Zacks Small-Cap Research

August 26, 2014
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Omni Bio Pharma, Inc. (OMBP-OTCQB)

OMBP: Preclinical Activities Continue On Plan…

Current Recommendation	**Neutral**
Prior Recommendation	N/A
Date of Last Change	04/15/2013
Current Price (08/26/14)	$0.23
Target Price	**$0.50**

UPDATE

Omni Bio Pharma is continuing with preclinical development of Fc-AAT, as well as keeping an eye on sponsored pilot studies with p-AAT in GvHD, post-MI, and Diabetes. 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	**$0.13**
One-Year Return (%)	**9.48**
Beta	**3.23**
Average Daily Volume (sh)	**14,461**
Shares Outstanding (mil)	**39**
Market Capitalization ($mil)	**$8**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**70**
Insider Ownership (%)	**17**
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	**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 E	0 E	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	-$0.02 E	-$0.02 E	-$0.02 E	-$0.08 A
2016					-$0.16 E
2017					-$0.20 E

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WHAT'S NEW

Financial Update

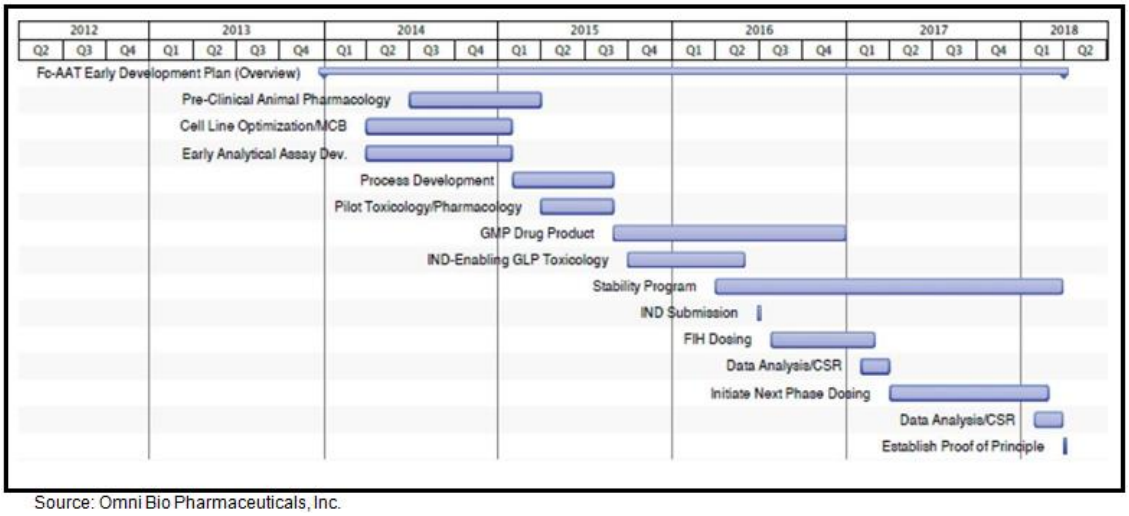
On August 15, 2014 Omni Bio Pharma (OMBP) reported financial results for the three month period ending June 30, 2014 and filed its From 10-Q with the U.S. SEC. This was the first quarter for the fiscal year ending March 31, 2015. Omni Bio did not report any revenues during the first quarter fiscal 2015. This was expected and in-line with expectations. The company reported a net loss of $0.834 million, of $0.02 per share based on 38.9 million shares outstanding. Loss was driven by $0.313 million in G&A expense and $0.220 million in R&D expense. Both line-items were slightly below expectations as the company focuses on keeping operations lean and focused on preclinical development of Fc-AAT. Loss also included $0.300 million in non-operating expenses, which included $0.241 million in non-cash amortization of debt discount expenses.

The company reported $0.652 million in cash and cash equivalents as of June 30, 2014. For the three month period, Omni Bio used $0.620 million in cash to fund operating activities. The company financed operations in the quarter by raising $0.652 million through various financing activities. We remind investors that on April 24, 2014, Omni Bio entered into a $3 million private financing plan that secured $2 million as needed and $1 million in future funds from yet to be named sources. The secured financing took place with Bohemian Investments, LLC, a Fort Collins, Colorado-based investment company with affiliation to Fort Collins-based BOCO Investments, LLC, a previous investor in Omni Bio Pharma. BOCO President, Mr. Joseph C. Zimlich is also CEO of Bohemian. BOCO Investments currently owns 39.1% of the outstanding shares. Directors and Executive Officers of the company own another combined 17% of the outstanding shares.

As of June 30, 2014, the company had borrowed $1.0 million from Bohemian Investments, LLC. We suspect that management will draw down on another $1.0 million in the next few months, providing enough cash to fund operations to the end of the year. As noted above, Omni Bio has access to another $1.0 million in future funds. We discuss the development plan and long-term financing needs below.

Preclinical Activities Continue

On April 29, 2014, the company announced an agreement with Gallus Biopharmaceuticals, LLC, a contract development and manufacturing organization (CMO), on a multi-stage manufacturing services agreement. According to the release, the agreement includes cell line optimization, analytical development, and manufacturing scale-up activities for Omni Bio's alpha-1 antitrypsin (AAT) Fc fusion protein. The goal is for Gallus to provide Omni cGMP product to support animal pharmacology and toxicology studies, and early human clinical trials of with Fc-AAT. Management has outlined these preclinical development plans in a slide from the June 2014 investor presentation – shown below:



Source: Omni Bio Pharmaceuticals, Inc.

Besides these initial process/formulation, manufacturing, and preclinical IND enabling activities, Omni Bio is also funding outside investigator studies of plasma-derived AAT (p-AAT) in steroid refractory graft-vs-host disease (GvHD). There are two ongoing pilot studies, one under the direction of Dr. Pavan Reddy, MD at the University of Michigan (NCT01700036) and another under the direction of Dr. H. Joachim Deeg at the Fred Hutchinson Cancer Research Center (NCT01523821). We discuss the opportunity for use of p-AAT in GvHD and how it relates to Omni Bio below. However, beside GvHD, several additional outside sponsored studies are currently ongoing. These programs include:

- ✓ A pilot study in patients with acute myocardial infarction under direction of lead investigator, Dr. Antonio Abbate, MD, PhD of the Virginia Commonwealth University (NCT01936896)
- ✓ A Phase 2/3 placebo controlled trial of Kamada (KMDA) & Baxter's (BAX) Glassia® in recent onset Type-1 diabetics (NCT02005848). This is a fairly-large trial that just began in November 2013 with a goal of enrolling 192 newly diagnosed Type-1 diabetics at four clinical sites in Israel. The primary endpoint is beta cell function at twelve months vs. baseline.
- ✓ A Phase 2 placebo controlled trial of Grifols Therapeutics' Prolastin-C® in recent onset Type 1 diabetics (NCT02093221). This is a slightly smaller trial at only 75 patients compared to the one Kamada and Baxter are running, but the trial is being conducted at a dozen sites in the U.S. so many garner more U.S. investor attention.

The reason we mention these outside studies is because Omni Bio Pharma controls the intellectual property to an issued method and composition patent for the treatment of diabetes (Type-1 or Type-2) using p-AAT. The company also controls the rights to method of use patents covering p-AAT 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.

That means if Kamada or Grifols wants to seek approval to market their respective p-AAT products for diabetes or GvHD, or any other indication for which Omni Bio Pharma controls the IP, they need to come to Omni Bio for a sub-license. This creates an interesting potential bidding war between some very big players in Baxter, Kamada, and Grifols, all with significant skin-in-the-game already with their p-AAT products for the treatment of alpha-1-antitripsan deficiency (AATD). In a sense, it's a race for these companies to generate data in these additional indications and then come to Omni Bio to negotiate a deal. While it's too early to strike a deal right now for any of these indications – Baxter, Kamada, and Grifols want to get the data first so they know if it is worth sub-licensing the IP – in two or three years, the IP alone that Omni Bio controls could be worth hundreds of millions.

For example:

…In GvHD…

GvHD is a common complication following allogeneic (genetically-mismatched) tissue transplant, most often associated with stem cell or bone marrow 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 or cancer relapse. Despite treatment with high-dose prednisone, 40% to 50% of unrelated allogeneic tissue transplant patients will develop GvHD.

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). Along with its potent anti-inflammatory profile, research shows that p-AAT has 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 p-AAT 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 control (see Figure-1 below). Furthermore, p-AAT treatment reduced the expansion of alloreactive T effector cells but enhanced the recovery of T regulatory T cells, thus reducing the pathological harmful response. Treatment with p-AAT also resulted in increased production of anti-inflammatory cytokines, IL-10 (Tawara I et al, 2012).

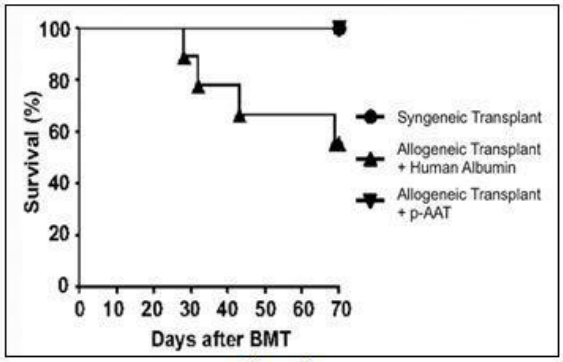


Figure-1

We see a number of key points and preclinical findings that support the utility of p-AAT in the treatment and prevention of GvHD:

- ✓ Data shows a loss of A1AT during intestinal GvHD in children (Hagen et al, 2011).
- ✓ Significant scientific evidence exists to support the involvement of IL-1 during the progression of GvHD, and treatment with IL-1 receptor antagonists have been shown to slow progression and reduce disease severity (Dinarello CA, 2011; Antin et al, 1994).
- ✓ Animal model data shows p-AAT provides a clear benefit to immune profile, body weight, and survival (Marcondes et al, 2011; Tawara et al, 2012).

We see GvHD as a potential enormous opportunity. According to data from the Center for International Blood and Marrow Transplant, the incidence of acute GvHD ranges from 26% to 34% in recipients of full matched sibling donor grafts, to 42% to 52% in recipients of matched unrelated donor grafts. The incidence is directly related to the degree of human leukocyte antigens (HLA) disparity. The median onset of acute GvHD is typically 21 to 25 days after transplantation. Chronic GvHD ranges in incidence from 30% in recipients of fully histocompatible transplants to 60% to 70% in recipients of mismatched hematopoietic cells or hematopoietic cells from an unrelated donor. The median time of diagnosis of chronic GvHD is 4.5 months after HLA-identical sibling transplantation and four months after unrelated donor transplantation.

GvHD is listed as a "rare disease" by the Office of Rare Diseases (ORD) of the NIH. Given the number of Americans that undergo a transplant per year, we estimate there are approximately 7,500 cases of acute GvHD per year in the U.S. According to data from the NIH, 30% to 50% of patients respond to high doses of corticosteroids, such as methylprednisolone. This would place the refractory-treatment population in the U.S. at approximately 4,000 patients per year. Including Europe and the rest of the developed world, Omni Bio is probably looking at an addressable patient population of approximately 10,000 individuals.

This represents a very attractive label expansion opportunity for the four big manufacturers of p-AAT: Grifols, Baxter, CSL Behring, and Kamada. It essentially doubles the market from the $600-700 million now with AATD. With positive proof-of-concept data, we think Omni Bio can strike a deal with one of these major players. A deal with Grifols, the market leader, makes sense from a defensive standpoint. Grifols could acquire the rights to use p-AAT in GvHD from Omni Bio to protect its market-leading position. A deal with Baxter or Kamada makes sense if one of these smaller players wants to make an offensive move on the market leader.

Above we noted an active pilot study being conducted by Dr. H. Joachim Deeg at the Fred Hutchinson Cancer Research Center (NCT01523821). Based on conversations with management, we believe initial data from this program will become available at the American Society of Hematology (ASH) meeting in December 2014.

…In post-MI…

Significant interest exists in seeking to reduce inflammatory response during and following an acute myocardial injury. Data shows that p-AAT may play a role in preserving tissue integrity and reducing scar formation thanks to its potent anti-inflammatory characteristic. Evidence also shows protection from ischemia reperfusion injury in renal mouse models by p-AAT (Daemen et al, 2000).

Work published in the Journal of Molecular Cell Cardiology shows the effects of exogenously administered p-AAT 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 p-AAT or albumin at reperfusion. Infarct size was evaluated after 1 and 7 days. AAT-treated mice had significantly smaller infarct sizes (-30% day 1 and -55% day 7) compared with mice treated with albumin (Figure-2). AAT-treated mice also exhibited a >90% 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).

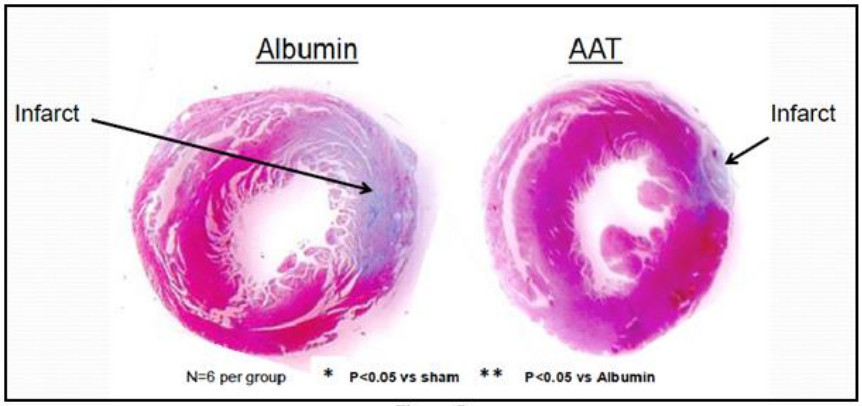


Figure-2

Above we noted an active pilot study being conducted by Dr. Antonio Abbate, MD, PhD of the Virginia Commonwealth University (NCT01936896). ClinicalTrials.gov lists this study as completed as of August 19, 2014. Based on conversations with management, we believe initial data from this study will be available at the American Heart Association (AHA) meeting in November 2014.

…In Diabetes…

Type-1 diabetes is an autoimmune disorder resulting in the destruction of insulin-producing B-cells of the pancreas. The lack of insulin leads to increased blood and urine glucose. Type-1 diabetes must be treated by injection (or inhalation) of insulin. Protection of the islet cells is the obvious target for new therapeutic methods of treatment, and new clinical studies are focusing on stopping the T-cell-mediated autoimmune attack. Anti-inflammatory agents, such as anti-CD3 antibodies, have shown encouraging signs of efficacy. Data show that blockage of the inflammatory molecule IL-1, a direct B-cell toxic agent, results in reduced insulin dependence when tested on children with early-stage disease (Sumpter et al, 2011).

Research on non-obese diabetic (NOD) mice show that inflammatory mechanisms trigger insulitis, insulin resistance, faulty insulin signaling, and the loss of immune tolerance to islets. The data demonstrate that treatment with p-AAT, an agent that dampens inflammation, does not directly inhibit T cell activation, ablates invasive insulitis, and restores euglycemia (normal blood glucose levels), immune tolerance to B cells, normal insulin signaling, and insulin responsiveness in NOD mice with recent-onset type-1 diabetes through favorable changes in the inflammation cascade (Figure-3) (Koulmanda et al., 2008).

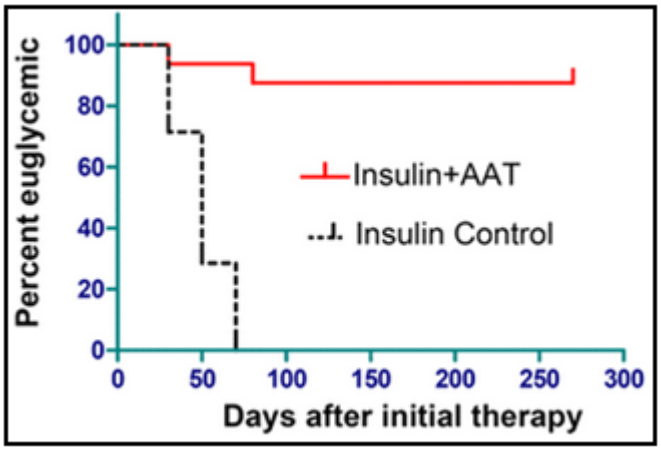


Figure-3

There are essentially three key points that lead us to believe p-AAT may have therapeutic utility in Type-1 diabetes.

- ✓ Firstly, non-functional circulating AAT has been shown to exist in most individuals with Type-1 diabetes (Yaghmaei et al, 2009), suggesting that AAT levels may play a role in disease progression.
- ✓ Secondly, multiple preclinical animal models also show consistent benefit of AAT on protection of insulin producing islet B-cells (Kamada, Ltd., November 2012; Koulmanda et al., 2008).
- ✓ Thirdly, AAT is endogenously produced under inflammatory stimuli by human islet cells (Bosco et al, 2005). These three conclusions strengthen the case for testing p-AAT in human clinical trials in children recently diagnosed with Type-1 diabetes.

On April 30, 2014, Omni Bio announce publication of a Phase 1 pilot study sponsored by the University of Colorado, Barbara Davis Center for Childhood Diabetes under the direction of Dr. Peter Gottlieb. Published results show that AAT may have a beneficial effect on Type-1 diabetes in recently diagnosed patients through the down-modulation of IL-1β and other pro-inflammatory cytokines. IL-1β is known to be harmful to insulin-producing cells. The authors further concluded that targeting inflammation with inhibitors of the innate immune system, such as p-AAT, might represent an efficient therapeutic strategy for disease prevention.

Key findings of the 12-patient, 8 week study (80 mg/kg per week) were as follows:

- ✓ AAT led to increased or unchanged levels of C-peptide responses in four patients during 18 months' follow up.
- ✓ The total content of TLR4-induced cellular IL-1β in monocytes at 12 months among all patients was reduced 3-fold compared to baseline ($p<0.05$).
- ✓ Monocyte production of IL-1β was reduced from 82% at baseline to 42% at 12 months.
- ✓ Similar reductions were seen using TLR7/8 and TLR3 agonists in monocytes and myeloid dendritic cells (mDCs).
- ✓ Unexpectedly, the reduction in cellular IL-1β was observed at 9-12 months post-treatment but not in a cohort of untreated diabetics.
- ✓ Improved islet cell function in the four responder patients correlated with the reduced monocytes and mDC production levels of IL-1β ($p<0.04$ and $p<0.02$, respectively).
- ✓ No significant adverse effects were reported in the study

Data from the two Phase 2 studies being conducted by Kamada & Baxter's using Glassia® (NCT02005848) and by Grifols using Prolastin-C® (NCT02093221) should offer data in 2015 or 2016.

Expanding The Management Team

On May 1, 2014, Omni Bio announced the appointment of Dr. Bruce Forrest to the position of Chief Development Officer (CDO). Omni Bio has retained Dr. Forrest for the previous 18 months to provide the company with scientific strategy and business development support. As Chief Development Officer, Dr. Forrest will now oversee implementation of the development plan for Omni Bio's Fc-AAT molecule, which will include interface and leadership responsibilities for work being performed by the company's cell line optimization and product manufacturing partner Gallus BioPharmaceuticals, Inc.

Dr. Forrest has over 25 years of industry experience in pharmaceutical development of vaccines and biopharmaceuticals. He was previously the Senior Vice President in Vaccines R&D at Wyeth Pharmaceuticals where he was responsible for clinical and developmental activities for all late phase programs. From 2004-2008 Dr. Forrest was the head of Wyeth's R&D organization in Japan where he oversaw the successful regulatory approval of several new drug entities. Dr. Forrest's background and wealth of experience should prove to be very helpful for Omni Bio as they continue the development of Fc-AAT.

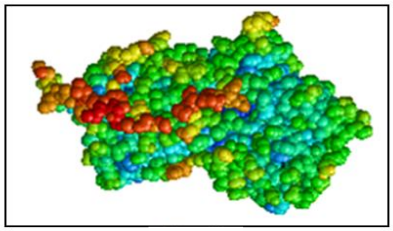
On August 14, 2014, Omni Bio announced the appointment of Jack Riccardi to the position of Chief Financial Officer (CFO). According to the press release, Mr. Riccardi brings more than 30 years of financial leadership experience. He was formerly CFO of ADial Pharmaceuticals and served most recently as CFO and Partner at Green Street Ventures. Mr. Riccardi holds a BS in Accounting from Pennsylvania State University, an MBA from the Wharton School of Business at the University of Pennsylvania, and an MS in Operations Science from London School of Economics and Political Science.

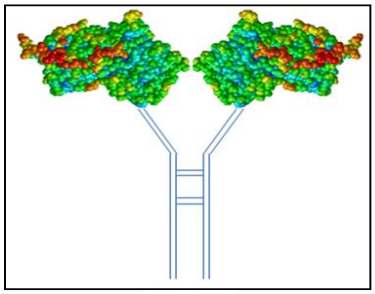
Fc-AAT – A Game Changer

Despite all the promise and potential for p-AAT, 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 AAT replacement therapy products are derived from pool human plasma. There are four manufacturers of p-AAT worldwide, Grifols, Baxter, CSL Behring, and Kamada. 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 AATD 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, which they refer to internally as Fc-AAT-2, 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).





Plasma-Derived A1AT (p-AAT) = 52 kDa
Recombinant Fc-AAT-2 = 174 kDa

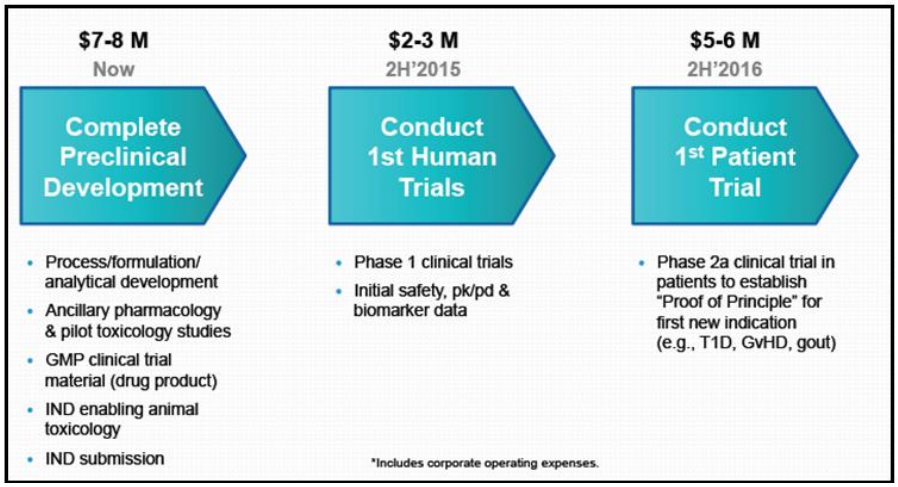
Omni Bio believes the technology used to construct Fc-AAT is similar to that already used to create highly successful drugs for human application, such as Amgen / Pfizer's Enbrel. Enbrel was jointly developed by Immunex (acquired by Amgen) and Wyeth (acquired by Pfizer). Omni Bio's CEO, Dr. Bruce Schneider, was a senior R&D executive at Wyeth and was intimately involved in the development of Enbrel. Through our conversations with Dr. Schneider, he tells us that many of the experiences with Enbrel are transferrable to the development of Omni Bio's Fc-AAT molecule. Other Fc drugs include Regeneron's Zaltrap, Biogen's Alprolix and Eloctate, and Eli Lilly's dulaglutide.

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 AAT molecule into new areas of therapeutic treatment such as Type-1 diabetes, GvHD, and myocardia infarction. Currently, all available supply of p-AAT is delegated for patients with AATD. 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. Plus, the drug has a simple and low-cost manufacturing that would allow for dramatically improved supply and economics to patients.

Conclusion

We really like the concept of Fc-AAT and Omni Bio's business strategy with cornering the intellectual property around the existing p-AAT products. We think there is potential for huge returns here. 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 June 2014 investor presentation.



Omni Bio needs at least another $10 million between now and mid-2016 to see this strategy through. That includes another $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 $0.20 per share. As of today, the basic share count is around 38.9 million, but the (reasonable) fully-diluted number is closer to 76 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 $0.20 per share, it would take issuing 50 million shares, plus most likely another 50 million warrants. Take note, on April 24, 2014, the company retained investment bank Dawson James to advise and assist the company in its capital market activities.

Unfortunately, that 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? That's not unrealistic to believe. Keep in mind data on the use of p-AAT post-MI is coming in November 2014 at AHA and data on the use in GvHD is coming at ASH in December 2014. Exciting data on either indication could be the prelude to a sub-licensing deal. In the meantime, we'll be keeping a close watch on the preclinical development of Fc-AAT.

PROJECTED FINANCIALS

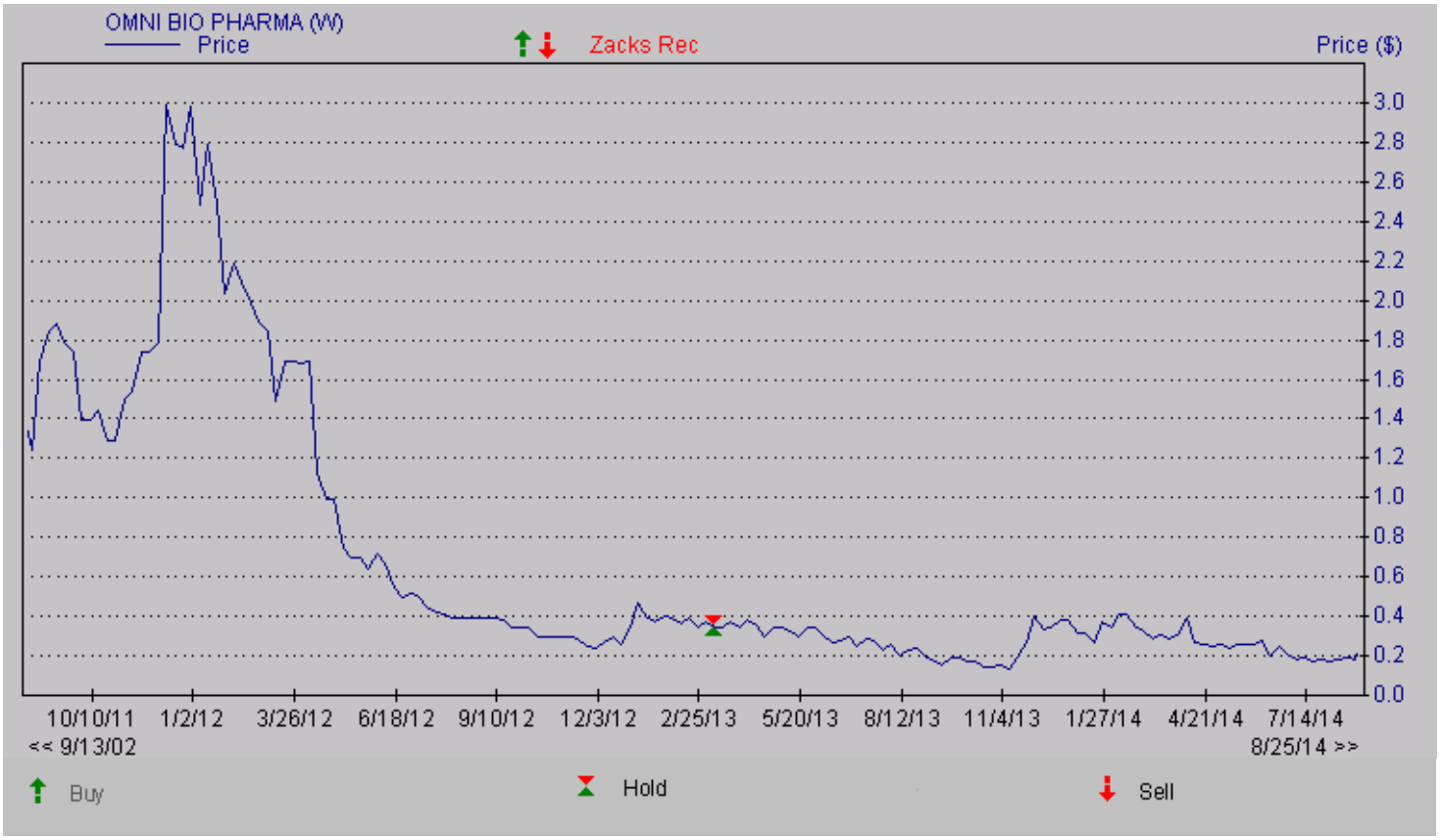
Omni Bio Pharmaceuticals, Inc.
Income Statement

Omni Bio Pharma	FY'14A	June-14A	Sept-14E	Dec-14E	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	$0.300	$0.400	$0.500	$1.420	$5.000
% R&D	-	-	-	-	-	-	-
SG&A	$1.750	$0.313	$0.350	$0.375	$0.400	$1.438	$2.500
% SG&A	-	-	-	-	-	-	-
Operating Income	**($1.912)**	**($0.533)**	**($0.650)**	**($0.775)**	**($0.900)**	**($2.858)**	**($7.500)**
Operating Margin	-	-	-	-	-	-	-
Interest & Other Income	($0.604)	($0.300)	($0.100)	($0.100)	($0.100)	($0.600)	($0.250)
Pre-Tax Income	($2.517)	($0.834)	($0.750)	($0.755)	($1.120)	($3.459)	($7.750)
Taxes & Other	$0	$0	$0	$0	$0	$0	$0
Tax Rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	($2.517)	($0.834)	($0.750)	($0.755)	($1.120)	($3.459)	($7.750)
Net Margin	-					-	-
Reported EPS	**($0.07)**	**($0.02)**	**($0.02)**	**($0.02)**	**($0.02)**	**($0.08)**	**($0.16)**
YOY Growth	-	-	-	-	-	-	-
Basic Shares Outstanding	37.3	38.9	41.0	43.0	45.0	42.0	50.0

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

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HISTORICAL ZACKS RECOMMENDATIONS



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