myos

Whitepaper

Fortetropin®: A Breakthrough in Muscle Health for Humans and Animals

Executive summary

There is a growing demand for safe, accessible solutions that can promote muscle health and prevent muscle loss across both human and veterinary applications. While muscle health has been overlooked in the past in the healthcare space, this is beginning to change as it is now recognized that maintaining muscle integrity has significant effects on mobility, recovery, quality of life, and longevity in both humans and animals.

Fortetropin®, a bioactive compound by MYOS, derived from fertilized chicken egg yolk via a patented process, represents a novel, all-natural approach to supporting muscle health. This white paper presents the key findings from over ten preclinical and clinical studies evaluating Fortetropin® and its source materials' mechanisms of action, efficacy, and safety. Across these studies, Fortetropin®'s key benefits include:

- Improved strength, mobility, and recovery.
- Increased lean muscle mass and protein synthesis.
- Activated anabolic signaling pathways, such as mTOR.
- Reduced muscle protein breakdown and modulated myostatin levels.
- Safe and well-tolerated in both animals and humans.

The findings from these studies highlight Fortetropin®'s potential as a safe, exercise-independent, muscle-preserving nutraceutical. Fortetropin®'s demonstrated ability to modulate key anabolic and catabolic pathways supports further investigation in longer-duration preclinical and clinical trials, particularly for neuromuscular disorders, disuse atrophy, weight-loss-related muscle decline, sarcopenia, age-related muscle decline, and for general muscle health in both humans and veterinary medicine.

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1. Overview and Unmet Needs for Muscle Health

Muscle health is central to maintaining physical capability, longevity, and quality of life in both humans and animals. Globally, more than 1.63 billion people are affected by musculoskeletal conditions, making them a leading cause of disability worldwide[1]. From 1990 to 2021, the number of reported cases of musculoskeletal disorders increased to over 367 million new cases annually, collectively accounting for a major proportion of global years lived with disability[2].

Muscle wasting and dysfunction can result from a number of potential causes[3] (Figure 1). These conditions range from the most common muscle disorders in humans, like sarcopenia, a progressive, age-related loss of muscle strength and mass, to rare, hereditary muscular dystrophies such as Duchenne, Becker, and facioscapulohumeral muscular dystrophy (FSHD). The global prevalence of sarcopenia in older adults ranges between 10 to 27%. Sarcopenia is closely associated with increased risks of mortality, falls, fractures, and reduced quality of life[4]. While genetic muscular dystrophies are associated with similar symptoms and risks, they typically appear much earlier in life and exhibit faster disease progression and, in many cases, accelerated mortality[5,6]. Similarly, in animals, muscle wasting and dysfunction can be the result of orthopedic injury, chronic disease, or inactivity during rehabilitation, with sarcopenia and cachexia now recognized as major contributors to morbidity in aging pets[7]. Muscle diseases significantly impact function and independence for humans and animals, often leading to loss of mobility, frailty, and increased need for medical care[8].

Current approaches to preserving muscle health primarily focus on symptomatic management rather than disease-modifying treatments. Exercise-based interventions, such as resistance training, and nutritional strategies, such as high-protein or leucine-rich diets, are some of the most common approaches[9,10]. However, these approaches are not suitable or accessible for all. While certain targeted therapies, such as exon-skipping and gene therapies for Duchenne muscular dystrophy[11,12], or testosterone replacement for hypogonadal men[13], can modify disease progression in a specific context, there remain no widely approved pharmacologic therapies aimed at prompting or preserving muscle mass across broader muscle-wasting conditions. Thus, there is a substantial unmet need for safe and effective interventions to support muscle health and recovery in both human and animal populations.

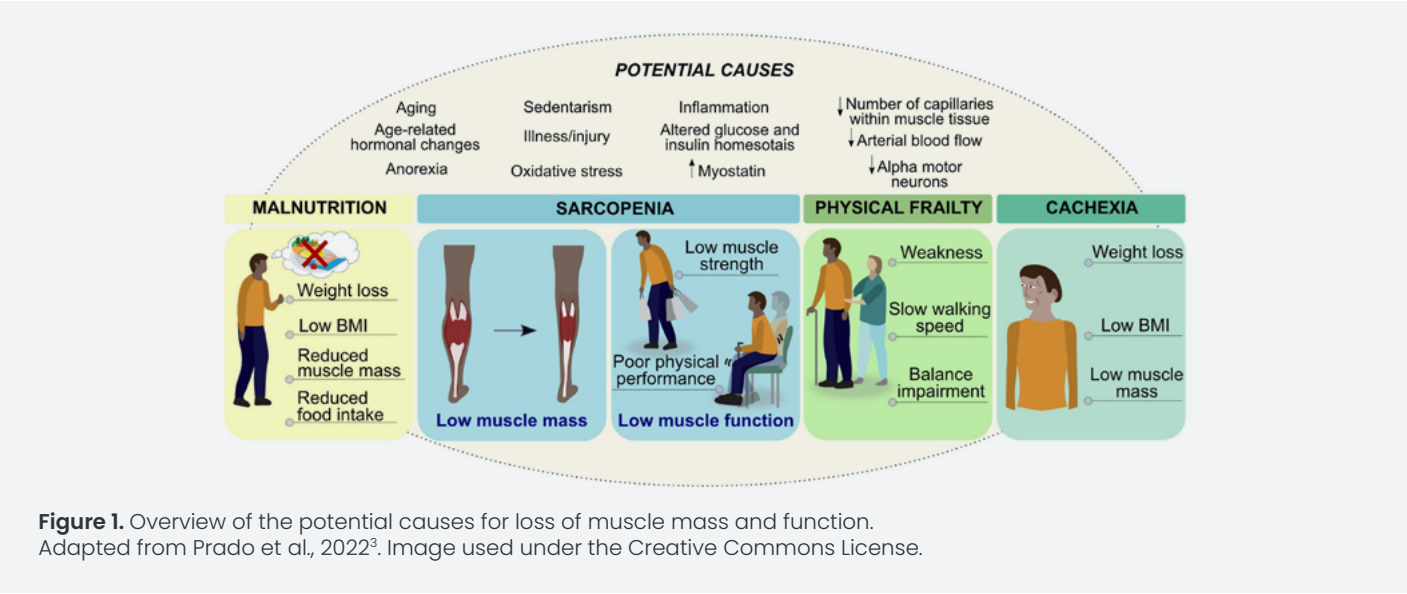

Figure 1. Overview of the potential causes for loss of muscle mass and function.
Adapted from Prado et al., 2022[3]. Image used under the Creative Commons License.

2. Fortetropin®: A Promising Nutraceutical for Muscle Health

With the lack of treatments that halt or reverse losses in muscle mass and function, there is growing interest in nutraceuticals and dietary supplements as complementary strategies for muscle health in both humans and animals.

Fortetropin® is a bioactive compound by MYOS, derived from fertilized chicken egg yolk via a patented process believed to preserve the natural structure and bioactivity of key proteins, peptides and lipids naturally present in fertilized egg yolk. The resulting product contains a high proportion of fat (55%), protein (33%), and carbohydrates (7%)[14].

Preclinical and clinical studies have shown that the nutraceutical, Fortetropin® can address the underlying biological drivers of muscle loss, enhancing muscle mass, endurance, and functional performance[14–16]. The favorable safety profile, accessibility, and potential for combination use with future pharmacological treatments make nutraceuticals a promising approach to address the current gap for muscle health and muscle loss management.

2.1. Unique Advantages of Fortetropin® in Muscle Health

No exercise requirement: Although exercise is strongly recommended, in animal and human studies, Fortetropin® has demonstrated muscle-preserving and muscle-building effects in both active individuals and those unable to exercise, such as during immobilization, disuse, or post-surgery periods[14,16,17].

Safe: Fortetropin® is a bioactive compound produced under patented pathogen-free manufacturing conditions. It is safe and well-tolerated in multiple species, including humans, dogs, cats, and horses, under a broad range of doses[14,15,17–20].

3. Benefits of Fortetropin®: Mechanistic and in vitro Evidence

In vitro and mechanistic studies have provided insight into the bioactivity and muscle-supporting properties of fertilized chicken egg yolk, the source material for Fortetropin®. Findings from these studies are indicative of fertilized chicken egg yolk containing components that activate anabolic and regenerative pathways central to muscle growth and recovery, which, when processed into Fortetropin®, provide biological bases for effects observed in animal and human studies.

3.1. Myoblast and Myotube Studies

Multiple in vitro studies have demonstrated that fertilized egg yolk, the source for Fortetropin®, promotes myoblast proliferation and differentiation, increasing the expression of myogenic markers such as MyoD and myogenin, and enhancing myotube formation and maturation[21–23].

The effect of different concentrations of fertilized chicken egg yolk extract on the viability, morphology, and myogenic gene expression of C2C12 myoblasts, a standard model for muscle regeneration, was evaluated[21]. Key findings from this study included enhanced proliferation and differentiation in both fertilized and unfertilized egg yolk extracts, while fertilized egg yolk led to more pronounced myotube formations, indicating enhanced muscle-like structure development[21]. Additionally, increased expression of muscle-specific genes such as MyoD and myogenin was observed with fertilized egg yolk extract, suggesting muscle growth pathway activation[21]. This study demonstrated the potential of fertilized egg yolk to address age-related muscle loss by promoting muscle cell growth and differentiation, as well as enhancing muscle recovery post-surgery through improved myoblast activity.

3.2. Proteomic Characterization

Proteomic analysis of fertilized egg yolk fractions, the source material for Fortetropin®, was conducted, successfully identifying bioactive proteins involved in angiogenesis and defense against pathogens[24]. From the study, 225 distinct proteins were identified. Eighteen proteins were significantly altered by fertilization: nine proteins enriched in angiogenesis and host-defense pathways were upregulated, and nine proteins associated with static nutrient storage were downregulated[24]. The pro-angiogenic and anti-microbial proteins upregulated included pleiotrophin, histidine-rich glycoprotein, mannose-binding lectin, β-defensin 11, serum amyloid p-component, and ovastatin[24]. This study demonstrated that fertilization activates early embryonic signaling in yolk plasma, shifting the proteome toward regenerative, immuno-protective function[24]. This supports MYOS' hypothesis that fertilization enriches biologically active proteins and peptides that may, via unknown or indirect mechanisms, contribute to processes relevant to tissue repair, angiogenesis, and muscle recovery.

4. Benefits of Fortetropin®: Animal and Human Evidence

Fortetropin® has been shown in preclinical and clinical studies to modulate anabolic and catabolic pathways that influence muscle protein turnover. These combined effects make Fortetropin® an ideal therapy to improve muscle health and alleviate symptoms of muscle-wasting diseases, offering a potentially disease-modifying approach to muscle preservation and regeneration.

4.1. Enhances muscle protein synthesis and anabolic signaling

The PI3K-Akt-mTOR pathway, activated by anabolic stimuli such as IGF-1, is a key, well-studied anabolic signaling cascade. With advancing age, this pathway becomes less responsive to stimuli, leading to reduced Akt phosphorylation and mTOR activation, which limits translation initiation and enhances proteolytic activity via FOXO-mediated gene expression[25,26].

The effects of Fortetropin® (0.26 g) supplementation, at the human equivalent dose of 13.2 g, on the molecular signaling mechanisms underlying muscle protein synthesis in rats were investigated in a study at the University of Tampa[15]. The mTOR pathway is central to skeletal muscle growth and repair, regulating translation initiation and protein synthesis through the phosphorylation of several downstream protein targets, including p70S6K, rpS6, and 4EBP1. In the Fortetropin® plus exercise group, higher phosphorylation levels of p70S6K, rpS6, and 4EBP1 were observed compared to the control[15] (Figure 2). This indicates greater mTOR activation and translation initiation, suggesting Fortetropin® amplified the exercise-induced anabolic signaling response. Additionally, the phosphorylation of eEF2, a protein associated with protein translation rates, was prevented in the Fortetropin® plus exercise group. Since phosphorylated eEF2 inhibits peptide chain elongation, lower phosphorylation indicates enhanced translational elongation, and therefore more efficient protein synthesis[15] (Figure 2).

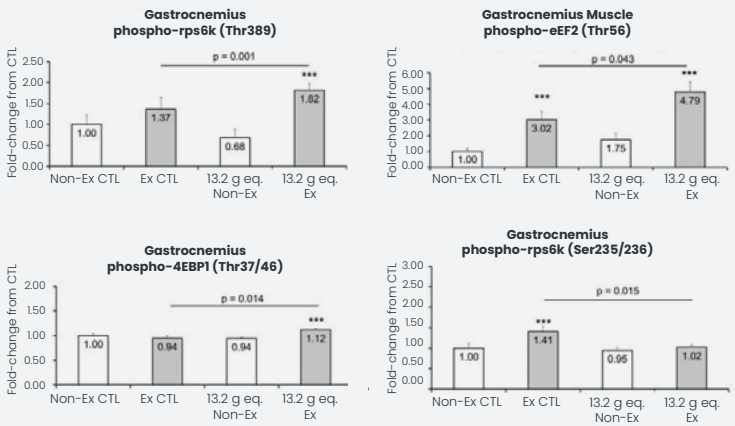

Figure 2. Fortetropin®, at the human-equivalent of 13.2 g, significantly increased the phosphorylation of p70S6K, rpS6, and 4EBP1, and decreased the phosphorylation of eEF2 in the rats plus exercise group. Adapted from Sharp et al., 2016[15].

In a randomized, placebo-controlled trial involving healthy young men (n=24), conducted at McMaster University over a 6-week study period, participants underwent a 2-week run-in, a 2-week single-leg immobilization phase, and a 2-week recovery phase[17]. Participants were randomized to receive a cheese powder placebo or Fortetropin® (19.8 g/day), and blood samples were collected on days 1 and 42[17]. At day 42, participants who received Fortetropin® had a significant 38% increase in phosphorylated p70S6K compared to placebo, indicating activation of the mTOR pathway[17] (Figure 3).

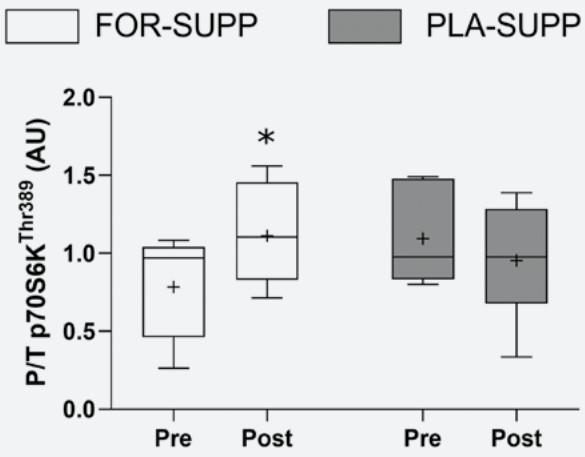

Figure 3. Fortetropin® supplementation increases the phosphorylation of p70S6K in humans post limb immobilization. FOR: Fortetropin®; PLA: Placebo. Adapted from Lim et al., 2023[17].

The effects of Fortetropin® (19.8 g/day) on muscle protein synthesis were also investigated in a randomized, double-blind, placebo-controlled trial involving older adults conducted at UC Berkeley[27]. Participants (n=20; 10 males and 10 females) were assigned to receive either Fortetropin® or a cheese powder placebo, daily, over 21 days[27]. Older healthy adults were selected as a model population for sarcopenia, as they exhibit age-associated declines in muscle protein synthesis. Fractional synthetic rate (FSR) of muscle proteins was measured using heavy water (^2H_2O) labeling and muscle biopsies of the vastus lateralis. Compared to placebo, Fortetropin® supplementation significantly increased protein synthesis rate across myofibrillar, sarcoplasmic, and mitochondrial gene ontologies[27]. The overall magnitude of this effect represented an 18% higher muscle protein FSR in the Fortetropin®-treated group compared to placebo[27]. A second unpublished study performed by MYOLOGICA and UC Berkeley also showed an increase in FSR in caloric-restricted rats administered Fortetropin®.

The data from these studies highlight Fortetropin®'s anabolic effects by increasing muscle protein FSR and enhancing phosphorylation of mTOR-related signaling molecules, thereby activating the mTOR pathway to support protein synthesis and muscle growth[15,17,27].

4.2.
Suppresses myostatin and muscle protein breakdown

Myostatin, a member of the TGF-β superfamily, acts as a negative regulator of muscle growth[28]. Myostatin inhibits the Akt/mTOR pathway, thereby suppressing protein growth, while also activating FOXO transcription factors that upregulate E3 ubiquitin ligases such as Atrogin-1 and MuRF1, promoting protein degradation through the ubiquitin-proteasome and autophagy pathways[26,28]. Aging muscle, and in individuals with genetic diseases such as FSHD, expression of ubiquitin E3 ligases is enhanced, which increases proteolysis and accelerates muscle protein degradation.

In the rat model described in the study at the University of Tampa, discussed previously in section 4.1, Fortetropin® suppressed the mRNA expression of Atrogin-1, MuRF-1, and poly-ubiquitinated protein, suggesting Fortetropin® may reduce the activation of the ubiquitin-proteasome degradation pathway[15]. The study also evaluated the effects of Fortetropin® in resistance-trained men (n=45) over a 12-week, double-blind, placebo-controlled trial[15]. Participants were randomized to the placebo group, Fortetropin® (6.6 g/day), or Fortetropin® (19.8 g/day), administered alongside a standardized resistance training program[15]. Both Fortetropin® doses produced significant post-training decreases in serum myostatin levels compared with the placebo group[15] (Figure 4).

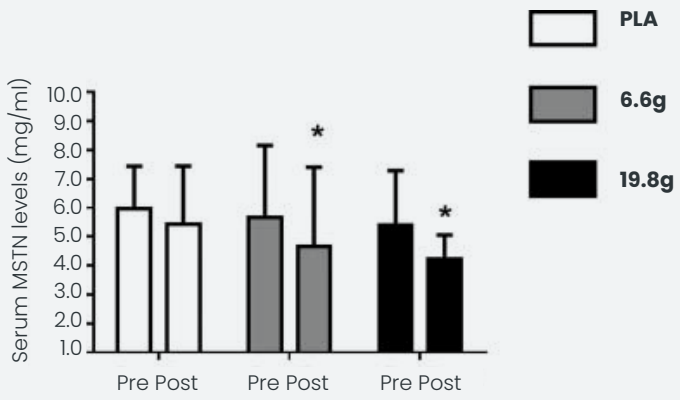

Figure 4. Fortetropin® (6.6 g and 19.8 g) significantly decreased serum levels of myostatin (MSTN) in resistance-trained humans. Adapted from Sharp et al., 2016[15].

A randomized, double-blind, placebo-controlled trial investigated 100 dogs post-orthopedic surgery, carried out at Kansas State University[14]. The 12-week study included 8 weeks of forced exercise restriction followed by a 4-week return to activity period[14]. Dogs were assigned to the placebo group where they received cheese powder, or Fortetropin® (300 mg/kg/day)[14]. During the immobilization phase (weeks 0-8), serum myostatin levels significantly increased, and there was a significant reduction in thigh circumference in the dogs of the placebo group. In contrast, myostatin levels remained stable, and thigh circumference was maintained or increased in the dogs of the Fortetropin® group[14].

Similarly, in the human limb immobilization study, previously discussed in section 4.1, Fortetropin® supplementation prevented the 59% rise in serum myostatin levels observed in the placebo group after 42 days[17] (Figure 5).

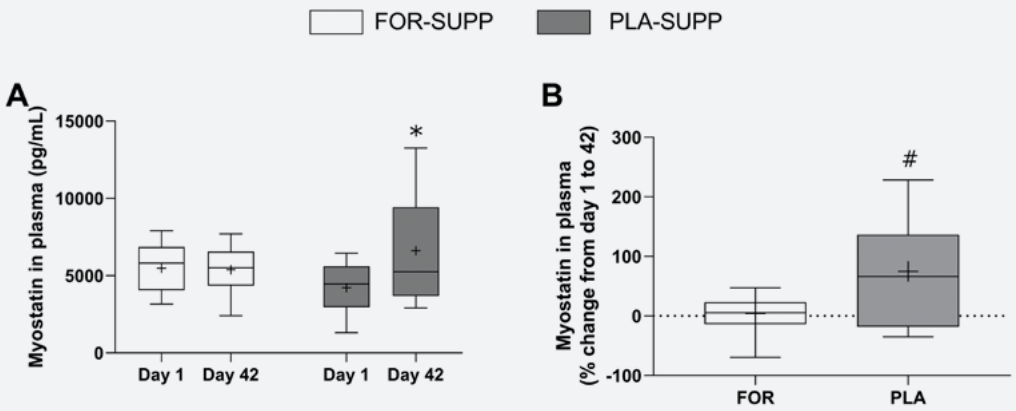

Figure 5. Fortetropin® supplementation prevented the rise in circulating myostatin in humans post limb immobilization. FOR: Fortetropin®; PLA: Placebo. Adapted from Lim et al., 2023[17].

Across human and animal models, Fortetropin® consistently suppresses myostatin levels and has also been shown to reduce the expression of key ubiquitin E3 ligases in a rat model, supporting a dual mechanism of catabolic inhibition and muscle preservation during disuse, rehabilitation, and recovery[14,15,17].

4.3.
Increases lean muscle mass and preserves strength

In the human resistance-training trial (University of Tampa, discussed in section 4.2), Fortetropin® supplementation (6.6 g/day and 19.8 g/day) significantly increased lean body mass and muscle thickness when compared to placebo[15]. Participants who received 19.8 g Fortetropin® demonstrated greater gains in lean body mass than those who received 6.6 g, indicating a dose-dependent anabolic effect[15] (Figure 6).

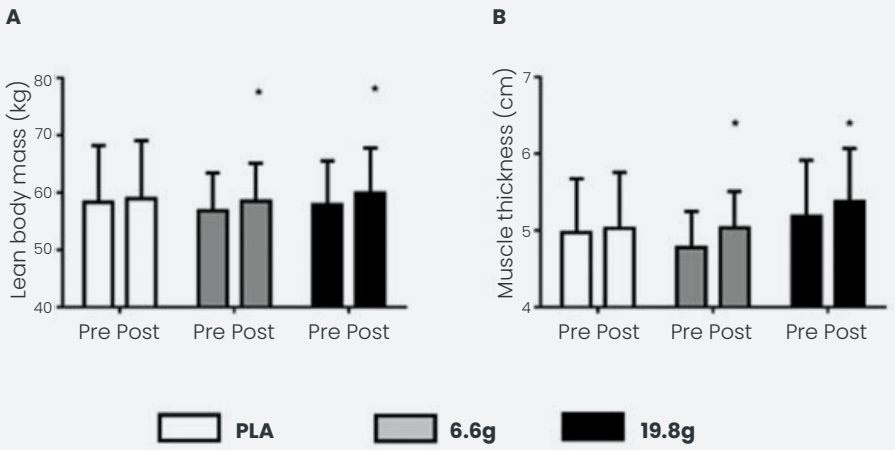

Figure 6. Fortetropin® (6.6 g and 19.8 g) significantly increased lean body mass and muscle thickness in resistance-trained humans. Adapted from Sharp et al., 2016[15].

The effects of Fortetropin® were also evaluated in cats with chronic kidney disease (CKD), a condition that affects 30 to 40% of cats over the age of 10 and up to 80% of cats over 15 years. As CKD progresses, cats often suffer from muscle wasting, reduced mobility, and diminished quality of life. Over a 12-week period, cats in either Stage II (n=6) or Stage III (n=6) of chronic kidney disease received Fortetropin® (2 g/day) in a study conducted at North Carolina State University[20]. Lean muscle mass increased in 67% of cats, with an average gain of 881 g in lean muscle mass per cat, and an average fat gain of 474 g per cat[20]. Importantly, this increase occurred without aggravating symptoms of the CKD observed to occur with protein supplementation.

Furthermore, a randomized, placebo-controlled trial was conducted involving horses (n=15) aged 9 to 27 years at a veterinary rehabilitation center[18]. Horses were randomized into either the placebo group, Fortetropin® (24 g/day), or Fortetropin® (48 g/day) for 2 weeks. In the 48 g/day group, body weight increased by 10.6% relative to baseline, suggesting rapid anabolic support even over a short supplementation period[18].

Across species, Fortetropin® consistently increases or preserves lean muscle mass, supporting its role as a cross-species nutraceutical for promoting muscle health, enabling muscle preservation, and aiding recovery.

4.4.
Improves mobility and functional performance

The dog study (Kansas State University, discussed in section 4.2) also investigated weight-bearing capacity in the dogs post-orthopedic surgery, and found that by the end of the immobilization period (week 8), those who received Fortetropin® had an enhanced weight-bearing capacity of 7% compared to 4.9% in the placebo group[14], thus indicating that Fortetropin® may also accelerate recovery.

An additional randomized, double-blind, placebo-controlled trial involving geriatric or senior dogs with reduced mobility (n=46) over 12 weeks was conducted at Kansas State University[16]. Participants received either placebo or Fortetropin® (3–12 g/day depending on size), and mobility was assessed using the Liverpool Osteoarthritis in Dogs questionnaire, a validated, owner-completed survey[16]. Those who received Fortetropin® had a significant improvement in mobility score at both week 6 and week 12 compared to baseline[16]. In contrast, the placebo group did not show any significant improvement in mobility throughout the study duration[16].

Functional improvements observed in the discussed studies mirror structural gains, supporting Fortetropin®'s role in recovery and performance maintenance, and as an adjunct to physical therapy or joint management programs.

5. Fortetropin®: Safety and Tolerability

Fortetropin® has demonstrated a favorable safety and tolerability profile in both preclinical and clinical studies across species, including mice, rats, cats, dogs, horses, and humans.

5.1. Safety Profile in Cats

The safety and tolerability of Fortetropin® in cats (n=12) were presented at the 2021 American College of Veterinary Internal Medicine Forum[19]. There were no adverse events observed in any of the cats that received 1 g/day (n=4) and 2 g/day doses (n=4) of Fortetropin® over the 2-week study[19]. There was a single episode of diarrhea observed on day 6 in one cat in the placebo group[19]. After the first phase of the study, the cats (n=4) that were initially in the placebo group were treated with a high dose of Fortetropin® (4 g/day) for 2 weeks[19]. On day 8, a single episode of vomiting was observed in one cat from this treatment group (4 g/day Fortetropin®). Therefore, the frequency of mild adverse events was no greater in the cats that received a high dose of Fortetropin® (4 g/day) relative to the cats that received the placebo treatment[19]. Additionally, in the study involving cats with CKD (n=12), only two cats exhibited brief gastrointestinal intolerance lasting 1 to 2 days, and there were no renal or systemic complications reported[20].

5.2. Safety Profile in Dogs

During the two canine studies outlined in the studies at Kansas State University, there were no adverse events reported with Fortetropin® (3–12 g/day)[14,16].

5.3. Safety Profile in Horses

In the equine study, horses were administered Fortetropin® 24 and 48 g/day (approximately 72–109 mg/kg) over two weeks[18]. The initial formulation containing only Fortetropin® was unpalatable; therefore, sugar (4.5–9 g/serving) was added to the Fortetropin®, and the study resumed. Fortetropin® with added sugar (for taste) was well-tolerated, with no adverse events reported[18].

5.4. Safety Profile in Humans

Across all clinical investigations, Fortetropin® was well-tolerated[15,17,27]. Notably, in the study carried out at McMaster University, Fortetropin® (19.8 g/day) was reported as safe and tolerable over the 6-week study period[17]. Additionally, no adverse events or dropouts were reported in the UC Berkeley study, supporting Fortetropin®'s safety profile[27].

6. Discussion and Clinical Translation

Fortetropin® represents a promising advancement in the support of muscle health, with consistent evidence demonstrated across cellular, animal, and human studies. The body of preclinical and clinical evidence indicates that Fortetropin® acts through multiple complementary mechanisms, by upregulating anabolic signaling pathways such as PI3/Akt/mTOR to enhance muscle protein synthesis, while also downregulating catabolic mediators, including myostatin and ubiquitin E3 ligases, to suppress muscle protein degradation[14,15,17]. The balanced modulation of anabolic and catabolic processes makes Fortetropin® a promising, exercise-independent, non-pharmacological approach for maintaining or increasing lean muscle mass and supporting overall muscle health.

Fortetropin®'s positive effects on muscle health have been observed across a range of species, underscoring its biological relevance and translational potential. In vitro studies demonstrate activation of myogenic genes and enhanced myotube formation[21–23], while animal studies reveal preserved muscle mass, improved mobility, and accelerated recovery following immobilization[14,16,18,20]. Human clinical trials add to these findings, showing increased muscle protein synthesis rates, decreased serum myostatin levels, and measurable gains in lean muscle mass[15,17,27]. Together, these findings highlight Fortetropin®'s cross-species efficacy and reinforce its potential role in both medical and veterinary applications.

Fortetropin® is the key ingredient in MYOS' portfolio of muscle health products, designed to translate scientific findings into practical, real-world solutions for both humans and animals. Across species, Fortetropin® shows promise as a supportive intervention for sarcopenia, rehabilitation following immobilization or surgery, and improving mobility, overall muscle health, and quality of life. As a safe, all-natural, exercise-independent nutraceutical, Fortetropin® offers a versatile approach to muscle preservation, serving as a powerful adjunct to rehabilitation and nutritional programs, or as a stand-alone intervention to support muscle health.

6.1. Limitations and Future Directions

While the preclinical and clinical studies outlined in this white paper demonstrate that Fortetropin® has been linked to significant positive effects on muscle health, there is still a need for longer-term studies to determine whether these observed shorter-term benefits translate into sustained improvements in muscle strength, fiber morphology, and physical performance. Future studies investigating fiber-type composition, satellite cell activation, and mitochondrial function would provide further understanding of Fortetropin®'s cellular and molecular mechanisms of action.

Further clinical trials in older adults diagnosed with sarcopenia are warranted to evaluate functional outcomes such as grip strength, gait speed, and mobility, as well as longer-term safety and tolerability. Additionally, studies in populations with genetic muscle diseases such as FSHD and possibly Duchenne Muscular Dystrophy will offer insight into how Fortetropin®'s potential to support disease management and slow progression.

Together, findings from the current studies position Fortetropin® as a safe, exercise-independent nutraceutical that may serve as an adjunct or stand-alone strategy for preserving and improving muscle health for both humans and animals.

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