CREDIT SUISSE

Esperion Therapeutics (ESPR)

SMALL & MID CAP RESEARCH

A New Pill to Lower Cholesterol

- **We Are Initiating Coverage of Esperion Therapeutics with an Outperform Rating and a $23 Target Price:** Our positive view is based on the large market opportunity and significant scarcity value of its novel oral pill for lowering cholesterol. If planned Phase IIb trials are successfully cleared and the FDA gives the "green light" for Phase III, we anticipate ESPR will seek a large global pharma partner or more likely be acquired.
- **Novel Cardiovascular Drug for a Mass Market Pharma Indication:** ETC-1002 has established efficacy in statin intolerant patients ($1B market opportunity), and upcoming data should better define the opportunity as an add-on to statins (multibillion dollar). Continued clean efficacy, better understanding of the safety, and a key end of Phase II meeting with FDA are the critical events to derisk ESPR. Assuming all goes well, Phase III trials could start in H1:15 for statin intolerant and potentially statin add-on patients.
- **More Derisking Is Necessary:** ETC-1002 is very well tolerated. However, modest decreases in hemoglobin and modest increases in uric acid need to be further described. Also, a partial clinical hold (limiting treatment duration to six months) remains in effect awaiting two-year animal tox studies.
- **Catalysts:** (1) Proof of concept Phase IIa statin add-on data in September 2013; (2) Phase IIb statin intolerant data in H2:14; (3) Phase IIb statin add-on data potentially in H2:14; and (4) end of Phase II meeting by early 2015.
- **Valuation:** Our $23 target price for ESPR is based on a probability adjusted DCF, assuming a 55% probability of success in statin intolerant and 25% probability in statin add-on patients. Additional data in 2013 and 2014 could increase our probabilities for both indications.

Rating	**OUTPERFORM* [V]**
Price (19 Jul 13, US$)	16.90
Target price (US$)	23.00[1]
52-week price range	17.31 - 14.00
Market cap. (US$ m)	259.07
Enterprise value (US$ m)	188.03

**Stock ratings are relative to the coverage universe in each analyst's or each team's respective sector.*
[1]Target price is for 12 months.
[V] = Stock considered volatile (see Disclosure Appendix).

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Financial and valuation metrics

Year	12/12A	12/13E	12/14E	12/15E
EPS (CS adj.) (US$)	-3.13	-2.23	-1.77	2.11
Prev. EPS (US$)	—	—	—	—
P/E (x)	-5.4	-7.6	-9.6	8.0
P/E rel. (%)	-34.1	-51.2	-71.7	66.2
Revenue (US$ m)	—	—	—	78.0
EBITDA (US$ m)	-10.1	-23.0	-30.2	42.1
OCFPS (US$)	-2.88	-1.98	-1.60	2.25
P/OCF (x)	—	-8.5	-10.6	7.5
EV/EBITDA (current)	-17.5	-7.6	-5.8	4.2
Net debt (US$ m)	16	-71	-119	-166
ROIC (%)	901.89	2,017.18	2,643.58	-3,685.26
EV/EBITDA (x)	-27.36	-8.17	-4.63	—
EV/EBITDA (x)	-27.36	-8.17	-4.63	—

Number of shares (m)	15.33	IC (current, US$ m)	-1.13
BV/share (Next Qtr., US$)	5.2	EV/IC (x)	-158.8
Net debt (Next Qtr., US$ m)	-77.6	Dividend (current, US$)	—
Net debt/tot cap (Next Qtr., %)	-101.5	Dividend yield (%)	—

Source: Company data, Credit Suisse estimates

Portfolio Manager Summary

Esperion's primary asset is a wholly owned, internally discovered oral pill for lowering cholesterol. ETC-1002 is currently in Phase II, and has already demonstrated convincing clinical efficacy in its lead indication for statin intolerant patients. Data in the statin add-on indication is expected in September 2013. Recent positive Phase IIa efficacy data in statin intolerant patients showed an approximate 30% average reduction in LDL-C, consistent with earlier studies. The efficacy of ETC-1002 appears roughly double that of Zetia (ezetimibe), the current standard of care in this setting.

We expect the following key value inflection catalysts in the next one to two years.

- **Data from the Phase IIa Study (Study 007):** Data in residual risk patients (statin add-ons) are expected in September 2013. This is the first data as an add-on to statins, and this market opportunity could be roughly three times that of statin intolerant. Our current probability of success for this indication is 25%.
- **Phase IIb Studies in Statin Intolerant Patients and Residual Risk Patients:** Both studies are expected to start in Q4:13. These studies will read out in H2:14, and are the key trials that we expect will drive a large pharma partnership or potential takeout.
- **More Regulatory Clarity after Its End of Phase II Meeting in Late 2014 / Early 2015:** This meeting will define the scope of the Phase III program and potentially lift the partial clinical hold. It is critical to the value of a potential takeout or partnership.

Exhibit 1: ESPR News Flow

Timing	Expected News Flow	Program
Statin intolerant program		
Q4:13	Start Phase IIb (Study-008)	ETC-1002
H2:14	Phase IIb data (Study-008)	ETC-1002
Residual risk (statin add-on)		
Q3:13 (Sept.)	Phase IIa data (Study-007)	ETC-1002
Q4:13	Start Phase IIb (Study-009 & 010)	ETC-1002
Q4:14	Phase IIb trial readout	ETC-1002
Pivotal program		
Mid-2015	Start Phase III study	ETC-1002
H2:16	Completion of Phase III efficacy study	ETC-1002
H1:17	Completion of Phase III safety study	ETC-1002
Regulatory		
Q1:15	End of Phase II meeting	ETC-1002
H2:14	2-year carc. study in animals	ETC-1002
H2:17	NDA filing	ETC-1002
H2:18	Approval and launch	

Source: Company data, Credit Suisse estimates

Exhibit 2: ESPR Pipeline

Drug	Indication	Stage	Partner
ETC-1002	Hypercholesterolemia (statin intolerant)	Phase IIa (completed)	Proprietary
	Hypercholesterolemia (Type II diabetics)	Phase IIa (completed)	
	Hypercholesterolemia (statin add-on)	Phase IIa	
ESP41091	Type II diabetes and obesity	Preclinical	Proprietary
4WF	Low HDL	Preclinical	Proprietary

Source: Company data, Credit Suisse research.

Investment Positives

- **Wholly Owned Cardiovascular Franchise with Focus on the Cholesterol Market:** ESPR owns 100% of the commercial rights to ETC-1002, which it acquired from Pfizer (Pfizer had previously acquired the program when it bought the original Esperion for $1.3B in 2004). ESPR does not owe any royalties to Pfizer for the drug, making this asset more attractive from a partnering or takeout perspective.
- **Potential for Blockbuster Sales:** If successful, ETC-1002 targets a mass market of 2-3M patients who are intolerant to statins and up to 12M patients with residual risk (statin add-on). Despite modest efficacy, Zetia has approximately $4B in annual sales (75% add-on therapy and 25% statin intolerant patients). We believe there is a substantial market for a new and more potent oral drug even in a market with projected generic Zetia and generic statins.
- **Solid Efficacy Data in a Blockbuster Indication:** Once-daily, oral ETC-1002 reduces cholesterol (LDL-C) by approximately 30% as a monotherapy. This is approximately twice the activity of Zetia, which is the current standard of care, second line treatment for high LDL-C (~18% reduction). Sales of Zetia are approximately $4B with $1B derived from the statin intolerant market.
- **Tolerability and Toxicity Appear Good:** ETC-1002 therapy is not associated with any major tolerability side effects that would limit its use, such as rash, nausea, etc. It is also not associated with any overt organ toxicity, such as muscle damage (the major limitation of statins) or liver enzyme elevations. There have been no discontinuations of ETC-1002 treatment due to muscle pain or weakness, a common side effect of statins.
- **Scarcity Value of Cardiovascular Biotechs:** ESPR is one of a small number of pure-play cardiovascular biotech companies. It is significant that the company has retained rights to its drug, ETC-1002, because this increases the chance of a takeout or favorable partnership deal. This management team was also responsible for selling the original Esperion to Pfizer in 2004 for $1.3B.
- **Near-Term Catalysts Could Drive Upside:** The Phase IIa statin add-on trial (Study 007) is expected to read out in September 2013. We currently use a low 25% probability of success in this indication, because of a lack of clinical data. A positive result could open this larger market opportunity beyond statin intolerant patients and trigger an upward revision in our probability of success.

Investment Risks

- **Potential Safety Signals with an Unknown Mechanism:** ETC-1002 treatment is associated with a modest decrease in hemoglobin and a modest increase in uric acid. While neither were cause for treatment disruption or led to any recorded adverse events, it is possible that these side effects could cause regulatory risks, longer-term

safety concerns, or limitations on Phase III enrollment. A better understanding of the mechanism for these physiologic changes is important.

- **Partial Clinical Hold:** ETC-1002 is on partial clinical hold with the FDA, limiting the maximum duration of treatment in ongoing trials to six months. The partial clinical hold results from the FDA having classified ETC-1002 as a PPAR activator (based on its chemical structure). This class of drugs has higher regulatory scrutiny because of potential cancer signals. ESPR is conducting the necessary two-year carcinogenicity studies and expects the hold to be lifted at the time of the end of Phase II meeting. ESPR is also collecting significant data from its clinical program and has substantial clinical evidence that ETC-1002 is not acting as a PPAR activator (no edema, no HDL effect, etc.).
- **Efficacy in the Statin Add-On Population Has Not Been Established:** Results from the statin add-on Phase IIa study are expected in September 2013. If this program is not successful, the market for the drug would be limited to statin intolerant patients, which is still a substantial opportunity (~2M US patients and 4-5M patients WW), but may make the company less attractive as an acquisition target. ESPR believes that a 20% reduction in LDL-C on top of statins is clinically meaningful.
- **Outcomes Studies May Be Required:** Our model assumes that ETC-1002 is approved for statin intolerant patients without the need for an outcomes study (on LDL-C lowering alone). To be conservative, we have forecast the need for an outcome study in our model for the statin add-on market. We expect ESPR will have better visibility on the regulatory requirements in its end of Phase II meeting in late 2014. If an outcome study is required, it would delay the launch by up to three years, substantially increase Phase III expenses, and increase clinical/regulatory risk for a partner or acquirer.
- **Binary Risk:** ESPR is highly levered to a single asset (ETC-1002). If there is a major setback in this program, it could put the value of the company at risk. Potential key risks include: (1) any new safety signals, (2) the inability to remove the partial clinical hold, and (3) failure to demonstrate efficacy in larger trials.

Valuation

Our target price of $23 is supported by a DCF methodology, using probability-weighted sales estimates for ETC-1002 modeled through 2030. We assume a 55% probability of success in statin intolerant patients and a 25% probability of success in the statin add-on market (Exhibit 3). The market opportunity in both indications is further modified by a 55% probability that ETC-1002 faces competition from CETP inhibitors (e.g., Merck's anacetrapib and Eli Lilly's evacetrapib). We model a commercial launch for statin intolerant in 2018 and for statin add-on in 2021.

We use a 38% tax rate and a 12% discount rate, and arrive at a target price of $23.

The most important levers in our valuations are as follows:

(1) **Probability of Success:** We use 55% probability of success for ETC-1002 in statin intolerant patients and 25% for ETC-1002 as a statin add-on. The value of the statin add-on will likely be adjusted after the Phase IIa data in September 2013.

(2) **Pricing of ETC-1002:** We assume that ETC-1002 is priced at a 30% premium to Crestor, an on-patent statin that is considered best-in-class in terms of efficacy.

(3) **Timing of U.S. and EU Approvals:** We assume U.S. and ex-US launches in 2018 for ETC-1002 in statin intolerant patients. The timing for ETC-1002 is forecast assuming an end of Phase II meeting in late 2014 or Q1:15, and completion of Phase III safety

and efficacy studies by H1:17. We assume patent protection until 2030. For the statin add-on market, we assume a launch in 2021, following longer-term outcome studies.

(4) **Competitive Entrants in the Cholesterol Market:** We assume CETP inhibitors, if approved, would compete directly with ETC-1002. If CETP inhibitors are not approved, we forecast a three times greater penetration rate in the statin add-on market and a 50% increase in the statin intolerant market. We assign a 55% probability of success for an approved CETP inhibitor. ETC-1002 would also share the market with generic statins, generic ezetimibe (Zetia), and generic combination products of statins plus ezetimibe.

(5) **We Assume that ESPR Enters into a Partnership for the Global Rights to ETC-1002 and that ESPR Receives a Royalty on Future Sales:** We have modeled probability-adjusted clinical and regulatory milestones for ETC-1002 and a 17.5% royalty on future global sales. In this scenario, the Phase III and launch costs are paid by the partner. If ESPR is not able to partner ahead of Phase III on favorable terms, then there is significant dilution risk from future equity raises to fund expensive late-stage development.

Exhibit 3: Probability of Success (POS) Valuation Matrix (per share)

		POS (statin intolerant)						
		10%	25%	40%	55%	70%	85%	100%
POS residual risk	10%	$1	$6	$12	$17	$22	$27	$32
	25%	$7	$13	$18	$23	$28	$33	$38
	40%	$13	$19	$24	$29	$34	$39	$44
	55%	$19	$25	$30	$35	$40	$45	$50
	70%	$25	$31	$36	$41	$46	$51	$56
	85%	$31	$36	$42	$47	$52	$57	$62
	100%	$37	$42	$47	$53	$58	$63	$68

Source: Company data, Credit Suisse estimates.

In our unadjusted sales model, we assume that both indications are successful in reaching the market.

In our probability-adjusted sales model, we assume a 55% probability of success in the statin intolerant indication and a 25% probability of success in the residual risk/statin add-on indication.

Exhibit 4: Unadjusted Sales Estimates (Assume 100% POS in Each Indication) (US$M)

		2018	2019	2020	2021	2022	2023	2024	2025
With CETP	Statin intolerant	$68.88	$150	$218	$287	$364	$449	$546	$655
	Residual risk	$0	$0	$0	$159	$272	$445	$569	$728
	Total (US)	**$69**	**$150**	**$218**	**$446**	**$635**	**$894**	**$1,115**	**$1,383**
	Total (ex-US)	**$18**	**$60**	**$116**	**$298**	**$423**	**$596**	**$743**	**$922**
	Grand Total	**$87**	**$210**	**$335**	**$744**	**$1,059**	**$1,490**	**$1,858**	**$2,306**
Without CETP	Statin intolerant	$103	$225	$328	$431	$545	$674	$819	$982
	Residual risk	$0	$0	$0	$455	$776	$1,270	$1,626	$2,081
	Total (US)	**$103**	**$225**	**$328**	**$886**	**$1,321**	**$1,944**	**$2,445**	**$3,064**
	Total (ex-US)	**$28**	**$90**	**$175**	**$590**	**$881**	**$1,296**	**$1,630**	**$2,042**
	Grand Total	**$131**	**$315**	**$502**	**$1,476**	**$2,202**	**$3,241**	**$4,074**	**$5,106**

Source: Company data, Credit Suisse estimates

Exhibit 5: Probability-Adjusted U.S. Sales (55% for Intolerant; 25% for Add-on; 55% for CETP) (US$M)

		2018	2019	2020	2021	2022	2023	2024	2025
With CETP	Statin intolerant	$38	$82	$120	$158	$200	$247	$300	$360
	Residual risk	$0	$0	$0	$40	$68	$111	$142	$182
	Total (US)	**$38**	**$82**	**$120**	**$198**	**$268**	**$358**	**$443**	**$542**
	Total (ex-US)	**$10**	**$33**	**$64**	**$132**	**$179**	**$239**	**$295**	**$362**
	Grand Total	**$48**	**$115**	**$184**	**$330**	**$446**	**$597**	**$738**	**$904**
Without CETP	Statin intolerant	$57	$124	$180	$237	$300	$371	$450	$540
	Residual risk	$0	$0	$0	$114	$194	$318	$406	$520
	Total (US)	**$57**	**$124**	**$180**	**$351**	**$494**	**$688**	**$857**	**$1,061**
	Total (ex-US)	**$15**	**$49**	**$96**	**$234**	**$329**	**$459**	**$571**	**$707**
	Grand Total	**$72**	**$173**	**$276**	**$584**	**$823**	**$1,147**	**$1,428**	**$1,768**

Probability Weighted for CETP

	2018	2019	2020	2021	2022	2023	2024	2025
Statin intolerant	$46	$101	$147	$193	$245	$303	$368	$441
Residual risk	$0	$0	$0	$73	$125	$204	$261	$334
Total (US)	**$46**	**$101**	**$147**	**$267**	**$370**	**$507**	**$629**	**$776**
Total (ex-US)	**$12**	**$40**	**$78**	**$178**	**$246**	**$338**	**$419**	**$517**
Grand Total	**$59**	**$141**	**$226**	**$444**	**$616**	**$845**	**$1,048**	**$1,293**

Source: Company data, Credit Suisse estimates

Key Modeling Assumptions

Our model projects 2022 probability-adjusted sales of ETC-1002 of approximately $616M, including $370M in the United States and $246M excluding the United States.

Our model starts with a projection of the total treated population based on current and projected prescription data and we include the following assumptions:

- **Pricing:** We assume that ETC-1002 is at a ~30% premium to Crestor ($153/mo WAC) and Zetia ($155/mo WAC). Crestor is the best-in-class statin, and Zetia is the standard therapy for statin intolerant and statin add-on patients. Although patent protection for Zetia is set to expire in 2017 before ETC-1002 reaches the market, we believe that this price point is a relevant precedent due to the high unmet medical need and the presumed higher efficacy of ETC-1002.
- **Competition:** Our base case assumes a 55% probability of competition from CETP inhibitors. Our model probability adjusts for with or without CETP inhibitors. In the scenario with CETP inhibitors, we use a lower penetration rate for ETC-1002, and this is most pronounced in the statin add-on model (see below).
- **Statin Intolerant Market (55% Probability of Success)**
    - Market size is 1.2M treated patients in 2013, growing to 2.6M patients in 2030 (8.9M to 18.9M prescriptions per year).
    - We assume ETC-1002 is approved for this indication in 2018. We assume competition from PCSK9 inhibitors, which could reach the market in 2016, and potentially CETP inhibitors, which could reach the market in 2017, pending ongoing outcome studies.
    - We assume a peak market share of 30% in this market, assuming CETP inhibitors do not make it to market. If CETP inhibitors make it to market (55% probability), we assume a 20% peak market share.
- **Statin Add-On/Residual Risk Market (25% Probability of Success)**
    - Market size is ~8.8M treated statin add-on patients in 2013, growing to 9.2M in 2030 (63M to 66M Rx per year).

- We assume ETC-1002 obtains a label for this indication in 2021, after the completion of an outcomes study (This is our conservative assumption)
- We assume a peak market share of 16%, assuming CETP inhibitors do not make it to market. If CETP inhibitors make it to market (55% probability), we assume a 5.6% peak market share.

Public Comps Support Upside Potential for ESPR Shares

Standalone biotech companies with marketed or late-stage drugs for cardiovascular diseases have enterprise values in the range of $1-3B (MDCO, ISIS, AEGR, etc.). In general, these companies have products that target more niche markets compared to Esperion. The biggest cardiovascular drugs all reside within big pharma or, more recently, large-cap biotech (e.g., AMGN and REGN).

Exhibit 6: Public Market Comps for CV Companies

Company	Ticker	Price 7/19/2013	Lead Product	Stage	Shares (M)	Cash (M)	Debt (M)	Mark. Cap (M)	Ent. Val (M)
Isis Pharmaceuticals Inc.	ISIS	$31.05	Kynamro	Approved	111.7	$413	$156	$3,149	$2,828
Aegerion Pharmaceuticals Inc.	AEGR	$79.11	Juxtapid	Approved	28.8	$82	$11	$2,265	$2,097
Amarin Corp. PLC ADS	AMRN	$5.54	Vascepa	Approved	135.5	$260	$219	$851	$872
Medicines Co.	MDCO-US	$30.92	Several products	Approved	56.0	$570	$226	$1,735	$1,651
Portola Pharmaceuticals, Inc.	PTLA	$22.95	Betrixaban	Phase 3	38.9	$238	$0	$893	$655
Esperion Therapeutics Inc.	ESPR	$16.90	ETC-1002	Phase 2	15.6	$83	$0	$264	$180
Average								**$1,778**	**$1,620**
Median								**$1,735**	**$1,651**

Source: Company data, Credit Suisse estimates

Scarcity Value of Cardiovascular Companies

There are relatively few new oral agents being developed in the cholesterol space and few cardiovascular-focused biotech companies. Historically, the majority of small companies in the cardiovascular space were ultimately acquired by larger pharma companies. This historical precedent offers a potential exit option for ESPR, pending Phase IIb clinical success. If there is no takeout, ESPR will likely need to partner for Phase III development and market launch, given the size of the market.

- The takeout thesis for Esperion is bolstered by this management team having sold the original Esperion to Pfizer in 2004 for $1.3B.

Exhibit 7: Acquisitions of CV Biotech Companies

Cardiovascular biotech	Acquirer	Year	$B	Indication	Stage
Centocor	JNJ	1999	$4.9	Anticoagulant	Marketed
COR Therapeutics	MLNM	2001	$2.0	Anticoagulant	Marketed
Scios	JNJ	2003	$2.4	Heart failure	Marketed
CV Therapeutics	GILD	2009	$1.3	Heart failure	Marketed
			$2.7		
Collateral Thereapeutics	Schering AG	2002	$0.1	PVD, CAD	Development
Esperion	PFE	2003	$1.3	Lipid agent	Development
Myogen	GILD	2006	$2.5	Hypertension	Development
NovoCardia	Merck	2007	$0.4	Heart failure	Development
Omthera	AZN	2013	$0.3	Lipid agent	Development
AlphaCore	AZN	2013	NA	Lipid agent	Development
			$0.9		

Source: Company data, Credit Suisse research

Unmet Need in Multibillion Dollar Cholesterol Market

The current market for LDL cholesterol reduction is huge, with >30M patients in the United States treated with statins. Statins are the first-line standard of care for the primary prevention of cardiovascular events in patients with elevated LDL cholesterol, total cholesterol, and/or triglycerides.

Despite the success of statins, there is a significant unmet medical need for new cholesterol drugs because many patients either cannot tolerate statins or do not meet their LDL-C goals on statin therapy alone. Approximately 2M patients in the United States are statin intolerant (4-5M WW), primarily due to muscle pain and weakness caused by statins. Another 9-11M patients in the United States (20M WW) do not meet their LDL-C goals on statin therapy alone (residual risk).

Zetia is the current standard of care for second-line therapy. Despite a modest benefit on LDL-C (~15-20% reduction), Zetia sales are around $4B annually, of which around $1B is for the statin intolerant market and $3B are for the add-on market.

Exhibit 8: Comparison to Market Standard (Zetia) and Emerging New Drug Classes

	PCSK9	CETP Inhibitors	Zetia (MRK)	ETC-1002
Launch timing	2016	2017	In-market	2018
Administration	Injectable	Oral	Oral	**Oral**
Frequency	Every 2-4 weeks	Once daily	Once daily	Once daily
LDL-c reduction	40-60%	25-35%	15-20%	**30%+**
Inflammation (C-reactive protein	No impact	Flat to increased inflammation	No impact	**40%+ reduction**
HDL	No impact	>100%	No impact	No impact
Triglycerides	Modest reduction	Modest impact	No impact	No impact
Tolerability	Good	Good	Good	Good
BP/Insulin	No impact	No impact	No impact	**Slight to modest benefit**
Notes	▪ Injectable therapy that is likely to be more expensive	▪ POS modest given prior category failures ▪ Impact of HDL increase unclear	▪ $4Bn+ agent – direct benchmark for ETC-1002	▪ Only oral agent with beneficial impact on broad CV risk factors

Source: Company data, Credit Suisse research.

Novel Dual Mechanism of Action

First Discovered Based on Functional Screen

ETC-1002 was discovered through a functional screening assay for compounds that caused inhibition of fatty acid and sterol synthesis. The structure of ETC-1002 was similar to known PPAR activators, leading to the early and potentially erroneous PPAR designation by the FDA.

Its initially described mechanism of action was inhibition of ATP Citrate Lyase (ACL), an enzyme upstream of the rate-limiting enzyme in cholesterol synthesis (HMG-CoA reductase), which is the target of statins. It was later discovered that it also activates AMP-Activated Protein Kinase (AMPK). (See Exhibit 9 and Exhibit 10.)

Favorable Impact on Cardiovascular Risk Markers

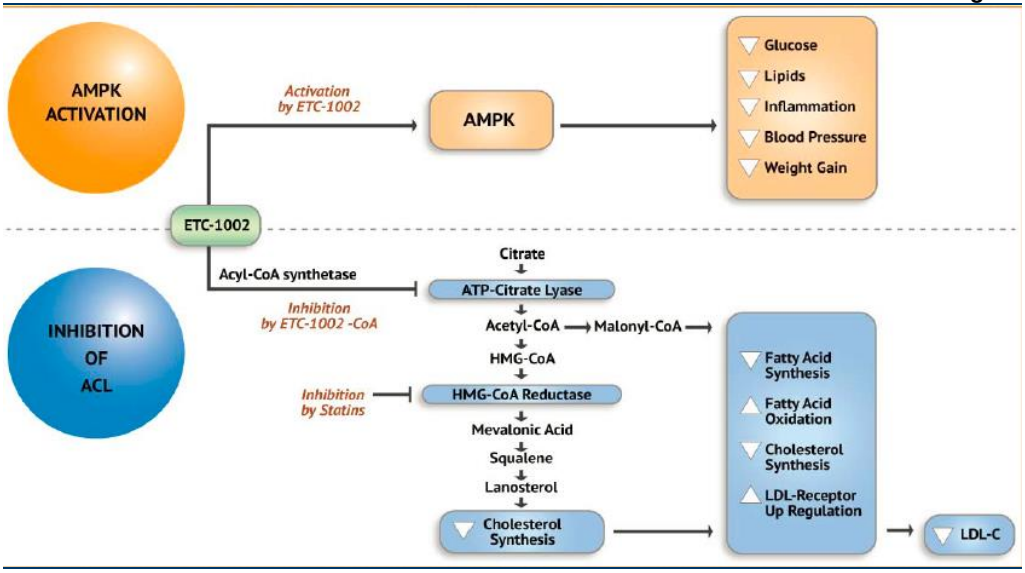
ETC-1002 has several unique properties that potentially improve its therapeutic profile either as an add-on to statins or a therapy for statin intolerant patients.

- **Lowers LDL-C:** The primary activity of ETC-1002 is to lower LDL-C. This is accomplished primarily by blocking ACL upstream of HMG-CoA reductase (the target of statins), which leads to reduced cholesterol synthesis, upregulation of LDL receptors in the liver, and subsequent reduction in LDL-C in the blood.
- **Reduces Fatty Acid Synthesis:** Inhibition of ACL blocks both fatty acid synthesis and cholesterol synthesis. This could potentially lead to additional therapeutic benefit, as LDL-C particles contain both a fatty acid component and a cholesterol component.
- **Additive with Statins:** In preclinical studies, ETC-1002 and statins are additive in upregulating LDL receptors, which is the primary mechanism by which statins lower LDL-C. This suggests the potential for an additive clinical benefit on LDL-C reduction.
- **Metabolic Effect:** The activation of AMPK may result in modest reductions in other cardio/metabolic risk factors. In clinical trials, ESPR has observed reductions in CRP, blood pressure, and insulin.

These favorable physiologic effects are achieved while maintaining important drug properties for a next generation cholesterol drug.

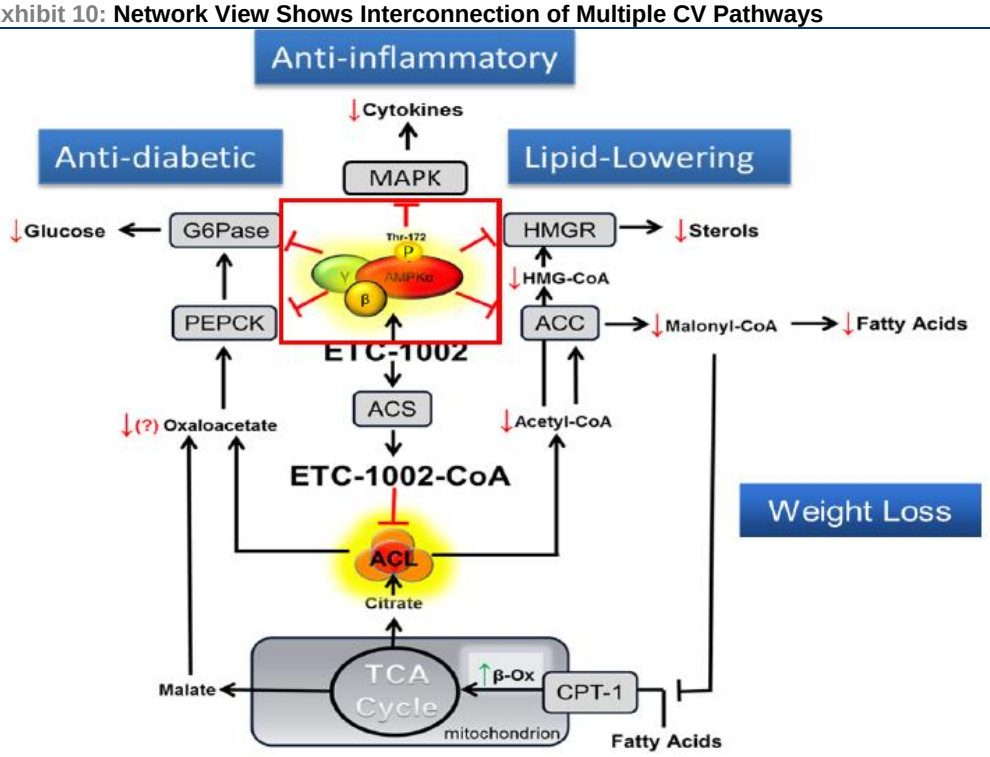
- **ETC-1002 Is an Oral Once-Daily Pill:** This is key for compliance and potentially future co-formulated products.
- **ETC-1002 Does Not Cause Muscle Toxicity:** This is key because muscle toxicity is a class effect of statins and the primary reason for statin intolerance.
- **ETC-1002 Is Taken Up by the Liver Using a Different Mechanism than Statins:** The mechanism is not the OATP1B1 transporter. Variations in the gene encoding the OATP1B1 transporter cause patient-to-patient variability in statin exposure and toxicity for patients on statins.
- **ETC-1002 is metabolized through a different pathway than statins:** The different metabolic pathways reduce the potential for additive toxicity.

Exhibit 9: Dual Action Causes LDL-C Reduction and Other Favorable Metabolic Changes



Source: Company data, Credit Suisse research.

Exhibit 10: Network View Shows Interconnection of Multiple CV Pathways



Source: Company data, Credit Suisse research.

Inhibition of ACL – The Branch Point of Cholesterol and Fatty Acid Synthesis

The inhibition of ACL causes downregulation of both sterol and fatty acid synthesis, which leads to less synthesis of cholesterol, increased expression of LDL receptors in the liver, and a subsequent reduction of LDL-C in the bloodstream. ACL is the enzyme at the

branch point immediately upstream of both fatty acid and sterol synthesis pathways. As such, ACL is upstream of HMG-CoA reductase, the target of statin therapy, in the sterol synthesis pathway. (See Exhibit 9.)

ACL inhibition occurs through an active metabolite of ETC-1002, called ETC-1002-CoA. This compound inhibits ACL from synthesizing acetyl-CoA, which is the key starting substrate for both sterol and fatty acid synthesis.

AMPK Activation – An Important Regulator of Energy Metabolism

AMPK activation regulates energy metabolism by deactivating energy consuming/anabolic pathways (e.g., fatty acid synthesis) and activating energy producing/catabolic pathways (e.g., fatty acid oxidation). Activated AMPK catalyzes inhibitory phosphorylation of multiple enzymes, which causes downstream effects on multiple pathways. Most significant for LDL-C reduction, AMPK catalyzes the phosphorylation of HMG-CoA reductase (HMGR), the target of statins, and acetyl-CoA carboxylase (ACC), a branch point in fatty acid and sterol synthesis. (See Exhibit 10.)

AMPK activation is believed to be responsible for ETC-1002's benefits on other cardio/metabolic risk factors, including reduction in C-reactive protein (a marker of inflammation), weight loss, and a low/modest decrease in blood pressure and blood glucose levels.

AMPK Activators in the Clinic

ETC-1002 is the most advanced direct activator of AMPK in the clinic. Rigel Pharmaceuticals, Kareus Therapeutics, and Debiopharm/Mercury Therapeutics have direct AMPK activators in preclinical development for metabolic conditions.

Metformin and Thiazolidinediones (TZD) are both indirect activators of AMPK. There has not been safety issues associated with AMPK activation in these drug classes, although it is difficult to discern if these drugs are activating AMPK under physiological conditions.

Exhibit 11: AMPK Activators, Marketed or in Development

Company/ Drug	Stage	Notes
Direct		
ESPR/ ETC-1002	Phase II	
Rigel Pharmaceuticals/ R118	preclinical	
Kareus Therapeutics	preclinical	
Debiopharm/Mercury Therapeutics Debio 0930	preclinical	
Indirect		
Metformin	Marketed	
GSK/ Rosiglitazone	Marketed	withdrawn from market due to PPAR related side effects
Takeda/ Pioglitazone	Marketed	
Pfizer/ Troglitazone	Marketed	

Source: Company data, Credit Suisse research.

Clear Efficacy in Multiple Studies

We believe the efficacy of ETC-1002 as a monotherapy for the reduction of LDL-C has been convincingly established. The key take-aways from the Phase I and Phase IIa studies reported to date are that:

- ETC-1002 is well tolerated and lowers LDL-C by approximately 30% (27-42%);
- Trials reported to date include nearly 400 patients (~275 on drug, ~115 on placebo);
- Efficacy has been demonstrated in different patient populations, including statin intolerant and diabetic patients;
- Additional potential benefits have been measured, including significant reductions in systemic inflammation (CRP); and
- Safety signals that require additional follow up and mechanistic explanation include a modest reduction in hemoglobin and a modest increase in uric acid.

Exhibit 12: Completed Clinical Trials of ETC-1002

Study	Phase	Title	Design	Indication	Duration (weeks)	Total Subjects	Treated Subjects
006	2A	Proof of Concept Clinical Trial in Patients with Hypercholesteremia and a History of Statin Intolerance	PBO-Controlled Forced Titration at 60mg, 120mg, 180mg, 240mg	Statin Intolerant	8	56	37
005	2A	Proof of Concept Clinical Trial in Patients with Type 2 Diabetes	PBO-Controlled Forced Titration at 80mg and 120mg	Type 2 Diabetes	4	60	30
004	1B	Multiple-Dose Tolerance Greater Than 120mg Clinical Trial	Multiple Ascending Doses >120mg	Healthy subjects	2	24	18
003	2A	Proof of Concept Clinical Trial in Hypercholesterolemic Patients	Three-doses in parallel PBO-controlled groups	Hypercholesterolemia	12	177	133
002	1B	Multiple-Dose Tolerance Clinical Trial	Multiple Ascending Doses up to 120mg	Healthy subjects	2/4	53	39
001	1A	Single-Dose Tolerance Clinical Trial	Single dose	Healthy subjects	1 dose	18	18
Total						**388**	**275**

Source: Company data, Credit Suisse research.

Study 003: Evidence of Dose Dependent Effects

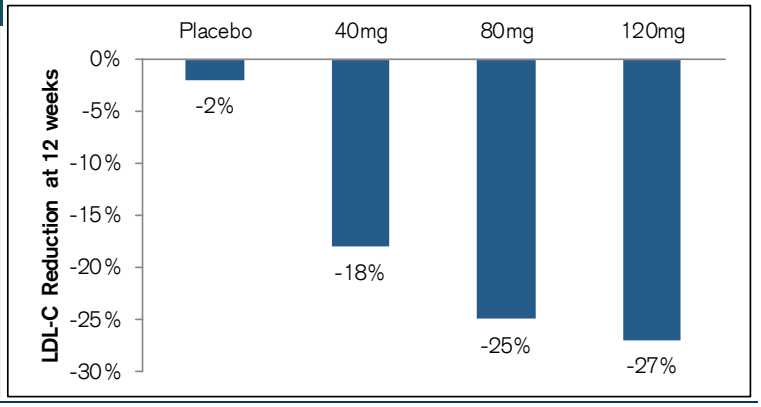
Study 003 was a Phase IIa study in patients with elevated LDL-C who were otherwise healthy. This was the first study demonstrating strong efficacy on par with statins (~30%). The trial tested three doses of ETC-1002 (40, 80, and 120mg) in three parallel placebo-controlled groups and enrolled 177 patients. At the top dose, LDL-C was reduced by 27% (p value < 0.0001).

We review key highlights from the trial.

- **Efficacy:** There was a dose-dependent lowering of LDL-C, with a 27% reduction in LDL-C at the highest dose. (See Exhibit 13.)
- **Safety:** ETC-1002 was well tolerated with no muscle toxicity.
- **Other Signals:** There was a mild decreases in hemoglobin and mild increases in homocysteine, which appeared to be dose dependent. There were mild increases in uric acid in patients in the ETC-1002 groups; the dose dependence was inconclusive.

Exhibit 13: LDL-C Reduction Is Dose Dependent

Study 003	
Stage	Ph 2a
Objective	Proof of concept in hypercholeserolemic patients
# of pts	177 (3:1)
Patients	Hypercholesterolemia LDL-C 130-220 mg/dL
Design	Randomized, pbo-controlled, parallel group
Duration	12 weeks
Doses	pbo, 40, 80, 120mg
Best dose	120mg
Pbo subtracted	-25%
Resulits	Pbo -2% 40mg -18% (p<0.0001) 80mg -25% (p<0.0001) 120mg -27% (p<0.0001)



Source: Company data, Credit Suisse research.

Exhibit 14: Dose Dependent Reduction in Hemoglobin and Homocysteine

ETC-1002 dose	Hemoglobin, g/dL (SE)	Homocysteine, µM (SE)
Placebo	-0.1 (0.7)	0.2 (1.3)
40mg	-0.3 (0.5)	1.3 (2.1)
80mg	-0.5 (0.6)	1.9 (1.8)
120mg	-0.6 (0.6)	2.4 (2.2)

Source: Company data, Credit Suisse research.

Study 005: Biggest Drop in LDL-C in Diabetics

The best reduction in LDL-C that has been seen to date was in diabetic patients (average 43% reduction). It is possible that this drop reflects the natural range of possible outcomes with ETC-1002 given the size of the trial or some other baseline characteristics that might predispose patients to a better response. However, it is also possible that ETC-1002 may have enhanced benefits in diabetics, potentially stemming from its unique dual mechanism of action.

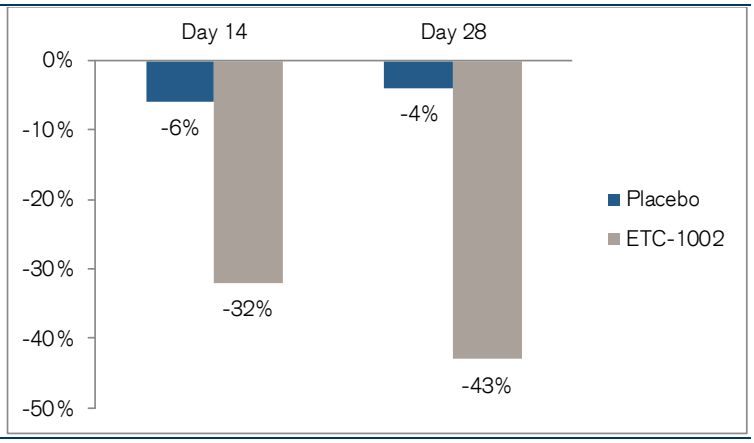
- **The Seemingly Enhanced Activity in Diabetics Is Unexplained:** One hypothesis is that diabetics have more substrate (blood glucose), so inhibition of the sterol and fatty acid synthesis pathway could result in a greater magnitude of LDL-C reduction.

More info on diabetic patients could come from subset analysis of the planned Phase IIb trials, which we do not expect will exclude these patients.

Study 005 enrolled 59 diabetics with high cholesterol. This placebo controlled trial used a forced dose escalation design, where patients were first treated at 80mg for two weeks and then the dose was escalated to 120mg for an additional two weeks. At week two, there was a 32% reduction in LDL-C, and at week four, there was a 43% reduction in LDL-C; both were highly statistically significant versus placebo. (See Exhibit 15.)

Exhibit 15: Biggest Absolute and Relative Drop in LDL-C in Diabetics

	Study 005
Stage	Ph 2a
Objective	Proof of Concept in Patients with Hypercholesterolemia & Type 2 Diabetes
# of pts	59 (1:1)
Patients	Type II diabetes (LDL-C > 100mg/dL)
Design	Randomized, pbo-controlled, forced titration
Duration	4 weeks
Doses	80mg for 2 wks, then 120mg for 2 wks vs. pbo
Best dose	120mg
Pbo subtracted	-39%
Resulits	Day 14: Placebo (-6%); 80mg (-32%) (p<0.0001) Day 28: Placebo (-4%); 120mg (-43%) (p<0.0001)



Source: Company data, Credit Suisse research.

Study 006: 30% LDL-C Reduction in Statin Intolerant Patients

Study 006 was designed to measure the activity of ETC-1002 in a population of patients who were intolerant to two or more statins. ESPR reported results from this trial in June 2013, and the study met its primary endpoint of LDL-C reduction. ESPR is moving forward with a planned Phase IIb trial, testing longer duration.

Key results from study 006 include:

- **Clear Activity:** There was an average 32% reduction in LDL-C at eight weeks in patients who were intolerant to two or more statins. There was a dose response up to 120mg (week four). Doses above 120mg had little additional impact on efficacy.
- **Well Tolerated:** The drug was well tolerated and there were no discontinuations due to muscle pain or weakness. The frequency of muscle-related complaints was the same in the treatment and placebo groups. This is important because all patients had failed prior statins due to adverse events.
- **No Overt Toxicities up to 240mg:** There were no adverse events that appeared with increased ETC-1002 doses up to 240mg, and lab measures associated with organ toxicity were not impacted (e.g., ALT/AST, bilirubin, CK, and creatinine).
- **Hemoglobin Decreased Modestly:** As has been previously observed, there was a dose dependent decrease in hemoglobin (0.7 g/dL reduction at the highest dose). Some patients had more significant drops, with roughly 1/3 of patients experiencing a ≥1 g/dL drop in hemoglobin compared to the baseline and 14% having a hemoglobin drop below the lower limit of normal.
- **Uric Acid Increase:** Uric acid increased modestly, though details of that data were not reported. One patient did have an adverse event of gout, which could have been related to increased uric acid. This patient had a history of gout, so it could be unrelated to treatment. It will be important to see if patients with a history of gout ultimately are excluded from Phase III trials.
- **Reduced Inflammation:** The study also demonstrated reductions in the levels of high sensitivity C-reactive protein (hsCRP), an important marker of inflammation.

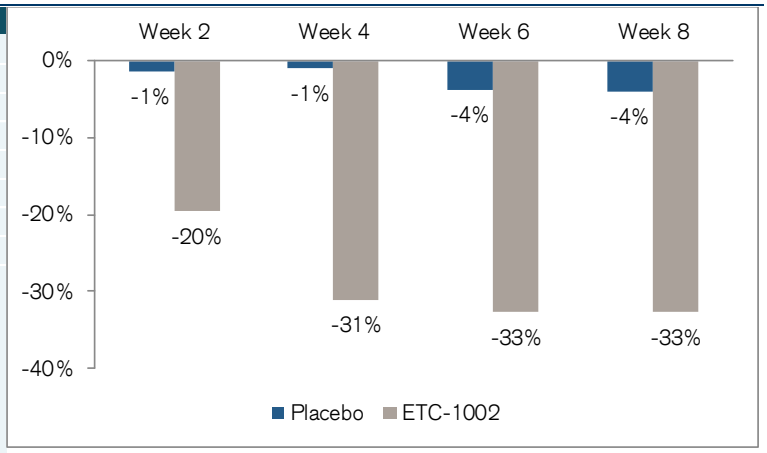
Forced Titration Dose-Response Design

In Study 006, each patient randomized to ETC-1002 was started at the low dose (60mg) and subsequently increased the dose every two weeks (60mg, 120mg, 180mg, and 240mg) for a total of eight weeks of treatment. This design has several strengths and weaknesses.

- **Strengths:** Each patient received each dose of the drug for two weeks, thus serving as his/her own control. Because there were not parallel groups at each dose, inter-group variability was not an issue when comparing across dose groups.
- **Weaknesses:** It is difficult to discern dose response from time dependence. We are confident in the dose response for efficacy. LDL-C levels continue declining up to 120mg and then the benefit tapers off. For safety, it is difficult to interpret if the drop in hemoglobin, seen at the highest dose (240mg) and at the longest time point (eight weeks) is dose or time dependent. We assume from prior experience that the impact is likely dose dependent.

Exhibit 16: Consistent Efficacy in Statin Intolerant Patients

	Study 006
Stage	Ph 2a
# of pts	54 (2:1)
Patients	Hypcholesterolemia (statin intolerant)
Design	Forced titration
Duration	8 weeks
Doses	60, 120, 180, and 240 mg
Best dose	120mg
Pbo subtracted	-29%
Results	Week 2: Placebo (-1.4%); 60mg (-19.5%) p<0.0001 Week 4: Placebo (-1.0%); 120mg (-31.0%), p<0.0001 Week 6: Placebo (-3.8%); 180mg (-32.6%), p<0.0001 Week 8: Placebo (-4.0%); 240mg (-32.6%), p=<0.0001



Source: Company data, Credit Suisse research.

Exhibit 17: Mean Change (SD) in Hemoglobin from Baseline

	ETC-1002 N=37	PBO N=19
Baseline	14.8 (1.0)	14.4 (1.2)
Wk 2 (60mg)	-0.2 (0.4)	0 (0.5)
Wk 4 (120mg)	-0.3 (0.5)	-0.1 (0.4)
Wk 6 (180mg)	-0.5 (0.5)	0 (0.6)
Wk 7 (180mg)	-0.5 (0.5)	-0.1 (0.3)
Wk 8 (240mg)	-0.7 (0.7)	-0.1 (0.6)

Source: Company data, Credit Suisse research.

Exhibit 18: Hemoglobin Abnormalities at Any Visit, Number (%) of Patients

	ETC-1002 N=37	PBO N=19
<LLN	5 (14%)	0
>1 g/dL below LLN	0	0
>2 g/dL below LLN	0	0
<10 g/dL	0	0
≥1 g/dL below baseline	12 (32%)	1 (5%)
≥2 g/dL below baseline	1 (3%)	0

Source: Company data, Credit Suisse research.

Study 007: Residual Risk Trial to Read Out in September 2013

Study 007 tests ETC-1002 in patients who do not achieve their LDL-C goal while on stable statin therapy. The trial has a similar forced dose titration as Study 006 (in statin intolerant patients). In this trial, patients are treated with escalating doses of ETC-1002 on top of a 10mg dose of atorvastatin calcium (Lipitor), the most commonly prescribed dose. Study 007 is expected to read out in September 2013.

Demonstration of clear efficacy and clean safety in this population is important in defining the market opportunity for ETC-1002 beyond the statin intolerant market.

Expectations and risks include:

- **Efficacy:** We believe that the minimum meaningful effect would be an approximate 20% reduction in LDL-C on top of statins. Clearly, the bigger the drop, the better, when considering efficacy. A smaller effect would call into question the market opportunity in this indication.
- **Safety:** We believe that the risk of increased toxicity is low. There is no evidence to date of overlapping toxicity, and both drugs are metabolized and transported by different mechanisms. It is possible that some new safety signal could arise by the more complete inhibition of the cholesterol synthesis pathway.

Exhibit 19: Study 007 Design

	Study 007
Stage	Ph 2a
# of pts	52 (3:1)
Patients	Hypcholesterolemia (residual risk)
Design	Forced titration
Duration	8 weeks
Doses	60, 120, 180, 240 mg
Best dose	NA
Pbo subtracted	NA
Results	Data expected Sept 2013

Source: Company data, Credit Suisse research

Phase IIb Plans: Larger, More Definitive Trials to Read out in H2:14

ESPR already has significant Phase II data on which to design a large randomized trial, and the planned Phase IIb studies in statin intolerant patients and residual risk patients (statin add-on) are intended to provide the support needed to go to Phase III in 2015.

ESPR plans to initiate the Phase IIb trial in statin intolerant patients in Q4:13, with top-line results in H2:14. The initiation of the statin add-on trial will be dependent on the results of Study 007 (data expected in September 2013).

ESPR plans to conduct two randomized, parallel group Phase IIb trials, testing two doses of ETC-1002 versus placebo.

- **Study 008** will treat statin intolerant patients (failed two or more statins) at two or three doses of ETC-1002 for 12 weeks versus Zetia as a control. The exact size and design of the trial have not been disclosed, but the company expects to start the study in Q4:13.
- **Study 009** will evaluate residual risk patients treated with both a statin and ETC-1002 (likely two different doses). The exact size and design of the trial have not been disclosed, but the company expects to start the study in Q4:13.

The details of both trials have not been fully described. Our expectation is that ESPR will test the 120mg dose and potentially one higher dose in both trials, though details have not been released.

Safety Is the Key Risk for this Program

While ETC-1002's efficacy appears relatively derisked at least in statin intolerant patients, questions about the safety and mechanism remain.

Typically, there are three types of safety issues that we consider when looking at drugs for chronic treatment: tolerability, overt toxicity, and unwanted physiologic effects. The balance of these side effects together with the target market helps define the clinical, regulatory, and commercial risks.

- **Tolerability:** Some drugs are hard to take long term because of symptoms such as rash, nausea, or other gastrointestinal side effects, for example. ETC-1002 does not appear to have any tolerability issues. Patients have taken the drug at up to 180mg for eight weeks and 240mg for two weeks. There have been no adverse events leading to drop-outs that have been directly tied to the drug.

- **Overt Toxicity:** Some drugs, including statins, can have direct toxic effects on certain tissues as measured by increased liver enzymes, pancreatic enzymes, or muscle enzymes. To date, ETC-1002 has no overt toxicity issues. Most importantly for the statin intolerant (and potentially add-on) market, there is no muscle toxicity observed with ETC-1002. ETC-1002 is not metabolized through the same pathway as statins, so drug and drug-related toxicities with statins are not expected.

- **Unwanted Physiological Effects:** Some drugs have "on target" or "off target" effects that impact physiology in ways not expected or desired. These can be benign or disastrous. There is evidence from Phase IIa trials that ETC-1002 causes a modest decrease in hemoglobin and a modest increase in uric acid. There have also been reports of mild abnormalities in alkaline phosphatase and homocysteine. There is no current on-target explanation for these observations, but no real clinical significance at this point (There was one patients with gout and a few patients with >2g/dL drops in hemoglobin)

Decreased Hemoglobin and Increased Uric Acid

As previously stated, ETC-1002 is associated with modest decreases in hemoglobin that have not been associated with any clinical adverse events and a modest increase in uric acid that is also asymptomatic (with the exception of one case of gout in a patient with a history of gout).

The biggest risks we see with these findings include:

- **Cumulative Effect:** Our current understanding of these effects is that they are dose related and relatively quick. If it turns out in longer studies that the effects on hemoglobin and uric acid increase over time, that would be a significant setback for a drug that is expected to be given chronically.

- **Exclusion of Patients:** If ESPR or the FDA becomes more concerned about these effects, they may exclude patients from the Phase III trials, for example, those with a history of gout or hemoglobin below a certain threshold. Any exclusion criteria are likely to become part of the eventual product label, and could limit the market opportunity.

- **Monitoring:** If the FDA believes that these lab abnormalities are significant enough, it could mandate that patients be monitored with blood tests periodically during treatment. This would introduce added commercial risk.

- **Potential FDA Concerns:** If the FDA believes that the side effects are significant enough and different enough from other lipid lowering drugs, it could mandate larger or longer trials in Phase III and potentially require an outcomes study for approval.

The FDA's view on these issues will become clearer after the end of Phase II meeting expected in late 2014/early 2015.

The lack of mechanistic understanding of the effect on hemoglobin and uric acid increases regulatory risk and the risk of more serious safety side effects as the drug is dosed longer in more patients. The hemoglobin and uric acid changes could be an unknown consequence of AMPK activation, due to its impact on numerous pathways, or these changes could be caused by an unknown off-target effect.

Partial Clinical Hold Presents Risk

Classification as a PPAR Limits Treatment Duration

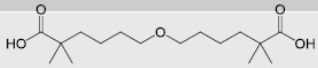
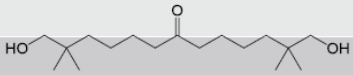
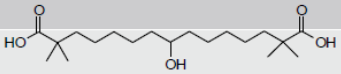
ETC-1002 was discovered through a functional screening assay of compounds that were derivatives of gemfibrozil, a PPAR agonist discovered in 1974 by Parke Davis. The assay was designed to look for compounds that inhibited sterol and fatty acid synthesis. The mechanism by with ETC-1002 was inhibiting sterol and fatty acid synthesis was unknown initially. Because of ETC-1002's structural history as a derivative of gemfiborzil, the FDA deemed the drug a PPAR agonist until proven otherwise.

- The PPAR designation comes with a partial clinical hold, which limits the duration of dosing in humans to six months.
- In order to have the partial clinical hold lifted, the FDA is requiring a two-year carcinogenicity study in two different animal models, rats and mice. The final results are due around YE:14 and are expected to be presented to the FDA at the end of Phase II meeting.

PPAR agonists alter the mitochondrial electron transport chain, which appears to induce a series of metabolic compensatory mechanisms. It is hypothesized that these mechanisms contribute to the toxicity of PPAR inhibitors (cancer induction and liver/cardio toxicity). PPAR agonists have been associated with increased risk of bladder cancer and cardiovascular events. For example, thiazolidinediones have been shown to cause bladder cancer in rats and are suspected to increase the risk in humans.

The company does not believe ETC-1002 activates PPAR at physiologically relevant concentrations, and ETC-1002 has not caused any of the hallmark side effects of PPAR agonists, such as edema, weight gain, increased triglycerides, and increased adiponectin.

Exhibit 20: **Structural Evolution of ETC-1002**

Compound	HLS IC50	Source	Year	Compound Structure
Gemfibrozil	67 µM	Parke Davis	1974	
CI-1027	13 µM	Parke Davis/ Pfizer	1995	
ETC-1001	5-8 µM	Esperion	1999	
ETC-1002 (ESP55016)	3-5 µM	Esperion	2001	
HLS IC50 denotes compound concentration that 50% inhibits lipid synthesis				

Source: Company data, Credit Suisse research.

Preclinical Toxicity Limits Dose to 240mg

There is also a hold on dosing above 240mg. This hold stems from preclinical toxicity that was seen in a batch of monkeys given a 60mg/kg dose of ETC-1002; the monkeys stopped eating and died after two to three weeks. ESPR has repeated these studies in other groups of monkeys at the same dose up to 29 weeks and has not seen this effect.

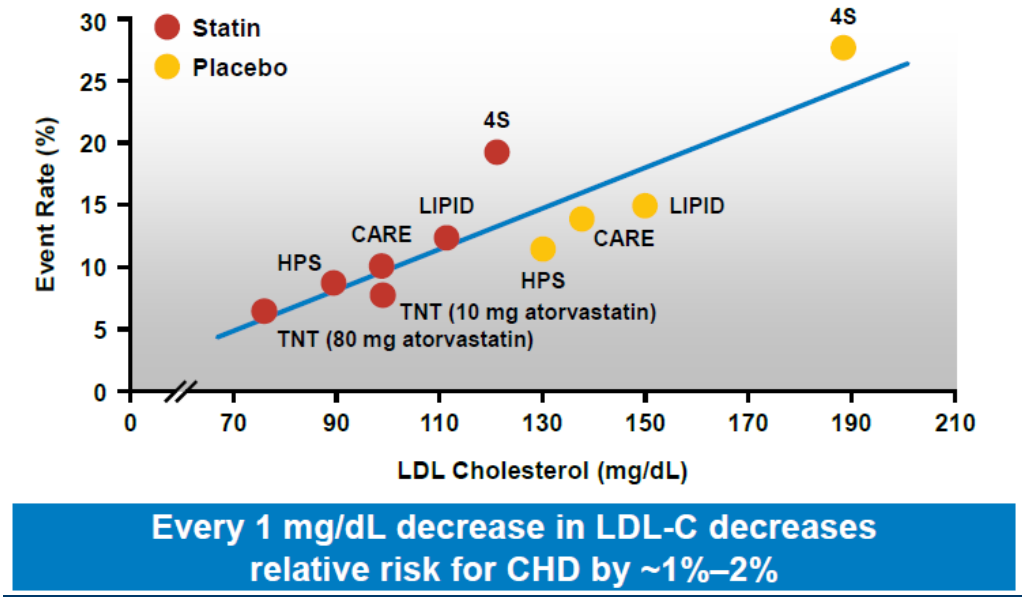
We do not view this as a major concern, no other monkeys have died when the dosing was repeated in longer-term studies. In addition, there appears to be no reason to dose above 240mg, since the drug seems to achieve maximum efficacy at lower doses.

Potential Risk of Outcomes Studies

Statin Intolerant Patients – Outcomes Study Not Expected

We are not expecting the FDA to require an outcomes study for the statin intolerant indication due to the unmet need in these patients and the substantial evidence supporting LDL-C reduction as a surrogate end point in predicting cardiovascular risk. Patients with lower LDL have a lower risk of heart attack, stroke, and other cardiac events. (See Exhibit 21.)

Exhibit 21: Lower LDL-C Correlated with Better Outcomes



Source: Company data, Credit Suisse research.

Statin Add-On/Residual Risk Patients – Outcomes Study Is a Possibility

ETC-1002's unique dual mechanism of action and the increased size of the statin add-on market could lead the FDA to require a pre-approval outcomes study in the statin add-on/residual risk population. We believe that the safety and tolerability data from the Phase IIb trials will be important in handicapping the probability for an outcomes study, assuming positive efficacy.

Regardless of whether it is a regulatory requirement for approval, we believe that the FDA will want ESPR (or its partner) to run an outcomes study post-approval, and most companies in the space are starting these trials in parallel with Phase III.

CETP inhibitors are conducting pre-approval outcomes studies and the PCSK-9 antibodies have ongoing outcomes studies, which are expected to read out after FDA approval.

In our model, we conservatively model a later entrance of ETC-1002 into the statin add-on market, assuming a large outcome study is required. This delays the expanded label until 2021. We may choose to bring forward the approval date as we obtain more data supporting the safety of ETC-1002.

ESPR Management

The management team at ESPR includes many founding members of the original Esperion, which was sold to Pfizer in 2004 for $1.3B. The current Esperion was founded when it acquired the rights to ETC-1002 from Pfizer in 2008. Roger Newton, the executive chairman and CSO, is best known for his discovery of Lipitor while at Warner Lambert.

- **Roger S. Newton, Ph.D. Executive Chairman and Chief Scientific Officer:** Dr. Newton was the co-discoverer of Lipitor (atorvastatin calcium), the most prescribed lipid lowering agent in the world, while at Warner-Lambert (acquired by Pfizer). He was previously the CEO at the first Esperion, which was founded in 1998 and acquired by Pfizer in 2004. Dr. Newton has a Ph.D. in nutrition from the University of California, Davis, an MS in nutritional biochemistry from the University of Connecticut, and a BS in biology from Lafayette College.
- **Tim M. Mayleben, President and Chief Executive Officer:** Mr. Mayleben has over a decade of experience as an executive officer at several life science and healthcare companies. Prior to Esperion, he was the CEO at Aastrom Biosciences. He was the COO of the original Esperion until its acquisition in 2004. Mr. Mayleben has an MBA from Northwestern University and a BBA from the University of Michigan.
- **Noah L. Rosenberg, Chief Medical Officer:** Dr. Rosenberg has prior experience as the medical director at Forest Laboratories, Pfizer, and Sanofi, where he worked in cardiovascular, metabolic, and diabetes drug development. Dr. Rosenberg has an MD from Drexel University and a BS in natural sciences from Johns Hopkins University.
- **Troy A. Ignelzi, VP of Business Development:** Mr. Ignelzi has prior experience in business development at Insys Therapeutics. He also has experience in sales and marketing with Eli Lilly's neuroscience division. He has a BS in accounting from Ferris State University.

Exhibit 22: ESPR Income Statement and Adjusted Product Projections ($M except for EPS)

	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E
Revenues											
Milestones and partnering revenue				78.0	4.0	56.5	59.0	4.0	4.0		
Royalty revenue							10.3	24.7	39.5	77.7	107.8
Total Net Revenues				78.0	4.0	56.5	69.3	28.7	43.5	77.7	107.8
Expenses											
R&D	**8.0**	15.0	20.9	24.6	25.8	27.1	28.5	29.9	31.4	33.0	34.6
G&A	**2.2**	8.1	9.3	11.3	11.9	12.5	13.1	13.7	14.4	15.1	15.9
Total Operating Expenses	**10.2**	23.0	30.2	35.9	37.7	39.6	41.6	43.6	45.8	48.1	50.5
Operating income (loss)	**(10.2)**	(23.0)	(30.2)	42.1	(33.7)	16.9	27.7	(14.9)	(2.3)	29.6	57.3
Total Other Income (Expense)	**(1.5)**	(0.3)	1.3	1.7	1.5	1.5	1.0	1.0	1.0	1.0	1.0
Pre Tax Income	**(11.7)**	(23.4)	(28.9)	43.8	(32.2)	18.4	28.7	(13.9)	(1.3)	30.6	58.3
Income tax expense (benefit)											20.4
Net Income	**(11.7)**	(23.4)	(28.9)	43.8	(32.2)	18.4	28.7	(13.9)	(1.3)	30.6	37.9
EPS - basic (proforma)	**($3.13)**	($2.23)	($1.77)	$2.14	($1.56)	$0.88	$1.37	($0.66)	($0.06)	$1.44	$1.77
EPS - diluted (proforma)	**($3.13)**	($2.23)	($1.77)	$2.11	($1.56)	$0.84	$1.30	($0.66)	($0.06)	$1.36	$1.68
Shares outstanding - basic (proforma)	**3.76**	10.51	16.34	20.46	20.66	20.87	20.97	21.08	21.18	21.29	21.40
Shares outstanding - diluted (proforma)	**3.76**	10.51	16.34	20.74	20.66	22.02	22.13	21.08	21.18	22.46	22.57

Product sales summary	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E
US (prob adjusted)						46.4	101.0	147.1	266.5	369.6
Ex-US (prob adjusted)						12.4	40.4	78.5	177.7	246.4
Total (prob adjusted)						58.8	141.4	225.6	444.2	616.0
Royalty						10.3	24.7	39.5	77.7	107.8

Source: Company data, Credit Suisse estimates

Exhibit 23: Abbreviated BS and CF ($M)

Abbreviated BS	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E
Cash	6.5	71.0	119.3	166.1	137.4	159.3	191.5	181.2	183.3	217.4
Other assets	0.8	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4	1.4
Total assets	7.3	72.4	120.7	167.5	138.8	160.7	192.9	182.5	184.7	218.8
Debt	22.8	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other liabilities	1.9	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
Total liabilities	24.7	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
Common and preferred stock	24.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Additional paid in capital	0.6	135.2	212.4	215.4	218.9	222.4	225.9	229.4	232.9	236.4
Accumulated deficit	(42.0)	(65.4)	(94.2)	(50.5)	(82.7)	(64.3)	(35.5)	(49.4)	(50.8)	(20.1)
Total Liabilities and Equity	7.3	72.4	120.7	167.5	138.8	160.7	192.9	182.5	184.7	218.8

Abbreviated CF	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E
Net income	(11.7)	(23.4)	(28.9)	43.8	(32.2)	18.4	28.7	(13.9)	(1.3)	30.6
Adjustments to net income	0.9	2.5	2.8	3.0	3.5	3.5	3.5	3.5	3.5	3.5
Cash flow from operations	(10.8)	(20.8)	(26.1)	46.8	(28.7)	21.9	32.2	(10.4)	2.2	34.1
Cash flow from investing	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cash flow from financing	15.8	85.4	74.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net cash increase (decrease)	4.9	64.5	48.3	46.8	(28.7)	21.9	32.2	(10.4)	2.2	34.1
Beginning cash	1.6	6.5	71.0	119.3	166.1	137.4	159.3	191.5	181.2	183.3
Ending Cash	6.5	71.0	119.3	166.1	137.4	159.3	191.5	181.2	183.3	217.4

Source: Company data, Credit Suisse estimates

Companies Mentioned *(Price as of 19-Jul-2013)*

Aegerion (AEGR.OQ, $79.81)
Amarin (AMRN.OQ, $5.54)
AstraZeneca (AZN.L, 3280.0p)
Esperion Therapeutics (ESPR.OQ, $16.9, OUTPERFORM[V], TP $23.0)
Gilead Sciences Inc. (GILD.OQ, $59.92)
Isis Pharma (ISIS.OQ, $31.14)
Johnson & Johnson (JNJ.N, $91.89)
Merck & Co., Inc. (MRK.N, $47.81)
Pfizer (PFE.ST, Skr189.5)
Portola Pharmaceuticals, Inc. (PTLA.OQ, $22.95)
Rigel (RIGL.O, $3.79)
Takeda Pharmaceutical (4502.T, ¥4,730)
The Medicines Company (MDCO.OQ, $30.92)

Disclosure Appendix

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Risk: Risks to our $23 TP for ESPR include factors that could decrease our probabilities of success for ETC-1002 in statin intolerant and statin add-on markets: 1) inability to remove the partial clinical hold, 2) lack of efficacy or any new toxicities in ongoing trials, 3) increased concern over known safety signals (decreases in hemoglobin and increases in uric acid), and 4) more stringent regulatory requirements (ie FDA requiring an outcome study for approval).

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