COWEN AND COMPANY

Anthera Pharmaceuticals

Outperform (1)

December 2, 2010

Varespladib And A-623 Keys To Success

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Conclusion: Anthera is a uniquely positioned biopharmaceutical company developing several drugs for the treatment of cardiovascular and inflammatory diseases, including varespladib for ACS, A-623 for SLE, and A-001 for sickle cell disease. Varespladib, which is actively enrolling a Phase III ACS trial (VISTA-16), targets an anti-inflammatory pathway. The interim analysis in H1:11 is likely to be a positive catalyst given that study continuation is probable. A-623 began Phase II (PEARL-SC) in Q4:10, but the trial was recently stopped for vial cracking; restart in H1:11 is expected. With much news flow on tap on Anthera's first or potentially best-in-class compounds, we believe the stock will outperform over the next 12-18 months.

- **Varespladib's VISTA-16 Key: Not Without Risk But Market Opportunity Sizable.** We believe VISTA-16 has up to a 40% probability of success given a reasonable scientific rationale and Phase II data that demonstrated robust anti-inflammatory activity and an incremental 5-7pp LDL-C reduction over a statin. The odds of success are capped at 40% given that an anti-inflammatory agent has yet to demonstrate a CV benefit, sPLA2 is a novel target, and VISTA-16 incorporates novel design elements. Should VISTA-16 be successful, we estimate sales of $1.7B+.

- **VISTA-16's Interim Analysis Likely To Be A Positive Catalyst.** VISTA-16's interim biomarker and futility analysis likely will report in H1:11. Should VISTA-16 largely replicate the FRANCIS data, we believe the study will continue. Study continuation would be a positive catalyst for the stock given that it would validate the mechanism as safe and potentially effective.

- **A-623 May Offer Advantages.** A-623, a BlyS peptibody, could have benefits over GSK/HGSI's Benlysta given subQ delivery and the fact that it binds both membranous and soluble BlyS. However, clinical data is necessary to prove the benefit of this differentiation.

ANTH (12/01)	**$5.49**
Mkt cap	**$213.6MM**
Dil shares out	38.9MM
Avg daily vol	20.0K
52-wk range	$2.8-8.6
Dividend	Nil
Dividend yield	Nil
BV/sh	NA
Net cash/sh	NA
Debt/cap	NA
ROE (LTM)	NA
5-yr fwd EPS growth (Norm)	NA
S&P 500	**1206.0**

Revenue $MM

FY Dec	2009 Actual	2010E Prior	2010E Current	2011E Prior	2011E Current	2012E Prior	2012E Current
Q1	0.0	—	0.0	—	0.0	—	—
Q2	0.0	—	0.0	—	0.0	—	—
Q3	0.0	—	0.0	—	0.0	—	—
Q4	0.0	—	0.0	—	0.0	—	—
Year	**0.0**	**—**	**0.0**	**—**	**0.0**	**—**	**80.0**
EV/S	—	—	—	—	—	—	—

EPS $

FY Dec	2009 Actual	2010E Prior	2010E Current	2011E Prior	2011E Current	2012E Prior	2012E Current
Q1	0.00	—	(0.83)A	(0.79)	(0.30)	—	—
Q2	0.00	—	(0.36)A	(0.62)	(0.27)	—	—
Q3	0.00	—	(0.36)A	(0.59)	(0.22)	—	—
Q4	0.00	(0.67)	(0.27)	(0.31)	(0.22)	—	—
Year	**(8.06)**	**(2.31)**	**(1.82)**	**(2.31)**	**(1.01)**	**(2.25)**	**(1.67)**
P/E	—	—	—	—	—	—	—

Please see addendum of this report for important disclosures.

Investment Thesis

Anthera is a uniquely positioned biopharmaceutical company developing several drugs for the treatment of cardiovascular and inflammatory diseases, including acute coronary syndrome (ACS), systemic lupus erythematosus (SLE), and acute chest syndrome in sickle cell disease. Anthera's lead compound, varespladib (A-002), a sPLA2 inhibitor, is enrolling a pivotal Phase III ACS trial, VISTA-16. As a novel anti-inflammatory, this approach is high risk but the ACS market opportunity is very large and thus the reward could be large. VISTA-16's interim analysis that will report in H1:11is likely to be a positive catalyst for the stock. We believe the trial is likely to continue should VISTA-16 replicate the FRANCIS data, validating the mechanism as safe and potentially effective. A-623 is an anti-BLyS peptibody for SLE. A-623 has potential advantages over GlaxoSmithKline/HGSI's Benlysta. The recent stoppage of a Phase II trial due to vial cracking should not result in a delay beyond H1:11. A-001 is an intravenous sPLA2 inhibitor in Phase II for acute chest syndrome due to sickle cell disease. The pipeline also includes additional potent sPLA2 inhibitors in pre-clinical development. With much news flow on tap on these first or potentially best-in-class compounds targeting either large and/or high unmet need markets, we believe ANTH will outperform over the next 12-18 months.

Anthera: Key Upcoming Events

Milestone	Timing
Varispladib (A-002)	
VISTA-16 Phase III initiated	June-10
1,000 patient interim biomarker analysis	H1:11
VISTA-16 final results	Q1:12
A-623	
Phase II initiated	Q4:10
Study stopped	November-10
Possible restart	H1:11
A-001	
Phase II data publication	Q1:11

Source: Company data

Varespladib Interim Biomarker Analysis The Next Event

Varespladib, a small molecule sPLA2-IIa inhibitor, is enrolling a pivotal Phase III study (VISTA-16) in ACS. The secretory PLA2's (sPLA2) are a family of enzymes directly involved in the acute and chronic steps of the inflammatory response. On balance, results of varespladib's proof-of-concept Phase II trials (PLASMA, PLASMA-2, and FRANCIS) support the move into Phase III. Despite broad adoption of statins and anti-platelet agents for the treatment of ACS, morbidity and mortality rates remain high. A novel, safe, and convenient approach that can reduce these rates by 15-20% could gain broad adoption in this large and well-established market. However, no data conclusively demonstrate that a reduction in inflammation alone - like sPLA2 inhibition - leads to a reduction in major adverse cardiovascular events (MACE), VISTA-16's primary endpoint. Therefore, we estimate the probability of success is 25-40%.

We believe the high-end of the probability of success range is more likely given that varespladib has demonstrated: (1) a reduction in inflammatory markers beyond sPLA2, including IL-6 and more importantly CRP; (2) a favorable impact on lipids, including an incremental 5-7 percentage point LDL reduction when used in combination with statins and potential synergies with Niacin; and (3) a side-effect profile presumably lacking cardiovascular signals demonstrated by a confidential DSMB review of safety from VISTA-16 which did not result in alternation or termination of the trial. Near term, we believe these attributes bode well for the 1,000+-patient interim biomarker efficacy analysis. While there are differences between VISTA-16 and FRANCIS, we take comfort in the consistency of the magnitude of inflammatory marker reduction and, more importantly, the enhanced LDL-C lowering power seen in FRANCIS. While VISTA-16's design encompasses added risk (discussed below) its composite endpoint excludes revascularization, an endpoint that plagued Atherogenic's AGI-1067 ARISE trial.

Nonetheless, this pivotal study is not without risk for the following reasons: (1) an agent targeting inflammatory markers has yet to demonstrate a CV outcomes benefit; (2) sPLA2 is a novel target and its inhibition has been unsuccessful in other inflammatory conditions; (3) many elements of VISTA-16's design, including the primary endpoint at 16-weeks, are novel; and (4) while varespladib has been studied in several thousand patients, its safety has yet to be confirmed in a single large study of high-risk patients.

Should VISTA-16 be successful, varespladib would face a large market opportunity. There are over 1.4MM ACS events in the U.S. per year and a similar amount internationally. A safe, effective, and convenient 16-week course of therapy that commands a premium price likely would have rapid and broad adoption. Anthera plans to commercialize varespladib in the U.S. but will partner it ex-U.S. Varespladib's market exclusivity periods - through February 2020 in the U.S. and 10 years of exclusivity in Europe - together with the potential absence of competing short-term therapies in ACS, are advantages. We estimate peak penetration of 25% ex-U.S., 35% in the U.S., and worldwide sales of $1.7B in 2018.

Varespladib ACS Market Buildup

ACS MARKET BUILD-UP

		2009	2010E	2011E	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
U.S.													
ACS Primary Diagnosis													
# of patients with MI		706,994	728,204	750,050	772,552	795,728	819,600	844,188	869,514	895,599	922,467	950,141	978,646
# of patients with UA		93,975	96,794	99,698	102,688	105,769	108,942	112,210	115,577	119,044	122,615	126,294	130,083
Total # of patients ACS primary diagnosis		800,969	824,998	849,748	875,240	901,498	928,542	956,399	985,091	1,014,643	1,045,083	1,076,435	1,108,728
ACS Secondary Diagnosis													
# of patients with MI		178,115	183,458	188,962	194,631	200,469	206,484	212,678	219,058	225,630	232,399	239,371	246,552
# of patients with UA		492,820	507,604	522,833	538,518	554,673	571,313	588,453	606,106	624,289	643,018	662,309	682,178
Total # of patients ACS secondary diagnosis		670,934	691,062	711,794	733,148	755,143	777,797	801,131	825,165	849,920	875,417	901,680	928,730
Total ACS Populations													
# of patients with MI	3%	885,109	911,662	939,012	967,182	996,198	1,026,084	1,056,866	1,088,572	1,121,229	1,154,866	1,189,512	1,225,198
# of patients with UA	3%	586,794	604,398	622,530	641,206	660,442	680,256	700,663	721,683	743,334	765,634	788,603	812,261
Estimated Total US ACS Population		**1,471,903**	**1,516,060**	**1,561,542**	**1,608,388**	**1,656,640**	**1,706,339**	**1,757,529**	**1,810,255**	**1,864,563**	**1,920,500**	**1,978,115**	**2,037,458**
Market Penetration U.S.						5%	10%	20%	25%	30%	35%	35%	20%
Number of patients on therapy						82,832	170,634	351,506	452,564	559,369	672,175	692,340	407,492
Price Per 16-week course ($)						1,600	1,600	1,600	1,600	1,600	1,600	1,600	1,600
A-002 U.S. Sales for ACS ($MM)						**133**	**273**	**562**	**724**	**895**	**1,075**	**1,108**	**652**
Ex-U.S.													
Estimated ACS Patients ROW (ex Japa	**90%**	**1,324,713**	**1,364,454**	**1,405,388**	**1,447,550**	**1,490,976**	**1,535,705**	**1,581,777**	**1,629,230**	**1,678,107**	**1,728,450**	**1,780,303**	**1,833,713**
16-Week ACS Market Penetration ex-U.S. (exclude Japan)						1%	10%	15%	20%	25%	25%	25%	25%
Number of patients on therapy						14,910	153,571	237,266	325,846	419,527	432,112	445,076	458,428
Price Per 16-week course						1,500	1,500	1,500	1,500	1,500	1,500	1,500	1,500
A-002 Ex-U.S. Sales for ACS ($MM)						**22**	**230**	**356**	**489**	**629**	**648**	**668**	**688**
Total W.W. Sales ($MM)						**$154.9**	**$503.4**	**$918.3**	**$1,212.9**	**$1,524.3**	**$1,723.6**	**$1,775.4**	**$1,339.6**

Source: Cowen and Company estimates; AHA; Company data

Anthera has two other clinical assets. A-623, licensed from Amgen, is an anti-BLyS mAb for the treatment of SLE that is administered by subcutaneous injection. In addition to the formulation advantage, A-623 binds soluble and membrane bound BLyS, and may have a more efficient and inexpensive *E. Coli*-based manufacturing process. However, in November 2010, A-623 experienced an important setback. A Phase IIb trial was stopped given that some vials were cracking and it was determined to be more than an isolated problem. Anthera is unsure when Phase IIB can restart given short supply of the drug, although patients dosing could resume in H1:11. Relatedly, management is close to disclosing a manufacturing agreement that will allow Anthera to supply sufficient amounts of A-623 to support Phase IIb studies. Anthera likely will license A-623 should the study be successful. Assuming a maximum 10% share of the BLyS mAb market and a royalty rate of 25%, A-623 could contribute 30% of total Anthera revenues by 2020. Last in the pipeline, A-001 is an intravenous sPLA2 inhibitor in Phase II development for acute chest syndrome in sickle disease. Anthera is not currently advancing A-001 and we do not model any revenues from A-001.

VISTA-16 Not Without Risk But Rationale Reasonable

Phase II Data Support Move Into Phase III

Anthera, under a Special Protocol Assessment (SPA), has commenced VISTA-16 (Vascular Inflammation Suppression to Treat Acute Coronary Syndrome -16 Weeks). The 6,500-patient study will evaluate short-term varespladib with Lipitor (atorvastatin) for the prevention of secondary major adverse cardiovascular events (MACE) in patients with recent ACS. An initial futility analysis is expected in H1:11. The decision to advance varespladib into Phase III was based on the results of Phase II studies, PLASMA, PLASMA-2, and FRANCIS. The majority of our physician consultants support the rationale to move into Phase III, although not all believe that VISTA-16's design, most notably its short duration, provides the best opportunity for success (discussed later).

Interim Biomarker Analysis Key Upcoming Event

VISTA-16's study design includes a 1,000-patient interim biomarker analysis. The staggered biomarker analysis begins post a minimum of 2 weeks of varespladib therapy and the data are likely in H1:11 at which point the DSMB has the option to stop the study if the biomarkers of inflammation and lipid profiles fail to meet pre-specified reductions. Each of the following needs to hit statistical significance in the arms: IL-6, CRP, sPLA2 and LDL. However, the timing for these measurements is different. IL-6 will be measured on weeks 2 and 4, sPLA2 and LDL on weeks 8 and 16, and CRP measurements will be done early and late. Anthera has not provided the expected magnitude of the prespecified reductions in the lipid and anti-inflammatory biomarkers or the delta between the arms required for continuation/futility.

Anthera estimates that the baseline LDL-C will be 115mg/dL - similar to that seen in FRANCIS. Patients have an opportunity to change their statin dose at week 8 based on the LDL-C reading if LDL-C levels are greater than 100mg/dL at that time. If we assume a baseline CRP of at least >2mg/dl and extrapolate the Phase II biomarker data, the trial likely will continue. We believe that there is a low probability (<5%) that the trial is stopped for futility. Continuation will reinforce the probability of success and likely will be a positive catalyst for the stock.

Phase II Biomarker Data Provide Confidence

Selective anti-inflammatory (e.g., cyclo-oxygenase 2 inhibitors) or antioxidant (e.g., Atherogenic's AGI-1067) therapies initially showed favorable effects on biomarkers and measures of efficacy, but were subsequently found to be adverse or neutral relative to cardiovascular disease events. We believe that there are several reasons why varespladib's Phase II data are different. The PLASMA (Phospholipase Levels and Serological Markers of Atherosclerosis) studies in stable cardiovascular disease and FRANCIS (Fewer Recurrent Acute coronary events with Near-term Cardiovascular Suppression) in ACS demonstrated reductions in sPLA2, IL-6, and CRP, and varespladib's impact on lipids was also favorable. Both these effects were synergistic in combination with a statin. In addition, there were no adverse cardiovascular signs. This is in contrast to AGI-1067, which increased CRP, LDL-C, and the rate of atrial fibrillation, and decreased HDL-C. In light of Crestor's JUPITER data, the combination of CRP and LDL lowering is very favorable, although JUPITER was a secondary prevention study in stable cardiovascular disease.

Comparison Of Anti-Inflammatory Biomarker And Safety Data In CV Trials

Drug Name	Company	LDL Change	CRP Change	sP-PLA2	LP-PLA2	IL-6	Blood Pressure	Arrhythmia	Malodor
AGI-1067	Atherogenics	↑	↑	?	?	?	↑	↑	?
Varespladib	Anthera	↓	↓	↓	?	↓	0	0	No
Darapladib	GlaxoSmithKline	0	↓	?	↓	0	↑	0	Yes

Source: Cowen and Company; Company data; Lancet; ESC; ACC

FRANCIS Supports Varespladib's Activity Through 16 Weeks, But Not Longer

FRANCIS, and to a lesser extent SPIDER-PCI, are more comparable to VISTA-16's design than the 8-week PLASMA and PLASMA-2 studies in stable cardiovascular disease. In FRANCIS, 625 patients with an ACS diagnosis within 96 hours were randomized to a minimum of 24 weeks of treatment with either varespladib 500 mg once-daily or placebo in combination with fixed-dose Lipitor (80 mg) and physician-directed standard of care. FRANCIS demonstrated robust biomarker results and favorable MACE trends through 16 weeks. However, the benefit of several of the marker analyses and MACE dissipated at 24 weeks. We view the incremental 5+pp LDL-C reduction in the varespladib arm as a major positive.

At 16 weeks, there were 13 (4.2%) MACE in the varespladib treated group versus 19 (6.1%) in the placebo group. However, at 24 weeks, this advantage narrowed and there were 23 (7.4%) MACE in the varespladib group versus 24 (7.7%) in the placebo group. With respect to safety, varespladib was well tolerated and there was no meaningful imbalance of overall adverse events. There was a higher incidence of mild and transient liver enzyme elevations (3x ULN). There were no effects on blood pressure or QT interval.

FRANCIS: Efficacy Data Trends Favorably Through 16 Weeks

	LDL-C Mean Percentage Change From Baseline			Median sPLA2 Percentage Change From			Median CRP Percentage Change From Baseline			IL-6 Median Percentage Change From Baseline		
	Varespladib + Lipitor 80mg	Lipitor 80mg	p-value	Varespladib + Lipitor 80mg	Lipitor 80mg	p-value	Varespladib + Lipitor 80mg	Lipitor 80mg	p-value	Varespladib + Lipitor 80mg	Lipitor 80mg	p-value
Week 2	~-46%	~-41%	p=0.0064	~-85%	~+5%	p<0.0001	~-40%	~-20%	p=0.183	~-18%	~-5%	p=0.18
Week 8	~-49%	~-44%	p=0.0023	~-83%	~-20%	p<0.0001	~-75%	~-70%	p=0.096	~-39%	~-34%	p=0.69
Week 16	~-44%	~-37%	p=0.017	~-63%	~-22%	p<0.0001	~-83%	~-72%	p=0.007	~-41%	~-38%	p=0.90
Week 24	NG	NG	NG	NG	NG	NG	NG	NG	NG	NG	NG	NG

Source: Company data

sPLA2 Favorable Correlation With CRP; Interaction With LDL-C Encouraging

Historical and recent clinical results have demonstrated a significant correlation between circulating levels of sPLA2 and the well-established inflammatory marker CRP. These and other clinical studies have also demonstrated that sPLA2 independently predicts coronary events in patients who have recently experienced an acute coronary syndrome and patients with stable CAD independent of other standard risk factors. In a stable cardiovascular patient, sPLA2 not only sustains chronic vascular inflammation, but it also adversely remodels lipoproteins such as low-density lipoprotein cholesterol, or LDL-C. Through a series of reactions, sPLA2 promotes the formation and oxidation of smaller, more pro-atherogenic and pro-inflammatory LDL-C particles. Moreover, these modified lipoproteins have a reduced affinity for LDL-C receptors, which are responsible for removal of cholesterol from the body. As a result, LDL-C remains in circulation longer and has a greater tendency to deposit in the artery wall. This increased LDL-C deposition and sustained chronic vascular inflammation may contribute to the development of atherosclerosis.

However, Results Of SPIDER-PCI Provide Reason For Pause

In May 2007, an investigator sponsored study, SPIDER-PCI (sPLA2 Inhibition to Decrease Enzyme Release after PCI), was initiated. The study compared varespladib once-daily to placebo for up to 10 days in 144 patients undergoing PCI. The study missed the primary endpoint, a reduction in the elevation of CK-MB or troponin I above the upper limit of normal at six to eight hours or 18 to 24 hours. Fifty-seven percent of patients in the varespladib arm versus 51% of placebo patients (p = 0.55)

had raised cardiac enzymes. Even though this is not a multi-center study and is focused primarily on PCI, the results are not favorable and raise caution. This is especially concerning given the results showed statistically significant reductions of sPLA2 as early as 18 hours post-PCI procedure, which persisted throughout the five days of dosing (-93.0%, $p < 0.001$) and numerical reductions in CRP from baseline versus placebo at three to five days (-82.1%, $p = 0.23$).

Phospholipase Biology Complex But Link With Atherogenesis Secure

Phospholipase A2s hydrolyse the sn–2 ester bond in phospholipids releasing fatty acid products and lysophospholipids, of which lysophosphatidyl choline (lysoPC) is the most important. In many cases the products are pro-inflammatory, and hence may have significant effects on atherogenesis, but there are also studies that suggest that in certain circumstances phospholipase activity may remove pro-inflammatory molecules. Thus, there is a debate about the pro- or anti-atherogenic role of these enzymes. There are five different types of phospholipase A2s, the secreted and cytosolic forms, the calcium-independent PLA2s (e.g. varespladib's target), the lysosomal PLA2s, and the platelet-activating factor acetylhydrolases (PAF-AH, which include Lp-PLA2, e.g. GlaxoSmithKline's darapladib's target).

Varespladib Targets sPLA2-IIA, An Important Acute Phase Protein Associated With Atherogenesis

There are nine different members of sPLA2 group found in humans. They have been associated with a range of inflammatory, autoimmune and allergic disorders with different isoforms being expressed and released by specific inflammatory cells. Effects relevant to atherogenesis include the promotion of chemokine production from monocytes and macrophages, and modification of plasma lipoproteins, making them more atherogenic. The three forms of sPLA2 that have so far been linked with atherogenesis are sPLA2-IIA, sPLA2-V and sPLA2-X. sPLA2-IIA, unlike the others, is an acute phase protein. Varespladib is a sPLA2-IIA inhibitor.

Lp-PLA2 Atherogenic Or Atheroprotective?

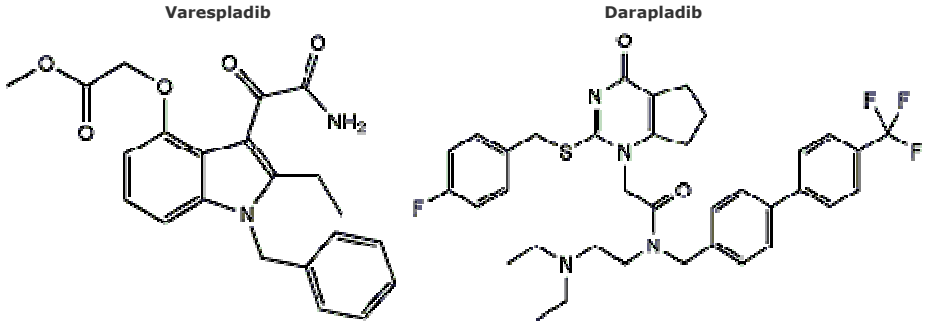
In early studies Lp-PLA2 was known as platelet-activating factor acetylhydrolase (PAF-AH). This indicates that the enzymatic activity studied was the hydrolysis of PAF. The relevance of the PAF-AH activity in humans is of significance in the debate about the atherogenic or atheroprotective roles of Lp-PLA2. It is secreted by monocyte-derived macrophage and mast cells, and it is associated in human plasma with lipoproteins, particularly LDL. Lp-PLA2 has been detected in atherosclerotic plaque, particularly in plaques with a necrotic core and in ruptured plaques. Lp-PLA2 has unusual substrate specificity, being unique in hydrolysing PAF and short-chain fatty acids in the sn–2 position. In LDL these fatty acyl chains may be shortened by oxidation of unsaturated acyl chains, and in this way Lp-PLA2 provides one mechanism by which oxidized LDL exerts its atherogenic effects.

sPLA2 And Lp-Pla2 Distinct But Varespladib Appears To Have The Clinical Edge Over Darapladib

The literature supports the distinction between sPLA2 and Lp-PLA2 but we believe the Phase II data in cardiovascular studies are most relevant. In contrast to varespladib's Phase II data, darapladib's Phase II biomarker data demonstrated no change in LDL-C and HDL and an increase in triglycerides. There was a decrease in CRP and IL-6. In addition, darapladib has been associated with an exacerbation of

asthma, and malodor in urine and feces. Varespladib has not been associated with these signs. Darapladib's intravascular ultrasound study missed the co-primary endpoint but the secondary endpoint, change in necrotic core size, was significantly lower in patients treated with darapladib (i.e., necrotic core size remained unchanged in the darapladib group but increased among those treated with placebo). In sum, the Phase II data are in varespladib's favor, but it is unclear whether these differences ultimately will translate into a clinical distinction. It is even possible that these agents are synergistic but at this stage we have no visibility into combination studies.

Chemical Structures Of Varespladib Versus Darapladib



Source: Thomson-Pharma

Darapladib's Phase II Biomarker Study Raises More Questions Than It Answers

The results of darapladib's Phase II biomarker study were presented at ACC 2008. The dose-ranging biomarker study enrolled 959 patients with CHD or CHD-risk-equivalent on Lipitor 20 mg or 80 mg and randomized them to oral darapladib 40 mg, 80 mg, 160 mg or placebo once daily for 12 weeks. At baseline, mean LDL-C was 67 ± 22 mg/dL (much lower than presumed in FRANCIS). Darapladib 40, 80, and 160 mg inhibited Lp-PLA2 activity by approximately 43%, 55%, and 66%, respectively, compared with placebo ($p<0.001$ at both weeks 4 and 12). Sustained dose-dependent inhibition was noted overall at different baseline LDL-C ($\geq$70 vs. <70 mg/dl) and HDL-C (<40 vs. $\geq$40 mg/dl). At 12 weeks, darapladib 160 mg decreased IL-6 by 12.3% (95% CI -22% to -1%; p=0.028) and hs-CRP by 13.0% (95% CI -28% to -5%; p = 0.15) compared with placebo. Treatment with darapladib did not modify total cholesterol, LDL-C, HDL-C, or triglyceride levels at week 4 or at week 12 as compared with placebo. This may be because Lp-PLA2 is firmly associated with ApoB-containing lipoproteins and patients were heavily pretreated with statins. Glaxo claims success in that darapladib was able independently to lower Lp-PLA2 without lowering the LDL-C.

IBIS-2: Primary Endpoint Missed But Necrotic Core Stabilization Encouraging

The IBIS-2 study was presented at ACC 2008 and ESC 2008. The Integrated Biomarker and Imaging Study-2 (IBIS-2) study compared the effects of 12 months of treatment with darapladib (160 mg daily) or placebo on coronary atheroma deformability and hs-CRP in 330 patients with angiographically documented coronary disease. The study used novel IVUS methodologies for the primary and secondary endpoints: IVUS-based palpography that measures the mechanical properties of the tissue during the cardiac cycle that shows high strain values when the dominant plaque type is fatty or fibrofatty, and IVUS-based virtual histology that uses radiofrequency spectral analysis of the ultrasound backscatter signals to

identify 4 different components of atherosclerotic plaque (fibrous, fibrofatty, dense calcium, and necrotic core).

IBIS-2 missed the primary endpoint. After 12 months, there were no significant differences between groups in plaque deformability (P=0.22) or hs-CRP (P=0.35). In the placebo-treated group, however, necrotic core volume increased significantly (4.5±17.9 mm3; P=0.009), whereas darapladib halted this increase (-0.5±13.9 mm3; P=0.71), resulting in a significant treatment difference of -5.2 mm3 (P=0.012). Sensitivity analyses suggested that this beneficial treatment effect was similar in various clinical subgroups as defined by established risk factors. The investigators did not analyze whether the treatment effect was affected by circulating oxidized phospholipid levels, which may be the main driving force behind the proatherogenic effects of Lp-PLA2 activity. Because the concentration of oxidized LDL, an important source of oxidized phospholipids, is ~70-fold higher in plasma than in atherosclerotic plaque, the effects of Lp-PLA2 activity and Lp-PLA2 inhibition may have substantially different, and perhaps even opposing, effects in plasma compared with atherosclerotic plaque.

The composite of cardiovascular death, myocardial infarction, stroke, and revascularization was 17% in the darapladib group versus 19% in the placebo group. The incidence of adverse events leading to withdrawal was similar with 7% (n=11) in placebo and 4% (n=7) in the darapladib group. A higher incidence of malodor (mainly feces or urine) was reported with darapladib (16%, n=28) compared with placebo (3%, n=5), but was not a common cause of withdrawal from the study (darapladib, 2%, n=3). Routine measurements of systolic blood pressure showed a difference between the groups (darapladib showed mean difference of 3.0 mm Hg, above placebo, p=0.031) that was not previously observed in other clinical trials with darapladib.

Darapladib Pivotal Program Significant But Several Years Away

Glaxo has approached darapladib's development more conservatively. Darapladib is in two large Phase III trials, STABILITY and SOLID. In December 2008, GlaxoSmithKline initiated a 15,500 patient chronic CVD trial, STABILITY. In December 2009, SOLID began recruiting 11,500 post-acute ACS patients with the first primary endpoint data expected in 2014. While SOLID is an ACS study there are several differences between it and VISTA-16. These differences include: (1) SOLID is studying post-acute ACS patients versus acute ACS for VISTA-16; (2) SOLID is significantly larger and plans to enroll 11,500 patients versus 6,500 for VISTA-16; (3) SOLID will stop at 1,500 major adverse cardiovascular events (MACE) versus 385 MACE for VISTA-16; and (4) length of therapy is a minimum of two years versus 16 weeks for VISTA-16.

STABILITY is an event driven trial (~1,500 events) in patients with stable chronic coronary heart disease. STABILITY completed enrollment in December 2009 and is scheduled for completion in 2012. GlaxoSmithKline conducted an interim review of the STABILITY study toward the end of 2009 that was the trigger to initiate the SOLID study. In SOLID-TIMI 52, men and women with ACS and receiving standard of care are randomized 1:1 to once-daily doses of darapladib 160 mg or placebo. The study will be stopped when approximately 1,500 reports of first occurrence of a major adverse cardiovascular event (MACE), including cardiovascular death, non-fatal heart attack or non-fatal stroke, have occurred. Glaxo estimates that patients will be on darapladib for at least two years and up to three years. Interim independent analyses are planned.

Despite Mostly Favorable Phase II Data, VISTA-16 Is Risky

In February 2010, Anthera reached agreement with FDA on a Special Protocol Assessment (SPA). Anthera began dosing patients in VISTA-16 in June 2010. VISTA-16 will enroll 6,500 patients over 14-15 months. VISTA-16's primary endpoint is a reduction in a composite MACE at week 16. Ultimately, we believe that VISTA-16 has a 40% chance of success for the following reasons: (1) the 16-week time point may be too short to detect a difference between the arms; (2) the assumption of the 8.5% placebo event rate may be aggressive and the hurdle for an 18-25% treatment effect is high; and (3) the variable use of Lipitor may add complexity.

Are 16-Weeks Enough To Determine A Benefit?

Anthera believes that VISTA-16 should be able to confirm varespladib's efficacy at 16 weeks. This is in part based on Lilly/Daiichi's TRITON and AstraZeneca's PLATO studies. These are contemporary ACS studies, with early intervention, and it appears (not measured) that at 120 days there is already a favorable MACE benefit. In TRITON, at 120 days (~16 weeks) the event rate was approximately 10.5% in the Plavix arm and approximately 8% in the Effient arm, a 25% reduction. In PLATO the event rate was approximately 8.5% and 7.0% in the Plavix and Brilinta arms, respectively; an 18% reduction. Patients with unstable angina were excluded from these studies, potentially resulting in a lower event rate than would be predicted in VISTA-16.

We are cautious about relying too heavily on these trials for the following reasons: (1) these studies investigated more aggressive platelet therapy and not early intensive statin therapy; (2) there was no predefined statistical analysis at the 120-day time point for either trial; and (3) the MACE endpoints are not identical to VISTA 16's endpoints.

We believe that studies in ACS comparing more intensive statin therapy are potentially better comparables because of varespladib's synergy with statins and its limited impact on platelets. In a meta-analysis conducted by Hulten et al (Arch Intern Med. 2006; 166:1815-1821), which is summarized in the table below, the benefit of more intensive statin therapy was consistently evident across trials only after four months of therapy. The meta-analysis incorporated 13 trials including the landmark PROVE-IT, MIRACL, and A to Z studies. We acknowledge that these trials are not identical to VISTA-16, but a few individual studies in this meta-analysis demonstrated a statistical benefit by four months. Nonetheless, the overall benefit begins to accrue after this period and is sustained through two years. The authors hypothesize that the reason for the delayed benefit is that it may take several months for plaque stabilization to occur with a statin. If varespladib stabilizes plaque earlier than when statins are presumed to do, this could lead to a benefit by 16 weeks. However, darapladib's Phase II IBIS-2 did not consistently demonstrate plaque stabilization, and it is unclear that the benefit seen on the necrotic core was seen within four months. We therefore conclude that an ACS trial of longer duration, more in line with darapladib's ACS SOLID trial, likely would increase the probability of success.

Pooled Hazard Ratios (HR) For Any Cardiovascular Event Over 24 Months

Month	HR (95% CI)	Heterogeneity
1	1.02 (0.95-1.09)	Q_{12}=64.8; I^2=86.1%
4	0.84 (0.72-1.02)	Q_{10}=591.6; I^2=98.5%
6	0.76 (0.70-0.84)	Q_6=79.4; I^2=92.4%
12	0.80 (0.76-0.84)	Q_5=78.7; I^2=93.6%
24	0.81 (0.77-0.87)	Q_3=58.5; I^2=94.9%
Pooled HR	0.84 (0.76-0.94)	Q_4=395; I^2=89.9%

Source: Hulten et al (Arch Intern Med. 2006; 166:1815-1821); Cowen and Company

High Hurdle But Not Impossible To Demonstrate 18-25% Improvement If MACE Rate Is 8.5%

VISTA-16 assumes a placebo MACE rate of 8.5%. The company calculates that approximately 385 events are required to detect an 18% treatment effect with 80% power and a type I error rate of 0.05 (Scenario 1). Three hundred ninety-five events are sufficient to detect a treatment effect of 25% with 80% power and a type I error rate of 0.05 (Scenario 2). A treatment effect of 18-25% is likely clinically relevant but the hurdle is significant given that Lipitor is used as the active control. Using crude statistics (i.e., a constant hazard ratio and no dropouts), we calculate that the varespladib arm will need to have an event rate of less than 6.7-7% in Scenario 1 and less than 6.4-6.5% in Scenario 2. This is a 1.5-2.1 percentage point (pp) difference. If we use varespladib's FRANCIS data that demonstrated a ~37% LDL-C reduction in the Lipitor arm and a 44% LDL-C reduction in the varespladib + Lipitor arm at week 16, and assume a baseline LDL-C of 110mg/dL (lower than in FRANCIS), we calculate a LDL-C difference of 7.7mg/dL at 16 weeks for VISTA-16. There is data that suggests a 1% benefit in relative risk for each 1.6mg/dL in LDL-C reduction. Using this assumption, the 8mg/dL LDC-C difference in FRANCIS would translate into a 4.8pp reduction in relative risk, more than double that required for statistical significance. However, we note that the 7.7mg/dL LDL difference likely will not be replicated for the following reasons: (1) Lipitor 80mg is not standard in VISTA-16 and the Lipitor dose can be modified at week 8 (potentially to the disadvantage of varespladib); (2) while the 7pp difference between the LDL-C reduction was significant at 16-weeks in FRANCIS, it was less significant than demonstrated in earlier weeks (i.e., an unfavorable trend); (3) we assume a baseline LDL-C of 110mg/dL which may not be replicated; and (4) the FRANCIS sample size is much smaller.

In addition, it is not clear that the relationship of a 1.6mg/dL reduction in LDL-C resulting in a 1pp increase in relative risk will hold. This relationship was established when baseline LDL-Cs were higher and in the context of using a statin to lower the LDL-C (i.e., not supercharged by an anti-inflammatory agent - a similar question is asked of Merck's Zetia ENHANCE trial). In the table below, we use different scenarios to test the sensitivity of these assumptions (baseline LDL-C, relative LDL-C reduction, and relationship of LDL-C to relative risk). Given the variables, the range of scenarios is broad, but other than our very conservative Scenario 3, it is possible that varespladib may achieve significance (>1.5-2.1pp MACE difference between arms).

VISTA-16 Primary Endpoint Sensitivity Analysis On LDL-C Reduction

	Scenario 1*	Scenario 2	Scenario 3
Baseline LDL-C (mg/dL)	110	110	105
Lipitor arm: 16 week LDL-C reduction	37%	45%	45%
Calculated 16 week LDL-C (mg/dL)	69.3	60.5	57.8
Lipitor+ varespladib arm: 16 week LDL-C reduction	44%	50%	48%
Calculated 16 week LDL-C (mg/dL)	61.6	55.0	54.6
LDL-C difference (mg/dL)	7.7	5.5	3.2
Relationship of LDL-C reduction to a 1pp relative risk benefit	1.6	2	2
Potential percentage point MACE benefit in the varespladib arm	4.8	2.8	1.6

* Assumes lower baseline LDL-C than in FRANCIS (~125mg/dL) but uses LDL-C percentage reduction seen in FRANCIS

Source: Cowen and Company; Company data; ENHANCE methodology paper

Variable Lipitor Dose May Confound Results

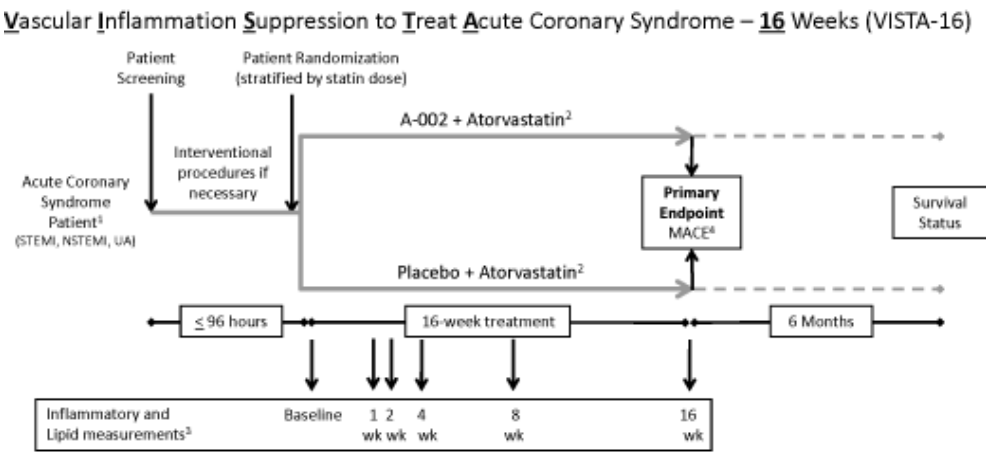
In an effort to replicate the real word, VISTA-16 does not fix the Lipitor dose. Instead, Lipitor doses will be reassessed at the 8 week time point and doses will be adjusted in patients where LDL-C is >100mg/dL. This is different from in FRANCIS, where Lipitor 80mg was standard across the treatment arms. Variable Lipitor dosing will therefore confound estimates regarding the predicted placebo rate and make achieving the target event rate of 8.5% less certain.

VISTA-16 Study Background

VISTA-16 will enroll up to 6,500 patients in up to 15 countries. Patients will be randomized to receive either varespladib 500mg once-daily or placebo in addition to a dose of Lipitor. The dose of Lipitor may be adjusted after eight weeks based on the subjects' LDL-C measurement. Patients will be treated with A-002 or placebo and a dose of Lipitor for 16 weeks and survival status will be obtained for patients six months after the completion of dosing.

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. 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. The number of subjects who undergo PCI following the index event and prior to randomization will be limited to no more than 40% of the total patient population.

The primary endpoint of the VISTA-16 study will be to determine whether 16 weeks of once-daily treatment with A-002 plus a dose of Lipitor is superior to placebo plus Lipitor 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, as defined by recent FDA draft guidance. Enrollment may be stopped anytime after a minimum of 395 adjudicated endpoint events as described in the protocol have occurred.



1 Patients will receive physician-directed interventional and therapeutic standard of care throughout the study
2 A 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, Major Adverse Coronary Events (MACE) is defined as Cardiovascular Death, Non-Fatal Myocardial Infarction, Non-Fatal Stroke, and Unstable Angina requiring urgent hospitalization

Source: Company Filing

A secondary endpoint for the VISTA-16 study is to determine whether varespladib plus a dose of Lipitor is superior to placebo plus Lipitor 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. A comparison between treatment groups will also be made for each component of the primary efficacy endpoint. Additionally, the time to multiple occurrences of any non-fatal component of the composite primary endpoint will also be explored. The biomarkers CRP, IL-6, LDL-C and sPLA2, will also be evaluated at each time point of the clinical study.

A DSMB will continually evaluate the performance of the VISTA-16 study over time to ensure patient safety and to review certain blinded laboratory data from the VISTA-16 study. The first interim DSMB review did not result in any alterations of trial design and bodes well for trial continuation. After a minimum of 1,000 patients have completed the 16-week treatment in the VISTA-16 study, the DSMB will conduct a biomarker futility analysis to ensure patient levels of inflammation, as measured by sPLA 2, CRP and IL-6, and lipid profiles, as measured by LDL-C, have met pre-specified reductions from baseline at various time-points.

Varespladib Strongly Positioned In Large, Established ACS Market

According to the American Heart Association, over 18MM people in the United States have experienced an acute coronary syndrome. About 800K patients will have a primary ACS diagnosis and 670K will have ACS as a secondary diagnosis for a total of 1.4MM diagnoses per year. We estimate this will grow by 3% per year. The American Heart Association estimates that worldwide, cardiovascular disease kills an estimated 17.5MM people each year. According to British Heart Foundation statistics, coronary artery disease (CAD), which often leads to acute coronary syndrome or heart attacks, accounts for 1.9MM deaths in Europe annually. According to the World Health Organization, cardiovascular disease is the most common cause of death in the Western world and a major cause of hospital admissions. These numbers are expected to increase given an aging population, as well as the rising epidemics of diabetes and obesity, two conditions known to increase the risk of acute coronary syndrome. Conservatively, we estimate the ACS rate ex-U.S.

(excluding Japan) to be 90% of the U.S. rate, or ~1.3MM events in 2010 growing at 3% per year.

We assume Anthera will launch varespladib in the U.S. in Q1:13 at a price of $1,600 for a 16-week course. This is approximately $14.50/day. This is a significant premium to statins, especially post Lipitor generics, but should VISTA-16 show varespladib to be effective, this price likely will be supported by health economics. We model initial penetration growing from 5% in year 1 to a peak of 35% in 2019. This is likely attainable given that this market is well established. We have not factored in potential competition from Glaxo's darapladib. We do not model varespladib use in stable cardiovascular disease. We model the ex-U.S. ACS launch in 2014 at a lower price point and with a peak penetration of 25%. In sum, we assume peak sales of $1.8B in 2019.

U.S. Patent Life Through Q1:20 Potentially Conservative

Varespladib was originally discovered and developed by Lilly (under license from Shionogi) for the treatment of several inflammatory disorders. Varespladib's U.S. composition of matter patent, '326, was issued in August 1997, and expires in August 5 2014. Anthera believes it has baseline protection through February 2020, based on five years of Hatch-Waxman restoration and a six-month pediatric extension. We assume exclusivity through February 2020. In Europe, varespladib likely will obtain a new chemical entity exemption for 10 years. If we assume a 2014 launch, we estimate European exclusivity through 2024. On the basis of varespladib's Phase II studies, the finding of LDL lowering, especially the synergy in combination with statins was unexpected. This is the basis for two new generations of patents that Anthera has filed with the PTO. If either is approved, varespladib will have IP protection through at least 2028.

Varespladib ACS Market Buildup

ACS MARKET BUILD-UP

		2009	2010E	2011E	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
U.S.													
ACS Primary Diagnosis													
# of patients with MI		706,994	728,204	750,050	772,552	795,728	819,600	844,188	869,514	895,599	922,467	950,141	978,646
# of patients with UA		93,975	96,794	99,698	102,688	105,769	108,942	112,210	115,577	119,044	122,615	126,294	130,083
Total # of patients ACS primary diagnosis		800,969	824,998	849,748	875,240	901,498	928,542	956,399	985,091	1,014,643	1,045,083	1,076,435	1,108,728
ACS Secondary Diagnosis													
# of patients with MI		178,115	183,458	188,962	194,631	200,469	206,484	212,678	219,058	225,630	232,399	239,371	246,552
# of patients with UA		492,820	507,604	522,833	538,518	554,673	571,313	588,453	606,106	624,289	643,018	662,309	682,178
Total # of patients ACS secondary diagnosis		670,934	691,062	711,794	733,148	755,143	777,797	801,131	825,165	849,920	875,417	901,680	928,730
Total ACS Populations													
# of patients with MI	3%	885,109	911,662	939,012	967,182	996,198	1,026,084	1,056,866	1,088,572	1,121,229	1,154,866	1,189,512	1,225,198
# of patients with UA	3%	586,794	604,398	622,530	641,206	660,442	680,256	700,663	721,683	743,334	765,634	788,603	812,261
Estimated Total US ACS Population		**1,471,903**	**1,516,060**	**1,561,542**	**1,608,388**	**1,656,640**	**1,706,339**	**1,757,529**	**1,810,255**	**1,864,563**	**1,920,500**	**1,978,115**	**2,037,458**
Market Penetration U.S.						5%	10%	20%	25%	30%	35%	35%	20%
Number of patients on therapy						82,832	170,634	351,506	452,564	559,369	672,175	692,340	407,492
Price Per 16-week course ($)						1,600	1,600	1,600	1,600	1,600	1,600	1,600	1,600
A-002 U.S. Sales for ACS ($MM)						**133**	**273**	**562**	**724**	**895**	**1,075**	**1,108**	**652**
Ex-U.S.													
Estimated ACS Patients ROW (ex Japa	**90%**	**1,324,713**	**1,364,454**	**1,405,388**	**1,447,550**	**1,490,976**	**1,535,705**	**1,581,777**	**1,629,230**	**1,678,107**	**1,728,450**	**1,780,303**	**1,833,713**
16-Week ACS Market Penetration ex-U.S. (exclude Japan)						1%	10%	15%	20%	25%	25%	25%	25%
Number of patients on therapy						14,910	153,571	237,266	325,846	419,527	432,112	445,076	458,428
Price Per 16-week course						1,500	1,500	1,500	1,500	1,500	1,500	1,500	1,500
A-002 Ex-U.S. Sales for ACS ($MM)						**22**	**230**	**356**	**489**	**629**	**648**	**668**	**688**
Total W.W. Sales ($MM)						**$154.9**	**$503.4**	**$918.3**	**$1,212.9**	**$1,524.3**	**$1,723.6**	**$1,775.4**	**$1,339.6**

Source: Cowen and Company; AHA; Company data

A-623 Targets Lupus; May Be Differentiated

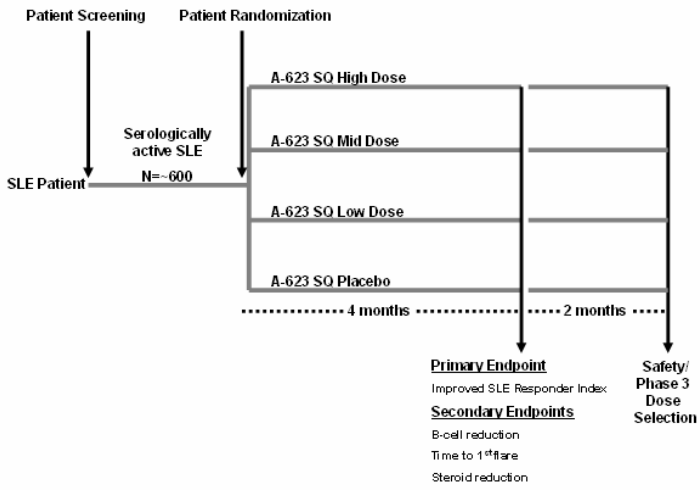
A-623, licensed from Amgen, is a selective peptibody antagonist of BLyS being developed for systemic lupus erythematosus or SLE. The BlyS/BAFF target has been validated by GlaxoSmithKline/Human Genome Science's Benlysta Phase III program. Anthera initiated a Phase II proof-of-concept study in Q4:10. This trial was recently suspended because of cracked vials. While a date for resuming the PEARL-SC trial has not been set, it is expected to restart sometime in H1:11. Two randomized, dose-ranging, placebo-controlled Phase 1 clinical studies of A-623 in 107 lupus patients have been completed. These studies demonstrated that A-623 generated anti-BLyS activity and showed statistically significant reductions in B-cells among lupus patients ($p < 0.001$).

Anthera believes A-623 may offer a number of points of differentiation over Benlysta and other BLyS antagonists. These include: (1) subcutaneous dosing, (2) improved pharmacodynamic benefits given binding to both membrane bound and soluble forms of BlyS, and 3) potential manufacturing benefits and lower cost of goods based on an *Escherichia coli* production process. It is unclear whether these factors will translate into clinical and commercial advantages especially given that A-623 lags Glaxo's Benlysta in development. Benlysta is an intravenous anti-BLyS/BAFF fully human IgG1 monoclonal antibody that only binds soluble BLyS. Glaxo filed global registration in Q2:10 and have since received FDA priority review designation, a favorable FDA advisory review committee vote on efficacy and safety, and has a PUDFA date of December 9, 2010, placing Benlysta at least 3-4 years ahead of A-623. Nonetheless, should Benlysta be approved, Street estimates range from $1B to $3B+ given the high unmet need. We believe that Benlysta will be approved despite its modest efficacy. Benlysta's regulatory success (or failure) will provide a road map for A-623. We conservatively estimate that A-623 obtains at peak a 10% share of the BlyS antibody market, which calculates to $585MM in worldwide sales. Anthera is actively pursuing a partnership with larger companies to develop and commercialize A-623. We model a deal in 2012 post completion of the Phase IIb trial.

A-623's Phase IIb (PEARL-SC) Stopped

Anthera initiated recruitment for its Phase IIb study of A-623, PEARL-SC, and enrollment had been successful in the U.S., Columbia, and Mexico. Six hundred patients with a diagnosis of serologically active SLE on stable therapy were to be randomized to one of three subcutaneous administration treatment groups of A-623 or placebo. All patients enrolled were to be treated with A-623 plus physician-directed standard of care, or placebo plus physician-directed standard of care, for at least four months, followed by a two-month safety follow-up after the treatment period.

A-623 Proposed Phase IIb SLE SubQ Administration Study



1Patients will receive physician-directed therapeutic standard of cared throughout the study

2 SELENA/SLEDAI: Safety of Estrogen in Lupus Erythematosus National Assessment (SELENA) and SLE Disease Activity Index (SLEDAI)

Source: Company Filing

A single vial of warehoused A-623 was discovered to have cracked unexpectedly. A thorough review of additional A-623 stocks revealed that this was not an isolated incident. As a result of vial instability, Anthera suspended the PEARL-SC trial indefinitely. It has not yet been determined what portion of existing drug is salvageable and what portion will need to be replaced. Management is actively seeking manufacturing solutions to provide an adequate supply of drug to continue to support Phase II trials. PEARL-SC is expected to resume in H1:11.

Estimated A-623 Sales Buildup

		2009	2010E	2011E	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Number of SLE Patients U.S.		325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000
Number of SLE Patients ROW		325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000	325,000
Estimated W.W. Total Lupus Population		**650,000**	**650,000**	**650,000**	**650,000**	**650,000**	**650,000**	**650,000**	**650,000**	**650,000**	**650,000**	**650,000**	**650,000**
Eligibile Patients for anti-Blyss mAb	**30%**	**195,000**	**195,000**	**195,000**	**195,000**	**195,000**	**195,000**	**195,000**	**195,000**	**195,000**	**195,000**	**195,000**	**195,000**
Benlysta share			100%	100%	100%	100%	99%	98%	97%	95%	90%	90%	90%
A-623 share							1%	2%	3%	5%	10%	10%	10%
Estimate number of patients on A-623							1,950	3,900	5,850	9,750	19,500	19,500	19,500
A-623 Price per year ($)							30,000	30,000	30,000	30,000	30,000	30,000	30,000
Total A-623 W.W Sales ($MM)							**$59**	**$117**	**$176**	**$293**	**$585**	**$585**	**$585**

Source: Company data, Cowen and Company estimates

Benlysta Likely First Novel Drug In SLE For Decades

Human Genome Sciences and Glaxo have successfully completed the largest ever Phase III program conducted in lupus, enrolling a total of approxmiately 1,620 patients. Benlysta, a BLyS/BAFF mAb, was filed in the E.U. and U.S. in Q2:10. BLyS is an endogenous protein that is necessary for the maturation of B-lymphocytes into plasma cells. Elevated levels of BLyS have been found in patients with active lupus, and may facilitate autoantibody production, disease activity via promoting B cell survival, B cell differentiation into plasma cells, Ig class switching, and Th1-associated inflammatory responses, and prevention of T cell effector responses.

Benlysta has completed two replicate randomized double-blind, placebo-controlled Phase III trials, BLISS-52 and BLISS-76. The Phase III trials have an SPA and the EMEA's approval as having an adequate study design. Although the two studies had similar protocols, BLISS-52 was a 52-week study and was conducted in Asia, South America,

and Eastern Europe while BLISS-76 was a 76-week trial (but included a prespecified primary analysis at week 52 for purposes of regulatory filings) and was conducted in the U.S. and Europe. In April 2010, GlaxoSmithKline and partner HGS announced that BLISS-76 missed the key secondary endpoint at week 76. There is much debate about the clinical/regulatory significance of this miss.

Phase III Program Demonstrates Statistical Benefit At Week 52

In BLISS-52 both doses of Benlysta met the primary endpoint at 52 weeks. At week 52, 57.6% of patients in the Benlysta 10mg/kg group were responders vs. 51.4% in the 1mg/kg group and 43.6% in the placebo group. The 10mg/kg treatment arm was statistically significantly different from placebo as early as week 16, and maintained significance throughout the trial. Consistent with Benlysta's Phase II experience, there were no imbalances in AEs, serious AEs, severe AEs, serious and/or severe AEs, dose interruptions, discontinuations, or death. The most common side effects (≥10%) were headache, arthralgias, upper respiratory tract infections, urinary tract infections, influenzea, diarrhea, nasopharyngitis, hypertension, and nausea. Rates of infection, including serious and severe infusion reactions, hypersensitivity, malignancy, and death were balanced.

In November 2009, Human Genome Science announced positive top-line results from BLISS-76, after 52 weeks of treatment. Benlysta 10mg/kg met the primary endpoint, a ≥4 point reduction in the SELENA-SLEDAI score. Unlike in BLISS-52 where approximately 70% of all patients were on doses of prednisone >7.5mg/day and 40% were on other immunosuppressive, in BLISS-76 46% of patients were on such a dose of prednisone and 56% were taking other immunosuppressive. Similar to BLISS-52, Benlysta was generally well-tolerated. The percent of patients with AEs, serious and/or severe AEs, infections, serious and/or severe infections, and discontinuations due to side effects were similar between treatment arms. Malignancies (breast cancer and ovarian within first few months of the trial beginning; cervical adenocarcinoma; two skin cancers; one gastric cancer) were documented in all three study arms (2 in the 10mg/kg, 3 in the 1mg/kg, and 1 in placebo) and one death occurred in the 10mg/kg arm, two deaths in the 1mg/kg arm, and none in placebo. No patients administered Benlysta have subsquently developed TB or PML.

Benlysta Could Be Used In 30% Of Lupus Patients

Assuming Benlysta is approved for the treatment of lupus, we anticipate the drug will be priced at $30K+/patient/year (on par with Roche's Rituxan). Experts note that lupus remains an unmet medical need since the disease is poorly treated with limited options. Our rheumatology consultants have different opinions, but are willing to use a new therapeutic of even modest efficacy, as long as it is generally well tolerated. If Benlysta is approved by the FDA, we estimate that up to 30% of patients will start therapy.

SLE A Significant Unmet Need

Systemic lupus erythematosus (SLE) is an idiopathic relapsing and remitting autoimmune disorder that predominantly affects women in their 20s and 30s. It has a varying prevalence depending on race (40-50 cases per 100,000 Northern Europeans, 200 per 100,000 Africans). We estimate 325K patients in the U.S. and similar number ex-U.S. are diagnosed each year. Fifteen-year survival is 80% and newly diagnosed 20-year-old patients have a 17% chance of dying before the age of

35. SLE patients are at increased risk for cardiovascular disease, malignancies, and infections. SLE is not one disease but rather a constellation of signs, symptoms, and pathology that vary with each patient. Severity depends on race (Africans and Hispanics worse off), gender (males worse off), age (older patients worse off), and geographic location. The exact etiology is thought to be multifactorial, with genetics, immunology (especially interferon alpha), hormonal imbalances, and environment all playing a role. Any organ system can be affected, with the most common manifestations being constitutional symptoms, arthritis, and mucocutaneous, renal, and hematologic disease.

Physicians use immunosuppressants to induce remission in patients with active disease as well as to maintain patients in a quiescent disease state. Current therapies, most of which are used off-label, have many unwanted side effects. During a flare, the goal of therapy is to use a combination of two or more medications (one usually being prednisone) to induce remission. Then after the patient has been in remission for about 6 months, rheumatologists consider scaling down therapy. While consultants indicate that steroids are responsible for a majority of the longer term morbidity issues associated with lupus, they are reluctant to withdraw immunosuppressive therapy for fear of risking a disease flare.

No new therapies for lupus have been approved in decades and numerous products have failed in clinical testing. The difficulties in developing drugs for lupus revolve, in part, around choosing an appropriate endpoint. While declining anti-dsDNA antibody levels are viewed as a favorable prognostic indicator in lupus nephritis, the FDA has been reluctant to accept this or any other biomarker as a surrogate endpoint of disease activity. Hence, the options for drug developers include undertaking an all-comers trial to evaluate disease activity using a broad index such as SLEDAI or BILAG scale, or enrolling patients with specific manifestations to investigate reduction of those symptoms (such as kidney function in lupus nephritis). Further challenges in study design include choosing whether to enroll patients during a lupus flare and measure improvement, or attract patients during a remission and study time to next flare.

A-001 In Sickle Cell Disease Represents Upside

A-001 is an intravenously administered, broad-spectrum inhibitor of sPLA2, including forms IIa, V and X. A-001 is currently being evaluated in a Phase II clinical study for the prevention of acute chest syndrome associated with sickle cell disease in at-risk patients. Phase II data likely will be published in Q1:11. Scientific evidence implicates sPLA 2 activity in the development of acute chest syndrome associated with sickle cell disease, as well as other forms of acute lung injury. FDA has granted A-001 orphan drug and fast-track designation. Anthera is unlikely to advance a 200-patient Phase III trial on its own and we have limited data to support A-001's effectiveness in reducing acute chest syndrome. We therefore do not forecast any revenues associated with A-001.

ANTHERA - ESTIMATED QUARTERLY P&L BUILDUP ($MM)

	2009	2010					2011				
	Total	Q1	Q2	Q3	Q4E	Total	Q1E	Q2E	Q3E	Q4E	Total
Total Revenue	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**
% Change	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**
Cost of Goods Sold	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Gross Profit	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Gross Margin	NM	0.0%	0.0%	0.0%	0.0%	NM	0.0%	0.0%	0.0%	0.0%	NM
R&D	$8.4	$5.2	$6.4	$6.9	$9.0	$27.6	$10.0	$9.0	$7.0	$7.0	$33.0
% Revenues	nm	nm	nm	nm	nm	nm	nm	nm	nm	nm	nm
SG&A	$3.4	$1.2	$1.5	$1.5	$1.5	$5.7	$2.0	$2.0	$2.0	$2.0	$8.0
% Revenues	nm	nm	nm	nm	nm	nm	nm	nm	nm	nm	nm
Total Operating Expenses	$11.8	$6.5	$7.9	$8.4	$10.5	$33.3	$12.0	$11.0	$9.0	$9.0	$41.0
% Revenues	nm	nm	nm	nm	nm	nm	nm	nm	nm	nm	nm
Operating Income	**($11.8)**	**($6.5)**	**($7.9)**	**($8.4)**	**($10.5)**	**($33.3)**	**($12.0)**	**($11.0)**	**($9.0)**	**($9.0)**	**($41.0)**
% Revenues	**nm**	**nm**	**nm**	**nm**	**nm**	**nm**	**nm**	**nm**	**nm**	**nm**	**nm**
Total Non-Operating Income	($0.4)	($4.64)	$0.03	$0.03	$0.03	($4.6)	$0.02	$0.03	$0.03	$0.03	$0.1
Pretax Income	($12.2)	($11.1)	($7.9)	($8.4)	($10.5)	($37.9)	($12.0)	($11.0)	($9.0)	($9.0)	($40.9)
Taxes	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Tax Rate	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Net Income - Operations	**($12.2)**	**($11.1)**	**($7.9)**	**($8.4)**	**($10.5)**	**($37.9)**	**($12.0)**	**($11.0)**	**($9.0)**	**($9.0)**	**($40.9)**
% Change	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
Extraordinary Items	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Diluted EPS -Operations	**($8.06)**	**($0.83)**	**($0.36)**	**($0.36)**	**($0.27)**	**($1.82)**	**($0.30)**	**($0.27)**	**($0.22)**	**($0.22)**	**($1.01)**
	NM	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**	**NM**
Diluted EPS - Reported	($8.06)	($0.83)	($0.36)	($0.36)	($0.27)	($1.82)	($0.30)	($0.27)	($0.22)	($0.22)	($1.01)
Shares (MM) - Diluted	1.5	13.3	22.2	23.0	38.9	38.9	40.0	40.0	40.0	40.0	40.0

Cash	**$14.3**	**$56.7**	**$52.1**	**$51.2**	**$63.9**	**$63.9**	**$53.5**	**$92.5**	**$82.0**	**$72.0**	**$72.0**
Cash/Share	**$9.45**	**$4.25**	**$2.34**	**$2.23**	**$1.64**	**$1.64**	**$1.34**	**$2.31**	**$2.05**	**$1.80**	**$1.80**

Source: Cowen and Company estimates; Company data

ANTHERA - ESTIMATED 2009-2020 P&L BUILDUP ($MM)

	2009	2010E	2011E	2012E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	Comments
A-002 Sales ACS U.S. ($MM)				$0.0	$132.5	$273.0	$562.4	$724.1	$895.0	$1,075.5	$1,107.7	$652.0	- Anthera to commercialize in U.S.; launch Q1:13; U.S. patent expiration Q1:2020
A-002 Sales ACS Ex-U.S. ($MM)				0.0	22.4	230.4	355.9	488.8	629.3	648.2	667.6	687.6	- Assume partner for ex-U.S. commercialization; assumes 10 year exclusivity post 2013 launch
A-623 Sales WW ($MM)				0.0	0.0	58.5	117.0	175.5	292.5	585.0	585.0	585.0	- Assumes out-licensed in 2012
Total Product Sales				**$0.0**	**$154.9**	**$561.9**	**$1,035.3**	**$1,388.4**	**$1,816.8**	**$2,308.6**	**$2,360.4**	**$1,924.6**	
A-002 Royalties ($MM)				0.0	6.7	69.1	106.8	146.6	188.8	194.5	200.3	206.3	
A-623 Royalties ($MM)				0.0	0.0	14.6	29.3	43.9	73.1	146.3	146.3	146.3	
Total Product Royalties				**$0.0**	**$6.7**	**$83.7**	**$136.0**	**$190.5**	**$261.9**	**$340.7**	**$346.5**	**$352.5**	
A-002 Milestones				50.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	- Assumes ex-U.S. partnership milestone
A-623 Milestones				30.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	
Total Milestones				**$80.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	
Total Anthera Revenues	**$0.0**	**$0.0**	**$0.0**	**$80.0**	**$139.2**	**$356.7**	**$698.4**	**$914.6**	**$1,156.9**	**$1,416.2**	**$1,454.3**	**$1,004.5**	
% Change	NM	NM	NM	NM	NM	+156%	+96%	+31%	+26%	+22%	+3%	-31%	
Cost of Goods Sold	$0.0	$0.0	$0.0	$16.0	$27.8	$71.3	$139.7	$182.9	$231.4	$283.2	$290.9	$200.9	
Gross Profit	**$0.0**	**$0.0**	**$0.0**	**$64.0**	**$111.4**	**$285.4**	**$558.7**	**$731.7**	**$925.5**	**$1,132.9**	**$1,163.4**	**$803.6**	
Gross Margin		nm	nm	80.0%	80.0%	80.0%	80.0%	80.0%	80.0%	80.0%	80.0%	80.0%	- Inclusive of a 20% blended royalty
R&D	$8.4	$27.6	$33.0	$55.0	$57.0	$40.0	$40.0	$40.0	$25.0	$25.0	$25.0	$25.0	- Assumes label expansion into CVD; Phase II for A-623; milestone to LLY/Shionogi and Amgen
% Revenues	nm	nm	nm	nm	40.9%	11.2%	5.7%	4.4%	2.2%	1.8%	1.7%	2.5%	
SG&A	$3.4	$5.7	$8.0	$76.0	$140.0	$150.0	$160.0	$170.0	$180.0	$190.0	$175.0	$100.0	- Assumes build of U.S. sales force, A-002 launch costs & marketing expenses
% Revenues	nm	nm	nm	nm	100.5%	42.0%	22.9%	18.6%	15.6%	13.4%	12.0%	10.0%	
Total Operating Expenses	**$11.8**	**$33.3**	**$41.0**	**$131.0**	**$197.0**	**$190.0**	**$200.0**	**$210.0**	**$205.0**	**$215.0**	**$200.0**	**$125.0**	
% Growth	nm	+181%	+23%	+220%	+50%	-4%	+5%	+5%	-2%	+5%	-7%	-38%	
% Revenues	nm	nm	nm	163.8%	141.5%	53.3%	28.6%	23.0%	17.7%	15.2%	13.8%	12.4%	
Operating Income/(Loss)	**($11.8)**	**($33.3)**	**($41.0)**	**($67.0)**	**($85.6)**	**$95.4**	**$358.7**	**$521.7**	**$720.5**	**$917.9**	**$963.4**	**$678.6**	
% Growth	nm	nm	nm	nm	nm	nm	nm	+45%	+38%	+27%	+5%	-30%	
% Revenues	nm	nm	nm	nm	nm	nm	51.4%	57.0%	62.3%	64.8%	66.2%	67.6%	
Non-Operating Income	($0.4)	($4.56)	$0.10	$0.10	$0.10	$0.10	$5.00	$10.00	$15.00	$20.00	$30.00	$40.00	- Assumes interest income increase as cash builds
Pretax Income	($12.2)	($37.9)	($40.9)	($66.9)	($85.5)	$95.5	$363.7	$531.7	$735.5	$937.9	$993.4	$718.6	
% Revenues	nm	nm	nm	nm	nm	27%	52%	58.1%	63.6%	66.2%	68.3%	71.5%	
Taxes	$0.0	$0.0	$0.0	$0.0	$0.0	$2.9	$109.1	$186.1	$257.4	$328.3	$347.7	$251.5	
Tax Rate	0.0%	0.0%	0.0%	0.0%	0.0%	3.0%	30.0%	35.0%	35.0%	35.0%	35.0%	35.0%	
Extraordinary Items	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	
Net Income - Operations	**($12.2)**	**($37.9)**	**($40.9)**	**($66.9)**	**($85.5)**	**$92.6**	**$254.6**	**$345.6**	**$478.1**	**$609.7**	**$645.7**	**$467.1**	
% Growth	nm	nm	nm	nm	nm	nm	nm	nm	nm	nm	nm	nm	
% Revenues	nm	nm	nm	nm	nm	nm	36.5%	37.8%	41.3%	43.0%	44.4%	46.5%	
Net Income - Reported	**($12.2)**	**($37.9)**	**($40.9)**	**($66.9)**	**($85.5)**	**$92.6**	**$254.6**	**$345.6**	**$478.1**	**$609.7**	**$645.7**	**$467.1**	
EPS -Operations	**($8.06)**	**($1.82)**	**($1.01)**	**($1.67)**	**($2.14)**	**$2.32**	**$6.37**	**$8.64**	**$11.95**	**$15.24**	**$16.14**	**$11.68**	
% Change	nm	nm	nm	nm	nm	nm	+175%	+36%	+38%	+28%	+6%	-28%	
EPS - Reported	**($8.06)**	**($1.82)**	**($1.01)**	**($1.67)**	**($2.14)**	**$2.32**	**$6.37**	**$8.64**	**$11.95**	**$15.24**	**$16.14**	**$11.68**	
Shares (MM) - Diluted	1.5	38.9	40.0	40.0	40.0	40.0	40.0	40.0	40.0	40.0	40.0	40.0	- Assumes financing in Q2:11 of $50MM at $9/share

Source: Cowen and Company estimates; Company data

Anthera R&D Pipeline

Therapeutic Class/Product	Indication	I	II	III	FILING	MKT	Comments
Cardiovascular							
Varespladib (A-002)	Acute coronary syndrome			•			Phase III (VISTA-16) enrolling; interim data H1:11
Inflammatory							
A-623	SLE		•				BLyS mAb; Phase II study on hold
A-001	Acute Chest Syndrome		•				Sickle cell disease; Phase II data Q1:11
Total Drugs In Development		**0**	**2**	**1**	**0**	**0**	

Hayward, CA **Investor Relations Contact: Julianne Snowden (212) 213-0006**

Source: Company data

Addendum

STOCKS MENTIONED IN IMPORTANT DISCLOSURES

Ticker	Company Name
ANTH	Anthera Pharmaceuticals

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Rating	Definition
Outperform (1)	Stock expected to outperform the S&P 500
Neutral (2)	Stock expected to perform in line with the S&P 500
Underperform (3)	Stock expected to underperform the S&P 500

(a) Assumptions: Time horizon is 12 months; S&P 500 is flat over forecast period.

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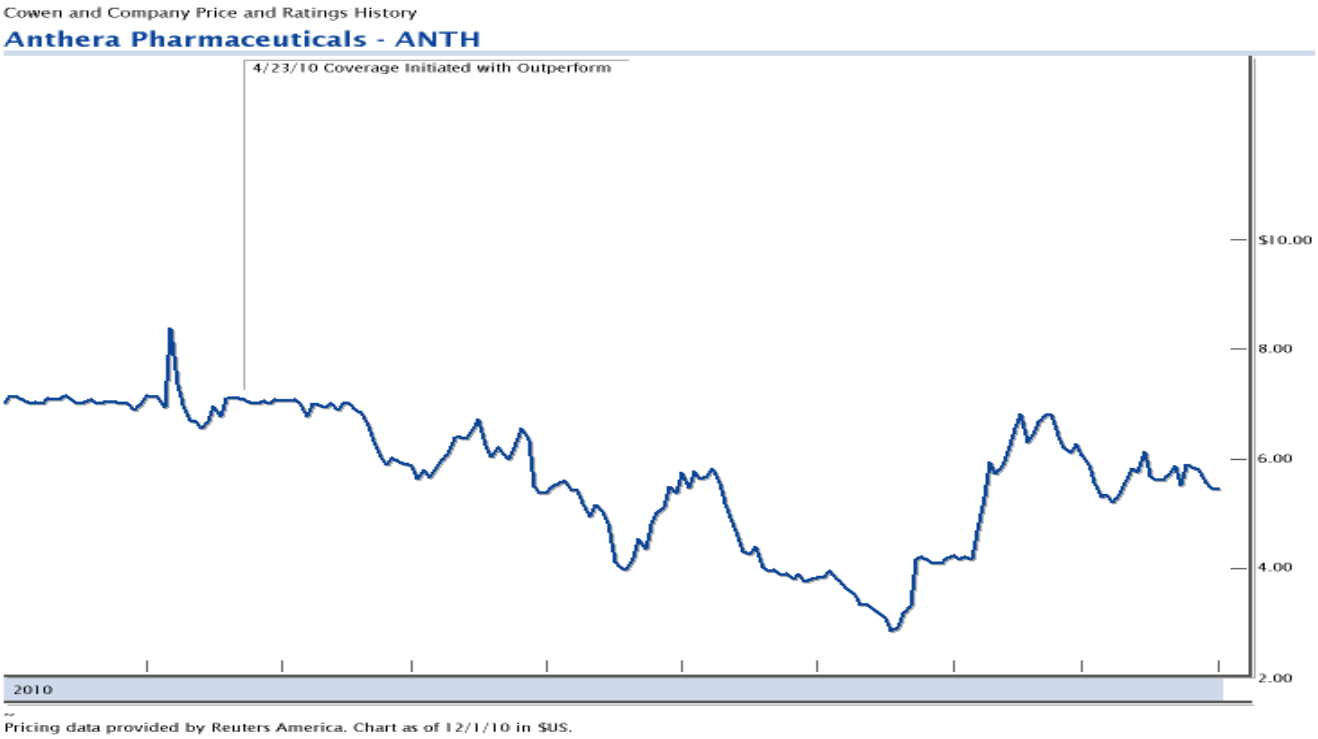
Rating	Pct of companies under coverage with this rating	Pct for which Investment Banking services have been provided within the past 12 months
Buy (b)	48.7%	3.8%
Hold (c)	47.7%	1.3%
Sell (d)	3.6%	0.0%

(a) As of 09/30/2010. (b) Corresponds to "Outperform" rated stocks as defined in Cowen and Company, LLC's rating definitions (see above). (c) Corresponds to "Neutral" as defined in Cowen and Company, LLC's ratings definitions (see above). (d) Corresponds to "Underperform" as defined in Cowen and Company, LLC's ratings definitions (see above). Note: "Buy," "Hold" and "Sell" are not terms that Cowen and Company, LLC uses in its ratings system and should not be construed as investment options. Rather, these ratings terms are used illustratively to comply with NASD and NYSE regulations.


Cowen and Company Price and Ratings History
Anthera Pharmaceuticals - ANTH
4/23/10 Coverage Initiated with Outperform
$10.00
8.00
6.00
4.00
2.00
2010
Pricing data provided by Reuters America. Chart as of 12/1/10 in $US.