Zacks Small-Cap Research

July 21, 2014
Jason Napodano, CFA
David Bautz, PhD
312-265-9421 / jnapodano@zacks.com

scr.zacks.com
10 S. Riverside Plaza, Suite 1600, Chicago, IL 60606

MYOS Corporation (MYOS - NASDAQ)

MYOS: Initiating coverage of MYOS Corporation, a company focused on the health and performance of muscles…

Current Recommendation	**Buy**
Prior Recommendation	N/A
Date of Last Change	07/16/2014
Current Price (07/21/14)	$14.72
Target Price	**$20.00**

INITIATION

We are initiating coverage of MYOS Corporation (NASDAQ: MYOS) with a Buy rating and a $20.00 price target. MYOS is a biotherapeutics and bionutrition company focusing on improving muscle health and function to combat muscle related diseases. The company's commercial division has products on the market, which have been clinically shown to reduce serum myostatin levels.

MYOS has differentiated itself from other bionutrition companies based on the solid scientific basis underlying the development of the currently marketed products as well as the current research initiatives underway targeting muscle health. Based on a discounted cash flow analysis, we feel the shares are worth $20.00 and would make a good choice for individuals looking to invest in the emerging area of muscle health.

SUMMARY DATA

52-Week High	**$15.65**
52-Week Low	**$11.25**
One-Year Return (%)	**N/A**
Beta	**N/A**
Average Daily Volume (sh)	**10,848**
Shares Outstanding (mil)	**3**
Market Capitalization ($mil)	**$44**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**0**
Insider Ownership (%)	**N/A**
Annual Cash Dividend	**$0.00**
Dividend Yield (%)	**0.00**
5-Yr. Historical Growth Rates	
Sales (%)	**N/A**
Earnings Per Share (%)	**N/A**
Dividend (%)	**N/A**
P/E using TTM EPS	**N/A**
P/E using 2014 Estimate	**N/A**
P/E using 2015 Estimate	**N/A**

Risk Level	**Above Average**
Type of Stock	**Small-Growth**
Industry	**Med-Biomed/Gene**

ZACKS ESTIMATES

Revenue
(In millions of $)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2013	$0.3 A	$0.7 A	$0.9 A	$1.4 A	$3.3 A
2014	$1.5 A	$1.7 E	$1.9 E	$2.1 E	$7.2 E
2015					$9.0 E
2016					$13.0 E

Earnings per Share
(EPS is operating earnings before non-recurring items)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2013	-$0.47 A	-$0.44 A	-$0.35 A	-$0.67 A	-$1.93 A
2014	-$0.16 A	-$0.20 E	-$0.12 E	-$0.15 E	-$0.62 E
2015					-$0.49 E
2016					-$0.04 E

© Copyright 2014, Zacks Investment Research. All Rights Reserved.

WHAT'S NEW

Initiating Coverage

We are initiating coverage of MYOS Corporation with a Buy rating and a $20.00 price target.

MYOS is a bionutrition and biotherapeutic company focused on characterizing regulatory pathways aimed at maintaining or improving the health and performance of muscle tissue. While research and development focuses on small molecules and pathways affecting muscle, the company's bionutritional arm is focusing on nutritional supplements along with functional and medical foods for consumers and hospitals. The company currently has two marketed products; MYO-X, powered by MYO-T12, which is distributed by Maximum Human Performance (MHP) to specialty retail and other outlets and a second private label product in the age management market.

MYO-T12 is a dietary supplement that has been shown in two clinical studies to temporarily decrease the levels of serum myostatin. Myostatin is a protein produced by most vertebrate organisms including humans. Research on animals and humans with a genetic deficiency for producing myostatin have shown an increased muscle mass, suggesting that myostatin is responsible for down-regulating muscle growth and development. In addition, myostatin increases with age, inhibiting muscle growth and contributing to muscle atrophy in the elderly.

The biotherapeutic division of MYOS is actively evaluating the role of myostatin in different muscle-related conditions including cachexia, sarcopenia, and degenerative muscle disease. Research is focused on developing strategies and therapeutic interventions to address muscle related conditions.

- Sarcopenia is the progressive loss of muscle mass with advancing age. The loss of muscle affects all individuals regardless of ethnicity or gender although the rate and degree of muscle loss varies between individuals and is affected by many factors. Those individuals who have lost significant amounts of muscle mass and strength often require assistance for accomplishing activities of daily living, which has a significant economic burden on a nation's healthcare system and impacts the overall economy. In addition to the many direct costs, sarcopenia adversely affects the overall quality of life.

- Cachexia is a syndrome that occurs in many diseases such as cancer, chronic heart failure, chronic kidney failure, and AIDS. It is characterized by a loss of body weight as a consequence of pathological changes in different metabolic pathways, with the loss of muscle mass as the core component of the syndrome.

- Degenerative muscle diseases, such as muscular dystrophy and muscle dysfunction that occurs as a consequence of denervation such as seen in amyotrophic lateral sclerosis (ALS), are conditions marked by the progressive deterioration of muscle tissue that results in weakness and impairs normal function. These diseases are typified by difficulty with walking, balance, and coordination with many affecting speech, swallowing, and breathing. There are currently no cures for degenerative muscle diseases outside of palliative care.

MYOS is a very early stage company; however, there are revenues to support pre-clinical and basic research studies through the sale of MYO-T12. Sales of MYO-X, the brand name for MYO-T12, increased 264% last year to $3.3 million. With the addition of a new age management market, sales in the first quarter 2014 came in at $1.5 million. The company reported $3.9 million in cash as of Mar. 31, 2014.

Our model indicates that MYOS is currently worth approximately $20.00 per share based on forecasted sales of two products and a price to sales target comparable to other clinically driven bionutrition companies. As all of the disease specific applications are pre-clinical, we do not include them in our valuation, however we do believe that the discovery program aimed at developing therapeutics targeting regulatory pathways involved in muscle health and performance differentiates MYOS from other bionutrition companies, thus making the stock a compelling option for those interested in the emerging area of muscle health.

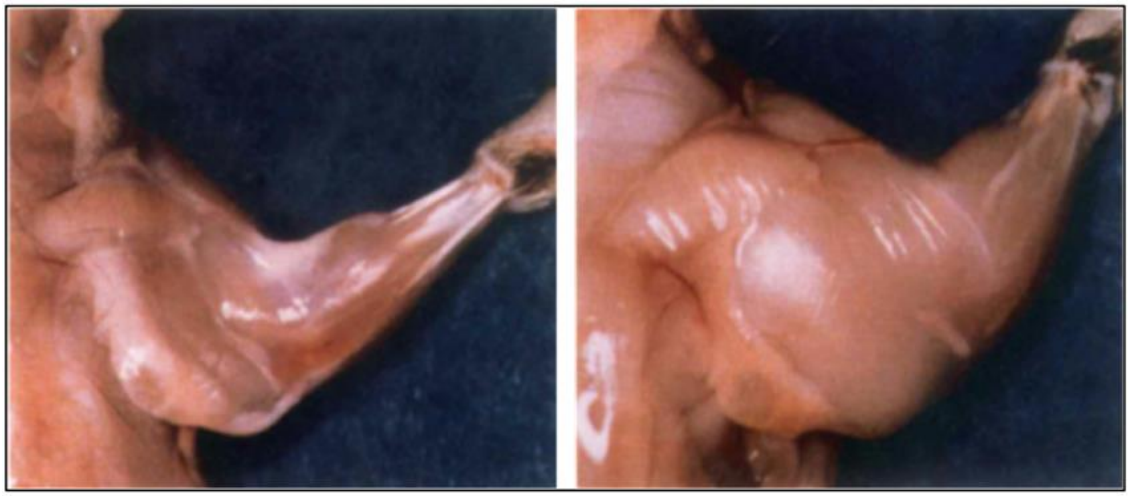
INVESTMENT THESIS

Myostatin

MYOS Corporation is developing nutritional and therapeutic products aimed at maintaining and improving the health and performance of muscle tissue. One current target of the research being conducted by MYOS is the inhibition of myostatin, a powerful negative muscle regulatory protein.

...Background on Myostatin…

A 1997 article in the journal Nature first described the discovery of a novel member of the transforming growth factor-β (TGF-β) superfamily of growth and differentiation factors. This factor was expressed specifically in adult skeletal muscle and referred to as growth/differentiation factor-8 (GDF-8) (McPherron *et al*., 1997). The researchers created "knockout" mice, whereby they disrupted the expression of GDF-8 throughout the organism, with the resulting mice showing a large and widespread increase in skeletal muscle mass. Individual muscles of mutant animals weighted 2-3 times more than those of wild-type animals, with the increase a result of both muscle cell hypertrophy and hyperplasia. The newly created mice were subsequently named "mighty mice". Based on the phenotype, the researchers dubbed the newly discovered protein myostatin.

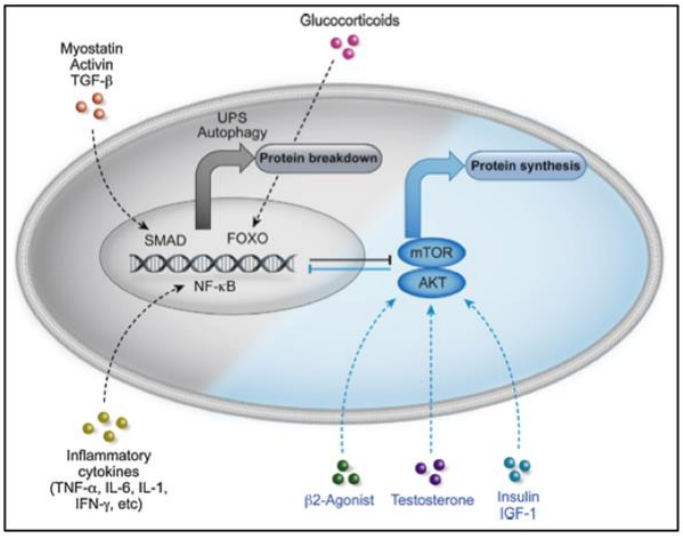


Upper limb muscle mass in myostatin wild-type (left) and myostatin knock-out (right).
Source: McPherron *et al*., 1997

This work suggests myostatin exerts an effect on both muscle hypertrophy and hyperplasia, as myostatin knock-out "mighty mice" were shown to have an increase in both the number of muscle fibers and in fiber sizes. Hypertrophy refers to the enlargement of a tissue or organ due to the enlargement of its component cells. In contrast, hyperplasia refers to an increase in the number of cells or a proliferation of cells. Both of these processes can lead to enlargement of an organ.

Skeletal muscle is the primary producer of myostatin, where it is secreted into the blood stream and acts as a negative regulator of muscle differentiation and growth. The protein begins as a 375 amino acid dimer that is cleaved by proteases to a 109 amino acid active domain. The active form of the protein binds to activin type II receptors, ActRIIA and ActRIIB (Lee *et al*., 2001). Binding to the receptors initiates a signaling cascade that results in an increase in protein breakdown and subsequent inhibition of protein synthesis.

The following figure shows how muscle mass is dynamically regulated by a host of extracellular signals that activate distinct intracellular signaling processes that alter protein balance in the muscle fibers. Insulin, IGF-1, testosterone, and β2-adrenergic agonist have anabolic actions on skeletal muscle, that is they induce protein synthesis and inhibit proteolysis leading to muscle hypertrophy. In contrast, catabolic signals such as glucocorticoids, a number of pro-inflammatory cytokines, and myostatin induce protein breakdown and muscle fiber atrophy.



Signaling pathways regulating muscle protein balance.
Source: Han *et al*., 2013

Myostatin has also been shown to negatively regulate satellite cell activation and self-renewal. Satellite cells are located between the sarcolemma and the basement membrane of individual muscle fibers and function as a stem cell population in muscles (Shultz *et al*., 1994). Muscle fiber growth causes satellite cells to re-enter the cell cycle, proliferate, and fuse with existing muscle fibers. *In vitro* studies using isolated satellite cells have shown myostatin to inhibit proliferation of these cells.

The ultimate role of myostatin and other muscle cell regulators is to balance muscle mass and skeletal integrity, which is important to prevent muscle growth from exceeding the structural integrity of bone. In evolutionary terms, myostatin was most likely selected for to prevent muscle fibers from continually building, which could lead to muscles exerting forces so strong they could damage the bones to which they are attached. There is also a potential Darwinian role for myostatin that vestigial control of muscle gains is needed to prevent an organism from being overly-muscled and thus fall more easily to predation because they were too bulky to speed away or because they just looked more delicious to predators. There are examples of naturally occurring myostatin mutations in otherwise healthy organisms that provide additional evidence for the role of myostatin in preventing muscle growth.

…Examples of Naturally Occurring Myostatin Mutations…

Since the initial discovery of myostatin, several naturally occurring "double-muscling" phenotypes have been reported in different cattle breeds, dogs, and even humans. An 11 base pair deletion in the bovine myostatin gene was shown to be the cause of a double-muscled phenotype in the Belgian Blue cattle breed (Grobet *et al*., 1997). These cattle are particularly intriguing to the agricultural industry due to the significant increase in meat obtainable from these animals, although the special care and feeding requirements of these animals has precluded their widespread adoption.



Double-muscled Belgian blue cow.
Source: www.occupyforanimals.com

A 2007 article identified a two base pair deletion in the myostatin gene in the whippet dog breed that results in a double-muscled phenotype (Mosher *et al*., 2007). In addition to the large effect on muscle size, researchers were also able to show that the increase in muscle led to a significant increase in speed in competitive racing events, marking the first time that a mutation in myostatin had been quantitatively linked to increased athletic performance.



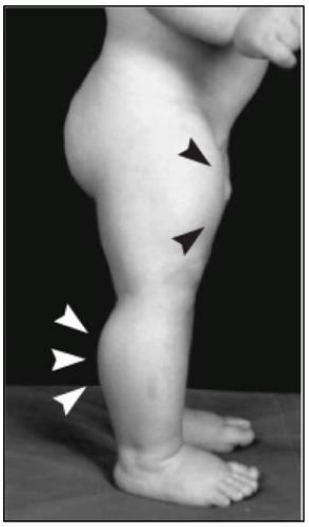
Bully whippet homozygous for wild-type myostatin



Bully whippet homozygous for myostatin mutation

Source: Mosher *et al*., 2007

A 2004 case report in the New England Journal of Medicine discussed a male infant with extraordinary muscularity at birth that included protruding muscles in his thighs, calves, and upper arms (Schuelke *et al*., 2004). The photograph to the right shows the child at seven months of age, with the arrowheads pointing to protruding muscles in the thigh and calf.



Child at 7 months of age

Source: Schuelke *et al*. 2004

At the time of the report, the child was 4.5 years of age, had normal motor and mental development, and was in good health. The child continued to have increased muscle bulk and strength, and was able to hold two 3-kg dumbbells in horizontal suspension with his arms extended. Upon further characterization it was found that the child's quadriceps muscle was 7.2 standard deviations (SD) above the mean value for 10 age- and sex-matched controls (6.72 cm^2 vs. 3.13±0.49 cm^2). In addition, the thickness of his subcutaneous fat pad was 2.88 SD below the mean value for controls (0.18 cm vs. 0.36±0.06 cm), with no difference seen in the diameter of the femur (0.57 cm vs. 0.50±0.13 cm.).

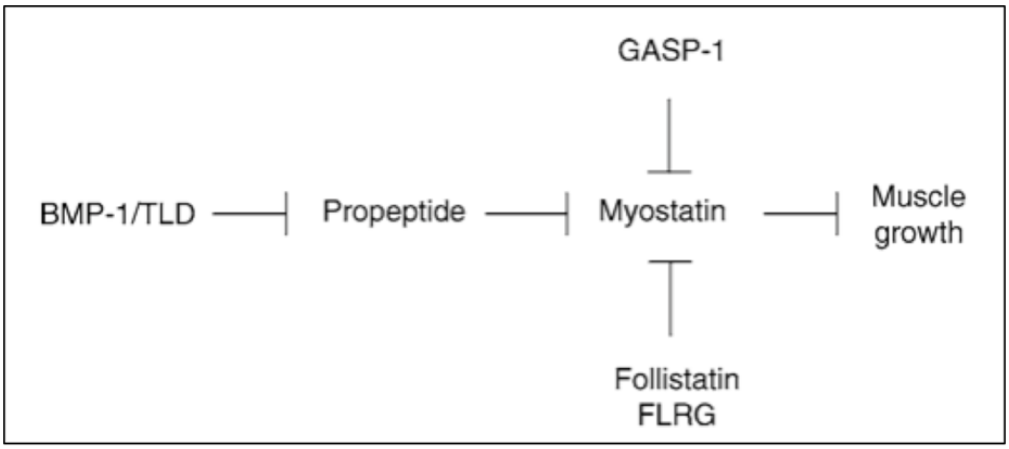
Molecular characterization identified a mutation in the non-coding portion of the myostatin gene that altered splicing of the myostatin messenger RNA and resulted in a non-functional form of the protein, with full-length myostatin not detectable in the patient's serum. We are unaware of any recent follow-up reports in regards to this patient; however, there are reports of additional children with myostatin mutations that exhibit similar phenotypes.

…Natural Myostatin Inhibitors…

There are a number of proteins that have been identified that regulate myostatin activity. As mentioned earlier, myostatin begins as an inactive precursor and is then cleaved by proteases to produce the active form. Proteolytic cleavage of myostatin produces an N-terminal propeptide and the biologically active C-terminal domain. The propeptide has been shown to play an important role in regulating the activity of the active domain. *In vitro* studies show that the propeptide is capable of blocking receptor binding (Thies *et al*., 2001), and mice overexpressing the propeptide in muscle have a similar increase in muscle mass compared to myostatin deficient mice (Yang *et al*., 2001). Analysis of myostatin isolated from human blood has shown it to be complexed with the propeptide *in vivo* (Hill *et al*., 2002). Cleavage of the propeptide appears to be one mechanism for how myostatin is activated (Wolfman *et al*., 2003).

In addition to the propeptide, several other proteins have been shown to be capable of binding and inhibiting the activity of myostatin. One of these proteins is follistatin, a secreted glycoprotein that has been shown to bind to a number of proteins in the TGF-β family. Follistatin is expressed in skeletal muscle like myostatin. Over-expression of follistatin in mice results in muscle enlargement, while follistatin knockout mice display muscle deficiency (Lee *et al*., 2001; Matzuk *et al*., 1995). In addition, follistatin has been shown to block myostatin activity in receptor binding assays.

In addition to propeptide and follistatin, two other proteins that appear to be involved in the regulation of myostatin activity are FLRG and GASP-1. Both of these proteins are complexed with myostatin in the blood of humans, and *in vitro* studies have shown that both proteins can bind to myostatin with high affinity and inhibit its activity. Outside of regulating myostatin activity, no other activities have been assigned to these proteins.



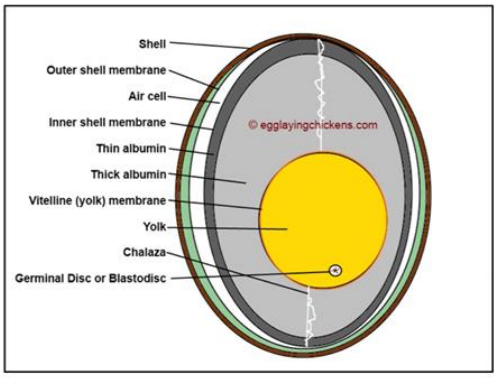
Myostatin is inhibited by a number of extracellular binding proteins.
Source: Lee *et al.*, 2004

With so many proteins capable of binding myostatin, the mechanism underlying myostatin activation is highly complex and is still being elucidated. It seems likely that the propeptide becomes bound to myostatin during the biosynthesis of the active form of the protein following its release into the circulation. Activation presumably requires dissociation from the propeptide with subsequent degradation of the propeptide to prevent re-association. The roles of FLRG and GASP-1 are still being investigated and a number of theories exist in regards to their role in regulating myostatin activity.

One theory suggests that myostatin, the propeptide, FLRG, and GASP-1 exist in a multimeric complex with each of the proteins having its own role in regulating myostatin activity. Another hypothesis is that the binding of the propeptide, FLRG, and GASP-1 may be mutually exclusive and thus the circulating form of myostatin may be a heterogeneous mixture of the various complexes. A third possibility is that FLRG and/or GASP-1 may be involved in terminating myostatin signaling after myostatin has already bound and activated its receptors.

…Discovery of a natural source of follistatin…

In 2005, Dr. Carlon Colker, MD, the former Chief Medical Officer of MYOS, discovered that standard store-bought fertilized eggs, but not unfertilized eggs, have a naturally high abundance of follistatin. The follistatin is mostly contained to the vitelline (yolk) membrane, and is released into the yolk to promote early embryo development and growth, with its expression terminated after a few days. This discovery was presented at the 2006 Annual Meeting of the American College of Nutrition (Colker, 2006).



Inside of a chicken egg.
Source: egglayingchickens.com

Dr. Colker then set out to produce a nutritional supplement by concentrating the follistatin from a large number of eggs, while preserving the bioactivity and bioavailability of follistatin. He began working with a company called Celldyne Biopharma, LLC to create a desiccated fertilized egg powder that maintained full follistatin activity. In another presentation at the 2006 Annual Meeting of the American College of Nutrition, Dr. Colker showed that this new product, Folstaxan™, contained biologically active follistatin that was capable of down-regulating serum myostatin levels (Colker-2, 2006). The study, which was conducted in only one test subject, showed a baseline serum myostatin level of 45 pg/mL and follistatin level of 0 pg/mL. Twelve hours after ingesting Folstaxan™ the subject's serum myostatin level decreased to 34 pg/mL and their follistatin level increased to 57 pg/mL. While preliminary, these results seem to indicate that 1) ingestion of this first generation product resulted in follistatin entering the bloodstream and 2) the follistatin appeared to be active and capable of down-regulating myostatin.

With these initial results, a second-generation product was produced that was less bulky and more refined. Using this product, a second study was performed on seven male subjects whereby they ingested a single 20-gram serving of Folstaxan™, with their myostatin levels followed for 24 hours. The subjects had an average baseline myostatin level of 27 pg/mL, at 12 hours the average serum myostatin level decreased to 24.4 pg/mL, and at 24 hours the myostatin level continued to decline to 23.9 pg/mL. Two of the seven subjects were "non-responders", meaning that their myostatin levels did not decrease. These results showed that it was possible to make a fertile egg yolk powder with active follistatin that decreases serum myostatin levels.

After successfully showing that Folstaxan™ was capable of down-regulating myostatin levels, the product was still not ready for commercial use yet as there were a number of issues to work out (taste, solubility, etc.). During this time, according to Dr. Colker, Celldyne filed a patent on an avian follistatin product without listing Dr. Colker as an inventor, created a Folstaxan™ website, and began marketing and selling the product without any consent from Dr. Colker. This product was vastly inferior to even the research grade product that Dr. Colker had utilized in his previous studies, thus resulting in a negative public profile for the product and Dr. Colker. With no other recourse, Dr. Colker sued Celldyne (Peak Wellness, Inc et al v. Green et al., Connecticut District Court), and at the preliminary hearing the judge granted an injunction against Celldyne, forcing them to shut down the website and to stop selling Folstaxan™. The case was eventually settled with Dr. Colker re-taking control of the intellectual property associated with avian derived follistatin products.

…A New and Improved Formulation…

A new and improved follistatin-rich product was formulated based on a proprietary high-grade handling process to improve all characteristics of the product, including taste, texture, and activity. This new product was tested in 10 healthy adult males, who each received one 10-gram bolus, with their serum myostatin levels again tracked for 24 hours. Results of this study were presented at the 2009 Annual Meeting of the American College of Nutrition. Average initial myostatin levels were 27.5 pg/mL, very similar to the study performed with Folstaxan™.

At the 12-hour time point, serum myostatin levels had decreased to an average of 12.6 pg/mL – a 46% decrease. In addition, all 10 subjects responded with a decrease in their myostatin level. Between 24-30 hours, the average serum myostatin level had normalized to an average value of 28.1 pg/mL. To underscore the dramatic drop in myostatin levels at 12 hours, the product was given the name MYO-T12.

...MYO-X…



MYO-T12 is the initial core product for MYOS Corp. The product is sold under the trade name **MYO-X** through a distribution agreement entered into on May 15, 2012 with Maximum Human Performance (MHP), a company engaged in the development, marketing, and distribution of nutritional and other supplemental products for consumer use. The distribution agreement with MHP allows the company to continue conducting clinical trials with MYO-T12 in support of marketing claims as well as to enhance the company's intellectual property, to develop product improvements and new products, and to reduce the cost of manufacturing.

MYO-X is currently available at specialty retail stores such as GNC and Vitamin Shoppe as well as websites such as bodybuilding.com and MHPStrong.com.

As part of the background research into MYOS Corporation, we purchased a bottle of MYO-X from Vitamin Shoppe in May 2014. The product is supplied in a 300-gram container, which is enough to last one month based on the recommended dosage of one 10-gram serving once per day. The product has a pleasant odor with a consistency best described as that of large breadcrumbs. The package recommends either mixing a serving into a cold liquid of choice or to eat it plain. For our test, we chose to consume each 10-gram serving plain and were pleasantly surprised that it was easily ingested. The product has vanilla flavoring with a slight "eggy" taste to it, but has a nice overall taste. We did not experience any type of gastrointestinal symptoms or any other type of adverse events based on ingestion of MYO-X. The analyst who consumed MYO-X felt that compared to other supplements on the market (protein shakes, creatine, etc.), MYO-X was the easiest to take, had the best taste, and had the least amount of unwanted side effects.

…Current Research Initiatives with MYO-T12…

As an early development-stage bionutritional and biotherapeutic company, MYOS is currently dedicated to basic and clinical research to support their current and potential future product portfolio. In support of this, the company recently constructed a state-of-the-art protein chemistry and proteomics laboratory. Research currently being conducted is focused on evaluating the properties of MYO-T12, and in November 2013 scientists from MYOS presented a poster at the 7th International Conference of the Society of Sarcopenia, Cachexia, and Wasting Disorders (SCWD) entitled "The Characterization of the Fertilized Chicken Egg Yolk Proteome and Lipidome by Accurate Mass, High Resolution Mass Spectrometry: Therapeutic Implications for the Treatment of Sarcopenia and Cachexia".

In addition, the company is actively building a small molecule and biologics discovery program aimed at myostatin and the different pathways that act upon muscle development. MYOS recently announced an agreement with privately held Cloud Pharmaceuticals, Inc. to discover product candidates related to the inhibition of targets in the myostatin regulatory pathway as well as inflammatory mediators associated with sarcopenia and cachexia. Cloud Pharmaceuticals utilizes cloud computing technology to initiate and design small molecule drug candidates based on their proprietary cheminformatics tool Inverse Design (Wang *et al*., 2006; Keinan *et al*., 2007; Keinan *et al*., 2008).

MYOS is targeting both furin, a convertase that plays a central role in the processing of myostatin to its biologically active form, and JAK3, a tyrosine kinase thought to be a factor in pathways central to inflammation. Candidate molecules selected from the partnership with Cloud Pharmaceuticals will be the company's first small molecules under development as treatments for sarcopenia and cachexia.

In addition to the discovery programs, MYOS is currently conducting a number of clinical studies. One such study in collaboration with the University of Tampa is designed to study the effects of MYO-T12 on skeletal muscle, growth, strength, and power. This is a double blind, placebo-controlled 12-week study involving 45 males who typically workout a few times a week (i.e., they are not bodybuilders). The subjects receive daily milkshake's containing placebo (n=15), a single serving of MYO-T12 (n=15), or a triple serving of MYO-T12 (n=15) along with supervised weight training sessions two times a week. The primary outcome for the study is change in lean body mass as assessed by whole body dual x-ray absorptiometry (DEXA) along with change in muscle size as determined through ultrasound imaging. Secondary outcomes include measurements of power and strength. In addition, blood work will be performed to examine inflammatory markers as well as myostatin level. The results from the study should be released during the third quarter of 2014.

Additional research initiatives currently being conducted include the following:

- In July 2013, the company entered into a pre-clinical study agreement with The Brigham and Woman's Hospital, Inc. to conduct a study on the effects of MYO-T12 alone or in combination with testosterone on skeletal muscle and fat mass in a rodent model. Results are expected in the fourth quarter of 2014.
- In September 2013, the company entered into a clinical study agreement with Hackensack University Medical Center to conduct a clinical study to determine the effects of MYO-T12 on blood chemistries and body mass index in a group of healthy adult women.
- In October 2013, the company entered into an agreement with Deutsches Institut fur Lebensmitteltechnik e.V. - the German Institute for Food Technologies (DIL) for the development of a liquid dietary supplement formulation based on MYO-T12. The study is expected to be completed in the fourth quarter of 2014.
- In June 2014, the company entered into a three year Master Service Agreement with Rutgers University with the initial project focused on the development of cell-based assays for high throughput screening studies of next

generation myostatin inhibitors such as proteins, peptides, and small molecules. The study is expected to be completed by the middle of 2015.

Potential Therapeutic Uses for Myostatin Inhibition

There is a wealth of pre-clinical data generated from numerous research groups on myostatin and the diseases that its modulation could potentially affect. MYOS is focused on three general areas: cachexia, sarcopenia, and degenerative muscle diseases, where targeting myostatin may turn out to be a promising therapeutic intervention.

Cachexia

Cachexia is the progressive loss of fat and muscle despite adequate nutrition. It is a common complication associated with cancer and leads to a poor quality of life and increased mortality. As skeletal muscle is diminished there is a reduced ability to move, a loss of strength, and an increase in conditions associated with immobility such as thrombosis, pneumonia, and respiratory failure. Cachexia is ultimately responsible for the deaths of 22% of cancer patients (Argiles *et al*., 2013).

Depending on the type of cancer, weight loss occurs in 30-80% of cancer patients and is severe (defined as a loss of >10% of initial body weight) in 15% of patients (DeWys, 1986). Weight loss is an important prognosticator in cancer therapy with the greater the weight loss than the shorter the survival time. A study of 109 disease-free breast cancer patients in stage II node positive and stage III disease showed unexplained body weight loss in 84% of patients developing recurrence, compared with 10% of patients remaining disease free (Marinoho *et al*., 2001).

Weight loss in cancer patients due to cachexia arises from the loss of both adipose tissue and skeletal muscle. Cachexia is further distinguished from simple starvation by the preservation of non-muscle protein (Fearon, 1992). The following table is a comparison between a group of lung cancer patients and healthy controls matched for age, sex, height, and pre-illness stable weight of the cancer patients (Fearon, 1992).

Parameter	Normal, kg	Cachectic, kg
Total body weight	65.6	44.9
Total fat	17.3	3.1
Muscle protein	2.8	0.7
Non-muscle protein	8.3	8.1
Intracellular water	19.1	12.9
Extracellular water	15.1	17.5
Minerals	3.0	2.6

Overall, the mean weight loss for the cancer patients was 32%, however this included an 85% loss of their total body fat and 75% of their skeletal muscle. The loss of skeletal muscle is indicative of why cachexia leads to an increase in mortality, since loss of respiratory muscle function can lead to death from hypostatic pneumonia (Windsor *et al*., 1988).

The most effective means for treating cancer-induced cachexia is to cure the cancer. Unfortunately, this remains an infrequent achievement in adults with solid tumors. A second option is to increase the nutritional intake, however since a lack of eating is only a small part of the problem, with cachexia representing an alteration in metabolism, this does not typically reverse cachexia completely.

Pharmaceutical treatment options are limited and include the following:

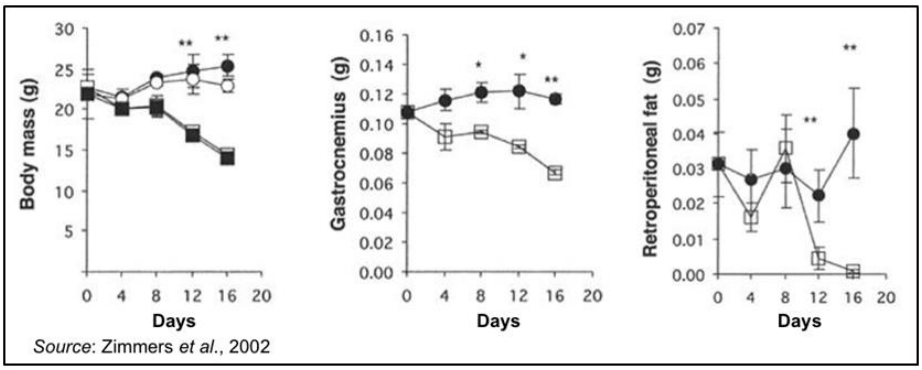
- Progestagens: Medroxyprogesterone acetate (MPA) and megestrol aceate (MA) are currently considered the best available treatment option for cachexia, and they are approved in Europe for treatment of cancer- and AIDS-related cachexia. While clinical trials have shown progestagens to be effective in increasing body mass (mainly as water and fat mass), they have not proved to be effective in increasing lean body mass, which is a critical target in the treatment of cancer cachexia.

- Corticosteroids: In randomized controlled studies, corticosteroids have been shown to improve appetite and quality of life compared with placebo. While progestagens and corticosteroids seem equally effective, corticosteroids have more side-effects associated with long term use including protein breakdown, insulin

resistance, water retention, and adrenal suppression, thus rendering their use only as a short-term treatment option.

…Myostatin and Cachexia…

Myostatin has been shown to have a direct result on cachexia. In a 2002 study, tumors were induced in mice with cells that either did or did not secrete myostatin (Zimmers *et al*., 2002). The following graphs show that the presence of myostatin-secreting tumors (open squares) resulted in the mice losing an average of 33% of their total body weight, while mice bearing control tumors (no secretion of myostatin; closed circles) had either maintained or gained weight. Part of the weight loss was due to a global decline in skeletal muscle mass, as examination of individual muscles (gastrocnemius) revealed declines of 35-50%. In addition, there was a near total loss of white adipose tissue (retroperitoneal fat).



Source: Zimmers *et al*., 2002

The authors cited four reasons why they believed that myostatin was responsible for the presence of cachexia in the mice.

1) Mice injected with 10 other similar types of tumor cells that did not secrete myostatin all had tumor growth with no weight loss.
2) The secreted myostatin from the tumor cell was under the control of a zinc-inducible promoter, and when the mice were provided water containing zinc sulfate their weight loss was accelerated, presumably due to an increase in the expression on myostatin from the tumor cells.
3) There was increased serum myostatin activity associated with increased weight loss.
4) Injections of small amounts of myostatin in non-tumor bearing animals resulted in a significant reduction in white fat, similar to what was seen in mice with myostatin-secreting tumors.

Lastly, the researchers determined whether they could control myostatin activity through administration of tumor cells secreting follistatin alongside the myostatin-secreting cells. Mice were injected with myostatin-secreting tumor cells 10 days following administration of follistatin-secreting tumor cells. The data shows that the presence of follistatin-secreting cells significantly slowed myostatin-induced weight loss, thus showing that targeting myostatin activity could be a novel mechanism for treatment of cachexia.

Additional studies examining the effect of myostatin inhibition in mouse models of cancer cachexia have shown similar results.

- ✓ Inhibiting myostatin activity with an anti-myostatin antibody prevented loss of muscle mass and function in lung tumor bearing mice (Benny Klimek *et al*., 2010).
- ✓ Use of a soluble actRIIB protein caused reversal of loss of muscle mass and strength and prolonged survival in multiple tumor models (Zhou *et al*., 2010).
- ✓ Myostatin inhibition using actRIIB antagonism resulted in the improvement of muscle wasting and physical performance in a mouse model of lung cancer (Busquets *et al*., 2012).

These results indicate that inhibiting myostatin activity represents a novel approach to treating cancer-induced cachexia.

Sarcopenia

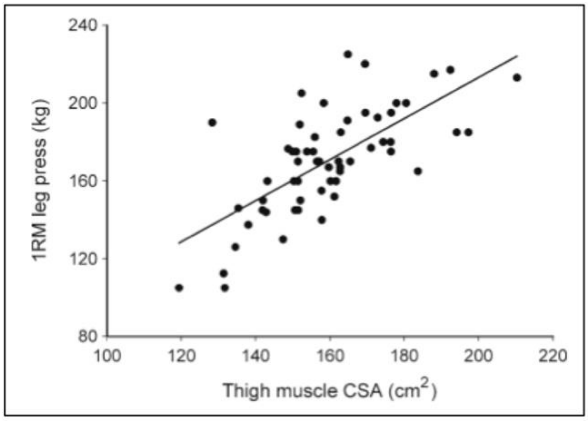
Sarcopenia is the progressive age-related loss of muscle mass and associated muscle weakness that results in a frail elderly population that is more susceptible to serious injury from sudden falls and fractures, potentially leading to a loss of functional independence. The causes of sarcopenia are multifactorial and can include disease, changing endocrine function, chronic diseases, inflammation, insulin resistance, and nutritional deficiencies (Evans, 2010). While cachexia may be a component of sarcopenia, the two conditions are not the same. Diagnosis of sarcopenia is considered in older patients who present with obvious declines in physical function, strength, or overall health, with a precise diagnosis being consistent in patients with a gait speed of <1 m/s and an appendicular lean/fat ratio <2 standard deviations of the average of a young adult (Evans, 2010).

The disease is a significant public health problem, particularly in the developed world. In 2000, it was estimated that healthcare costs in the U.S. associated with sarcopenia were $18.5 billion, or approximately 1.5% of total healthcare expenditures (Janssen *et al*., 2004). The elderly population in the U.S. is expected to increase to approximately 70 million individuals by 2030, and since sarcopenia affects all elderly regardless of ethnicity, gender, or wealth, this will continue to put an added strain on the healthcare system.

The molecular mechanisms underlying sarcopenia appear to be the result of the progressive atrophy and loss of individual muscle fibers associated with some loss of motor units, and a reduction in muscle "quality" due to the infiltration of fat other non-contractile tissues. Age-related changes in skeletal muscle are associated with a complex interaction of factors affecting neuromuscular transmission, protein synthesis and degradation, muscle architecture, fiber composition, increased generation of reactive oxygen species, myonuclear apoptosis, altered excitation-contraction coupling, and metabolism (Lynch *et al*., 2008; Ryall *et al*., 2008; Arnold *et al*., 2010; Wenz *et al*., 2009).

As individuals age, the gradual loss of muscle is typically exacerbated by normal decreases in physical activity with advancing age and also by metabolic changes and oxidative stresses that can result in the accumulation of cellular damage from free radicals (Meng *et al*., 2010). Even former professional athletes and otherwise healthy older adults exhibit a progressive loss of muscle mass, strength, and power output (Runge *et al*., 2004). The loss of muscle mass is most notable in the lower limbs, with the cross-sectional area of the *vastus lateralis* (largest part of the quadriceps) being reduced by as much as 40% between the age of 20 and 80 years (Lexell, 1995).

The following graph shows the correlation between thigh muscle thickness and leg press strength.



Correlation between thigh muscle size and strength.
Source: Koopman *et al*., 2011

Since muscle strength is proportional to muscle mass and cross-sectional area, the loss of skeletal muscle mass is always accompanied by a subsequent decrease in muscle strength, a decline in functional capacity, and a reduction in skeletal muscle oxidative capacity.

There is substantial evidence to indicate that older adults who are less physically active are more likely to have lower skeletal muscle mass and strength with an increased risk of developing sarcopenia (Rolland *et al*., 2008). Thus, exercise appears to be an effective intervention for the prevention and treatment of sarcopenia, with most studies indicating that resistance training, as opposed to aerobic activity, has a larger effect on muscle mass and strength (Sipila *et al*., 1995).

In addition to strength training, maintaining proper nutrition is another approach that has been attempted for preventing sarcopenia. Most older adults do not consume sufficient quantities of dietary protein, which can lead to a reduction in lean body mass. The current recommended daily allowance (RDA) of protein is 0.8 g/kg/day, and almost 40% of people >70 years of age do not meet this RDA (Houston *et al*., 2008). Interestingly, when daily protein intake is standardized at the RDA, exercise induced increases in muscle mass become apparent and a further increase in protein intake does not seem to have any beneficial effect (Iglay *et al*., 2007). In fact, additional protein supplementation may reduce subsequent voluntary food consumption in the elderly (Fiatarone Singh *et al*., 2000), thus protein supplementation above the RDA is not recommended.

Testosterone replacement therapy in men is another approach that has been tested with mixed results. A significant number of older men (20% of those >60 years and 50% of those >80 years) are hypogonadal, meaning they have a total testosterone concentration that is at least 2 standard deviations below the mean for healthy young men (Harman *et al*., 2008). Testosterone decreases gradually at approximately 1% per year from the age of 30 years, with this reduction associated with loss of muscle strength, muscle mass, and a reduction in bone mineral density (Srinivas-Shankar *et al*., 2010). However, the evidence to support testosterone supplementation is highly variable, with some studies showing an increase in lean body mass and hand grip strength (Gruenewald *et al*., 2003), with others showing no increase in muscle strength but an improvement in lean body mass (Emmelot-Vonk *et al*., 2008). Thus, testosterone supplementation is typically not utilized for sarcopenia treatment.

…Myostatin and Sarcopenia…

Myostatin protein levels have been shown to change with age in humans. Numerous studies have indicated there is a significant increase in both myostatin messenger RNA and/or protein levels during aging in humans and rodents (Baumann *et al*., 2003; Leger *et al*., 2008; Raue *et al*., 2006). However, other studies have been unable to corroborate these findings, suggesting that there may not be a difference in myostatin expression between young and old individuals (Welle *et al*., 2002; Ratkevicius *et al*., 2011).

One possible explanation for the discrepancies in these studies could be due to the complexities associated with myostatin and its activity. Myostatin abundance may not be indicative of myostatin activity, as the mature form of the protein is found complexed to numerous regulatory proteins, thus methods to quantify myostatin may not discriminate between the active and latent form. Whether the proportion of active to latent myostatin changes with age is yet to be determined.

While the role that myostatin plays in aging are poorly understood, several of the protein's characteristics make it a unique and desirable therapeutic target for sarcopenia:

1) It has been shown that postnatal inhibition of myostatin unequivocally increases skeletal muscle mass in adult and older mice (Whittemore *et al*., 2003). Aged animals administered a myostatin neutralizing antibody for four weeks saw increases in muscle mass by up to 17% with subsequent improvement in muscle performance.
2) The effects of myostatin inhibition are muscle specific. Even with large increases in muscle mass in animals containing myostatin mutations, there does not appear to be an increase in the masses of other organs or in the prevalence of cancer. In addition, aged myostatin deficient mice exhibit increased bone mineral density, suggesting that disrupting myostatin signaling could have other positive influences on age-related changes in other tissues (Morissette *et al*., 2009).
3) Myostatin is a viable target for pharmacological intervention as it is secreted into the bloodstream.
4) Partial reductions in myostatin activity promote meaningful changes in skeletal mass and function. Whippet dogs with a myostatin mutation in only one copy of the myostatin gene (resulting in ~50% overall myostatin activity) are still more muscular and run faster than the wild-type animals.

While more research is necessary to determine the exact role of myostatin in age-related muscle loss, the characteristics of myostatin render it an intriguing potential target for sarcopenia.

Degenerative Muscle Diseases

Degenerative muscle diseases, which include those illnesses known as muscular dystrophy (MD), are conditions that result in the progressive damage to muscle cells and eventually to weakness and loss of control. The muscular dystrophy's are a related group of nine diseases, each with a different cause with the conditions being inherited and genetic in nature. Other degenerative muscle diseases include amyotrophic lateral sclerosis (ALS) and myasthenia gravis (MG). There are currently no cures for degenerative muscle diseases.

Symptoms of MD are associated with the progressive weakness of the affected muscle groups, which is dictated by the underlying condition. Patients with Duchenne muscular dystrophy (DMD) have muscle degeneration, walking difficulties that typically leads to the patient being wheelchair bound by the time they are 12, breathing problems, and eventually death. Those suffering from facioscapulohumeral muscular dystrophy have problems with facial muscles, scapula, and upper arms that includes eyelid drooping, shoulder weakness, hearing loss, and loss of strength in leg muscles.

There are no cures currently available for any form of MD, with a limited number of medications that can slow the course of disease. The only drug proven to shown some benefit is prednisone, a synthetic corticosteroid. A randomized, double-blind multicenter study of 103 male patients with DMD showed that patients on prednisone have improvement in muscle strength at 1, 2, and 3 months compared to those subjects receiving placebo (Mendell *et al*., 1989). However, the benefit derived from prednisone treatment was short-lived, with muscle strength leveling off at the 3-month mark and then decreasing after that.

…Myostatin and Muscular Dystrophy…

The same characteristics that make myostatin a desirable therapeutic target in cachexia and sarcopenia are responsible for the interest in myostatin as a target for MD therapy. However, the two sets of clinical trials that have been carried out using myostatin antagonists in muscular dystrophies have yielded disappointing results.

A Phase 1/2 trial of the human anti-myostatin antibody MYO-29 was carried out in adult muscular dystrophies of the Becker, facioscapulohumeral and limb girdle type (Wagner *et al*., 2008). The double-blind, placebo-controlled, multinational, randomized study included 116 subjects divided into sequential dose-escalation cohorts each receiving MYO-29 or placebo at 1 mg/kg, 3 mg/kg, 10 mg/kg, or 30 mg/kg. The results showed that MYO-29 was safe and well tolerated, with the exception of cutaneous hypersensitivity at the 10 and 30 mg/kg doses. There were no improvements noted in exploratory end points of muscle strength or function, however the study was not powered to examine efficacy. The authors did not that there was a trend toward increased muscle size in MYO-29 treated patients, but MYO-029 is no longer being developed as a treatment for DMD.

A Phase 2 clinical trial of the recombinant ActRIIB reagent (ACE-031) was conducted in boys with Duchenne muscular dystrophy but was stopped early due to unexpected and unexplainable side effects. The trial began in November 2010 but was stopped in May 2011 due to minor nose and gum bleeding and dilation of blood vessels in the skin. While the side effects were not considered dangerous in and of themselves, the companies and regulatory agencies wanted to fully understand the events before continuing clinical studies. In May 2013, Acceleron Pharma and Shire announced that they would not be continuing development of ACE-031 as a treatment for DMD.

…Other Myostatin Inhibitors Under Development…

Given the large patient populations that could potentially benefit from treatments targeted to myostatin, a number of pharmaceutical companies are currently developing various types of myostatin inhibitors:

- LY2495655: A humanized anti-myostatin monoclonal antibody being developed by Eli Lilly for the treatment of muscle wasting associated with cancer and other disorders. Results from a phase 1 trial in healthy volunteers demonstrated the drug to be well tolerated and led to an increase in thigh muscle volume (Jameson *et al*., 2012). Interim results from a Phase 1 safety study in advanced cancer patients reported increased muscle volume with concomitant increases in hand grip strength and other functional measures. A Phase 2 trial of LY2495655 in patients with locally advanced or metastatic pancreatic cancer combined with chemotherapy is ongoing with overall survival as the primary endpoint. That study is expected to be completed in February 2015.

- BYM338: A fully human anti-ActRIIB monoclonal antibody that is being developed by Novartis for the treatment of sporadic inclusion body myositis (sIBM), chronic obstructive pulmonary patients with cachexia, sarcopenic

adults, and cancer cachexia (both lung and pancreatic). The antibody was granted breakthrough therapy designation for sIBM based on the results of a Phase 2 proof-of-concept study that showed BYM338 substantially benefited patients with sIBM compared to placebo, including an increase in thigh muscle volume, lean body mass, and quadriceps strength. The compound is also being tested in a Phase 2 trial for the treatment of cachexia in patients with stage IV nonsmall cell lung cancer or Stage III/IV adenocarcinoma of the pancreas.

- PF-06252616: An anti-myostatin monoclonal antibody being developed by Pfizer currently in Phase 1 testing in healthy volunteers, with plans to test the compound as a treatment for DMD. The drug was granted orphan drug designation by the European Medical Agency for treatment of DMD.

- REGN1033: An anti-myostatin monoclonal antibody being developed by Regeneron. The antibody has completed Phase 1 testing in healthy volunteers and is currently in a Phase 2 trial in patients with sarcopenia.

- FS344: This is a gene therapy under development by Milo Biotech that is currently in a Phase 1 trial with enrollment by invitation only. Treatment consists of the follistatin gene being delivered directly into the thigh muscle by the adeno-associated virus in patients suffering from Becker muscular dystrophy or sIBM. One hundred and eighty days after gene delivery a biopsy will be performed to determine if the muscle fibers are increasing in size. The study is expected to be completed in December 2016.

VALUATION AND RECOMMENDATION

We are initiating coverage of MYOS Corporation with a Buy rating and a price target of $20. MYOS is a bionutrition and biotherapeutic company focused on the development and characterization of regulatory pathways aimed at maintaining or improving the health and performance of muscle tissue. While research and development focuses on small molecules and pathways affecting muscle, the company's bionutritional arm is focusing on nutritional supplements as well as functional and medical foods for consumers and hospitals. The company currently has two marketed products; MYO-X, powered by MYO-T12, which is distributed by Maximum Human Performance (MHP) to specialty retail and other outlets and a second private label product in the age management market.

MYO-T12 is a dietary supplement that has been shown in two clinical studies to temporarily decrease the levels of circulating myostatin. Myostatin is a protein produced by most vertebrate organisms including humans, and research on animals and human with deficient myostatin have shown it to down-regulate muscle production. In addition, myostatin increases with age, thereby inhibiting muscle growth and contributing to muscle atrophy in the elderly.

…More than just OTC products…

We believe MYOS has differentiated itself from other bionutrition companies that market over the counter nutritional products based on the solid scientific basis underlying the development of the currently marketed products as well as the current research initiatives underway targeting muscle health. We believe the science behind myostatin inhibition and muscle development has been validated through a number of pre-clinical and clinical studies. However, in order to move the company forward it will be necessary to convince the scientific community at large of the importance of focusing on muscle health and the optimal way to treat muscle disorders. In support of this, MYOS recently appointed Dr. J. Craig Venter to the Board of Directors. Dr. Venter is a world-renowned scientist best known for sequencing the first draft human genome, the first complete human genome, and construction of the first synthetic cell. Having an individual with the stature of Dr. Venter on the Board of Directors further validates the science and approach that MYOS is taking to target muscle related conditions.

A useful comparison to show the potential for MYOS is to compare it to other companies that have successfully sold nutritional products with a solid scientific rationale. Abbott Laboratories (NYSE: ABT) markets the Ensure® line of adult nutritional products aimed at a number of specific nutritional goals. Ensure® products are one of a number of adult nutritional products that generated approximately $1.4 billion in 2013 U.S. sales for Abbott. The Ensure® line is backed by a number of clinical studies showing how certain components included in the drinks can affect such conditions as sarcopenia and cachexia. This is particularly true for the Ensure® Muscle Health drink, which contains

β-hydroxy-β-methylbutyrate, a compound that has been shown to reduce muscle protein breakdown (Nissen *et al*., 1996) and could potentially help reverse cachexia (May *et al*., 2002).

This is quite comparable to the work being done by MYOS and the studies underway with MYO-T12, thus the Ensure® line is an interesting case study of what is possible for an adult nutritional product that is based on a solid scientific basis. In addition, it helps to show how products with strong scientific rationale's supporting their use are capable of differentiating themselves in a crowded marketplace and attaining robust revenues.

…Upcoming catalysts to drive the stock forward…

MYOS has a number of potential catalysts being reported in the near future involving development of MYO-T12, including:

- The "Tampa Study": a double-blind, placebo-controlled study examining the effects of MYO-T12 on skeletal muscle development in 45 male subjects being conducted at the University of Tampa. With quantitative measurements assessing the change in lean body mass and muscle size, this study should leave little doubt about whether MYO-T12 is an effective supplement for augmenting muscle growth. The results from this study are due to be reported in the third quarter of 2014.

- A liquid formulation of MYO-T12 is currently under development in collaboration with the German Institute for Food Technologies with results expected in the fourth quarter of 2014.

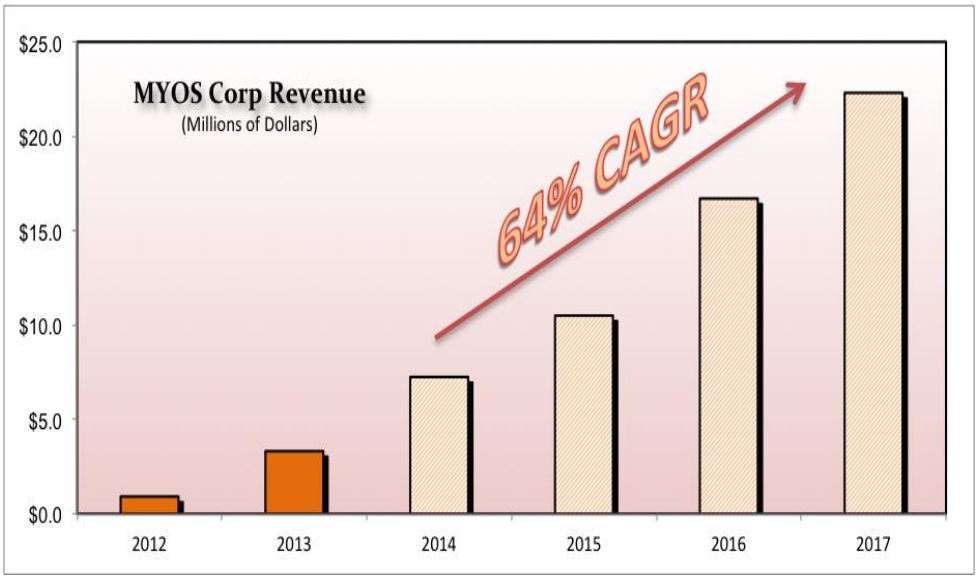
The "Tampa Study" will help shed more light on how effective MYO-T12 is on skeletal muscle growth, with positive results likely to end up being touted in a number of exercise/bodybuilding media outlets that could help drive sales of MYO-X higher.

We believe that a liquid formulation based on MYO-T12 is something that the company could sell in a hospital setting, similar to Ensure® shakes. With a recent study showing oral supplemental nutrition in hospitalized patients improving readmission rates in the elderly (Lakdawalla *et al*., 2013), there is likely to be an increased demand for supplemental nutrition in the hospital setting, and could open an entirely new revenue stream for MYOS in the future.

…Growing revenues make MYOS attractively valued…

MYO-T12 has been distributed by MHP since May 2012, and is currently available in specialty retail stores such as GNC, Vitamin Shoppe, bodybuilding.com, and MHPStrong.com under the brand name MYO-X. Sales of MYO-X in 2012 and 2013 were $0.9 and $3.3 million, respectively. In the first quarter of 2014, MYOS reported sales of $1.5 million, which we forecast will lead to a more than doubling of revenues in 2014 compared to 2013.

Looking ahead, we forecast MYOS to continue to have strong revenue growth, as we are estimating sales of $10.5, $16.5, and $22.0 million in 2015, 2016, and 2017, respectively.



MYOS has a clean capital structure with a low number of shares outstanding. As of Mar. 31, 2014 there were only 2.919 million common shares. In addition, there are very few warrants and stock options outstanding, meaning future dilution to existing shareholders should be minimal.

Basic Share Count	2.919
Preferred Stock	0.000
Stock Options	0.030
Employee Incentive Plan	0.252
Stock Warrants	0.316[a] 0.158[b]
Fully Diluted	**3.517**

[a]Strike Price = $15.00

[b]Strike Price = $45.00

A simple valuation for MYOS can be constructed based upon a price to sales ratio. With a stock price close to $16, that puts the fully diluted market cap at approximately $60 million. This values the company at a price to sales ratio of approximately 6x 2015 revenues and 3.6x 2016 revenues.

Were MYOS to solely focus itself as an OTC bionutrition company it may be considered fairly valued at the current price. However, MYOS is positioning itself to be a leader in muscle science through a dedicated research and discovery group that is focused on developing therapeutics based on scientifically validated pathways for improving muscle development and health, which we believe warrants a higher valuation multiple.

When comparing MYOS to other companies, we believe that the company should be looked at alongside other bionutrition companies that are backed up by solid clinical science, such as Amarin Corp. (Nasdaq: AMRN) and Neptune Technologies and Bioressources (TSX: NTB). Both of those companies have utilized clinical science to advance the marketing of a natural product and are currently trading at a price to sales ratio of approximately ten.

Company	Ticker	2013 Sales	Market Cap	P/S ratio
Amarin Corp.	NASDAQ: AMRN	$26.4	$271	10.3
Neptune Technologies & Bioressources	TSX: NTB	$19.5	$194	9.9

We forecast that MYOS will have 2014 revenues of approximately $7.2 million. If we were to apply a multiple of 10x to these sales that would give MYOS a valuation of $72 million, which based upon the fully diluted share count gives us a valuation of approximately $20 per share.

To further validate our price target, we have constructed a more detailed discounted cash flow model based on the future revenues of MYO-X and the liquid formulation product currently under development. We forecast peak revenues of approximately $63 million and using a 15% discount rate and the fully diluted share count we calculate the shares to be worth approximately $20 (model available in back of report).

MANAGEMENT PROFILES

Dr. Robert J. Hariri – Executive Chairman

Dr. Hariri joined the company in July 2011 and was elected Chairman of the Board in April 2012. Dr. Hariri is currently the Chairman and Founder of Celgene Cellular Therapeutics, a division of Celgene Corporation, and previously served as the Chief Executive office from 2005 to 2013. Prior to joining Celgene Cellular Therapeutics as president in 2002, Dr. Hariri was founder, chairman and chief scientific officer at Anthrogenesis Corporation/LIFEBANK, Inc., a privately held biomedical technology and service corporation involved in the area of human stem cell therapeutics, which was acquired by Celgene in 2002. He has also served as co-founder, vice chairman and chief scientific officer of Neurodynamics, a privately held medical device and technology corporation. Dr. Hariri has also held key academic positions at Weill Medical College of Cornell University and the Cornell University Graduate School of Medical Science, including serving as the director of the Center for Trauma Research. Dr. Hariri is Co-founder and Vice-chairman of Human Longevity, Inc. and serves on the board of directors of Provista Diagnostics.Dr. Hariri is a member of the board of trustees of the J.Craig Venter Institute and board of visitors of the Columbia University Fu Foundation School of Engineering and Applied Sciences and the Science and Technology Council of the Columbia University College of Physicians and Surgeons and is a member of the scientific advisory board for the Archon X Prize for Genomics, which is awarded by the X Prize Foundation. Dr. Hariri was recently appointed to the New Jersey Commission for Cancer Research by Governor Chris Christie. Dr. Hariri received his undergraduate training at Columbia College and Columbia University School of Engineering and Applied Sciences and was awarded his M.D. and Ph.D. degrees from Cornell University Medical College. Dr. Hariri received his surgical training at The New York Hospital-Cornell Medical Center and directed the Aitken Neurosurgery Laboratory and the Center for Trauma Research.

Peter Levy – President and Chief Operating Officer

Mr. Levy joined the company in February 2012 as Chief Operating Officer and Executive Vice President and has served as President since February 2013. From October 2010 to January 2012, Mr. Levy served as Executive Vice President of Empire Sports and Entertainment Company, a promotional and entertainment firm focused on live events. From April 2010 to October 2010, he served as head of research and development for JMP Holdings, a real estate development firm maintaining a portfolio of retail, entertainment, sports, education, government projects, and residential properties. From January 1999 until April 2010, Mr. Levy was a partner and principal of Sobel & Co., LLC, Certified Public Accountants and Consultants, a regional CPA firm, where he was responsible for the firm's Sarbanes-Oxley practice, Strategic Planning, and the Corporate Integrity Unit. Mr. Levy graduated from Harvard University in 1982 with honors, and was a recipient of the John Harvard Scholarship for Academic Distinction. Mr. Levy graduated from Cornell Law School in 1985.

Joseph DosSantos – Chief Financial Officer

Mr. DosSantos joined MYOS as the Chief Financial Officer in May 2014. Prior to joining MYOS, Mr. DosSantos served as Executive Director, Finance Operations, at Actavis plc, a New York Stock Exchange listed global specialty pharmaceutical company focused on developing, manufacturing, and distributing generic, brand, and biosimilar products. Previously, he served as Vice President, Corporate Controller, of Alvogen, a multi-national, privately-owned pharmaceutical company focused on developing, manufacturing, and distributing generic, over-the-counter and biosimilar pharmaceutical products. Mr. DosSantos has also served as Senior Director, Assistant Corporate Controller at Celgene Corporation, a NASDAQ-listed global biopharmaceutical company engaged in the discovery, development and commercialization of innovative therapies for the treatment of cancer and immune-inflammatory related diseases. Additionally he has held positions of increasing responsibilities at Cytec Industries and National Starch & Chemical, two multi-national chemical companies. Mr. DosSantos is a licensed certified public accountant in New Jersey, graduated from Kean University in 1991 with a B.S. in Accountancy and holds an M.B.A. in Finance from Seton Hall University.

Dr. Robert Ashton – Chief Medical Officer

Dr. Ashton joined MYOS in February 2014 as the Chief Medical Officer. From April 2012 to January 2014, Dr. Ashton served as Chief Medical Officer of Advanced Practice Strategies, Inc., a company focused on lifelong learning for clinicians and risk management solutions for hospitals. From March 2009 through January 2012, Dr. Ashton served as Director of Thoracic Surgery in the Moses Division of Montefiore Medical Center where he practiced thoracic surgery and general surgery. From July 2005 to February 2009, Dr. Ashton served as a thoracic surgeon at Hackensack University Medical Center, in Hackensack, New Jersey. From January 2002 to June 2005, Dr. Ashton worked as a surgeon and served as a Director of Minimally Invasive and Robotic Thoracic Surgery at St. Luke's Roosevelt Hospital Center, in New York, New York. From July 2000 to December 2001, Dr. Ashton worked as an attending surgeon at Rockland Thoracic Associates. In these positions, Dr. Ashton has experience in the clinical practice and scientific development in medicine, with a background in cardiovascular disease, oncology, obesity, transplantation and chronic disease states. Dr. Ashton has published over 75 original manuscripts and abstracts and has a comprehensive understanding of wellness and preventive medicine and is a contributor on Fox News Channel along with appearances on the Today Show, NBC Nightly News, CBS World News, and MSNBC. Dr. Ashton is currently a member of the board of directors at Jenrin Discovery, a preclinical drug development company focused on a variety of metabolic syndromes, and CytImmune Sciences, a clinical stage drug development company focused on oncology. Dr. Ashton was the co-founder of MDLinx, Inc., an online healthcare media company that was acquired in 2006 by So-Net M3, a Sony Communication Network Group company. Dr. Ashton received a B.S. degree in Biology and Philosophy, with Honors, from Muhlenberg College in 1987, and received a M.D. degree from The Medical College of Pennsylvania in 1992.

PROJECTED FINANCIALS

MYOS Corporation – Income Statement

MYOS Corporation	2012 A	2013 A	Q1 A	Q2 E	Q3 E	Q4 E	2014 E	2015 E	2016 E
Myo-X	**$0.9**	**$3.3**	**$1.5**	**$1.7**	**$1.9**	**$2.1**	**$7.2**	**$9.5**	**$13.5**
YOY Growth	-	*263.9%*	-	-	-	-	*117.8%*	*31.5%*	*42.1%*
Future Liquid Product	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$1.0**	**$3.0**
YOY Growth	-	-	-	-	-	-	-	-	*200.0%*
Total Revenues	**$0.9**	**$3.3**	**$1.5**	**$1.7**	**$1.9**	**$2.1**	**$7.2**	**$10.5**	**$16.5**
YOY Growth	-	*263.9%*	-	-	-	-	*117.8%*	*45.3%*	*57.1%*
CoGS	$0.9	$1.5	$0.4	$0.5	$0.5	$0.6	$1.9	$2.6	$3.6
Product Gross Margin	*5.3%*	*54.2%*	*73.8%*	*73.2%*	*73.7%*	*73.8%*	*73.6%*	*75.2%*	*78.2%*
R&D	$0.2	$0.8	$0.4	$0.4	$0.5	$0.6	$1.9	$3.5	$5.5
% R&D	*22.7%*	*22.7%*	*28.1%*	*26.2%*	*23.7%*	*28.6%*	*26.6%*	*33.3%*	*33.3%*
SG&A	$2.9	$5.3	$1.1	$1.3	$1.3	$1.4	$5.2	$6.0	$7.5
% SG&A	*315.6%*	*160.0%*	*73.1%*	*79.8%*	*68.4%*	*66.7%*	*71.6%*	*57.1%*	*45.5%*
Other expenses	$0.9	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.1	$0.1
Operating Income	($3.9)	($4.3)	($0.4)	($0.6)	($0.4)	($0.5)	($1.8)	($1.7)	($0.2)
Operating Margin	*0.0%*	*-128.6%*	*-27.4%*	*-32.7%*	*-18.4%*	*-21.4%*	*-24.5%*	*-16.2%*	*-1.2%*
Total Other Income	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Pre-Tax Income	($3.9)	($4.3)	($0.4)	($0.5)	($0.3)	($0.4)	($1.8)	($1.7)	($0.2)
Taxes & Other	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Tax Rate	*0%*	*100%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	($3.9)	($4.3)	($0.4)	($0.5)	($0.3)	($0.4)	($1.8)	($1.7)	($0.2)
Reported EPS	**($2.13)**	**($1.93)**	**($0.16)**	**($0.20)**	**($0.12)**	**($0.15)**	**($0.62)**	**($0.49)**	**($0.04)**
YOY Growth	-	-	-	-	-	-	-	-	-
Shares Outstanding	1.8	2.2	2.7	2.8	2.9	3.0	2.9	3.4	4.0

Source: Zacks Investment Research, Inc. *Jason Napodano, CFA*

© Copyright 2014, Zacks Investment Research. All Rights Reserved.

MYOS Corporation – DCF Model

MYOS Corp	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023
Myo-X											
Total Myo-X Revenues	3.29	6.97	9.61	13.24	16.85	21.43	24.99	29.13	33.97	39.61	46.18
Liquid Nutrition Product	0.00	0.00	1.00	3.50	5.50	7.00	9.00	11.00	13.00	15.00	17.00
Total Revenues	**$3.29**	**$6.97**	**$10.61**	**$16.74**	**$22.35**	**$28.43**	**$33.99**	**$40.13**	**$46.97**	**$54.61**	**$63.18**
Cost of Good Sold	$1.5	$1.9	$2.5	$3.7	$4.8	$5.9	$7.0	$8.3	$9.7	$11.3	$13.0
Research & Development	$0.8	$2.1	$3.5	$5.5	$6.0	$6.5	$7.0	$7.5	$8.3	$9.1	$10.7
Selling, General & Administrative	$5.3	$5.0	$6.0	$7.5	$7.8	$8.1	$8.4	$8.8	$9.1	$9.5	$9.9
Other Expenses	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
EBITDA	($4.3)	($2.0)	($1.4)	$0.0	$3.7	$7.9	$11.5	$15.6	$19.8	$24.7	$29.6
Depreciation and amortization	*$0.0*	*$0.1*	*$0.1*	*$0.1*	*$0.1*	*$0.1*	*$0.1*	*$0.1*	*$0.1*	*$0.1*	*$0.1*
EBIT	**($4.3)**	**($2.1)**	**($1.5)**	**($0.1)**	**$3.6**	**$7.8**	**$11.4**	**$15.5**	**$19.7**	**$24.6**	**$29.5**
EBITDA margin	-	*-30.5%*	*-14.0%*	*-0.3%*	*16.3%*	*27.5%*	*33.7%*	*38.5%*	*42.0%*	*45.1%*	*46.7%*
Cash Taxes	$0	$0	$0	$0.0	($1.2)	($2.5)	($3.7)	($4.9)	($6.3)	($7.9)	($9.4)
Tax Rate	-	-	-	-	*32%*	*32%*	*32%*	*32%*	*32%*	*32%*	*32%*
Discounted Cash Flow											
Net Income	($4.3)	($2.1)	($1.5)	($0.1)	$2.5	$5.3	$7.8	$10.5	$13.4	$16.7	$20.1
Depreciation and amortization	$0.0	$0.1	$0.1	$0.1	$0.1	$0.1	$0.1	$0.1	$0.1	$0.1	$0.1
Net Other Cash Adjustment	$1.3	$1.1	$0.9	$0.7	$0.5	$0.3	$0.1	($0.1)	($0.3)	($0.5)	($0.7)
Post-Tax Operating Cash Flow	**($3.0)**	**($0.9)**	**($0.5)**	**$0.7**	**$3.1**	**$5.7**	**$8.0**	**$10.5**	**$13.2**	**$16.3**	**$19.5**
Dividends	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Free Cash Flow	**($3.0)**	**($0.9)**	**($0.5)**	**$0.7**	**$3.1**	**$5.7**	**$8.0**	**$10.5**	**$13.2**	**$16.3**	**$19.5**

Assumptions	
Equity Risk Premium:	6.0%
Risk Free Rate:	2.0%
Terminal Growth:	2.0%
Discount Rate:	**15.0%**

Valuation	
10-Year Model:	$25
Terminal Value:	$37
Net Cash - LT Debt:	$9
Firm Value:	**$71**

Price Target	
Basic Outstanding:	2.9
Options / Warrants:	0.6
Fully Diluted:	3.5
Target Price:	**$20.13**

HISTORICAL ZACKS RECOMMENDATIONS



© Copyright 2014, Zacks Investment Research. All Rights Reserved.

DISCLOSURES

The following disclosures relate to relationships between Zacks Small-Cap Research ("Zacks SCR"), a division of Zacks Investment Research ("ZIR"), and the issuers covered by the Zacks SCR Analysts in the Small-Cap Universe.

ANALYST DISCLOSURES

I, Jason Napodano, CFA, hereby certify that the view expressed in this research report accurately reflect my personal views about the subject securities and issuers. I also certify that no part of my compensation was, is, or will be, directly or indirectly, related to the recommendations or views expressed in this research report. I believe the information used for the creation of this report has been obtained from sources I considered to be reliable, but I can neither guarantee nor represent the completeness or accuracy of the information herewith. Such information and the opinions expressed are subject to change without notice.

INVESMENT BANKING, REFERRALS, AND FEES FOR SERVICE

Zacks SCR does not provide nor has received compensation for investment banking services on the securities covered in this report. Zacks SCR does not expect to receive compensation for investment banking services on the Small-Cap Universe. Zacks SCR may seek to provide referrals for a fee to investment banks. Zacks & Co., a separate legal entity from ZIR, is, among others, one of these investment banks. Referrals may include securities and issuers noted in this report. Zacks & Co. may have paid referral fees to Zacks SCR related to some of the securities and issuers noted in this report. From time to time, Zacks SCR pays investment banks, including Zacks & Co., a referral fee for research coverage.

Zacks SCR has received compensation for non-investment banking services on the Small-Cap Universe, and expects to receive additional compensation for non-investment banking services on the Small-Cap Universe, paid by issuers of securities covered by Zacks SCR Analysts. Non-investment banking services include investor relations services and software, financial database analysis, advertising services, brokerage services, advisory services, investment research, investment management, non-deal road shows, and attendance fees for conferences sponsored or co-sponsored by Zacks SCR. The fees for these services vary on a per client basis and are subject to the number of services contracted. Fees typically range between ten thousand and fifty thousand USD per annum.

POLICY DISCLOSURES

Zacks SCR Analysts are restricted from holding or trading securities in the issuers which they cover. ZIR and Zacks SCR do not make a market in any security nor do they act as dealers in securities. Each Zacks SCR Analyst has full discretion on the rating and price target based on his or her own due diligence. Analysts are paid in part based on the overall profitability of Zacks SCR. Such profitability is derived from a variety of sources and includes payments received from issuers of securities covered by Zacks SCR for services described above. No part of analyst compensation was, is or will be, directly or indirectly, related to the specific recommendations or views expressed in any report or article.

ADDITIONAL INFORMATION

Additional information is available upon request. Zacks SCR reports are based on data obtained from sources we believe to be reliable, but are not guaranteed as to be accurate nor do we purport to be complete. Because of individual objectives, this report should not be construed as advice designed to meet the particular investment needs of any investor. Any opinions expressed by Zacks SCR Analysts are subject to change without notice. Reports are not to be construed as an offer or solicitation of an offer to buy or sell the securities herein mentioned.

ZACKS RATING & RECOMMENDATION

ZIR uses the following rating system for the 1047 companies whose securities it covers, including securities covered by Zacks SCR:
Buy/Outperform: The analyst expects that the subject company will outperform the broader U.S. equity market over the next one to two quarters.
Hold/Neutral: The analyst expects that the company will perform in line with the broader U.S. equity market over the next one to two quarters.
Sell/Underperform: The analyst expects the company will underperform the broader U.S. Equity market over the next one to two quarters.

The current distribution is as follows: Buy/Outperform- 16.5%, Hold/Neutral- 77.7%, Sell/Underperform – 5.1%. Data is as of midnight on the business day immediately prior to this publication.