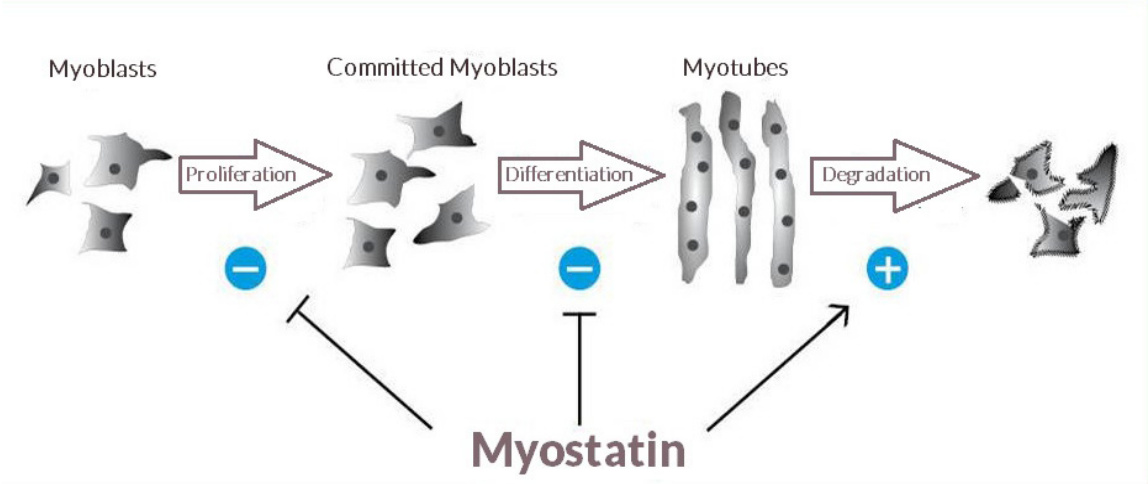
Myostatin

Myostatin belongs to the transforming growth differentiation factor-ß (TGF-ß) super family and is known as a negative regulator of muscle growth. The protein is synthesized in muscle and released into circulation where it binds locally or systemically to specific receptors. One of its major effects is the inhibition of proliferation and differentiation of myoblasts. A pharmacological or genetic blockade of the myostatin signal transduction pathway leads to a hypermuscular phenotype.

Studies have shown measurement of myostatin as an indicator in:

- Bone and muscle strength
- Limb wound recovery
- Muscular distrophies
- Diabetes and exercise
- Muscle wasting diseases
- Sports relevant research

Negative Regulation of Muscle Growth by Myostatin



Assays are For Research Use Only

Myostatin

Myostatin as a Negative Regulator of Muscle Growth

The protein is an important negative regulator of muscle mass by inhibiting muscle growth and promoting muscle protein degradation. Studies have shown myostatin knockout mice are significantly larger than wild-type animals and exhibit a profound and widespread increase in skeletal muscle mass due to an increase of muscle fiber number (hyperplasia) and thickness (hypertrophy).[1]

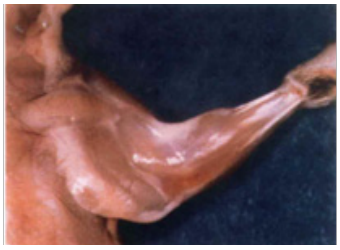


Figure 1: *Wild-type mouse muscle*[1]

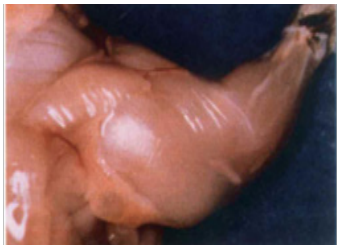


Figure 2: *Knockout mouse muscle*[1]

Featured Assay

Myostatin ELISA

The Myostatin ELISA is a sensitive and quantitative assay for same day determination in small sample volumes.

Catalog #:	30-1012
Sample Type:	Plasma, serum
Sample Size:	30 µL
Range:	0.27 - 195 ng/mL
Sensitivity:	0.27 ng/mL
Incubation:	5 hours

Myostatin and Bone Action

Another tissue which is sensitive to Myostatin action is the bone. Studies have shown myostatin knockout mice have a higher bone density than control mice and they display a change in bone architecture. This could be an indirect effect of myostatin on the bone as the bigger muscles of the knockout animals exert more force on the bones. However, recent evidence demonstrates that the changed bone phenoptype could also be a direct effect of myostatin. Myostatin and its receptor are expressed in bone in the early phase of bone repair and one hypothesis is that myostatin represses the differentiation of precursor cells in the fracture callus.[2]

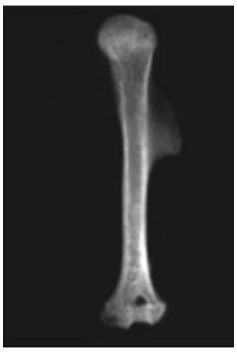


Figure 3: *Humerus of Wild-type mouse*[2]

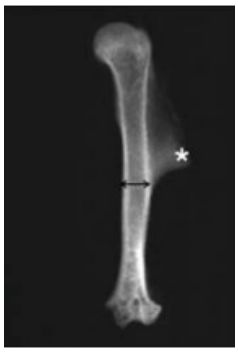


Figure 3: *Humerus of Knockout mouse*[2]

References

1. Mc Pherron AC, et al. Nature. 1997; Vol 387 No 6628: 83-90
2. Elkasrawy MN, Hamrick MW. J Musculoskelet Neuronal Interact . 2010; Vol 10 No 1: 56-63
3. Gonzales-Cadavid NF et al. (1998) Proc Natl Acad Sci USA. 1998; Vol 95: 14938-14943
4. Yarasheski KE.J Nutr Health Aging. 2002; Vol 6: 343-348
5. Zhao B et al. Biochem Biophys Res Comm. 2005; Vol 337 No 1:248–255
6. Hittel et al. Medicine & Science in Sports & Exercise. 2010; DOI: 10.1249/MSS.0b013e3181e0b9a8

Myostatin and obesity/insulin resistance

7. Ehehalt et al. Investigation of Myostatin Serum Levels Before and After a 6-Month Lifestyle Intervention Program in Obese Children. Exp Clin Endocrinol Diabetes. 2011; Vol 119 No 4: 238-42
8. Hittel et al. Myostatin decreases with aerobic exercise and associates with insulin resistance. Med Sci Sports Exerc. 2010; Vol 42 No 11: 2023

Myostatin and heart failure

9. Wintgens et al. Plasma myostatin measured by a competitive ELISA using a highly specific antiserum. Clin Chim Acta. 2012; Vol 413 No 15-16: 1288-94
10. Gruson et al. Increased plasma myostatin in heart failure. European Journal of Heart Failure. 2011; Vol 13 No 7: 734-736
11. Zamora et al. Serum myostatin levels in chronic heart failure. Rev Esp Cardiol. 2010; Vol 63 No 8: 992-6

Myostatin and kidney failure

12. Cheung et al. Modulation of melanocortin signaling ameliorates uremic cachexia. Kidney International. 2008; Vol 74 No 2: 180-186

Myostatin and cancer

13. Lokireddy et al. Myostatin is a novel tumoral factor that induces cancer cachexia. Biochem J. May 23, 2012

Assays are For Research Use Only