Research Note

Cardiovascular, Devices and Regenerative

Anthera Pharmaceuticals (ANTH)

Attractive Pipeline in ACS and Lupus; Initiating with OUTPERFORM and $8 Fair Value

- **Anthera has two unpartnered drugs in late-stage development, both targeting blockbuster markets**. Anthera is well positioned as a development stage biotech company, possessing two late-stage drug candidates, each of which could be best-in-class with blockbuster potential. As noted below, the company now has sufficient cash to sustain itself through several critical near-term milestones. We believe investors should seriously consider ANTH, as we believe success in either A-002 in Acute Coronary Syndrome (ACS) or A-623 in Lupus could provide significant returns.
- **Varespladib (A-002) in SPA-backed pivotal study for the reduction of adverse events in ACS.** A-002 is an oral inhibitor of sPLA2 that has been shown to hold potent anti-inflammatory, lipid-lowering and lipid-modulating effects. Anthera is currently studying A-002 in a pivotal Phase III trial, VISTA-16, for the short-term (16-week) treatment of patients experiencing an acute coronary syndrome where it is poised to become the first anti-inflammatory therapeutic approved for prevention of MACE (major adverse cardiovascular events), where we predict peak sales could exceed $1 billion per year. In addition to these acute applications, A-002 may also prove useful as chronic therapy where sales could possibly reach several billions of dollars per year.
- **A-623 has exciting potential in Lupus.** Anthera is rapidly progressing with A-623 in Lupus following the success of Human Genome Sciences' Benlysta, which stands to be the first drug approved in Lupus in decades. Like Benlysta, A-623 targets BLyS, but A-623 may be superior in terms of efficacy (by also targeting membrane-bound BLyS) and ease-of-use (SC administration). While Lupus drug development has historically been a difficult endeavor, we are encouraged by the validation of BLyS as a target, while the blockbuster potential for A-623 should drive interest from big pharma.
- **We predict that Anthera has cash runway well past potentially valuable near-term milestones.** We estimate that Anthera has roughly $71 million in cash, which we predict to last the company through several critical milestones, including: (1) interim Phase III data for A-002; (2) interim Phase IIb data for A-623; and (3) initiation of a Phase IIb program of A-001 in Acute Chest Syndrome. Additional potential sources of cash, which could include licensing fees for partnership of A-002 and/or A-623 may even support the company into profitability.
- **Initiating with OUTPERFORM rating and fair value of $8 per share.** Our fair value is calculated using a sum-of-parts analysis, applying a 30% annual discount to our peak annual sales estimate for A-002 in ACS, and A-623 in SLE, incorporating a 1-10 multiple for each based on stage of clinical risk. At a current market price of $4.15 we believe that Anthera is at an attractive valuation with the potential to increase 93% in the next 12 months. We are initiating research coverage with an OUTPERFORM rating.
- **Risks to the attainment of our fair value include risks that**: Anthera's products obtain disappointing clinical trial results and or fail to obtain regulatory approval; Physicians are not be impressed with the products' clinical profiles; Anthera or a partner fails to effectively commercialize Anthera's drug candidates; third-party patents prevent the timely commercialization; superior clinical results are obtained by a third-party competitor; Anthera is unable to raise needed capital.

	2009A	2010E			2011E		
REV. ($m)	ACTUAL	CURR.	PREV.	CONS.	CURR.	PREV.	CONS.
Q1 Mar		$0.0E			$0.0E		
Q2 Jun		0.0E			0.0E		
Q3 Sep		0.0E		$0.0E	0.0E		
Q4 Dec		0.0E		0.0E	0.0E		
Year	**$0.0**	**$0.0E**		**$0.0E**	**$0.0E**		**$0.0E**
	2009A	**2010E**			**2011E**		
EPS	**ACTUAL**	**CURR.**	**PREV.**	**CONS.**	**CURR.**	**PREV.**	**CONS.**
Q1 Mar		($0.83)A			($0.36)E		
Q2 Jun		(0.36)A			(0.40)E		
Q3 Sep		(0.43)E		$(0.51)E	(0.41)E		
Q4 Dec		(0.33)E		(0.67)E	(0.46)E		
Year	**($8.06)**	**($1.74)E**		**$(2.26)E**	**($1.64)E**		**($2.21)E**

October 6, 2010

Price (intraday 10/6/10)
$4.15

Rating
OUTPERFORM

Fair Valuation
$8

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Company Information

52-Week Range	$2.82 - $8.55
Shares Outstand.	38.6 million
Avg. daily volume	51,600
Market Cap.	$160 million
ST / LT Debt	$0 M / $0 M
Debt/Capital	N/A
ROE	(44.6%)
Cash & Inv/Share	$1.84
Book Value/Share	$2.56

Company Description

Anthera Pharmaceuticals is biopharmaceutical company focused on developing products to treat inflammatory disorders, including cardiovascular and autoimmune diseases. Anthera currently has one Phase 3 clinical program, A-002, as well as two Phase 2 clinical programs, A-623 and A-001.

Source: Nasdaq.com

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Investment Summary

- **With two late-stage clinical programs, each currently unpartnered yet holding blockbuster potential, we believe Anthera presents a compelling investment opportunity**. Anthera is well positioned as a development stage biotech company, possessing two late-stage drug candidates, each of which is poised to become best-in-class, and each of which holds blockbuster potential. As noted below, the company now has sufficient cash to sustain itself through several critical near-term milestones. We believe investors should seriously consider ANTH, as we believe success in either A-002 (Varespladib) or A-623 could provide significant returns.
- **Anthera's Phase III drug candidate, Varespladib (A-002), could be first drug approved for potentially blockbuster indication of reduction of adverse events in Acute Coronary Syndrome.** A-002 is an orally-administered inhibitor of the IIa, V and X forms of the sPLA2 enzyme, which has been shown to hold potent anti-inflammatory, lipid-lowering and lipid-modulating effects. When combined with lipid-lowering therapies such as the statin drugs, A-002 offers a unique and synergistic approach targeting inflammation, elevated lipid levels and atherosclerosis, each of which has been shown to play an important role in the generation of adverse events. Anthera is currently studying A-002 in a pivotal Phase III trial for the short-term (16-week) treatment of patients experiencing an acute coronary syndrome where it is poised to become the first anti-inflammatory therapeutic approved for prevention of MACE (major adverse cardiovascular events), where we predict peak sales could exceed $1 billion per year. In addition to these acute applications, A-002 may also prove useful as chronic therapy where sales could possibly reach several billions of dollars per year.
- **Anthera's Phase IIB drug candidate, A-623, may prove superior to much-anticipated Benlysta**. After 50 years without a new drug approved for the treatment of Systemic Lupus Erythematosus, Benlysta is widely anticipated to receive approval around December 2010. Benlysta is the first of a new class of drugs directed to inhibiting the BLyS cytokine, and is anticipated to both provide patients a new a therapeutic option, as well as a lucrative commercial opportunity for developers Glaxo-Smith-Kline and Human Genome Sciences. That said, Anthera's own BLyS antagonist, A-623, may prove superior to Benlysta in both efficacy (unlike Benlysta, A-623 binds to both soluble and membrane-bound BLyS) and ease-of-use (while Benlysta is administered via a monthly intravenous infusion, A-623 is given via subcutaneous injection). For these reasons, we expect excitement to grow for Anthera as Benlysta reaches the market, and as investors begin to focus on the next-generation of drugs which could displace it.
- **We believe Anthera is poised to benefit from several potentially-positive near-term catalysts**. By mid-2011, we expect two data sets, each of which will provide important feedback on one of ANTH's ongoing clinical trials. First, a biomarker data set for the ongoing A-002 pivotal VISTA-16 trial is expected in Q1:2011. Although the trial's primary endpoint is directed to a potential reduction in cardiovascular events, A-002 is expected to achieve this effect through reductions in both lipid levels and inflammation, which can be measured via various biomarkers. The results of a preliminary examination of these biomarkers in VISTA-16 trial patients will be revealed early next year. A positive result will provide persuasive evidence that A-002 continues to perform as expected, and significantly derisk the ongoing trial. Second, in Q2:2011, we're expecting preliminary data from the ongoing Phase IIb trial of A-623 focusing on the drug candidate's effects on B-cell reduction. Should significant decreases be seen here, we believe this program will be incrementally derisked. Finally, given their relatively late stages in clinical development, potential partnerships for both A-002 and A-623 are possible next year, particularly for the former.
- **We project potential profitability in 2014, with potential for acquisition beginning in early 2012**. Should ANTH remain independent, we anticipate that the company could reach profitability in mid-2014 based on future royalties for A-002. However, A-002's blockbuster potential and the fact that it has already begun its pivotal clinical program, we believe that acquisition of ANTH remains a likely outcome, particularly once the story is sufficiently derisked. Such derisking should occur by Q1:2012, when VISTA-16 data is expected, although the preliminary biomarker data (expected in Q1:2011) could possibly inspire acquisition before then.
- **We believe that Anthera has relatively low near-term financing risk.** Anthera recently raised $29.3 million in net proceeds through a PIPE financing in late September 2010. We estimate that Anthera currently has roughly $71 million in cash, which should provide most of the financing needs for the ongoing late stage A-002 and A-623 clinical studies. While we do not expect Anthera to seek additional financing until late 2011, we note that the company may satisfy its capital needs by signing one or more partnership agreements which could provide the company with a significant up-front cash payment.
- **We believe that Anthera has sufficient liquidity.** The company has a roughly $160 million market capitalization, with an average daily volume of approximately 52,000 shares.
- **We calculate ANTH is trading at an attractive valuation with 93% potential upside to our fair value estimate of $8/share.** Our fair value calculated using a sum-of-parts analysis of Anthera's clinical pipeline. A 30% annual discount is applied to our estimate of peak annual sales for each clinical stage product/indication and a 1-10x multiple is applied to our current value based on stage of clinical development to reflect risk. While we list fair value estimates for each product in the pipeline, our overall fair value for the stock only includes fair value estimates for product candidates/indications which, in our view, have at least positive clinical proof-of-concept data. As a result, this fair value does not include A-002 in stable coronary artery disease or A-001 and A-003 in any indications (*Please contact your Wedbush PacGrow LifeSciences salesperson for full calculations*.)

Introduction

Anthera Pharmaceuticals is biopharmaceutical company focused on developing drug candidates to treat inflammatory disorders, including cardiovascular and autoimmune diseases. Anthera currently has one Phase III clinical program, A-002, as well as two Phase II clinical programs, A-623 and A-001. The company's product development portfolio is summarized in the table below:

Exhibit 1. Anthera's Clinical Pipeline.

	Product Candidate	Co-therapy	Development Phase	Description	Next Milestones
Lead Programs	A-002-(varespladib methyl)	Atorvastatin (Lipitor)	Phase III	Orally administered sPLA2 inhibitor being evaluated for the prevention of secondary MACE following an acute coronary syndrome (16-week treatment).	Biomarker data in late Q1:2011.
	A-623	None	Phase IIb	Selective peptibody antagonist of BLyS cytokine being developed for the treatment of systemic lupus erythematosus.	Interim B-cell reduction data in Q2:2011.
	A-001-(varespladib sodium)	None	Phase II	Intravenous sPLA2 inhibitor with orphan drug and fast track status. Being evaluated for the prevention of acute chest syndrome in hospitalized patients with sickle cell disease.	Initiate Phase IIb clinical study in H1:2011.
Additional Programs	A-002-(varespladib methyl)	Niacin	Phase II ready	Orally administered sPLA2 inhibitor being study as potential therapy to improve lipid profiles and reduce flushing in cardiovascular patients on niacin therapy.	Potential label expansion study post VISTA-16 data.
	A-003	None	Preclinical	Second generation sPLA2 inhibitor being developed for cardiovascular diseases.	Continue preclinical development.

Source: Anthera Pharmaceuticals, Wedbush Securities research.

As shown above, three of Anthera's product candidates (A-002, A-001 and A-003) are designed to inhibit a novel enzyme target known as secretory phospholipase A2 (sPLA2), which acts to directly amplify inflammation and abnormally modify lipids. Elevated levels of sPLA2 have been shown to play a central role in a variety of acute and chronic inflammatory conditions, including: (1) acute coronary syndrome; (2) acute chest syndrome associated with sickle cell disease; and (3) stable coronary artery disease. Anthera is currently developing sPLA2 inhibitors for the first two of these indications, with plans to also pursue the third.

Anthera's most advanced sPLA2 inhibitor, A-002, operates through a novel mechanism of action to offer both targeted anti-inflammatory activity and incremental lipid reductions when used in combination with commonly prescribed statin drugs. Indeed, when combined with statins, A-002 is one of only a few therapeutics in development with the potential to offer a unique and synergistic approach to targeting inflammation, elevated lipid levels and atherosclerosis. A Phase III trial of A-002 (VISTA-16) is currently underway and we expect Anthera to report interim biomarker data in late Q1:2011. We believe that A-002 provides a compelling, differentiated, and relatively rapid pathway for a company to enter the lucrative cardiovascular drug market.

Second, Anthera's Phase IIb product candidate, A-623, targets elevated levels of B-lymphocyte stimulator (BLyS), which has been associated with a variety of B-cell mediated autoimmune diseases including systemic lupus erythematosus, lupus nephritis, rheumatoid arthritis, multiple sclerosis, Sjögren's Syndrome, and Graves' Disease. Anthera initiated a 600 patient Phase IIb study of A-623 in July 2010, and we expect the company to report interim data from the study in Q2:2011. Anthera may also opportunistically enter into collaborations with third parties for development of this compound in lupus or in other B-cell mediated diseases.

Finally, Anthera is also developing A-001, an intravenous sPLA2 inhibitor for prevention of acute chest syndrome associated with sickle cell disease, where elevations in sPLA2 activity are known to precede and predict disease progression. Because there are currently no approved drugs for the prevention of acute chest syndrome associated with sickle cell disease, Anthera has received orphan drug designation and fast track status from the FDA for this drug candidate and indication.

Anthera was incorporated in Delaware on September 9, 2004 and the company's headquarters are located in Hayward, California. The company underwent its IPO in March 2010, and currently trades on Nasdaq under the symbol ANTH.

Near-Term Milestones, Pipe-line Predictions, Valuation and Cash Runway

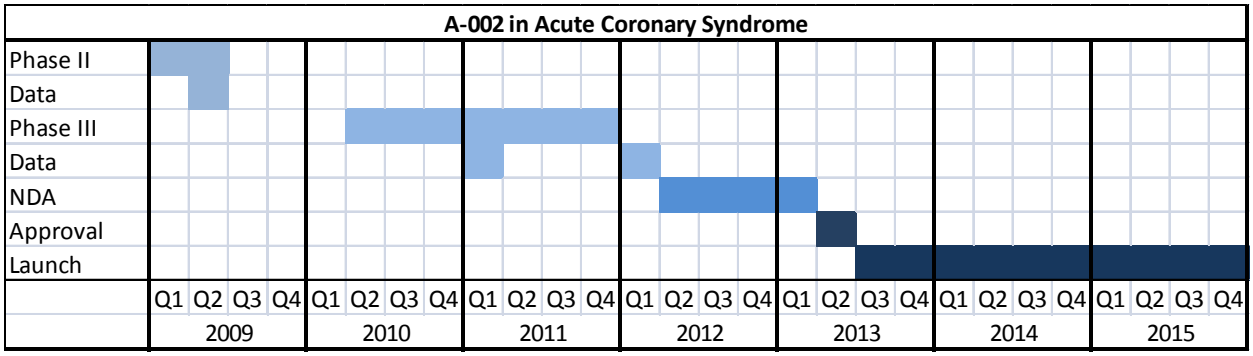
Near-Term Milestones

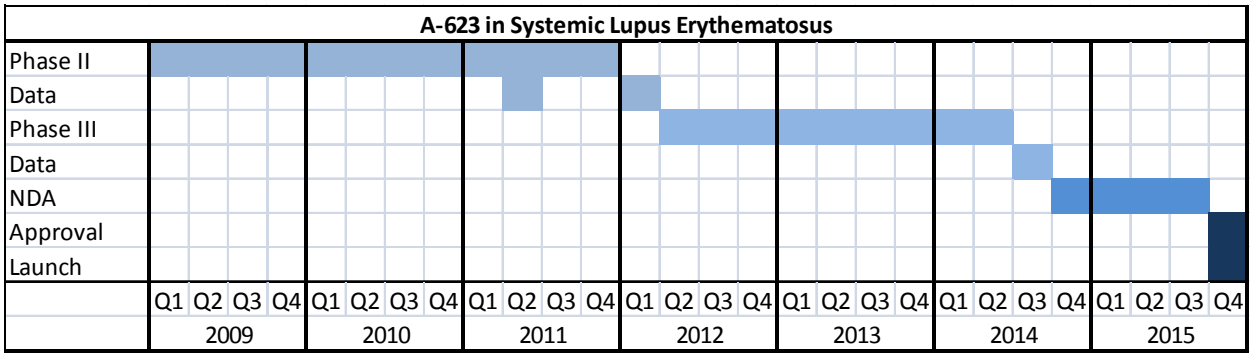
We estimate the following near-term milestones for Anthera:

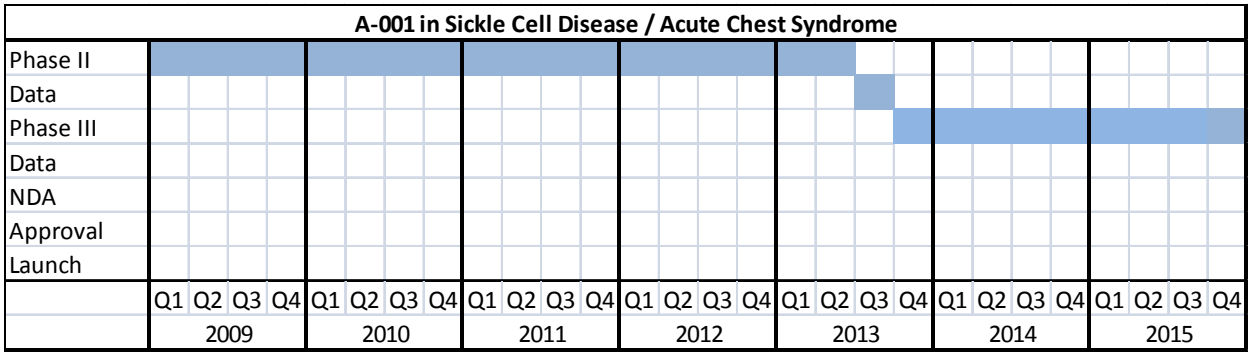
2010	Q4:2010	Release of full Phase II IMPACTS data of A-001 in Acute Chest Syndrome
	Q4:2010	Expected approval and launch of Benlysta in Lupus (GSK and HGSI)
	YE:2010	Expect 1,000 patients enrolled in VISTA-16 Study
2011	Q1:2011	Biomarker data from interim analysis of VISTA-16 in late Q1:2011
	Q2:2011	B-cell reduction data for A-623 in Lupus
	H1:2011	Initiate Phase IIb Study of A-001

Pipeline Predictions

We predict the following timeline for ANTH's product pipeline:







Valuation

Our fair value of $8 per share is calculated using a sum-of-parts analysis of Anthera's clinical pipeline. A 30% annual discount is applied to our estimate of peak annual sales for each clinical stage product/indication and a 1-10x multiple is applied to our current value based on stage of clinical development to reflect risk. While we list fair value estimates for each product in the pipeline, our overall fair value for the stock only includes fair value estimates for product candidates/indications which, in our view, have at least positive clinical proof-of-concept data. As a result, this fair value does not yet include A-002 in stable coronary artery disease, or A-001 and A-003 in any indications. (*Please contact your Wedbush PacGrow LifeSciences salesperson for full calculations*.)

We use multiples to account for clinical and regulatory risk at various stages of development.

NOVEL DRUGS

1: in preclinical testing	6: In Pivotal Trial
2: passed preclinical	7: Pivotal data
3: IND filing	8: regulatory review
4: Phase I data	9: approved
5: Phase II data	10: launched

Today: 10/6/10	Stock	MktCap ($000)	Upside
Wedbush Current Fair Value for ANTH	$7.57	$291,788	82%
Current Full Pipeline Value:	$7.57	$291,788	82%
Cash:	$1.84	$71,108	
ANTH Total Value:	$9.41	$362,896	127%
Current ANTH Stock:	$4.15	$160,024	
ANTH Shares Outstanding (000):	38,560		

Anthera Product Pipeline Valuation

Product		Indication	Eligible # Annual WW Treatments Est	Pricing $ per Patient per Year Est/Actual	Peak Penetration Est	Gross WW Peak Sales Est ($000)	ANTH Net Peak Revs Est WW ($000)	Est/Actual Launch	Multiple	Annual Discount Rate	Wedbush MktCap Fair Value ($000)	Wedbush Stock Fair Value
sPLA2 Antagonists	A-002	Acute Therapy	3,500,000	$2,114	15%	$1,110,000	$194,250	7/1/2013	6	30%	$199,011	$5.16
		Chronic Therapy	10,000,000	$2,363	5%	$1,181,250	$206,719	6/1/2015	5	30%	$106,707	$2.77
	A-001	Sickle Cell Crisis	175,000	$22,143	20%	$775,000	$135,625	1/1/2017	4	30%	$47,987	$1.24
BLyS Antagonist	A-623	Systemic Lupus Erythematosus	600,000	$17,083	10%	$1,025,000	$205,000	12/1/2015	5	30%	$92,777	$2.41

Source: Wedbush Securities research

Cash Runway

At the end of Q2:2010, Anthera reported $51 million in cash and equivalents. Moreover, Anthera recently raised $29.3 million in net proceeds through a PIPE on September 24, 2010.

Meanwhile, we predict that Anthera needs roughly $150 million in to reach profitability, anticipated in Q2:14. We estimate that the Phase III Vista-16 Study will cost roughly $70 to $85 million, and that the Phase IIb study in A-623 will cost approximately $25 to $28 million. Both of these trial results may represent attractive partnership and/or acquisition opportunities, which could provide Anthera with sufficient cash from upfront payments and milestones to support the company until profitability.

Role of sPLA2 in Inflammation and Disease

Inflammation and Disease

Inflammation is a powerful and complex biological response of vascular tissue to harmful stimuli. It serves both a protective role and as a critical initiator of the healing process. However, inflammation is also a somewhat messy process, with widespread and often deleterious effects on nearby tissues. As a result, the inflammatory process is typically kept under tight control by the body to ensure appropriate activation and prompt resolution.

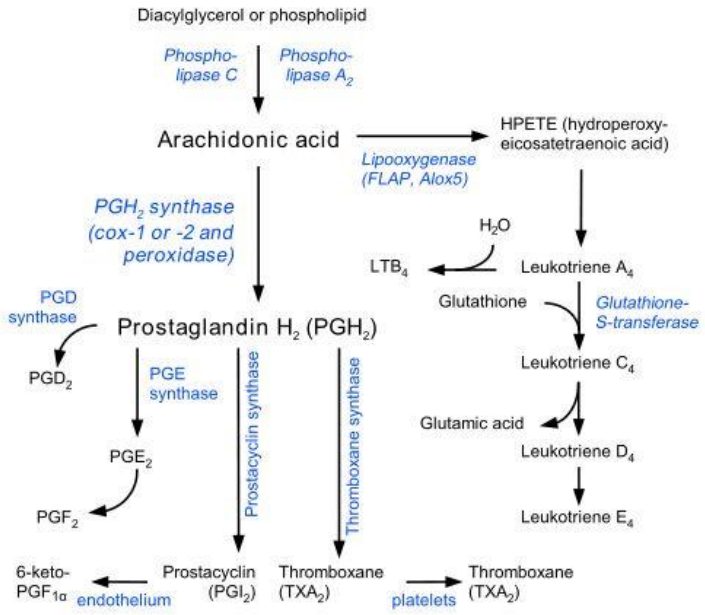
However, under certain circumstances, such as with autoimmune and also cardiovascular diseases, control over the inflammatory process malfunctions, leading to inappropriate activation and/or prolongation. In this event, the inflammatory process damages the body's own cells and tissues without an underlying and ongoing need. While designed to protect, in this case inflammation actually does more harm than good.

Building upon the company's knowledge of inflammatory pathways and the growing body of evidence that links inflammation to multiple disease states, Anthera believe that it has have developed a leadership position with respect to inhibition of secretory phospholipase A2 (sPLA2), an enzyme thought to play a key role in the pathogenesis of a variety of medical disorders. Anthera is believed to currently have the two most advanced sPLA2 inhibitors in clinical development.

sPLA2 Biology

sPLA2 is a family of enzymes thought to play a key role in both acute and chronic inflammation. sPLA2 enzyme activity is elevated during the early stages of inflammation, and the enzyme is produced in response to inflammatory signals such as IL-1 and TNF. sPLA2's acute effects amplify the inflammatory process by catalyzing the breakdown of phospholipids into arachidonic acid, the first step in the arachidonic acid pathway. As shown in Exhibit 2, arachidonic acid is then further metabolized into a variety of inflammatory mediators such as Leukotrienes, Prostacyclins, Prostaglandins and Thromboxanes. These mediators are directly responsible for causing damage to cells and tissue during the inflammatory process. By creating the final common source (arachidonic acid) for each of these mediators, sPLA2 activity plays a key role in amplifying early inflammatory signals.

Exhibit 2. Arachidonic Acid Pathway.



Source: Wikipedia.

The known family of sPLA2 enzymes includes at least three forms that have been shown to play a role in inflammation and the development of cardiovascular disease and lung injury.

While both sPLA2 and lipoprotein associated phospholipase A2 (Lp-PLAs) are members of the same enzyme family, and while both are present in the blood, important differences do exist between them. In particular, Lp-PLA2 mostly binds to LDL-C and high-density lipoprotein (HDL) while sPLA2 does not. Even more importantly, clinical studies suggest that unlike with Lp-PLA2, inhibition of sPLA2:

- is synergistic with statins in reducing LDL-C, total cholesterol, and non-HDL cholesterol in patients with CAD;
- lowers circulating small, dense and pro-atherogenic, or plaque-building LDL-C particles; and
- has been shown to lower CRP in a statistically significant manner.

As a result, a drug designed to inhibit sPLA2 activity may offer a unique opportunity to reduce complications associated with certain inflammatory diseases, above and beyond that allowed through standard approaches. As discussed in more detail below, Anthera is exploring this opportunity among both cardiovascular patients with acute coronary syndrome, and sickle cell patients with acute chest syndrome.

A-002 – Presents Compelling Opportunity in Acute Coronary Syndrome

As described in more detail in a subsequent section, A-001 is a broad-spectrum, once-daily inhibitor of the IIa, V and X forms of the sPLA2 enzyme. A-002, discussed here, is an orally administered pro-drug of A-001. Both of these compounds have demonstrated potent anti-inflammatory, lipid-lowering and lipid-modulating treatment effects in multiple clinical studies.

When combined with lipid-lowering therapies, A-002 is one of only a few therapeutics in development with the potential to offer a unique and synergistic approach targeting inflammation, elevated lipid levels and atherosclerosis as part of physician-directed standard of care. Anthera is currently developing A-002 for short-term (16-week) treatment of patients experiencing an acute coronary syndrome. Indeed, if Anthera's Phase III program proves successful, A-002 is poised to become the first anti-inflammatory therapeutic approved for prevention of MACE.

Acute Coronary Syndrome

According to the American Heart Association, over 18 million people in the United States have experienced an acute coronary syndrome and roughly 1.5 million Americans will have a new or recurrent heart attack this year alone. The AHA also estimates that worldwide, cardiovascular disease kills an estimated 17.5 million people each year. Likewise, coronary artery disease is thought to account for 1.9 million deaths in Europe annually. These numbers are expected to only increase given the aging population, as well as rising epidemics of diabetes and obesity.

The AHA defines acute coronary syndrome as any group of clinical signs and symptoms related to acute myocardial ischemia. Acute coronary syndrome covers a spectrum of clinical conditions that include ST-elevated myocardial infarction (STEMI), non-ST-elevated myocardial infarction (NSTEMI), and unstable angina. Both STEMI and NSTEMI are types of heart attack, where damage to the heart muscle occurs due to lack of sufficient blood flow. Meanwhile, unstable angina results in chest pain from ischemia, but does not cause permanent damage to the heart muscle.

For patients who experience an acute coronary syndrome, the risk of a secondary major adverse cardiovascular events (MACE) is significantly increased immediately after the initial event. For people over the age of 40, the AHA estimates that 20% will die within one year of an initial heart attack, and over one-third will die within the first five years. Moreover, large clinical outcome studies such as MIRACL and PROVE-IT have reported the 16-week rate of secondary MACE in acute coronary syndrome patients to be between 6.1% and 14.8%.

Current treatments for coronary artery disease include a variety of drugs such as aspirin, statins as well as anti-platelet and anti-coagulant agents. For patients presenting with acute coronary syndrome, therapy is administered quickly to improve blood flow to the heart and limit the risk associated with continued ischemia and clotting. Moreover, medications such as statins are prescribed in an attempt to provide chronic protection against secondary MACE through improvement in lipid profiles such as lowering LDL-C. Finally, interventional procedures such as PCI, and surgical procedures such as CABG, are employed as a last resort because of their invasiveness, expense and risk to patients.

Inflammation in Cardiovascular Disease

In patients experiencing an acute coronary syndrome, the relationship between higher levels of inflammation and increased risk for MACE has been demonstrated extensively. For example, reductions in c-reactive protein (CRP) have been correlated with reductions in subsequent MACE in numerous clinical studies among a variety of clinical interventions.

Exhibit 3. Relationship between CRP and Cardiovascular Disease.

Study	Design	Results
MIRACL-1 (JAMA 2001)	3,086 patients with acute coronary syndrome were randomized within 96 hours of their index event to treatment with high-dose atorvastatin or placebo.	Atorvastatin significantly reduced secondary MACE after 16 weeks (14.8% vs. 17.4%).
MIRACL-2 (Circulation 2003)	Same as above.	After 16 weeks, CRP was 34% lower with atorvastatin compared to placebo.
PROVE-IT (NEJM 2005)	3,745 patients were randomized to either intensive statin therapy with 80 mg atorvastatin or moderate statin therapy with 40 mg pravastatin.	Patients with low CRP or LDL-C had fewer MACE than those with higher levels (2.7 vs. 4.0 events per 100 person-years). Patients who had both LDL-C < 70 mg/dL and CRP < 1 mg/L had the fewest number of secondary events overall.

Sources: Schwartz GG et al JAMA 2001; Kinlay S et al Circulation 2003; Ridker PM et al NEJM 2005

CRP is the most commonly used marker of inflammation.

Although a causative role for CRP in cardiovascular disease has not yet been established, inflammation is known to promote acute coronary syndrome. Accordingly, CRP may play a direct role in both vascular inflammation as well as plaque rupture.

LDL-C in Cardiovascular Disease

The direct relationship between lower LDL-C levels and reduced risk for major MACE has been even more clearly demonstrated. In particular, results from large clinical outcome studies demonstrate that lowering LDL-C levels reduces the risk of future cardiovascular events and provides continued patient benefit.

Moreover, studies also show that this benefit is incremental to reductions in CRP. For example, the results of the JUPITER study published in November 2008 in the New England Journal of Medicine, showed that reducing CRP resulted in a statistically significant reduction in MACE events, well above that which would be predicted from historical data evaluating LDL-C reductions alone.

Exhibit 4. Relationship between CRP, LDL-C and Cardiovascular Disease.

Study	Design	Results
JUPITER (NEJM 2008)	17,000 patients with relatively normal levels of LDL-C, but elevated levels of inflammation (based on CRP) were randomized to either statin therapy or placebo.	The study was stopped early because those patients randomized to statin therapy demonstrated a statistically significant reduction in CRP (which also translated to a statistically significant reduction in cardiovascular events) versus those patients on placebo. The reduction in events was well in excess of that which would be predicted from historical data evaluating LDL-C reductions alone.

Source: Ridker PM et al, New England Journal of Medicine 2008

In light of the growing appreciation of LDL-C's role in MACE, lipid treatment guidelines have been revised to establish more aggressive LDL-C treatment goals over time. The most recent guidelines from the National Cholesterol Education Program's Adult Treatment Panel III (updated in 2004) advocate treatment goals for LDL-C below 100 mg/dL for high-risk patients and 70 mg/dL for very high-risk patients.

sPLA2 Activity in Cardiovascular Disease

Excess sPLA2 activity appears to have acute and chronic implications on disease progression and patient outcomes in ACS. For instance, significant elevations in sPLA2 activity and mass have been seen from 24 hours to two weeks following an ACS event, and have been shown to persist for up to an additional 12 weeks thereafter. Shortly after a heart attack, sPLA2 increases dramatically, amplifying inflammatory activity that is itself associated with more frequent and secondary cardiovascular events. Indeed, clinical studies also demonstrate that sPLA2 independently predicts coronary events in patients that have recently experienced an ACS event and in patients with stable CAD independent of other standard risk factors.

Clinical Development

Eli Lilly and Shionogi & Co., Ltd. conducted at least 17 Phase I and Phase II clinical studies evaluating A-002 and A-001 in various inflammatory conditions, including sepsis, rheumatoid arthritis, asthma and ulcerative colitis. These studies provide a large body of safety data for A-002 and A-001, and demonstrate that A-002 was generally well-tolerated among over 1,000 healthy volunteers and patients.

Phase 2b Acute Coronary Syndrome Study—FRANCIS (Fewer Recurrent Acute coronary events with Near-term Cardiovascular Inflammation Suppression)

In July 2008, Anthera initiated a Phase IIb study designed to evaluate the safety and efficacy of A-002 when co-administered with the highest dose (80 mg) of atorvastatin among patients with high levels of inflammation and dyslipidemia. The study was structured as a randomized, double-blind, placebo-controlled trial, and enrolled 625 acute coronary syndrome patients across 35 centers in three countries.

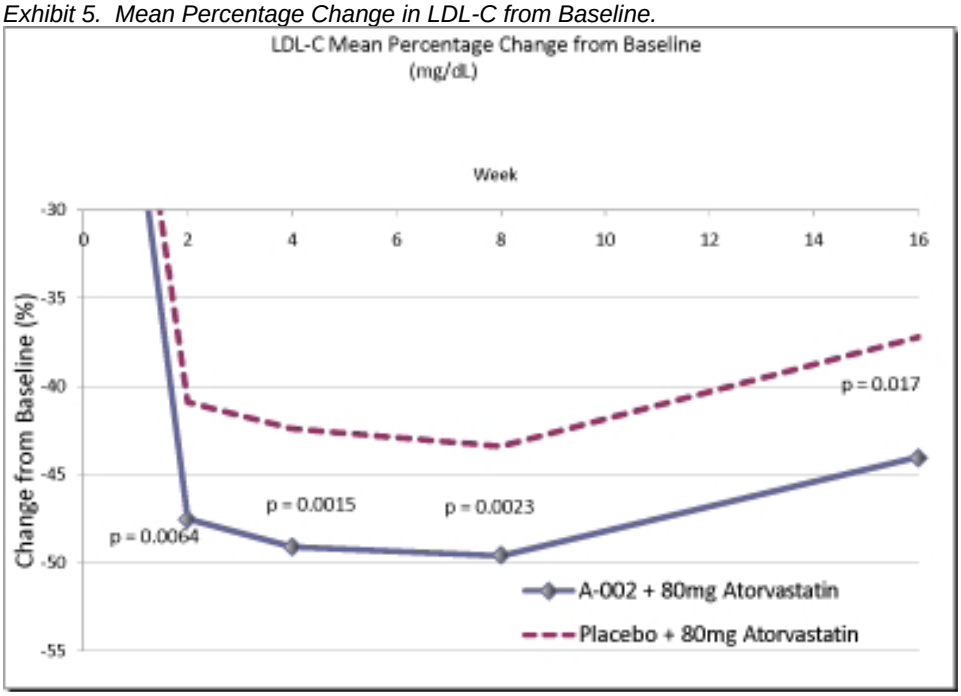
Patients were eligible for enrollment in the trial if they had a diagnosis of either UA, NSTEMI or STEMI. In addition, patients must have had one of the following risk factors: diabetes, body mass index (BMI) ≥ 25 kg/m2, CRP ≥ 2 mg/L (NSTEMI/STEMI) or CRP ≥ 3 mg/L (UA); as well as the presence of three pre-defined characteristics of metabolic syndrome. Finally, subjects could only be enrolled within 96 hours of hospital admission for the index event, or, if already hospitalized, within 96 hours of index event diagnosis.

Patients were randomized to a minimum of 24 weeks of treatment with either: (1) 500 mg once-daily A-002; or (2) placebo; both in combination with 80 mg atorvastatin and physician-directed standard of care.

The primary efficacy endpoint focused on the change in LDL-C after 500 patients completed eight weeks of therapy. LDL-C is the most widely recognized surrogate for predicting cardiovascular risk and percentage reductions in LDL-C have been highly correlated with reductions in future cardiovascular risk. Secondary endpoints included changes in established markers of inflammation (sPLA2, CRP and IL-6) and the occurrence of secondary MACE (all-cause mortality, non-fatal myocardial infarction, documented UA requiring urgent hospitalization, revascularization occurring ≥ 60 days post the index event or non-fatal stroke).

As shown in Exhibit 5 below, results of the primary endpoint demonstrated a statistically significant incremental LDL-C reduction of 5.7% (p = 0.0023) in A-002 treated patients versus those treated with 80 mg atorvastatin alone after eight weeks of therapy. Indeed, a statistically significant difference was observed in LDL-C reduction from baseline as early as two weeks after treatment, and this treatment effect was maintained throughout the observation period.

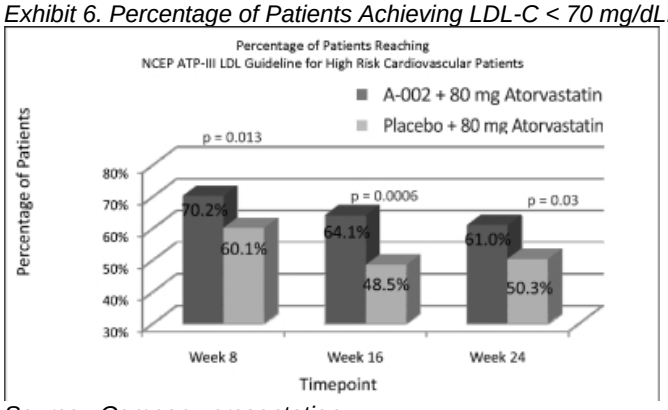
Exhibit 5. Mean Percentage Change in LDL-C from Baseline.



Source: Company presentation

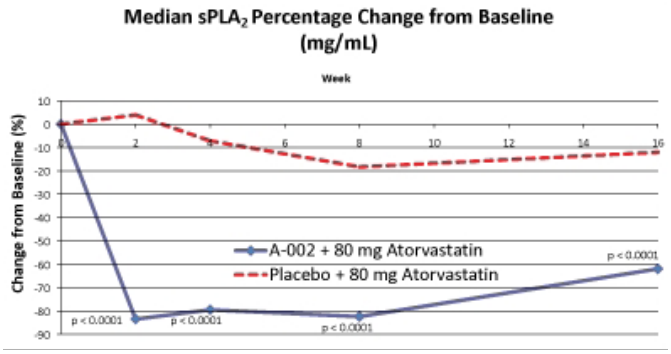
As shown in Exhibit 6, treatment with A-002 along with atorvastatin also resulted in significantly more subjects with LDL-C levels less than 70 mg/dL (in accordance with the optional target for very high risk patients as per NCEP ATP III Guidelines) than those on placebo (atorvastatin alone) at eight, 16 and 24 weeks of treatment.

Exhibit 6. Percentage of Patients Achieving LDL-C < 70 mg/dL.



Source: Company presentation

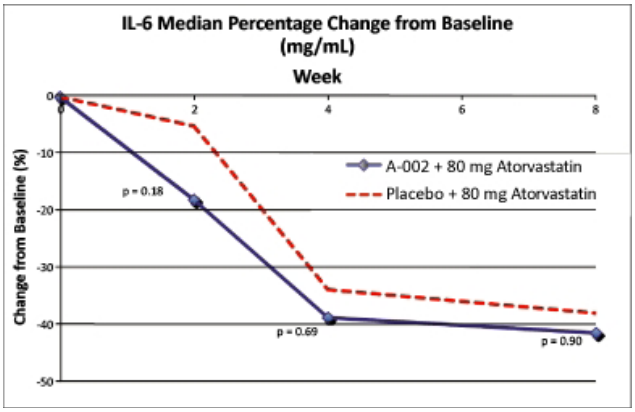
Exhibit 7. Median Percentage Change in sPLA2 Concentration from Baseline



Source: Company presentation

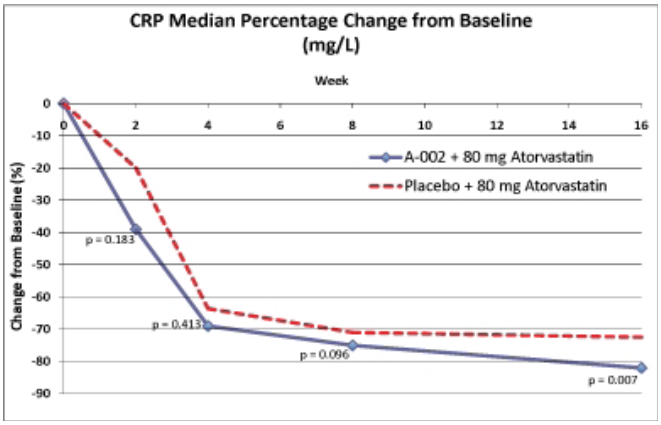
Regarding secondary endpoints, sPLA2 concentration was statistically significantly reduced from the earliest time point of two weeks through the 16-week time point ($p < 0.0001$) as compared to high-dose statin (80 mg atorvastatin) therapy alone. Moreover, the percent decrease in IL-6 in patients on A-002 at week two was more than three times the reduction in IL-6 in patients on placebo (-18% versus -5.1%, p = 0.18). Likewise, CRP reductions were consistently greater among the treatment group than among placebo.

Exhibit 8. Median Percentage Change in IL-6 Concentration from Baseline



Source: Company presentation

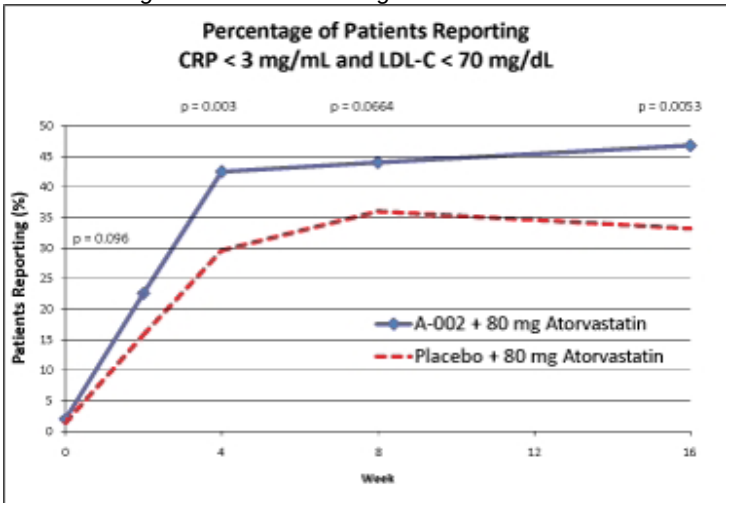
Exhibit 9. Median Percentage Change in CRP Concentration from Baseline.



Source: Company presentation

Finally, Anthera examined the proportion of patients in the clinical study that were able to achieve both LDL-C levels less than 70 mg/dL and CRP levels below 3 mg/L. As shown in Exhibit 10 below, more patients treated with A-002 and 80 mg atorvastatin achieved these dual goals than those treated with placebo and 80 mg atorvastatin alone at all time points in the clinical study with statistically significantly greater percentages of patients achieving these levels at week four and week 16 ($p = 0.003$ and $p = 0.005$).

Exhibit 10. Percentage of Patients Achieving Combined Targets of CRP < 3.0 mg/L and LDL-C < 70 mg/dL



Source: Company presentation

Anthera also conducted an exploratory analysis of MACE. At 16 weeks, there were 13 (4.2%) MACE events in the A-002 treated group as compared to 19 (6.1%) in the placebo group. Meanwhile, at the completion of the clinical study, all patients had received at least six months of therapy and there were 23 (7.4%) MACE in the A-002 treated group as compared to 24 (7.7%) MACE in the placebo group.

Overall, A-002 was generally well-tolerated and no imbalance was seen in dropouts due to drug effects. At week four and week eight, occasional mild and transient elevations in liver enzymes, defined as elevations three times the upper limit of normal, were seen among more patients taking A-002, but the frequency and magnitude of the elevations were not meaningfully different between the active and control groups at the end of the clinical study. The frequency of the elevations was also similar to that reported for atorvastatin and other currently approved lipid-lowering agents. Finally, there were no effects on blood pressure or the QT interval, an electro-cardiographic safety endpoint.

Phase 2 Stable Coronary Artery Disease Study—PLASMA (Phospholipase Levels and Serological Markers of Atherosclerosis): A-002 Twice-Daily Versus Placebo

Anthera's Phase 2 PLASMA study was designed to confirm the safety of A-002, as well as its effect on sPLA 2 concentration, other inflammatory biomarkers, and lipids. The study was structured as a randomized, double-blind, placebo-controlled trial evaluating four

doses of A-002 (50 mg, 100 mg, 250 mg and 500 mg) administered twice-daily versus placebo among 396 patients with stable CAD from 38 centers in two countries. The clinical study enrolled patients more than 12 weeks after a myocardial infarction or six weeks after an episode of unstable angina. Following randomization, patients were treated for eight weeks and safety and efficacy evaluations were conducted at weeks two, four and eight. The primary endpoint the change in sPLA2 concentration from baseline to week eight, while secondary endpoints included change in lipids (including LDL-C), lipoprotein subclasses and certain inflammatory biomarkers, as measured from baseline to each of weeks two, four and eight.

Of note, physician-directed standard-of-care therapies were permitted during the clinical study, and 259 patients were treated with background statin therapy.

The PLASMA results were selected for a late-breaking presentation at the American Cardiology Conference, and also published in the Lancet journal in February 2009. In brief, treatment with A-002 led to statistically significant reductions in sPLA2, LDL-C and various plaque-building and pro-inflammatory forms of LDL-C. In particular, among patients receiving A-002:

- There were incremental reductions in CRP versus placebo (-55.6% versus -24.8%, p = 0.47) from baseline to eight weeks.
- Median sPLA2 concentration decreased by 86.7% from baseline to week eight, as compared to 4.8% in the placebo group (p < 0.0001).
- Median sPLA2 concentration decreased in a dose-dependent manner
- At week eight, LDL-C was reduced by 9.7% versus placebo (p = 0.0035). In a subgroup of patients taking statins with LDL-C > 70 mg/dL, LDL-C was reduced by 12.0% (p = 0.0065) versus placebo at the eight week time point. Notably, the reductions in LDL-C appear to be driven primarily by a shift in the distribution of LDL-C particles with fewer pro-atherogenic, pro-inflammatory small LDL-C particles present in the circulation.
- Statistically significant reductions from baseline to week eight were seen in total cholesterol and non-HDL cholesterol.

A-002 was generally well-tolerated. Adverse effects were reported to be mild or moderate with no imbalance in the A-002 groups as compared to placebo. The most commonly reported adverse effects seen in the A-002 groups were headache (6.4%) and nausea (5.4%). Moreover, there were also mild and transient elevations of liver function tests in patients taking A-002.

Phase 2 Stable Coronary Artery Disease Study—PLASMA-2 (Phospholipase Levels and Serological Markers of Atherosclerosis -2): Once-Daily A-002 versus Placebo

Based on data the first PLASMA study, Anthera initiated a second Phase 2 clinical study (PLASMA-2) to evaluate the effect of once-daily A-002 treatment on inflammatory and lipid biomarkers. This second study was structured as an eight-week, randomized, double-blind, placebo-controlled Phase 2 trial evaluating two doses of A-002 versus placebo amongst 138 patients with stable CAD.

The study was conducted in the United States at 13 clinical sites, and physician-directed standard of care therapies were permitted: 123 patients (89.1%) were on background statin therapy.

The primary endpoint was change in sPLA2 concentration (at week eight), while secondary endpoints included measurements of lipids including LDL-C and certain other inflammatory biomarkers (at each of weeks two, four and eight).

Patients on A-002 demonstrated a 77.8% reduction in sPLA2 concentration as compared to an increase of 8.3% in placebo treated patients (p < 0.0001). Moreover, pharmacokinetic data suggested that once-daily dosing with A-002 is sufficient to achieve over 90% inhibition of sPLA2 mass and activity over a 24-hour period. Moreover, LDL-C was decreased by 8.3% compared to 0.7% in placebo (p = 0.014). However, due to the study's small size and the low baseline of inflammation present, no meaningful changes with CRP could be detected between the active and control groups.

A-002 was generally well-tolerated and there was no imbalance of adverse events among the A-002 groups and placebo. The most common effects seen in the A-002 groups were diarrhea (6.7%), nausea (5.6%), any increase in alanine aminotransferase (5.6%), and any increase in aspartate aminotransferase (5.6%).

Exhibit 11. Placebo-corrected Percent Decrease from Baseline to Week Eight in Biomarkers

	sPLA2	LDL Cholesterol	Total Cholesterol	Non-HDL Cholesterol	Oxidized LDL-C
PLASMA (All doses A-002)	81.9% (p < 0.0001)	9.7% (p = 0.0035)	4.9% (p = 0.0069)	7.2% (p = 0.0009)	5.4% (p = 0.0065)
PLASMA-2 (500 mg A-002) (Dose selected for Phase III)	86.1% (p < 0.0001)	13.9% (p = 0.0007)	9.2% (p = 0.0006)	14.2% (p = 0.0001)	7.3% (pNS)

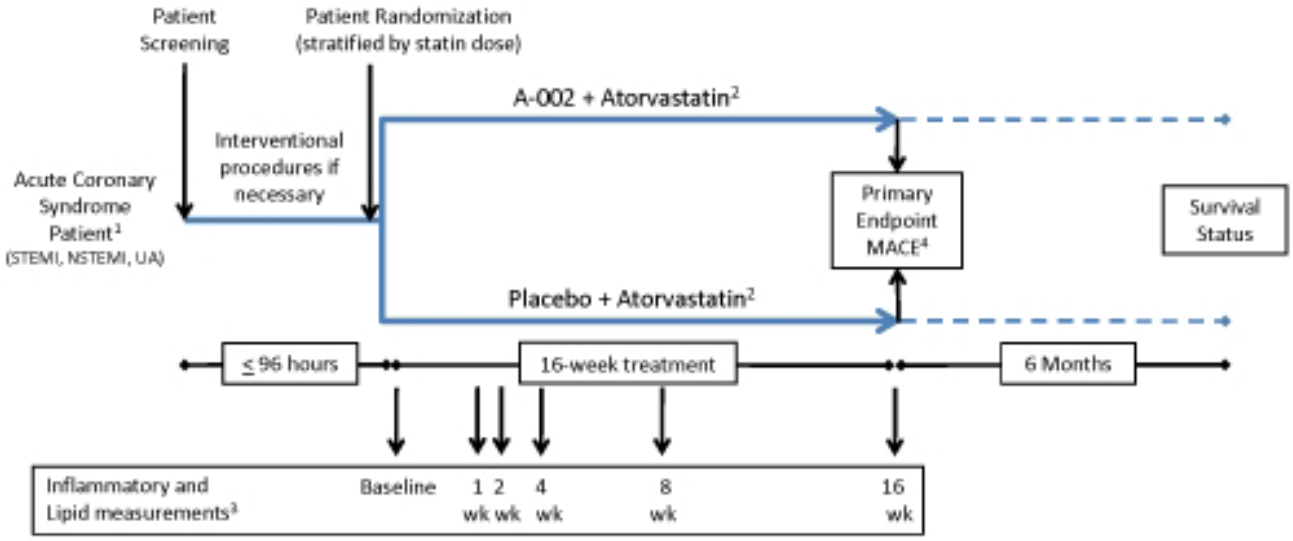
Source: Anthera Pharmaceuticals.

Pivotal VISTA-16 Study—Acute Coronary Syndrome

Anthera recently launched the Phase III VISTA-16 study to evaluate A-002 in combination with statin therapy for the short-term (16-week) treatment of acute coronary syndrome. The VISTA-16 trial began enrolling patients in June 2010, after Anthera received an SPA agreement from the FDA in February 2010. This SPA is designed to expedite the approval process by having the FDA agree to consider the trial design, with its shorter durational endpoint, sufficient for approval should the trial generate a positive outcome.

VISTA-16 is structured as a multinational, randomized, double-blind, placebo-controlled Phase III study. Patients will be randomized at entry to receive either 500 mg once-daily A-002 or placebo in addition to any dose of atorvastatin. While the study is planned to enroll of up to 6,500 patients with acute coronary syndrome, in up to 15 countries and at up to 500 centers, enrollment may be stopped anytime after a minimum of 395 primary endpoints have occurred. Alternatively, Anthera may increase sample size if the adjudicated endpoint events occur at a lower rate than the company expects.

Exhibit 12. VISTA-16 Study Design.



1 Patients will receive physician directed interventional and therapeutic standard of care throughout the study
2 Any dose of atorvastatin
3 There will be a DSMB review of safety and selected biomarkers after a minimum of 1,000 patients have completed 16 weeks of treatment.
4 As per FDA Guidance, MACE is defined as Cardiovascular Death, Non-Fatal Myocardial Infarction, Non-Fatal Stroke, and UA requiring urgent hospitalization

Source: Company presentation

The study is recruiting a population of high-risk cardiovascular patients with acute coronary syndrome similar to those enrolled in the FRANCIS study. As in FRANCIS, randomization must occur within 96 hours of hospitalization for the acute coronary syndrome event, or if already hospitalized, within 96 hours of event diagnosis. Patient blood chemistry will be evaluated at baseline, 24 hours, 48 hours and weeks one, two, four, eight and 16. Moreover, randomization will be stratified by the presence or absence of lipid-lowering therapy prior to the index event as well as the type of acute coronary syndrome event, such as UA, NSTEMI or STEMI. Finally, the number of subjects who undergo percutaneous coronary intervention following the index event and prior to randomization will be limited to no more than 40% of the total patient population.

During the study, the dose of atorvastatin may be adjusted after eight weeks based on the subjects' LDL-C measurement in accordance with local country treatment guidelines.

The primary endpoint will be to determine whether 16 weeks of once-daily treatment with A-002 plus atorvastatin is superior to placebo plus atorvastatin in the time to the first occurrence of the combined endpoint of cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, or documented UA with objective evidence of ischemia requiring hospitalization.

Meanwhile, a secondary endpoint will examine whether A-002 plus atorvastatin is superior to placebo plus atorvastatin in the time to the first occurrence of the combined endpoint of all cause mortality, non-fatal myocardial infarction, non-fatal stroke, or documented UA with objective evidence of ischemia requiring hospitalization. Moreover, a comparison between treatment groups will also be made for each component of the primary efficacy endpoint and, the time to multiple occurrences of any non-fatal component of the composite primary endpoint will also be explored. Finally, the biomarkers CRP, IL-6, LDL-C and sPLA 2, will also be evaluated at each time point of the clinical study.

A Data Safety Monitoring Board (DSMB) will evaluate the performance of VISTA-16 over time to ensure patient safety and to review certain blinded laboratory data. In particular, after a minimum of 1,000 patients have completed 16 weeks of treatment, the DSMB will conduct a biomarker futility analysis to ensure patient levels of inflammation, as measured by sPLA2, CRP and IL-6, and lipid profiles, as measured by LDL-C, have met pre-specified reductions from baseline at various time-points. Anthera expects to report the results of this interim analysis in late Q1:11. Finally, we note that the trial is powered at 80% (at $p<0.05$) to detect a treatment effect of 25% (we understand the trial is also powered to detect a treatment effect as low as 17%), and assumes the placebo arm will have an event rate of 8.5% at 16 weeks.

We expect the VISTA-16 trial to complete enrollment in H2:11 and report final data in Q1:12 leading to potential approval in Q2:13.

Commercial Expectations

Upon clinical trial success and ultimate regulatory approval, we anticipate commercial launch of A-002 in the United States in Q3:2013 and in Europe in Q3:2014. We also assume pricing of roughly $2,100, as well as peak penetration of roughly 15%, from a total US population of 1.6 million patients. We also assume that Anthera will license A-002 for a 17.5% royalty.

Based on these estimates, our royalty predictions for A-002 are below:

A-002 in ACS - Acute								
	FY:2010	FY:2011	FY:2012	FY:2013	FY:2014	FY:2015	FY:2016	FY:2017
U.S Patients	1,630,251	1,671,478	1,713,747	1,757,085	1,801,519	1,847,076	1,893,786	1,941,677
Treated	0	0	0	15,364	67,105	143,424	220,367	266,684
Penetration	0.00%	0.00%	0.00%	0.87%	3.72%	7.76%	11.64%	13.73%
Sales ($K)	0	0	0	34,568	150,985	322,704	495,825	600,039
ANTH Royalty ($K)	$0	$0	$0	$6,049	$26,422	$56,473	$86,769	$105,007
E.U. Patients	1,935,923	1,984,880	2,035,074	2,086,538	2,139,304	2,193,403	2,248,871	2,305,742
Treated	0	0	0	0	18,706	81,702	174,871	268,324
Penetration	0.00%	0.00%	0.00%	0.00%	1.99%	5.12%	9.59%	12.93%
Sales ($K)	0	0	0	0	37,411	163,404	349,743	536,648
ANTH Royalty ($K)	$0	$0	$0	$0	$6,547	$28,596	$61,205	$93,913
Total Royalty ($K)	$0	$0	$0	$6,049	$32,969	$85,069	$147,974	$198,920

Source: Wedbush Securities research

Competitive Landscape

Anthera's A-002 faces competition from a number of drugs in development (Exhibit 13). Although there are no sPLA2 inhibitors currently on the market, a similar anti-inflammatory approach to ACS is being studied in a number of later-stage compounds. In our view, however, the market for cardiovascular drugs is sufficiently large to allow for several products to compete successfully. Moreover, we also note, as elaborated below, that A-002 offers theoretical advantages over its competition.

The farthest advanced of these, darapladib is a small molecule inhibitor of Lp-PLA2 and, being developed by GlaxoSmithKline, using technology from Human Genome Sciences. Lp-PLA2 is member of the same enzyme family as sPLA2, and differs from sPLA2 in that Lp-PLA2 mostly binds to LDL-C and HDL while sPLA2 does not.

A Phase II study of darapladib showed dose-dependent inhibition of Lp-PLA2 in patients receiving the cholesterol lowering drug atorvastatin. Although darapladib lingered in early stages of development for several years, GSK moved the compound into Phase III in December 2008 with the launch of the STABILITY trial, involving 15,500 patients in 39 countries with chronic coronary heart disease. This double-blind trial completed enrollment in Q4:09, and will evaluate darapladib vs. placebo (added to standard of care including statin, aspirin, and blood pressure medications) on a primary endpoint of first occurrence of major adverse cardiovascular events (MACE), including death, heart attack, and stroke.

GSK also initiated a second Phase III trial of darapladib in December 2009 called SOLID-TIMI 52. The study will enroll 11,500 patients from 40 countries with ACS. As with the STABILITY trial, SOLID-TIMI 52 has a primary endpoint of MACE. The study will be stopped when approximately 1,500 reports of MACE occur, and we currently expect data from this trial in 2013.

Other than darapladib, we also note two later stage compounds in development for treating ACS through anti-inflammatory means. First, VIA-2291 from Via Pharmaceuticals is a 5-lipoxygenase inhibitor is in Phase II trials for ACS and other cardiovascular indications. Via is seeking to find a partner to take the compound into Phase III trials but the likelihood of this appears low.

Second, Eisai is developing E-5555, a PAR-1 antagonist that modulates thrombin-platelet-endothelial interactions. Eisai completed a Phase II safety and tolerability study in patients with ACS in August 2009, and we expect the company to update its plans regarding moving the drug into Phase III during 2010.

Exhibit 13. Competitive Landscape for Acute Coronary Syndromes.

Drug Candidate	Company	Stage	Indications	Comments
Darapladib	GlaxoSmithKline / Human Genome Sciences	Phase III	Acute coronary syndrome and chronic heart disease	• Lp-PLA2 inhibitor •15,500 patient study in CHD (STABILITY). •11,500 patient study in ACS (SOLID-TIMI 52). Data expected in 2013.
VIA-2291	Via Pharmaceuticals	Phase II	Acute coronary syndrome or atherosclerosis	•5-lipoxygenase inhibitor •Seeking partnership to initiate Phase III trials
E-5555	Eisai	Phase II	Acute coronary syndrome or atherosclerosis	•PAR-1 inhibitor •600 patient study completed in August 2009 • Evaluating biomarkers and events

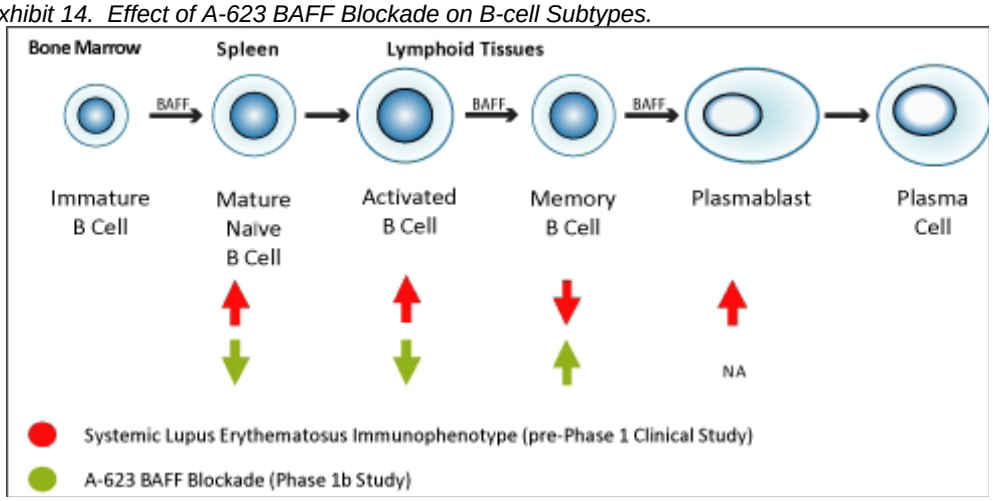
Sources: Company reports, Wedbush Securities research

Additionally, A-002 may also compete with approved statins and other lipid-lowering drugs that are often used in combination. While the anti-inflammatory effect of A-002 is thought to work synergistically with statins, it is possible that the native anti-inflammatory effects of statins may be viewed as competitive to A-002. Finally, lipid-lowering drugs such as Vytorin, Tricor, and Zetia are often used in combination with statins to lower LDL-C levels. For ACS patients who are put on a LDL-C lowering regimen, these drugs may be viewed as competitive with A-002. That said, given the relatively attenuated effect that these readily available therapies have on LDL-C and particularly CRP, we believe that their use alone will prove inferior to therapy that also includes an sPLA2 inhibitor such as A-002.

A-623 – Targeting BLyS in Lupus

A-623 is a selective peptibody antagonist of the BLyS cytokine, also known as B-cell activating factor, or BAFF. A peptibody is a novel fusion protein that is distinct from an antibody. Meanwhile, BLyS is a tumor necrosis family member is critical to the development, maintenance and survival of a type of white blood cell (B-cells). BLyS is primarily expressed by other types of white blood cells (macrophages, monocytes and dendritic cells) and interacts with three different receptors on B-cells including BAFF-R, MCMA and TACI.

Exhibit 14. Effect of A-623 BAFF Blockade on B-cell Subtypes.



Source: Company presentation

BLyS has been associated with a wide range of B-cell mediated autoimmune diseases, including systemic lupus erythematosus (SLE), where Anthera is currently targeting A-623.

Systemic Lupus Erythematosus

SLE is a chronic autoimmune disorder characterized by inflammation, swelling, pain and tissue damage. Although inflammation typically targets connective tissues, SLE can affect any part of the body and patients can experience a broad range of symptoms. For example, inflammation of the brain can cause seizures while inflammation of the heart can lead to heart failure or sudden death. Meanwhile, inflammation of the lungs, joints and skin causes shortness of breath, swelling and rashes, respectively. Finally, inflammation of the kidneys, termed Lupus Nephritis, can lead to kidney damage requiring dialysis or transplantation.

The course of SLE tends to be unpredictable, with periods of illness (flares), alternating between periods of remission. According to the Lupus Foundation, approximately 1.5 million people in the United States and five million worldwide suffer from SLE. Although lupus may affect people of either gender, women are 10 times more likely to suffer from the disease than men.

The cause of SLE is not completely understood. However, B-cell activation and autoantibody production are known to be central to the process and evidence has emerged that over-expression of BLyS plays an important role. For example, in preclinical studies, transgenic mice created to over-express BLyS begin to exhibit symptoms similar to SLE. Meanwhile, treatment of these same mice with BLyS antagonists appears to ameliorate the disease.

Clinical Development

Prior to Anthera's in-licensing of A-623, Amgen completed two Phase I clinical studies of A-623 in lupus patients. These studies were designed to evaluate the safety and pharmacokinetics of single/multiple doses of intravenous and subcutaneous formulations of A-623.

Completed Phase Ia Study

Anthera's Phase Ia trial was structured as a randomized, placebo-controlled, dose-escalation study evaluating A-623 as a single intravenous (1, 3 and 6 mg/kg) or subcutaneous therapy (0.1, 0.3, 1 and 3 mg/kg) among 54 patients with SLE. The primary endpoint was safety and tolerability, while secondary endpoints were designed to assess the plasma pharmacokinetic profile and immunogenicity of A-623.

This study suggested that the safety and tolerability of single doses of A-623 was comparable to placebo. Both intravenous and subcutaneous doses exhibited linear pharmacokinetics, and no neutralization antibodies were seen. No deaths were reported, and

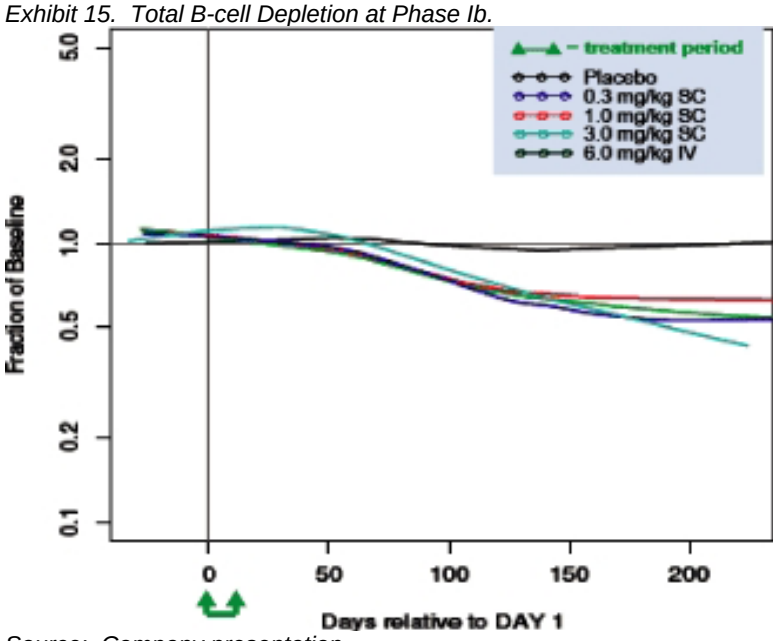
there were comparable adverse events between the A-623 and placebo groups with the most commonly reported adverse events being nausea (15%), headache (10%), upper respiratory tract infection (10%) and diarrhea (8%).

Completed Phase Ib Study

Next, A-623 was evaluated in a Phase Ib clinical trial. This trial was structured as randomized, placebo-controlled, multi-dose trial, involving A-623 as an intravenous (6 mg/kg) or subcutaneous therapy (0.3, 1 and 3 mg/kg) one weekly for four weeks among 61 patients with SLE.

The primary endpoint was safety and tolerability of multiple dose administrations of A-623, while secondary endpoints were designed to assess pharmacokinetics and immunogenicity after multiple doses.

Exhibit 15. Total B-cell Depletion at Phase Ib.



Source: Company presentation

Multiple doses of A-623 given either intravenously or subcutaneously exhibited dose-proportional pharmacokinetics and results demonstrated a dose-dependent decrease in total B-cells as early as 15 days of treatment. Total B-cell reduction (up to approximately 60-70% of baseline) reached its nadir after about 160 days of therapy, and by six months after treatment, the B-cell populations had returned to baseline levels.

Results also demonstrated that A-623 selectively modulates certain B-cell subsets and induced trends toward normal that are consistent with findings in the pre-Phase 1 clinical study.

Finally, the tolerability of multiple doses of intravenous or subcutaneous A-623 was generally comparable to placebo. No deaths reported among either the A-623 or placebo groups. Moreover, few neutralization antibodies were seen, and all resolved in subsequent visits.

Phase IIb PEARL-SC Clinical Study in Patients with SLE

Following positive results in the Phase Ia and Ib clinical studies described above, Anthera initiated a Phase IIb study of A-623, entitled PEARL-SC, in July 2010.

PEARL-SC is a randomized, double-blind, placebo-controlled study scheduled to enroll 600 patients (up to 60 centers worldwide) with serologically active SLE. Patients will be randomized to one of three treatment groups of subcutaneous A-623 (high-dose weekly, low-dose weekly or high-dose monthly) or placebo, with all patients being additionally treated with physician-directed standard of care for at least four months, followed by a two-month safety follow-up.

The primary endpoint is the clinical improvement in the SLE responder index at 24 weeks. The SLE responder index is a composite of changes in clinical disease severity as measured by disease activity indices. Secondary endpoints include safety, other clinical efficacy measures, changes in B-cells and immunoglobulins, time to first flare and flare rates. The primary endpoint is the same used in the pivotal Benlysta studies and would likely also be used for future Phase III trials of A-623. Finally, we note that the trial has a power of 85% (at $p<0.05$), and assumes a 14% treatment effect of A-623 over placebo.

We expect the PEARL-SC trial to complete enrollment in H1:2011 and report final data in Q1:2012. The company will also conduct an interim analysis to measure B-cell reduction, and we expect this data to be released in Q2:2011.

Commercial Expectations

We anticipate commercial launch of A-623 in the United States in Q4:2015 and Europe in Q4:2016. We also assume pricing of roughly $20,000, as well as peak penetration of roughly 10%, from a total US population of 250,000 patients. We also assume that Anthera will license A-623 for a 20% royalty.

Based on these estimates, our royalty predictions for A-623 are below:

A-623 in SLE								
	FY:2010	FY:2011	FY:2012	FY:2013	FY:2014	FY:2015	FY:2016	FY:2017
U.S Patients	250,000	260,194	265,446	270,804	276,270	281,846	287,535	293,339
Treated	0	0	0	0	0	218	4,796	11,600
Penetration	0.000	0.000	0.000	0.000	0.000	0.001	0.017	0.040
Sales ($K)	0	0	0	0	0	4,355	95,929	231,992
ANTH Royalty ($K)	$0	$0	$0	$0	$0	$871	$19,186	$46,398
E.U. Patients	366,096	357,065	364,272	371,624	379,125	386,778	394,584	402,549
Treated	0	0	0	0	0	0	311	6,833
Penetration	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.08%	1.70%
Sales ($K)	0	0	0	0	0	0	4,665	102,494
ANTH Royalty ($K)	$0	$0	$0	$0	$0	$0	$933	$20,499
Total Royalty ($K)	$0	$0	$0	$0	$0	$871	$20,119	$66,897

Source: Wedbush Securities research.

Competitive Landscape

No new therapies have been approved for SLE in the last 50 years, although we expect the FDA to approve HGSI's Benlysta later this year (PDUFA 12/09/2010). Meanwhile, traditional therapies, such as non-steroidal anti-inflammatory drugs, corticosteroids and immunosuppressants lack substantial efficacy, and are associated with significant adverse events and broad immune suppression.

More recently, newer biological agents that target BLyS are being evaluated for the treatment of SLE and Lupus Nephritis. While Benlysta proved successful, as described below, many of these drugs have been fraught with problems resulting in studies being halted or terminated.

Exhibit 16. Competitive Landscape for SLE.

Drug Candidate	Company	Stage	Indications	Comments
Benlysta	Human Genome Sciences	BLA (accelerated approval)	SLE	• Monoclonal antibody against BLyS • Positive results reported in two Phase 3 clinical studies • Approval expected Dec. 2010
Atacicept	Merck KGaA	Phase III	SLE	• Fusion protein against BLyS and APRIL • Phase III clinical study in LN stopped due to safety issues • Data from Phase II/III study in SLE expected in 2011
LY2127399	Eli Lilly	Phase III	SLE	• Monoclonal antibody against BLyS • Phase III trial initiation expected Q4:2010
Ocrelizumab	Roche/Biogen Idec	Phase III	Lupus Nephritis	• Humanized CD-20 monoclonal antibody • Phase 3 clinical study in lupus halted due to infection (restart unlikely)
Epratuzumab	UCB/ Immunomedics	Phase IIb	SLE	• Humanized CD-22 monoclonal antibody •Phase III initiation expected Q4:10
Lupuzor	Cephalon/ ImmuPharma	Phase IIb	SLE	• Modulates CD4 T cells • Phase III initiation expected FY:11

Sources: Wedbush Securities research.

Benlysta is a human monoclonal antibody to BLyS that was evaluated in two successful Phase III studies: BLISS-76 (treatment duration of 76 weeks; conducted in the U.S.) and BLISS-52 (52 week treatment period; conducted outside of the U.S. and Europe). HGSI presented positive data from both studies in 2009 and we expect Benlysta to be approved later this year, on the December 9

PDUFA date. While the results of the BLISS-5 2 and BLISS-76 trials are impressive, we see room for improvement from other drugs in development. In sum, while we see Benlysta rapidly dominating the Lupus market following its launch, we believe their will be room for new treatments to show improved and differentiated profiles.

Exhibit 17 Trial Design for Benlysta Phase III Studies

	BLISS-52	BLISS-76
Site locations	Asia Pacific and South America	Europe and North America
Size	N = 867	N = 810
Trial design	Randomized, double-blind, placebo-controlled, multicenter, superiority study	
Treatment arms	1mg/kg or 10mg/kg Benlysta or placebo, IV, monthly (d1, 15, 28 loading)	1mg/kg or 10mg/kg Benlysta or placebo, IV, monthly (d1, 15, 28 loading)
Treatment Duration	52 weeks	76 weeks
Primary endpoint	Composite response rate (SLE responder index) at 12 months	
Secondary endpoint	Response rate at 12 months, SF-36 score, fatigue measures, steroid dose reduction	
Regulatory	PDUFA Date: 12/09/2010	
Data	57.6% response rate for Benlysta 10 mg/kg vs. 43.6% for placebo (p=0.0006)	43.2% response rate for Benlysta 10 mg/kg vs. 33.8% for placebo (p=0.02)
Partnership	GSK and HGSI 50/50 profit share, worldwide	

Source: Company reports, Wedbush Securities estimates.

Finally, as shown in Exhibit 16 above, companies with other products in development that are being tested for potential treatment of SLE, include Merck Serono, whose dual BLyS/APRIL antagonist fusion protein, Atacicept, is in a Phase III clinical study for SLE and may report data in 2011; and Immunomedics and UCB., who recently reported favorable results for their CD-22 antagonist humanized antibody, epratuzumab, which completed a Phase 2b clinical study in SLE.

However, we believe that A-623 may prove superior to each of these agents. In particular, we note A-623's:

- Demonstrated dosing flexibility with both subcutaneous and intravenous delivery;
- Selective modulation and reduction of relevant B-cell types in SLE patients;
- Ability to bind to both membrane-bound and soluble BLyS; and
- Smaller size as compared to a full antibody, which may confer differentiating pharmacokinetic and pharmacodynamic characteristics.

We hope to see evidence of this superiority in the ongoing Phase IIB trial, which would clear the way for A-623 to enter a large Phase III program shortly thereafter.

A-001 for the Prevention of Acute Chest Syndrome Associated with Sickle Cell Disease

Anthera's next product candidate, varespladib sodium, A-001, is an intravenously administered, potent and broad-spectrum inhibitor of sPLA2, including forms IIa, V and X. A-001 is currently being evaluated by Anthera in a Phase II trial for the prevention of acute chest syndrome associated with sickle cell disease in at-risk patients. Sickle cell disease is a genetic disorder which leads to the structural alteration, or "sickling," of otherwise healthy red blood cells. As a result, patients with sickle cell disease experience periods of intense pain known as vaso-occlusive crisis (VOC) as structurally-altered red blood cells bind together and occlude small blood vessels that supply blood and nutrients to vital tissue and bone. The most common cause of death in patients with sickle cell disease is acute chest syndrome, which occurs with VOC of the pulmonary vessels.

Substantial scientific evidence implicates sPLA2 activity in the development of acute chest syndrome as well as other forms of acute lung injury. For instance, sPLA2 levels are dramatically elevated in sickle cell patients during an episode of VOC as well as within 24 to 48 hours of the onset of acute chest syndrome. Moreover, during VOC, microscopic droplets of fat from the bone marrow can break free and become lodged in the lung. These particles are substrates for sPLA2 enzymes and are thought to provide fuel for an already established inflammatory response, thereby increasing lung injury. Finally, sPLA2 has been shown to degrade human lung surfactant, a component necessary in maintaining appropriate lung function.

Anthera believes that early intervention with A-001 to inhibit sPLA2 activity may offer a novel preventative therapy to improve outcome among sickle cell disease patients presenting with a high risk of acute chest syndrome. In support, the FDA has granted

orphan drug and fast-track designation for A-001 for the prevention of acute chest syndrome associated with sickle cell disease in at-risk patients.

Market Opportunity

Sickle cell disease is a genetically inherited blood disorder which is of lifelong duration and typically diagnosed during early childhood. According to the Sickle Cell Information Center, approximately 1,000 children are born with the disease annually in the United States, and over 70,000 people currently suffer from the disease. Meanwhile, in Europe, over 200,000 people are thought to suffer from the disease, while another 200,000 children are born with sickle cell disease each year in Africa. Life expectancy for those with sickle cell disease is only in the mid-40s.

Sickle cell disease is characterized by an altered form of hemoglobin that results in structural alterations in red blood cells and causes them to assume an abnormal shape, similar to a sickle. These sickled red blood cells have a shortened life-cycle, and also become relatively inflexible, preventing them from easily passing through the body's small blood vessels. At times, obstructions can occur, leading to significant damage in tissue and bone in an event termed-Occlusive Crisis (VOC). During VOC, blockages within the circulation of the long bones can cause microscopic fragments of bone or bone fat to break free and embolize to the lungs, resulting in acute chest syndrome.

Acute chest syndrome exhibits symptoms and characteristics similar to acute lung injury, and represents both the most common cause of death in sickle cell patients, and the second most common cause of hospitalization. There are an estimated 10,000 episodes of acute chest syndrome each year in the United States.

A majority of sickle cell patients experience at least one episode of acute chest syndrome in their lifetimes and repeated episodes can result in progressive lung disease. Acute chest syndrome is most common in the 2-4 year old age group and gradually declines in incidence with age.

There are currently no marketed therapies for acute chest syndrome. The most common therapeutic regimen includes heavy doses of corticosteroids, opiates, blood transfusions and antibiotics. In addition, a chemotherapeutic agent, hydroxyurea, was found to reduce the frequency of VOC as well as the need for blood transfusions in adult patients. However, existing therapies are associated with significant adverse effects while only offering limited patient benefit.

Clinical Development

Anthera's clinical program for A-001 is summarized below.

Phase II Acute Chest Syndrome in Hospitalized Patients with Sickle Cell Disease Study—Investigation of the Modulation of Phospholipase in Acute Chest Syndrome, or IMPACTS.

In January 2007, Anthera initiated a randomized, double-blind, placebo-controlled Phase II clinical study to assess the safety and tolerability of escalating doses of A-001. The clinical study enrolled 30 hospitalized sickle cell disease patients, at risk for acute chest syndrome on the basis of VOC, fever and serum sPLA2 concentration level greater than 50 mg/mL, across approximately 30 sites in the United States. The primary endpoint for the clinical study was safety and tolerability, while secondary endpoints included the absence of acute chest syndrome, suppression of sPLA2, reduced need for blood transfusions and assessment of pharmacokinetics.

The first group of patients was randomized 2:1 to receive either low dose A-001 or placebo as a 48-hour continuous infusion. A pre-specified interim analysis, conducted in February 2009 after the 30th patient completed treatment, indicated that serum levels of A-001 (when dosed at 55 mug/kg/hr) reduced sPLA2 activity levels by more than 80% from baseline within 48 hours. Moreover, the prevention of acute chest syndrome associated with sickle cell disease appeared to be related to the level of sPLA2 activity.

Exhibit 18. Reductions of sPLA-2 activity compared with Incidence of Acute Chest Syndrome.

48-Hour sPLA2 Activity as a Percentage of Baseline	0.0% < 25.0%	≥ 25% < 50%	≥ 50% < 75%	≥ 75%
Number of Subjects	7	7	3	12
Number of Subjects Developing Acute Chest Syndrome (%)	0 (0)	2 (28)	1 (33)	4 (25)

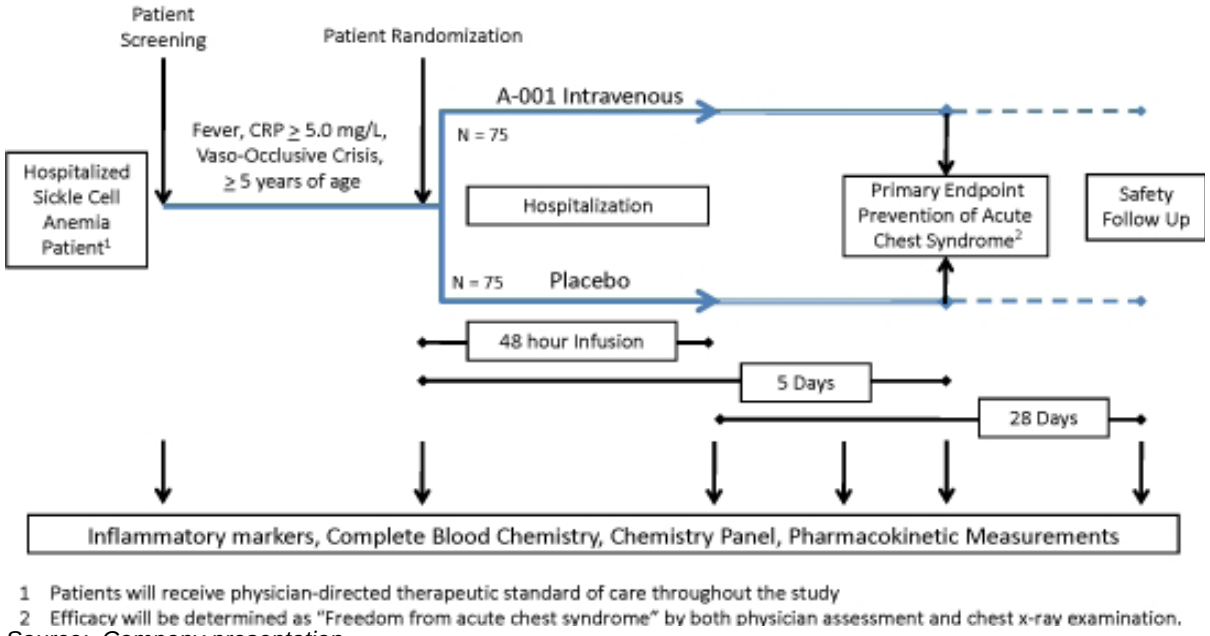
Source: Anthera Pharmaceuticals.

The DSMB also recommended that the clinical study continue based on safety and tolerability, and approved the addition of a higher dose group of 110 mug/kg/hr via continuous infusion during the second half of the clinical study. However, based on the study's relative dramatic interim results and after subsequent discussions with the FDA, the company decided to forgo the second half of this study, and move directly into the Phase IIb/III trial, described below.

Upcoming Phase IIb Study of A-001 in the Prevention of Acute Chest Syndrome

Anthera's planned multinational, randomized, double-blind, placebo controlled Phase IIb study will enroll patients with sickle cell disease who are at an elevated risk of developing acute chest syndrome as a result of fever, vaso-occlusive crisis, and CRP ≥ 5.0 mg/L at the time of hospitalization. Up to 200 patients will be randomized to receive a continuous infusion of A-001 or placebo for 48 hours after randomization and the primary endpoint will be freedom from acute chest syndrome as determined by physician assessment and independent review of chest X-rays.

Exhibit 19. Phase IIb Study in Prevention of Acute Chest Syndrome in Patients with Sickle Cell Disease.



1 Patients will receive physician-directed therapeutic standard of care throughout the study
2 Efficacy will be determined as "Freedom from acute chest syndrome" by both physician assessment and chest x-ray examination.

Source: Company presentation

We expect the study to begin enrolling patients before H1:2011, and note that, based on conversations with the FDA, Anthera believes that the trial may qualify a registration study. If this is the case, we anticipate A-623 could obtain approval as early as Q1:2014. Alternatively, if a separate Phase III is required, we anticipate approval in 2017. For purposes of modeling, we have taken the more conservative approach.

Commercial Expectations

We anticipate commercial launch of A-001 in the United States in 1Q:2017 and in Europe in 1Q:2018. We also assume pricing of roughly $25,000, as well as peak penetration of roughly 20%, from a total US population of 75,000 patients. We also assume that Anthera will license A-001 for a 17.5% royalty.

Based on these estimates, our royalty predictions for A-001 are below:

A-001 in Sickle Cell Crisis								
	FY:2010	FY:2011	FY:2012	FY:2013	FY:2014	FY:2015	FY:2016	FY:2017
U.S Patients	76,387	77,928	79,501	81,106	82,743	84,413	86,117	87,855
Treated	0	0	0	0	0	0	0	4,311
Penetration	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.049
Sales ($K)	0	0	0	0	0	0	0	107,776
ANTH Royalty ($K)	$0	$0	$0	$0	$0	$0	$0	$18,861
E.U. Patients	102,018	104,078	106,178	108,321	110,508	112,738	115,014	117,335
Treated	0	0	0	0	0	0	0	0
Penetration	0%	0%	0%	0%	0%	0%	0%	0%
Sales ($K)	0	0	0	0	0	0	0	0
ANTH Royalty ($K)	$0	$0	$0	$0	$0	$0	$0	$0
Total Royalty ($K)	$0	$0	$0	$0	$0	$0	$0	$18,861

Source: Wedbush Securities research

Competitive Landscape

There are currently no agents approved specifically for the treatment or prophylaxis of acute chest syndrome associated with sickle cell disease. That said, Droxia (hydroxyurea) is approved for the prevention of VOC in sickle cell disease. In addition, there is evidence that blood transfusions may prevent the occurrence of acute chest syndrome associated with sickle cell disease, and a randomized clinical study is underway by the National Heart, Lung and Blood Institute to explore this possibility.

However, as the major cause of mortality in sickle cell disease, acute chest syndrome represents a clear unmet medical need.

Preclinical Portfolio

Anthera is also developing a series of preclinical sPLA2 inhibitors, including the A-003, the company's most advanced preclinical drug candidate. A-003, is chemically distinct from both A-001 and A-002, and has shown both increased potency against target enzymes and higher drug exposure after dosing in preclinical studies. As a result, A-003 which can be formulated for oral or intravenously administered use, may confer beneficial pharmacodynamic effects in patients.

However, in the absence of clinical proof-of-concept data, we have not yet included A-003 or any of the company's other, undisclosed preclinical compounds in our pipeline valuation.

Company Background

Intellectual Property

A-002 and A-001

Anthera holds exclusive worldwide licenses (except Japan) from Eli Lilly and Shionogi & Co., Ltd. to a portfolio of intellectual property directed to A-002 and A-001. This licensed portfolio includes:

- Thirteen granted U.S. patents (with scheduled expiration dates between 2014 and 2021);
- One pending U.S. non-provisional patent application;
- Four European Patent, or EP, patents, each validated in one or more of Austria, Belgium, Denmark, France, Germany, Greece, Ireland, Italy, Liechtenstein, Luxembourg, the Netherlands, Portugal, Spain, Sweden, Switzerland and the United Kingdom;
- Three pending EP patent applications;
- Fourteen non-EP foreign patents in Argentina, Australia, Brazil, Canada, China, Finland, Malaysia, Mexico, the Philippines, South Korea, Taiwan and Turkey; and
- Eight pending non-EP foreign patent applications in Brazil, Canada, China, India, Mexico, South Korea, Taiwan and Thailand.

These patents and pending applications contain claims directed to A-002 and A-001 compositions of matter, as well as to various methods of making and using A-002 and A-001, including methods of treating various inflammatory conditions.

In addition, Anthera also holds several internally developed A-002 and A-001 related patent applications, including:

- Two pending U.S. non-provisional patent applications;
- Four pending U.S. provisional patent applications; and
- One pending Patent Cooperation Treaty, or PCT, patent application.

These patent applications contain claims directed to A-002 and A-001 compositions of matter and methods of treating various cardiovascular indications.

A-003

Anthera holds exclusive worldwide licenses (except Japan) from Eli Lilly and Shionogi & Co., Ltd. to a portfolio of intellectual property directed to A-003. This licensed portfolio includes:

- Two granted U.S. patents (with scheduled expiration dates between 2017 and 2018);
- One pending U.S. non-provisional patent application (also covering A-002 and A-001);

- Four EP patents (one of which also covering A-002 and A-001), each validated in one or more of Albania, Austria, Belgium, Denmark, France, Germany, Greece, Ireland, Italy, Liechtenstein, Lithuania, Luxembourg, the Netherlands, Portugal, Romania, Slovenia, Spain, Sweden, Switzerland and the United Kingdom;
- Two pending EP patent applications (also covering A-002 and A-001);
- Eight non-EP foreign patents (one of which also covers A-002 and A-001) in Argentina, Australia, Canada, China, Mexico, South Korea and Taiwan; and
- Eight pending non-EP foreign patent applications (six of which also cover A-002 and A-001) in Argentina, Brazil, Canada, China, India, Mexico, South Korea, and Taiwan.

These patents and applications contain claims directed to A-003 compositions of matter, as well as to various methods of making and using A-003, including methods of treating various inflammatory conditions.

In addition, Anthera also holds several internally developed A-003 related patent applications, including:

- Two pending U.S. non-provisional patent applications (also covering A-002 and A-001)
- Four pending U.S. provisional patent applications (also covering A-002 and A-001)
- One pending Patent Cooperation Treaty, or PCT, patent application (also covering A-002 and A-001)

These patent applications contain claims directed to A-003 compositions of matter and methods of treating various cardiovascular indications.

New sPLA2 Compounds

Anthera holds exclusive worldwide licenses (except Japan) from Eli Lilly and Shionogi & Co., Ltd. to a portfolio of intellectual property directed to other sPLA2 compounds. This licensed portfolio includes:

- Over 30 granted U.S. patents (with scheduled expiration dates between 2013 and 2024); and
- One granted EP patent.

These patents contain claims directed to various second generation sPLA2 compounds covering composition of matter and use in certain indications.

A-623

Anthera hold exclusive worldwide licenses from Amgen to a portfolio on intellectual property directed to A-623. This licensed portfolio includes:

- One U.S. patent
- One pending U.S. non-provisional patent application
- One EP patent validated in Albania, Austria, Belgium, Cyprus, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Latvia, Lichtenstein, Lithuania, Luxembourg, Monaco, the Netherlands, Portugal, Romania, Slovenia, Spain, Sweden, Switzerland, Turkey and the United Kingdom
- One pending EP patent application
- Eight non-EP foreign patents in Australia, China, Eurasia, New Zealand, Singapore, South Korea and South Africa
- 17 pending non-EP foreign patent applications

Licensing and Partnerships

Anthera licenses technology related to sPLA2 inhibitors from Eli Lilly and Shionogi & Co. and has licenses related to A-623 from Amgen. Please see the table below for a description of the terms for these licenses.

Exhibit 20: Anthera licensing agreements

Drug	Company	ANTH Rights	Terms
A-001, A-002, A-003, preclinical	Eli Lilly and Shionogi & Co.	Worldwide except Japan	• Anthera made upfront payment of $250,000 in cash and $2.3 million in convertible stock. • Anthera is responsible for various clinical, regulatory and sales milestone payments: • A-002 $3.5 million within 12 months of starting A-002 Phase III study, and up to $32 million upon approval and post-approval sales milestones. • A-001 $3 million for certain clinical development milestones, and up to $25 million upon approval and post-approval sales milestones. • A-003 and preclinical compounds $2 million for clinical development milestones, and up to $35.5 million upon approval and post-approval sales milestones.
A-623	Amgen	Worldwide	• Anthera made upfront payment of $6 million in cash • Anthera is responsible for certain clinical and approval milestones, $10 million pre-clinical and up to $23 million post-approval. • Anthera pays tiered royalty payments on net sales, ranging from high single digits to low double digits.

Source: Company reports, Wedbush Securities research.

Recent Financing History

Prior to the company's IPO, Anthera raised approximately $55.4 million through private placements of preferred stock and convertible debt. Anthera went public in March 2010, raising $57.8 million in net proceeds from the IPO and concurrent private placement. Details of Anthera's IPO and subsequent financings are shown in the table below:

Exhibit 21. Recent financing events

Date	Financing event
September 2010	Anthera raised $29.3 million in net proceeds in conjunction with a PIPE offering of 10.5 million units. Each unit consisted of one share of Anthera common stock and one warrant to purchase 0.40 shares of common stock. The warrants have an exercise price of $3.30 per share and expire five years from the date of issuance.
April 2010	As granted per the IPO, the underwriters exercised the over-allotment option to purchase an additional 604,492 shares of common stock at a price of $7.00 per share, resulting in approximately $4 million in net proceeds to Anthera
March 2010	Concurrent with the closing of the IPO, Anthera completed a private placement of 2.6 million shares of common stock to certain of its existing investors at $6.58 per share, resulting in an additional $17.1 million in net proceeds.
March 2010	Anthera completed $36.7 million IPO (net of fees), offering 6 million shares of common stock at $7.00 per share. In addition, Anthera granted the underwriters a 30-day option to purchase up to an additional 90,000 shares to cover over-allotments.
October 2009	Approximately 2.3 million Series A preferred shares were converted into common shares (1:1 basis) by CR Intrinsic Investments. No series A preferred shares remain following the conversion.

Source: Company reports

Sales and Marketing

As an early stage biotechnology company, Anthera does not have a commercial sales force or distribution capabilities. The company plans to wait until after it receives Phase III data before initiating a sales and marketing strategy, which may include in-house programs as well as external partnerships.

If Anthera chooses to develop its own sales strategy, we would expect the company to do so for acute care (e.g., acute coronary syndrome) and orphan indications (e.g., sickle cell disease) that could be served by a small but specialized sales force. Additionally, Anthera could pursue a co-commercialization strategy with a partner in these areas. The company estimates that the US and Western European ACS market could be served by approximately 300 sales reps. We expect Anthera to seek partners to serve Eastern European and Asian markets. We expect Anthera to out-license commercialization rights for larger, chronic conditions to large pharmaceutical companies with the capability to address these markets.

Management

Anthera is led by what we consider to be an experienced Management team with a valuable background in developing pharmaceutical pipelines.

Exhibit 22. Anthera Management

Name	Position	Experience
Paul F. Truex	Chief Executive Officer	Mr. Truex has served as CEO since Anthera's inception in September 2004, and also serves as a member of the board of directors. Prior to founding Anthera, Mr. Truex was CEO of Peninsula Pharmaceuticals which went on to be acquired by Johnson and Johnson in 2005. Prior to working at Peninsula, Mr. Truex was VP of Commercial Development for Vicuron and held various positions at Eli Lilly and Company. Mr. Truex is also a director of Trius Therapeutics and Eiger Biopharmaceuticals. Mr. Truex received and M.B.A. from Indiana University.
Christopher P. Lowe	Chief Financial Officer	Mr. Lowe was appointed CFO in November 2007. Mr. Lowe previously worked as the VP of Finance and then CFO of Asthmatx from September 2005 through November 2007. Mr. Lowe also worked at Peninsula Pharmaceuticals from June 2004 until June 2005, first as Corporate Controller and then as Chief Announcting Officer. Mr. Lowe holds an M.B.A. from Saint Mary's University in Texas. Mr. Lowe is a director of Hansen Medical Corporation.
Colin Hislop, MD	Chief Medical Officer	Dr. Hislop was promoted to CMO in June 2010, having previously served as Anthera's Senior VP of Cardiovascular Products since 2005. Prior to working at Anthera, Dr. Hislop served as VP of clinical development for Peninsula Pharmaceuticals where he oversaw three global development programs in the company's anti-infective portfolio. Prior to Peninsula, Dr. Hislop worked at CV Therapeutics, Proctor & Gamble, and Eli Lily. Dr. Hislop has a B.Sc. from University of Surrey and an M.D., B.S. from the University of London.
Debra Odink, PhD	VP of Pharmaceutical R&D	Dr. Odink has worked as Anthera's VP of Pharmaceutical Research and Development since December 2005. From September 2002 until July 2005, she was the VP of Pharmaceutical Chemistry and Product Development at Peninsula Pharmaceuticals. Dr. Odink received a PhD in Inorganic Chemistry from the University of California at Davis.
Joaquim Trias, PhD	Senior VP of Preclinical Development	Dr. Trias has served as Anthera's Senior Vice President of Preclinical Development since December 2004. From July 1996 until July 2004, Dr. Trias was VP of Drug Discovery Research at Vicuron Pharmaceuticals where he directed internal discovery projects. Dr. Trias holds a PhD in microbiology from the University of Barcelona.
Stephen Lau	VP of Corporate and Business Development	Mr. Lau was appointed VP of Corporate and Business Development in February 2008. From October 2003 until February 2008, Mr. Lau worked in business development at Amgen, where he negotiated licensing deals. Prior to working at Amgen, Mr. Lau was an investment banker at Adams, Harkness & Hill. Mr. Lau received an M.S. in immunology from the University of California at Davis and a Master's degree in health care management from Harvard University.
Ursula Fritsch, Pharm. D	VP Global Regulatory and Compliance	Dr. Fritsch has served as Anthera's VP of Global Regulatory and Compliance since April 2005. Prior to working at Anthera, Dr. Fritsch was the Senior Director of Regulatory Affairs at Peninsula Pharmaceuticals from 2003 to 2005. Dr. Fritsch has also held various regulatory positions at Genentech, Oclassen Pharmaceuticals, and Onyx Pharmaceuticals (as head of regulatory). Dr. Fritsch received a Pharm. D. from Creighton University.

Source: Anthera Pharmaceuticals

Risks to Investment Thesis

Intellectual Property Risk. Although we believe that Anthera holds an adequate intellectual property position, there is some inherent uncertainty in both the interpretation of patent claims and the application of patent law. Moreover, when the company's licensed patents expire, Anthera may be unable to prevent third-parties from copying its products. Furthermore, competitors might challenge the validity or scope of Amarin's patents in court, or simply find ways around them. Finally, third-party patents that are not licensed to Anthera and which could prevent Anthera from commercializing its product candidates, or continue to do so, could be granted in the future or even already exist.

Commercialization Risk. Although Anthera may be able to secure FDA approval of its compounds, the company may face unpredictable challenges in successfully commercializing its drugs. These risks include the presence of a competing drug with a more favorable efficacy and/or safety profile, lower cost, stronger physician preference and greater ease of reimbursement.

Manufacturing Risk. Anthera does not yet posses its own manufacturing capabilities to supply sufficient quantities of its drugs and/or other pharmaceutical preparations in development. Any disruption to the agreements between Anthera and its supplies, or the emergence manufacturing problems at its suppliers could disrupt the company's ability to produce sufficient quantities of drug in a timely manner.

Clinical Risk. Anthera does not yet have a track record of generating positive data in large, pivotal clinical trials. If the results of the company's ongoing Phase III trial is negative, A-002 will likely be unable to seek FDA approval and may be forced to conduct additional studies requiring significant capital and lengthening the timeline for approval.

Regulatory Risk. Anthera has not yet received regulatory approval for its drugs in any jurisdiction, and it may never obtain such approval. The required regulatory process for drug candidates is complex, and requires the collection and production of extensive amounts of data from time-consuming and expensive clinical trials. Moreover, decisions on approval are at the sole discretion of the regulatory agencies, who can act unpredictably at times. Finally, even after drugs are approved for marketing, regulatory agencies retain jurisdiction and the right to remove these drugs from the market if they are viewed to present sufficient danger.

Competition Risk. The indications being targeted by Anthera are also being targeted by several large pharmaceutical companies with superior resources. Anthera may be unable to compete for market share as effectively as these companies, even if Anthera's products have superior clinical profiles.

Financing Risk. Anthera's current cash balance is insufficient to fund operations through profitability and the company will likely require a significant amount of additional cash before it is able to generate revenues from the commercialization of its drugs. Such financing may to be dilutive and may be on terms deemed unfavorable to investors. If the company is unable to raise cash, it may be forced to curtail or even cease operations.

Anthera Pharmaceuticals (NASDAQ: ANTH)

Historical and Projected Income Statement

(In thousands except per share data)

(Fiscal Year Ends on December 31)

Wedbush PacGrow LifeSciences

Duane Nash, MD JD MBA

	2009A	2010					2011E	2012E
	FY:09A	Q1A	Q2A	Q3E	Q4E	FY:10E	FY:11E	FY:12E
Revenues:								
Royalties on Product Sales	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
A-002 in Acute Coronary Syndrome - Acute Therapy	-	-	-	-	-	-	-	-
A-002 in Acute Coronary Syndrome - Chronic Therapy	-	-	-	-	-	-	-	-
A-001 in Acute Chest Syndrome (Sickle Cell Disease)	-	-	-	-	-	-	-	-
A-623 in Systemic Lupus Erythematosus	-	-	-	-	-	-	-	-
Collaboration Revenue	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
Milestones	-		-	-	-	-	-	-
Total Revenues	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**
Operating Expenses								
R&D	8,415	5,242	6,438	8,460	9,358	29,498	47,082	27,063
Sales, General and Administrative	3,425	1,224	1,510	1,631	1,761	6,126	8,571	11,660
Other	-	-	-	-	-	-	-	-
Total Operating Expenses	**11,840**	**6,466**	**7,948**	**10,091**	**11,119**	**35,624**	**55,653**	**38,723**
Operating Income (Loss)	(11,840)	(6,466)	(7,948)	(10,091)	(11,119)	(35,624)	(55,653)	(38,723)
Interest income	24	3	12	67	76	159	238	142
Interest expense	(386)	(4,641)	-	-	-	(4,641)	-	-
Beneficial conversion feature	-	-	-	-	-	-	-	-
Income Before Income Taxes	**(12,203)**	**(11,104)**	**(7,936)**	**(10,023)**	**(11,043)**	**(40,106)**	**(55,415)**	**(38,581)**
Other comprehensive income (loss)								
Provision for Income Taxes (benefit)	-	-	-	-	-	-	-	-
Net Income (Loss)	**$ (12,203)**	**$ (11,104)**	**$ (7,936)**	**$ (10,023)**	**$ (11,043)**	**$ (40,106)**	**$ (55,415)**	**$ (38,581)**
EPS (Basic & Diluted)	(8.06)	(0.83)	(0.36)	(0.43)	(0.33)	(1.74)	(1.64)	(1.11)
Shares Outstanding (Basic)	**1,514**	13,344	22,224	23,474	33,225	**23,067**	**33,850**	**34,850**
Fully Diluted Shares Outstanding (Pro forma)	**10,190**	14,953	23,664	24,914	38,560	**25,523**	**38,810**	**39,210**

Source: Company reports, Wedbush Securities estimates

ANALYST CERTIFICATION

I, Duane Nash, certify that the views expressed in this report accurately reflect my personal opinion and that I have not and will not, directly or indirectly, receive compensation or other payments in connection with my specific recommendations or views contained in this report.

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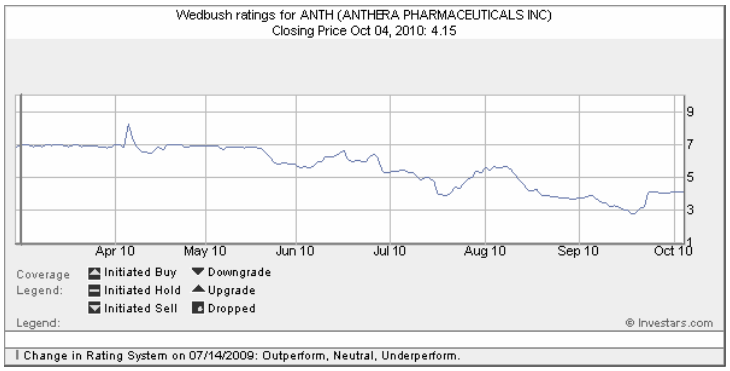

Disclosure information regarding historical ratings and price targets is available at
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OUTPERFORM – Expect the total return of the stock to outperform relative to the median total return of the analyst's (or the analyst's team) coverage universe over the next 6-12 months.

NEUTRAL – Expect the total return of the stock to perform in-line with the median total return of the analyst's (or the analyst's team) coverage universe over the next 6-12 months.

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