Zacks Small-Cap Research

October 30, 2014
Jason Napodano, CFA
312-265-9421
jnapodano@zacks.com

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

Matinas BioPharma Holdings, Inc. (MTNB-OTCQB)

Initiating Coverage of Matinas BioPharma – Pioneering Designer Omega-3 Fatty Acids for Cardiovascular Protection…

Current Recommendation	**Outperform**
Prior Recommendation	N/A
Date of Last Change	10/28/2014
Current Price (10/29/14)	$0.60
Target Price	**$1.25**

INITIATION

We are initiating coverage of Matinas BioPharma Holdings, Inc, with an Outperform rating and a price target of $1.25. Led by senior management and scientists who developed the pioneering omega-3 fatty acid product Lovaza and sold it GlaxoSmithKline for $1.65B, Matinas BioPharma is developing a 3rd generation, designer omega-3 fish oil product with enhanced properties, including the potential for better control of triglyceride and cholesterol levels, anti-coagulant activity, and synergy with statins. The company expects to complete first in-human PK/PD studies in the next several months, paving the way for a pivotal Phase 3 trial in 2015. We expect the product to have superior lipid lowering properties compared to existing omega-3 products. Should the company succeed in demonstrating improvement in cardiovascular outcomes, we believe this could be a blockbuster product.

SUMMARY DATA

52-Week High	**$0.90**
52-Week Low	**$0.55**
One-Year Return (%)	**N/A**
Beta	**N/A**
Average Daily Volume (sh)	**2,864**
Shares Outstanding (mil)	**32**
Market Capitalization ($mil)	**$22**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**5**
Insider Ownership (%)	**35**
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 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2013	0 A	0 A	0 A	0 A	0 A
2014	0 A	0 A	0 E	0 E	0 E
2015					0 E
2016					0 E

Earnings per Share
(EPS is report figures)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2013					-$0.20 A
2014	-$0.07 A	-$0.08 A	-$0.07 E	-$0.09 E	-$0.30 E
2015					-$0.23 E
2016					-$0.17 E

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

WHAT'S NEW

Initiating Coverage

We are initiating coverage Matinas BioPharma Holdings, Inc (MTNB-OTCQB) with an 'Outperform' rating and a $1.25 per share price target.

Matinas is developing MAT9001, a proprietary prescription grade omega-3 fatty acid composition, comprised of a complex mixture of omega-3 fatty acids, predominantly eicosapentaenoic acid ("EPA"), and docosapentaenoic acid ("DPA"), a rare and potent omega-3 fatty acid. The product also contains several other omega-3 fatty acids, including relatively small amounts of docosahexaenoic acid ("DHA"). The initially targeted indication is severe hypertriglyceridemia, for which Matinas expects to initiate a Phase 3 clinical trial in Q3 2015. Severe hypertriglyceridemia is a condition in which patients have elevated serum triglycerides (TG ≥500 mg/dl) that put them at high risk of pancreatitis, and is recognized as an independent risk factor for cardiovascular disease. A second potential indication is the treatment of patients with mixed hyperlipidemia (triglycerides > 200 mg/dL in spite of ongoing statin treatment). The addressable treatment populations for these two indications in the United States are approximately 5 million and 30 to 35 million, respectively.

Prescription omega-3 fatty acids are historically valued for their ability to potently reduce triglyceride levels with a very clean side effect profile. The market is currently dominated by Lovaza®, a mixture of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) ethyl esters that is approved for the treatment of both severe hypertriglyceridemia (>500 mg/dL) and for the treatment of mixed hyperlipidemia in combination with a statin. Lovaza sales in 2013 were $917M at GlaxoSmithKline. Other competitors in the space include Vascepa® (EPA ethyl ester) by Amarin Pharmaceuticals and Epanova® (EPA and DHA free acids) by AstraZeneca, each of which are currently labeled for treatment of severe hypertriglyceridemia only. Vascepa, approved in 2012, generated $23M in revenue in 2013 and $24M in revenue in the first half of 2014. It is currently being examined for its ability to reduce cardiovascular events in an 8,000 patient, 7-year cardiovascular outcomes study. Competing non-omega-3 products that are used in these indications include fibrates (Tricor®, gemfibrozil) and extended release niacin (Niaspan®) sold by AbbVie Pharmaceuticals.

The unique properties of DPA confer unique properties on MAT9001 that potentially offer significant competitive advantages. Unlike the current approved therapies in this product category, many of which have been repurposed following clinical failures in their originally intended indications, MAT9001 was specifically designed and developed to treat hypertriglyceridemia and mixed dyslipidemia (described below). Based on publicly available preclinical and clinical data associated with one of the key components of MAT9001, the product has the potential to exceed the performance of available omega-3 fatty acid products by:

- ✓ Better controlling cholesterol, potentially decreasing low density lipoprotein (LDL) levels.
- ✓ Better controlling triglycerides.
- ✓ Providing anti-coagulant effects.

In addition, MAT9001 contains very low levels of DHA, a component of some competing omega fatty acid products that has been shown to increase LDL cholesterol. This differentiated profile is expected to provide significant commercial advantages in a potential market of up to 40 million people in the U.S.

INVESTMENT THESIS

Matinas BioPharma (MTNB) is developing MAT9001, a novel prescription omega-3 fatty acid treatment for familial hypertriglyceridemia (high serum triglycerides) and mixed hyperlipidemia (high serum triglycerides and high cholesterol). Unlike currently marketed omega-3 fatty acid mixtures that were repurposed after failing in other indications, MAT9001 is a designer omega-3 fatty acid mixture that fully exploits recent clinical and preclinical findings in omega-3 fatty acid pharmacology. Specifically, preclinical and clinical findings suggest that MAT9001 has the potential to outperform existing omega-3 prescription fatty acid products such as GlaxoSmithKline's $900M+ per year Lovaza by:

- ✓ Providing better control of triglycerides
- ✓ By synergistically interacting with statins to lower LDL cholesterol
- ✓ Through anti-coagulant effects and prophylaxis of acute ischemic events

MAT9001 differs from currently marketed prescription omega-3 fatty acid products by incorporation of N-3 docosapentaenoic acid (DPA), a rare omega-3 fatty acid whose properties have historically been difficult to elucidate due to its low natural abundance in marine sources containing large numbers of structurally related acids. *In vitro, in vivo*, and clinical studies have demonstrated important differences in the pharmacological properties of omega-3 fatty acids in spite of their structural similarity. DPA lacks the LDL cholesterol-raising properties of docosahexaeneoic acid (DHA), found in Glaxo's Lovaza. Compared to eicosapentaenoic acid (EPA), found in both Lovaza and in Amarin Pharmaceuticals 96% EPA product Vascepa®, DPA shows more potent triglyceride and LDL cholesterol lowering ability. It also exhibits anti-coagulant activity not seen for EPA or DHA.

Matinas is led by executives and senior scientists with deep experience in the omega-3 fatty acid therapeutics area. Most of the founding senior management played key roles at Reliant Pharmaceuticals, and in the development and / or launch of Lovaza, which formed the basis of GlaxoSmithKline's $1.65B takeover of Reliant Pharmaceuticals in November 2007.

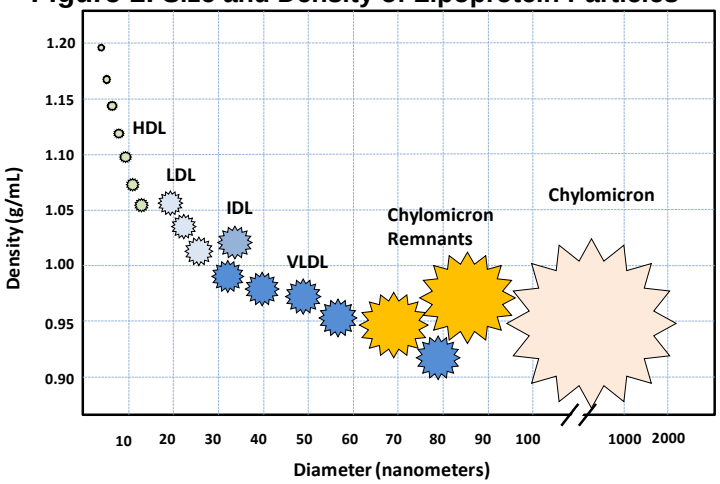
Matinas management believes the market for treatment of severe triglyceridemia in the United States is 5 million patients. At current pricing for dyslipidemia treatments ($200/month), this corresponds to an addressable market of $12 billion. The mixed hyperlipidemia patient population is 7 times as large. Given the limited efficacy of current treatment options, we believe there is considerable commercial opportunity for new agents with superior clinical profiles.

The Role of Lipoproteins in Normal Metabolism

Nutrients such as cholesterol, long chain fatty acids, and fat-soluble vitamins are critical to the growth and function of the body's cells, but being very poorly soluble in water, cannot be delivered to (or when present in excess, removed from) the tissues without special handling. These substances are packaged into particles for transport through the bloodstream (Daniels, TF, 2009). As the particles contain both lipids and proteins, they are commonly called lipoprotein particles. Lipoprotein particles play an essential role in the absorption of dietary lipids; the transport of lipids from the liver (where many are synthesized) to the peripheral tissues, and the transport of cholesterol from the tissues to the liver.

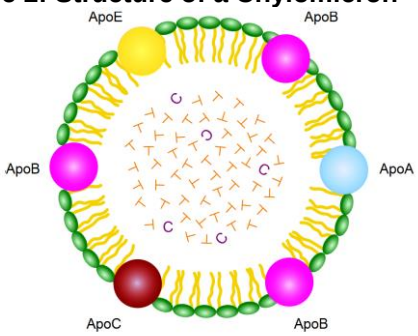
Lipoproteins vary widely in size and function, but all lipoproteins consist of a core of hydrophobic lipids (cholesterol esters and triglycerides) surrounded by an envelope of hydrophilic lipids (phospholipids and free cholesterol) and proteins. The density of each particle type correlates with its size, and is determined by the ratio of protein to lipid. There are five main types of lipoprotein particle that differ in their size and the nature of the proteins on their surface. The proteins on the surface of each particle interact with receptors in the liver and peripheral tissues to ensure that each particle type interacts with tissues selectively and thereby fulfills its particular function.

Figure 1: Size and Density of Lipoprotein Particles



1) **Chylomicrons** are large lipoprotein particles that are formed in the intestine and serve to carry dietary lipids through the blood stream to the tissues. They contain a very high ratio of triglycerides relative to cholesterol. Upon arrival in the peripheral tissues, the proteins on the surface of the chylomicrons bind to specific receptors in fat and muscle tissue. The triglycerides they carry are hydrolyzed to fatty acids that are then transported into the tissue, where they are stored for future use in energy production.

Figure 2: Structure of a Chylomicron



Abbreviations: T = triglyceride; C = cholesterol; ApoB, ApoC and ApoE are proteins

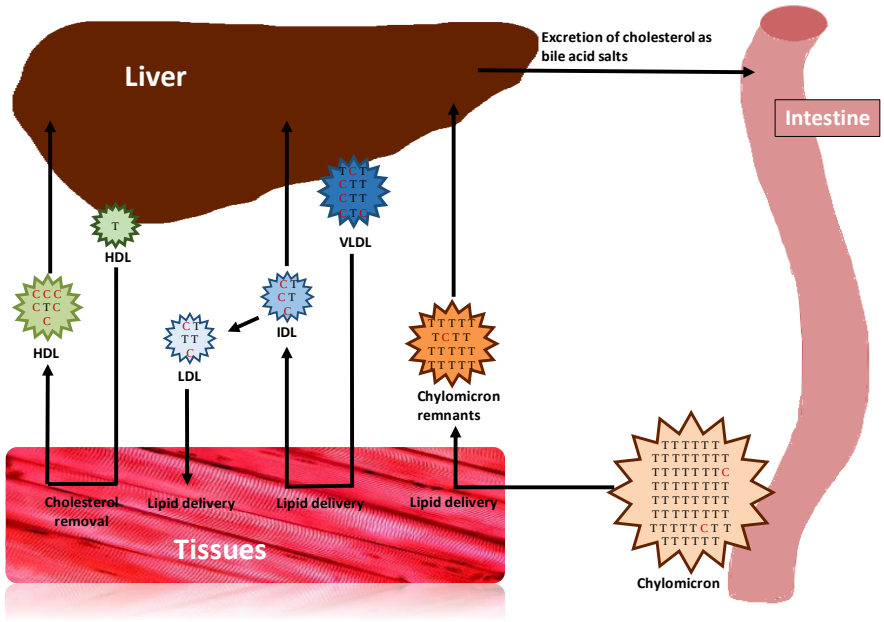
2) The chylomicron shrinks as it delivers its triglyceride payload into the tissues, and with the loss of triglycerides the fraction of its content that is cholesterol increases. These intermediate sized particles are sometimes called **Chylomicron Remnant**s. These gradually transfer their phospholipid and protein content to HDL particles. After this they eventually get absorbed by the liver where the remaining triglycerides and cholesterol may be recycled or excreted via the bile into the intestines.

3) **Very Low Density Lipoprotein (VLDL)** also delivers lipids to the tissues, but in this case it is lipids synthesized or stored in the liver rather than those recently absorbed from the diet. VLDL is the principle carrier of triglycerides in the bloodstream. In people consuming a high fat diet, cholesterol may substitute for triglycerides as the main component of the lipid core. Like chylomicrons, VLDL particles shrink as they give up their payload to the peripheral tissues. Some of them eventually transfer their surface proteins and phospholipids to HDL particles, becoming **IDL Particles** and then are absorbed by the liver.

4) Alternatively, the depleted VLDL particles can be processed by an enzyme called LPL to form **Low Density Lipoprotein (LDL)** particles. LDL is the primary plasma cholesterol carrier, because these particles are relatively abundant and because they have a very high ratio of cholesterol to triglycerides. As the concentration of LDL correlates strongly with the risk of heart disease, LDL is often referred to as "bad cholesterol". It serves as a source of cholesterol to most of the body. High levels of LDL are associated with the formation of atherosclerotic plaques that cause heart attacks and strokes. In contrast to chylomicrons and VLDL particles, LDL particles are completely absorbed and degraded by the tissues that they deliver lipids to.

5) **High density lipoproteins (HDL)** are lipoprotein particles produced by the liver that scavenge excess cholesterol and triglycerides from the tissues and from other lipoprotein particles. The excess is carried to the liver, where the cholesterol is converted to bile salts and excreted in the bile. This housecleaning function underlies the inverse relationship between HDL levels and cardiovascular risk. In contrast to chylomicrons and VLDL particles, which give up their contents and then get absorbed by the liver, HDL particles grow larger over time and are taken up by the liver when they are filled to capacity.

Figure 3: Key Lipoprotein Particles and Their Role in Lipid Transport



Source: Zacks Investment Research / John Tucker, PhD

Lipid Disorders and Their Health Consequences

Excessive or unbalanced levels of lipoprotein particles in the bloodstream are known as dyslipidemias. They may involve increased levels of serum cholesterol (hypercholesterolemia), increased levels of triglycerides (hypertriglyceridemia), or increased levels of both (mixed hyperlipidemia). High serum levels of cholesterol and triglycerides are strongly correlated with increased risk of atherosclerosis and heart disease; lifestyle changes and pharmacological management of serum lipid levels are an important part of cardiovascular disease prevention. In addition, a high ratio of LDL to HDL cholesterol has been associated with increased cardiovascular risk (Nelson RH, et al, 2013).

Owing to their potent triglyceride-lowering capabilities, omega-3 fatty acids are most widely used for lowering triglyceride levels in patients with severe hypertriglyceridemia (triglycerides > 500 mg/dL), or in patients with moderate triglyceridemia that persists in spite of being treated with a statin (mixed hyperlipidemia).

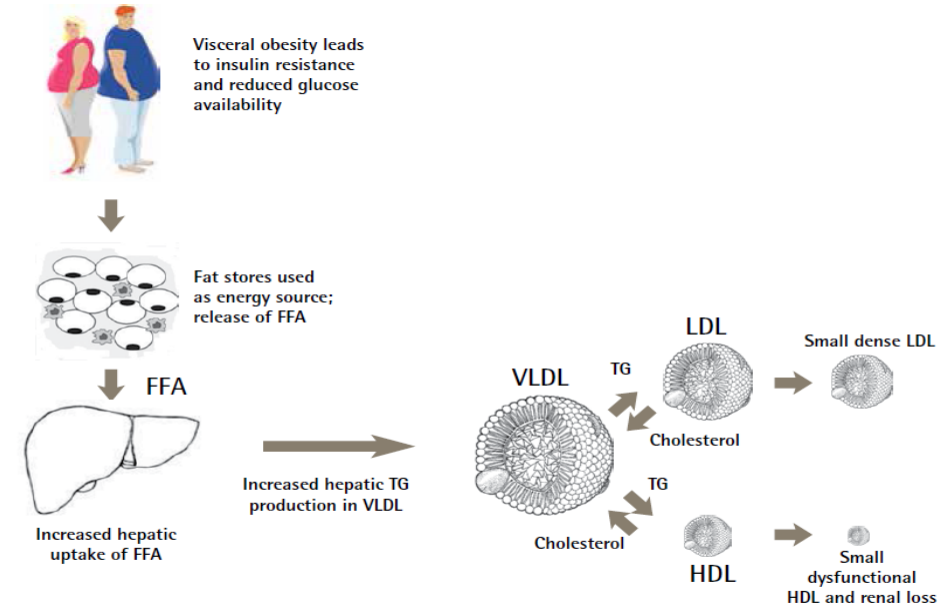
Causes of Dyslipidemia

…Obesity and Type 2 Diabetes…

Obesity and Type 2 diabetes are by far the most common cause of dyslipidemia in Western societies (source: NHLDI/NIH, 2002). These conditions lead to a state of insulin resistance in which the tissues fail to properly take up and process serum glucose to meet their energy needs. With reduced glucose metabolism, fatty acid metabolism increases in order to provide energy needed by the tissues. In response to this increased tissue demand for fatty acids, the liver produces large amounts of VLDL and LDL particles. The LDL particles are smaller and denser than those found in healthy people, contain a higher ratio of triglycerides relative to cholesterol, and are intensely atherogenic because they penetrate into the blood vessel walls especially well.

The combination of insulin resistance, increased fatty acid utilization, and highly atherogenic serum lipoprotein pattern shared by diabetics and the obese is commonly referred to as “metabolic syndrome”, as illustrated in Figure 4 below.

Figure 4: Metabolic Syndrome



FFA—free fatty acids, HDL—high-density lipoprotein, LDL—low-density lipoprotein, TG—triglyceride, VLDL—very low-density lipoprotein.

Diagnosis requires the presence of three of the five markers in Table 1 below. Diabetics and others with metabolic syndrome commonly exhibit mixed hyperlipidemia with high triglyceride and LDL levels. Statin monotherapy is frequently insufficient to reduce triglyceride levels to recommended levels of <150 mg/dL, and omega-3 fatty acid products are commonly used for further triglyceride reduction.

Table 1: Diagnostic Criteria for Metabolic Syndrome.

MEASURE	DIAGNOSTIC CUT POINT
Waist Circumference	>90-94 cm (men); >80 cm women
Triglycerides	>150 mg/dL
HDL Cholesterol	<40 mg/dL
Blood Pressure	Systolic >130 mm or diastolic >85 mm
Fasting Glucose	>100 g/dL

…Genetic Causes…

Isolated severe hypertriglyceridemia is commonly caused by a genetic syndrome called *Familial hypertriglyceridemia.* Its prevalence in Western societies is about one in 500 persons (source: CDC). It is characterized by elevated levels of triglycerides (250-1000 mg/dL), normal or only slightly elevated cholesterol levels (<250 mg/dL), and low HDL cholesterol. The elevated triglyceride levels are due to excess VLDL and/or chylomicrons. Interestingly, patients with familial hypertriglyceridemia are not found to be at exceptionally high risk of cardiovascular disease, but their high levels of serum triglycerides require treatment to avoid the risk of pancreatitis. Treatment normally consists of dietary interventions, fibrate drugs, or omega-3 fatty acids (see below).

- *Familial combined hyperlipidemia* is typically characterized by moderate elevations in plasma triglycerides (mainly VLDL), "bad cholesterol" (LDL), and reduced levels of HDL cholesterol. Approximately 20% of patients who develop coronary artery disease by age 60 have familial combined hyperlipidemia.

- *Familial hypercholesterolemia* is caused by defects in the protein responsible for directing the uptake of IDL particles into the liver, ultimately leading to very high LDL levels. This syndrome is less important to our current discussion as it is not widely treated with omega-3 products.

Treatment Guidelines

…NCEP-ATP3 - Cholesterol Guidelines…

In 2001, the United States National Heart, Lung, and Blood Institute organized an expert panel on the detection, evaluation, and treatment of high blood cholesterol in adults. The panel set threshold values for the pharmacological treatment of dyslipidemias. These guidelines are often referred to by the acronym NCEP-ATP3 (National Cholesterol Education Project - Adult Treatment Panel 3). A minor update of the guidelines was performed in 2004. Serum lipid levels as classified by NCEP-ATP3 are shown in Table 2.

Table 2: ATP 3 Classification of Serum Lipid Levels

LDL Cholesterol		Triglycerides	
< 100	Normal	<150	Normal
100 - 129	Near optimal	150 - 199	Borderline high
130 - 159	Borderline high	200-499	High
≥ 190	Very high	≥ 500	Very high
HDL Cholesterol			
< 40	Low		
≥ 60	High		

NCEP-ATP3 treatment guidelines are based on a stratification of patients according to underlying non-LDL cholesterol-related risk factors. For each risk class there is a serum LDL cholesterol goal value, above which lifestyle interventions including increased exercise, weight loss, and improved diet is recommended. For the highest risk category, pharmacological treatment is initiated simultaneously with lifestyle interventions. Recommended agents include statins, fibrates, nicotinic acid, and bile acid sequesterants.

In lower risk categories, the patient is further evaluated after a 3 month trial of lifestyle intervention. At this point the patient is evaluated for pharmacological treatment of high LDL cholesterol levels, high triglyceride levels, and metabolic syndrome. Metabolic syndrome is diagnosed if three of the five signs shown in Table 1 above are present, and is treated as needed with lifestyle interventions. Other cardiovascular risk factors are treated as needed, including the use of aspirin to reduce pro-thrombotic state, the use of antihypertensive drugs, and the treatment of hypertriglyceridemia.

Table 3: Treatment Guidelines from NCEP-ATP3

10 year CHD risk	Characteristics	LDL Level to Begin Lifestyle Interventions (mg/dL)	LDL Level to Begin Drug Treatment (mg/dL)
Low	• 0 or 1 risk factors (calculated ten year CHD risk <10%)	160	190 mg/mL
Medium	• 2 or more risk factors, *and* ○ Calculated ten year risk of CHD <10%, *or* ○ Calculated ten year risk of CHD 10% to 20%	130	130 if risk >10% 160 if risk 10-20%
High	• Calculated ten year risk of CHD >20%, *or* • Presence of established coronary artery or other atherosclerotic disease, *or* • Diabetic	100	130

…Triglyceride Guidelines…

The NCEP-ATP3 guidelines explicitly refer to elevated triglycerides as an independent risk factor for cardiovascular disease. Hypertriglyceridemia treatment goals from NCEP-ATP3 include the following:

- ✓ In all patients with triglyceride levels >150 mg/dL, the primary goal of therapy is to reduce LDL levels, ideally through lifestyle interventions.
- ✓ In those with triglyceride levels above 200 mg/dL even after the LDL goal is reached, total non-HDL cholesterol goals are set at 30 mg/dL greater than the LDL goals in Table 3.
- ✓ In those with triglyceride levels 200-449 mg/dL, consider intensifying anti-LDL therapy or adding a second drug (a fibrate or nicotinic acid) to lower VLDL
- ✓ In those with triglyceride levels >500 mg/dL, prioritize lowering triglycerides to minimize the risk of pancreatitis, using lifestyle interventions and LDL lowering therapy and/or nicotinic acid or fibrates

Thus the NCEP-ATP3 guideline explicitly recommend the treatment of high triglyceride levels, whether they occur in isolation in severe hypertriglyceridemia or in combination with high cholesterol levels in those with mixed hyperlipidemia.

Guidelines from the European Society for Cardiology, the National Lipid Association, and the American Diabetes Association

…Cholesterol Guidelines…

More recently issued guidelines from other professional medical societies such as the European Society for Cardiology (ESC) (European Heart Journal, 2011; Atherosclerosis, 2011), the American Diabetes Association (ADA) (Diabetes Care Volume 37, Supplement 1, January 2014), and the National Lipid Association (NLA) (source) represent relatively modest variations from the NCEP-ATP 3 paradigm. In each case patients are stratified into risk groups based on non-LDL risk factors such as overt cardiovascular disease, smoking, age, and diabetes.

Target LDL cholesterol values are assigned to each group according to its calculated 10 year cardiovascular risk, with higher risk groups receiving lower target LDL values. Patients are treated with lifestyle interventions and/or pharmacologically, in most cases using statins as first line therapy.

…Triglyceride Guidelines…

All three guidelines recommend that triglyceride lowering be given first priority in those with severed hypertriglyceridemia in order to reduce the risk of pancreatitis. The ESC guidelines are broadly supportive of combination therapy with a statin plus nicotinic acid, a fibrate, or omega-3 fatty acid in those with low HDL-C or high triglycerides that persist despite statin treatment. The ADA generally recommends against combination treatments, stating that the risk of side effects outweighs any benefit. The NLP is generally strongly supportive of reducing serum triglycerides to target irrespective of whether LDL targets have been reached.

The 2013 American Heart Association Guidelines

Based in part on calls by the United States Institute of Medicine to align treatment guidelines more strictly around the paradigm of evidence-based medicine, the American Heart Association and the American College of Cardiology (AHA/ACC) released 2013 treatment guidelines (link) that are a radical departure from those of NCEP-ATP3. These guidelines have been somewhat controversial as they make minimal use of cholesterol levels to determine which patients should undergo lipid lowering therapy, do not set serum lipid treatment targets, and make little provision for treatment of low HDL or high triglyceride levels.

Instead, they are aligned around strictly following evidence from randomized clinical trials, most of which were performed by pharmaceutical companies for the purpose of demonstrating that their products can reduce cardiovascular events and mortality. Since the pharmaceutical trial entry criteria were based more on estimated risk calculated from lifestyle factors and cardiovascular history than on baseline cholesterol levels, a strict evidence-based treatment paradigm necessarily selects patients for treatment based on these same criteria. Similarly, as the pharmaceutical trials used fixed drug doses, mainly of statins, the new AHA guidelines contain few provisions for modifying dose to achieve specified LDL targets, for treating hypertriglyceridemia, or for treating patients with any drug class other than a statin.

The AHA/ACC guidelines recommend that all patients be encouraged to adhere to a healthy lifestyle, including regular exercise, avoidance of smoking, and a diet that is low in fat, red meats, and sugars. Statin therapy is recommended for four groups:

- ✓ All patients with established cardiovascular disease
- ✓ All patients with LDL cholesterol $\geq$ 190 mg/dL
- ✓ Diabetics over 40 years of age with LDL cholesterol $\geq$ 70 mg/dL
- ✓ In patients without established cardiovascular disease or diabetes who have an estimated 10 year risk of cardiovascular disease >7.5%

For those with an initial LDL cholesterol level in excess of 190 mg/dL, a second LDL lowering agent such as niacin, fish oil, or a fibrate may be added to further reduce LDL levels. An LDL cholesterol level reduction of at least 50% is recommended in this group.

For patients with high triglycerides and healthy cholesterol levels, medications other than fish oils are recommended in relatively narrow circumstances. This reflects the modest available clinical evidence that pharmacological treatment of high triglyceride levels improves cardiovascular outcomes. Limited evidence suggests that a fibrate or statin may be beneficial to patients whose primary lipid abnormality is high triglyceride levels or low HDL.

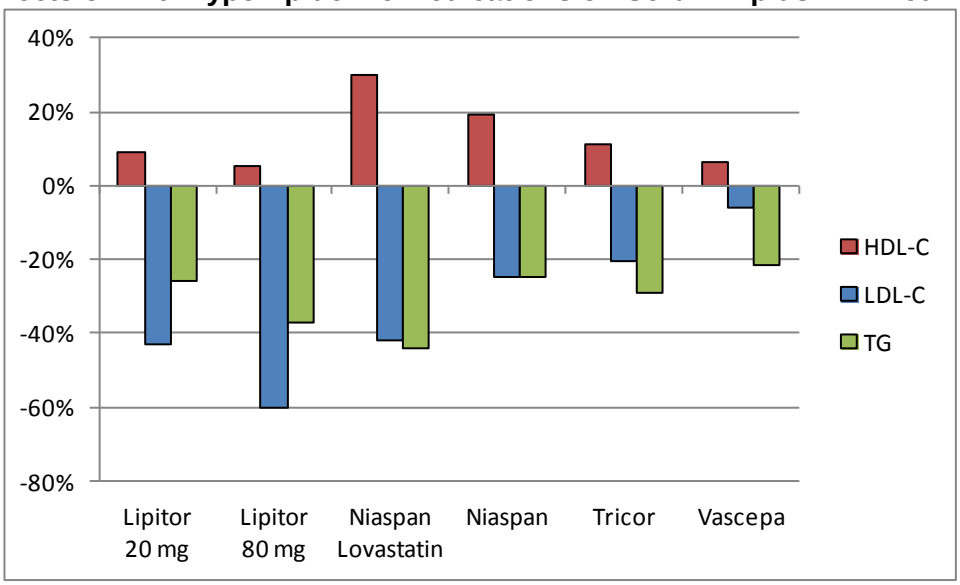
For patients with mixed dyslipidemia (high LDL cholesterol and high triglycerides), the AHA / ACC guidelines recommend treatment with a statin. If high triglyceride levels or low HDL levels persist while under treatment with a statin, combination therapy with a second agent can be instituted, but is recommended only in those with triglyceride levels in the top third or HDL levels in the bottom third.

Like other organizations, the AHA recommends aggressive treatment of severe triglyceridemia to reduce the risk of pancreatitis. However, the AHA guidelines generally oppose combination treatments or any effort to modulate HDL-C or triglyceride levels via pharmacological methods.

Approved Therapies for Dyslipidemia

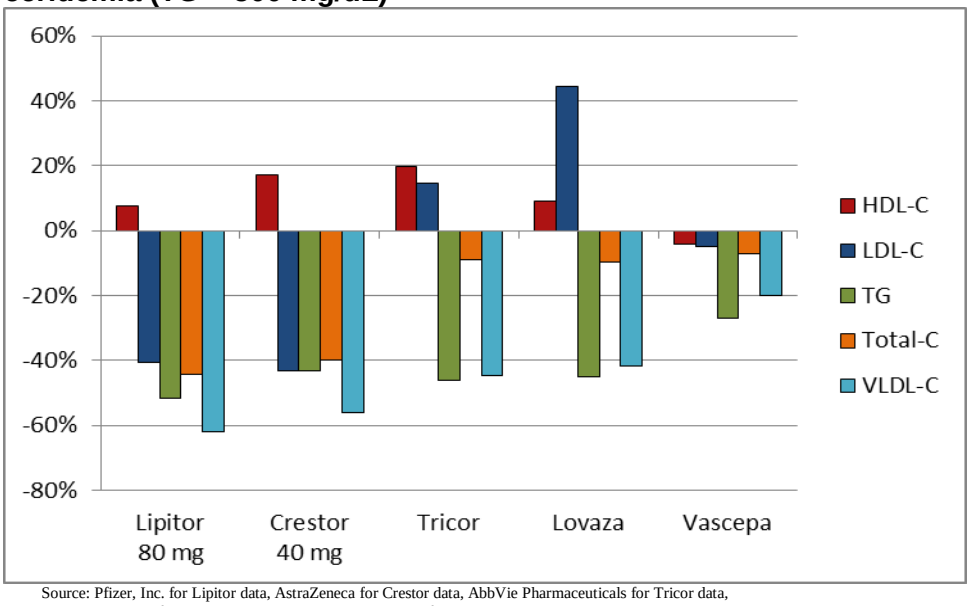
The effects of commonly used therapies for the treatment of simple dyslipidemia and for the treatment of severe triglyceridemia on key lipid parameters are shown in Figures 5 and 6 below.

Figure 5: Effects of Anti-Hyperlipidemic Medications on Serum Lipids in Mixed Hyperlipidemia



Source: Pfizer, Inc. for Lipitor data, AbbVie Pharmaceuticals for Niaspan & Tricor data, Amarin Pharmaceuticals for Vascepa data.

Figure 6: Effects of Antihyperlipidemic Medications on Serum Lipids in Severe Hypertriglyceridemia (TG > 500 mg/dL)



Source: Pfizer, Inc. for Lipitor data, AstraZeneca for Crestor data, AbbVie Pharmaceuticals for Tricor data, GlaxoSmithKline for Lovaza data, Amarin Pharmaceuticals for Vascepa data.

…Statins…

Statins are the most well-characterized and most widely used treatments for lipid disorders. As seen in Figures 5 and 6 for the statins Lipitor® and Crestor®, statins potently reduce LDL cholesterol, VLDL cholesterol, triglycerides, and total cholesterol, while modestly increasing HDL. In addition to their potency, statins are first-line agents for the treatment of dyslipidemia because they have been shown to reduce cardiovascular events and mortality both in patients with existing cardiovascular disease and in the primary prevention setting. Lipitor, for example, at the 10 mg dose, reduced the surrogate endpoint of non-fatal myocardial infarction or cardiovascular death by 36% in the Anglo-Scandanavian Cardiac Outcomes Trial (ASCOT) (The Lancet: Sever PS, et al, 2003).

…Fibrates…

Tricor and other fibrates produce modest increases in HDL cholesterol and reduce LDL cholesterol and triglycerides to an extent similar to statins in patients with mixed hyperlipidemia. In those with very high triglycerides, fibrates tend to raise, rather than lower, LDL cholesterol. Fibrates potently reduce serum triglycerides, and are first-line agents for this purpose in many hypertriglyceridemia and mixed hyperlipidemia treatment guidelines.

The evidence for fibrates having a favorable impact on cardiovascular outcomes is mixed. In the Helsinki Heart Study (New England Journal of Medicine: Frick MH, et al, 1987), gemfibrozil monotherapy produced a 34% reduction in the composite endpoint of cardiovascular events or mortality, which was increased to 71% in the subgroup with high triglycerides. It also produced a 24% reduction in cardiovascular events and death in the VA-HIT trial.

Other studies looking at fibrates in general or at Tricor (phenofibrate) in particular have failed to demonstrate a favorable impact on outcomes when used as monotherapy (vs placebo) or in combination with a statin (vs. statin only). In the ACCORD-LIPID trial (Diabetes Care: Margolis KL, et al, 2014), the combination of a fibrate and a statin failed to demonstrate reduced cardiovascular events in diabetics relative to a statin plus placebo.

…Sustained Release Niacin…

High-dose niacin (vitamin B-3) 1500 or more mg/dL decreases triglyceride levels by at least 40% and can raise HDL cholesterol levels by 40% or more. Niacin also reliably and significantly lowers LDL cholesterol levels, which the other major triglyceride-lowering medications do not (Maki KC, et al, 2012). These are commonly used in situations where HDL increase is desired, but they are also first-line treatments in many guidelines for treating hypertriglyceridemia. Flushing and itching are important tolerability issues.

Modest to weak evidence supports the ability of niacin monotherapy to favorably impact cardiovascular outcomes. The data mainly comes from studies of immediate release niacin used as monotherapy in the pre-statin era. The Coronary Drug Project, initiated in 1975, found a 27% reduction in non-fatal myocardial infarction in patients who had previously had a heart attack. Total mortality was the same in the two groups at 5 years.

A reduction in atherosclerotic plaque reduction for patients treated with niacin and cholestipol compared to cholestipol alone was observed in the Cholesterol-Lowering Atherosclerosis Study (CLAS) (Azen SP, et al, 2006). However, two more recent trials failed to demonstrate a reduction in cardiovascular events for patients treated with niacin plus a statin compared to a statin alone (AIM-HIGH) (American Heart Journal, 2011) and HPS-THRIVE (Boden WE, et al, 2014) trial.

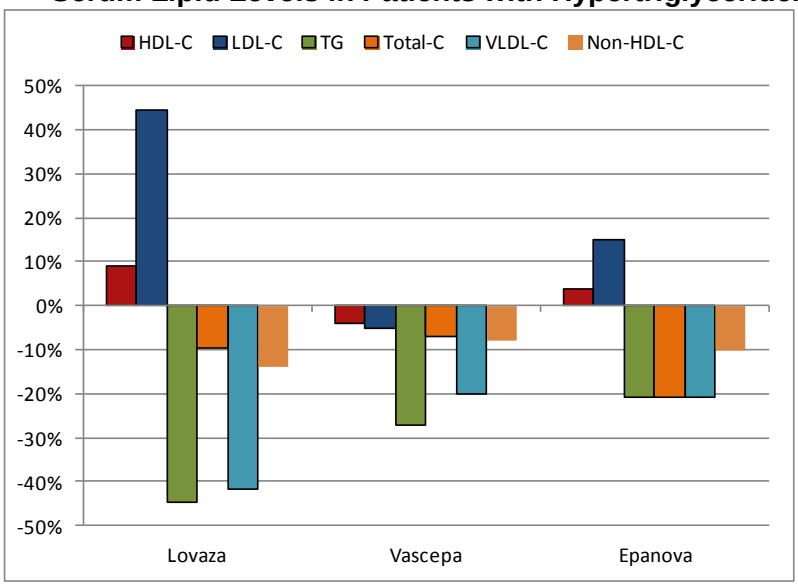
…Omega-3 Fatty Acids (Fish Oil)…

Available fish oil products are described in Table 4 below. Lipid lowering properties of these drugs are shown in Figures 7 below. The predominant constituent in all three products is EPA or its pro-drug EPA hydrochloride. The compositions differ mainly in the amount of DHA and other fish oil fatty acids present. All three are approved for the treatment of very high triglyceride levels (≥500 mg/mL), but only Lovaza is approved for the treatment of borderline high triglycerides in patients whose LDL levels have already achieved target with statin administration.

Table 4: Approved Fish Oil (Omega-3 Fatty Acid)

Product	Company	Composition	Form
Lovaza	GlaxoSmithKline	51% EPA 42% DHA 7% other marine fatty acids	Ethyl esters
Vascepa	Amarin	96% EPA	Ethyl ester
Epanova	AstraZeneca	55% EPA 20% DHA 25% other marine fatty acids	Free acids

Figure 7: Effects of Lovaza, Vascepa, and Epanova on Serum Lipid Levels in Patients with Hypertriglyceridemia



Source: AstraZeneca for Epanova data, GlaxoSmithKline for Lovaza data, Amarin Pharmaceuticals for Vascepa data.

Characteristic differences in the impact of these three drugs on serum lipid levels in severe hypertriglyceridemia (TG ≥ 500 mg/dL) patients appear to be related to their differing DHA content. Lovaza, which has the largest amount of DHA, is more potent at reducing triglycerides and VLDL than are Vascepa or Epanova, but it raises LDL. Overall however, total non-HDL cholesterol is thought to be a better predictor of cardiovascular risk in hyperlipidemic patients that is LDL, and the effects of the three dugs on non-HDL cholesterol are very similar.

- **Lovaza** was developed by Reliant Pharmaceuticals and approved by the U.S. FDA in 2004. Reliant was acquired by GlaxoSmithKline in November 2007 for $1.65 billion. Sales have run in the $900 million range since 2008. No cardiovascular outcome trial has been performed to determine whether the attractive impact of Lovaza on serum lipids translates into improved survival and reduced cardiovascular disease in patients with hyperlipidemia. The GISSI Heart Failure Project demonstrated increased survival in heart failure patients, but Lovaza was never approved for this indication in the U.S. Teva Pharmaceuticals launched an authorized generic formulation of Lovaza in April 2014.

- **Vascepa**, >96% EPA, was developed by Amarin Pharmaceuticals and approved by the FDA for treatment of patients with severe hypertriglyceridemia in September 2012. It is also in development for the treatment of patients whose triglyceride levels remain above 200 mg/dL in spite of being under treatment with a statin. Amarin sought and pursued a SPA with the FDA for this indication, but the SPA was revoked in light of new data from trials of other drugs that convinced the agency that triglyceride lowering was unlikely to lead to improved outcomes in this patient population.

  Data shows that Vascepa combined with a statin, compared to a placebo combined with statin, lowered triglycerides by 33%, LDL by 2%, and non-HDL cholesterol by 18%. The ongoing global REDUCE-IT (NCT01492361) cardiovascular outcomes trial will examine the efficacy of Vascepa vs. placebo against a background of stable statin therapy for reducing a composite endpoint of CV death, MI, stroke, coronary revascularization, and hospitalization for unstable angina. Expected enrollment is 8,000 patients and top-line results are expected in 2018. The effect of EPA on cardiovascular outcomes has previously been examined in the Japan EPA Lipid Intervention Study (JELIS), a prospective, randomized, open label, blinded endpoint trial including 18,645 primary and secondary prevention patients with mixed hyperlipidemia. After 5 years of follow-up, a 19% reduction in major coronary events was observed in the EPA treatment arm (Ohnishi, H, et al, 2013).

- **Epanova** was developed by Omthera Pharmaceuticals and approved by the FDA in May 2014. Omthera was acquired by AstraZeneca in July of 2013 for $323 million plus a potential contingent milestone of $120 million based on global sales of the drug. Currently approved for use in patients with TG > 500 mg/dL, AstraZeneca is seeking approval for use in combination with a statin in patients whose triglyceride level remains above 200 mg/dL in spite of ongoing treatment with a statin. The design of the cardiovascular outcomes trial, dubbed STRENGTH (NCT02104817), is very similar to that of the corresponding Vascepa trial, only larger with an enrollment goal of 13,000 patients. Lead investigation of STRENGTH is taking place at the Cleveland Clinic. Data is expected in 2019.

Matinas Biopharma's New Approach

The team at Matinas has taken a new approach based on the company's proprietary techniques for purifying higher homologues of omega-3 fatty acids. This ability allows the company to prepare "designer" prescription omega-3 fatty acid products having optimized properties for the treatment of cardiovascular disease and improved patentability based on the identification of novel mixtures with improved properties.

Table 5 below shows some of the diversity of omega-3 fatty acids found in fish oil. Many of these fatty acids are formed in low abundance, and the similarity of their properties to much more abundant close analogs has hindered evaluation of their biological properties. Matinas Biopharma has developed proprietary methods for purifying these rare omega-3 fatty acids, which allows their unique biological properties to be elucidated for drug discovery efforts. The very limited prior work with these rare acids offers the opportunity for discovering new therapeutic paradigms, and to avoid much of the complications of intellectual property management that have been faced by companies working with the well-characterized EPA and DHA.

Table 5: Omega-3 Fatty Acids

Name	Abbreviation	No. of Carbons	No. of Double Bonds
Hexadecantrienoic acid	HTA	16	3
Alpha-Linolenic acid	ALA	18	3
Stearidonic acid	SDA	18	4
Eicosatrienoic acid	ETE	20	3
Eicosatetraenoic acid	ETA	20	4
Eicosapentaenoic acid	EPA	20	5
Heneicosapentaenoic acid	HPA	21	5
Docosapentaenoic acid	DPA	22	5
Docosahexaenoic acid	DHA	22	6
Tetracosapentaenoic acid	TPA	25	5
Tetracosahexaenoic acid	THA	24	6

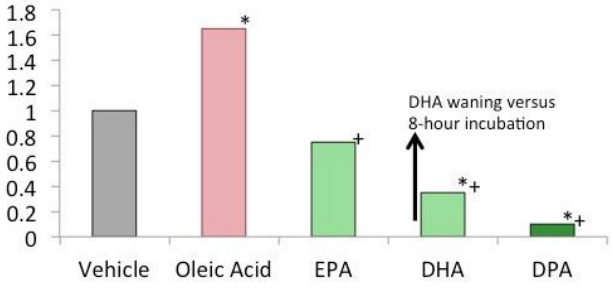
Docosapentaenoic Acid and MAT9001

One omega-3 fatty acid of particular interest is docosapentaenoic acid (DPA). DPA is a key component of MAT9001, a next generation prescription grade omega-3 fatty acid that Matinas is developing for the treatment of mixed hyperlipidemia and hypertriglyceridemia. Preclinical studies suggest that DPA has the potential to confer a unique therapeutic profile compared to existing omega-3 products, particularly in its ability to exert synergistic anti-hyperlipidemic effects in combination with statins.

Lipids have historically been regarded as mainly having structural and functional roles. They serve as part of cellular and organelle membranes and as metabolic intermediates for energy production and the formation of signaling molecules. The omega-3 and omega-6 fatty acids have additionally been understood to serve as inhibitors of enzymes involved in triglyceride processing. But importantly, recent discoveries have pointed toward the ability of certain omega-3 fatty acids to modulate the expression of genes involved in lipid synthesis.

The gene regulatory effects of DPA in rat liver cells include the down-regulation of genes such as SREBP-1c, acetyl coenzyme-A carboxylase, ChREBP, and fatty acid synthetase that are known targets of other omega fatty acids. Notable, however, is DPA's unusual potency in reducing the expression of RNA encoding HMG-CoA reductase, the enzyme target of statins. By down-regulating gene expression DPA has the potential to act synergistically with statins in reducing the activity of this well-established drug target. Reduced enzyme production is a result of the action of DPA at the gene level, and the protein that is produced is inhibited by the statin.

Figure 8: Effect of Various Omega-3 Fatty Acids on HMG-CoA Reductase RNA Levels in Rat Liver Cells

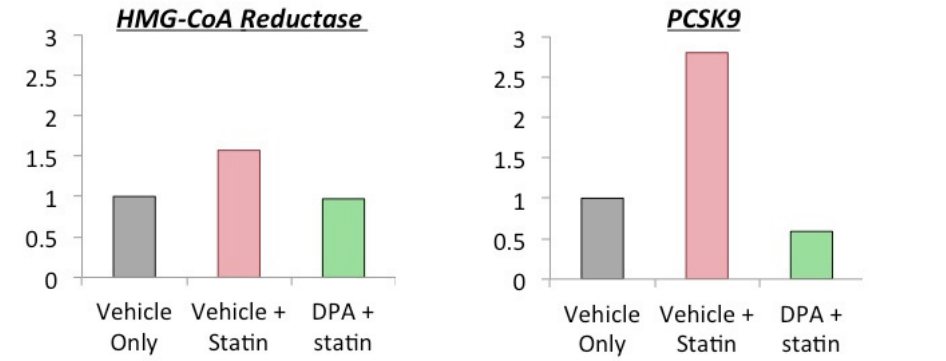


Relative mRNA expression levels after 48 hour incubation of Rat Liver Cells with FA
* Significant difference (p<0.05) from Control (EC)
\+ Significant difference (p<0.05) from Oleic Acid (OAC)

Source: Kaur et al.; PLEFA (2011) 85, 155-161

In vivo results support the *in vitro* finding and its suggestion of potential synergy with statins in man. Figure 9 shows HMG-CoA reductase and PCSK9 gene levels in fatty Zucker rats treated with vehicle (control), vehicle plus statin, or DPA plus statin. The Figure shows that the RNA encoding each drug target is up-regulated, probably as a compensatory mechanism, in rats treated with statin only. The addition of DPA abolishes this compensatory response, and in the case of PCSK9, drives RNA levels below those of rats not treated with statin.

Figure 9: Relative Levels of mRNA for HMG-CoA Reductase and PCSK9 in Rat Liver - In Vivo

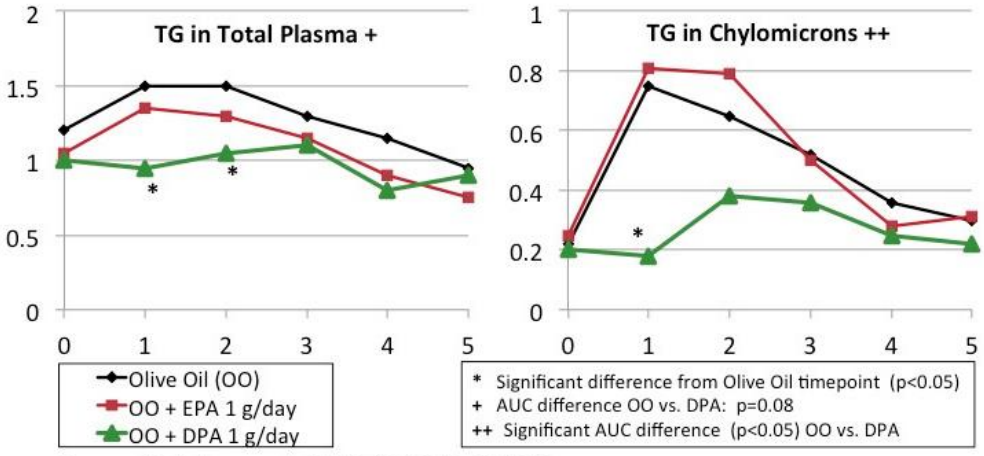


Source: Matinas BioPharma research; unpublished

In addition to the effects described above, DPA has several other types of activity. These include an aspirin-like platelet inhibitory and an anti-angiogenic effect through the suppression of the expression of the gene for the VEGF-2 receptor.

Pure DPA has been examined for antihyperlipidemic activity in a small 10 patient Australian cross-over trial. DPA was observed to prevent post-meal excursions in plasma triglyceride levels to a greater extent than EPA. This effect appears to arise from reduction in the production of chylomicrons.

Figure 10: Human Post-Meal TG levels (mmol/L) over 5 Hours



Source: Linderborg et al.; PLEFA (2013) 88, 313-319

The potential of DPA for synergism with statins in the treatment of hyperlipidemias is being exploited in Matinas' lead development candidate, MAT9001. MAT9001 is a proprietary mixture of DPA, EPA, low levels of DHA and certain other omega-3 fatty acids. **HYPOTHESIS:** Given the known properties of EPA and DHA, and the potent ability of DPA to regulate RNA expression for HMG-CoA reductase and other hyperlipidemia targets, this formulation has the potential to be a highly differentiated drug candidate. MAT9001 is in development for therapeutic applications in dyslipidemia with hypertriglyceridemia as the primary indication.

On October 20, 2014 the company filed its MAT9001 IND with the US FDA. The company anticipates completing the Canadian PK/PD study in the first quarter of 2015, followed by the initiation of a U.S.-based Phase 3 registration program by the middle of the year. On October 29, 2014, the company received approval from Health Canada to initiate the aforementioned PK/PD study.

WE CONCLUDE: Taken in sum, these data suggest that MAT-9001 has the potential to provide a highly differentiated profile in patients with severe hypertriglyceridemia and in those with mixed hyperlipidemia. We think this is a promising, though very early stage opportunity, with significant upside in the event that clinical results reflect this superior potential.

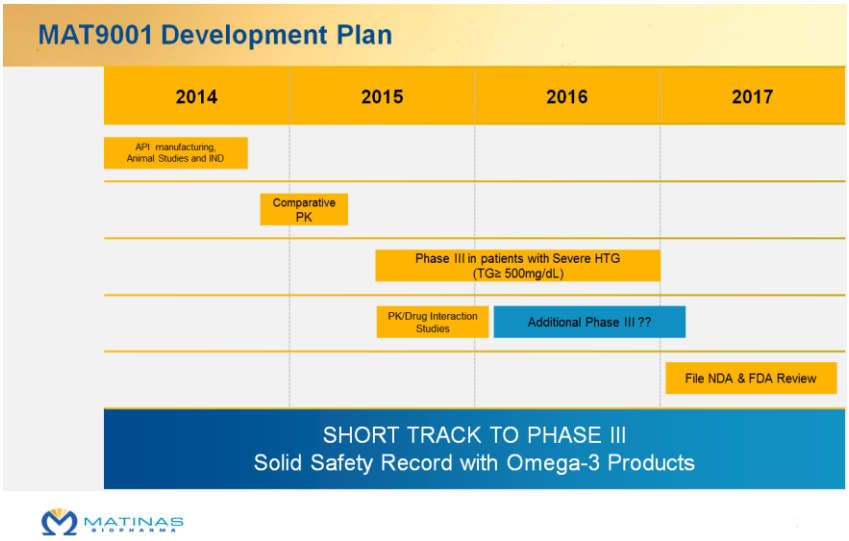
Intellectual Property

Potential concerns with respect to intellectual property for those working in the omega-3 fatty acid area are suggested by Amarin Pharmaceutical's development of 96% pure eicosapentaenoic acid (Vascepa). The company was denied the 5 years of administrative exclusivity that pertains to the approval of a New Chemical Entity (NCE) because the FDA does not consider any active ingredient found in a previously approved drug to be an NCE. Eiscosapentaenoic acid (EPA) is found as one of several omega-3 fatty acids in GlaxoSmithKline's Lovaza. Opportunities for patent protection were also limited, as the law does not permit patenting of natural products, and obtaining use patents was rendered difficult by the widespread use of ethyl EPA in Japan for 15 years prior to its development in the United States. Amarin has been able to obtain a number of formulation and utility patents, but a mere one and a half years post approval, Vascepa has already become the subject of several paragraph IV challenges.

In contrast to the difficulties faced by Amarin with Vascepa, GlaxoSmithKline's had a relatively smooth time protecting Lovaza's IP up until late 2013. As an optimized mixture of omega-3 fatty acids, Lovaza was protected by U.S. Patent No. 5656667, which claims the discovery of a uniquely useful mixture of omega-3 fatty acids of optimized properties. Glaxo and Teva came to an agreement in 2013 allowing the launch of an authorized generic formulation in April 2014. A similar model focusing on an optimized mixture of omega-3 fatty acids seems likely to apply to Matinas and MAT9001. We note that the company has already filed at least 13 patent applications, mostly covering mixtures of docosapentaenoic acid (DPA) with other omega-3 fatty acids.

Development Timeline

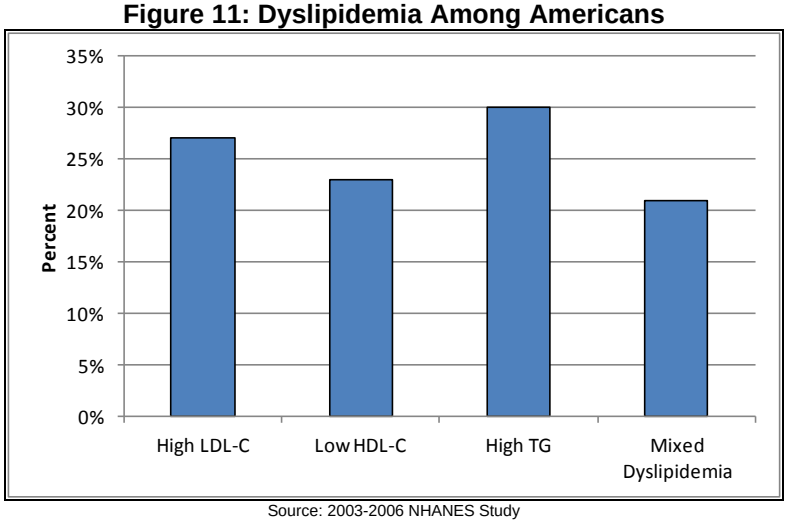
On October 20, 2014, the company announced the filing of its IND covering the severe hypertriglyceridemia development program to the U.S. FDA. The company has guided to initiating a comparative a PK study in late 2014. On October 29, 2014, the company received approval from Health Canada to initiate the aforementioned PK/PD study. The plan is then to move directly into a Phase 3 trial in severe hypertriglyceridemia around the middle of 2015 in the U.S. If all goes well, the company could be in position to file the New Drug Application (NDA) late in 2016, with a U.S. FDA PDUFA action date around the end of 2017.



Market Analysis & Peak Sales Opportunity

According to data from the AHA/ACC, roughly 5 million Americans have severe hypertriglyceridemia (>500 mg/dL). Another 35 million Americans have mixed dyslipidemia, with a triglyceride level in excess of 200 mg/dL. Approximately 25 million Americans are taking statin drugs such as Lipitor or Crestor to control their high cholesterol and triglyceride levels. However, according to New Guidelines issued in November 2013 from the AHA and ACC, the number of Americans that actually qualify for statin therapy and should be taking the drugs is closer to 70 million! According to the 2003-2006 NHANES study, 30% of Americans have serum lipid profiles that exceed NCEP-ATP3 guidelines shown above in Table 2.

Figure 11: Dyslipidemia Among Americans

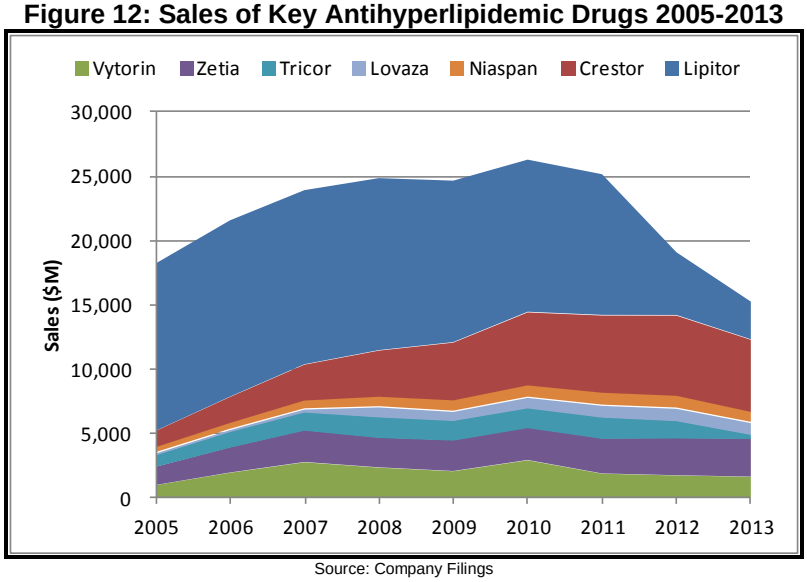


Source: 2003-2006 NHANES Study

The fish oil / omega-3 fatty acid market is currently dominated by Lovaza, a mixture of 47% EPA, 38% DHA, and 17% other fish oil in the form of ethyl esters. Lovaza is approved for the treatment of both severe hypertriglyceridemia (>500 mg/dL) and for the treatment of mixed hyperlipidemia in combination with a statin. Lovaza sales in 2013 were $917M at GlaxoSmithKline. Based on prescription tracking data by IMS Health, the total number of Lovaza prescriptions in the U.S. in 2013 was roughly 4.79 million, down slightly (-8%) from the 5.18 million in 2012 due in part to added competition from recently approved Vascepa at Amarin and Epanova at AstraZeneca, as well as the high profile failures of the ACCORD-LIPID trial (with TriCor) and the AIM-HIGH and HPS-THRIVE trials (with extended release niacin).

Sales of antihyperlipidemic products peaked in 2010 just before Pfizer's mega-blockbuster Lipitor lost patent exclusivity. AbbVie's TriCor has also lost patent exclusivity and is now available as a generic. Nevertheless, prescriptions for cholesterol and triglyceride lowering agents remain relatively strong, even in the face of the high profile failures noted above for fenofibrate and niacin products. Prescriptions for fish oil products should still eclipse 5 million in 2014. Amarin's Vascepa, with comparably better head-to-head data than Lovaza, has only captured roughly 10% of the market (~500,000 TRx). The outcome of Amarin's REDUCE-IT cardiovascular outcome trial in 2018 will have a profound impact on the future prescription and revenue potential for all fish oil / omega-3 fatty acid products.

Figure 12: Sales of Key Antihyperlipidemic Drugs 2005-2013



Source: Company Filings

With clearly superior data, we believe MAT9001 could easily capture 20% of the fish oil market for patients with severe hypertriglyceridemia once approved in 2018. We estimate the market for severe hypertriglyceridemia in the U.S. in 2020 will be approximately 7 million patients. However, less than 10% of the patients are actually taking fish oil products. With 20% market share, Matinas Biopharma has an existing market opportunity of ~150,000 patients or 1.8 million prescriptions. Assuming $200 per prescription, the market opportunity to Matinas Biopharma looks roughly $360 million in size.

Matinas will not, however, have cardiovascular outcome data to support expanding the label for MAT9001 into the mixed dyslipidemia market and legally target the 35 million American's with triglyceride levels between 200 mg/dL and 500 mg/dL. Nevertheless, some of these patients are on prescription fish oil products like Lovaza and Vascepa "off label" per the recommendations of their primary care physician or cardiologist. We estimate approximately 20% of Lovaza's 5.18 million prescriptions in 2012 are in the mixed dyslipidemia population. With superior data, assuming Matinas captures the same 20% market share away from Lovaza, off label indications for MAT9001 look to be about another $140 million. As such, we see the current peak market opportunity for MAT9001, prior to any outcome data, at $500 million. We caution investors that this $500 million figure assumes a vastly superior clinical profile for MAT9001 vs. generic Lovaza (authorized now, full in 2015), Vascepa, and Epanova.

Matinas Financial Position & Capital Structure

Matinas Biopharma exited the second quarter ending June 30, 2014 with $6.65 million in cash and investments. Burn from operations in the first and second quarter of 2014 totaled $2.3 and $1.9 million, respectively. We forecast burn in the third quarter 2014 totaled around the same amount. Thus, the cash position as of September 30, 2014 probably stood around $4.8 million. We expect burn in the fourth quarter will increase slightly as the company conducts the PK/PD study on MAT9001 outside the U.S. (in Canada). Therefore, we forecast cash at year end 2014 will be around $2.5 million. This should be enough to continue to fund operations into March 2015.

All-in, we suspect Matinas will require roughly $30 million to fund the required Phase 3 trial with MAT9001 prior to filing the NDA in 2016. This does not include the potential for a second Phase 3 trial, if required, prior to the NDA filing. We believe the company will seek to raise a small amount of money in the near-term to fund operations through the Canadian PK/PD data expected in April 2015. We suspect that a bridge financing of $5 million gets the company well into the second half of 2015, putting them in a much better position to then fund the Phase 3 program in 2015 and 2016.

As of August 8, 2014, Matinas Biopharma had 32.0 million basic shares outstanding. The company has another 8.25 million issuable shares under the existing employee stock option program. Of this amount, 3.1 million is outstanding, 0.8 million of which is currently exercisable at $0.94 per share. There are 1.5 million warrants at $1.00 per share outstanding that expire in July 2018 and 13.75 million warrants at $2.00 per share that expire in July and August 2018. The current stock price is $0.60 per share, so all outstanding options and warrants are out-of-the-money. However, should all outstanding warrants be exercised it would generate approximately $29.0 million in cash to the company. The fully diluted market capitalization at $2.00 per share would be $96.1 million (includes $29.0 million in cash).

The current **Basic Market Capitalization** is $19.2 million (32.0 million shares x $0.60 per share)

MATINAS BIOPHARMA HOLDINGS, INC
(Formerly Matinas BioPharma, Inc.)
Condensed Consolidated Balance Sheets

	June 30, 2014 Unaudited	December 31, 2013 Audited
Assets		
Current assets		
Cash and cash equivalents	$ 6,647,479	$ 10,840,428
Prepaid expenses and other current assets	38,065	84,493
Total current assets	6,685,544	10,924,921
Property, plant and equipment, net	361,645	93,057
Other long term assets	316,105	315,778
Total assets	$ 7,363,294	$ 11,333,756
Liabilities, Preferred Stock and Stockholders' Equity		
Current liabilities		
Accounts payable	$ 281,090	$ 396,768
Accrued expenses	547,326	462,200
Lease liability - current	43,815	-
Total current liabilities	872,231	858,968
Lease liability - long term	33,580	-
Total liabilities	905,811	858,968
Stockholders' equity		
Preferred stock - $0.001 par value, 10,000,000 shares authorized, 0 shares issued and outstanding at June 30, 2014 and December 31, 2013	-	-
Common stock - $0.001 par value, 150,000,000 shares authorized, 32,000,000 shares issued and outstanding at June 30, 2014 and December 31, 2013	3,200	3,200
Additional paid-in capital	14,849,896	14,302,307
Deficit accumulated during development stage	(8,395,613)	(3,830,719)
Total stockholders' equity	6,457,483	10,474,788
Total liabilities and stockholders' equity	$ 7,363,294	$ 11,333,756

Matinas Biopharma Management Profiles

Roelof Rongen has served as President and Chief Executive Officer and as a Director of Matinas BioPharma since April 2012. He is also the Founder and Chairman of Essential Fatty Acid Therapeutics LLC, a biotech company focused on the development of innovative fatty acid derivatives. Prior to Matinas BioPharma, Mr. Rongen was Executive Vice President North American Operations for Trygg Pharma AS (subsequently named EPAX AS) (2009-2012) and Vice President of Life Cycle Management and Intellectual Property at Reliant Pharmaceuticals, Inc. (2000-2008). While at Reliant, Mr. Rongen held various earlier positions, including head of the Omacor®/Lovaza® launch team, Executive Director of Marketing for Lescol® and Executive Director of Business Development. Prior to Reliant, Mr. Rongen was Global Product Director for Humira® at BASF Pharma (1998-2000), later acquired by Abbott Laboratories. He was also a consultant at The Wilkerson Group in New York (1995-1998) and Arthur D. Little in Amsterdam (1990-1993), and a Research Fellow in biochemistry at Baylor University in Texas (1989-1990). Mr. Rongen earned an MBA from Kellogg GSM at Northwestern University in Evanston, IL, and a graduate degree in Molecular Sciences from Wageningen University in the Netherlands.

Jerome D. Jabbour, JD has served as Executive Vice President, Chief Business Officer, General Counsel and Secretary since October 2013 and as a Director from November 2013. Mr. Jabbour is also a Co-Founder of Matinas BioPharma. Prior to joining the management team, he was the Executive Vice President and General Counsel of MediMedia USA from 2012 to October 2013, a privately held/ diversified health care services company. Prior to MediMedia, he was the Senior Vice President, head of Global Legal Affairs and US General Counsel of Wockhardt Limited (2008-2012) and Senior Counsel at Reliant (2004-2008). Earlier in his career, he held positions as Commercial Counsel at Alpharma, Inc. (2003-2004) and as a Corporate Associate at Lowenstein Sandler LLP (1999-2003). Mr. Jabbour earned his J.D. from Seton Hall University School of Law in New Jersey and a B.A. in Psychology from Loyola University in Baltimore.

George Bobotas, PhD has served as our Executive Vice President and Chief Scientific Officer since August 2011. Dr. Bobotas is a Co-Founder of Matinas BioPharma. Prior to Matinas BioPharma, Dr. Bobotas was a founder of expert consulting firm Demelle BioPharma, LLC ("DeMelle BioPharma") (2008-2012) and Vice President Scientific Affairs at Reliant (2000-2008). Prior to Reliant, he was the founder and Executive Director of the Covance Center for CNS Research (1997-2000). Earlier in his career, Dr. Bobotas held senior positions at Somerset Pharmaceuticals, Inc. (1994-1997), Mylan Laboratories Limited (1988-1994), and Forest Laboratories Inc. (1981-1988). He is the inventor on 22 published patents and patent applications all related to the health and pharmaceutical development and manufacturing processes. Dr. Bobotas received his Ph.D. in Biochemistry from the City University of New York, an M.A. in Physical Chemistry from Smith College, Northhampton, MA, and a B.A. in Chemistry from Windham College, Vermont.

Abdel A. Fawzy, PhD has served as Executive Vice President for Pharmaceutical and Supply Chain Development since August 2011. Dr. Fawzy is a Co-Founder of Matinas BioPharma. Prior to Matinas BioPharma, Dr. Fawzy was a founder of expert consulting firm DeMelle BioPharma (2008- 2012) and Executive Director Pharmaceutical Development at Reliant, from 2000 to 2008. Earlier in his career, Dr. Fawzy held pharmaceutical development positions at Ascent Pharmaceuticals, Inc. (1994-2000), DuPont (1990-1994) and Squibb Marsam Pharmaceuticals (1989-1990). He is the inventor on 15 published patents and patent applications all related to the health and pharmaceutical development and manufacturing processes. Dr. Fawzy received his Ph.D. in Pharmaceutical Technology from Tuebingen University in Germany, a Pharmacy degree from Temple University in Philadelphia, PA, and a MS in Pharmaceutical Technology from the Cairo School of Pharmacy in Egypt.

Gary Gaglione, CPA has served as Interim Chief Financial Officer, Vice President of Finance & Accounting on a consulting basis from April 2013 until October 2013 at which time he became a full time employee. Prior to joining Matinas, Mr. Gaglione was President of MCM Consulting LLC from 2011 until October 2013. Prior to MCM Consulting, Mr. Gaglione was Senior Director of Finance at Shionogi USA, Inc., responsible for budgeting and planning (2011). In 2009 and 2010, he was Vice President of Finance and Controller for Phytomedics, Inc., a startup botanical pharmaceutical company. Prior to Phytomedics, he was Controller for ProStrakan Inc.'s U.S. operations (2008-2009). From 2001 to 2008, Mr. Gaglione was an Executive Director at Reliant, initially as head of Planning, Budgets and Analysis, then, from 2006 on, as head of Internal Audit and Sarbanes Oxley Compliance in preparation for a potential Reliant initial public offering. Before Reliant, he held numerous finance positions of increasing responsibility at the U.S. subsidiary of Hoffmann-La Roche Inc. (1976-2001), including Vice President of R&D Finance (1997-2001), Director of Compensation with responsibility for executive payroll, payroll, benefits, and exempt/non-exempt compensation systems (1995-1997), and Controller for the US pharmaceutical division and sites (1985-1997). Mr. Gaglione earned a B.S. degree in Business Administration with a major in Accounting from Villanova University, Villanova, PA, and an MBA in Finance from Seton Hall University, West Orange, NJ.

Herbert Conrad became the Chairman of the Board of Matinas BioPharma in 2012. He also serves as a Director on the Board of Celldex and as an Advisor to the Seaver Autism Center at Mount Sinai Hospital. Mr. Conrad was the former President of Roche Pharmaceuticals in the United States from 1982 until his retirement in 1993. Since then he has served on the boards of Pharmasset (Chairman), Savient, Dura, UroCor, GenVec (Chairman), Sicor, Bone Care International (Chairman), Sapphire Therapeutics (Chairman), the medical advisory board of Henry Schein, and he was a Director and co-founder of Reliant Pharmaceuticals. He received B.S. and M.S. degrees from the Brooklyn College of Pharmacy and an honorary Doctorate in Humane Letters from Long Island University.

SUMMARY, VALUATION & RECOMMENDATION

We are initiating coverage Matinas BioPharma Holdings, Inc (MTNB:OTCQB) with an 'Outperform' rating and a $1.25 per share price target. Matinas is developing MAT9001, a proprietary prescription grade omega-3 fatty acid composition, comprised of a complex mixture of omega-3 fatty acids, predominantly eicosapentaenoic acid ("EPA"), and docosapentaenoic acid ("DPA"), a rare and potent omega-3 fatty acid. The product also contains several other omega-3 fatty acids, including relatively small amounts of docosahexaenoic acid ("DHA"). The initially targeted indication is severe hypertriglyceridemia, for which Matinas expects to initiate a Phase 3 clinical trial in the middle of 2015. Severe hypertriglyceridemia is a condition in which patients have elevated serum triglycerides (TG > 500 mg/dL) that put them at high risk of pancreatitis, and is recognized as an independent risk factor for cardiovascular disease. A second potential indication is the treatment of patients with mixed hyperlipidemia (triglycerides > 200 mg/dL in spite of ongoing statin treatment). The addressable treatment populations for these two indications in the United States are approximately 5 million and 30 to 35 million, respectively.

…Fish Oil & Why MAT9001 Is Different…

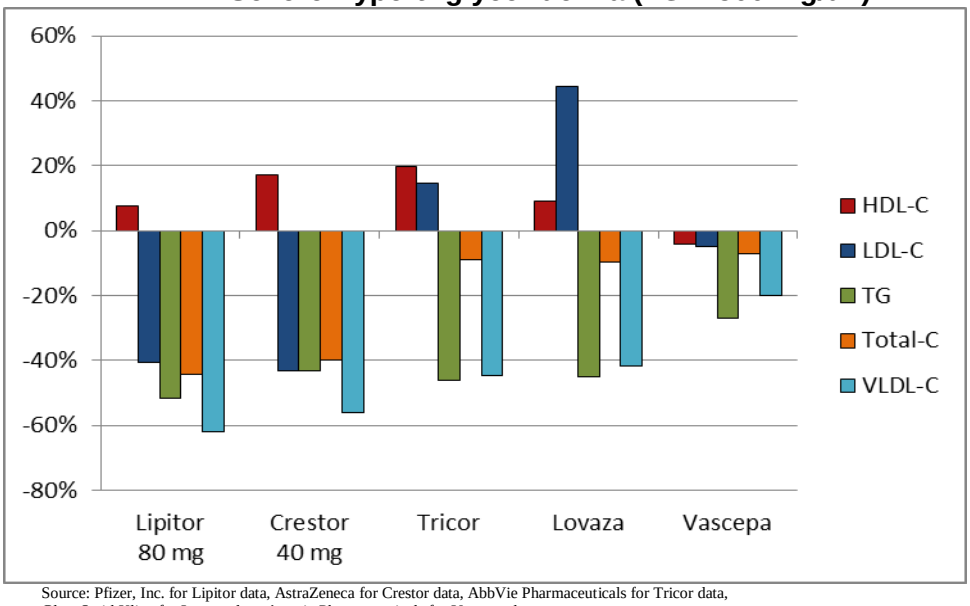
Individuals with hypertriglyceridemia and mixed dyslipidemia have a number of FDA approved prescription therapeutic options to treat their disease. The leading prescription products available for mixed dyslipidemia are statins, one of the most well-characterized and widely used classes of pharmaceutical agents. Statin drugs, such as Pfizer's Lipitor® and AstraZeneca's Crestor®, potently reduce LDL cholesterol, VLDL cholesterol, triglycerides, and total cholesterol, while modestly increasing HDL. In addition to their potency, statins are first-line agents for the treatment of dyslipidemia because they have been shown to reduce cardiovascular events and mortality both in patients with existing cardiovascular disease and in the primary prevention setting. Lipitor, for example, at the 10 mg dose, reduced the surrogate endpoint of non-fatal myocardial infarction or cardiovascular death by 36% in the Anglo-Scandanavian Cardiac Outcomes Trial (ASCOT) (Sever PS, et al, 2003).

Besides statins, fibrate products such as AbbVie's TriCor® and extended-release niacin products such as AbbVie's Niaspan® also have been shown to potently reduce triglycerides. However, unlike statins, cardiovascular outcome studies with fibrate and extended-release niacin products have been mixed with respect to demonstrating a reduction in morbidity and mortality with long-term use. The high profile failures of the ACCORD-LIPID trial with TriCor (Margolis KL, et al, 2014) and the HPS-TRIVE trial with Niaspan (Boden WE, et al, 2014) in 2013 have led to a meaningful decline in prescriptions for these two classes of drugs.

Prescription fish oil and omega-3 fatty acid products are the final class of pharmaceutical agents used to treat hypertriglyceridemia and mixed dyslipidemia. The market is currently dominated by Lovaza®, a mixture of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) ethyl esters that is approved for the treatment of both severe hypertriglyceridemia (> 500 mg/dL) and for the treatment of mixed hyperlipidemia in combination with a statin. Lovaza sales in 2013 were $917 million at GlaxoSmithKline. It is now available as an authorized generic. Other competitors in the space include Vascepa® (EPA ethyl ester) by Amarin Pharmaceuticals and Epanova® (EPA and DHA free acids) by AstraZeneca, each of which are currently labeled for treatment of severe hypertriglyceridemia only.

Cardiovascular outcome data with fish oil products is limited. Amarin is currently investigating Vascepa in an ongoing 8,000 patient, 7-year cardiovascular outcomes study called REDUCE-IT (NCT01492361). The primary endpoints of this study are CV-related death, myocardial infarction, stroke, coronary revascularization, and hospitalization for unstable angina. Data is expected in 2018. The effect of EPA on cardiovascular outcomes has previously been examined in the Japan EPA Lipid Intervention Study (JELIS), a prospective, randomized, trial including 18,645 primary and secondary prevention patients with mixed hyperlipidemia. After 5 years of follow-up, a 19% reduction in major coronary events was observed in the EPA treatment arm (Ohnishi, H, et al, 2013).

Figure 13: Effects of Antihyperlipidemic Medications on Serum Lipids in Severe Hypertriglyceridemia (TG > 500 mg/dL)

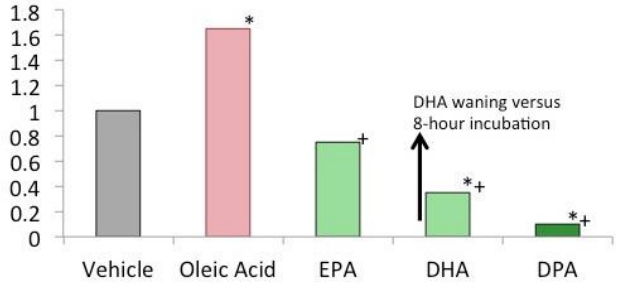


Source: Pfizer, Inc. for Lipitor data, AstraZeneca for Crestor data, AbbVie Pharmaceuticals for Tricor data, GlaxoSmithKline for Lovaza data, Amarin Pharmaceuticals for Vascepa data.

Unlike both Lovaza and Vascepa, Matinas' MAT9001 contains a unique component of docosapentaenoic acid, or "DPA". Preclinical studies suggest that DPA has the potential to confer a unique therapeutic profile compared to existing omega-3 products, particularly in its ability to exert synergistic anti-hyperlipidemic effects in combination with statins. For example, omega-3 fatty acids have been shown to serve as inhibitors of enzymes involved in triglyceride processing.

Recently, research has pointed toward the ability of certain omega-3 fatty acids to modulate the expression of genes involved in lipid synthesis. The gene regulatory effects of DPA in rat liver cells include the down-regulation of genes such as SREBP-1c, acetyl coenzyme-A carboxylase, ChREBP, and fatty acid synthetase that are known targets of other omega fatty acids. Notable, however, is DPA's unusual potency in reducing the expression of RNA encoding HMG-CoA reductase, the enzyme target of statins. By down-regulating gene expression DPA has the potential to act synergistically with statins in reducing the activity of this well-established drug target. Reduced enzyme production is a result of the action of DPA at the gene level, and the protein that is produced is inhibited by the statin.

Figure 14: Effect of Various Omega-3 Fatty Acids on HMG-CoA Reductase RNA Levels in Rat Liver Cells

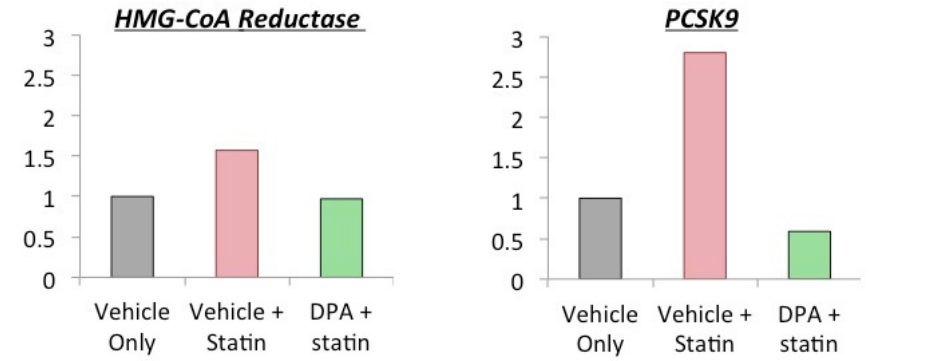


Relative mRNA expression levels after 48 hour incubation of Rat Liver Cells with FA
* Significant difference (p<0.05) from Control (EC)
\+ Significant difference (p<0.05) from Oleic Acid (OAC)

Source: Kaur et al.; PLEFA (2011) 85, 155-161

In vivo results support the in this *in vitro* finding and its suggestion of potential synergy with statins in man. Figure 15 shows HMG-CoA reductase and PCSK9 gene levels in fatty Zucker rats treated with vehicle (control), vehicle plus statin, or DPA plus statin. The Figure shows that the RNA encoding each drug target is up-regulated, probably as a compensatory mechanism, in rats treated with statin only. The addition of DPA abolishes this compensatory response, and in the case of PCSK9, drives RNA levels below those of rats not treated with statin.

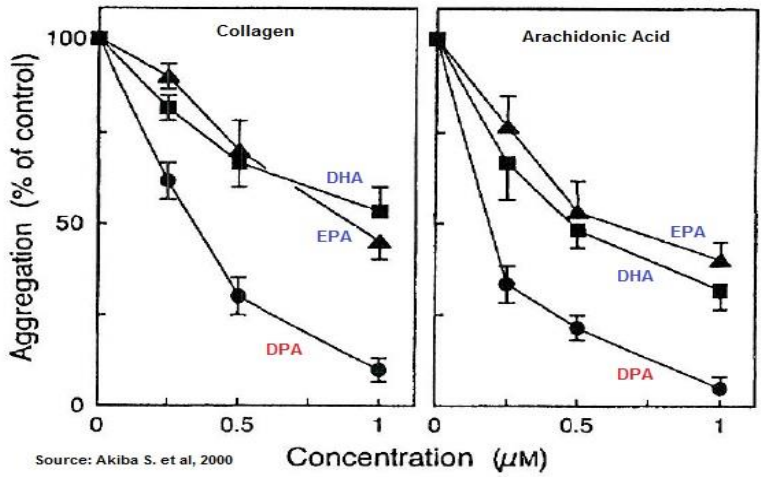
Figure 15: Relative Levels of mRNA for HMG-CoA Reductase and PCSK9 in Rat Liver - In Vivo



Source: Matinas BioPharma research; unpublished

In addition to the effects described above, DPA has several other types of activity. These include an aspirin-like platelet inhibitory and an anti-angiogenic effect through the suppression of the expression of the gene for the VEGF-2 receptor. A study conducted by Satoshi Akiba, et al, 2000, compared the effects of DPA on platelet aggregation and arachidonic acid metabolism to EPA and DHA. Collagen- or arachidonic acid-stimulated platelet aggregation was inhibited dose-dependently by n-3 fatty acids, among which data shows DPA to be the most potent. Furthermore, these fatty acids suppressed thromboxane A2 formation by platelets which were exposed to collagen, thrombin, or by platelets to which arachidonic acid was added. In these experiments, DPA was the most potent inhibitor. DPA also enhanced formation of 12-hydroxyeicosatetraenoic acid in response to collagen or from arachidonic acid by intact platelets, while the other two acids had less of an effect.

Figure 16: Effects of n-3 Fatty Acids on Platelet Aggregation Induced by Collagen or Arachidonic Acid



The potential of DPA for synergism with statins in the treatment of hyperlipidemias is being exploited in Matinas' lead development candidate, MAT9001. MAT9001 is a proprietary mixture of DPA, EPA, low levels of DHA and certain other omega-3 fatty acids. Matinas believes that the known properties of EPA and DHA, and the potent ability of DPA to regulate RNA expression for HMG-CoA reductase and other hyperlipidemia targets, as well as inhibit platelet aggregation has the potential to be a highly differentiated drug candidate for the treatment of hypertriglyceridemia (primary indication) and mixed dyslipidemia (potential label expansion).

On October 20, 2014 the company filed its MAT9001 IND with the US FDA. The company anticipates commencing PK/PD studies in the first quarter of 2015 in Canada, followed by the initiation of a Phase 3 registration program by the middle of 2015. PK/PD studies are currently taking place in Canada in an effort to better characterize MAT9001 prior to U.S. Phase 3 studies in 2015.

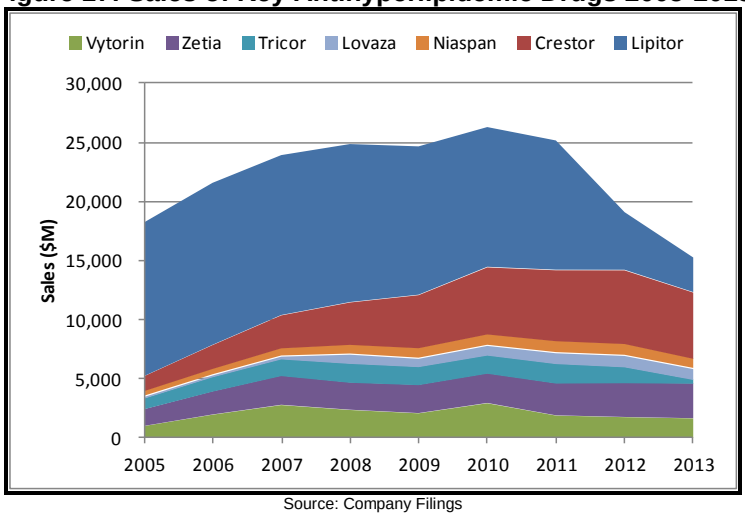
Taken in sum, we believe the data above suggest that MAT-9001 has potential to provide a highly differentiated profile in patients with severe hypertriglyceridemia and in those with mixed hyperlipidemia. We think this is a promising, though very early stage opportunity, with significant upside in the event that clinical results reflect this superior potential.

…With A Differentiated Profile – MAT9001 Has Blockbuster Potential…

According to data from the AHA/ACC, roughly 5 million Americans have severe hypertriglyceridemia with a serum triglyceride level > 500 mg/dL. Another 35 million Americans have mixed dyslipidemia, with a triglyceride level in excess of 200 mg/dL. Approximately 25 million Americans are taking statin drugs such as Lipitor or Crestor to control their high cholesterol and triglyceride levels. However, according to New Guidelines issued in November 2013 from the AHA and ACC, the number of Americans that actually qualify for statin therapy and should be taking the drugs is closer to 70 million!

Sales of antihyperlipidemic products peaked in 2010 just before Pfizer's mega-blockbuster Lipitor lost patent exclusivity. AbbVie's TriCor has also lost patent exclusivity and is now available as a generic. Nevertheless, prescriptions for cholesterol and triglyceride lowering agents remain relatively strong, even in the face of the high profile failures noted above for fenofibrate and niacin products. Prescriptions for fish oil products should still eclipse 5 million in 2014. Amarin's Vascepa, with comparably better head-to-head data than Lovaza, has only captured roughly 10% of the market (~500,000 TRx). The outcome of Amarin's REDUCE-IT cardiovascular outcome trial in 2018 and AstraZeneca's STRENGTH cardiovascular outcome trial in 2019 will have a profound impact on the future prescription and revenue potential for all fish oil / omega-3 fatty acid products.

Figure 17: Sales of Key Antihyperlipidemic Drugs 2005-2013



Source: Company Filings

With clearly superior data, we believe MAT9001 could easily capture >20% of the fish oil market for patients with severe hypertriglyceridemia once approved in 2017. The market for severe hypertriglyceridemia in the U.S. is approximately 5 million patients. However, less than 10% of the patients are actually taking fish oil products. With 20% market share, Matinas Biopharma has an existing market opportunity of ~150,000 patients or 1.8 million prescriptions. Assuming $200 per prescription, the market opportunity to Matinas Biopharma looks roughly $360 million in size.

Matinas will not, however, have cardiovascular outcome data to support expanding the label for MAT9001 into the mixed dyslipidemia market and legally target the 35 million American's with triglyceride levels between 200 mg/dL and 500 mg/dL. Nevertheless, some of these patients are on prescription fish oil products like Lovaza and Vascepa "off label" per the recommendations of their primary care physician or cardiologist. We estimate approximately 20% of Lovaza's 5.18 million prescriptions in 2012 were in the mixed dyslipidemia population. With superior data, assuming Matinas captures the same 20% market share away from Lovaza, off label indications for MAT9001 look to be about another $140 million. As such, we see the current peak market opportunity for MAT9001, prior to any outcome data, at $500 million. We caution investors that this $500 million figure assumes a vastly superior clinical profile for MAT9001 vs. generic Lovaza (authorized now, full in 2015), Vascepa, and Epanova.

…NPV Analysis Shows Matinas Attractively Valued…

Below we present a model for the moderate, high, and severe hypertriglyceridemia market in the U.S. We forecast in 2018, when MAT9001 will likely be on the market, that there will be ~7 million severe hypertriglyceridemia patients and ~38 million high hypertriglyceridemia or mixed dyslipidemia patients in the U.S. Only a small percentage of these patients are on therapy. However, with superior data, we believe MAT9001 can capture 20% of the market away from generic Lovaza and branded Vascepa and Epanova. That puts MAT9001 as a potential $500 million drug. As of now, prior to seeing the MAT9001 data or the results of cardiovascular outcome data from REDUCE-IT or STRENGTH, we believe this is a fair forecast. Assuming industry average margins, which include manufacturing, ongoing R&D, and selling, general, and administrative expenses, we believe Matinas can achieve ~50% net margins at peak sales. Using an aggressive 30% discount rate, we believe the company is worth $90 million in value.

Figure 18: MAT9001 Revenue Model

YEAR	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
U.S. Adult Population (> 18 yrs) In Millions	**242**	**244**	**247**	**249**	**252**	**254**	**257**	**259**	**262**	**265**	**267**	**270**
% w/ Normal TG's (0-149 mg/dL)	*70%*	*70%*	*69%*	*69%*	*68%*	*68%*	*67%*	*67%*	*66%*	*66%*	*65%*	*65%*
Target U.S. Adult Population	73	75	77	79	81	83	85	87	89	91	94	96
% w/ Moderate TG's (150-199 mg/dL)	*14%*	*14%*	*15%*	*15%*	*15%*	*15%*	*16%*	*16%*	*16%*	*16%*	*17%*	*17%*
American's with Moderate TG Levels	34	35	36	37	38	39	40	41	42	43	44	45
% w/ High TG's (200-499 mg/dL)	*14%*	*14%*	*14%*	*14%*	*15%*	*15%*	*15%*	*15%*	*15%*	*15%*	*16%*	*16%*
American's with High TG Levels	34	35	35	36	37	38	38	39	40	41	41	42
% w/ Ultra High TG's (>500 mg/dL)	*2%*	*2%*	*2%*	*2%*	*2%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*	*3%*
American's with Ultra High TG Levels	5	5	5	6	6	6	7	7	7	8	8	8
Stage of Development	**P2**	**P3**		**NDA**	**Approval**							
Currently On Rx Fish Oil (Moderate TG's)	*0.05%*	*0.05%*	*0.05%*	*0.05%*	*0.05%*	*0.05%*	*0.05%*	*0.05%*	*0.05%*	*0.05%*	*0.05%*	*0.05%*
On Rx Fish Oil (High TG's)	*0.50%*	*0.52%*	*0.54%*	*0.56%*	*0.58%*	*0.60%*	*0.62%*	*0.64%*	*0.66%*	*0.68%*	*0.70%*	*0.72%*
On Rx Fish Oil (Ultra High TG's)	*5.00%*	*5.20%*	*5.40%*	*5.60%*	*5.80%*	*6.00%*	*6.20%*	*6.40%*	*6.60%*	*6.80%*	*7.00%*	*7.20%*
On Rx with Moderate TG Levels	0.017	0.017	0.018	0.018	0.019	0.019	0.020	0.020	0.021	0.022	0.022	0.023
On Rx with High TG Levels	0.169	0.180	0.191	0.202	0.213	0.225	0.237	0.250	0.263	0.276	0.290	0.304
On Rx with Ultra High TG Levels	0.242	0.267	0.293	0.321	0.351	0.382	0.414	0.448	0.484	0.522	0.561	0.603
Penetration Rate (Moderate TG's)	*0%*	*0%*	*0%*	*0%*	*0.00%*	*0.00%*	*0.00%*	*0.00%*	*0.00%*	*0.00%*	*0.00%*	*0.00%*
Penetration Rate (High TG's)	*0%*	*0%*	*0%*	*0%*	*2.00%*	*6.00%*	*12.00%*	*20.00%*	*20.00%*	*20.00%*	*20.00%*	*20.00%*
Penetration Rate (Ultra High TG's)	*0%*	*0%*	*0%*	*0%*	*5.00%*	*10.00%*	*15.00%*	*20.00%*	*20.00%*	*20.00%*	*20.00%*	*20.00%*
Active Patients on MAT9001	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
Price per Year	*$0*	*$0*	*$0*	*$0*	*$2,400*	*$2,520*	*$2,621*	*$2,699*	*$2,753*	*$2,781*	*$2,809*	*$2,837*
Estimated MAT9001 Sales (In Millions)	**$0**	**$0**	**$0**	**$0**	**$52.3**	**$130.2**	**$237.4**	**$377.0**	**$411.4**	**$443.9**	**$478.3**	**$514.5**

Source: Jason Napodano, CFA / Zacks Small-Cap Research

Matinas Biopharma exited the second quarter ending June 30, 2014 with $6.65 million in cash and investments. Burn from operations in the first and second quarter of 2014 totaled $2.3 and $1.9 million, respectively. We forecast burn in the third quarter 2014 totaled around the same amount. Thus, the cash position as of September 30, 2014 probably stood around $4.8 million. We expect burn in the fourth quarter will increase slightly as the company conducts the PK/PD study on MAT9001. Therefore, we forecast cash at year end 2014 will be around $2.5 million. This should be enough to continue to fund operations into March 2015.

All-in, we suspect Matinas will require roughly $30 million to fund the required Phase 3 trial with MAT9001 prior to filing the NDA in 2016. This does not include the potential for a second Phase 3 trial, if required, prior to the NDA filing. We believe the company will seek to raise a small amount of money in the near-term to fund operations through the PK/PD study data expected in April 2015. We suspect that a bridge financing of $5 million gets the company into the second half of 2015, putting them in a much better position to then fund the Phase 3 program in 2015 and 2016.

As of August 8, 2014, Matinas Biopharma had 32.0 million basic shares outstanding. The company has another 8.25 million issuable shares under the existing employee stock option program. Of this amount, 3.1 million is outstanding, 0.8 million of which is currently exercisable at $0.94 per share. There are 1.5 million warrants at $1.00 per share outstanding that expire in July 2018 and 13.75 million warrants at $2.00 per share that expire in July and August 2018. To fund operations through the U.S. NDA filing in late 2016 we believe Matinas will need to issue another 20+ million shares. This assumption includes the issuance of shares to raise the $5 million bridge loan at today's valuation plus raising a new $15 to $20 million in 2015 at slightly higher valuations post the U.S. Phase 1 data. By 2018, we suspect the basic outstanding share count will be in the area of 50 to 55 million. Including another 15 to 20 million in estimated warrants brings our fully-diluted share count to roughly 70 to 75 million.

As such, we believe the shares are fairly valued today at $1.25 per share. Our target is subject to revision based on the terms of the $5 million bridge financing, the results of the PK/PD data, and the timing and results of the pivotal U.S. Phase 3 study.

…Risks To Consider…

Today's price represents as attractive valuation for the long-term Matinas shareholder. However, we caution investors that the fish oil / omega-3 fatty acid industry has been a tumultuous one. Investors in Omthera did very well to see the company acquired by AstraZeneca for $323 million + $125 million in milestones in 2013. The $1.65 billion sales of Reliant to GlaxoSmithKline in 2007 set the bar very high. However, other players like Amarin and Acasti have not fared so well. Amarin's stock is down >95% from its summer 2011 highs. Matinas Biopharma, with a candidate that has yet to even enter U.S. clinical trials, is clearly late-to-the-game with MAT9001. Investors in Matinas should be excited about the science and market opportunity, but appreciate the risks in both clinical development and financial position. Right now, Matinas requires more cash today to develop MAT9001 through NDA filing that its current market capitalization. As such, investment in Matinas is a high risk / high reward endeavor.

Figure 19: Comparison Valuations In The Fish Oil / Omega-3 Industry Segment

Company	Product	Stage Of Development	Valuation
Reliant Pharma	Lovaza®	Approved (in 2004) Posted ~$250M in TTM sales prior to Sale	Sold to Glaxo for $1.65 billion (in 2007)
Omthera Pharma	Epanova®	Approved (in 2014) Post-Phase 3 at time of Sale to AstraZeneca	Sold to AstraZeneca for $323 million + $125 million in potential milestones (in 2013)
Amarin Pharma	Vascepa®	Approved (in 2012) Sales currently annualizing at ~$70M.	Current market capitalization = $182 million (based on 174M basic x $1.05 per share)
Acasti Pharma	CaPre®	Completed Phase 2 Trials. Phase 3 Planned for 2015 in U.S.	Current market capitalization = $57 million (based on 105M basic x $0.54 per share)
Matinas Biopharma	**MAT9001**	**PK/PD in Canada Phase 3 U.S. in 2H-2015**	**Current market capitalization = $19 million (based on 32M basic x $0.60 per share)**

Source: Jason Napodano, CFA / Zacks Small-Cap Research

PROJECTED FINANCIALS

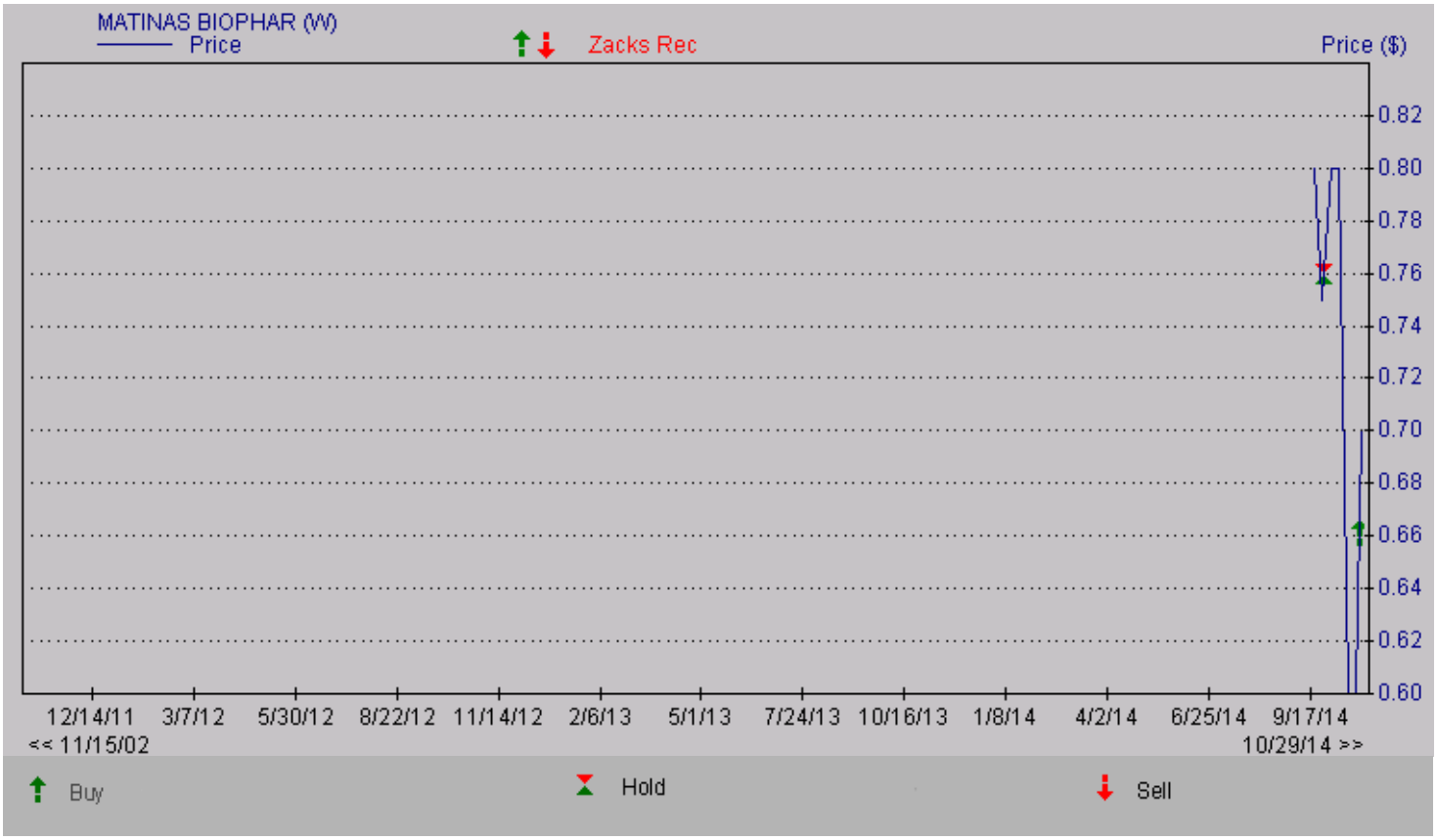
Matinas BioPharma – Income Statement

Matinas Biopharma	2013 A	Q1 A	Q2 A	Q3 E	Q4 E	2014 E	2015 E	2016 E
MAT9001	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**
YOY Growth	-	-	-	-	-	-	-	-
Total Revenues	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**
YOY Growth	-					-	-	-
Cost of Goods	$0	$0	$0	$0	$0	$0.0	$0.0	$0.0
Product Gross Margin	-	-	-	-	-	-	-	-
Research & Develop	$1.8	$1.1	$1.1	$1.2	$1.5	$4.9	$6.5	$7.5
General & Admin	$2.0	$1.1	$1.3	$1.1	$1.2	$4.7	$5.0	$5.5
Operating Income	($3.7)	($2.1)	($2.4)	($2.3)	($2.7)	($9.5)	($11.5)	($13.0)
Net Other Income	($0.0)	($0.0)	($0.0)	($0.0)	($0.0)	($0.1)	($0.1)	($0.1)
Pre-Tax Income	($3.7)	($2.1)	($2.4)	($2.3)	($2.7)	($9.6)	($11.6)	($13.1)
Taxes	$0	$0	$0	$0	$0	$0	$0	$0
Tax Rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	($3.7)	($2.1)	($2.4)	($2.3)	($2.7)	($9.6)	($11.6)	($13.1)
Net Margin	-	-	-	-	-	-	-	-
Reported EPS	**($0.20)**	**($0.07)**	**($0.08)**	**($0.07)**	**($0.09)**	**($0.30)**	**($0.23)**	**($0.17)**
YOY Growth	-	-	-	-	-	-	-	-
Wt. Ave Shares Outstanding	19.0	32.0	32.0	32.0	32.0	32.0	50.0	75.0

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

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

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, 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, equity 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 placed on the ZIR, SCR, or Zacks & Co. restricted list, which may include issuers in the Small-Cap Universe. 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 1126 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- 15.8%, Hold/Neutral- 78.3%, Sell/Underperform – 5.7%. Data is as of midnight on the business day immediately prior to this publication.