Esperion Therapeutics, Inc. (ESPR)

Initiating Coverage at Market Outperform; Potential for a Blockbuster Cholesterol-Lowering Drug

MARKET DATA

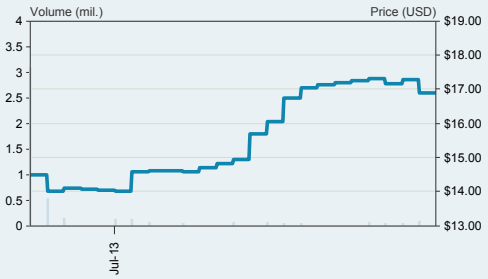
Price	$16.90
52-Week Range:	$13.55 - $18.95
Shares Out. (M):	15.3
Market Cap ($M):	$258.6
Average Daily Vol. (000):	85.0
Cash (M):	$84
Cash/Share:	$5.48
Enterprise Value (M):	$175

Source: Thomson Reuters and JMP Securities LLC

FY DEC		2012A	2013E	2014E
Revenue ($M)	1Q	--	$0.0A	--
	2Q	--	$0.0	--
	3Q	--	$0.0	--
	4Q	--	$0.0	--
	FY	**$0.0**	**$0.0**	**$0.0**
EPS	1Q	--	($0.84)A	--
	2Q	--	($0.58)	--
	3Q	--	($0.35)	--
	4Q	--	($0.56)	--
	FY	**($3.13)**	**($2.02)**	**($3.68)**

Source: Company reports and JMP Securities LLC

STOCK PRICE PERFORMANCE



MARKET OUTPERFORM | Price: $16.90 | Target Price: $25.00

INVESTMENT HIGHLIGHTS

We are initiating coverage on Esperion Therapeutics with a Market Outperform rating and $25 price target. Esperion's lead product candidate is ETC-1002, a novel small molecule therapy in Phase 2 development for the treatment of high cholesterol (LDL-C). The company has completed three Phase 2a trials demonstrating consistent LDL-C lowering efficacy and results from a fourth trial are anticipated in 3Q13. In addition, the safety and tolerability profile seen to date is favorable and may enable differentiation from current standards of care, specifically with respect to the muscle-related side effects associated with statins. We believe that ETC-1002 may offer a novel and attractive option in the treatment of high cholesterol, an established multi-billion dollar market. The company completed its IPO on June 26 and we believe ESPR shares will continue to outperform peers as results from the Phase 2a trial in residual risk patients are announced and the program advances into Phase 2b development. Our $25 price target is derived through a risk-adjusted NPV analysis of ETC-1002.

Demonstrated efficacy, with favorable safety/tolerability profile seen so far. In completed Phase 2a trials, ETC-1002 has demonstrated LDL-C lowering efficacy of 30-40%, which we believe can enable competitive positioning of the drug among current and emerging treatment options. The first indication for the drug is in patients with elevated LDL-C who do not tolerate statins due to muscle-related side effects, affecting up to an estimated 20% of patients receiving these drugs (~ six million people in the U.S.). In our view, a key determining factor of the drug's success will be confirming the favorable safety and tolerability profile seen to date, especially given the advantages of convenient once-daily oral administration.

Additional Phase 2a results anticipated in 3Q13, representing significant upside to our valuation. Results from a Phase 2a trial evaluating ETC-1002 as an add-on to statins in patients who are not adequately controlled on statins alone, are anticipated in 3Q13. The commercial opportunity in this patient population, referred to as residual risk, could be substantially greater than in the statin-intolerant opportunity. It is estimated that as many as 11 million patients have not achieved the National Institutes of Health's (NIH) cholesterol treatment goal despite statin therapy. Currently, sales of ETC-1002 in the residual risk setting represent upside to our revenue estimates and valuation.

ETC-1002 can become a highly sought-after asset. We view ETC-1002 as an asset with high scarcity value and business development potential. Furthermore, we believe that a large pharmaceutical partner is necessary to successfully complete late-stage development of a cholesterol-lowering drug, given the likely size and cost of a Phase 3 program, and to effectively commercialize the product. We view results from the soon-to-be-initiated Phase 2b trials, expected by the end of 2014, as a key value-inflection point for the asset and likely catalyst for partnership and/or acquisition transactions.

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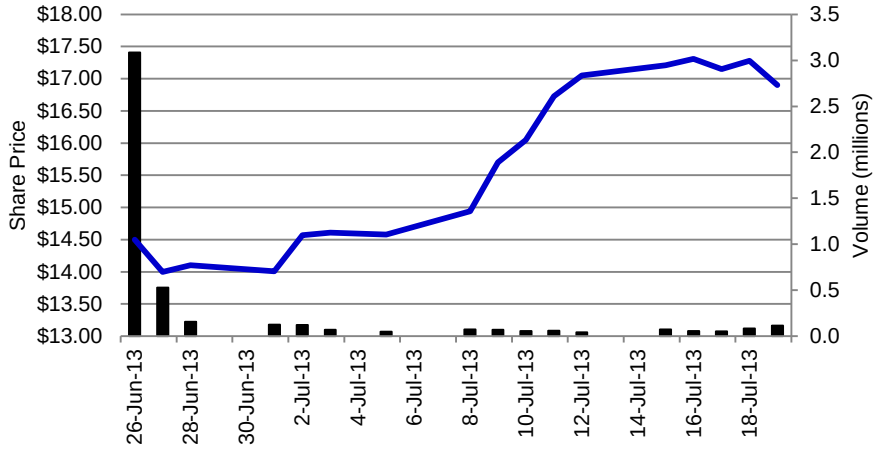
COMPANY DESCRIPTION

Esperion Therapeutics is a biopharmaceutical company focused on the discovery, development and commercialization of novel treatments for patients with elevated levels of low-density lipoprotein cholesterol (LDL-C) and other cardiometabolic risk factors. The company's lead development candidate is ETC-1002, an orally available small molecule therapy being developed for patients with elevated levels of LDL-C. The drug acts in the liver to: 1) inhibit ATP-citrate lyase, a key enzyme that supplies substrate for cholesterol and fatty acid synthesis, as well as glucose production, and 2) activate AMP kinase. The initial development focus for ETC-1002 is in patients who are not able to tolerate therapy with statins (the standard of care treatment for elevated LDL-C) and as an add-on to statins in patients who remain inadequately controlled on therapy. ETC-1002 is a wholly owned asset with an issued composition-of-matter patent providing protection in the U.S. at least through December 2025.

FIGURE 1. Historical Stock Chart



Source: Thomson Reuters

KEY UPCOMING MILESTONES

3Q13	Phase 2a (ETC-1002-007) top-line data readout in residual risk subjects
4Q13	Phase 2b (ETC-1002-008) trial initiation in statin-intolerant subjects
4Q13	Phase 2b (ETC-1002-009) trial initiation in residual risk subjects
YE14	Phase 2b (ETC-1002-008) top-line data in statin-intolerant subjects
YE14	Phase 2b (ETC-1002-009) top-line data readout in residual risk subjects
1H15	Potential end-of-Phase 2 FDA meeting

INVESTMENT THESIS

Esperion is a biopharmaceutical company focused on the development of ETC-1002, a novel small molecule drug candidate for the treatment of elevated LDL cholesterol. The company has completed six Phase 1 and 2 trials with ETC-1002 demonstrating consistent improvements in LDL cholesterol levels. The safety of the drug candidate seen to date is also favorable, with no evidence of the muscle-related adverse events (pain and weakness) associated with statin therapy. The commercial opportunity for ETC-1002 is attractive, potentially addressing blockbuster markets with unmet medical needs in patients who are intolerant to or inadequately controlled with current standard of care treatments.

Development focus on patients who do not tolerate or inadequately respond to statins. It is well established that lowering LDL-C reduces the risk of adverse cardiovascular outcomes, including heart attacks, stroke and deaths from cardiovascular disease. Statins are the current standard of care for lowering LDL-C for approximately 30 million patients in the U.S. Muscle-related symptoms (pain or weakness) occur in up to 20% of patients on statin therapy and two million patients have discontinued treatment due to these adverse effects. A further 11 million adults in the U.S. are not adequately controlled on statins and could benefit from an additional treatment option. ETC-1002 works by a different mechanism of action to statins, one that is upstream in the biomolecular pathway of LDL cholesterol synthesis, and therefore may provide a differentiated treatment option. Another class of injected drugs, PSCK9s, is also being developed for statin-intolerant and/or statin-resistant patients; however, we believe an oral therapy such as ETC-1002 could be an attractive alternative.

Results from a Phase 2a trial for ETC-1002 demonstrated ~30% reduction in LDL cholesterol in patients intolerant to statin therapy. In June 2013, the company reported top-line results from a Phase 2a trial for ETC-1002 in patients intolerant to two or more statins. Results demonstrated that ETC-1002 lowered LDL-C by an average of 32%. ETC-1002 was well tolerated and no patients treated with ETC-1002 discontinued the trial because of muscle pain or weakness. There were also no signals of elevated liver enzymes. The most commonly used drug in this patient population is Merck's Zetia, which lowers LDL cholesterol by ~18%. In our view, these results support the consistent evidence of potent LDL-C lowering efficacy observed in clinical trial experience with ETC-1002 to date. Results from a Phase 2a trial in patients who are not adequately controlled on statin therapy (residual risk patients) are anticipated in 3Q13 and, in our view, the results could substantially broaden the commercial potential for the drug and drive upside to our current valuation assumptions.

ETC-1002 development funded by IPO through the end-of-Phase 2 meeting with the FDA. Phase 2b trials in statin intolerant/residual risk populations are expected to begin in 4Q13. We expect Phase 2b results by the end of 2014 and an end-of-Phase 2 meeting with the FDA in 1H15. In our view, the regulatory pathway for a novel LDL-C lowering drug such as ETC-1002 has become increasingly well defined. The FDA has communicated a clear definition for the statin intolerant indication as being patients who have a history of intolerance to two or more statins, including at least one at the lowest approved dose, due to muscle pain or weakness. Furthermore, we gain comfort from the precedent being established with the PCSK9 inhibitors that a cardiovascular outcomes trial will not be required prior to approval for ETC-1002. We also note that since PCSK9 inhibitors are more advanced in development than ETC-1002, the regulatory reviews for these drugs could provide insights into regulatory questions or hurdles that must be addressed.

Management has demonstrated success in cardiometabolic diseases, including a key involvement in the discovery of statins and the prior sale of Esperion to Pfizer for $1.3 bil. The founder of Esperion, and the company's Executive Chairman and Chief Scientific Officer is Roger S. Newton, Ph.D., FAHA, who co-discovered Lipitor. Previously, Esperion was focused on the development of drugs targeting HDL cholesterol and was sold to Pfizer in 2004 for $1.3 bil. The company and its assets, including ETC-1002, were spun out into an independent company in 2008.

VALUATION

We value Esperion through a risk-adjusted NPV analysis of ETC-1002 revenues. Our valuation includes sales of ETC-1002 in the statin-intolerant LDL-C indication alone.

As outlined in our revenue model (see page 24), we project sales of the drug from launch in 2019 through to patent expiration in April 2030. We estimate that the drug achieves peak U.S. sales of ~$1.8 bil. in 2029 and global sales in that year in excess of $3.5bil.

Our model assumes that Esperion secures a partner for the drug following the end-of-Phase 2 meeting with the FDA in 1H15 and receives royalties on global sales at a rate of 20%. Following patent expiration, we assume a terminal growth rate of -70%, reflecting likely generic competition. Our NPV analysis of royalty revenue to Esperion includes a 55% probability of success and a contribution margin ranging from 65-70%, which takes into consideration the costs of maintaining the business and taxes. We include a discount rate of 12.5% and divide by our estimate for fully diluted shares outstanding of ~17.5million.

FIGURE 2. Risk-Adjusted NPV Valuation

	Sales ($MM)	Royalty rate	Royalties ($MM)	Revenue year	rNPV ($MM)	rNPV per share
ETC-1002						
U.S.	1,786	20%	357	2029	212	**$12.14**
Europe	1,261	20%	252	2029	127	**$7.25**
ROW	630	20%	126	2029	65	**$3.72**
Enterprise value						**$23.11**
Net cash (YE14)					26	**$1.47**
Price target						**$24.59**
Shares outstanding					17.5	

Source: JMP Securities LLC

The potential for ETC-1002 in the residual risk market opportunity currently reflects upside to our revenue projections and valuation. We look to results from the ongoing Phase 2a trial (ETC-1002-007), anticipated in 3Q13, to validate the potential for the drug in this patient population. We believe that the sales potential for ETC-1002 in the U.S. residual risk market could exceed $715MM at peak in 2029, assuming only a mid-single digit percentage market share and positive results from the Phase 2a trial could represent upside to our valuation of ~$10-15 per share. We do not currently include the company's early-stage pipeline assets in our valuation.

Financials

Capital Structure

Through its IPO, Esperion sold 5.75 million shares, including the full exercise of the over-allotment option, at $14 per share, raising gross proceeds of $80.5MM and receiving net proceeds of approximately $72.8MM.

Following the completion of the IPO, the total basic shares outstanding was approximately 15.3 million. There are an additional 0.7 million stock options outstanding and 0.3 million shares issuable upon the exercise of warrants. About 1.2 million shares of common stock were reserved for future issuance under equity incentive plans. The fully diluted number of shares outstanding is approximately 17.5 million.

Balance Sheet

Following the completion of the IPO, Esperion has a pro forma cash position of approximately $84MM. We view current cash as sufficient to fund operations through 1H15. This includes completing the two planned Phase 2b trials in 2014 and participating in an end-of-Phase 2 meeting with the FDA in 1H15.

Intellectual Property for ETC-1002

ETC-1002 is claimed in U.S. Patent No. 7,335,799, covering the drug's composition of matter. This patent expires in December 2025 and may be eligible for a patent term extension period of up to five years. At least one pending U.S. patent application claims a method of treatment using ETC-1002. There are currently three issued patents and four pending applications in countries outside the United States that relate to ETC-1002.

INVESTMENT RISKS

Clinical Risk. Esperion's product candidates may fail to demonstrate adequate efficacy, safety, and/or tolerability in one or more clinical studies.

Regulatory risk. The FDA and/or other ex-U.S. regulatory agencies could reject any of the firms', or its partners', future regulatory filings or require additional studies prior to granting approval.

Industry Risk. The biopharmaceutical industry is highly competitive, with many firms developing novel therapies that may address Esperion's target diseases. It is possible that breakthrough competitor products or therapies may render the company's products obsolete and affect the future survival of the company.

Balance Sheet Risk. The company has a history of losses, and has not yet established a track record of consistent profitability. While we project that the company will not need to raise additional capital to maintain profitability, it may be necessary to do so to fund the business model. Following the closing of the IPO, Esperion had a pro forma cash position of approximately $84MM and we project that the company will end 2013 with cash of $82.7MM.

CHOLESTROL-LOWERING THERAPIES

Cardiovascular Disease and Hypercholesterolemia

Cardiovascular disease is the leading cause of death and disability in western societies, leading to heart attacks, strokes and other adverse cardiovascular events. The American Heart Association (AHA) estimates that in 2009, approximately 800,000 deaths in the U.S. were caused by cardiovascular disease. Elevated LDL-C is a recognized as a significant risk factor for cardiovascular disease. According to estimates from the Centers for Disease Control (CDC), 71 million adults in the U.S. have elevated LDL-C levels. Elevated LDL-C can cause atherosclerosis, where excess cholesterol and other lipids are deposited in the walls of arteries as plaque, which can lead to cardiovascular disease.

It has been well established, through a broad body of clinical research, including many large clinical trials, that lowering elevated levels of LDL-C translates into reduced risk of cardiovascular disease. This was first shown in the early 1980's through the Lipid Research Clinics Coronary Primary Prevention Trial which demonstrated that treatment with cholestyramine, a bile acid sequestrant, led to a 20% reduction in LDL-C with a 19% reduction in risk of cardiovascular disease death or nonfatal myocardial infarction, or heart attack. Common therapies for hypercholesterolemia and the associated key side effects are shown in Figure 3.

FIGURE 3. Common Hypercholesterolemia Therapies

Class of Therapy	Exemplary products	Labeled Indication	Average LDL-C Change from Baseline	Key Side Effects
Statins	Lipitor (atorvastatin), Crestor (rosuvastatin), Zocor (simvastatin)	Reduction in LDL-C	Up to 63%	• Skeletal muscle effects (e.g., myopathy and rhabdomyolysis) • FDA recently warned that people being treated with statins may have an increased risk of raised blood sugar levels and the development of type 2 diabetes
Fixed combination therapies	Vytorin (ezetimibe / simvastatin)	Reduction in LDL-C	Up to 63%	• Includes a statin as one of the underlying therapies and therefore contains the same side effects outlined above
Bile acid sequestrants	Welchol (colesevelam), Questran (cholestyramine)	Reduction in LDL-C*	Up to 20%	• Gastrointestinal disorders
Cholesterol absorption	Zetia (ezetimibe)	Reduction in LDL-C	Up to 18%	• Limited
Niacin	niacin, Niaspan (niacin ER)	Reduction in LDL-C; Reduction in recurrent myocardial infarction	Up to 17%	• Flushing (i.e., warmth or redness) hepatic toxicity and skeletal muscle effects
Fibrates	Bezalip (bezafibrate), Modalim (ciprofibrate), Tricor (fenofibrate)	Reduction in triglycerides and LDL-C	Up to 21%	• Gallstones, skeletal muscle effects and liver disorders

** Welchol, a bile acid sequestrant, is also approved for improving glycemic control in adults with type 2 diabetes.*

Source: Company reports

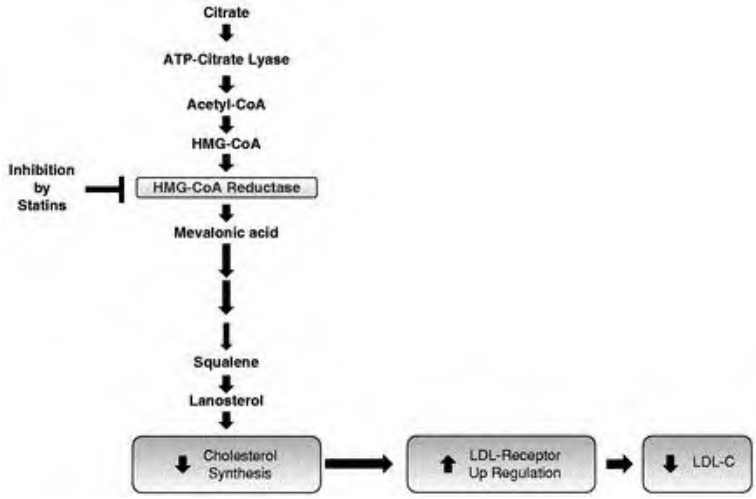
According to the most widely followed clinical practice guidelines for the management of dyslipidemia in the U.S., the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III), cholesterol is a primary target for the treatment of cardiovascular disease and LDL-C is the primary focus of cholesterol-lowering therapy. Together with other cardiovascular risk factors, including a familial history of cardiovascular disease, additional lipid parameters, as well as a history of smoking or type 2 diabetes, the ATP III guidelines provide a structure for the medical management of cholesterol. The guidelines recommend multiple pharmacotherapy approaches to achieve LDL-C lowering, beginning with statins and also including bile acid sequestrants, nicotinic acid, and fibric acids.

Statins

Statins represent the first line of therapy in treating hyperlipidemia, and are generally highly effective at lowering LDL-C. This class of drugs includes simvastatin (Zocor), atorvastatin (Lipitor), fluvastatin (Lescol), lovastatin (Mevacor), pitavastatin (Livalo), pravastatin (Pravachol), and rosuvastatin (Crestor). According to a National Health and Nutrition Examination Survey, about 25% of Americans over the age of 45 were treated for elevated LDL-C levels with a statin therapy from 2005 to 2008.

Statins are selective, competitive inhibitors of HMG-CoA reductase, a rate-limiting enzyme in the cholesterol biosynthesis pathway, and function primarily in liver cells (see Figure 4). Statin-mediated inhibition of cholesterol biosynthesis leads to an increase in the number of LDL receptors on the surface of liver cells, which enhances uptake of LDL particles into liver cells from the circulation, thereby decreasing levels of LDL-C.

FIGURE 4. Mechanism of Statin Inhibition



Source: Company reports

While statins have proven to be efficacious across a broad population, there are significant subgroups of the population for whom statins are intolerable due to muscle pain or weakness, memory loss or increased glucose levels (statin intolerant population). Moreover, a significant number of patients are unable to reach their LDL-C goal on statin therapy alone (residual risk population). Additionally,

safety/tolerability concerns are becoming more exposed, leading to the FDA issuing an update on the risks of statin. In February 2012, the agency issued a communication advising consumers and health care professionals that:

- Routine monitoring of liver enzymes in the blood is no longer needed, as has not been found to be effective in predicting or preventing the rare occurrences of serious liver injury associated with statin use,
- Cognitive impairment, such as memory loss, forgetfulness and confusion, has been reported by some statin users,
- Statin patients may have an increased risk of raised blood sugar levels and development of type 2 diabetes, and
- Certain drug/drug interactions with lovastatin can increase the risk of muscle damage.

As such, we believe that there is a substantial opportunity for new therapeutic alternatives and believe that ETC-1002 could provide a compelling treatment option. We discuss the commercial opportunities for ETC-1002 in further detail on pages 20-22.

Other Therapies

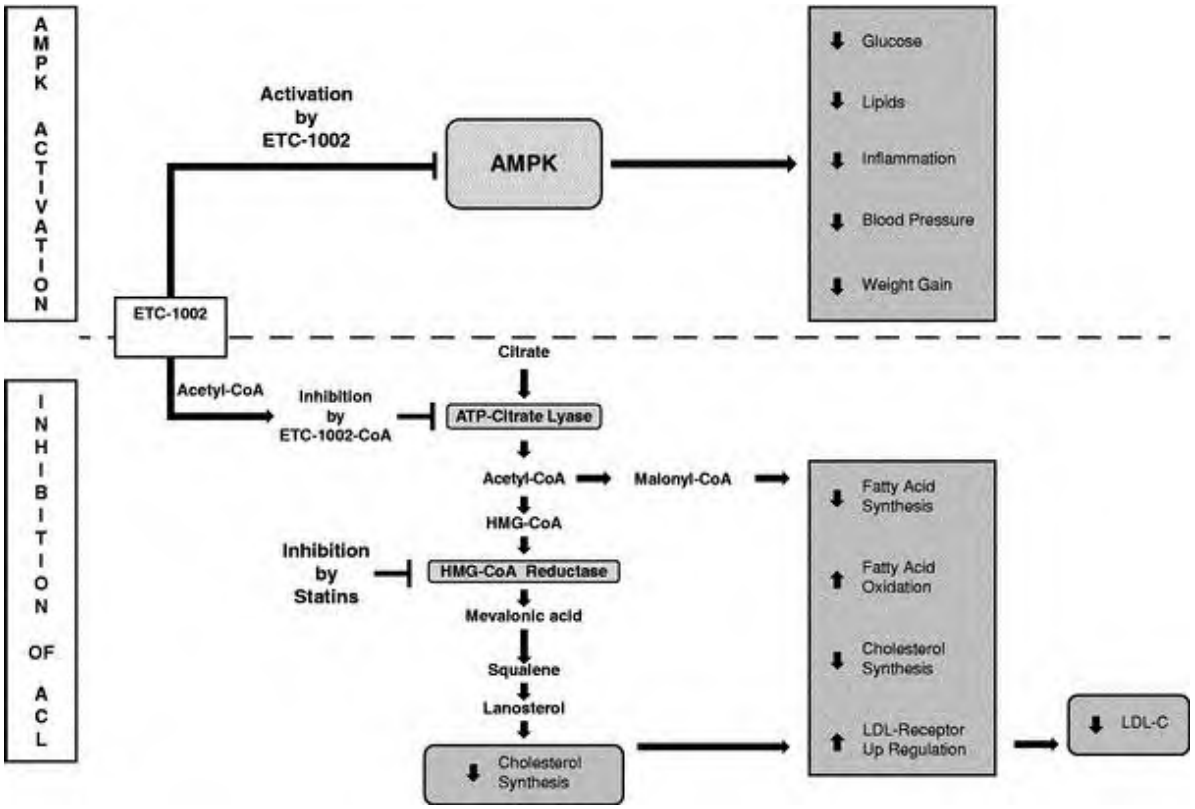
Figure 3 illustrates common therapies used to treat hypercholesterolemia: Beyond the front-line treatment option of statins, we are focused on drugs that are currently most commonly used in patients who do not tolerate or adequately respond to statin therapies. The first of these drugs is Merck's Zetia (ezetimibe), which is the most broadly used alternative therapeutic among statin-intolerant subjects. Ezetimibe works by inhibiting the absorption of cholesterol in the intestine. In particular, it appears to bind to a mediator of cholesterol absorption, the Niemann-Pick C1-Like 1 (NPC1L1) protein, on both the gastrointestinal tract epithelial cells as well as in hepatocytes. Secondary to this effect, decreased cholesterol absorption leads to an upregulation of LDL-receptors on the surface of cells, which increase LDL-cholesterol metabolism, thereby decreasing levels of LDL in the blood plasma. Another currently used treatment alternative to statins is the bile acid sequestrant Welchol (colesevelam), marketed in the U.S. by Daiichi Sankyo. While both of these alternatives are viable, their efficacy is inferior to statins leaving, in our view, a significant opportunity for new therapeutic options.

Additionally, two drugs were recently approved for the treatment of severely high levels of cholesterol in patients with familial hypercholesterolemia. The first is Aegerion's Juxtapid (lomitapide), approved in December 2012 for treating patients with homozygous familial hypercholesterolemia (HoFH). LDL-C was shown to be lowered by approximately 40% after 26 weeks of treatment. However, besides significant tolerability issues associated with Juxtapid, the drug is associated with liver enzyme abnormalities and accumulation of fat in the liver, and carries a boxed warning regarding serious risk for hepatic toxicity. The second drug is Sanofi Aventis/Isis' Kynamro (mipomersen sodium), approved in January 2013. This is a once-per-week injection for treating HoFH. Out of 51 patients, Kynamro reduced LDL-C levels by approximately 25% after 26 weeks. Like Juxtapid, Kynamro also carries a boxed warning on the serious risk of liver toxicity as it is associated with liver enzyme abnormalities and accumulation of fat in the liver, which could lead to progressive liver disease with chronic use. Given these two products' safety and tolerability profiles, we anticipate that the use of these drugs will remain in patients with severely high levels of cholesterol, a point we believe is supported by the orphan drug pricing strategy these companies have adopted.

ETC-1002 – A Novel and Differentiated Option

ETC-1002 is a novel, first-in-class, orally available, once-daily LDL-C lowering, small molecule therapy. It possesses dual mechanisms of action that can potentially regulate both lipid and carbohydrate metabolism. ETC-1002 inhibits ATP citrate lyase (ACL) and activates 5'-adenosine monophosphate-activated protein kinase (AMPK; see Figure 5). Pre-clinical studies have demonstrated that upon entering the liver, ETC-1002 is converted to a derivative coenzyme, ETC-1002-CoA, which then directly inhibits ACL, a key enzyme that supplies substrate for cholesterol and fatty acid synthesis, as well as glucose production in the liver. Both ACL and AMPK play critical roles in cholesterol and glucose synthesis in the liver, therefore regulation of these enzymes is complementary. ETC-1002-mediated cholesterol synthesis Inhibition in the liver causes the liver to take up LDL particles from the blood, thereby lowering blood LDL-C levels.

FIGURE 5. ETC-1002 Mechanism of Action - ACL-Dependent Inhibition of Hepatic Cholesterol Synthesis and AMPK Activation



Source: Company reports

ETC-1002's activation of AMPK complements the effects of its ACL inhibition in the liver and contributes to the beneficial effects on other cardiometabolic risk factors including hsCRP, insulin sensitization, blood pressure and weight. While the relative contributions of ACL inhibition and AMPK activation are not yet known, this mechanism is supported by pre-clinical and clinical observations that have been published in peer reviewed publications and presented at scientific conferences. We are not aware of any alternative explanations regarding ETC-1002's dual mechanism of action or the preliminarily accepted conclusion in the scientific community that inhibiting ACL and activating AMPK have the potential to regulate metabolic imbalances in both the lipid and carbohydrate metabolic pathways, which do not function normally in particular patient populations with specific cardiometabolic risk factors.

ETC-1002 and Statins Lower LDL-C via Different Mechanisms

While ETC-1002 and statins lower LDL-C levels to a similar extent, they target different enzymes in the cholesterol synthesis pathway. ETC-1002 has dual mechanisms of action that activate AMPK and inhibit ACL, whereas statins act solely through direct inhibition of HMG-CoA reductase. Both forms of therapy reduce LDL-C by lowering cholesterol synthesis and thereby increasing LDL receptors in the liver. Thus, ETC-1002 and statins may be complementary, and potentially could be used in combination to achieve further lowering of LDL-C.

ETC-1002 CLINICAL DEVELOPMENT PROGRAM

To date, Esperion has completed six clinical trials evaluating ETC-1002, including three Phase 1 trials and three Phase 2a trials. A fourth Phase 2a trial is ongoing and results are anticipated in 3Q13. The ETC-1002 clinical program is outlined in Figure 6 and LDL-C lowering efficacy is summarized in Figure 7. Trial designs and results and detailed below.

Based on the results of these seven trials, the company intends to initiate two Phase 2b trials in 1H14, evaluating ETC-1002 in two patient populations with elevated LDL-C levels:

- patients with a history of statin intolerance to two or more statins
- patients who do not adequately respond to statin treatment

The two Phase 2b trials are intended to inform the design of a Phase 3 program and the company currently intends to request an end-of-Phase 2 meeting with the FDA in 1H15, following the completion of these trials.

FIGURE 6. ETC-1002 Clinical Program

				Subjects	
Trial	**Phase**	**Objective**	**Treatment duration**	**Total**	**Treated**
Completed					
ETC-1002-006	2a	PoC in Hypercholesterolemia Subjects with a History of Statin Intolerance	8 weeks	56	37
ETC-1002-005	2a	PoC in Type 2 Diabetes Subjects	4 weeks	60	30
ETC-1002-004	1b	Multiple-Dose Tolerance Greater Than 120 mg	2 weeks	24	18
ETC-1002-003	2a	PoC in Hypercholesterolemic Subjects	12 weeks	177	133
ETC-1002-002	1b	Multiple-Dose Tolerance	2 weeks / 4 weeks	53	39
ETC-1002-001	1a	Single-Dose Tolerance	Single dose	18	18
Ongoing					
ETC-1002-007	2	PK Interaction of ETC-1002 and atorvastatin	8 weeks	52	52
Planned					
ETC-1002-008	2b	Dose-Ranging Study in Statin Intolerant Subjects	12 weeks	n/a	n/a
ETC-1002-009	2b	Dose-Ranging Study in Residual Risk Subjects	12 weeks	n/a	n/a

Source: Company reports

Ongoing Phase 2a trial and planned Phase 2b program

ETC-1002-007 - In addition to the completed Phase 2a trial in patients with a history of muscle-related intolerance to statins, Esperion is completing a Phase 2a trial (ETC-1002-007; NCT01779453) in patients who do not adequately respond to statin therapy. This trial is evaluating ETC-1002 as an add-on to statins and top-line results are anticipated in 3Q13.

The primary goal of this ~50 patient trial is to assess for any pharmacokinetic interactions between ETC-1002 and atorvastatin (10 mg). Patients enrolled into the trial received atorvastatin (10 mg) for four weeks to achieve steady-state levels and were then randomized (3:1) to receive atorvastatin in combination with ETC-1002 or placebo for eight weeks. Patients randomized to the ETC-1002 arm will be titrated. During this clinical trial, patients in the drug arm will be forced titrated (60 mg, 120 mg, 180 mg, and 240 mg doses) every two weeks.

Planned Phase 2b trials – Esperion expects to initiate two Phase 2b trials for ETC-1002 in 4Q13. The first (ETC-1002-008) is in patients with a history of intolerance to statins. As demonstrated in the ETC-1002-006 trial (discussed below), ETC-1002 has demonstrated LDL-C lowering efficacy and a favorable safety/tolerability profile in patients who are not able to tolerate statins due to muscle-related pain and/or weakness. The goal of this trial will be to demonstrate superior efficacy to Zetia, the current standard of care in statin-intolerant patients, along with comparable tolerability.

The trial will enroll patients with elevated LDL-C levels who are intolerant to two or more statins due to muscle-related adverse events. The trial is expected to evaluate two or three doses of ETC-1002, vs. Zetia, over a 12-week treatment period.

A second Phase 2b trial is planned to be initiated based on the results of the ongoing Phase 2a trial (ETC-1002-007) in patients who do not adequately respond to statin therapy (the residual risk population. This trial is also expected to be 12 weeks in duration and is intended to demonstrate that ETC-1002 added onto statin therapy improves LDL-C goal achievement.

These two Phase 2b trials are expected to have results by the end of 2014 and inform the design of a Phase 3 program, to be discussed in an end-of-Phase 2 meeting with the FDA, potentially in 1Q15.

Summary of clinical results seen to date

In our view, the clinical profile of ETC-1002 demonstrated in clinical trials to date support consistent, statistically significant evidence of lowering LDL-C levels, as well as improvements in additional markers of cardiometabolic risk including blood pressure, glycemic control, body weight and the inflammation marker C-reactive protein (hsCRP). As summarized in Figure 7, trials to date have shown LDL-C reductions of ~30-40% including a 32% LDL-C lowering in the most recently completed Phase 2a trial in patients intolerant to statins.

FIGURE 7. Summary of ETC-1002 Phase 2a Results

Patient Population	Avg. Reduction in LDL-C from Baseline	p-value
Elevated LDL-C and Statin Intolerant	Up to 32%	<0.0001
Elevated LDL-C	Up to 27%	<0.0001
Type 2 Diabetes and Elevated LDL-C	Up to 43%	<0.0001

Source: Company reports

Safety/Tolerability Discussion

We believe that the safety and tolerability profile of ETC-1002 will be a key determinant of the drug's commercial potential. Other drugs including Aegerion's Juxatpid and Sanofi/Isis's Kynamro have demonstrated potent LDL-C lowering efficacy. However, we believe their use will remain restricted to very high-risk patient populations due to unfavorable safety profiles. Below we address what we expect to be the key safety topics of discussion.

In our view, while we acknowledge that additional confirmatory data are required, the data available support a favorable and differentiated safety and tolerability profile, with no evidence of adverse effects on liver enzymes or muscle intolerability issues.

Pre-clinical toxicology studies

ETC-1002 is currently subject to two partial clinical holds by the FDA. While we are not overly concerned by the reasoning for either of the clinical holds, we look to gain greater clarity in 2014. Positively, in our view, we anticipate that both pre-clinical issues will have been addressed prior to the completion of the planned Phase 2b trials and the end-of-Phase 2b meeting with the FDA.

The first clinical hold currently limits the company's ability to include doses of greater than 240 mg. This relates to a pre-clinical primate study and, in our view, the issue raised was unlikely drug related. More importantly, we believe the issue has been adequately addressed by two subsequent studies that evaluated appropriate doses and durations of therapy and did not raise the same issue. Furthermore, based on the dose-ascending Phase 1 and II trials completed and the potent LDL-C lowering efficacy observed, Esperion does not anticipate pursuing doses of ETC-1002 above this threshold.

The second clinical hold limits the duration of clinical trials evaluating ETC-1002 to six months. This hold related to a 2009 determination by the FDA that ETC-1002 was sufficiently similar to a potential peroxisome proliferator activated receptor, or PPAR, agonist. The FDA requires that all drugs in the PPAR class, or that may be deemed to have PPAR-like qualities, must complete two-year, pre-clinical animal carcinogenicity studies before initiating Phase 3 clinical trials of longer than six months. This is due to prior experiences with drugs in the PPAR class causing such toxicities. In our view, this concern is theoretical and we note that the PPAR class includes a broad array of compounds of which many do not impart carcinogenicity risks. For example, the fibrates are PPAR-alpha agonists and are widely used therapies for dyslipidemia.

Furthermore, and in our view specifically relevant to ETC-1002, clinical data available for the drug so far support an absence of PPAR-mediated pharmacology, which typically includes triglyceride decreases, adiponectin increases, mild ALT increases, or toxicity (e.g., weight gain, edema, creatinine kinase/creatinine increases). While we acknowledge that results from the animal carcinogenicity will be necessary to fully alleviate this issue, we are confident that it is only a theoretical risk. Moreover, results from both the rat and mouse studies will be available by mid-2014, prior to the completion of the planned Phase 2b trials and the end-of-Phase 2 meeting with the FDA.

Liver function enzymes

Given the liver's central role in lipid metabolism, drugs that target dyslipidemia, and commonly lipid metabolism, therefore typically act on this organ predominantly or specifically. As a result, the effect of the impact and potential for adverse effects on the liver are of primary consideration when evaluating novel treatments for dyslipidemia and hypercholesterolemia. As with statins, the mechanism of ETC-1002 targets LDL-C metabolism and has it primary activity in the liver. In pre-clinical and clinical studies to date, assessment of liver function enzymes has been a key focus of the development program.

Based on the available data, including three completed Phase 2a trials with approximately 200 patients exposed to the drug for up to eight weeks, we do not believe there is any evidence for concern for liver toxicity with ETC-1002. Specifically, we gain comfort from the finding that no patient in the Phase 2a trials treated so far with ETC-1002 has experienced substantial elevations in liver function tests greater than three times the upper level of normal that have been drug-related and no patient experienced substantial elevations of creatine kinase greater than five times the upper limit of normal. Furthermore, only one patient, in the ETC-1002-003 trial, experienced liver function tests more than three times the upper limit of normal, and this was determined by the investigator not to be drug related as it coincided with a confirmed acute viral infection.

However, we acknowledge that investors will be looking to both larger number of patients and increased durations of treatment to gain greater certainty on this issue in the Phase 2b and Phase 3 development program. We will also be focused on the upcoming results of the ETC-1002-007 trial, evaluating ETC-1002 as an add-on to statin therapy. Given that both drugs act primarily in the liver, we view it as important to ensure that there is no evidence of pharmacokinetic interaction between these drugs and no unexpected impact on liver function enzymes.

Muscle-related adverse events

In our view, the most important tolerability debate for ETC-1002 is and will continue to be the incidence and severity of muscle related pain and weakness. This is because we view these adverse events to be an important limitation of statin therapy and potential point of clinical and commercial differentiation for ETC-1002.

While statin-induced myalgia is strongly dose-dependent, its mechanism of action is not clear, which have made it difficult to develop treatments or preventative measures. One hypothesis centers on the portion of the population having genetically impaired organic anion transporters. This results in an inability for statins to be taken up in the liver, and they therefore accumulate in the blood and ultimately concentrate in the muscles. This build-up of drug in muscle may then be causing pain and weakness. However, it should also be noted that statin-associated myalgia also occurs in patients who do not have impaired organic anion transporters. Again, the cause of pain and weakness is thought to be related to stating accumulation in muscle tissue.

Importantly, to date there has been no evidence of muscle pain or weakness due to ETC-1002. It should be noted that although both statins and ETC-1002 have their primary activity in the liver, they are taken up by different cell surface receptors and transporters. Additionally, ETC-1002 does not appear to accumulate in patients with impaired organic anion transporters.

Hemoglobin and uric acid changes

In clinical studies conducted with ETC-1002 to date, there have been observations of changes in biomarkers, including modest average increases in uric acid and modest average decreases in hemoglobin. While we believe that further data in a larger number of patients and longer durations of therapy would clarify results, we gain comfort from the absence of any concerning clinical implications of these changes.

Completed Phase 2a Studies

ETC-1002-006—Proof of Concept in Hypercholesterolemia Patients with a History of Statin Intolerance

ETC-1002-006 (NCT01751984) was a Phase 2a trial evaluating ETC-1002 in patients with elevated levels of LDL-C and a history of intolerance to statins due to muscle pain or weakness. Top-line results were announced in June 2013, and demonstrated further evidence of LCL-C lowering efficacy with ETC-1002 treatment. Furthermore, the trial showed that the occurrence rates of muscle-related adverse events were similar between the drug and placebo arms. In our view, the results from the trial support the advancement of the program into the Phase 2b trial in statin intolerant patients, expected to begin by the end of 2013.

The trial was a U.S.-based multicenter, randomized, double-blind, placebo-controlled study that enrolled 56 patients, randomized (2:1) to receive treatment for eight weeks with ETC-1002 or placebo. All patients underwent a two-week lipid-lowering therapy wash-out period followed by two weeks of dosing with placebo. Patients randomized to the ETC-1002 arm received increasing doses of drug (60 mg, 120 mg, 180 mg and 240 mg) titrated ever two weeks. The primary endpoint was LDL-C lowering and secondary endpoints included an assessment of LDL-C throughout the eight-week treatment period, the impact on other lipid and cardiometabolic biomarkers, muscle-related adverse events, safety and tolerability.

Results: Top-line results demonstrated that ETC-1002 reduced LDL-C levels after eight weeks by an average of 32%, compared to an average reduction of 3% for patients dosed with placebo ($p<0.0001$; see Figure 8). LDL-C levels were lowered by an average of 20%, 31%, 33% and 33% by ETC-1002 versus 1%, 1%, 4% and 4% by placebo in patients completing two, four, six and eight weeks of dosing, respectively. In addition, treatment with ETC-1002 also reduced hsCRP, a marker of inflammation, by 42% after eight weeks of therapy, compared to no change in placebo treated patients ($p=0.0022$). No significant changes in HDL-C or triglyceride levels were observed.

FIGURE 8. ETC-1002-006 Results – Patients with History of Statin Intolerance

Trial Arm	Time Period	Number of Patients	Baseline LDL-C (mg/dL)	Average LDL-C Change from Baseline	p-value
Placebo	Week 2	18	184	-1.4%	-
ETC-1002 (60 mg)		34	177	-19.5%	<0.0001
Placebo	Week 4	15	184	-1.0%	-
ETC-1002 (120 mg)		30	178	-31.0%	<0.0001
Placebo	Week 6	15	184	-3.8%	-
ETC-1002 (180 mg)		31	180	-32.6%	<0.0001
Placebo	Week 8	13	180	-4.0%	-
ETC-1002 (240 mg)		26	175	-32.6%	<0.0001

Source: Company reports

Safety and Tolerability Profile: Results from the ETC-1002-006 trial continue to support the previously observed favorable safety and tolerability profile with ETC-1002. There were no dose-limiting side effects associated with treatment and drop-out rates were comparable to placebo (14% versus 16%). Importantly, in our view, the frequency muscle-related adverse events were similar in drug and placebo arms (27% versus 32%) and no patients treated with ETC-1002 discontinued due to muscle-related adverse events.

Laboratory tests raised no points of concern for the drug. Consistent with prior clinical studies of ETC-1002, modest average increases in uric acid and homocysteine and a modest average decrease in alkaline phosphatase were observed. Additionally, modest reductions in hemoglobin were observed that were greater in the ETC-1002 group. No patient treated with ETC-1002 experienced substantial elevations in liver function tests greater than three times the upper level of normal and no patient experienced substantial elevations of creatine kinase greater than five times the upper limit of normal. There were no discontinuations due to changes in uric acid, homocysteine, hemoglobin, or alkaline phosphatase.

One ETC-1002 patient developed a gout flare-up. This patient had a history of gout since 2006 and elevated uric acid was noted at baseline. The patient was receiving treatment with colchicine, a standard gout treatment, at entry to the trial. The gout flare occurred on Day 36 and persisted for three days, resolving with an increase in colchicine medication, a standard medical response to gout flares. Recall that the patient completed the study. One patient treated with ETC-1002 was reported to have thyroid cancer, deemed a serious adverse event. The patient had a thyroid cyst history and only received treatment with ETC-1002 for 15 days. This SAE was assessed by the investigator and determined to be unrelated to ETC-1002.

ETC-1002-005—Proof of Concept in Patients with Type 2 Diabetes

ETC-1002-005 (NCT01607294) was a Phase 2a trial that evaluated ETC-1002 in patients with type 2 diabetes who had elevated levels of LDL-C. Top-line results were announced in January 2013 and full data were presented in May at the Arteriosclerosis, Thrombosis, and Vascular Biology (ATVB) 2013 Scientific Sessions. The trial demonstrated statistically significant LDL-C lowering with ETC-1002 compared to placebo.

This was a four-week randomized, double-blind, placebo-controlled trial that enrolled 60 patients at a single clinical site. All patients underwent a 28-day washout of all glucose- and lipid-regulating drugs and supplements before being randomized (1:1) to receive ETC-1002 (80 mg) or placebo once-daily for 14 days, after which patients randomized to the ETC-1002 arm were uptitrated up to 120 mg once-daily. Patients were confined to the clinical site from the morning of Day (-7) to the morning of Day (29) in order to stabilize diet and lifestyle, monitor safety continuously, and complete key efficacy assessments. The primary endpoint was LDL-C levels after four weeks of treatment.

Results: The results demonstrated that after four weeks of treatment with ETC-1002 LDL-C levels were reduced by an average of 43%, compared to an average of 4% for patients dosed with placebo ($p<0.0001$; see Figure 9). In addition, of the approximately 80% of patients not at their NCEP ATP III LDL-C goal of less than 100 mg/dL at the beginning of the study, 88% of patients treated with ETC-1002 achieved this goal by the end of the study, compared to 4% of placebo patients ($p<0.0001$). Improvements in other cardiometabolic risk factors were also reported with ETC-1002 treatment, including a 41% reduction in hsCRP (vs. 11% with placebo; $p=0.001$) and modest trends toward improved glycemic control and insulin resistance. No changes in HDL-C and triglyceride levels were observed and there was no increase in blood pressure with ETC-1002 treatment (as measured using 24-hour Ambulatory Blood Pressure Monitoring), vs. placebo. A retrospective analysis of the ABPM data indicated a trend towards lowering of diastolic blood pressure with ETC-1002 compared to placebo (7.8 mmHg vs. 0.4 mmHg; $p=0.047$).

FIGURE 9. ETC-1002-005 Results - Type 2 Diabetes Subjects

Trial Arm	Time Period	Number of Patients	Baseline LDL-C (mg/dL)	Average LDL-C Change from Baseline	p-value
Placebo	Days 1 to 14	30	128	-6%	-
ETC-1002 (80 mg)		29	125	-32%	<0.0001
Placebo	Days 15-28	30	128	-4%	-
ETC-1002 (120 mg)		29	125	-43%	<0.0001

Source: Company reports

Safety and Tolerability Profile: ETC-1002 was safe, well-tolerated and associated with no dose-limiting side effects. No patient dosed with ETC-1002 reported myalgia and importantly no patient treated with ETC-1002 experienced substantial elevations in liver function tests greater than three times the upper level of normal or creatine kinase greater than five times the upper limit of normal. Consistent with other clinical trials of ETC-1002, modest average increases in uric acid and homocysteine and modest average decreases in alkaline phosphatase and hemoglobin were observed.

No severe adverse events were observed in patients dosed with ETC-1002. All patients completed the study except for one patient treated in the placebo arm who withdrew due to an SAE of heart attack. A further patient, also in the placebo arm, had an SAE of kidney stones, but completed the clinical trial.

ETC-1002-003—Phase Proof of Concept in Hypercholesterolemic Patients

The Phase 2a ETC-1002-003 (NCT01262638) trial was a U.S.-based multicenter, randomized, double-blind, placebo-controlled trial designed to evaluate the efficacy of ETC-1002 in patients with hypercholesterolemia (LDL-C 130-220 mg/dL) and either normal (<150 mg/dL) or elevated triglycerides (150-400 mg/dL). Patients enrolled in the trial underwent a six-week washout period of all lipid-regulating therapies, after which they were stratified into a normal or elevated triglyceride stratum and randomized (1:1:1:1) to receive treatment with one of three ETC-1002 doses (40 mg, 80 mg, or 120 mg) or placebo. The trial demonstrated that ETC-1002 significantly reduced LDL-C levels.

Results: Treatment with ETC-1002 resulted in lowering of LDL-C levels by an average of 18-27%, compared to an average of 2% for patients treated with placebo ($p<0.0001$; Figure 10). The LDL-C lowered efficacy of ETC-1002 was maintained independent of baseline triglycerides levels. In addition, ETC-1002 demonstrated dose-dependent lowering of cardiometabolic risk biomarkers including hsCRP (reduction of 20-26% vs. 2% with placebo), apolipoprotein (apo) B, non-HDL-C, and LDL particle number ($p<0.0001$). The improvements in hsCRP were greatest in the sub-group with elevated hsCRP, where treatment with ETC-1002 demonstrated a reduction of 43-64%, vs. a decrease of 7% for placebo-treated patients. HDL-C and triglyceride levels were unchanged across all treatment arms.

FIGURE 10. ETC-1002-003 Results - Hypercholesterolemia

Trial Arm	Number of Patients	Baseline LDL-C (mg/dL)	Average LDL-C Change from Baseline	p-value
Placebo	42	168	-2%	-
ETC-1002 (40 mg)	42	163	-18%	<0.0001
ETC-1002 (80 mg)	44	170	-25%	<0.0001
ETC-1002 (120 mg)	42	165	-27%	<0.0001

Source: Company reports

Safety and Tolerability Profile: ETC-1002 was safe, well-tolerated and associated with no dose-limiting side-effects. The numbers of discontinuations were similar in the drug and placebo arms. The incidence of myalgia was greater in patients treated with ETC-1002 than placebo: it was reported in two patients in the 40 mg arm, two patients in the 80 mg arm, three patients in the 120 mg arm, and in no patients in the placebo arm. One patient, in the 80 mg arm, discontinued treatment due to myalgia. No patient reporting myalgia experienced concurrent creatine kinase elevations more than twice the upper limit of normal. No SAEs were observed in patients treated with ETC-1002.

A single ETC-1002 patient experienced a substantial elevation in liver function tests more than three times the upper limit of normal. However, this lab finding was determined by the investigator as not related to treatment as it coincided with a confirmed acute cytomegalovirus infection. No patient experienced creatine kinase increases greater than five times the upper limit of normal. Consistent with other clinical trials for ETC-1002, modest average increases in uric acid and homocysteine and modest average decreases in alkaline phosphatase and hemoglobin were observed.

Completed Phase 1 Studies

Esperion has completed three Phase 1 trials evaluating ETC-1002, including one, single-dose study and two, multiple-dose trials. Results from these trials provided initial evidence of LDL-C lowering efficacy with the drug. Additionally, no dose-limiting toxicities were identified.

ETC-1002-004 (NCT01485146) was a Phase 1b multiple-ascending dose trial in 24 healthy volunteers. The placebo-controlled trial evaluated 14 days of treatment with three ascending cohorts of ETC-1002 (140, 180, or 220 mg) and placebo, each with eight healthy subjects. The result demonstrated that LDL-C levels were reduced by an average of 21-36% in the increasing dose cohorts, compared to an average 4% increase for subjects receiving placebo ($p<0.0001$; see Figure 11). Note that the reductions in LDL-C were achieved in patients with non-elevated baseline LDL-C levels.

FIGURE 11. ETC-1002-004 Results - Multiple-Dose Tolerance Greater than 120mg

Trial Arm	Number of Subjects	Baseline LDL-C (mg/dL)	Average LDL-C Change from Baseline	p-value
Placebo	6	121	4%	-
ETC-1002 (140 mg)	6	113	-21%	0.0012
ETC-1002 (180 mg)	6	100	-27%	0.0001
ETC-1002 (220 mg)	6	105	-36%	<0.0001

Source: Company reports

The ETC-1002-002 (NCT01105598) trial was also a placebo-controlled, multiple-ascending dose study. The trial evaluated four doses of ETC-1002 (20, 60, 100, or 120 mg) in patients with mildly elevated LDL-C. The results demonstrated that LDL-C levels were reduced by an average of 11-17% in the increasing dose cohorts.

FIGURE 12. ETC-1002-002 Results - Multiple-Dose Tolerance Clinical Trial

Trial Arm	Time Period	Number of Patients	Baseline LDL-C (mg/dL)	Average LDL-C Change from Baseline	p-value
Placebo	2 Weeks	8	114	11%	--
ETC-1002 (20 mg)		6	124	4%	0.2975
ETC-1002 (60 mg)		6	138	-11%	0.0035
ETC-1002 (100 mg)		6	135	-17%	0.0003
ETC-1002 (120 mg)		6	127	-15%	0.0004
Placebo	4 Weeks	6	146	-1%	--
ETC-1002 (120 mg)		15	122	-16%	0.0317

Source: Company reports

In both of these multi-dose Phase 1b trials, no subjected receiving ETC-1002 reported myalgia or experienced substantial elevations in liver function tests greater than three times the upper limit of normal. Additionally, no ETC-1002 treated subject experienced creatine kinase greater than five times the upper limit of normal.

ETC-1002 MARKET OPPORTUNITY AND COMPETITIVE LANDSCAPE

We assess the potential for ETC-1002 in two patient populations with elevated levels of LDL-C: first, we consider the opportunity in patients who do not tolerate statins due to muscle-related adverse events and second, we consider the use of the drug as an add-on to current statin therapy in patients who have not achieved adequate LDL-C lowering and remain above their LDL-C treatment goals.

Statin-Intolerant Opportunity

We forecast WW peak sales of $3.7bil. for ETC-1002 in the statin-intolerant market

The lead indication for ETC-1002 is in patients with hypercholesterolemia who are intolerant to statin therapies. Given the limited precedent for a drug addressing this commercial opportunity, the size of the market is somewhat uncertain. It is known that compliance and persistence on statin therapy is substantially below the lay understanding of a chronic therapy. It is estimated that more than 50% of patients stop taking statins within one year of initiating treatment. There is a growing body of evidence to support that a common reason for patients discontinuing stating therapy is muscle pain or weakness. As such, we believe this market represents a compelling commercial opportunity for ETC-1002.

The USAGE survey, published in 2012 in the *Journal of Clinical Lipidology,* was an approximately 10,000-patient academic study of current and former statin users. The study found that 12% of patients on statins discontinue therapy, of which 62% cited side effects as the reason for discontinuation and of this, more than 86% of patients cited muscle pain or weakness as the reason. This implies that more than two million adults in the U.S. have discontinued statin therapy due muscle pain or weakness. We believe that these two million people represent the initial market opportunity for ETC-1002.

Upside in this initial commercial opportunity could come from the sizeable number of patients who experience muscle-related side effects, but nonetheless remain on statin therapy. The USAGE survey estimated that 25% of statin users remain on statin therapy despite experiencing muscle-related side effects. In addition, a study published in August 2008 in the *Journal of General Internal Medicine* estimated that muscle pain was a complaint for up to 20% of statin-treated patients in clinical practice. Thus, the market opportunity for ETC-1002 in this population of adverse-event sufferers who continue on statin therapy could be at least six million adults in the U.S.

The competitive landscape in the statin intolerant opportunity includes currently approved therapies and a new class of injectable drugs called PCSK9 (proprotein convertase subtilisin/kexin type 9) inhibitors, currently in Phase 3 development. The PCSK9 inhibitor class is discussed below. The currently approved and, reported to be, most commonly prescribed treatments for statin-intolerant patients are Zetia (Merck & Co.) and Welchol (Daiichi Sankyo). Together, these drugs had 2012 U.S. sales approaching $1.7 bil. and Esperion estimates that ~50% of these revenues were derived from the statin intolerant opportunity. As such, the company estimates that the current U.S. market for the statin intolerant indication is in excess of $800MM.

Should ETC-1002 be successfully developed and approved in the U.S., we believe that, based on the initial clinical evidence, the drug could offer a superior LDL-C lowering profile vs. these approved therapies. As shown in Figure 3, these drugs lower LDL-C levels by approximately 20% and in Phase 2a trials, ETC-1002 lowered LDL-C by ~30-40%. As discussed earlier in this report, we believe a key determinant of the commercial success of ETC-1002 will be confirmation of a favorable safety and tolerability profile.

We see potential for WW sales of nearly $1.5 bil. for ETC-1002 in the residual risk market

Residual Risk Opportunity

The second commercial opportunity we consider for ETC-1002 is an add-on therapy for hypercholesterolemic patients who cannot achieve their recommended LDL-C goals via statin therapy alone. This segment is referred to as the “residual risk” patient population. The CDC estimates that there are approximately 11 million residual risk patients in the U.S. Using data from the Centers for Disease Control and Prevention study, "Vital Signs: Prevalence, Treatment, and Control of High Levels of Low-Density Lipoprotein Cholesterol—United States, 1999 – 2002 and 2005 – 2008," Esperion estimates that 70% of the 11 million residual risk patients in the U.S., or 7.7 million people, are within 30% of their LDL-C goal.

The current standard of care for patients who do not achieve LDL-C treatment goals is fixed-combination therapies including: Vytorin (ezetimibe and simvastatin), Advicor (niacin extended release and lovastatin), and Simcor (niacin simvastatin). As with the statin intolerant patient population, we believe that demonstrating superior LDL-C lowering efficacy compared to current oral fixed-combination therapies, as well as an attractive safety and tolerability profile, will be key to gaining market traction.

Emerging PCSK9 Inhibitor Therapies

A new class of biologic therapies for hyperlipidemia are being developed that target PCSK9 (proprotein convertase subtilisin/kexin type 9), an enzyme that binds LDL receptors. These PCSK9 inhibitors are injectable, fully-human antibodies intended to lower LDL-C. Several monoclonal antibodies directed against PCSK9 are in development, as shown in Figure 13. The earliest market entrants could be Amgen’s AMG 145 or Sanofi/ Regeneron’s REGN727, which are both in Phase 3 development.

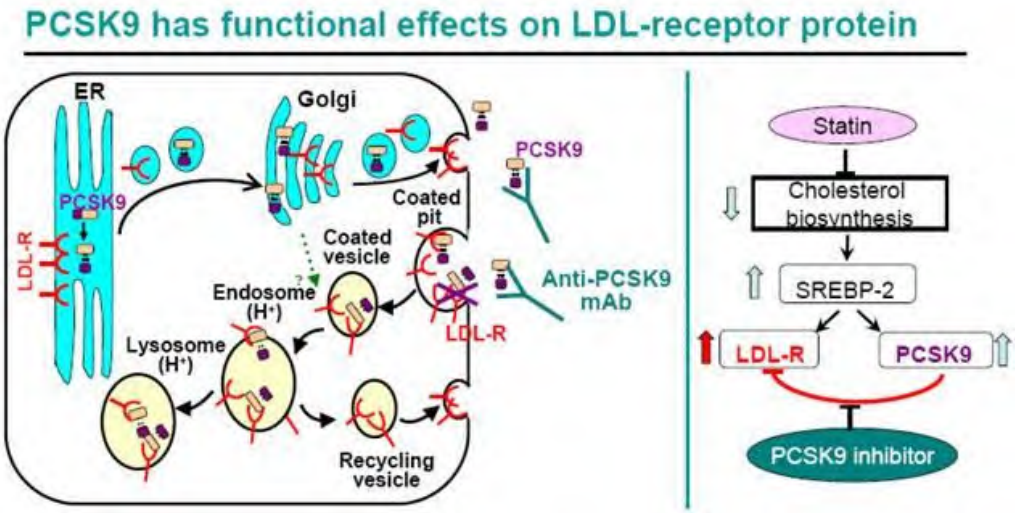
FIGURE 13. Anti-PCSK9 Antibody Innovators

Company	Product	Development stage	Date of Next Catalyst	Catalyst
Amgen	AMG 145	Phase 3	2H13	Data readouts expected for ongoing Phase 2 and 3 clinical trial program
Sanofi/ Regeneron	REGN727	Phase 3	2H13	Data readout expected for Phase 3 MONO trial (head-to-head with ezetimibe)
Pfizer	RN316	Phase 2b	unknown	Plans for further development have not been disclosed
Roche (Genentech)	RG7652 (MPSK3169A)	Phase 2	4Q13	Data readout expected for Phase 2 EQUATOR trial

Source: JMP Securities LLC and Company reports

The PCSK9 gene encodes a proprotein convertase belonging to the proteinase K subfamily of the secretory subtilase family. The protein is synthesized as a soluble zymogen that undergoes autocatalytic intramolecular processing in the endoplasmic reticulum. The protein may function as a proprotein convertase. PCSK9 plays a major regulatory role in cholesterol homeostasis. LDL cholesterol is removed from the blood when it binds to LDL receptors on the surface of liver cells, and is taken inside the cells. PCSK9 binds to the epidermal growth factor-like repeat A (EGF-A) domain of the low-density lipoprotein receptor (LDLR), targeting LDLR and the LDL for degradation (Figure 14). Reduced LDLR levels result in decreased metabolism of LDL, which could lead to hypercholesterolemia. However, if PCSK9 does not bind to LDLR, the receptor can return to the surface of the cell and remove more cholesterol.

FIGURE 14. PCSK9 Mechanism of Action



Source: http://www.ijbs.com/v08p0310.htm

While acknowledging that ETC-1002 is substantially earlier in clinical development than the most advanced PCSK9 inhibitors, comparing the two will likely be a commonplace occurrence as ETC-1002 progresses through its development. In our view, there are three attributes of these drugs that will likely take center stage in such a debate:

- Efficacy
- Safety/tolerability
- Convenience

Considering efficacy first, the clinical trials completed to date for AMG 145 and REGN727 have demonstrated LDL-C lowering efficacy in the range of 40-60%. This compares with the available data for ETC-1002 which has demonstrated LDL-C lowering efficacy in the range of 30-40% (~30% in the statin-intolerant population). While the efficacy for ETC-1002 is modestly lower than the PCSK9 inhibitors, we believe it may also be important to consider the proportion of patients achieving treatment goals on therapy. To this point, Esperion estimates that 70% of the 11 million residual risk patients in the U.S., or 7.7 million people, are within 30% of their LDL-C goal.

As discussed previously, we believe that the safety/tolerability profile of ETC-1002, in comparison to current therapies and new therapies such as PCSK9 inhibitors, will be the most important determinant of the drug's clinical and commercial success.

Should later stage clinical trials confirm the encouraging safety/tolerability and efficacy profile seen with ETC-1002 to date, we believe that the oral, once-daily administration of the drug will be an attractive attribute commercially. The current market for dyslipidemia pharmacotherapies is dominated by the oral drug and extending the cardiometabolic treatment landscape to include anti-diabetic medications. Oral therapies are preferred and commercially superior to injectable alternatives, especially in the primary care setting. We believe that with a favorable safety/tolerability profile, the convenient administration of ETC-1002 will make the drug competitive, even assuming slightly less efficacy than PCSK9 inhibitors.

In our view, there remains insufficient data to compare ETC-1002 to PCSK9 inhibitors, especially in reference to the amount of safety data available for ETC-1002. However, with additional data supporting the current clinical profile of the drugs, it could be hypothesized that PCSK9 inhibitors may be most appropriate for patients with more severe hypercholesterolemia, including familial (homozygous and heterozygous) hypercholesterolemia, with the opportunity for ETC-1002 in the broad population of patients with high cholesterol for whom statins are not tolerable or sufficient therapies.

ETC-1002 Revenue Models

Statin-intolerant market

As discussed above, of the approximately 30 million patients in the U.S. currently receiving treatment with statins, muscle-related symptoms occur in up to 20% of patients. For the purposes of our revenue projections, we initially focus on the approximately two million patients in the U.S. who are estimated to have discontinued statin therapy due to muscle-related side effects. However, we believe that with the introduction of novel, safe, tolerable, and effective treatments, the proportion of patients who experience muscle-related side effects on statins and therefore seek alternative treatment options will increase. Our model assumes that the number of statin intolerant patients seeking alternative therapies increases by approximately 50% within five years of ETC-1002's launch, after which we assume annual population growth of 0.5%.

We expect Phase 3 trials for ETC-1002 to be initiated by the beginning of 2016 and anticipate that the product will be launched in the U.S. in 2019 and in Europe in 2020. We assume that the price per day at launch in the U.S. will be $5.50, which is approximately in line with current, branded statin drugs and Zetia. We include 3% annual price increases in the U.S. In Europe, we assume a 25-30% discount to the U.S. price ($4 per day) and do not include any price increases. We also assume that the average persistence on therapy peaks at six months.

We model sales through to patent extension in April 2030 (and assume five years of patent term extension) after which we anticipate generic competition and rapid erosion of market share (Figure 15). Based on these assumptions, we project peak sales in the U.S. of $1.8 bil. in 2029, with worldwide sales approaching $3.7 bil.

FIGURE 15. ETC-1002 U.S. Revenue Model – Statin-Intolerant Indication

U.S. revenue estimates	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
Addressable patient population	2,091,309	2,195,874	2,415,461	2,777,781	3,055,559	3,208,337	3,272,503	3,305,228	3,321,754	3,338,363	3,355,055	3,371,830
Population growth	*2.5%*	*5.0%*	*10.0%*	*15.0%*	*10.0%*	*5.0%*	*2.0%*	*1.0%*	*0.5%*	*0.5%*	*0.5%*	*0.5%*
Patients on ETC-1002	104,565	219,587	362,319	555,556	763,890	962,501	1,145,376	1,322,091	1,328,702	1,335,345	1,342,022	1,348,732
ETC-1002 market share	*5.0%*	*10.0%*	*15.0%*	*20.0%*	*25.0%*	*30.0%*	*35.0%*	*40.0%*	*40.0%*	*40.0%*	*40.0%*	*40.0%*
Average treatment persistence (months)	3.0	4.5	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	6.0	2.0
Cost per day	5.50	5.67	5.83	6.01	6.19	6.38	6.57	6.76	6.97	7.18	7.39	7.61
Price increase	0	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*	*3.0%*
Average annual cost per patient	495	765	1,050	1,082	1,114	1,148	1,182	1,218	1,254	1,292	1,330	457
ETC-1002 sales ($MM)	**51.8**	**167.9**	**380.5**	**601.0**	**851.2**	**1,104.6**	**1,354.0**	**1,609.7**	**1,666.3**	**1,724.9**	**1,785.5**	**616.1**
Royalties to Esperion	**10.4**	**33.6**	**76.1**	**120.2**	**170.2**	**220.9**	**270.8**	**321.9**	**333.3**	**345.0**	**357.1**	**123.2**
Royalty rate	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*	*20%*

Source: JMP Securities LLC

Residual risk market

As discussed above, the CDC estimates that there are approximately 11 million whose LDL-C levels remain above their goal, and whom we consider relevant to the residual risk population. While this patient population is substantially larger than the statin intolerant opportunity, we are maintaining more conservative market penetration assumptions as we look to results from the Phase 2a ETC-1002-007 trial in 3Q13. Note that revenue from the residual risk opportunity is not currently included in our valuation assumptions. Our model assumes that the number of residual risk patients increases by 0.5% per year based on population growth. Also note that there will be overlap between the statin intolerance and residual risk patient populations.

Assuming positive results from the ETC-1002-007 trial, we expect a Phase 2b trial in this indication to begin in late 2013 or early 2014, and for Phase 3 trials to be initiated by the beginning of 2016. In line with the statin intolerance indication, we expect product launch in 2019 at a daily cost of $5.50. We include 3% annual price increases in the U.S. In Europe, we assume a 25-30% discount to the U.S. price ($4 per day) and do not include any price increases. We again assume that the average persistence on therapy peaks at six months.

We model sales through to patent extension in April 2030 (assuming five years of patent term extension) after which we anticipate generic competition and rapid erosion of market share. We believe that the residual risk opportunity could drive upside to our current valuation of ~10-15 per share, depending on the strength of the Phase 2a data. A summary of scenarios for peak penetration and probability of success and the respective upside to our price target is shown in Figure 16.

FIGURE 16. Residual Risk Indication – Potential Upside to Valuation

		Peak Penetration				
		2.5%	**5.0%**	**7.5%**	**10.0%**	**12.5%**
Probability of success	**45%**	4.11	8.22	11.63	14.60	18.21
	50%	4.57	9.13	12.93	16.22	20.23
	55%	5.02	10.05	14.22	17.84	22.26
	60%	5.48	10.96	15.51	19.46	24.28
	65%	5.94	11.87	16.80	21.09	26.30

Source: JMP Securities LLC

EARLY-STAGE PIPELINE ASSETS

ESP41091

The company is investigating ESP41091 as a therapy for type 2 diabetes and obesity. Exclusive worldwide rights to the asset were acquired from Pfizer in April 2008. In pre-clinical pharmacology studies, treatment with ESP41091 resolved hyperglycemia and reduced body weight, following a four-week treatment in a diet-induced, obese mouse model of insulin resistance. Further, treatment with ESP41091 also resulted in beneficial effects on lipid metabolism and body weight in obese Zucker rats.

Intellectual property: ESP41091 is claimed in U.S. Patent Nos. 7,119,221 and 7,405,226. Various methods of treatment using ESP41091 are claimed in U.S. Patent Nos. 8,153,690 and 8,309,604 and in at least one other pending application in the U.S. There are currently two issued patents and four pending applications in countries outside the United States that relate to ESP41091.

4WF

4WF is being developed as a synthetic HDL therapy designed to preserve the function of HDL and its primary apolipoprotein (apoA-I) and to deliver oxidation-resistant synthetic HDL therapy. A potential myeloperoxidase oxidation-resistant apoA-I mimetic, 4WF is a pre-clinical stage, next-generation synthetic HDL therapy via injection instead of intravenous infusion. Esperion acquired the exclusive worldwide rights to 4WF from the Cleveland Clinic Foundation in June 2011.

Intellectual property: The 4WF patent portfolio currently consists of 19 issued patents and pending patent applications in the U.S. and other foreign jurisdictions regarding apolipoprotein mixtures, dimeric oxidation-resistant apolipoprotein variants and oxidant resistant apolipoprotein A1 variants and mimetic peptides thereof.

MANAGEMENT TEAM

Roger Newton - Executive Chairman and Chief Scientific Officer

Roger Newton, Ph.D., F.A.H.A., is founder, Executive Chairman and Chief Scientific Officer of Esperion and is a fellow of the American Heart Association. Prior to founding Esperion in 2008, Mr. Newton was Senior Vice President, Pfizer Global R&D from 2004 to 2008. Previously, Mr. Newton was co-founder, President & CEO of the original Esperion from July 1998 until its acquisition by Pfizer in 2004. Prior to founding the original Esperion, Mr. Newton was Chairman of the Atherosclerosis Drug Discovery Team at Warner Lambert from 1981 to 1998. Mr. Newton is a director of a number of life science companies including Juventas Therapeutics, Inc. and Rubicon Genomics, Inc. and is also a member of the Technology Advisory Boards for Arboretum Ventures and Metagenics, Inc. Mr. Newton earned a Ph.D. in Nutrition from the University of California, Davis, an M.S. in Nutritional Biochemistry from the University of Connecticut, and a B.S. in Biology from Lafayette College.

Tim Mayleben - President & Chief Executive Officer

Tim Mayleben is President and Chief Executive Officer and has been a member of the board of directors since February 2010. Prior to joining Esperion, Mr. Mayleben was President, CEO and a Director of Aastrom Biosciences, Inc. Previously, Mr. Mayleben was President, Chief Operating Officer and a director of NightHawk Radiology Holdings, Inc. Prior to joining NightHawk, Mr. Mayleben was the COO of the original Esperion, until its acquisition by Pfizer in 2004. Mr. Mayleben is an advisor to, investor in, and member of the board of directors of several life science companies, including DeNovo Sciences, Intelliject Corporation, Lycera Corporation and Marinus Pharmaceuticals. Mr. Mayleben earned an M.B.A., with distinction, from the J.L. Kellogg Graduate School of Management at Northwestern University, and a B.B.A. from the University of Michigan, Ross School of Business.

Noah Rosenberg, M.D. - Chief Medical Officer

Noah Rosenberg, M.D. is Chief Medical Officer. Prior to joining Esperion, Dr. Rosenberg served as Executive Medical Director, head of cardiovascular/metabolism at Forest Laboratories. Previously, Dr. Rosenberg was Senior Medical Director at Sanofi where he led the development of diabetes compounds. Prior to joining Sanofi, Dr. Rosenberg worked as a medical director at Pfizer Inc., developing compounds in cardiovascular/metabolism. Dr. Rosenberg earned an M.D. from Drexel University and a B.A. in Natural Sciences from The Johns Hopkins University.

Troy Ignelzi - Vice President, Business Development

Troy Ignelzi is Vice President, Business Development. Prior to joining Esperion, Mr. Ignelzi served as Vice President, Business Development and Strategic Planning of Insys Therapeutics. Mr. Ignelzi previously worked as a sales and marketing professional in the neuroscience division at Eli Lilly. Mr. Ignelzi earned a B.S. in Accounting from Ferris State University.

Source: Excerpted from Company website

FIGURE 17. Esperion Therapeutics Earnings Model ($MM, except per share data)

	2012	1Q:13	2Q:13E	3Q:13E	4Q:13E	2013E	1Q:14E	2Q:14E	3Q:14E	4Q:14E	2014E	2015E
Revenue												
Revenue	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Grant income	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total Revenue	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**
Cost of goods sold	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	
Gross Profit	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**
Operating expenses												
R&D	8.0	2.1	2.2	4.0	7.1	15.4	8.9	11.1	13.9	17.4	51.3	59.0
SG&A	2.2	1.3	1.3	1.4	1.6	5.5	1.6	1.6	1.7	1.7	6.7	7.4
Total Operating Expenses	**10.2**	**3.3**	**3.5**	**5.4**	**8.7**	**20.9**	**10.5**	**12.8**	**15.6**	**19.1**	**58.0**	**66.4**
Operating income (loss)	**(10.2)**	**(3.3)**	**(3.5)**	**(5.4)**	**(8.7)**	**(20.9)**	**(10.5)**	**(12.8)**	**(15.6)**	**(19.1)**	**(58.0)**	**(66.4)**
Interest expense	(1.5)	(0.8)	0.0	0.0	0.0	(0.8)	0.0	0.0	0.0	0.0	0.0	0.0
Change in fair value of warrant liability	0.0	(0.0)	0.0	0.0	0.0	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0
Other income (expense), net	(0.1)	(0.0)	0.0	0.0	0.0	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0
Total other income (expense)	**(1.5)**	**(0.9)**	**0.0**	**0.0**	**0.0**	**(0.9)**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**
Net Income Before Taxes	**(11.7)**	**(4.2)**	**(3.5)**	**(5.4)**	**(8.7)**	**(21.8)**	**(10.5)**	**(12.8)**	**(15.6)**	**(19.1)**	**(58.0)**	**(66.4)**
Income tax provision	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net income (loss)	**(11.7)**	**(4.2)**	**(3.5)**	**(5.4)**	**(8.7)**	**(21.8)**	**(10.5)**	**(12.8)**	**(15.6)**	**(19.1)**	**(58.0)**	**(66.4)**
EPS												
Basic	**($3.13)**	**($0.84)**	**($0.58)**	**($0.35)**	**($0.56)**	**($2.32)**	**($0.67)**	**($0.81)**	**($0.99)**	**($1.20)**	**($3.68)**	**($3.30)**
Diluted	**($3.13)**	**($0.84)**	**($0.58)**	**($0.35)**	**($0.56)**	**($2.02)**	**($0.67)**	**($0.81)**	**($0.99)**	**($1.20)**	**($3.68)**	**($3.30)**
Weighted shares outstanding												
Basic	3.8	5.0	6.1	15.5	15.6	9.4	15.7	15.7	15.8	15.9	15.8	20.1
Diluted	3.8	5.0	6.1	15.5	15.6	10.8	15.7	15.7	15.8	15.9	15.8	20.1
Cash Flow												
GAAP Net Income	(11.7)	(4.2)	(3.5)	(5.4)	(8.7)	(21.8)	(10.5)	(12.8)	(15.6)	(19.1)	(58.0)	(66.4)
Depreciation & amortization	0.1	0.5	0.5	0.5	0.5	2.0	0.5	0.5	0.5	0.5	2.0	2.0
Stock-based compensation	0.1	0.0	0.1	0.1	0.1	0.3	0.5	0.5	0.5	0.5	2.0	2.0
Other adjustments	0.7	1.1	0.0	0.0	0.0	1.1	0.0	0.0	0.0	0.5	0.5	0.5
Operating Burn	**(10.8)**	**(2.6)**	**(2.9)**	**(4.8)**	**(8.1)**	**(18.3)**	**(9.5)**	**(11.8)**	**(14.6)**	**(17.6)**	**(53.5)**	**(61.8)**
Cash at start of period	1.6	6.5	3.9	18.0	86.0	6.5	78.0	68.5	56.7	42.1	78.0	24.5
Cash from operations	(10.8)	(2.6)	(2.9)	(4.8)	(8.1)	(18.3)	(9.5)	(11.8)	(14.6)	(17.6)	(53.5)	(61.8)
Cash from investing	(0.0)	0.0	0.0	0.0		0.0					0.0	0.0
Cash from financing	15.8	0.0	17.0	72.8		89.8					0.0	95.0
Shares issued				5.8								4.0
Price per share				14								25.0
Effect of Fx	0.0											
Cash at end of period	**6.5**	**3.9**	**18.0**	**86.0**	**78.0**	**78.0**	**68.5**	**56.7**	**42.1**	**24.5**	**24.5**	**57.7**
Marketable securities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	
Cash and ST investments	**6.5**	**3.9**	**18.0**	**86.0**	**78.0**	**78.0**	**68.5**	**56.7**	**42.1**	**24.5**	**24.5**	**57.7**

Source: JMP Securities LLC, Company reports

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