently been shown to help regrow hair in addition to PRP.

PROTOCOL:

Content & Potency: 3000mcg/ml subcutaneous injection provided in a 5ml vial.
Suggested dosage: Inject 0.25ml subcutaneously daily for 20 days.

CLINICAL RESEARCH:

Differential expression of the human thymosin-beta 4 gene in lymphocytes, macrophages, and granulocytes.

A cDNA clone encoding human thymosin-beta 4 was isolated from a cDNA library prepared from peripheral blood leukocytes of a patient with acute lymphocytic leukemia. This clone contained the entire coding sequence of 43 amino acid residues of thymosin-beta 4 and had an initiation codon and two termination codons. The amino acid and nucleotide sequences in the coding region were well conserved between rat and human. Nine of 132 nucleotides were different in the coding sequences (93% homology), but the deduced amino acid sequences were identical. No signal peptide was found in the deduced protein sequence. Human thymosin-beta 4 mRNA, approximately 830 nucleotides in length, was about 30 nucleotides larger than rat thymosin-beta 4 mRNA. Expression of the human thymosin-beta 4 gene in various primary myeloid and lymphoid malignant cells and in a few human hemopoietic cell lines was studied. Northern

blot analyses of different neoplastic B lymphocytes revealed that steady state levels of thymosin-beta 4 mRNA varied as a function of differentiation stage. Thymosin-beta 4 mRNA levels were decreased in myeloma cells as are class II human leukocyte antigen, Fc receptor, and complement receptor, suggesting a relationship between thymosin-beta 4 and the immune response. Thymosin-beta 4 mRNA was more highly expressed in mature granulocytes than in immature blastic cells. Treatment of THP-1 cells, a human monocytic cell line, with recombinant human interferon-lambda reduced the levels of thymosin-beta 4 mRNA. Its level decreased after differentiation of THP-1 cells into Ia+ macrophages, but increased after differentiation of HL-60 cells into Ia- macrophages. The pattern of thymosin-beta 4 gene expression suggests that it may play a fundamental role in the host defense mechanism.

H Gondo, J Kudo, J W White, C Barr, P Selvanayagam, G F Saunders The Journal of Immunology December 1, 1987, 139 (11) 3840-3848;

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.

Purity: >98% (HPLC on request) | Molecular Formula: C147H237N43O43S
Molecular Weight: 3326.831 g/mol | Sequence: HSDAVFTDNYTRLRQMAVKKYLN SILN-NH2

DESCRIPTION:

Vasoactive intestinal polypeptide (VIP) is a naturally produced neuropeptide that functions as a neuromodulator and neurotransmitter. It is a potent vasodilator, regulates smooth muscle activity, epithelial cell secretion, and blood flow in the gastrointestinal tract. As a chemical messenger, it functions as a neurohormone and

paracrine mediator. Therapeutically, it is often dosed nasally in patients with mold toxicity and other biotoxin illnesses. In these patients, exogenous administration can help support healthy hormone levels, works to limit inflammation, regulates the immune system, and help in the healing activity of the brain.

PROTOCOL:

Content & Potency: 500mcg/ml provided in a 12 ml nasal spray applicator.
Suggested dosage: Instill 50mcg intranasally in alternating nostrils up to 4 times daily.

CLINICAL RESEARCH:

Vasoactive intestinal polypeptide (VIP) corrects chronic inflammatory response syndrome (CIRS) acquired following exposure to water-damaged buildings

Exposure in water-damaged buildings (WDB) to airborne bioaerosols including metabolic products of toxigenic fungi, bacteria and actinomycetes; and inflammagens, can lead to a persistent innate immune inflammatory illness. This illness, termed a chronic inflammatory response syndrome (CIRS-WDB), is systemic with symptoms acquired from multiple organ systems. Treatment of CIRS-WDB has progressed rapidly as a better understanding of the inflammatory pathophysiology has led to targeted, sequential therapies. The fundamental basis of uncontrolled innate immune responses, the humoral deficiency of regulatory neuropeptides melanocyte stimulating hormone (MSH) or vasoactive intestinal polypeptide (VIP), seen in over 98% of patients, has not consistently responded to any treatment modality. Use of replacement VIP has been attempted anecdotally; VIP replacement therapies show promise in short term studies but longer therapies have not been attempted. Here we report an open label trial of 20 patients with refractory

CIRS-WDB illness who took replacement VIP in a nasal spray for at least 18 months with confirmation of durable efficacy and absence of significant side effects. These 20 patients were similar in symptoms and lab findings to three previously published cohorts involving 1829 patients and 169 controls. Dosage of VIP was titrated downwards from four to zero doses a day to determine minimum effective dose, and re-titrated upwards for maximum improvement over time. The trial showed that VIP therapy safely 1) reduced refractory symptoms to equal controls; 2) corrected inflammatory parameters C4a, TGF beta-1, VEGF, MMP9; 3) corrected estradiol, testosterone and 25-OH Vitamin D; 4) returned pulmonary artery systolic pressure (PASP) during exercise to normal; and 5) enhanced quality of life in 100% of trial patients. Subsequent identification of correction of T-regulatory cell levels supports the potential role of VIP in both innate and adaptive immune function.

Ritchie C. Shoemaker, Dennis House, James C. Ryan

A full copy of all trials are available from Tailor Made Compounding Pharmacy. Please contact us for more info.



Zinc Thymulin

Purity: >98% (HPLC on request) | Molecular Formula: C₃₃H₅₄N₁₂O₁₅
Molecular Weight: 858.85 g/mol | Sequence: Non-Peptide

DESCRIPTION:

Thymulin is a nonapeptide produced by two distinct epithelial populations in the thymus first described by Bach in 1977. It requires zinc for biological activity. The hormone is involved in T-cell differentiation and enhancement of T and NK cell actions. Thymulin has neuroendocrine effects as well. It follows a circadian rhythm and physiologically elevated ACTH levels correlate positively with thymulin plasma levels and vice versa. A recent study was done on Zinc Thymulin

to test its efficacy in the treatment of hair loss. The study indicated that topical treatment with zinc thymulin significantly increased hair growth over 6 months; further, there were no systemic or local side effects from the treatment. The zinc thymulin metallo-peptide optionally also improves endogenous hair pigmentation. For example, by stimulating melanogenesis in grey or greying hair.

PROTOCOL:

Content & Potency: Topical foam provided in a quantity of 50ml foaming applicator.
Suggested dosage: Apply 1ml (2 pumps) to scalp once daily at night.

CLINICAL RESEARCH:

An Analysis of the Safety and Efficacy of Topical Zinc-Thymulin to treat Androgenetic Alopecia

To assess the safety and efficacy of the metallopeptide zinc-thymulin (ZT) for treating androgenetic alopecia (AGA). Previous in vitro studies have described that different thymic peptides can both increase and decrease anagen (thymulin and thymosin beta-4, respectively). Zinc is an essential element and serum zinc deficiency can cause hair loss.

Eighteen consecutive adult subjects were recruited, 17 males and 1 female, age range 35-90 years (mean 55.4, SD 13.3) with a diagnosis of AGA, Norwood classification 2-7, and hair loss duration range of 3-40 years (mean 15.8, SD 9.6). The trial duration for each subject ranged from 4-10 months. The test compound ZT was synthesized by standard Fmoc peptide protocols and administered in water based topical spray to the scalp. Baseline and after treatment images for hair growth were graded by two blinded assessors using two validated scales: 1. numerical visual analog scale (VAS) for global assessment 2. hair growth

index (HGI) of images under higher magnification for percentage changes of vellus, intermediate and terminal hair.

ZT demonstrated no adverse systemic effects or local side effects of redness or scalp irritation in any subject over a total of 3,300 treatment days. Three subjects who were concurrently using minoxidil (N=2) and minoxidil / finasteride (N=1) did not report any drug interaction with ZT. VAS hair assessment improvement was significant in subjects who completed 6 months of treatment (P=0.045, t-test). HGI assessment showed a significant increase in the number of newly observed intermediate hairs in previous "absent hair" regions (P<0.0001) with an average increase of vellus type (32%) and intermediate type (23%) hairs at 6 months. Melanogenesis was observed in several subjects. Topical applications of ZT demonstrated safety and established efficacy for initiating and maintaining anagen to treat male pattern baldness when applied for >6 months.

Vickers ER

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DOSING

CHARTS

Please contact Tailor Made Compounding with dosing inquiries for any products you may be interested in.

support@tailormadecompounding.com

PRODUCT	QUANTITY	DOSAGE	TIMING OF DOSING	APPLICATION	INDICATION
2-Methoxyestradiol					
Capsule	3mL	100mcg daily	Before bed	Suppository	Cancer
3-Desoxy DHEA					
Capsules	100mg (30 capsules)	1 capsule daily	Patient preference	Oral	Aromatase inhibitor
5-amino-1MQ					
Injection	10ml at 300mg/ml	150mcg (.5ml) daily for 20 days	30 minutes after oral administration of NAD+ precursor	Subcutaneous injection	Weight Loss & Performance
Amlexanox					
Capsules	40mg (90 capsules)	1 capsule TID	Patient preference	Oral	Insulin sensitivity, weight loss
Ammonium Tetrathiomolybdate					
Capsules	40mg (90 capsules)	1 capsule TID	Patient preference	Oral	Cancer
Aniracetam					
Capsules	375mg (60 capsules)	2 capsules daily	Patient preference	Oral	AMPA receptor modulator (Neurogenic)
AOD-9604					
Cream	30mL at 600mcg/mL	4 clicks (1mL) daily	Patient preference	Rubbed between inner forearms	Repair
Injection	5mL at 1200mcg/mL	0.25mL daily	Patient preference	Subcutaneous Injection	Repair
Injection with HA	AOD 1000mcg/mL + HA10mg/mL	0.5mL-0.75mL weekly for 4 weeks	Done by physician	Intra-articular	Repair hyaline cartilage
Argireline/Leuphasyl					
Cream	0.5%/0.5% cream in 30ml TopiClick	1ml applied daily	Patient preference	Topical	Reduce wrinkle depth and fine lines
Argireline/GHK-Cu/Leuphasyl					
Cream	0.5%/0.2% / 0.5% cream in 30ml TopiClick	1ml applied daily	Patient preference	Topical	Reduce wrinkle depth and fine lines
BPC-157					
Injection	3mL at 2000mcg/mL	0.15mL daily	Patient preference	Subcutaneous injection into site of injury	Repair tendon + ligaments
Injection	5mL at 2000mcg/mL	0.15mL daily	Patient preference	Subcutaneous injection into site of injury	Repair tendon + ligaments
Capsules	500mcg (30 capsules)	1 capsule daily	Patient preference	Oral	Repair bowel/gut
Cerebrolysin					
Injection	4x10 mL at 215mg/mL	1mL daily	Patient preference	Subcutaneous injection	Neurogenic
CJC 1295					
Injection	2mL at 2000mcg/mL	0.10mL 5 nights a week	Nightly, on an empty stomach before bed	Subcutaneous injection	GHRH

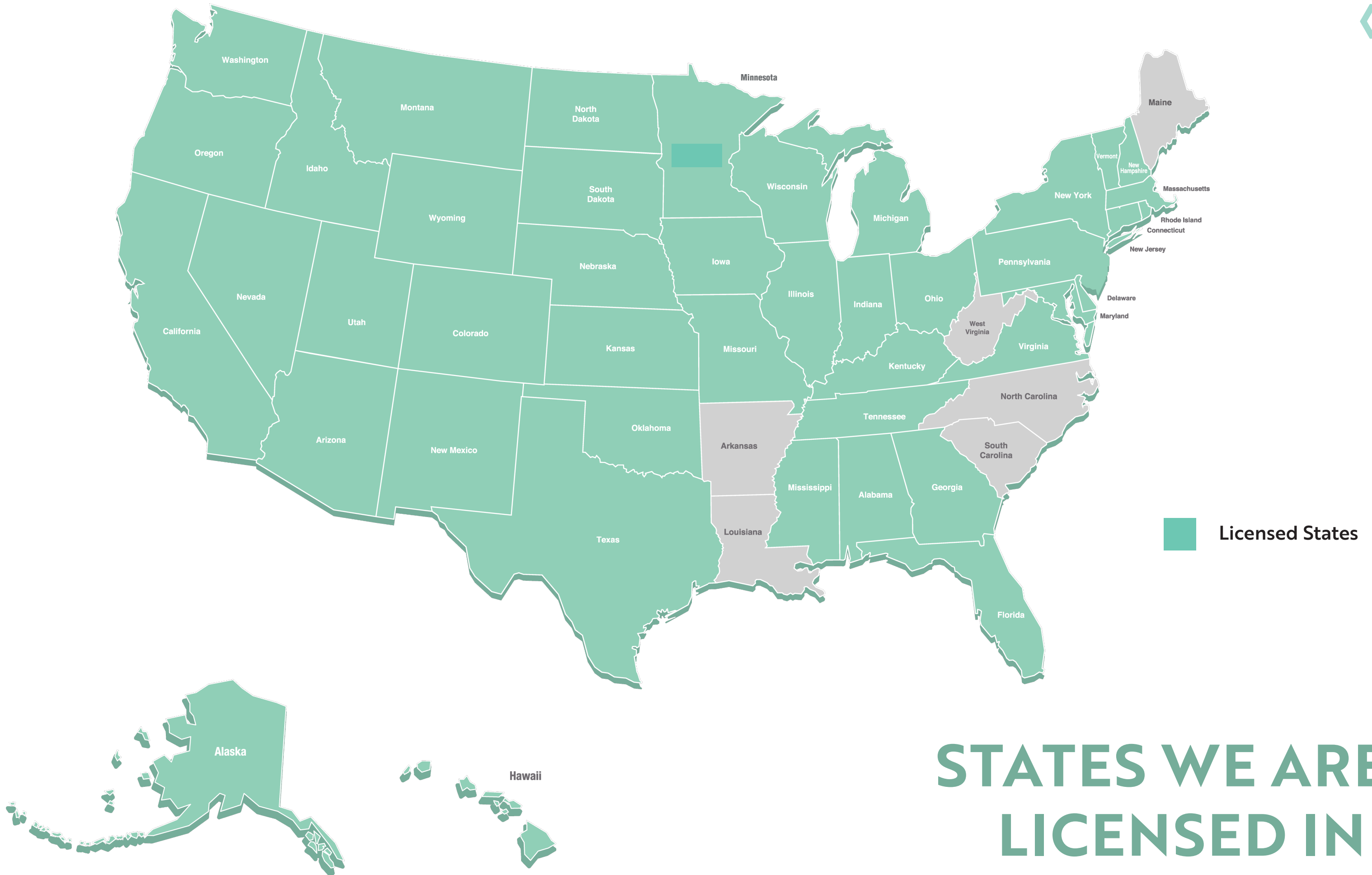
PRODUCT	QUANTITY	DOSAGE	TIMING OF DOSING	APPLICATION	INDICATION
CJC 1295					
Injection	5mL at 2000mcg/mL	0.10mL 5 nights a week	Nightly, on an empty stomach before bed	Subcutaneous injection	GHRH
CJC 1295/Ipamorelin					
Injection	2mL (or 5ml) at 1000mcg/1000mcg/mL	0.10mL 5 nights a week	Nightly, on an empty stomach before bed	Subcutaneous injection	GHRH, weight loss
Injection	2mL (or 5ml) at 2000mcg/1000mcg/mL	0.10mL 5 nights a week	Nightly, on an empty stomach before bed	Subcutaneous injection	GHRH, weight loss
Injection	2mL (or 5ml) at 2000mcg/2000mcg/mL	0.10mL 5 nights a week	Nightly, on an empty stomach before bed	Subcutaneous injection	GHRH, weight loss
DHH-B					
Capsules	30 7.5mg capsules	1-2 capsules as needed	Patient preference	Oral	Anti-Anxiety
Dihexa					
Capsules	5mg (30 capsules)	1-2 capsules daily	Patient preference	Oral	Neurological function/repair
Cream	30mL at 20mg/mL	4 clicks (1mL) daily	Patient preference	Transdermal	Neurological function/repair
DSIP					
Injection	3mL at 1000mcg/mL	0.1mL daily, 2-3 times a week	Patient preference	Subcutaneous injection	Endocrine regulation, sleep
Enclomiphene					
Capsules	25mg (30 capsules)	1 capsule daily	Patient preference	Oral	Aromatase inhibitor (Reproductive system)
Epicatechin					
Injection	100mg/ml	100mg (1ml) daily	Patient preference	Subcutaneous injection into the abdomen	Sarcopenia, Muscle gain
Epitalon					
Injection	5mL at 3000mcg/mL	0.1mL daily	Patient preference	Subcutaneous injection into site of injury	Repair tendon + ligaments
Cerebrolysin					
Injection	4x10 mL at 2.15.2mg/mL	1mL daily	In the morning	Subcutaneous injection into the abdomen	Hormone regulation, telomere extension
Fat Loss Cream (Aminophylline/Glycrrhetic Acid)					
Cream	60mL at 0.5% aminophylline glycrrhetic acid 2.5%	2 pumps (1mL) twice daily	Patient preference	Transdermal	Fat reduction
FGL(I)					
Injection	5mL at 10mg/mL	0.25mL daily	Patient preference	Subcutaneous injection into the abdomen	Neurogenic/ neurorestorative

PRODUCT	QUANTITY	DOSAGE	TIMING OF DOSING	APPLICATION	INDICATION
FOXO4-DRI					
Injection	10ml vial at 10mg/ml	300mcg/kg	3 doses every other day over 5 days	Intravenous	Clear senescent cells
GHK-Cu					
Injection	5mL at 10mg/mL	0.2mL daily	Patient preference	Subcutaneous injection	Skin elasticity
Foam	50mL at 5mg/mL	2 pumps (1mL) daily	Patient preference	Foam (for scalp application)	Hair loss
IGF-1					
Injection	Two 6.2mL vials at 620mcg/mL	0.4mL-0.8ml daily	Patient preference	Subcutaneous injection into the abdomen	Muscle building, weight loss
IGF-1 LR3					
Injection	6.2mL at 100mcg/mL	0.4-0.8mL daily	Post workout if applicable	Subcutaneous injection into the abdomen	Weight loss, muscle building, autocrine + paracrine hormone signaling
Ipamorelin					
Injection	5mL at 2000mcg/mL	0.1mL daily 5 days a week	Patient preference	Subcutaneous injection	GHRH, weight loss
iRGD					
Injection	10mL at 2.5mg/mL	40mcg/kg daily	Dose alongside concurrent therapy	Subcutaneous injection	Cancer
Kisspeptin-10					
Injection	5mL at 100mcg/mL	0.1mL daily	Patient preference	Subcutaneous injection into abdomen	LH increase
KPV					
Topical	30ml TopiClick at 15mg/ml	0.5ml applied twice daily	Patient preference	Transdermal Cream	Psoriasis
Liraglutide					
Injection	5mL at 3600mcg/mL	Varies	Varies	Subcutaneous injection	Insulin resistance, weight loss
LL-37					
Injection	5mL at 2000mcg/mL	Varies with indication and patient	Physician preference	Subcutaneous injection	Antimicrobial
Melanotan I					
Injection	5mL at 2000mcg/mL	0.15mL daily with UVB light exposure	Patient preference	Subcutaneous injection into the abdomen	Vitiligo
Melanotan I					
Injection	0.15mL daily for 1-2 weeks then 0.25mL weekly for maintenance	0.15mL daily for 1-2 weeks	Patient preference	Subcutaneous injection into the abdomen	Libido, tanning, weight loss

PRODUCT	QUANTITY	DOSAGE	TIMING OF DOSING	APPLICATION	INDICATION
Met-Enkephalin					
Injection	10mg Lyophilized vial	10mg IV once weekly	N/A	Intravenous	Cancer
MK-677					
Capsules	25 mg (30 capsules)	1 capsule daily	Patient preference on empty stomach	Oral	Growth hormone IGF-1 increase
MOTS-c					
Injection	4mL at 10mg/mL	1mL once weekly	Patient preference	Subcutaneous injection into the abdomen	Energy, weight loss
Myristyl					
Injection	5ml vial at 6mg/ml	100mcg/kg once weekly intravenously	N/A	Intravenous	Cholesterol Reduction
NMN					
Injection	10ml vial at 200mg/ml	200mg (1ml) BID subcutaneously	Patient preference	Oral	Anti-aging/ Weight loss
Capsule	30 capsules at 500mg	1 capsule daily	Patient preference	Oral	Anti-aging/ Weight loss
PEG-MGF					
Injection	5mL at 2000mcg/mL	0.1mL daily, 5 days a week	Patient preference	Subcutaneous injection into the abdomen	Building muscle
Pentosan Polysulfate (PPS)					
Injection	10mL at 250mg/mL	250mg	N/A	Intramuscular	Osteoarthritis
PNC-27					
Injection	5mL at 1000mcg/mL	2.5mg-5mg daily	Patient preference	Subcutaneous injection	Cancer
PT-141 (Bremelanotide)					
Injection	2mL at 10mg/mL	0.2mL (female) as needed	1-5 hours before sexual activity (first dose will establish timing)	Subcutaneous injection into abdomen	Erectile dysfunction, libido
Injection	2mL at 10mg/mL	0.1mL (male) as needed	1-5 hours before sexual activity (first dose will establish timing)	Subcutaneous injection into abdomen	Erectile dysfunction, libido
PTD-DBM					
Cream	5mL at 0.5% spray	Spray a sufficient amount to cover area	Applied after microneedling	Transdermal	Hair Loss
RG3					
Nasal	15mL	One spray into each nostril 2-4 times daily	Patient preference	Nasal	Neurogenic/ neurorestorative
Nasal	30mL	One spray into each nostril 2-4 times daily	Patient preference	Nasal	Neurogenic/ neurorestorative
RG3/Methylcobalamin/NAD+					
Nasal	15mL	One spray into each nostril 2-4 times daily	Patient preference	Nasal	Neurogenic/ neurorestorative



PRODUCT	QUANTITY	DOSAGE	TIMING OF DOSING	APPLICATION	INDICATION
RG3/Methylcobalamin/NAD+					
Nasal	30mL	One spray into each nostril 2-4 times daily	Patient preference	Nasal	Neurogenic/ neurorestorative
SARMS LGD-4033					
Capsules	0.5mg (30 capsules)	1 month on, 1 month off rotation	Patient preference	Oral	Muscle Building
Selank					
Injection	5mL at 1000mcg/1mL	0.1mL daily	Patient preference	Subcutaneous injection into the abdomen	Neurorestorative
Nasal	3mL at 7500mcg/mL	One spray into each nostril once daily	Patient preference	Nasal	Neurogenic
Semax					
Injection	5mL at 1000mcg/1mL	0.1mL daily	Patient preference	Subcutaneous injection into the abdomen	Neurorestorative
Nasal	3mL at 7500mcg/mL	One spray into each nostril once daily	Patient preference	Nasal	Neurogenic
Tesamorelin					
Injection	24 vials (lyophilized) at 1mg/vial	1 (1mg) vial 6 days a week	Before bed, 90 minutes after last food intake	Subcutaneous injection into the abdomen	Muscle building, weight loss
Tesofensine					
Capsules	500mcg (30 capsules)	1 capsule daily	In the morning	Patient preference	Weight loss
Thymosin Alpha-1					
Injection	5mL at 3000mcg/mL	0.15mL daily or 1.5mg twice a week	Patient preference	Subcutaneous injection into the abdomen	Immune modulation
Thymosin Beta-4					
Injection	5mL at 3000mcg/mL	0.25mL daily for 20 days	Patient preference	Subcutaneous injection into the abdomen	Repair soft tissue
TMB					
Capsules	40mg (30 capsules)	40mg twice daily	Patient preference	Oral	Insulin resistance, weight loss
TTA/Amllexanox					
Capsules	200mg/40mg (30 capsules)	1 capsule 3 times daily	Breakfast, lunch, dinner	Oral	Insulin resistance, weight loss
VIP					
Nasal	12mL at 50mcg/mL	One spray into each nostril up to 4 times daily	Patient preference	Nasal	Mold toxicity
Zinc Thymulin					
Foam	30mL at 50 mcg/mL	2 pumps (1mL) daily	Applied right before bed	Foam (for scalp application)	Hair loss



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