Zacks Small-Cap Research

February 18, 2015
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Omni Bio Pharma, Inc. (OMBP-OTCQB)

OMBP: Preclinical Development of FC-AAT Continues…

Current Recommendation	**Neutral**
Prior Recommendation	N/A
Date of Last Change	04/15/2013
Current Price (02/18/15)	$0.27
Target Price	**$0.50**

UPDATE

Omni Bio Pharma is continuing with preclinical development of Fc-AAT, as well participating in outside studies testing the use of pAAT in things like post-acute STEMI and GvHD. The company has secured enough cash to fund operations into 2015. However, in order to push Fc-AAT forward into advanced preclinical development – and file the IND for human studies – significant capital is required. Data on the use of p-AAT in GvHD and post-MI is coming in the fourth quarter 2014. Could that data lead to a sub-licensing agreement and significant non-dilutive capital for Omni Bio?

SUMMARY DATA

52-Week High	**$0.45**
52-Week Low	**$0.16**
One-Year Return (%)	**-20.00**
Beta	**2.11**
Average Daily Volume (sh)	**28,418**
Shares Outstanding (mil)	**40**
Market Capitalization ($mil)	**$12**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**34**
Insider Ownership (%)	**22**
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 2015 Estimate	**N/A**
P/E using 2016 Estimate	**N/A**

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

ZACKS ESTIMATES

Revenue
(In millions of $)

	Q1 (Jun)	Q2 (Sep)	Q3 (Dec)	Q4 (Mar)	Year (Mar)
2014	0 A	0 A	0 A	0 A	0 A
2015	0 A	0 A	0 A	0 E	0 E
2016					0 E
2017					0 E

Earnings per Share
(EPS is operating earnings)

	Q1 (Jun)	Q2 (Sep)	Q3 (Dec)	Q4 (Mar)	Year (Mar)
2014	-$0.02 A	-$0.02 A	-$0.02 A	-$0.01 E	-$0.07 A
2015	-$0.02 A	-$0.03 A	-$0.04 A	-$0.02 E	-$0.10 A
2016					-$0.10 E
2017					-$0.14 E

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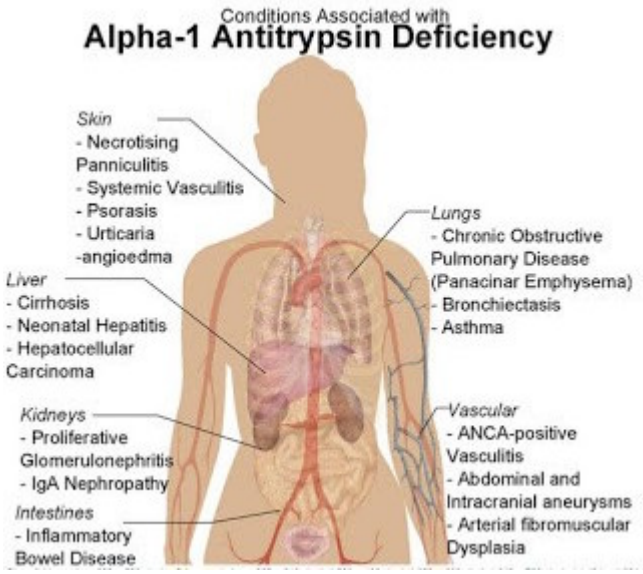
WHAT'S NEW

Brief Background On A1AT

Alpha-1 Antitrypsin (A1AT) is a protease inhibitor belonging to the serpin family. A1AT is a single-chain 52-kDa / 394 amino acid glycoprotein produced by the liver with a circulating reference range in blood between 1.0 and 3.5 mg/mL. A1AT acts as a serum trypsin inhibitor, inhibiting a wide variety of proteases, protecting tissue from enzymes of inflammatory cells, especially neutrophil elastase. Research shows that A1AT modifies dendritic cell maturation and promotes T-cell differentiation. Specifically, A1AT decreases the production of important inflammatory cytokines such as tumor necrosis factor (TNF)-α and interleukin (IL)-1β, two prototypical upstream mediators of inflammation. A1AT also lowers the levels of the chemokines and monocyte chemotactic protein (MCP)-1, two major chemokines in the trafficking of inflammatory cells (Lewis EC, 2011). And now we finally have human data confirming these animal model studies. For example, data presented at AHA in November is suggestive of A1AT also lowering C-Reactive Protein (CRP) and data at ASH in December is suggestive of A1AT lowering IL-1Ra.

In the absence or low levels of circulating A1AT, proteases such as neutrophil elastase are free to break down the connective tissue fiber elastin, which contributes to the elasticity of the lungs, resulting in respiratory complications such as emphysema or chronic obstructive pulmonary disease (COPD) in adults and hepatic cirrhosis in children. Genetic disorders that result in low levels of A1AT allow for the degradation of lung tissue that leads to the characteristic manifestation of pulmonary emphysema (Tuder et al, 2010).



A1AT deficiency (A1AD) has no cure. Instead, physicians attempt to manage the symptoms of the disease, either by treating the related lung or liver conditions or augmenting circulating A1AT levels with weekly infusions of plasma-derived A1AT (p-AAT). However, because p-ATT is in limited quantities (it is pooled from donated human plasma), augmentation by weekly infusions of pAAT is recommended only for patients with severe A1AD. It's also incredibly expensive at around $2,000 per dose. And, since the half-life of pAAT is roughly 3 to 5 days, A1AD patients require weekly infusions. This puts the yearly cost at over $100,000 for the average 60-70 kg patient.

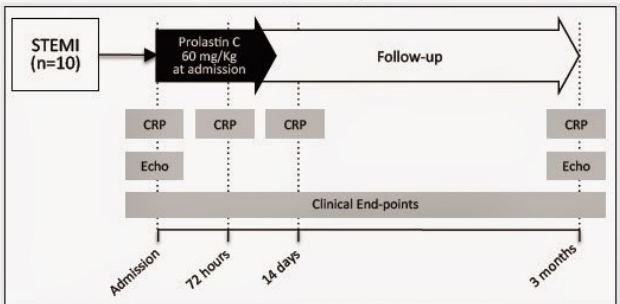
There are four manufacturers of pAAT worldwide, Grifols, Baxter, CSL Behring, and Kamada. The largest providers is Grifols' with a product they call **Prolastin-C**. Annual sales of Prolastin-C are roughly $500 million.

Phase 1 Data At AHA – Evidence Of Effect On CRP

On November 19, 2014, Omni BioPharmaceuticals, Inc. presented data from a Phase 1 clinical trial studying the effects of a single dose of plasma-derived alpha-1 antitrypsin (pAAT) in ten patients post-acute ST-elevation myocardial infarction ("STEMI"). The goal of the study (**NCT01936896**) was to determine whether treatment with pAAT reduces inflammatory response in patients with acute STEMI. The study took place at The Virginia Commonwealth University under direction of principal investigator Antonio Abbate, M.D., Ph.D. The data were presented in poster form at the American Heart Association Scientific Sessions 2014, in Chicago, IL.

Prolastin-C was provided to Dr. Abbate's lab at VCU free of charge, courtesy of Grifols. The study itself was sponsored by the American Heart Association. As noted above, the goal of the study was to determine whether treatment with pAAT reduces inflammatory response in patients with acute STEMI. A total of 10 patients were enrolled. The primary endpoint was AUC for C-Reactive Protein (CRP) with data points at baseline, day 3, and day 14. Secondary endpoints included safety and both cardiac and non-cardiac adverse events over a 3 month period following the single infusion of Prolastin-C at 60 mg/kg. Below is a cartoon depicting the trial design. We note there was no placebo group in the trial. Instead, investigators chose to compare the results with 20 historical STEMI patients (likely at the same hospital this trial took place). We also show patient characteristics below.

Study Design



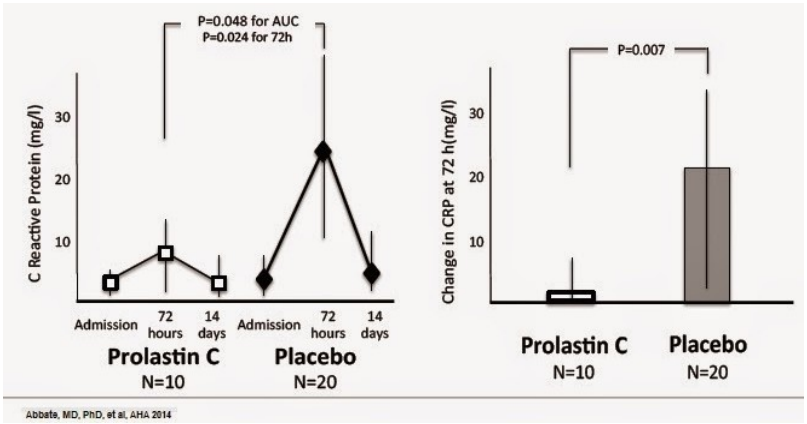
Study design. Single-arm, open-label, treatment with Prolastin C in patients with STEMI.

Abbate, MD, PhD, et al AHA 2014

Patient Characteristics

	Prolastin C (10 pts)	Historic Placebo Controls (20 pts)	p value
Number of patients	10	20	
Age (years)	52 (45-61)	58 (50-65)	0.42
Gender (males)	5 (50%)	18 (90%)	0.026
Race (caucasians)	7 (70%)	11 (55%)	0.69
Hyperlipidemia	6 (60%)	13 (65%)	1.0
Hypertension	4 (40%)	14 (70%)	0.14
Diabetes mellitus	1 (10%)	4 (20%)	0.64
Tobacco use	7 (70%)	7 (70%)	1.0
LVEF (%)	49 (45-61)	54 (41-63)	0.90
Time from pain to PCI (min)	85 (58-141)	155 (82-387)	0.10
Time from pain to drug (min)	456 (325-575)	480 (375-608)	0.67
Location of ST elevation Anterior Inferior/Posterior Lateral	 2 (20%) 7 (70%) 1(10%)	 6 (30%) 10 (50%) 4 (20%)	0.56
Peak CK-MB (ng/ml)	123 (30-195)	123 (71-213)	0.71

The data show that the area-under-the-curve (AUC) of CRP levels was significantly lower in the Prolastin-C treated group when compared to historic controls. The graph below on the left shows the AUC for CRP over the three tested time periods, infusion, day 3 and day 14. The graph on the right shows the absolute change in CRP at day 3. Investigators reported that both data sets are statistically significant when compared to historic controls.



Abbate, MD, PhD, et al, AHA 2014

Obviously it would have been ideal to compare these ten patients vs. another ten patients on placebo instead of 20 historic placebo patients. As stock analysts, we are skeptical of calling single-center data statistically significant when compared to historic controls. The trial design of this Phase 1 study was clearly not the gold-standard randomized, multi-center, placebo-controlled design that is required for FDA approval, let alone true proof-of-concept. However, we believe the marked reduction in CRP is clearly evidence as demonstrated in the graph above on the right. We look forward to seeing future data in this regard from next stage clinical trials.

In terms of how a reduction in CRP relates to real-world outcomes for patients, study investigators followed all 10 patents for 12 weeks after Prolastin-C infusion. One subject died during sleep at week 8. Study investigators believe this individual suffered stent thrombosis. Another subject suffered a pulmonary embolism at week 11. No subjects in the study showed new onset heart failure or recurrent myocardial infarction at week 12.

Safety results show that Prolastin C infusion was well tolerated, with no related adverse events. This was expected considering the U.S. FDA has already approved the use of Prolastin-C in A1AD patients. Below is a table showing patient vital signs prior to and at the end of infusion. The average time from percutaneous coronary intervention to drug infusion was 320 minutes [260-444] with the average infusion time taking between 12 and 15 minutes.

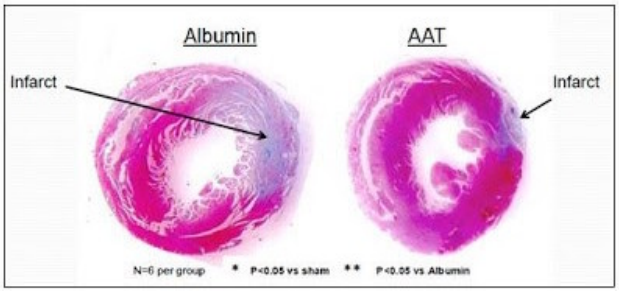
	Prior to Infusion	End of Infusion
Systolic blood pressure (mmHg)	128 [108-139]	125 [115-135]
Diastolic blood pressure (mmHg)	77 [64-89]	70 [61-75]
Heart rate (_/min)	74 [61-89]	70 [64-78]
Oxygen saturimetry (%)	98 [96-99]	98 [96-99]
Temperature (oral, C°)	36.7 [36.6-36.8]	36.6 [36.4-36.9]
Pain/Discomfort (0/10 scale)	0 [0-0]	0 [0-0]

Abbate, MD, PhD, et al, AHA 2014

Animal Data Show Promise In Ischemic Heart Disease

Data shows that pAAT may play a role in preserving tissue integrity and reducing scar formation thanks to its potent anti-inflammatory characteristic (Lewis, EL, 2012). Evidence also shows protection from ischemia reperfusion injury in renal mouse models by pAAT (Daemen et al, 2000).

Work published in the Journal of Molecular Cell Cardiology shows the effects of exogenously administered pAAT on caspase-1 activity and on the outcome of ischemia-reperfusion injury in a mouse model of acute myocardial infarction. Mice underwent 30 min of coronary artery ligation followed by reperfusion and were randomly assigned to receive clinical-grade A1AT or albumin at reperfusion. Infarct size was evaluated after 1 and 7 days. A1AT-treated mice had significantly smaller infarct sizes (-30% day 1 and -55% day 7) compared with mice treated with albumin (below). AAT-treated mice also exhibited a smaller increase in left ventricular end-diastolic diameter and end-systolic diameter, and smaller reduction in ejection fraction. The authors conclude that exogenous administration of clinical grade A1AT reduces caspase-1 activity in the ischemic myocardium leading to preservation of viable myocardium and prevention of adverse cardiac remodeling (Toldo S et al, 2011).



Phase 1/2 Data At ASH – Evidence Of Utility In GvHD

Earlier this week, at the American Society of Hematology (ASH) meeting, investor got yet another interesting look at the potential utility of pAAT, this time for the treatment of refractory Graft-vs-Host Disease (GvHD). The data were presented in poster form at ASH, and the study (**NCT01523821**) was sponsored by the Fred Hutchinson Cancer Research Center, pAAT manufacturers Baxter International (BAX) and Kamada Ltd (KMDA), and Omni BioPharma.

Graft-versus-host disease is a common complication following allogeneic (genetically-mismatched) tissue transplant, most often associated with stem cell or bone marrow transplant. Symptoms in both acute and chronic GvHD range from mild to severe, and usually show up within the first three months after a transplant. Common treatment options for patients post allogenic stem cell transplant includes drugs that suppress the T cell mediated immune onslaught on the host tissues, including glucocorticoids such as prednisone. These drugs often have poor tolerability, may illicit harmful side effects on the kidneys and liver, and create risk of infection of cancer relapse.

Despite treatment with high-dose prednisone, 60% to 80% of unrelated allogeneic tissue transplant patients will develop GvHD. Even when the subjects are related, GvHD still occurs in 30% to 40% post transplant (source: **NLM-NIH**). Thus, there exists a significant unmet medical need to develop a new immune modulating agent that can allow the T cell mediated graft-versus-leukemia response while sparing the recipient from an injurious inflammatory and immunological assault (Lewis EC, 2011). Previous animal research has shown potent anti-inflammatory activity with pAAT, along with the ability to modify dendritic cells toward a tolerogenic phenotype (Subramanian et al, 2011; Lewis et al, 2008).

Data published in the Proceedings of the National Academy of Sciences (PNAS) shows that administration of pAAT early after bone marrow transplant decreased mortality in three models of GvHD and reduced serum levels of pro-inflammatory cytokines such as TNF-a and IL-1b in the allogeneic recipients compared with the controls. Furthermore, pAAT treatment reduced the expansion of alloreactive T effector cells but enhanced the recovery of T regulatory cells, thus reducing the pathological harmful response. Treatment with pAAT also resulting in increased production of anti-inflammatory cytokines, IL-10 (Tawara I et al, 2012).

Data At ASH Builds On Animal Model Studies

The Fred Hutchinson Cancer Research Center, along with pAAT manufacturers Baxter and Kamada, and Omni Bio were looking to confirm the encouraging animal model data in GvHD with this early-stage clinical study. The purpose of the study was to evaluate the feasibility and safety of pAAT given to hematopoietic cell transplant (HCT) recipients with steroid non-responsive acute GvHD. Study investigators aimed at characterizing pharmacodynamic effects of pAAT while assessing clinical responses as determined by changes in stool volume and overall clinical improvement.

In the study, seven patients (3 female, 4 male) with hematologic malignancies were given transplants from HLA-matched siblings (n=5) or received cord blood (n=2) along with standard of care GvHD prophylaxis. Acute GvHD of grades III-IV developed at 49 to 71 (median of 48) days, and treatment with systemic methylprednisolone, 2 mg/kg/day was instituted. Patients showing no clinically satisfactory responses after 5 days were given pAAT (Baxter's **Glassia®**) at 90 mg/kg IV on day 1, followed by 30 mg/kg (first cohort) or 60 mg/kg (second cohort) every other day for a total of 8 doses (15 days). Below we provide a breakdown of the initial results:

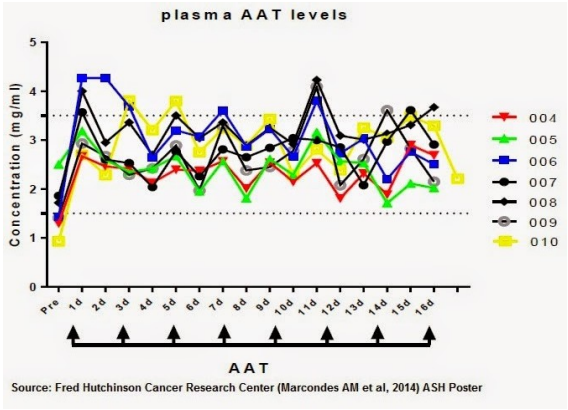
Table 1: Clinical Parameters in AAT-Treated Subjects

Patient ID	Diagnosis	Age	Donor Type	Conditioning Regimen	GVHD prophylaxis	GVHD onset	Follow-up (m)	GVHD (I-IV)	GVHD site	AAT Dose	Response
JJC001 (004)	CMML	35	PBSC	Cy/TBI	CSA/MMF	D+49	6	III	Liver/Gut	90-30 mg/kg	PR (deceased)
BAS002 (005)	CLL	58	PBSC	Flu/TBI	CSA	D+221	6	III	Gut	90-30 mg/kg	CR
DM003 (006)	AML	50	PBSC	Bu/Cy	CSA/MMF	D+71	1	III	Gut	90-30 mg/kg	CR
WC004 (007)	NHL	59	PBSC	Flu/TBI	CSA/MMF/Siro	D+37	1	IV	Gut	90-30 mg/kg	CR (CMV/Perf)
JW005 (008)	ALL	50	MMUCB	Flu/Cy/Thi/TBI	CSP/MMF	D+34	1	IV	Liver/Gut	90-30 mg/kg	PR (deceased)
ML006 (009)	AML	58	PBSC	Clo/TBI	CSA/MMF	D+93	2	III	Gut	90-30 mg/kg	CR (ARDS/CMV/Perf)
XX007 (010)	T-ALL	35	MMUCB	Flu/Cy/TBI	CSA/MMF	D+70	1	III	Liver/Gut	90-60 mg/kg	PR

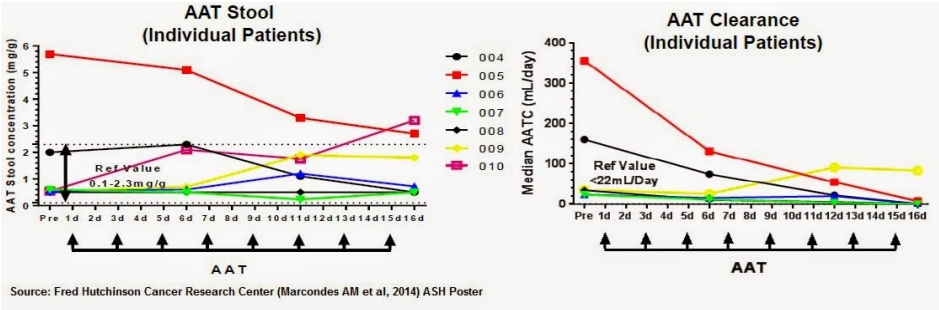
Source: Fred Hutchinson Cancer Research Center (Marcondes AM et al, 2014) ASH Poster

Four patients were deemed to have complete response to treatment with pAAT. Another patient had partial response with analysis still ongoing. Two patients died, which is not unexpected considering all patients had advanced hematologic cancers and severe GvHD. Nevertheless, a 60-70% response rate is outstanding.

If we delve deeper into the data we see some additional encouraging signs. Firstly (seen in the figure below), the dosing schedule resulted in meaningful (~80%) increase in circulating serum alpha-1 antitrypsin for all patients. Normal circulating A1AT levels are around 1.5 to 2.5 mg/ml. The IV infusions with pAAT resulted in 6 of 7 patients seeing an increase in serum circulating A1AT. The one subject that did not see an increase in alpha-1 antitrypsin serum concentration (#005) had a baseline pre-dose concentration at 2.5 mg/ml, the high end of the normal range.



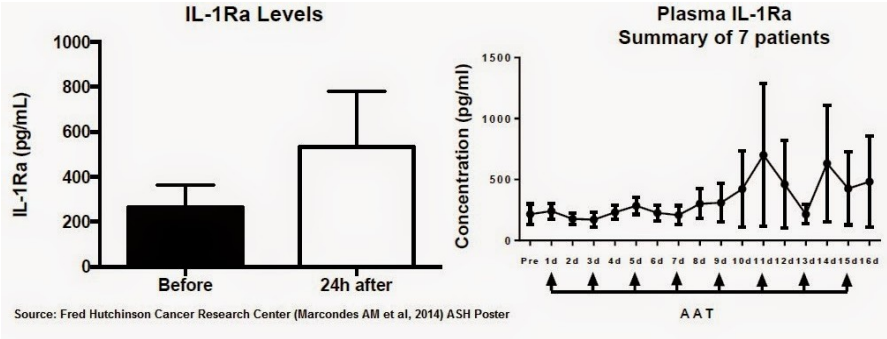
The data are not surprising, essentially showing that if you give subjects infusions of pAAT every other day at concentrations of 30 mg/kg or 60 mg/kg the circulating level of protein increases. However, more important is that A1AT stool concentrations were below plasma levels, suggesting an ability of the patients to uptake excess circulating pAAT post-dose, for whatever reason, and that the dose was not overloading the system to potentially toxic or intolerabiley levels. Also, A1AT clearance rates declined meaningfully with treatment (see below).



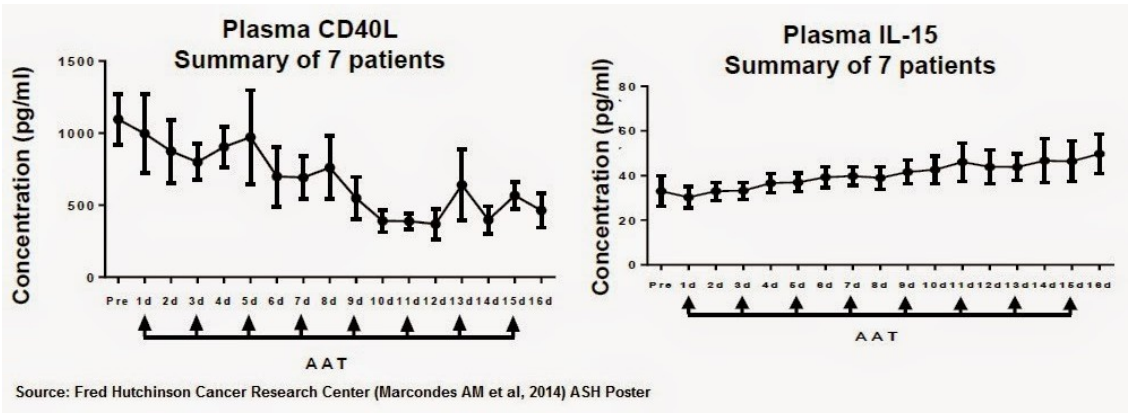
Patients in the study all had severe GvHD with inflamed gastrointestinal tract. Inflammation of the GI tract can lead to a condition called Protein Losing Enteropathy (PLE). PLE is characterized by the severe loss of serum proteins into the intestine. Normal protein loss in the gastrointestinal tract mainly consists of sloughed enterocytes and pancreatic and biliary secretions. Albumin loss through the gastrointestinal tract normally accounts for 2-15% of the total body degradation of albumin, but, in patients with severe protein-losing gastrointestinal disorders, the enteric protein loss may reach up to 60% of the total albumin pool. The serum protein level reflects the balance between protein synthesis, metabolism, and protein loss. Protein Losing Enteropathy is characterized by more loss of proteins via the gastrointestinal tract than synthesis leading to hypoalbuminemia. It is not a single disease, but an atypical manifestation of other diseases, such as GvHD.

For this pilot study, PLE was quantified by nephelometric assay and defined as levels > 22 ml/day. All seven patients had PLE at baseline, with six of the seven patients showing a marked reduction in protein loss by the end of the study. We believe this represents evidence of meaningful GI healing and GvHD symptom remission.

Besides reduction in evidence of PLE, patients showed a meaningful increase in IL-1Ra (see below). Increasing levels of IL-1Ra is a commercially validated anti-inflammatory mechanism of action for rheumatoid arthritis in Amgen's **Kineret®** (anakinra) and in Cryopyrin Associated Periodic Syndrome and Active Systemic Juvenile Idiopathic Arthritis in Novartis' **Ilaris®** (canakinumab).

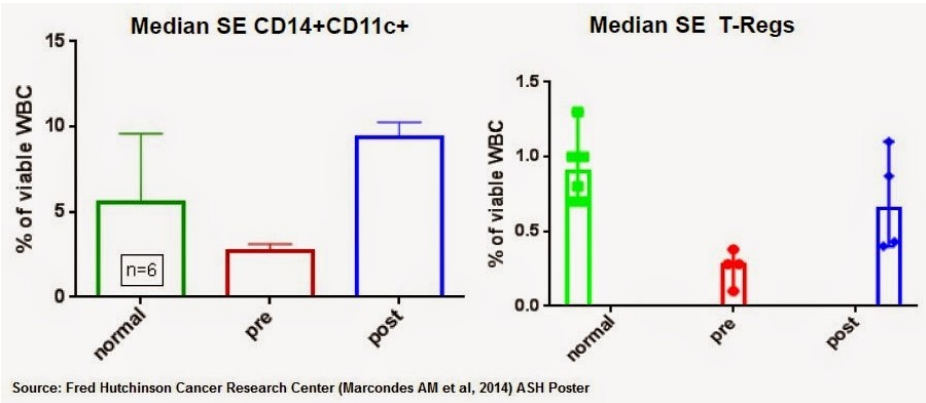


Data showing a reduction in CD40L, a biomarker of T cell activation, and an increase in IL-15, a predictor of GvHD risk factor, were also observed in the seven patients (pooled data). CD40L down regulation is indicative of the reduction in T cell activation associated with the underlying immunological basis for the GvHD. Interleukin 15 (IL-15) regulates T and natural killer (NK) cell activation and proliferation. IL-15 has been shown to enhance the anti-tumor immunity of CD8+ T cells in preclinical models (Klebanoff CA, et al, 2004). A study by Pratt et al, 2013, showed that patients with low IL-15 levels (< 30.6 ng/L) on day 7 post transplant had a 2.7-fold high likelihood of developing chronic GvHD.

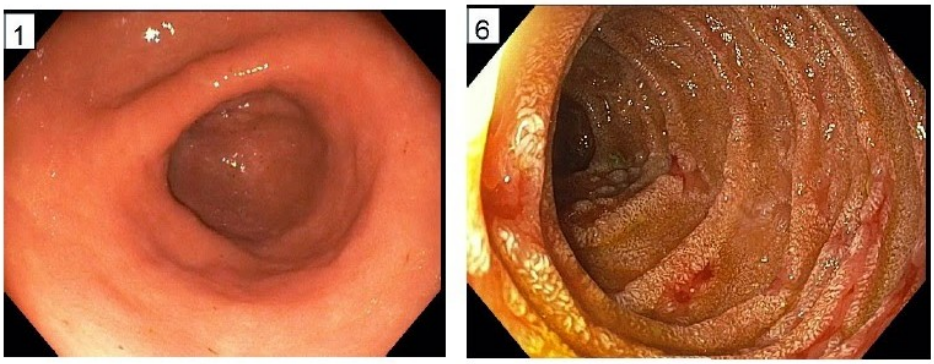


Patient samples were also analyzed by fluorescence activated cell sorter (FACS) to examine the changes in CD14+/CD11c+ (dendritic cells) and CD4+/CD25+/Fox3P+ (T regulatory cells) populations before and after treatment with pAAT. Munneke et al, 2014, showed that an increase in these dendritic cells was associated with a significant reduction in susceptibility to acute GvHD. Likewise, prior results showed that CD4+/CD25+/Fox3P regulatory T cells are able to suppress GvHD (Hoffman et al., 2002; Taylor et al., 2002). Thus, an increase in either of these cell types could be a potential explanation for the clinical response after AAT therapy.

The following two graphs show that after pAAT treatment both the CD14+/CD11c+ and CD4+/CD25+/Fox3P+ populations increase, with the percentage of dendritic cells increasing to a level greater than that found in a group of healthy controls and the percentage of T regulatory cells rising to a level similar to the group of healthy controls.



In summary, the presented biomarker and cytokine data are supportive of the underlying mechanism of pAAT as an effective treatment for in patients with steroid refractory chronic GvHD disease. There is a shift towards reduced inflammatory cytokines, improved T regulatory cells and innate immune cell function that are both associated with improved clinical outcomes and reduction in risk of chronic GvHD based on available literature. Lastly, the pictures below show an endoscopic cross section of the upper gastrointestinal tract for one of the patients pre-dose with severe GvHD (1). Note the inflammation and narrowed passage along the tract with lack of visible villus. Post treatment with pAAT the inflammation has dramatically reduced and intestinal villi return (6). These are quite impressive results.



Source: Fred Hutchinson Cancer Research Center (Marcondes AM et al, 2014) ASH Poster

Study Conclusions And What These Data Mean For Omni Bio

Study investigators concluded that continuous administration of pAAT as salvage therapy for steroid resistant gut GvHD is feasible without clinical toxicity. Stool showed a decrease in intestinal alpha-1 antitrypsin loss with pAAT treatment, as measured by AAT clearance and endoscopic evaluation suggesting healing of the bowel mucosa. Plasma-derived AAT administration also suppressed serum levels of pro-inflammatory cytokines. We regard preliminary results as encouraging, and eagerly await further exploration of pAAT therapy in the treatment of steroid refractory or acute GvHD. We remind investors that another clinical study with pAAT in GvHD is currently ongoing at the University of Michigan (**NCT01700036**). This is a slightly larger study being sponsored by CSL Behring and The Leukemia and Lymphoma Society. Data are expected in 2016.

Following the encouraging data on the use of pAAT in the treatment of post acute STEMI, Omni Bio controls various method of use and composition patents for pAAT. For example, Omni Bio controls the intellectual property to an issued **method and composition** patent for the treatment of diabetes (Type-1 or Type-2) using pAAT. The company also controls the rights to method of **use patents** covering pAAT in GvHD, non-organ transplant rejection, post myocardial infarction remodeling, certain bacterial and viral infections, and radioprotection. On May 8, 2014, Omni Bio **announced** receipt of a Grant Notice from the European Patent Office for the use of AAT in reducing the risks of non-organ transplant rejection and graft versus host disease (GvHD) in patients who have received a cornea, bone marrow, or pancreatic islet cell transplant.

There are four manufacturers of pAAT worldwide, Grifols, Baxter, CSL Behring, and Kamada. The largest provider is Grifols' with a product called **Prolastin-C**. Annual sales of Prolastin-C are roughly $500 million. We noted the post acute STEMI data presented at AHA was sponsored by The American Heart Association, with drug provided by Grifols. The data just recently presented at ASH was sponsored by the Fred Hutchinson Cancer Research Center, Baxter, and Kamada. The study ongoing at the University of Michigan is being sponsored by the CSL Behring and The Leukemia and Lymphoma Society.

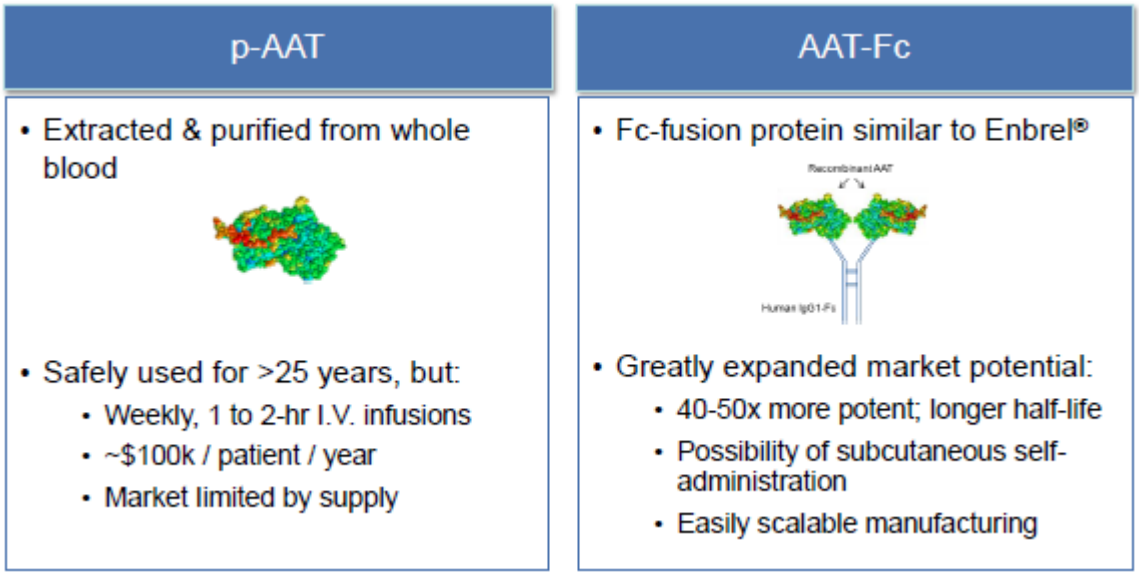
However, despite all this interest in pAAT, if anyone wants to seek approval to market their respective pAAT products for MI or GvHD, or any other indication for which Omni Bio controls the IP like diabetes, they need to come to Omni Bio for a sub-license. This creates an interesting potential bidding war between some very big players all with significant skin-in-the-game already with their respective pAAT products for the treatment of genetic alpha-1 antitrypsin deficiency.

Fc-AAT – A Game Changer

Despite all the promise and potential for pAAT, the drug remains horribly inefficient to manufacture. In an era of recombinant insulin, synthetic clotting factors, monoclonal antibodies, interference RNA, exon-skipping technology, and the ability to transplant living human neural stem cells, all of the currently available A1AT replacement therapy products are derived from pool human plasma. The market leading product, Prolastin-C, is prepared by cold ethanol fractionation from pooled human plasma purified by polyethylene glycol (PEG) precipitation, anion exchange chromatography, and cation exchange chromatography. Two additional steps, a solvent / detergent treatment and 15 nm virus removal nanofiltration are also included in the process to reduce risk of transmission of enveloped and non-enveloped viruses.

The product also has a short half-life of only 3-5 days, meaning that administration for patients with A1AD must be done through weekly infusions. The infusion process takes about 2 hours and must be performed at an infusion clinic. The average cost of therapy is around $2,000 per 60 mg/kg weekly infusion, or around $100,000 per year! It's pretty simple, p-ATT is expensive, inconvenient, and though the products are treated extensively to reduce the risk, potentially exposes patients to blood-borne pathogens.

Omni Bio's lead Fc-AAT molecule is a fusion protein that combines human AAT (below left) with an Fc fragment of human IgG1 immunoglobulin molecule. This fusion protein spontaneously binds together to form a dimer. Each dimer contains two AAT molecules and two Fc molecules connected by molecular bonds (below right).



Omni Bio believes the technology used to construct Fc-AAT is similar to that already used to create highly successful drugs for human application, such as Amgen / Pfizer's Enbrel. Enbrel was jointly developed by Immunex (acquired by Amgen) and Wyeth (acquired by Pfizer). Omni Bio's CEO, Dr. Bruce Schneider, was a senior R&D executive at Wyeth and was intimately involved in the development of Enbrel.

We think the company's Fc-AAT molecule has the potential to be a game-changer in this market; not only for replacement therapy in patients with AAT deficiency, but also in expanding the use of the A1AT molecule into new areas of therapeutic treatment such as Type-1 diabetes, GvHD, and myocardial infarction. Currently, all available supply of p-AAT is delegated for patients with A1AD. The new indications noted above have significant interest, but uptake would be limited by supply and cost. That's why we think the Fc-AAT formulation has the potential to big a big seller, and we suspect even quality for 'Breakthrough Therapy' designation by the U.S. FDA.

Omni Bio's recombinant Fc-AAT is **40-50x more potent** than p-AAT based on early-stage animal trials. For example, Fc-AAT in models of gout and myocardial infarction that show good dose response and also that a 50 ug dose of Fc-AAT performed at least as well as a 2 mg dose of p-AAT. The drug offers improved stability and longer duration of effect. Omni Bio expects that Fc-AAT will be administered via self-administered subcutaneous injections, a huge improvement over 2+ hour trips to the local infusion center.

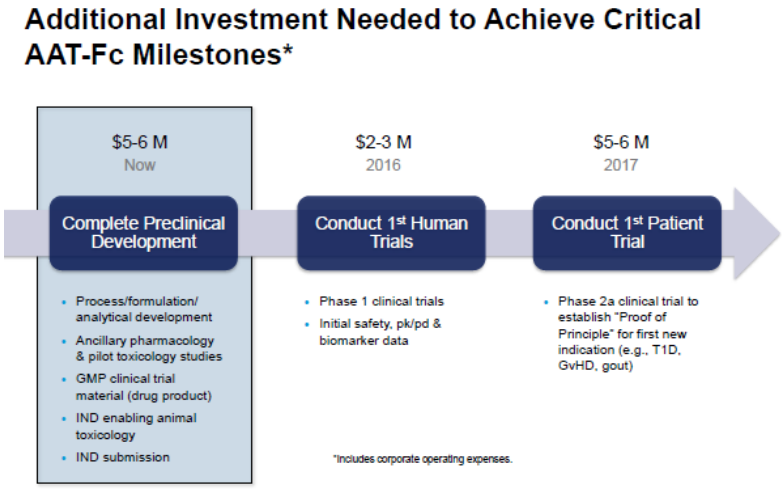
The company hit a **major milestone** earlier in November with respect with to manufacturing. Omni Bio has teamed up with Patheon and Sigma-Aldrich Fine Chemicals (SAFC) to product Fc-AAT. On November 18, Omni Bio announced that they have successfully identified several high-expression cell lines able to produce recombinant alpha-1 antitrypsin (Fc-AAT) in large enough quantities to be considered commercially viable. The news enables Omni Bio to move toward IND-enabling toxicology studies with the first and only pharmaceutically feasible recombinant A1AT candidate currently in development. We expect that Omni Bio and Patheon will move into manufacturing scale-up activities in early 2015 to support further preclinical investigations and early clinical studies. We believe a simple and low-cost manufacturing of recombinant A1AT would allow for dramatically improved supply and economics to patients.

Conclusion

Omni BioPharma remains squarely on our radar for potential break-out names in 2015. The recent data presented at AHA and ASH provide an interesting look at the mechanism of action and potential utility of pAAT outside of just use in patients with genetic alpha-1 antitrypsin deficiency. We believe this will eventually lead to a licensing agreement or out-right acquisition of the entire company by the four major pAAT players: Grifols, Baxter, CSL, or Kamada. After all, the current market capitalization is only $9 million on a basic level. This is peanuts, and a licensing deal or acquisition will result in a major move higher for the stock.

However, the story is not without risk. Omni Bio recently secured $2 million. That's not enough. They need more. Take a look at the following slide from the company's most recent investor presentation.

Develop Alone Through Phase 2a	Co-Develop with Partner	Out-License To Pharma Co.
• 3-4 year outlook • Additional funding of ~$15M required	• 1-3 year outlook • Any time from cell line optimization onward • Lowers standalone funding requirement	• Most likely from Phase 1 onward • Leveraged exit point for Omni investors



Omni Bio needs at least another $15 million between now and when they expect to sign a co-development partnership to see this strategy through. That includes another $5-6 million to complete preclinical development and $2-3 million to conduct the first human clinical trial. That's going to be a challenge given the current stock price of $0.30 per share. As of today, the basic share count is around 40.5 million, but the fully-diluted number is closer to 77 million, and will be going up as the company raises more money. For example, if Omni Bio were to go out and raise $10 million in early 2015 at today's prices of $0.25 per share, it would take issuing 50 million shares, plus most likely another 40 million warrants.

The company is making strategic advancements worth noting:

- ✓ In April 2014, the company retained investment bank Dawson James to advise and assist the company in its capital market activities.

- ✓ In August 2014, the company hired Tiberend Strategic Advisors, Inc. to provide investor relations and corporate communication services.

- ✓ In January 2015, the company engaged Spencer Edwards, Inc. to offer and sell to qualified, accredited investors up to $1 million of Units consisting of 12% Secured Convertible Promissory Notes and warrants to purchase shares of Omni's common stock equal to 10% of the face value of 2015 Notes sold. Spencer Edwards will receive a sales commission of 10% of the gross proceeds.

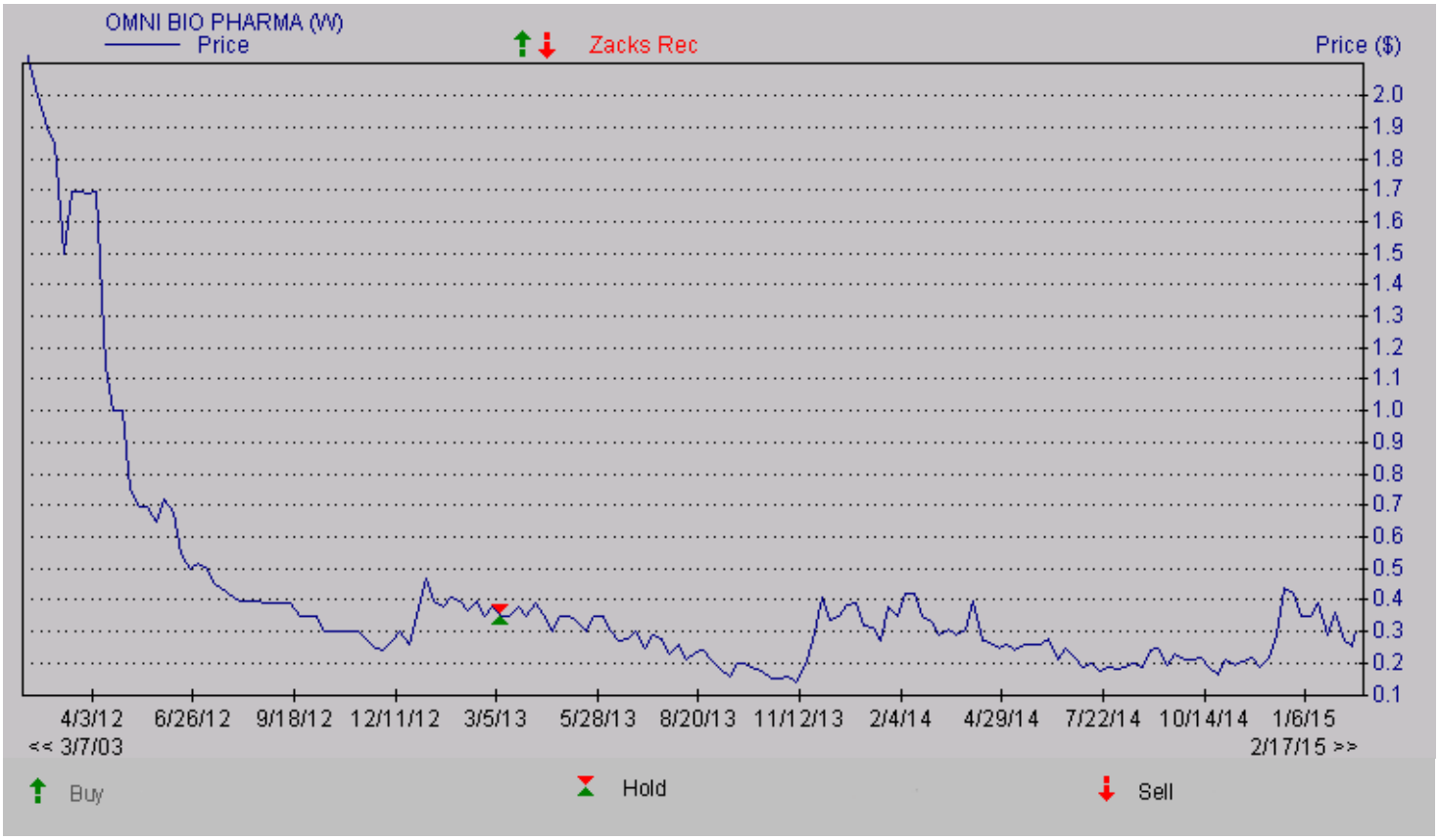
Unfortunately, the need for cash makes it difficult for us to tell investors to buy the shares today. That's an awful lot of new stock coming to the market, no matter how good the science is. What Omni Bio needs is a "White Knight" – perhaps Grifols or Kamada will generate impressive data in Type-1 diabetes and decide Omni is too good to pass up at even 3-4x this price? The data in GvHD was a major step in that direction. Thus, it's not unrealistic to believe a deal could get done in 2015.

Omni Bio Pharmaceuticals, Inc.
Income Statement

Omni Bio Pharma	FY'14A	June-14A	Sept-14A	Dec-14A	Mar-15E	FY'15E	FY'16E
Product Sales (Fc-AAT)	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-
Licensing & Collaborative Payments	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-
Total Revenues	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**
YOY Growth	-					-	-
R&D	$0.163	$0.220	$0.138	$0.112	$0.350	$0.821	$2.500
% R&D	-	-	-	-	-	-	-
SG&A	$1.750	$0.313	$0.475	$0.410	$0.450	$1.648	$2.250
% SG&A	-	-	-	-	-	-	-
Operating Income	($1.912)	($0.533)	($0.614)	($0.522)	($0.800)	($2.469)	($4.750)
Operating Margin	-	-	-	-	-	-	-
Interest & Other Income	($0.604)	($0.300)	($0.376)	($0.877)	($0.100)	($1.653)	($0.250)
Pre-Tax Income	($2.517)	($0.834)	($0.990)	($1.399)	($0.900)	($4.122)	($5.000)
Taxes & Other	$0	$0	$0	$0	$0	$0	$0
Tax Rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	($2.517)	($0.834)	($0.990)	($1.399)	($0.900)	($4.122)	($5.000)
Net Margin	-					-	-
Reported EPS	**($0.07)**	**($0.02)**	**($0.03)**	**($0.04)**	**($0.02)**	**($0.10)**	**($0.10)**
YOY Growth	-	-	-	-	-	-	-
Basic Shares Outstanding	37.3	38.9	38.9	39.2	45.0	40.5	50.0

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

HISTORICAL ZACKS RECOMMENDATIONS



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Sell/Underperform: The analyst expects the company will underperform the broader U.S. Equity market over the next one to two quarters.

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