Edward A. Tenthoff, Sr Research Analyst
212 284-9403, edward.a.tenthoff@pjc.com
Piper Jaffray & Co.
Chad J. Messer, Ph.D., Research Analyst
212 284-9326, chad.j.messer@pjc.com
Piper Jaffray & Co.

Reason for Report:
Initiating Coverage

Changes	Previous	Current
Rating	--	Overweight
Price Tgt	--	$12.00
FY10E Rev (mil)	--	$0.0
FY11E Rev (mil)	--	$0.0
FY10E EPS	--	($1.57)
FY11E EPS	--	($2.01)

Price:	$6.99
52 Week High:	$8.55
52 Week Low:	$6.86
12-Month Price Target:	$12.00
Proj EV of $$288M + mid'11E cash	
Shares Out (mil):	22.2
Incl recent IPO	
Market Cap. (mil):	$155.2
Avg Daily Vol (000):	67
Book Value/Share:	$2.49
Cash Per Share:	$2.90
Debt to Total Capital:	0%
Est LT EPS Growth:	NA
P/E to LT EPS Growth (FY10):	NA
Est Next Rep Date:	08/13/2010
Fiscal Year End:	Dec
Cash/sh incl recent IPO	

Rev (mil)	2009A	2010E	2011E
Mar	$0.0A	$0.0E	$0.0E
Jun	$0.0A	$0.0E	$0.0E
Sep	$0.0A	$0.0E	$0.0E
Dec	$0.0A	$0.0E	$0.0E
FY	$0.0A	$0.0E	$0.0E
CY	$0.0A	$0.0E	$0.0E
FY RM	NM	NM	NM
CY RM	NM	NM	NM

EPS	2009A	2010E	2011E
Mar	($0.34)A	($0.21)E	($0.59)E
Jun	($0.30)A	($0.24)E	($0.50)E
Sep	($0.22)A	($0.44)E	($0.42)E
Dec	($0.09)A	($0.64)E	($0.50)E
FY	($1.24)A	($1.57)E	($2.01)E
CY	($1.24)A	($1.57)E	($2.01)E
FY P/E	NM	NM	NM
CY P/E	NM	NM	NM

Quarterly EPS does not add to full year result

Anthera Pharmaceuticals (ANTH - $6.99)
Overweight

Promising Cardiac Inflammation and Lupus Play; Initiating w/ Overweight Rating

CONCLUSION:
Anthera is developing drugs to treat life-threatening inflammatory and autoimmune diseases. The company is preparing to initiate the Phase III VISTA-16 trial of Varespladib in Acute Coronary Syndrome (ACS) under an SPA agreement with the FDA. Anthera is also developing A-623, an injectable BLyS inhibitor ready to begin a Phase II lupus study. Following the recent successful IPO, Anthera now has cash of $64 million, sufficient to begin these potential value creating studies. We are initiating coverage of Anthera with an Overweight rating and $12 price target.

- **Phase II Biomarker Data Gives Confidence in Pivotal Study.** There is a growing body of evidence that inflammation plays an important role in cardiovascular disease (CVD) and heart attack. Varespladib is an sPLA2 inhibitor that acts by lowering cardiac inflammation. The Phase II PLASMA and FRANCIS studies showed varespladib in combination with statins reduced target sPLA2, C-Reactive Protein (CRP) and LDL cholesterol. These results give us confidence that varespladib can successfully prevent recurrent Major Adverse Coronary Events (MACE) in a larger Phase III study.

- **FDA Granted an SPA for Phase III VISTA-16 Trial.** Anthera received a Special Protocol Assessment (SPA) from the FDA for a single Phase III ACS study. VISTA-16 will compare 500mg QD Varespladib to placebo on top of *Lipitor* for 16 weeks in high CRP patients treated within 96 hours of event. VISTA-16 will enroll up to 6,500 ACS patients yielding an expected 385 events powered to show a 25% improvement in MACE. We expect VISTA-16 will begin this summer with data in late 2011 or early 2012. If successful, we anticipate U.S. launch in 2013 with U.S. and EU sales of $830 million in 2020E.

- **A-623 Potential Blockbuster in Lupus.** Based on the success of Human Genome Sciences' *Benlysta*, Anthera has accelerated development of A-623. This peptibody also targets BLyS, but is an injectable drug versus Benlysta's monthly i.v. infusion. Anthera intends to initiate a Phase II lupus study in 3Q:10 providing a data milestone next year for potential value appreciation.

- **IPO Brings in Sufficient Cash to Begin Studies.** Following the recent successful IPO, Anthera holds *pro forma* cash of $64 million, sufficient to begin these studies. We expect Anthera to either partner A-623 or varespladib overseas or raise additional capital in order to complete the studies.

INVESTMENT RECOMMENDATION:
We are initiating coverage of Anthera with an Overweight rating and $12 price target. We value varespladib at $288 million by applying a standard 5x multiple to 2015 U.S. sales of $307 million discounted back at 45% annually. To this we add mid'11E cash. At present, we assign no value for European sales of varespladib or A-623 in lupus providing potential upside.

RISKS TO ACHIEVEMENT OF TARGET PRICE:
Risks include clinical, regulatory and commercial. Varespladib and/or A-623 may fail in the clinic. Anthera may require additional cash from the capital markets.

COMPANY DESCRIPTION:
Anthera is a biopharmaceutical company developing varespladib and A-623.

PROMISING CARDIAC INFLAMMATION AND LUPUS PLAY

Anthera is a biopharmaceutical company developing novel drugs to treat life–threatening inflammatory and autoimmune diseases. The company's lead drug, *varespladib*, is a first-in-class secretory phospholipase A2 (sPLA2) inhibitor. sPLA2 is a family of enzymes that cause inflammation. Varespladib works through a dual mechanism by reducing cardiac inflammation and LDL cholesterol (LDLc). The Phase II PLASMA and FRANCIS studies showed varespladib in combination with statins reduced target sPLA2, C-Reactive Protein (CRP) and LDLc. We see varespladib as a novel cardiovascular agent that may lower Major Adverse Coronary Events (MACE) in patients with coronary artery disease (CAD).

Under a Special Protocol Assessment (SPA), Anthera is preparing to initiate the Phase III VISTA-16 trial of varespladib in Acute Coronary Syndrome (ACS) patients. This single 16-week pivotal study will compare 500mg once daily (QD) Varespladib to placebo on top of *Lipitor* (atorvastatin) in high CRP patients (>10 mg/L) treated within 96 hours of event. The VISTA-16 trial will enroll up to 6,500 ACS patients yielding an expected 385 events powered to show a 25% improvement in MACE. We expect VISTA-16 will begin this summer with data in late 2011 or early 2012. There is an interim biomarker futility analysis after the first 1,000 patients have completed 16 weeks of therapy, likely to take place in 1H:11, which could reassure investors on the progress of the pivotal study. If VISTA-16 is successful, we anticipate Anthera will partner varespladib for significant financial terms. We anticipate U.S. launch in 2013 with ACS sales reaching $830 million in 2020E. Ultimately we foresee blockbuster opportunity for chronic varespladib in CAD.

Anthera is also developing A-623 for lupus, a peptibody that targets BLyS. Based on the success of Human Genome Sciences' (HGSI) *Benlysta*, Anthera has accelerated development of A-623. A-623 is an injectable drug versus Benlysta's monthly i.v. infusion. Anthera intends to initiate Phase II studies in 3Q:10 providing a data milestone for potential value appreciation by mid-2011. If successful, A-623 represents a potential blockbuster drug with potential applicability beyond lupus.

We are initiating coverage of Anthera with an Overweight rating and $12 price target. Anthera recently raised over $60 million in net proceeds in a successful IPO priced at $7 per share including insider participation and exercise of a partial green shoe. We look for Anthera to create value by reporting Phase II A-623 lupus data in 2011 and Phase III varespladib ACS data by early 2012, as well as potentially partnering either drug in the interim. We value varespladib at $288 million by applying a standard 5x multiple to 2015 U.S. sales of $307 million discounted back at 45% annually. To this we add mid'11E cash of $3 million. At present, we assign no value for European varespladib sales or A-623 in lupus providing potential upside.

Expected Upcoming Events:
- Initiate Phase III trial of varespladib in ACS under an SPA this summer
- Commence Phase II study of A-623 in lupus in 3Q:10
- DSMB to conduct interim futility analysis after first 1,000 patients in ACS pivotal study likely in 1H:11
- Report Phase II A-623 lupus data by mid-2011
- Potentially partner either European rights to varespladib or A-623 retaining significant value
- Report Phase III varespladib ACS data in late 2011 or early 2012
- Potentially file an NDA for varespladib by mid-2012 with U.S. launch in 2013

ACUTE CORONARY SYNDROME

Acute Coronary Syndrome (ACS) encompasses a group of clinical conditions including unstable angina, ST segment elevation myocardial infarction (STEMI) or heart attack, and non-ST segment elevation myocardial infarction (NSTEMI). These life-threatening disorders are caused by coronary artery disease (CAD), which is insufficient blood flow to the heart primarily due to atherosclerosis.

Atherosclerosis is a complex lipid driven disease that has significant inflammatory and immunological inputs. A critical first step in plaque formation is the deposition and oxidation of low density lipoprotein cholesterol (LDLc) in the arterial vessel walls. It is for this reason that LDLc lowering therapy such as statins has become a cornerstone in the management of CAD. Oxidized LDLc particles recruit and activate leukocytes causing endothelial cell death. These dying cells secrete inflammatory cytokines and adhesion molecules that perpetuate the cycle by attracting more macrophages and further growing the atherosclerotic plaque.

If the plaque partially occludes the vessel, the lesion may go undetected until it results in symptoms of angina or chest pain. This may lead to unstable angina, cerebral or pulmonary infarction and episodes of ischemia. If the plaque fully occludes the artery or ruptures causing a blockage, the downstream cardiac tissue will no longer receive oxygen resulting in a myocardial infarction (MI) or heart attack.

THE GROWING EVIDENCE OF INFLAMMATION IN CARDIOVASCULAR DISEASE

There are 2 types of validated cardiac biomarkers. Biomarkers that reflect cardiac injury include Troponin, CK-MB-2 isomers and myoglobin. In patients presenting within 6 hours of onset of symptoms troponin, CK-MB-2 isoform and myoglobin are elevated. Troponin T (cTnT) and I (cTnI), along with CK-MB-2 (mass assay) are reliable markers of cardiac muscle injury and tissue necrosis. Troponin release is similar to that of CK-MB, but remains elevated longer (~7 to 10 days) after an ACS event. Cardiac inflammatory biomarkers include Interleukin-6 (IL-6), C-reactive protein (CRP), secretory phospholipase A2 (sPLA2) and lipoprotein associated phospholipase A2 (LpPLA2).

The JUPITER (**J**ustification for the **U**se of Statins in **P**revention: an **I**ntervention **T**rial **E**valuating **R**osuvastatin) trial published in the *New England Journal of Medicine* in November 2008 demonstrated the importance of lowering inflammation to prevent CVD. This landmark study randomized 17,802 healthy individuals with "normal" LDLc levels (<130mg/dL) but elevated high sensitivity CRP (>2mg/L) to either 20mg *Crestor* (rosuvastatin) or placebo. Importantly, Crestor reduced LDLc levels by 50% and hsCRP levels by 37%. JUPITER was stopped early because of a dramatic reduction in events. Specifically, Crestor showed a 43% reduction in the incidence of MACE with 0.77 events per 100 person years of follow-up vs. 1.36 events on placebo (HR 0.56, 95% confidence interval 0.46 to 0.69; p<0.00001). In our view the JUPITER study proves the importance of preventing inflammation in CVD and supports the mechanism and use of veraspladib.

Interleukin-6 (IL-6)

IL-6 is a cytokine and an inflammatory intercellular mediator. IL-6 is secreted by T cells and macrophages to stimulate immune response to trauma. IL-6 is also a "myokine," a cytokine produced by muscle, and is elevated in response to muscle contraction. Most significantly IL-6 is thought to promote the hepatocyte production of CRP, which is then secreted into systemic circulation. IL-6 plasma concentrations reflect the integrity and the vulnerability of plaque to rupture and restenosis following percutaneous coronary intervention (PCI). IL-6 is involved in the pathogenesis of the ACS by stimulating the linear production of clotting factors such as fibrinogen and CRP. Further, IL-6 stimulates macrophages to produce tissue factor and matrix metalloproteinases, platelet aggregation, adhesion molecules, tumor necrosis factor (TNF) and vascular smooth muscle cell proliferation. Elevation of circulating IL-6 is a strong and independent marker of increased mortality with acute coronary events. IL-6 predicts future heart attacks in healthy men as well as total mortality in the elderly *(Arnaud et al, Arterioscler Thromb Vasc Biol 2005).*

C-Reactive protein (CRP)

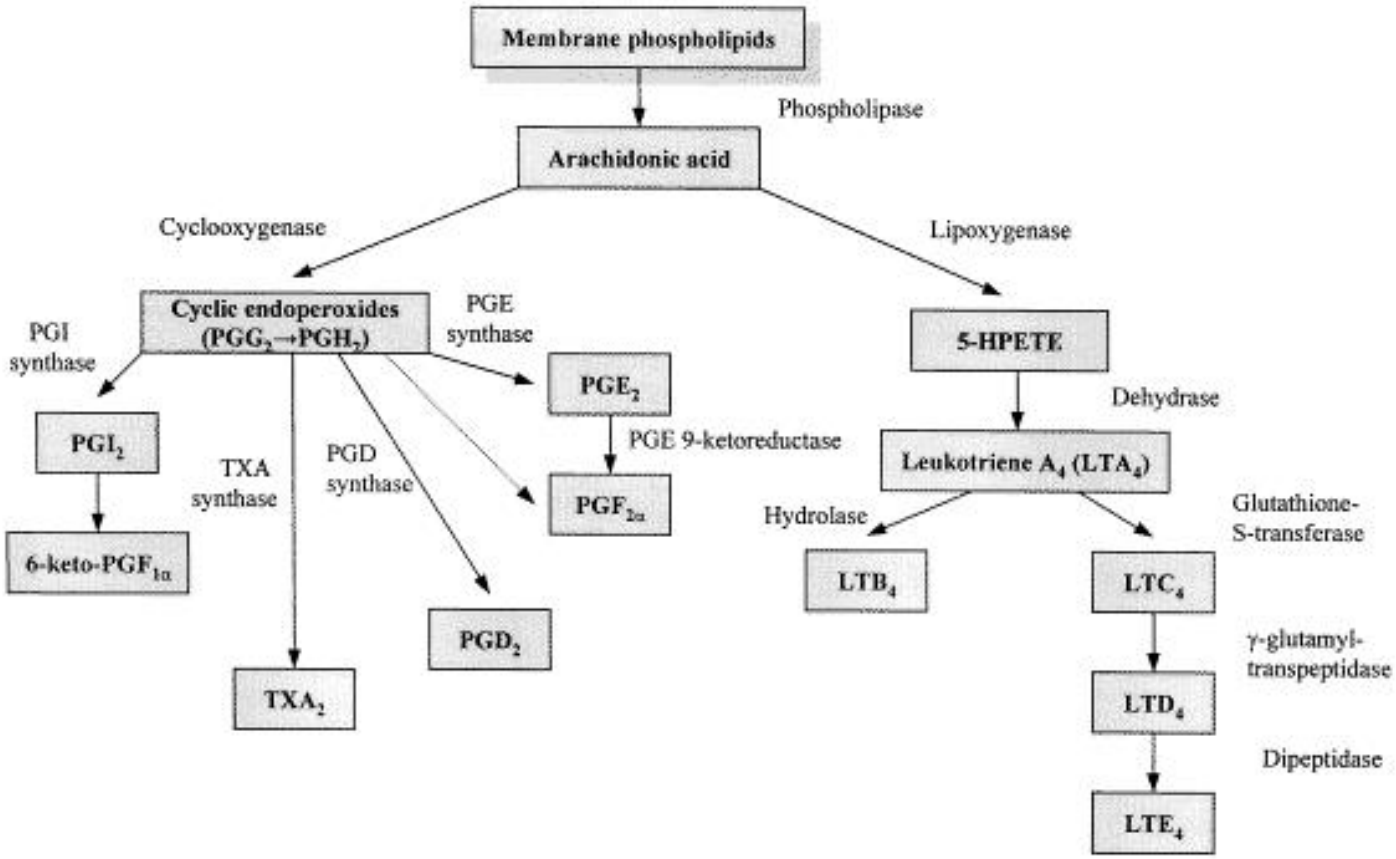
CRP is an important biomarker in CVD and ACS settings indicating the onset of an inflammatory process. CRP is an acute-phase reactant, whose levels rise dramatically with acute and chronic inflammation throughout the body. CRP rises above normal limits within 6 hours and peaks at 48 hours. The half-life of CRP is constant, and therefore plasma levels are mainly determined by the rate of production, which is dependent upon the severity of the precipitating cause. The CRP rise is thought to be correlated to a rise in plasma concentration of IL-6.

CRP is identified for predicting atherothrombotic events in apparently healthy individuals and for adding predictive value at all levels of risk based on Framingham score. CRP levels can also help grade the presence and severity of ACS. hsCRP has demonstrated a strong correlation as an independent risk factor for future cardiac events. Randomized controlled trials have demonstrated that CRP may play a direct role in atherothrombosis and that hsCRP values less than 0.5 mg/L may suggest decreased cardiovascular risk and absence may be protective. (*Riker et al Circulation, 2004*) Higher CRP levels (>10 mg/L) correlate with an increased risk of a cardiovascular event and may reflect inflammatory process in patients. Based on the Centers for Disease Control/AHA recommendations, the risk adjusted cut off points for hsCRP are <1.0 mg/L for low risk, 1-3 mg/L for average risk, and >3.0 mg/L for high risk. Importantly existing data supports the predictive value of hsCRP of new coronary events in patients with ACS independent of troponin T and helps in the risk stratification of patients with established CVD.

Secretory Phospholipase A2 (sPLA2)

Phospholipases (PLA2) are a class of enzymes that break down phospholipids into fatty acids. One such fatty acid is arachidonic acid. The arachidonic acid pathway mediates an inflammatory cascade that causes cells and tissue damage. (Please see Figure 1 below.)

Figure 1: ARACHIDONIC ACID PATHWAY



Source: Drugs Today 1998

The sPLA2 family of enzymes include at least three extracellular forms (IIa, V and X) that play a major role in inflammation. Specifically, $sPLA_2$ increases the ability of LDLc to aggregate, oxidize, and promote plaque formation. In humans plasma sPLA2 levels are higher in patients with increased risk of CAD. In patients with unstable CAD, sPLA2 levels correspond to recurring events after an initial acute coronary event. Mallat et al evaluated the relationship between sPLA2 activity and CAD in healthy individuals in the EPIC-NORFALK study. They showed that the combined measurement of sPLA2 activity and CRP allowed a better assessment of the risk of incident CAD than either biomarker alone.

In ACS patients, excess sPLA2 has both acute and chronic implications on disease progression and patient outcomes. Shortly after an acute coronary event, sPLA2 levels increase significantly within the first 24 hours through 2 weeks and remain elevated for up to an additional 12 weeks. This heightened cardiac inflammation can have several adverse implications including further destabilization of atherosclerotic plaques and death of cells on the margin. sPLA2 also adversely modifies lipids to smaller more pro-thrombotic fragment that may lead to the recurrence of a MACE.

In a study by Lima et al investigating the correlation between the sPLA2 activity with atheromatosis in subjects with CAD undergoing coronary angiography, plasma sPLA2 activity was significantly higher in subjects with severe and mild/moderate atheromatosis than in controls. Importantly in a multiple logistic regression model, adjusted for age, gender, body mass index, hypertension, Quality of life, family history for CAD, diabetes mellitus, total cholesterol, HDLc, LDLc, triglycerides, hsCRP and PLA2, **only sPLA2 was observed to be independently associated with severe CAD (70% of stenosis) ($p<0.0001$).** (Please see Figure 2)

Figure 2: CAUSATIVE FACTORS OF CORONARY ARTERY DISEASE

Variable	Odds-ratio (95% confidence interval)	*p*-value
Gender	0.740 (0.286–1.914)	0.534
Age (years)	0.667 (0.453–2.169)	0.141
Body mass index (kg/m^2)	0.981 (0.383–2.511)	0.968
Smoking	0.466 (0.173–1.253)	0.079
Hypertension	0.726 (0.203–2.600)	0.622
Sedentarism	1.894 (0.597–6.007)	0.278
Family history for CAD	0.841 (0.344–2.056)	0.705
Diabetes mellitus	0.986 (0.282–3.453)	0.983
Total cholesterol (mmol/l)	0.995 (0.985–1.005)	0.304
LDL cholesterol (mmol/l)	1.034 (0.984–1.087)	0.184
Triglycerides (mmol/l)	1.002 (0.995–1.009)	0.626
sPLA2 (U/ml)	10.781 (3.270–35.547)	<0.0001
hs-CRP (g/dl)	0.997 (0.953–1.043)	0.903

Source: Thromb Thrombolysis May 2009

Higher plasma sPLA2 activity and hsCRP levels are associated with CAD. In addition, there is a correlation between higher levels of both sPLA2 and CRP and the degree of CAD. Importantly, only sPLA2 activity was significantly higher in subjects with one or two affected vessels and was shown to be independently associated with CAD. These results confirm findings of recent studies that indicated an independent association of PLA2 with CAD.

Lipoprotein Associated Phospholipase A2 (LpPLA2)

LpPLA2 is related to the secretory form; however, differs in subtle but important ways. LpPLA2 is primarily bound to LDLc and high-density lipoprotein cholesterol (HDLc). LpPLA2 is highly expressed in the necrotic core of atherosclerotic lesions. Epidemiological studies have shown that LpPLA2 levels can predict future cardiovascular events. In a study involving 466 patients with stable coronary heart disease patients with higher LpPLA2 levels were associated with a greater risk.

GlaxoSmithKline (GSK) and HGS are developing *Darapladib*, which targets LpPLA2. In March 2008, GSK presented positive results from a 959 patient Phase II trial with heart disease or equivalent CV risk to 20mg or 80mg Lipitor. At 12 weeks, darapladib showed dose-dependent reductions in LpPLA2 levels to 43 nmol/min/ml with the 160mg darapladib dose, 56 nmol/min/ml with the 80mg dose and 68 nmol/min/ml with the 40mg dose versus 124 nmol/min/ml with placebo ($p<0.001$, all doses.) While not powered to show a reduction, cardiovascular events were 1% in 160mg group and <1% in the 80 and 40 mg groups versus 2% in placebo.

Darapladib is currently being investigated in 2 large Phase III outcomes studies. This STABILITY study began enrollment in December 2008. Over 15,000 patients are being randomized (1:1) to 160mg QD darapladib or placebo. The primary endpoint is MACE and the study will be stopped when 1,500 events have been reported. GSK has guided that data should be available in 2012. Secondary endpoints in the study include individual components of MACE, death, non-fatal MI, coronary revascularization, unstable angina, and all-cause mortality.

The SOLID TIMI 52 study was initiated in December 2009 and will enroll 11,500 patients who have had ACS within the last 30 days. Patients will receive 160mg QD darapladib or placebo on top of standard of care. The primary endpoint is secondary MACE and this study will also be stopped at 1,500 events. GSK has guided that data should be available in 2013.

CURRENT TREATMENT PARADIGM OF ACUTE CORONARY SYNDROME

When an individual presents at a hospital ER with symptoms, standard ACS guidelines dictate treatment. Aggressive early pharmacologic intervention has been proven to save lives and prevent recurrent MACE. Upon classification via cardiac biomarkers and electrocardiogram, patients are typically treated with anti-platelet therapy including aspirin (300mg) and often Plavix (clopidogrel) in patients with STEMI and NSTEMI. Heparin and GPIIb/IIIa antagonists such as ReoPro, Integrilin or Aggrastat play a role. Depending upon diagnosis, Beta blockers and angiotension-converting enzyme (ACE) inhibitors are initiated. As a result of clinical evidence, statin therapy to lower LDL is today standard to prevent recurrent event. (Please see Figure 3 below.)

Figure 3: EARLY PHARMACOLOGIC INTERVENTION GUIDELINES FOR ACS

Antiplatelet Therapy:
A - Following an acute coronary syndrome all patients should be maintained on long term aspirin therapy.
B - In addition to long term aspirin, clopidogrel therapy should be continued for three months in patients with non-ST elevation acute coronary syndromes.
A - In addition to long term aspirin, clopidogrel therapy should be continued for up to four weeks in patients with ST elevation acute coronary syndromes.

Statin Therapy:
B - Patients with an acute coronary syndrome should be commenced on long term statin therapy prior to hospital discharge.
Beta-Blocker and Antianginal Therapy:
C - Patients with unstable angina or evidence of myocyte necrosis should be maintained on long term beta-blocker therapy.
A - Patients with clinical myocardial infarction should be maintained on long term beta-blocker therapy.

Angiotensin-Converting Enzyme (ACE) Inhibitors:
B - Patients with unstable angina or myocyte necrosis should be commenced on long term angiotensin-converting enzyme inhibitor therapy.
A - Patients with clinical myocardial infarction should be commenced on long term angiotensin-converting enzyme inhibitor therapy within the first 36 hours.

Angiotensin Receptor Blockers:
A - Patients with clinical myocardial infarction complicated by left ventricular dysfunction or heart failure should be commenced on long term angiotensin receptor blocker therapy if they are intolerant of angiotensin converting-enzyme inhibitor therapy.

Aldosterone Receptor Antagonists:
B - Patients with clinical myocardial infarction complicated by left ventricular dysfunction (*ejection fraction <0.40*) in the presence of either clinical signs of heart failure or diabetes mellitus should be commenced on long term eplerenone therapy.
Source: www.guideline.gov
Note: A, B, C indicate Grades of Recommendation.

Statins play a significant role in ACS therapy and have been incorporated into standard guidelines. By inhibiting the cholesterol synthesis in the liver and increasing clearance of LDLc, statins are thought to reduce the lipid core and stabilize the plaque.

In our view ACS represents a significant market opportunity for cardiac anti-inflammatory agents. We believe veraspladib could be added to current standard of care to "cool" down the heart post-MI and reduce recurrence of secondary MACE.

VARESPLADIB (A-002)

Varespladib is a first-in-class oral sPLA2 inhibitor. Varespladib has a dual mechanism of action that both reduces cardiac inflammation as well as LDLc, total cholesterol and non-HDLc. Specifically varespladib lowers pro-atherogenic LDLc particles. This is an important point of differentiation from LpPLA2 inhibitors like Darapladib that have not demonstrated similar effects on lipids. The effect of Varespladib on inflammation has been demonstrated in human clinical trial by a statistically significant reduction in IL-6, CRP and sPLA2. In animal studies, Varespladib has shown synergistic reductions of plaque volume when added to statins.

PLASMA-1 Study

The Phase II PLASMA (**P**hospholipase **L**evels **A**nd **S**erological **M**arkers of **A**therosclerosis) study randomized 396 stable CAD patients to 50mg, 100mg, 250mg, or 500mg BID Varespladib or placebo for 8 weeks on top of standard-of-care background therapy. 259 (65.4%) patients in the study were on statins. The primary endpoint was a change in sPLA2 with secondary endpoints including changes in lipids, lipoprotein subclasses and other inflammatory biomarkers.

PLASMA results published in the *Lancet* in February 2009 showed Varespladib significantly reduced sPLA2 and LDLc as well as several LDLc subtypes known to cause inflammation. A dose dependent decrease in sPLA2 was observed. Across all Varespladib doses the reduction in sPLA2 was -86.7% versus only -4.8% in placebo ($p<0.0001$). A non significant reduction in CRP of -55.6% on Varespladib versus -24.8% in placebo was seen ($p=0.47$). LDLc was reduced by 9.7% versus placebo ($p=0.0035$) with those patients having the highest baseline LDLc (>70mg/dL) despite statin therapy showing an even higher 12% reduction ($p=0.0065$). Importantly, an analysis of the lipid subtypes showed the decrease in overall LDLc was driven by a shift in particle size away from smaller pro-atherogenic LDLc. Statistically significant reductions were also seen in total cholesterol and non-HDLc.

Varespladib was found to be safe and well-tolerated with no difference in adverse events (AEs) between the treatment groups and placebo. The most common AEs were headache (6.4%) and nausea (5.4%). Mild and transient increases in liver function tests of >3X upper limit of normal were observed in the Varespladib arm, although there were no observed increases in bilirubin and Hys Law criteria for liver injury were not met.

PLASMA-2 Study

The Phase II PLASMA-2 study enrolled an additional 138 patients with stable CAD to one of two once daily doses of Varespladib or placebo for 8 weeks on top of standard-of-care therapy including 123 (89.1%) patients on background statins. As in PLASMA, the primary endpoint was change in sPLA2 with lipids and other inflammatory biomarkers measured. In PLASMA-2 Varespladib treatment resulted in a -77.8% reduction in sPLA2 versus an 8.3% increase in placebo ($p<0.0001$). Other results were also consistent with PLASMA including a significant decrease of 8.3% in LDLc versus 0.7% in placebo ($p=.0.014$), as well as significant reductions in total cholesterol and non-HDLc.

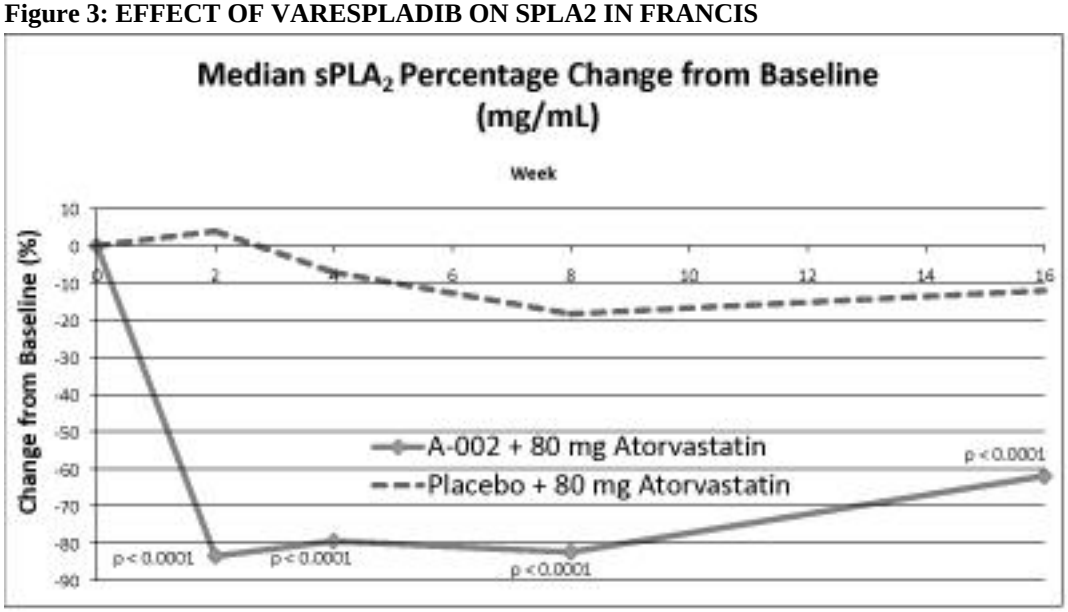
Varespladib was found to be safe and well tolerated with the most common AEs including diarrhea (6.7%), nausea (5.6%), and increases in liver enzymes (5.6%). Again, there was no raise in bilirubin and Hys law criteria were not met.

FRANCIS Study

The Phase IIb FRANCIS (**F**ewer **R**ecurrent **A**cute coronary events with **N**ear-term **C**ardiovascular **I**nflammation **S**uppression) study enrolled 625 ACS patients to 500 mg QD Varespladib or placebo on top of high dose 80mg Lipitor and any other standard-of-care therapy for 24 weeks. Patients had to be randomized within 96 hours of coronary event or hospitalization. The primary endpoint was change in LDLc after the first 500 patients completed 8 weeks of therapy. Changes in inflammatory markers such as sPLA2, CRP and IL-6 were also measured as well as secondary MACE.

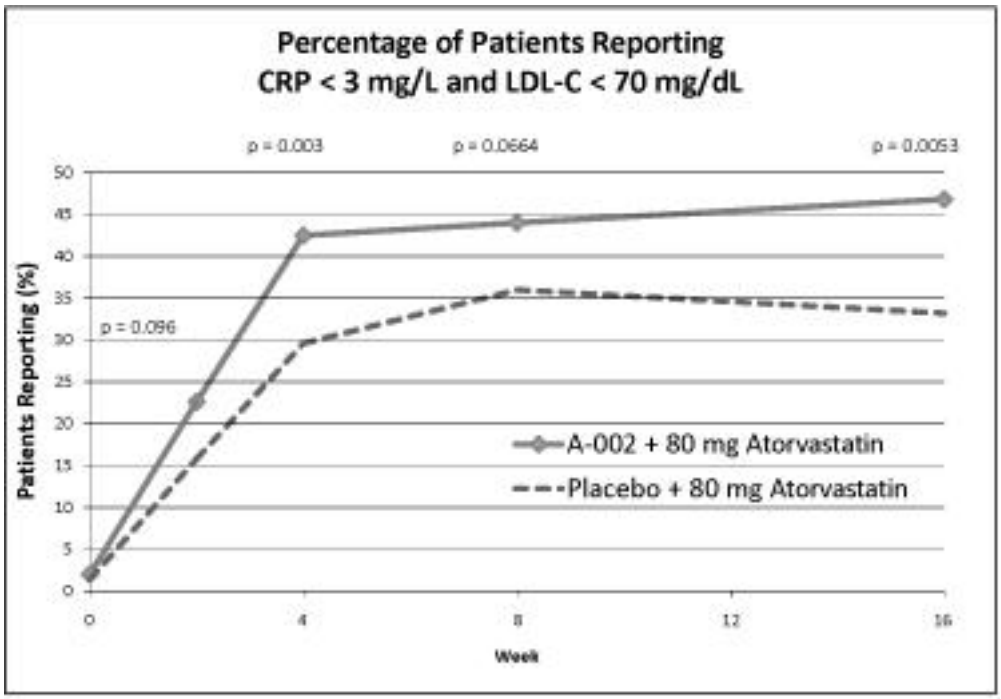
Varespladib showed a significant reduction in sPLA-2 by week 2 ($p<0.0001$) that persisted through the study (Please see Figure 3 below), as well as LDLc by week 8 (-5.7%, $p=0.0023$) and CRP by week 16 ($p=0.0067$). A significant number of patients reached their LDLc goal of <70 mg/do at week 8 ($p=0.013$) and week 16 ($p=0.0023$) and met the JUPITER study defined criteria of CRP <3mg/dL and LDLc <70mg/dL ($p=0.005$). (Please see Figure 4 below) An 18% overall reduction in MACE, a 33% reduction in unstable angina and a 50% reduction in MI were also seen with Varespladib, although the study was not powered to show these effects and did not reach significance. There was a confounding higher rate of sudden death on varespladib, but the study was too small to demonstrate significance.

Figure 3: EFFECT OF VARESPLADIB ON SPLA2 IN FRANCIS



Source: Company Reports

Figure 4: PERCENTAGE OF PATIENTS REACHING JUPITER DEFINED CRP & LDL-C IN FRANCIS



Source: Company Reports

Varespladib was found to be safe and well-tolerated with no imbalance in dropouts due to drug effects. Transient LFT increases of >3X upper limit of normal were seen at week 4 and 8 in the Varespladib group, but resolved by end of treatment. There were no increases in bilirubin and Hys law criteria were not met. Importantly, no effects on blood pressure or the cardiac QTc interval were observed.

VISTA-16 Trial

Anthera received a Special Protocol Assessment (SPA) from the FDA for a single Phase III ACS study. The VISTA-16 trial will randomize up to 6,500 ACS patients within 96 hours of event or hospitalization to either 500mg QD Varespladib or placebo for 16 weeks on top of Lipitor and background standard-of-care. The number of subjects who undergo PCI following MI and prior to randomization will be limited to <55%. Physician will determine Lipitor dose of 20mg, 40mg or 80mg QD with a one-time dose adjustment permitted after 8 weeks if LDLc remains above target 100mg/dL. The primary endpoint of the trial is reduction in MACE at week 16. All cause mortality is a key secondary endpoint. The study is expected to yield a minimum of 385 events and is powered to show a 25% improvement in MACE. We expect VISTA-16 will begin this summer with data in late 2011 or early 2012.

An independent Data Safety Monitoring Board (DSMB) will conduct safety reviews and one interim biomarker efficacy analysis. The interim analysis will occur when at least 1,000 patients have completed treatment and 50% of the number of target events has occurred, which we expect will occur in 1H:11. The DSMB may also assess futility. The survival status for all subjects who have not withdrawn consent will be measured at the end of the study and 6 months after they complete the study.

ANALYSIS OF ACS OUTCOME STUDIES

Inflammation is thought to play an important role in morbidity and mortality leading up to or following ACS. In patients with ACS several outcome studies have been published in the recent past showing benefits of intensive lipid lowering therapy with statins. The bulk of the evidence points to improved outcomes in relation to lowering of the LDLc, however we see a clear opportunity for cardiac anti-inflammatory agents to work synergistically with statins in ACS and ultimately more broadly in CVD.

The MIRACL (**M**yocardial **I**schemia **R**eduction with **A**cute **C**holesterol **L**owering) trial was published in JAMA in April 2001. A total of 3,086 ACS patients were randomized within 96 hours of event to either 80mg Lipitor or placebo. There were 228 (14.8%) MACE with Lipitor vs. 269 (17.4%) MACE on placebo (RR 0.84; 95% confidence interval 0.70-1.00; *p*=0.048). The effect was largely driven by reduction of recurrent symptomatic ischemia requiring re-hospitalization. Lipitor lowered mean LDLc 41.9% from 124 mg/dL to 72mg/dL. Subsequent analysis reported at ACC in 2002 showed that Lipitor reduced CRP 83% in 16 weeks compared to 74% on placebo. In heart attack patients, Lipitor reduced CRP by 89% vs. 84% on placebo (p<0.0001), and in unstable angina patients of 71%

vs. 55% with placebo (p<0.0001).

The **PROVE IT**-TIMI 22 (**PR**avastatin **O**r Ator**V**astatin **E**valuation and **I**nfection **T**herapy-**T**hrombolysis **I**n **M**yocardial **I**nfarction **22**) published in the *New England Journal of Medicine* in April 2004 randomized 4,162 hospitalized ACS patients to standard therapy 40mg Pravachol (pravastatin) vs. high dose 80mg Lipitor. High-dose Lipitor achieved LDLc of 62mg/dL vs. 95mg/dL on standard-dose of Pravachol (p<0.001). High-dose Lipitor proved superior with a 16% reduction in CV events at 22.4% vs. 26.3% on Pravachol (p=0.005; 95 percent confidence interval 5-26%).

VISTA is the first outcome based study in ACS primarily targeting inflammatory biomarkers along with reduction of LDLc. In Figure 5 below, we highlight five ACS outcome studies for comparison. Based on the design parameters to show a delta of 25% (HR:0.75) and an estimated 8.5% MACE event rate in the control arm, we conservatively estimate a minimum event rate of 6.38% in the varespladib and statin arm for statistical significance to attain a P value of 0.05. Although not powered for significance, the varespladib plus statin arm in FRANCIS did show an 18% overall reduction in MACE. In our view the hurdle rate for VISTA-16 is achievable given the increase in power, existing precedence of the JUPITER trial demonstrating a 44% delta with statins alone and due to the proven synergistic activity of varespladib in combination with statins.

Figure 5: ANALYSIS OF ACS OUTCOMES STUDIES

Study	Year of Publication	N	Primary Indication	Treatment Arm	Comparator Arm	Outcomes	PEP Treatment Arm	PEP Control Arm	Delta	P value	HR	Source
The PROVE IT#TIMI 22 (Pravastatin or Atorvastatin Evaluation and Infection Therapy#Thrombolysis In Myocardial Infarction 22)	2009	N = 4162	ACS	Atorvastatin 80mg	Pravastatin 40mg	MACE	21.50%	26.5%	23.2%	P = 0.002	0.78	*JACC, 2009*
MERLIN-TIMI 36	2008	N= 3279	ACS	Rano lazine	Placebo	MACE	Non Inferior to Placebo					*ACC, 2007*
Early intensive Statin vs Dalayed Conservative Statin	2004	N = 4497	ACS	Simvastatin 40mg/80mg	Placebo/Simvastatin 20mg	MACE	14.40%	16.7%	*15.9%*	P = 0.14	0.89	*JAMA, 2004*
						CVS Death	4.10%	5.4%	31.7%	P = 0.05	0.75	
Intensive vs Moderate Lipid Lowering with Statins in ACS	2004	N = 4162	ACS	Atorvastatin 80mg	Pravastatin 40mg	MACE	22.40%	26.3%	17.4%	P = 0.005	0.84	*NEJM, 2004*
The Myocardial Ischemia Reduction with Aggressive Cholesterol Lowering (MIRACL) study	2001	N = 3086	ACS	Atovastatin 80mg 24 to 96hrs after admission	Placebo	MACE	14.80%	17.4%	17.5%	P = 0.048	0.84	*JAMA, 2001*
Varespladib -VISTA Study	2010-11	N = 6500	ACS	Varespladib + Lipitor	Lipitor	MACE`	6.38%	8.50%	25.0%	P = 0.05	0.75	

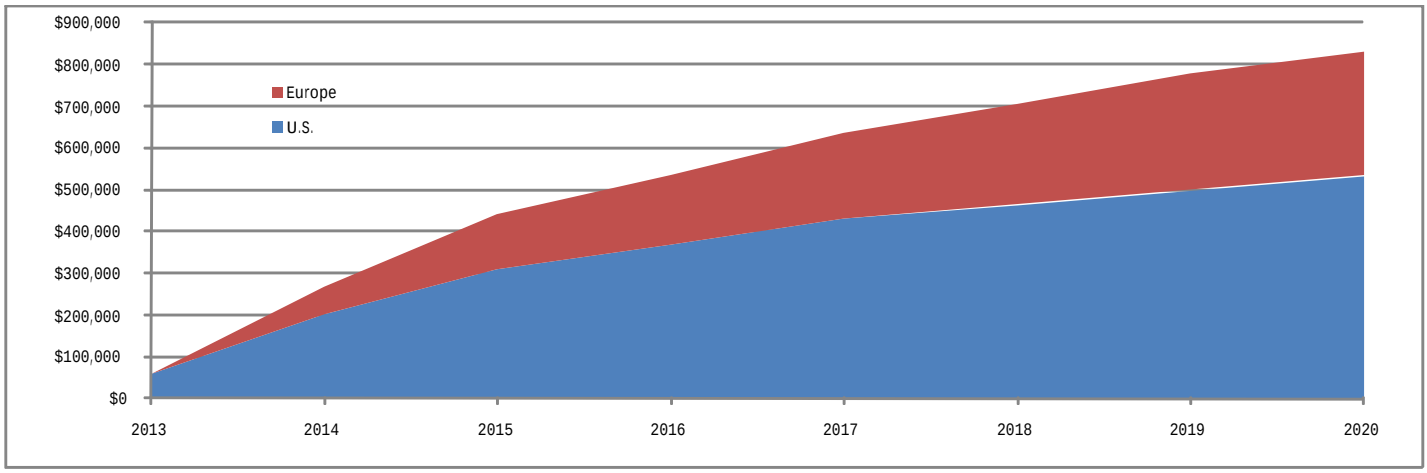
Source: Piper Jaffray analysis. Please see above.

VARESPLADIB SALES FORECAST

CAD is the leading cause of death in the developed world and ACS is a major cause of emergency medical care and hospitalization. Annual incidence of ACS in the U.S. based on hospital discharges is approximately 1.3 million with 41% of the admissions due to Unstable Angina, 36% due to STEMI and 40% due to NSTEMI. (*Circulation* Jan. 2009) An estimated 15% of ACS patients have congestive heart failure (CHF) or other co-morbidities that are excluded from VISTA-16 enrollment criteria and thus may not be included on the Varespladib label.

We expect VISTA-16 will begin this summer with pivotal data in late 2011 or early 2012. Based on positive pivotal data, we expect NDA filing in 2012 and U.S. approval in 2013 and in Europe by 2014. Shionogi retains rights in Japan. Assuming modest 15% penetration of the U.S. ACS market with a selling price of ~$1,700 and 10% penetration of the European ACS market with a lower selling price of less than $1,400, we forecast global sales of $438 million in 2015. We forecast global Varespladib sales will grow to $830 million by 2020 and exceed $1 billion in 2023. (Please see Figure 6 below.)

Figure 6: U.S. VARESPLIDIB SALES FORECAST



	2013	2014	2015	2016	2017	2018	2019	2020
Annual Incidence of ACS in U.S.	**1,363,794**	**1,377,432**	**1,391,206**	**1,405,118**	**1,419,169**	**1,433,361**	**1,447,695**	**1,462,172**
Patients Eligible for Varespladib therapy	**1,159,225**	**1,170,817**	**1,182,525**	**1,194,351**	**1,206,294**	**1,218,357**	**1,230,541**	**1,242,846**
Percetage of ACS Patients	*85.0%*	*85.0%*	*85.0%*	*85.0%*	*85.0%*	*85.0%*	*85.0%*	*85.0%*
ACS patients treated with Varespladib	**34,777**	**117,082**	**177,379**	**209,011**	**241,259**	**255,855**	**270,719**	**285,855**
Penetration of Varespladib	***3.0%***	***10.0%***	***15.0%***	***17.5%***	***20.0%***	***21.0%***	***22.0%***	***23.0%***
Cost of Varespladib therapy per day	**$15.00**	**$15.23**	**$15.45**	**$15.69**	**$15.92**	**$16.16**	**$16.40**	**$16.65**
Total Varespladib Per Patient Cost	$1,680	$1,705	$1,731	$1,757	$1,783	$1,810	$1,837	$1,865
Varespladib U.S. Sales ($000s)	**$58,425**	**$199,648**	**$307,003**	**$367,179**	**$430,186**	**$463,056**	**$497,307**	**$532,987**
Annual Incidence of ACS in Europe	**1,124,487**	**1,125,611**	**1,126,737**	**1,127,864**	**1,128,991**	**1,130,120**	**1,131,251**	**1,132,382**
Patients Eligible for Varespladib therapy	**955,814**	**956,770**	**957,726**	**958,684**	**959,643**	**960,602**	**961,563**	**962,525**
Percetage of ACS Patients	*85.0%*	*85.0%*	*85.0%*	*85.0%*	*85.0%*	*85.0%*	*85.0%*	*85.0%*
ACS patients treated with Varesladib		**47,838**	**95,773**	**119,836**	**143,946**	**168,105**	**192,313**	**202,130**
Penetration of Varespladib		***5.0%***	***10.0%***	***12.5%***	***15.0%***	***17.5%***	***20.0%***	***21.0%***
Cost of Varespladib therapy per day		**$12.00**	**$12.18**	**$12.36**	**$12.55**	**$12.74**	**$12.93**	**$13.12**
Total Varespladib Per Patient Cost		$1,344	$1,364	$1,385	$1,405	$1,426	$1,448	$1,470
Varespladib European Sales ($000s)		**$64,295**	**$130,649**	**$165,927**	**$202,301**	**$239,798**	**$278,444**	**$297,048**
Worldwide Varespladib Sales ($000s)	**$58,425**	**$263,943**	**$437,652**	**$533,105**	**$632,487**	**$702,854**	**$775,750**	**$830,035**

Source: Piper Jaffray company reports.

We expect Anthera will enter into a major Varespladib distribution alliance bringing in significant capital and a cardiology sales force. Importantly, we ultimately believe that a partner may explore use of varespladib more broadly in CAD much like darapladib representing significant upside to our forecast.

SYSTEMIC LUPUS ERYTHEMATOSUS

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease in which an individual's immune system attacks the body's own tissues. Lupus is a debilitating disease that affects multiple tissues including the skin, joints, and multiple organ systems including the lungs, kidneys, and blood. Lupus causes inflammation, atherosclerosis, organ damage/failure, and can potentially result in death. Lupus has been a particularly difficult disease to treat due to the heterogeneity of disease. This complex autoimmune disease targets organs in each individual differently and unpredictably, making drug development and clinical trial design challenging.

The CDC estimates lupus affects approximately 1.5 million Americans and at least 5 million people worldwide. The death rate from lupus remains relatively low at 52 people per million with a total of 22,861 deaths reported from 1979-1998. Approximately 36% of deaths occur under the age of 45 and are caused by active SLE, organ failure, infection, or heart disease related to accelerated atherosclerosis. Lupus predominantly affects women (5x) and especially those of African descent (3x). It is estimated that 1 in 250 African-American women between the ages of 18 and 65 are affected by the disease.

No new therapies for the treatment of lupus have been approved by the FDA over the last 40 years, presenting a massive

unmet clinical need. Today, most treatments for lupus only provide symptomatic relief including NSAIDs, corticosteroids, and immunosuppressants. The FDA states that current treatments for SLE remain inadequate as many patients have incompletely controlled disease, progression to end-stage organ involvement continues, and current therapies carry potential risks of debilitating side effects. Lupus can be extremely difficult to diagnose as patients do not display identical symptoms. Often, different symptoms occur over a period of time, and it may take years for a definitive diagnosis.

Physical symptoms of SLE are often accompanied by elevated concentrations of antinuclear antibodies (ANA). An immunoflorescent test is available to measure concentrations of ANA and to confirm SLE when other symptoms are seen. Almost 97% of lupus patients have elevated concentrations of ANA. However, the test is not definitive as elevated ANA concentrations are associated with numerous ailments such as rheumatoid arthritis, thyroid disease, viral infections, autoimmune diseases and certain drug use. Further, an ANA test will be weakly positive in approximately 20% of healthy individuals. Difficulty in diagnosing the disease is further complicated by the fact that lupus can enter periods of inactivity or remission and can quickly "flare up" due to some external stimuli.

Several potentially fatal diseases are associated with SLE as a result of increased inflammatory response and cellular damage. These include increased risk of atherosclerosis and inflammation of the heart (myocarditis, endocarditis, and pericarditis), kidney (nephritis), the lungs (pleuritis), blood (vasculitis), and central nervous system. During periods of flare, many of these organ systems are susceptible to damage, which can accumulate over years and eventually lead to organ failure. Flares are treated with increased immunosuppressive therapy, which in turn has additional toxicities. The most commonly studied organ-specific manifestation of lupus is lupus nephritis, which can lead to end-stage renal disease and death.

BLYS TARGETING IS THE NEXT BIG ADVANCE IN LUPUS THERAPY

B-Lymphocyte Stimulator (BLyS) is a highly specific B-cell growth factor that causes progenitor cells to differentiate into B-lymphocytes. B-cells are immune cells that play a central role in the inflammatory component of lupus. Plasma B cells produce antibodies that act as the first line of defense for the body's immune system. In lupus and certain other autoimmune diseases, elevated levels of BLyS are believed to contribute to the production of auto-antibodies that attack and destroy the body's own healthy tissues. The presence of auto-antibodies appears to correlate with disease severity. Extensive preclinical studies have demonstrated that BLyS promotes B-cell count, as well as production of the serum immunoglobulin IgA, IgG, and IgM.

BLyS is a clinically validated lupus target, as evidenced by the recent success of Human Genome Science's *Benlysta*. Benlysta is a fully human monoclonal antibody directed against soluble BLyS which has completed two Phase III trials. HGS and partner GSK intend to file a BLA for 10mg/kg Benlysta in the U.S. this quarter. Importantly, BLys targeting agents may have applicability in other autoimmune diseases such as rheumatoid arthritis and multiple sclerosis.

In two Phase III trials, BLISS-52 and BLISS-76, SLE patients with confirmed antinuclear antibodies (ANA+) were randomized to one of three treatment groups: 10 mg/kg Benlysta (in BLISS-52, n=290), 1 mg/kg Benlysta (BLISS-52, n=288), or placebo (BLISS-52, n=287). Patients were dosed intravenously on Days 0, 14 and 28, then every 28 days thereafter for the duration of the study. All receive standard of care therapy in addition to the study medication.

In the Phase III BLISS-52 study, Benlysta met the composite primary endpoint of response at 52 weeks. Benlysta also significantly reduced SLE disease activity versus placebo based on the SELENA SLEDAI and Physician's Global Assessment scores. The composite primary endpoint was defined by 1.) a reduction in baseline SELENA SLEDAI score of at least 4 points, 2.) no worsening in the Physician's Global Assessment (PGS) score, 3.) no new BILAG-A severe organ domain score, and 4.) no >1 new BILAG-B moderate organ domain score from baseline. Benlysta showed a composite response of 57.6% for 10 mg/kg (p=0.0006), 51.7% for 1 mg/kg (p=0.013) vs. 43.6% for placebo. Benlysta response was driven by a significant 4-point reduction in SLEDAI at both 10mg/kg (p=0.0024) and 1mg/kg (p=0.019). Benlysta delayed time to first SLE Flare with median 119 days for 10 mg/kg (p=0.0055), 126 days for 1 mg/kg (p=0.1342) vs. 84 days for placebo. On Physician's Global assessment (PGA), Benlysta showed a 45.7% improvement at 10mg/kg (p<0.0001) and 39.3% at 1mg/kg (p=0.004) vs. 27.8% for placebo. Steroid dose reduction trended at 10mg/kg, but was not statistically significant. Both Benlysta doses did significantly improve fatigue (p<0.05) and Health Related Quality of Life as measured by SF36 (p=0.025 and p=0.027, respectively). Benlysta was generally well tolerated, with rates of overall adverse events, comparable between Benlysta and placebo treatment groups. Serious infection was 6.2% in Benlysta patients vs. 5.9% with placebo.

The BLISS-76 study reported patient response of 43.2% for 10 mg/kg (p=0.021), 40.6% for 1 mg/kg (p=0.10) versus 33.8% for placebo. While these results came in below the first BLISS-52 trial, 10mg/kg was statistically significant and supports approval. BLISS-76 has precisely the same design as BLISS-52, however is slightly smaller with 826 SLE ANA+ patients primarily recruited in the developed world - in U.S., Canada and Europe, and only 5-10% from and Mexico and Costa Rica. Benlysta response was driven by a significant 4-point reduction in SLEDAI at the 10mg/kg (p=0.0062) with a non-significant trend at 1mg/kg (p=0.087). On the secondary PGA endpoint, Benlysta showed non-significant trends at both doses with a 0.49 point improvement at 10mg/kg and 0.55 point improvement at 1mg/kg vs. 0.46 point improvement for placebo. Steroid dose reduction also trended at 10mg/kg in BLISS-76, but reached significance when pooled with BLISS-56.

A-623 CLINICAL DATA AND DEVELOPMENT PLAN

A-623 is a peptibody antagonist against BlyS. Anthera in-licensed A-623 from Amgen in December 2007 and holds worldwide rights. A-623 offers several benefits over Benlysta. Most importantly, A-623 has shown proof-of-concept clinical data when formulated as both an intravenous (IV) infusion and a subcutaneous injection while Benlysta will only be available as a less convenient IV infusion that requires a monthly visit to a doctor's office. A-623 also targets both soluble and receptor bound BLyS while Benlysta only binds soluble BLys, a difference that may increase efficacy.

In two Phase I studies of 107 lupus patients, A-623 showed anti-BLys activity and statistically significant reductions in B cells using either single IV or single subcutaneous administration ($p<0.001$). A Phase Ib study of 63 lupus patients investigated IV doses of A-623 0.3, 1.0, and 3.0 mg/kg and an subcutaneous dose 6.0 mg/kg and demonstrated significant and selective decreases in B-cells as early as 15 days.

Anthera is planning a Phase II study of subcutaneous A-623 in 120 serologically active lupus patients on top of standard of care, which could provide a value inflection point in 2011. The trial will randomize patients with a SELENA SLEDAI > 6 who are ANA+ or dsDNA positive to one of three undisclosed doses of A-623 or placebo. The primary endpoint of the Phase II study will be percent changes in B-cell counts, as well as other relevant immunological biomarkers, such as changes in double-stranded DNA, IgG and IgM levels. A key secondary endpoint will mimic the composite response endpoint used in the Benlysta trials after 4 months of treatment. Following the 4 month treatment phase of the trial, patient will be followed for an additional 2 months for further safety assessments.

A-623 MARKET FORECAST

The size of the lupus market is the subject of some controversy. The Lupus Foundation of America estimates there are 1.5 million patients with SLE in the U.S. and 5 million worldwide, although many question the methodology used to arrive at these figures. In our opinion, the most reliable recent estimates come from a meta-analysis of available national surveys done in 2008 by the National Arthritis Data Workgroup (Helmick et al. (2008), *Arthritis and Rheumatism*, **58**: 15-25.) According to the Workgroup's analysis, 161,000 Americans have definitive SLE and up to 322,000 people have definitive or probable SLE. We conservatively estimate the addressable systemic lupus erythmatosus (SLE) population was ~200,000 in the U.S. in 2007, 70% of whom are seropositive and therefore eligible for A-623. Assuming a price of $25,000 annually and modest peak penetrations of 30% of the addressable SLE market, we forecast A-623 sales could reach $1.2 billion by 2020. We point out that we do not include any potential sales of A-623 outside of Lupus or in territories outside of the U.S. in our current estimates. Importantly, we do not include potential A-623 sales in our current price target valuation providing potential upside.

Figure 7: A-623 U.S. SALES FORECAST

	2015E	2016E	2017E	2018E	2019E	2020
Population with Lupus	216,571	218,737	220,924	223,134	225,365	227,619
Patients Eligible for A-623 Therapy	**151,600**	**153,116**	**154,647**	**156,194**	**157,756**	**159,333**
% Seropositive	***70.0%***	***70.0%***	***70.0%***	***70.0%***	***70.0%***	***70.0%***
A-623 Patients (000)	7,580	22,967	30,929	39,048	47,327	47,800
A-623 Penetration	***5.0%***	***15.0%***	***20.0%***	***25.0%***	***30.0%***	***30.0%***
Estimated A-623 Price	$25,000	$25,000	$25,000	$25,000	$25,000	$25,000
U.S. Lupus Sales ($000's)	**$189,500**	**$574,185**	**$773,235**	**$976,210**	**$1,183,166**	**$1,194,998**

Source: Piper Jaffray Estimates

EXPERIENCED MANAGEMENT; DONE THIS BEFORE

Paul Truex, President and CEO has served as the President and CEO of Anthera since its founding in September 2004. Previously, Truex served as a Director, President and CEO of Peninsula Pharmaceuticals, which was sold to Johnson & Johnson (JNJ) and as VP of Commercial Development for Vicuron. Prior to this, Truex held various positions at Eli Lilly and Company (LLY).

Chris Lowe, Chief Financial Officer has served as the CFO and VP of Administration of Anthera since November 2007. Previously Lowe served as VP of Finance & Administration and CFO of Asthmatx. Before this he worked as the Corporate Controller of Peninsula Pharmaceuticals.

James E. Pennington, MD, Chief Medical Officer. Dr. Pennington has served as the EVP and CMO of Anthera since March 2007. Previously he was the EVP and CMO at CoTherix. Dr Penningon obtained his medical degree from the University of Oregon School of Medicine and is board certified in internal medicine and infectious disease.

Colin Hislop, PhD, Chief Scientific Officer. Dr. Hislop has served as the SVP of Cardiovascular Products at Anthera since November 2005. Previously, Dr. Hislop was VP, Clinical Development at Peninsula Pharmaceuticals and as VP of Clinical Development at CV Therapeutics. Dr. Hislop holds a degree in medicine from the University of London.

SCIENTIFIC ADVISORY BOARD

Stephen J. Nicholls, M.D., Ph.D. Dr. Nicholls has been Assistant Professor of Molecular Medicine and Associate Director of the Cleveland Clinic Coordinating Center for Clinical Research since 2006. Dr. Nicholls holds a medical degree from the University of Adelaide in Australia and completed his doctoral studies at the Heart Research Institute in Sydney, Australia.

John J.P. Kastelein, M.D., Ph.D. Dr. Kastelein has been a Professor of Medicine and Chairman of the Department of Vascular Medicine at the Academic Medical Center of the University of Amsterdam since January 2003, where Dr. Kastelein holds the Strategic Chair of Genetics of Cardiovascular Disease. Dr. Kastelein holds a medical degree from the University of Amsterdam.

Robert S. Rosenson M.D., FACC, FACP, FAHA. Dr. Rosenson has been Professor of Medicine at the State University of New York at the Downstate Campus in Brooklyn, where Dr. Rosenson is the Chief in the Division of Endocrinology, Diabetes and Metabolism and serves as a Senior Cardiologist. Dr. Rosenson holds a medical degree from Tulane University

David D. Waters, M.D. Dr. Waters was Chief of Cardiology at San Francisco General Hospital and the Maurice Eliaser Jr. Distinguished Professor of Medicine at UCSF from 1999 to 2007, and is now Emeritus Professor in the Department of Medicine. He completed medical school at the University of Western Ontario.

FINANCIALS:

Revenues. Anthera reported no revenues in 2009 and we do not forecast any revenues for 2010 or 2011.

Operating Expenses. In 2009, Anthera reported R&D expense of $8.4 million and G&A expense of $3.4 million. We forecast operating expense will increase substantially in 2010 and 2011 as the company conducts the Phase III VISTA-16 study for varespladib and a Phase II lupus study for A-623. We project R&D expense of $27.5 million in 2010 and $40 million in 2011 and G&A expense of $7.5 million in 2010 and $8 million in 2011.

Net Loss. Anthera reported a net loss of $12.2 million or ($1.24) per adjusted share in 2009. We forecast increased net losses of $34.8 million or ($1.57) per share in 2010 and $47.8 million or ($2.01) per share in 2011.

Balance Sheet. Anthera ended 2009 with $3.8 million in cash. Anthera subsequently raised over $60 million in net proceeds in a successful IPO priced at $7 per share including insider participation and exercise of a partial green shoe bringing *pro forma* cash to $64.2 million. We forecast this cash will last into mid-'11. After conversion of convertible debt at the IPO, Anthera currently holds no debt.

VALUATION:

We are initiating coverage of Anthera with an Overweight rating and $12 price target. Anthera recently raised over $60 million in net proceeds in a successful IPO priced at $7 per share including insider participation and exercise of a partial green shoe. We value varespladib at $288 million by applying a standard 5x multiple to 2015 U.S. sales of $307 million discounted back at 45% annually. We believe this discount rate is on the high side (30-45%) for Phase II-ready drugs. To this we add mid'11E cash of $3 million. At present, we assign no value for European varespladib sales or A-623 in lupus providing potential upside.

A comp group of small cap cardiovascular and autoimmune companies is currently trading at an average enterprise value of $267 million. (Please see Figure 8 below.) At Friday 4/9 close, Anthera is trading at an enterprise value of $91 million, representing a significant discount to the comp group. We look for Anthera to create value by reporting Phase II A-623 lupus data in 2011 and Phase III varespladib ACS data by early 2012, as well as potentially partnering either drug in the interim.

Figure 8: ANTHERA COMP ANALYSIS

ANTHERA Comparable Company Analysis

$ in millions except per share Company	Ticker	Price 04/09/10	Market Cap.	Cash	Mkt. Cap./ Cash	Tech. Value	LTD	Net Cash	Ent. Value
Human Genome Sciences	HGSI	$32.86	$5,847	$1,075	5.4	4,772	$207	869	4,979
Ardea	RDEA	$22.12	$407	$49	8.3	358	$5	44	363
Cardiome	CRME	$7.27	$437	$84	5.2	353	$0	84	353
Arena Pharmaceuticals	ARNA	$3.18	$295	$114	2.6	181	$90	24	271
Rigel	RIGL	$7.67	$397	$157	2.5	241	$2	155	243
Cytokinetics	CYTK	$3.49	$213	$121	1.8	92	$14	107	106
Average (excl. HGSI & INCY)			**$350**	**$105**	**4.1**	**$245**	**$22**	**$83**	**$267**
Anthera Pharmaceuticals	**ANTH**	**$6.99**	**$155**	**$64**	**2.4**	**91**	**$0**	**64**	**91**

Source: Company Reports, First Call Corp., ILX and Piper Jaffray & Co. estimates

Source: Company Reports, Piper Jaffray estimates.

INVESTMENT RISKS:

Risks associated with Anthera are typical with all drug discovery companies including clinical, regulatory and commercial. Anthera may require additional capital to complete the pivotal ACS and Phase II lupus studies. VISTA-16 may fail and varespladib may not gain regulatory approval. Anthera may be unable to partner varespladib based on pivotal data. Cardiovascular disease is a competitive space with new and entrenched agents. Varespladib may not meet sales forecast and as a result fail to achieve profitability. A-623 may fail in Phase II studies. Anthera may be unable to partner A-623, which may not compete effectively against competitive agents. Anthera may face future litigation.

Anthera Pharmaceuticals

Quarterly Earnings Estimates

12-Apr-10 ($ in thousands except per share)

	1Q	2Q	3Q	4Q	2009A	1Q	2Q	3Q	4Q	2010E	1Q	2Q	3Q	4Q	2011E
Total Revenues	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
Operating Expenses:															
R&D Expense	2,915	2,286	2,526	688	8,415	2,000	4,000	8,500	13,000	27,500	12,000	10,000	8,000	10,000	40,000
SG&A Expense	846	999	885	695	3,425	2,000	1,700	1,800	2,000	7,500	2,000	2,000	2,000	2,000	8,000
Total Operating Expenses	**$3,761**	**$3,286**	**$3,411**	**$1,383**	**11,841**	**$4,000**	**$5,700**	**$10,300**	**$15,000**	**35,000**	**$14,000**	**$12,000**	**$10,000**	**$12,000**	**48,000**
Operating Loss	**($3,761)**	**($3,286)**	**($3,411)**	**($1,383)**	**($11,841)**	**($4,000)**	**($5,700)**	**($10,300)**	**($15,000)**	**($35,000)**	**($14,000)**	**($12,000)**	**($10,000)**	**($12,000)**	**($48,000)**
Interest and Other Income	13	9	(0)	2	24	25	50	45	35	155	45	50	40	35	170
Interest Expense	(37)	(59)	(193)	(96)	(385)	0	0	0	0	0	0	0	0	0	0
Total Other Income (Expense)	(24)	(50)	(193)	(94)	(362)	25	50	45	35	155	45	50	40	35	170
Pre-Tax Loss	**($3,785)**	**($3,336)**	**($3,604)**	**($1,477)**	**($12,203)**	**($3,975)**	**($5,650)**	**($10,255)**	**($14,965)**	**($34,845)**	**($13,955)**	**($11,950)**	**($9,960)**	**($11,965)**	**($47,830)**
Income Tax Expense	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0	$0
Net Loss	**($3,785)**	**($3,336)**	**($3,604)**	**($1,477)**	**($12,203)**	**($3,975)**	**($5,650)**	**($10,255)**	**($14,965)**	**($34,845)**	**($13,955)**	**($11,950)**	**($9,960)**	**($11,965)**	**($47,830)**
Net Loss per Share	**($0.34)**	**($0.30)**	**($0.22)**	**($0.09)**	**($1.24)**	**($0.21)**	**($0.24)**	**($0.44)**	**($0.64)**	**($1.57)**	**($0.59)**	**($0.50)**	**($0.42)**	**($0.50)**	**($2.01)**
Shares Outstanding	11,087	11,095	16,415	16,542	9,854	18,750	23,250	23,350	23,500	22,212	23,600	23,700	23,800	24,000	23,775

Source: Company reports and Piper Jaffray estimates.

Note: CY:09A results include adjusted pro forma share count. As a result, quarterly EPS does not add to full year result.

For up-to-date company disclosure information, please visit http://www.piperjaffray.com/researchdisclosures

Important Research Disclosures

Notes: The boxes on the Rating and Price Target History chart above indicate the date of the Research Note, the rating, and the price target. Each box represents a date on which an analyst made a change to a rating or price target, except for the first box, which may only represent the first Note written during the past three years.

Legend:

I: Initiating Coverage

R: Resuming Coverage

T: Transferring Coverage

D: Discontinuing Coverage

S: Suspending Coverage

OW: Overweight

N: Neutral

UW: Underweight

B: Buy (Piper Jaffray discontinued use of the B, N, and S ratings on June 30, 2009)

N: Neutral

S: Sell

OP: Outperform (Piper Jaffray discontinued use of the OP, MP and UP ratings on November 15, 2007)

MP: Market Perform

UP: Underperform

AL On/AL Off: Placed on/removed from the Alpha List maintained by Piper Jaffray

NA: Not Available

UR: Under Review

Distribution of Ratings/IB Services
Piper Jaffray

			IB Serv./Past 12 Mos.	
Rating	Count	Percent	Count	Percent
BUY [OW]	334	48.00	69	20.66
HOLD [N]	275	39.50	18	6.55
SELL [UW]	87	12.50	1	1.15

Note: Distribution of Ratings/IB Services shows the number of companies currently in each rating category from which Piper Jaffray and its affiliates received compensation for investment banking services within the past 12 months. FINRA rules require disclosure of which ratings most closely correspond with "buy," "hold," and "sell" recommendations. Piper Jaffray ratings are not the equivalent of buy, hold or sell, but instead represent recommended relative weightings. Nevertheless, Overweight corresponds most closely with buy, Neutral with hold and Underweight with sell. See Stock Rating definitions below.

Important Research Disclosures

Analyst Certification — Edward A. Tenthoff, Sr Research Analyst

The views expressed in this report accurately reflect my personal views about the subject company and the subject security. In addition, no part of my compensation was, is, or will be directly or indirectly related to the specific recommendations or views contained in this report.

Research Disclosures

Piper Jaffray was making a market in the securities of Anthera Pharmaceuticals, Inc. at the time this research report was published. Piper Jaffray will buy and sell Anthera Pharmaceuticals, Inc. securities on a principal basis.

Piper Jaffray expects to receive or intends to seek compensation for investment banking services from Anthera Pharmaceuticals, Inc. in the next 3 months.

Piper Jaffray has received compensation for investment banking services from or has had a client relationship with Anthera Pharmaceuticals, Inc. within the past 12 months.

Within the past 12 months Piper Jaffray was a managing underwriter of a public offering of, or dealer manager of a tender offer for, the securities of Anthera Pharmaceuticals, Inc. or the securities of an affiliate.

Within the past 3 years Piper Jaffray participated in a public offering of, or acted as a dealer manager for, Anthera Pharmaceuticals, Inc. securities.

Affiliate Disclosures: This report has been prepared by Piper Jaffray & Co. and/or its affiliates Piper Jaffray Ltd. and Piper Jaffray Asia Securities Limited, all of which are subsidiaries of Piper Jaffray Companies (collectively Piper Jaffray). Piper Jaffray & Co. is regulated by FINRA, NYSE, and the United States Securities and Exchange Commission, and its headquarters is located at 800 Nicollet Mall, Minneapolis, MN 55402. Piper Jaffray Ltd. is registered in England, no. 3846990, and its registered office is One South Place, London, EC2M 2RB. Piper Jaffray Ltd. is authorised and regulated by the UK Financial Services Authority ("FSA"), entered on the FSA's register, no. 191657 and is a member of the London Stock Exchange. Piper Jaffray Asia Securities Limited is a licensed corporation regulated by the Securities and Futures Commission of Hong Kong ("SFC"), entered on the SFC's register, no. ABO154, and is an exchange participant of The Stock Exchange of Hong Kong Limited. Its headquarters is located at 39/F Tower 1 Lippo Centre, 89 Queensway, Hong Kong. Disclosures in this section and in the Other Important Information section referencing Piper Jaffray include all affiliated entities unless otherwise specified.

Piper Jaffray research analysts receive compensation that is based, in part, on overall firm revenues, which include investment banking revenues.

Rating Definitions

Stock Ratings: Piper Jaffray ratings are indicators of expected total return (price appreciation plus dividend) within the next 12 months. At times analysts may specify a different investment horizon or may include additional investment time horizons for specific stocks. Stock performance is measured relative to the group of stocks covered by each analyst. Lists of the stocks covered by each are available at www.piperjaffray.com/researchdisclosures. Stock ratings and/or stock coverage may be suspended from time to time in the event that there is no active analyst opinion or analyst coverage, but the opinion or coverage is expected to resume. Research reports and ratings should not be relied upon as individual investment advice. As always, an investor's decision to buy or sell a security must depend on individual circumstances, including existing holdings, time horizons and risk tolerance.

- **Overweight (OW):** Anticipated to outperform relative to the median of the group of stocks covered by the analyst.
- **Neutral (N):** Anticipated to perform in line relative to the median of the group of stocks covered by the analyst.
- **Underweight (UW):** Anticipated to underperform relative to the median of the group of stocks covered by the analyst.

An industry outlook represents the analyst's view of the industry represented by the stocks in the analyst's coverage group. A Favorable industry outlook generally means that the analyst expects the fundamentals and/or valuations of the industry to improve over the investment time horizon. A Neutral industry outlook generally means that the analyst does not expect the fundamentals and/or valuations of the industry to either improve or deteriorate meaningfully from its current state. An Unfavorable industry outlook generally means that the analyst expects the fundamentals and/or valuations of the industry to deteriorate meaningfully over the investment time horizon.

The Alpha List consists of Overweight-rated stocks that have been selected by analysts from among their best ideas within their universe of covered stocks, with an eye toward above-average returns over the next three to six months.

Other Important Information

The material regarding the subject company is based on data obtained from sources we deem to be reliable; it is not guaranteed as to accuracy and does not purport to be complete. This report is solely for informational purposes and is not intended to be used as the primary basis of investment decisions. Piper Jaffray has not assessed the suitability of the subject company for any person. Because of individual client requirements, it is not, and it should not be construed as, advice designed to meet the particular investment needs of any investor. This report is not an offer or the solicitation of an offer to sell or buy any security. Unless otherwise noted, the price of a security mentioned in this report is the market closing price as of the end of the prior business day. Piper Jaffray does not maintain a predetermined schedule for publication of research and will not necessarily update this report. Piper Jaffray policy generally prohibits research analysts from sending draft research reports to subject companies; however, it should be presumed that the analyst(s) who authored this report has had discussions with the subject company to ensure factual accuracy prior to publication, and has had assistance from the company in conducting diligence, including visits to company sites and meetings with company management and other representatives.

This report is published in accordance with a conflicts management policy, which is available at http://www.piperjaffray.com/researchdisclosures.

Notice to customers: This material is not directed to, or intended for distribution to or use by, any person or entity if Piper Jaffray is prohibited or restricted by any legislation or regulation in any jurisdiction from making it available to such person or entity. Customers in any of the jurisdictions where Piper Jaffray and its affiliates do business who wish to effect a transaction in the securities discussed in this report should contact their local Piper Jaffray representative. **Europe:** This material is for the use of intended recipients only and only for distribution to professional and institutional investors, i.e. persons who are authorised persons or exempted persons within the meaning of the Financial Services and Markets Act 2000 of the United Kingdom, or persons who have been categorised by Piper Jaffray Ltd. as professional clients under the rules of the Financial Services Authority. **Asia:** This report is distributed in Hong Kong by Piper Jaffray Asia Securities Limited, which is regulated by the Hong Kong SFC. This report is intended only for distribution to professional investors as defined in the Hong Kong Securities and Futures Ordinance and is for the use of intended recipients only. **United States:** This report is distributed in the United States by Piper Jaffray & Co., member SIPC, FINRA and NYSE, Inc., which accepts responsibility for its contents. The securities described in this report may not have been registered under the U.S. Securities Act of 1933 and, in such case, may not be offered or sold in the United States or to U.S. persons unless they have been so registered, or an exemption from the registration requirements is available.