

Geriatric Syndrome—Bone Up, Muscle Up, and More with Myostatin Inhibition

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How Myostatin Inhibition Supports Bone Density, Prevents Sarcopenia, and Enhances Musculoskeletal Health in Aging Populations

Osteoporosis and sarcopenia are major concerns for aging individuals, often leading to fractures and loss of independence. This article explores myostatin inhibition's role in improving bone density and muscle mass and highlights Fortetropin as a promising natural intervention for musculoskeletal health.

The most common metabolic bone disease in the United States,¹ osteoporosis, leads to significant morbidity and mortality among senior patients and is responsible for costing them their independence. Osteoporosis often goes undetected until a patient suffers from a fracture. In adults 50 or older, any new fracture increases the risk of subsequent fractures, especially in the year after the first fracture.¹

Many people experience upset stomachs or heartburn when taking medications to treat osteoporosis. Rarer side effects include atypical femoral fracture (a break in the thighbone) and jaw osteonecrosis (the slowness or failure to heal after dental work).

Clearly, a better alternative is needed, which may very well be the inhibition of myostatin. Myostatin inhibition is a vital clinical consideration for functional medicine providers who want to get at one of the root causes of low bone density.

What Is Myostatin?

Myostatin is a growth differentiation factor (GDF-8) in the transforming growth factor-beta (TGF- β) superfamily. It blocks skeletal muscle growth and is involved in sarcopenia (muscle wasting).² However, emerging research suggests that myostatin also involves bone breakdown and osteoporosis.

From Building Muscle to Protecting Bone

A lot of the initial research on the inhibition of myostatin indicates that blocking myostatin can lead to increased protein synthesis, improved lean body mass, and muscle growth. It is becoming apparent that inhibiting myostatin also can play a role in bone health.

Muscle and bone health are connected. By increasing muscle mass, more mechanical load is generated on bones. When this happens, it stimulates the activity of bone-building cells known as osteoblasts, which enhances bone mineral density (BMD). Myostatin is highly expressed in sites of fractures.³ Increasing muscle mass by inhibiting myostatin improves bone mineral density and reduces fracture risk.³

In addition, inhibiting myostatin improves bone resorption, the process by which damaged bone is broken down and absorbed by the body.³ Bone remodeling regulates both the quantity and quality of bone.⁴ During bone remodeling, osteoclasts first resorb bone, and then bone-building osteoblasts fill in the spaces created by this process.⁴

Bone formation is a balancing act between the activity of bone-building osteoblasts and bone-destroying osteoclasts. Blocking myostatin can lead to bone formation by increasing the activity of osteoblasts and suppressing osteoclasts.³

The relationship between the muscles and bones is more than just mechanical; muscle increases the load, and bone acts as an attachment site. Emerging evidence suggests that bone and muscle act as secretory endocrine organs that affect each other's function.⁵ The crosstalk that occurs between muscle and bone occurs due to a number of factors, including bone-destroying signals conveyed by myostatin.⁵

Sarcopenia's Connection to Bone Loss

Sarcopenia is a progressive decline of skeletal muscle mass and function. Up to 29% of older persons have this muscle disease.² The presence of low muscle mass plus low muscle strength or low physical performance indicates a sarcopenia diagnosis.²

Inhibiting myostatin leads to the prevention of sarcopenia, which indirectly supports bone health.⁶ The connection between muscle and bone health is evident in the newly created term osteosarcopenia, which refers to the existence of both osteoporosis and sarcopenia in a patient.⁶ Muscle-bone interactions at the biomechanical, cellular, paracrine, endocrine, neuronal, or nutritional levels may play a role in the development of osteosarcopenia.⁶

A Novel Way To Support Muscles and Bones

Fortetropin is a bioactive myostatin inhibitor derived from fertilized egg yolk extract. Fortetropin increases muscle protein synthesis and inhibits muscle atrophy by reducing circulating myostatin. Fortetropin increases lean body mass, reduces markers of protein breakdown consistent with inhibition of myostatin, and increases mTOR signaling, which is associated with muscle growth. All of these actions are beneficial to both muscle and bone strength.⁷⁻⁹

Research demonstrates Fortetropin's effects on protein synthesis and muscle health. For example, 21 days of Fortetropin supplementation in 10 healthy older men and 10 women resulted in 18% higher muscle protein synthesis compared with a placebo.⁸ According to the lead researcher, "Stimulating muscle protein synthesis in elderly patients could potentially result in increased muscle mass and better outcomes for those with sarcopenia."

In another human study of 37 resistance-trained college-age males, lean body mass dramatically increased in patients given Fortetropin compared with placebo.⁷ The men taking 19.8 grams had greater lean body mass improvements than those given 6.6 grams. In both groups, there were pronounced increases in muscle thickness, and Fortetropin decreased markers of protein breakdown.

In 24 healthy young men who underwent single-leg immobilization for two weeks, Fortetropin also blocked the rise in circulating myostatin that would have normally occurred.¹⁰

Animal research supports Fortetropin's use, such as a study in dogs suffering from disuse muscle atrophy after undergoing tibial plateau leveling osteotomy (TPLO) surgery.⁹ The dogs given Fortetropin experienced no change in myostatin levels. By comparison, myostatin levels increased over eight weeks in the placebo-treated dogs. Thigh circumference did not change in the animals given Fortetropin, while the placebo-treated dogs had a reduction in thigh circumference over eight weeks.

Fortetropin's Effects on Bones

By inhibiting protein breakdown and promoting protein synthesis, Fortetropin may also benefit bone health.⁸ A low-protein diet or a reduced ability to synthesize protein can suppress bone formation, resulting in lower bone density and fragility.¹¹ Additionally, Fortetropin's ability to inhibit muscle loss is connected to protein metabolism⁷ and can potentially improve bone weakness and reduce fracture risk.

Inhibiting Myostatin Through Lifestyle

In addition to supplementing with Fortetropin, I encourage patients to focus on lifestyle-related approaches that can support myostatin inhibition. These include:

- Engaging in resistance exercise
- Eating a high-protein diet
- Consuming a nutritious diet with a variety of phytonutrients
- Getting enough sleep to help the body heal

Conclusion

Emerging evidence indicates that inhibition of myostatin is an effective way to promote muscle and bone health. Osteoporosis and fractures can severely limit the independence of older patients, and taking a proactive approach to supporting bone health can lead to greater mobility as patients age. Fortetropin, a bioactive myostatin inhibitor derived from fertilized egg yolk extract, effectively inhibits myostatin and promotes lean body mass, protein synthesis, and muscle health. Combining Fortetropin with lifestyle approaches such as resistance exercise and eating enough protein can yield excellent results.

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Bio

Dr. Chris D. Meletis is an educator, international author, and lecturer. His mission is “Changing the World’s Health One Person at a Time.” He believes that when people become educated about their bodies, actual change and wellness begin.

He has written over 200 nationally published articles and over a dozen books to share his passion and amazement of the human body and what is required to maintain health. Dr. Meletis served as Dean of Naturopathic Medicine and Chief Medical Officer for seven years at NCNM, the oldest naturopathic university in North America. The American Association of Naturopathic Physicians awarded him the 2003 Physician of the Year.