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Reason for Report:
Company Update

Changes	Previous	Current
Rating	--	Overweight
Price Tgt	$12.00	$9.00
FY10E Rev (mil)	--	$0.0
FY11E Rev (mil)	--	$0.0
FY10E EPS	($2.20)	($1.91)
FY11E EPS	($2.10)	($1.44)

Price:	$4.22
52 Week High:	$8.55
52 Week Low:	$2.82
12-Month Price Target:	$9.00
Proj EV of $288M + mid'11E cash	
Shares Out (mil):	32.7
Market Cap. (mil):	$138.0
Avg Daily Vol (000):	31
Book Value/Share:	$2.20
Net Cash Per Share:	$2.46
Debt to Total Capital:	0%
Est LT EPS Growth:	NA
P/E to LT EPS Growth (FY11):	NA
Est Next Rep Date:	11/01/2010
Fiscal Year End:	Dec
Includes recent PIPE	

Rev (mil)	2009A	2010E	2011E
Mar	$0.0A	$0.0A	$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	($2.57)A	($0.83)A	($0.42)E
Jun	($2.22)A	($0.36)A	($0.36)E
Sep	($2.40)A	($0.43)E	($0.30)E
Dec	($0.98)A	($0.45)E	($0.36)E
FY	($8.06)A	($1.91)E	($1.44)E
CY	($8.06)A	($1.91)E	($1.44)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 - $4.22)

Overweight

Promising Lupus and Cardiac Inflammation at Attractive Valuation

CONCLUSION:

Anthera recently raised $31.5 million and now holds *pro forma* cash of ~$80 million, removing any near-term financing overhang. Anthera is enrolling a larger and longer Phase IIb PEARL study of A-623 in lupus. The company also started the 6,500-patient Phase III VISTA-16 study of *varespladib in* Acute Coronary Syndrome (ACS) patients. A safety analysis will be performed after the first 1,000 patients next year and we expect final data by early 2012.

- **Successful Deal Removes Near-Term Financing Overhang.** Anthera issued 10,500 shares with 40% warrant coverage for gross proceeds of $31.5 million bringing *pro forma* cash to ~$80 million. We believe this cash will last into 2012 and top-line PEARL and VISTA-16 data.

- **Larger and Longer A-623 Lupus Study.** In July, Anthera began the Phase IIb PEARL SC trial of A-623 for the treatment of lupus. PEARL will randomize up to 600 systemic lupus erythmatosus (SLE) patients to 3 doses of A-623 or placebo for 24 weeks on top of standard background therapy. The primary endpoint is the SLE responder index used by HGS in the *Benlysta* BLISS studies. PEARL is both larger (600 vs. 120 SLE patients) and longer (6 vs. 4 months) than envisioned at the time of the IPO in our view dramatically increasing the likelihood of success.

- **VISTA-16 Rapidly Enrolling Patients.** In July, Anthera also initiated the pivotal VISTA-16 trial of varespladib. Anthera has a Special Protocol Assessment (SPA) agreement with the FDA for a single Phase III ACS study comparing 500mg once-daily (QD) varespladib to placebo on top of *Lipitor* for 16 weeks. VISTA-16 will enroll up to 6,500 ACS patients yielding an expected 385 events and is 80% powered to show a 25% improvement in MACE. A safety analysis will be performed after the first 1,000 patients, likely by mid-2011, and we expect final VISTA-16 data by early 2012.

- **FRANCIS Study Results Published.** Last month, full results of the Phase IIb FRANCIS study were published in the *Journal of the American College of Cardiology*. The study enrolled 625 ACS patients to 500mg QD varespladib or placebo on top of 80mg Lipitor for 24 weeks. Varespladib showed a significant reduction in sPLA-2 by week 2 ($p<0.0001$) that persisted through the study, as well as LDLc by week 8 (-5.7%, $p=0.0023$) and CRP by week 16 ($p=0.0067$).

INVESTMENT RECOMMENDATION:

We reiterate our Overweight rating, but are reducing our price target to $9 from $12 to reflect the recent deal and assuming exercise of the warrants. We still value varespladib at $288 million and add mid'11E net cash.

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 LUPUS AND CARDIAC INFLAMMATION PLAY AT AN ATTRACTIVE VALUATION

Anthera is a biopharmaceutical company developing novel drugs to treat life–threatening autoimmune and inflammatory 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 initiated the Phase III VISTA-16 trial of varespladib in Acute Coronary Syndrome (ACS) patients. This 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 and is 80% powered to show a 25% improvement in MACE. 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, and we expect top-line data by early 2012. If VISTA-16 is successful, we anticipate Anthera will partner varespladib for significant financial terms. We currently project U.S. launch in 2013 with ACS sales reaching $830 million in 2020E. Ultimately we foresee blockbuster opportunity for chronic varespladib in CAD.

We see investor interest shifting to A-623, a peptibody in-licensed from Amgen that targets BLyS for lupus. Based on the success of Human Genome Sciences' (HGSI) *Benlysta*, Anthera has accelerated development of A-623. In July, Anthera began the Phase IIb PEARL SC trial to randomize up to 600 SLE patients to one of three sub-cutaneous doses of A-623 or placebo for 24 weeks. The primary endpoint is the approvable SLE responder index used by HGS in the *Benlysta* BLISS studies. PEARL is both larger (600 vs. 120 SLE patients) and longer (6 vs. 4 months) than envisioned at the time of the IPO in our view dramatically increasing the likelihood of success. Importantly, Anthera has validated drug supply to conduct the larger Phase IIb study and will make announcement soon on a supply contract. Since the peptibody in produced in *e.coli*, there is significant manufacturing capacity to supply the drug. If successful, A-623 represents a potential blockbuster drug with potential applicability beyond lupus.

Anthera recently raised $31.5 million and now holds ~$80 million in *pro forma* cash, removing any near-term financing overhang. We reiterate our Overweight rating, however are reducing our price target to $9 from $12 to reflect dilution from the offering. We still value varespladib at $288 million by applying a 5x multiple to 2015E U.S. sales of $307 million discounted back at 45% annually. From this we add mid'11E net cash. Our target assumes exercise of outstanding warrants. At present, we assign no value for European varespladib sales or A-623 in lupus providing potential for significant upside. We look for Anthera to create value by reporting Phase IIb lupus data in 2011 and Phase III ACS data by early 2012, as well as potentially partnering either drug in the interim.

Expected Upcoming Events:
- Announce supply contract for A-623
- Interim blinded A-623 lupus biomarler look in 2Q:11
- DSMB to conduct interim futility analysis after first 1,000 patients in VISTA-16 pivotal study likely in mid-'11
- Potentially partner either European rights to varespladib or A-623 retaining significant value
- Report Phase IIb A-623 lupus data in 2H:11
- Report Phase III varespladib ACS data in early 2012
- Potentially file an NDA for varespladib by mid-2012 with U.S. launch in 2013

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.

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 BLISS studies of *Benlysta.* Benlysta is a fully human monoclonal antibody directed against soluble BLyS which has completed two Phase III trials. HGS and partner GSK filed a BLA for 10mg/kg Benlysta in the U.S. and the FDA has scheduled for November 16h. 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. 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 worldwide rights to A-623 from Amgen in December 2007. A-623 offers several potential 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 monthly IV infusion that requiring a 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 0.3, 1, and 3mg/kg IV doses and a 6mg/kg subcutaneous dose showing significant and selective decreases in B-cells as early as 15 days.

Anthera has begun the Phase IIb PEARL SC study of A-623 in 600 serologically active lupus patients on top of standard of care. The trial will randomize patients with a SELENA SLEDAI score of >6 who are ANA+ or dsDNA positive to one of three undisclosed doses of A-623 or placebo on top of standard background therapy. Anthera has disclosed that there will be mid- and high-dose once-weekly sub-cu cohorts, as well as a high-dose once-monthly sub-cu cohort. The primary endpoint is the approvable SLE responder index used by HGS in the *Benlysta* studies. The study is 85% powered to show the same 14% benefit exhibited by Benlysta in the pivotal BLISS studies with a 95% confidence interval. Anthera will measure several secondary endpoints including safety, SLEDAI and BILAG scores, fatigue, steroid utilization and time to flare. A blinded interim biomarker look will measure B-cells and potentially remove any inactive arm(s). PEARL SC is both larger (600 vs. 120 SLE patients) and longer (6 vs. 4 months) than envisioned at the time of the IPO, in our view, dramatically increasing the likelihood of success.

Importantly, Anthera has validated drug supply to conduct the larger and longer Phase IIb study and will make announcement soon on a supply contract. Since the peptibody in produced in e.coli, there is significant manufacturing capacity to supply the drug. Anthera will owe Amgen ~$30 million in regulatory and commercial milestones as well as a low double digit royalty.

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 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. (Please see Figure 1 below.) 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 until we see further validation providing potential upside.

Figure 1: 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

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. 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 below.)

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.

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 PLA SMA-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.

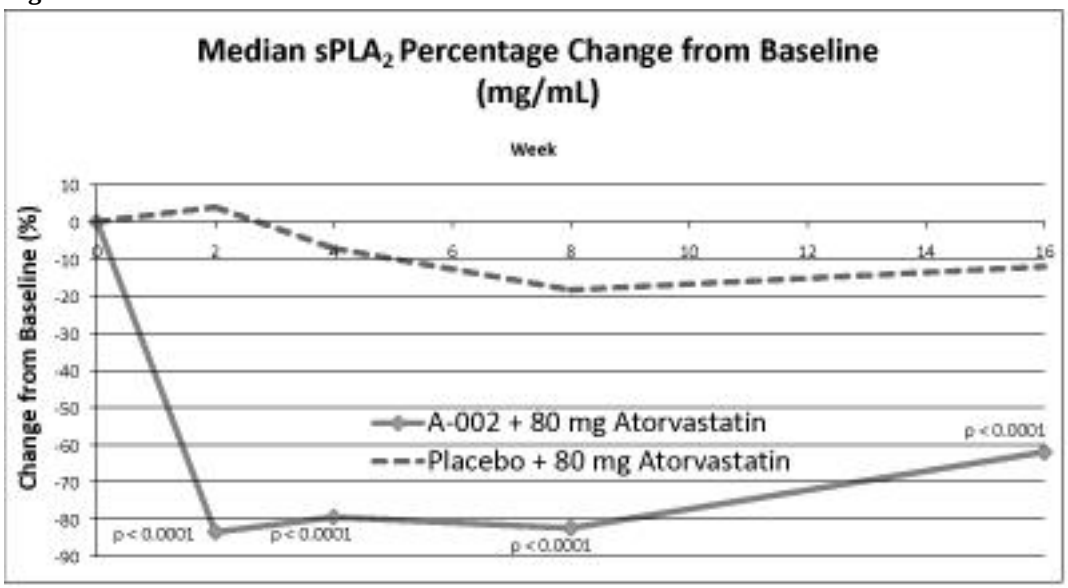
FRANCIS Study

Results from the Phase IIb FRANCIS (**F**ewer **R**ecurrent **A**cute coronary events with **N**ear-term **C**ardiovascular **I**nflammation **S**uppression) study were reported in September 2010 in the *Journal of the American College of Cardiology* (Rosenson et al., *JACC* (2010)**14**: 1079-88.) The 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.

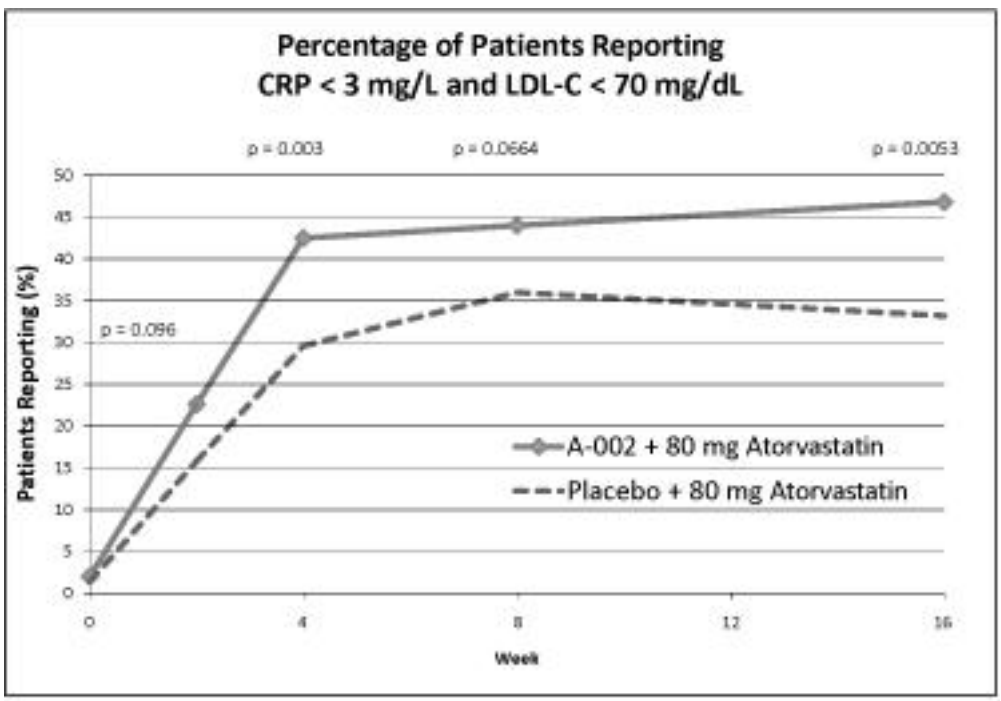
Varespladib showed a significant reduction in sPLA-2 by week 2 ($p<0.0001$) that persisted through the study (Please see Figure 3 below), as well as LDLc by week 8 (-5.7%, $p=0.0023$) and CRP by week 16 ($p=0.0067$). A significant number of patients reached their LDLc goal of <70 mg/do at week 8 ($p=0.013$) 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 be 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 QTc interval were observed.

VISTA-16 Trial

Anthera initiated the VISTA-16 trial in June under a SPA agreement with the FDA. 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 repeat Major Adverse Coronary Events (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 80% powered to show a 25% improvement in MACE. We look for final data by 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 tobe by mid-'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.

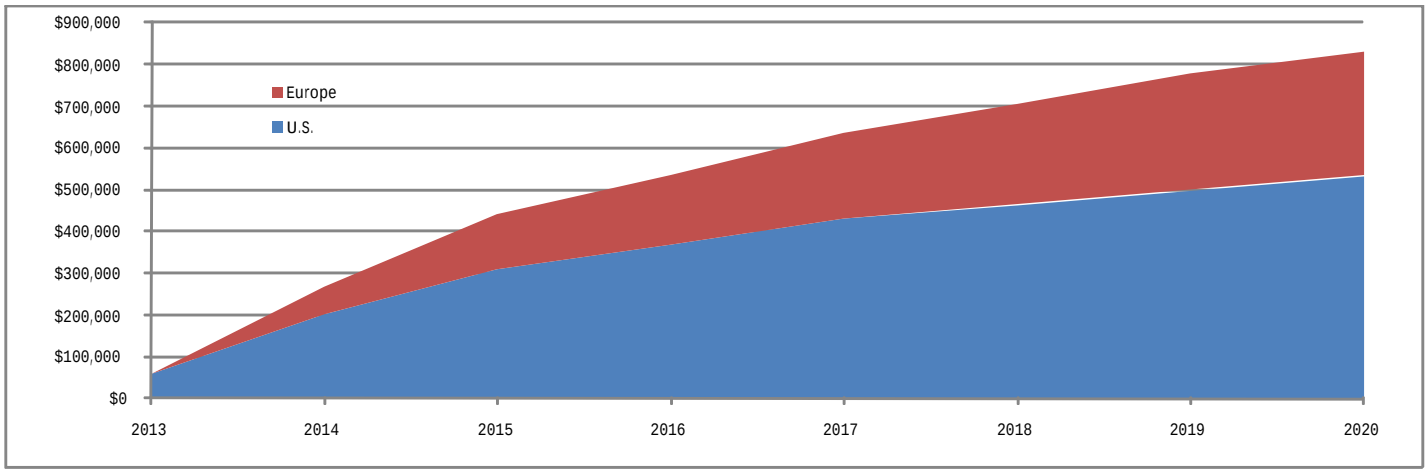
VARESPLADIB SALES FORECAST

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.

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 5 below.)

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.

Figure 5: 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.

VALUATION:

We reiterate our Overweight rating, however are reducing our price target to $9 from $12 to reflect the recent pipe. We still value varespladib at $288 million by applying a standard 5x multiple to 2015E U.S. sales of $307 million discounted back at 45% annually. From this we add mid'11E net cash. Our target assumes exercise of outstanding warrants. At present, we assign no value for European varespladib sales or A-623 in lupus providing potential upside.

Anthera Pharmaceuticals

Quarterly Earnings Estimates

($ in thousands except per share)

4-Oct-10

	1QA	2QA	3QA	4QA	2009A	1QA [1]	2QA	3QE	4QE	2010E	1QE	2QE	3QE	4QE	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	$5,242	$6,438	8,500	13,000	33,180	12,000	10,000	8,000	10,000	40,000
SG&A Expense	846	999	885	695	3,425	1,224	1,510	1,800	2,000	6,534	2,000	2,000	2,000	2,000	8,000
Total Operating Expenses	**$3,761**	**$3,286**	**$3,411**	**$1,383**	**11,841**	**$6,466**	**$7,948**	**$10,300**	**$15,000**	**39,714**	**$14,000**	**$12,000**	**$10,000**	**$12,000**	**48,000**
Operating Loss	**($3,761)**	**($3,286)**	**($3,411)**	**($1,383)**	**($11,841)**	**($6,466)**	**($7,948)**	**($10,300)**	**($15,000)**	**($39,714)**	**($14,000)**	**($12,000)**	**($10,000)**	**($12,000)**	**($48,000)**
Interest and Other Income	13	9	(0)	2	24	3	$12	45	35	95	45	50	40	35	170
Interest Expense	(37)	(59)	(193)	(96)	(385)	(4,641)	0	0	0	(4,641)	0	0	0	0	0
Total Other Income (Expense)	(24)	(50)	(193)	(94)	(362)	(4,638)	12	45	35	(4,546)	45	50	40	35	170
Pre-Tax Loss	**($3,785)**	**($3,336)**	**($3,604)**	**($1,477)**	**($12,203)**	**($11,104)**	**($7,936)**	**($10,255)**	**($14,965)**	**($44,260)**	**($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)**	**($11,104)**	**($7,936)**	**($10,255)**	**($14,965)**	**($44,260)**	**($13,955)**	**($11,950)**	**($9,960)**	**($11,965)**	**($47,830)**
Net Loss per Share	**($2.57)**	**($2.22)**	**($2.40)**	**($0.98)**	**($8.06)**	**($0.83)**	**($0.36)**	**($0.43)**	**($0.45)**	**($1.91)**	**($0.42)**	**($0.36)**	**($0.30)**	**($0.36)**	**($1.44)**
Shares Outstanding	1,471	1,500	1,500	1,500	1,514	13,344	22,224	24,050	33,000	23,155	33,100	33,200	33,300	33,500	33,275

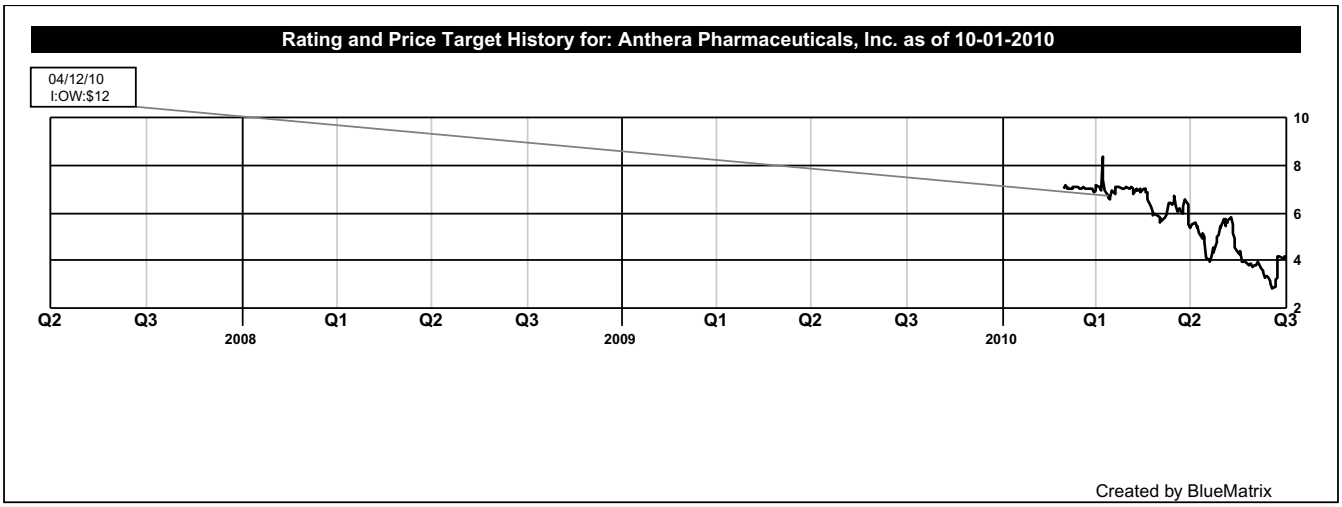
Source: Company reports and Piper Jaffray estimates.

Note: CY:09A results include adjusted primary share count excluding preferred stock. As a result, quarterly EPS does not add to full year result.

1. 1Q:10 R&D expense includes $3.5 million non-cash milestone payment in IPO stock to Lilly and Shinogi.

Current disclosure information for this company can be found at 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)

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Distribution of Ratings/IB Services
Piper Jaffray

			IB Serv./Past 12 Mos.	
Rating	Count	Percent	Count	Percent
BUY [OW]	351	49.80	82	23.36
HOLD [N]	293	41.60	27	9.22
SELL [UW]	61	8.70	3	4.92

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Analyst Certification — Edward A. Tenthoff, Sr Research Analyst

— Chad J. Messer, Ph.D., Research Analyst

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