Vitae Pharmaceuticals (VTAE)

Initiating Coverage with an OUTPERFORM Rating and $21 PT; Experienced Management, State-of-the-Art Platform, Strong IP, and Big Pharma Support

- **Vitae is a clinical-stage emerging pharmaceutical company leveraging its proprietary Contour® structure-based drug design platform to discover and develop optimized orally-dosed small-molecule drug candidates for large market indications.** Partnered with Boehringer Ingelheim (BI), the two lead programs include VTP-34072 for type 2 diabetes and VTP-37948 for Alzheimer's. The company anticipates release of phase-2 type 2 diabetes results in H1 2015 and phase-1 results for Alzheimer's by year-end–which we consider to be potential catalysts. Due to target validation for both programs, we estimate a 60% probability of a positive outcome for each trial.
- **Vitae's earlier-stage pipeline consists of three candidates for autoimmune, CNS and cardiovascular disorders–potentially providing multi-billion dollar sales potential for long-term growth.** We project VTP-43742 in autoimmune disorders such as psoriasis and/or multiple sclerosis can potentially achieve gross peak annual worldwide sales of about $370 million and/or over $4 billion, respectively. VTP-38443 in acute coronary syndrome (ACS) can potentially achieve gross peak annual worldwide sales of about $750 million. VTP-38543 in atopic dermatitis can potentially achieve gross peak annual worldwide sales of about $150 million.
- **We project cash runway through 2016 covering material catalysts for the two lead programs.** We estimate Vitae ended Q3:14 with about $78 million in cash covering the phase 1 VTP-37948/Alzheimer's biomarker data release by year-end and the phase 2 VTP-34072/type 2 diabetes data release in H1:15, which we believe is likely to validate the drug candidates and the platform (in addition to the $150+ million already paid to Vitae from BI for these two programs).
- **We are initiating coverage of VTAE with an OUTPERFORM rating and a 12-month price target of $21.** Our price target is a 12-month projection of our current fair value calculated using a sum-of-parts with each treatment/indication's value calculated using a 30% annual discount from our net peak sales year to the present day, then applying a 1x-10x premium depending on stage of development to reflect risk.

FYE Dec	2013A	2014E			2015E		
REV (M)	ACTUAL	CURR.	PREV.	CONS.	CURR.	PREV.	CONS.
Q1 Mar	$1.0A	$1.2A		--	$0.8E		--
Q2 Jun	1.0A	1.2A		--	0.8E		--
Q3 Sep	10.3A	7.2E		--	0.8E		--
Q4 Dec	10.3A	1.2E		--	0.8E		--
Year*	**$22.5A**	**$10.7E**		**--**	**$3.1E**		**--**
Change	--	--			--		
	2013A	2014E			2015E		
EPS	ACTUAL	CURR.	PREV.	CONS.	CURR.	PREV.	CONS.
Q1 Mar	$(0.66)A	$(0.47)A		--	$(0.43)E		--
Q2 Jun	(0.34)A	(0.48)A		--	(0.44)E		--
Q3 Sep	0.26A	(0.05)E		--	(0.45)E		--
Q4 Dec	0.46A	(0.40)E		--	(0.47)E		--
Year*	**$0.11A**	**($1.27)E**		**--**	**$(1.79)E**		**--**
P/E	--	--			--		
Change	--	--			--		

Consensus estimates are from Thomson First Call.
** Numbers may not add up due to rounding.*

October 20, 2014

Price
$6.00

Rating
OUTPERFORM

12-Month Price Target
$21

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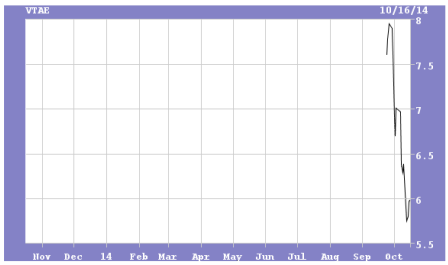
Company Information	
Shares Outst (M)	17.4
Market Cap (M)	$104
52-Wk Range	$5.51 - $8.48
Book Value/sh	$-3.23
Cash/sh	$4.49
Enterprise Value (M)	$163
LT Debt/Cap %	(17)

Company Description

Vitae is a clinical stage emerging pharmaceutical company discovering and developing small-molecule drug candidates to treat large market indications with unmet medical needs.



Source: Thomson Reuters

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Table of Contents

INVESTMENT THESIS

Vitae, located in Fort Washington, Pennsylvania, is a clinical-stage emerging pharmaceutical company focused on discovering and developing novel, small molecule drugs for large market diseases with significant unmet medical needs. The company's proprietary structure-based drug design platform called Contour® has provided multiple, high-quality product candidates which have attracted over $150 million in collaboration funding from big pharma. Vitae has two partnered product candidates in the clinic and several wholly-owned product candidates in preclinical development. The most advanced product candidates include VTP-34072, currently being tested in phase 2 for the treatment of type 2 diabetes and VTP-37948 which is in phase 1 for the treatment of Alzheimer's disease. Both products are being developed by Vitae's partner Boehringer Ingelheim GmbH (BI). Earlier-stage product candidates are currently unpartnered and include VTP-43742 for the treatment of autoimmune disorders, VTP-38443 for the treatment of acute coronary syndrome (ACS) and VTP-38543 for the treatment of atopic dermatitis (eczema). Vitae intends to develop and commercialize these programs and/or to strategically partner programs as appropriate. We have projected clinical development and potential regulatory approvals so that the first product could be launched in late 2019. We project the first full year of profitability in 2021 from revenues of about $119 million. We estimate Vitae ended Q3 2014 with about $78 million in cash and equivalents which we project can last through 2016. We project cash runway could cover transforming clinical data releases from multiple product candidates. We also anticipate upside potential from additional partnerships around their currently unpartnered product candidates and that Vitae's future clinical success could result in the company's acquisition.

KEY POINTS

1. **We consider VTAE to be an attractive investment due to its state-of-the-art structure-based drug design Contour® platform which rapidly produces high-quality small molecule drug candidates with solid intellectual property and has, in our view, been validated by investment of over $150 million from big pharma.**
2. **We believe Vitae's cutting-edge Contour® platform creates best-in-class drug candidates.** Contour® is based on state-of-the-art structural-based drug design in which the structure of the x-ray crystallized target is matched with optimized drug candidates. The company applies its Contour® technology to biologically validated therapeutic targets which have historically been difficult to develop drugs against due to potency, selectivity, pharmacokinetics, or patentability issues. Contour®'s computational software uses artificial intelligence and sophisticated algorithms to model the assembly of molecular fragments into fully elaborated, drug-like structures that precisely fit each target's 3-dimensional binding site. These molecules are then assessed by Contour®'s state-of-the-art scoring function to identify the most promising and drug-like structures. Together, these functions allow Vitae to rapidly focus on structures with the highest potential. The company chemically synthesizes, comprehensively tests and critically evaluates these novel structures rapidly, iterating each new data set back into the design process until it identifies product candidates with demonstrable first- or best-in-class potential. Vitae has leveraged Contour® to rapidly achieve preclinical proof-of-concept with a qualified lead product candidate in less than 18 months.
3. **The company's large and diversified pipeline reduces risk, in our view.** Currently, the company's lead product candidates include VTP-34072 (in phase 2a for type 2 diabetes) and VTP-37948 (in phase 1 for Alzheimer's). Both product candidates are partnered with Boehringer Ingelheim GmbH (BI), which has provided Vitae with over $150 million, including upfront cash, research funding and success-based milestone payments as well as equity investment. We believe this level of non-dilutive funding validates the Contour® platform and the competency of Vitae. In addition, Vitae has its own preclinical candidates, including VTP-43742 for autoimmune disorders, VTP-38443 for acute coronary syndrome (ACS), and VTP-38543 for atopic dermatitis.
4. **Due to the partnership with Boehringer Ingelheim for the two lead drug candidates, we believe commercial risks are reduced and project over one billion dollars in gross peak annual sales beginning in 2023.** As Boehringer Ingelheim has a commercial salesforce for diabetes and CNS disorders, we believe its experience reduces commercial risk for VTP-34072 in diabetes/metabolic syndrome and for VTP-37948 in Alzheimer's disease. With launches in late 2019 and 2020, respectively, we project both drug candidates could achieve almost $1 billion in gross sales each in 2023 and VTP-34072 can achieve over $2 billion gross peak annual worldwide sales in 2025 and VTP-37948 can achieve almost $3 billion.
5. **With about $78 million in cash estimated by us at the end of Q3, we project runway through 2016 which covers multiple clinical data releases** including: phase 2 data releases from VTP-34072 in diabetes/metabolic syndrome and phase 1 and potentially phase 2 data releases for VTP-37948 in Alzheimer's plus phase 1 data releases from VTP-43742 in autoimmune diseases, VTP-38443 in ACS and VTP-38543 in atopic dermatitis.
6. **We believe execution risk is reduced by management's quality and experience.** Jeff Hatfield is CEO and transformed Vitae from a start-up in 2004 to a product-focused discovery and development machine which has been funded by over $150 million from big pharma since the last venture round in 2004. Prior to joining Vitae in 2004, Mr. Hatfield held various positions at Bristol-Myers Squibb and contributed to the success of Pravachol®, Plavix®, Avapro®, Abilify®, Reyataz®, and Atripla®. Given his industry experience and his ability to attract significant partnership revenues to drug candidates discovered using the Contour® platform,

we believe he is likely to continue his success. Hatfield has also surrounded himself with an impressive management team who we believe reduce execution risk.

7. **We consider intellectual property around Vitae's product candidates to be strong due to multiple patents, including composition of matter, expiring in 2030 and beyond.** The most advanced product candidates have composition of matter and methods of treatment in major markets worldwide. These patents and patent applications, if granted, are expected to provide Vitae with intellectual property protection until at least 2030 and beyond.

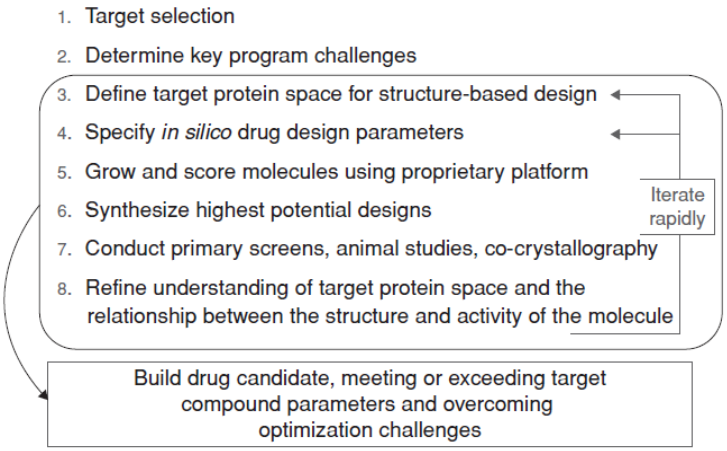
8. **In our view, VTAE, currently trading around $6 per share, is at an attractive valuation compared with our 12-month price target of $21 per share.** Based on the sum of a 30% annual discount and a 1x-10x premium range on our net peak annual sales estimate for each product and indication in the clinic, and projecting 12 months, we calculate VTAE's 12-month price target to be about $21 per share. We consider VTAE's peers to be platform biotech companies including ARNA, LXRX, SGMO, and RGLS. We project a current acquisition value at about $500 million market cap or about $29 per share.

CONTOUR® PLATFORM

SMALL MOLECULE CLINICAL CANDIDATES ARE OF PARTICULAR INTEREST TO BIG PHARMA.

Small-molecule drugs represent the majority of FDA drug approvals and continue to do so, with 25 out of the 27 FDA approved drugs in 2013. This class of drug candidates is the most desirable among big pharma, in our view–especially new chemical entities (NCEs) with oral bioavailability and strong IP. Historically, structure-based drug design techniques have been used to create NCEs as part of the drug discovery process.

FIGURE 1: CONTOUR® DISCOVERY PROCESS



Source: Company data; Wedbush Securities, Inc.

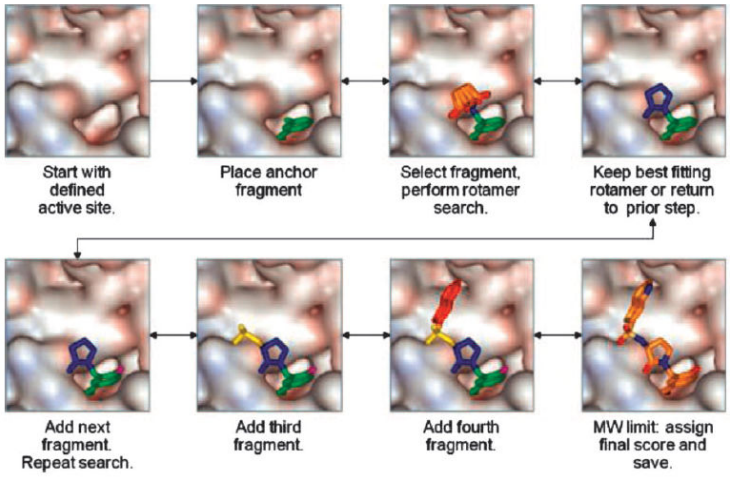
THE CONTOUR® PLATFORM OFFERS SEVERAL ADVANTAGES OVER TRADITIONAL DRUG DISCOVERY APPROACHES

The efficiency of conventional structure-based drug design may be limited by several factors: 1) screening thousands-to-millions of molecules one at a time, 2) use of molecules generated from a different target, 3) lack of high affinity molecules in the library, 4) having to test all potential structures of each molecule, and 5) lack of a useful scoring function to predict binding. Vitae's Contour® platform addresses these inefficiencies with proprietary design and modeling software coupled with a state-of-the-art scoring system. Instead of using the conventional trial and error approach to drug optimization, the Contour® platform accelerates the optimization of lead compounds through an iterative process guided by the structure and desired biological effect.

The software component of the Contour® platform increases the probability of constructing an NCE that best fits into the target. X-ray crystal structures and/or homology modeling are coupled with Dynamic Fragment Selection software which uses the physical characteristics of the binding site to assemble viable fragments synthesized during the growth process. In addition, with each process, Contour®'s Artificial Intelligence improves performance for the next round of iteration.

The initial fragment growth process begins with a starting site in the binding pocket which is derived from a crystal structure of a target with its ligand or by selecting a biologically defined interaction site. Contour® then starts selecting fragments from a library based on a defined list of sequences, using probability-based selection, or through the active guiding.

FIGURE 2: CONTOUR® GROWTH ALGORITHM MOLECULAR ASSEMBLY



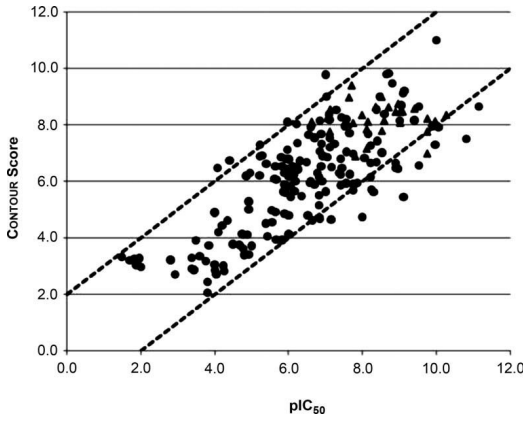
Source: Company data; Wedbush Securities, Inc.

Contour® performs intelligent fragment selection to limit the time it takes to build a fragment. On average, each fragment has 6 incoming, 6 outgoing attachment points and 6 possible rotational isomers for each bond. Therefore, for a library of 10,000 fragments there would be 2.6 x 10^6 different orientation possibilities for each fragment. In order to reduce the library to a more manageable set an algorithm is used for random sampling to reduce the number of calculations needed and decrease the number of less desirable fragments that have to be evaluated. In order to favor the best fit, Vitae has developed and implemented Dynamic Fragment Selection into the Contour® platform that uses the physical characteristics of the binding pocket to narrow down and select fragments for the growth process. Once a fragment is added, Contour® performs a rotamer search to optimize location and orientation to minimize the molecule's energy state and optimize the target-ligand binding.

CONTOUR®'S SCORING FUNCTION

Fragments are assessed using Contour®'s Scoring Function and a prediction of the fragments binding affinity and/or potency of enzyme activity is provided as a numerical score that is approximately equivalent to pKi (-logK_1). Plotting the Contour® score as a function of IC_{50} shows how accurate the scoring function is in determining fragment binding affinity. The Contour® Scoring Function has shown utility in scoring target-ligand complexes across several classes of protein targets and has allowed rapid and effective engineering and assessment of target interactions to optimize product candidates. Once the compounds are engineered and ranked, a chemist synthesizes and then screens them using the appropriate biological and cell based assays. Those with the best in-vitro profiles are then assessed for their pharmacokinetic profile in mice and rats. Further optimization includes co-crystallization with the target.

FIGURE 3: CONTOUR® SCORE IS A STRONG INDICATOR FOR THE MOLECULES IC_{50}



Source: Company data; Wedbush Securities, Inc.

In addition to the Contour® platform, Vitae's scientific staff includes experts in x-ray crystallography, molecular modeling, medicinal chemistry and biology. We believe that the Contour® platform and internal expertise allow efficiently designed molecules. In addition, initial drug design to animal testing takes only months as opposed to years seen with traditional drug discovery approaches. Vitae's drug discovery approach accelerates the time to chemistry solution and animal proof of concept. The company has consistently been able to engineer novel compounds in 7 months or less and provide animal proof of concept data with oral dosing in 16 months or less.

FIGURE 4: DEVELOPMENT TIMING

Selected Target	Novel Chemistry Solution	Animal Proof-of-Concept
Renin(1)	7 months	14 months
11β HSD1	2 months	16 months
BACE	6 months	14 months
RORγt	2 months	10 months
LXRβ	6 months	12 months

Source: Company data; Wedbush Securities, Inc.

PIPELINE OVERVIEW

FIGURE 5: REDUCED PIPELINE RISK FROM MULTIPLE CANDIDATES

PRODUCT PIPELINE			
PRODUCT CANDIDATE	**INDICATION (TARGET)**	**COMMERCIAL RIGHTS**	**STAGE OF CLINICAL DEVELOPMENT AND ANTICIPATED MILESTONES**
VTP-34072	Type 2 Diabetes and metabolic syndrome (11β HSD1)	BI: Global	• Phase 1 clinical trial completed. All primary endpoints met • Phase 2 clinical trial expected to be initiated in July 2014
VTP-37948	Alzheimer's Disease (BACE1)	BI: Global	• Phase 1a clinical trial initiated January 2014 • Phase 1b clinical trial expected to begin in second half of 2014
VTP-43742	Psoriasis, Multiple Sclerosis, other autoimmune diseases (RORγt)	Vitae: Global	• Phase 1a clinical trial expected to begin in first half of 2015 • Phase 1b clinical trial expected to begin in second half of 2015
VTP-38443	Acute Coronary Syndrome (LXRβ)	Vitae: Global	• Phase 1 clinical trial expected to begin in first half of 2016
VTP-38543	Atopic Dermatitis (LXRβ)	Vitae: Global	• Phase 1 clinical trial expected to begin in second half of 2015

Source: Company data; Wedbush Securities, Inc.

Vitae has multiple product candidates in development, including the two lead programs which are partnered with Boehringer Ingelheim--VTP-34072 for type 2 diabetes and metabolic syndrome and VTP-37948 for Alzheimer's disease. Vitae has additional early-stage candidates for diseases, including autoimmune disorders, acute coronary syndrome, and atopic dermatitis.

MODEL, VALUATION, RISKS AND CAPITALIZATION

WE PROJECT FULL-YEAR PROFITABILITY IN 2021 FOLLOWING PRODUCT LAUNCHES STARTING IN LATE 2019

Our 2021 full-year profitability estimate depends on the clinical, regulatory, and commercial success for projected US launches of VTP-34072 in type 2 diabetes in late 2019, VTP-37948 in Alzheimer's disease in 2020, and VTP-43742 in autoimmune diseases in 2020. We project net revenues of about $119 million and GAAP EPS of $0.98 in 2021.

Presuming positive phase-2 results by year-end 2014 and subsequent clinical, regulatory, and commercial success, we project a potential US launch of VTP-34072 in type 2 diabetes in late 2019 with the potential to achieve gross peak annual sales of about $2.4 billion worldwide in 2025. For VTP-37498 treatment of Alzheimer's disease, we project US launch in 2020 and the potential to achieve gross peak annual worldwide sales of about $3 billion in 2025.

With achievement of clinical proof-of-concept efficacy for additional indications and drug candidates, we see significant upside to our revenue projections. For VTP-43742, for autoimmune indications, we estimate a potential launch in 2020 and gross peak annual worldwide sales could reach about $370 million to about $4 billion in 2025 depending on indication. For VTP-38443, there is the potential to treat acute coronary syndrome and achieve about $750 million in gross peak annual worldwide sales in 2025 and VTP-38543 could achieve about $150 million for atopic dermatitis.

FIGURE 6: MODEL

Vitae, Inc. (VTAE:NASDAQ) — **Wedbush Securities, Inc.**
Historical and Projected Income Statement — Liana Moussatos, PhD
(In thousands except per share data)

	2013A	2014E					2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
	FY:13A	Q1A	Q2A	Q3E	Q4E	FY:14E	FY:15E	FY:16E	FY:17E	FY:18E	FY:19E	FY:20E	FY:21E	FY:22E	FY:23E	FY:24E	FY:25E
Gross Sales:																	
VTP-34072	-					-	-	-	-	-	1,483	62,046	411,760	1,396,469	3,858,191	7,898,438	13,037,176
VTP-37948	-					-	-	-	-	-	1,483	36,952	136,071	402,006	923,761	1,643,207	2,428,129
VTP-43742	-					-	-	-	-	-	-	10,889	83,056	288,888	811,169	1,745,285	2,966,804
VTP-43742	-					-	-	-	-	-	-	2,120	26,886	84,339	180,099	274,499	333,909
VTP-38443	-					-	-	-	-	-	-	12,085	162,600	558,210	1,459,452	2,859,636	4,405,808
VTP-38543	-					-	-	-	-	-	-	-	1,711	39,989	128,026	288,181	474,199
	-					-	-	-	-	-	-	-	1,437	8,929	26,210	54,814	89,850
	-																
Revenues:	-																
Product Sales/Royalties																	
VTP-34072	-	-	-	-	-	-	-	-	-	-	148	3,660	13,480	46,402	116,557	205,678	293,353
VTP-37948	-	-	-	-	-	-	-	-	-	-	-	1,089	8,344	32,262	105,948	228,466	375,189
VTP-43742	-	-	-	-	-	-	-	-	-	-	-	1,060	13,087	37,802	77,183	113,533	133,809
VTP-43742	-	-	-	-	-	-	-	-	-	-	-	5,982	79,357	260,959	667,871	1,257,619	1,830,916
VTP-38443	-	-	-	-	-	-	-	-	-	-	-	-	847	19,569	57,995	123,877	193,097
VTP-38543	-	-	-	-	-	-	-	-	-	-	-	-	711	4,134	11,320	22,387	34,042
	-	-	-	-	-	-	-	-	-	-	-	-					
Total Net Product Revenues	-	-	-	-	-	-	-	-	-	-	148	11,791	115,826	415,235	1,361,488	2,867,056	4,782,300
Collaborative Revenues	22,513	1,165	1,165	7,165	1,165	10,658	3,058	3,058	3,058	3,058	3,058	3,058	3,058	3,058	3,058	3,058	3,058
Total Revenues	$ 22,513	$ 1,165	$ 1,165	$ 7,165	$ 1,165	$ 10,658	$ 3,058	$ 3,058	$ 3,058	$ 3,058	$ 3,206	$ 14,849	$ 118,884	$ 418,293	$ 1,364,546	$ 2,870,114	$ 4,785,358
Total COGS	-	-	-	-	-	-	-	-	-	-	15	1,179	11,583	41,523	136,149	286,706	478,230
Gross Margin	$ 22,513	$ 1,165	$ 1,165	$ 7,165	$ 1,165	$ 10,658	$ 3,058	$ 3,058	$ 3,058	$ 3,058	$ 3,191	$ 13,670	$ 107,301	$ 376,769	$ 1,228,397	$ 2,583,409	$ 4,307,128
Operating Expenses:																	
R&D	14,917	4,713	4,713	5,807	5,923	21,155	26,034	29,128	31,529	26,141	28,296	30,628	33,153	35,886	38,844	42,046	45,512
SG&A	5,406	1,315	1,315	2,353	2,376	7,358	9,528	9,784	10,181	14,948	29,774	42,141	43,852	45,633	47,486	49,414	51,420
Acquired in-process R&D	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Operating Expenses	$ 20,322	$ 6,027	$ 6,027	$ 8,159	$ 8,299	$ 28,512	$ 35,562	$ 38,912	$ 41,710	$ 41,089	$ 58,070	$ 72,769	$ 77,005	$ 81,519	$ 86,330	$ 91,460	$ 96,932
Operating Income (Loss)	2,190	(4,863)	(4,863)	(995)	(7,135)	(17,854)	(32,504)	(35,854)	(38,652)	(38,031)	(54,879)	(59,099)	30,296	295,251	1,142,067	2,491,949	4,210,196
Other Income / (Expense), net	327	109	109	60	73	351	325	320	319	319	319	319	319	319	319	319	319
Interest Income	69	15	15	7	(2)	34	103	38	(54)	(149)	(261)	(415)	(488)	(317)	558	3,015	7,788
Interest (Expense)	(1,425)	(271)	(271)	101	78	(362)	98	-	-	-	-	-	-	-	-	-	-
Total other (expenses) income	(1,028)	(147)	(147)	168	149	23	526	358	265	170	59	(95)	(169)	2	877	3,334	8,107
Income Before Income Taxes	$ 1,162	$ (5,010)	$ (5,010)	$ (827)	$ (6,986)	$ (17,832)	$ (31,978)	$ (35,496)	$ (38,387)	$ (37,860)	$ (54,820)	$ (59,195)	$ 30,127	$ 295,252	$ 1,142,945	$ 2,495,283	$ 4,218,303
Deemed Dividend to preferred stockholders	-																
(Provision)/benefit for Income Taxes	-	-	-	-	-	-	-	-	-	-	-	-	(9,074)	(115,148)	(445,748)	(973,160)	(1,645,138)
Tax Rate	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	12.3%	39.0%	39.0%	39.0%	39.0%
Net Income (Loss)	$ 1,162	$ (5,010)	$ (5,010)	$ (827)	$ (6,986)	$ (17,832)	$ (31,978)	$ (35,496)	$ (38,387)	$ (37,860)	$ (54,820)	$ (59,195)	$ 21,052	$ 180,104	$ 697,196	$ 1,522,123	$ 2,573,165
Stock-based compensation	100	56	56	1,875	1,875	3,862	5,013	5,265	5,310	5,317	5,317	5,317	5,317	5,317	5,317	5,317	5,317
EPS	$ 0.12	$ (0.48)	$ (0.48)	$ (0.16)	$ (0.51)	$ (1.55)	$ (2.07)	$ (2.20)	$ (2.29)	$ (2.19)	$ (2.96)	$ (3.09)	$ 0.73	$ 7.91	$ 30.48	$ 65.11	$ 107.46
GAAP EPS	$ 0.11	$ (0.47)	$ (0.48)	$ (0.05)	$ (0.40)	$ (1.27)	$ (1.79)	$ (1.92)	$ (2.01)	$ (1.92)	$ (2.70)	$ (2.83)	$ 0.98	$ 8.15	$ 30.72	$ 65.34	$ 107.68
Weighted Average Shares Outstanding	10,674	10,578	10,482	17,371	17,521	13,988	17,896	18,496	19,096	19,696	20,296	20,896	21,496	22,096	22,896	23,296	23,896
Cash	$32,454	$19,264	$18,141	$77,945	$69,462	$69,462	$32,584	($2,711)	($40,896)	($78,555)	($133,215)	($194,718)	($183,311)	($34,229)	$580,938	$1,983,693	$4,422,373
Cash Per Share	$3.04	$1.82	$1.73	$4.49	$3.96	$4.97	$1.82	($0.15)	($2.14)	($3.99)	($6.56)	($9.32)	($8.53)	($1.55)	$25.60	$85.15	$185.06
Net Cash	$27,650	$14,460	$4,470	$58,371	$54,292	$54,292	$31,740	($2,711)	($40,896)	($78,555)	($133,215)	($194,718)	($183,311)	($34,229)	$580,938	$1,983,693	$4,422,373
Net Cash Per Share	$2.59	$1.37	$0.43	$3.36	$3.10	$3.88	$1.77	($0.15)	($2.14)	($3.99)	($6.56)	($9.32)	($8.53)	($1.55)	$25.60	$85.15	$185.06
Cash Burn (Generation)	$28,593					($208)	$73,679	$72,095	$74,984	$74,459	$91,460	$98,302	$25,393	($112,282)	($578,367)	($1,365,955)	($2,401,880)

Source: Company data; Wedbush Securities, Inc.

FIGURE 7: VALUATION

Vitae Product Pipeline Valuation		Eligible #	Pricing	Gross Sales		Net Revs	Peak		Estimated / Actual	Discount	Estimate	Fair Value
Product	Indication	Patients	$/Patient	($000)	Year	($000)	Penetration	Multiple	Launch	Rate	Fair Value	per Share
VTP-34072 (WW)	Diabetes / Metabolic Syndrome	67,152,070	$1,952	$2,428,129	2025	$293,353	2%	4	12/4/2019	30%	$195,304	$11.15
VTP-37948 (WW)	Alzheimer's Disease	8,730,000	$5,226	$4,318,067	2026	$910,566	10%	3	9/4/2020	30%	$164,829	$9.41
VTP-43742 (WW)	Psoriasis	609,167	$4,978	$369,925	2027	$144,635	11%	2	11/4/2020	30%	$14,478	$0.83
VTP-43742 (WW)	MS	857,143	$49,683	$4,405,808	2025	$1,830,916	8%	2	11/4/2020	30%	$115,764	$6.61
VTP-38443 (WW)	ACS	600,000	$30,500	$754,409	2028	$274,449	4%	1	12/4/2021	30%	$7,994	$0.46
VTP-38543 (WW)	Atopic Dermatitis	1,235,000	$4,978	$148,850	2028	$50,055	2%	1	8/4/2021	30%	$1,543	$0.09

We use multiples to account for clinical and regulatory risk at various stages of development.			Stock	MktCap ($000)	Upside
1: in preclinical testing	6: in Phase 3	12-month Price Target	$20.55	$360,134	243%
2: passed preclinical	7: Phase 3 data	Total Pipeline Value	$28.53	$499,913	376%
3: IND filing/stable mature product	8: regulatory review	Current Cash	$4.45	$77,945	
4: Phase 1 data	9: approved	Current Stockprice:	$6.00	$104,228	
5: Phase 2 data	10: launched	Primary Sharecount	17,371		

Source: Company data; Wedbush Securities, Inc.

In our view, VTAE trading at about $6 per share, or about $100 million market cap is at an attractive valuation compared with our price target of $21 per share, or about $360 million market cap. Based on the sum of a 30% annual discount and a 1x-10x premium range on our net peak annual sales estimate for each product and indication in the clinic, and projecting 365 days, we calculate VTAE's 12-month price target. We see VTAE's peers to be platform biotech companies including ARNA, LXRX, SGMO, and RGLS. We project a current acquisition value at about $500 million market cap or $29 per share for VTAE.

RISK TO THE ATTAINMENT OF OUR PRICE TARGET

Clinical Risks: Despite producing high-quality product candidates and encouraging initial clinical and preclinical data, Vitae has not completed phase-3 testing with any product candidate and, in general, the majority of clinical candidates fail. Vitae with BI are also developing a treatment for Alzheimer's disease in which the vast majority of clinical candidates have failed. Vitae is also dependent on BI for the proper development of their two lead product candidates VTP-34072 and VTP-37948. Both Vitae and BI use third parties to conduct preclinical and clinical testing which we view as higher risk as we believe third parties may be less motivated to reduce execution risk. Near-term clinical risks including data releases from the two lead programs are the highest risks to our price target at this time, in our view.

Regulatory Risks: Despite Vitae's management having big pharma experience, Vitae has not achieved regulatory approval for any product candidate.

Manufacturing Risks: On one hand, we view manufacturing risk to be lower for small molecule drug candidates versus biologics and oligonucleotides; however, Vitae relies on third parties for the manufacture of their product candidates for preclinical, clinical, and potential commercial manufacture and we view third parties as less motivated, in general. Also, if Vitae succeeds at obtaining regulatory approval for a product candidate, the current purchase order supply arrangements will need to be augmented with long-term supply arrangements. Vitae intends to also work with additional manufacturers to provide active pharmaceutical ingredients (APIs) and fill-and-finish services prior to pursuing regulatory approval. BI is responsible for the manufacture of API and fill-and-finish services for both 11β-HSD1 and BACE.

Commercial Risks: For their unpartnered programs, Vitae anticipates retaining US commercial rights in specialty markets and establish regional partnerships to commercialize outside the United States. At this time, Vitae does not have a sales force or marketing capabilities. For the two lead programs, Vitae expects BI to commercialize these products with their sales and marketing group.

Competition Risks: Vitae's product candidates, if approved, will compete with currently marketed treatments and potentially with product candidates currently in development focusing on the same mechanism of action which include: 1) 11β-HSD1 competition from Bristol-Myers Squibb, Eli Lilly & Co., and Roche Holding AG which are also testing their inhibitors in clinical trials; 2) BACE competition from Merck & Co., AstraZeneca PLC and Eisai Co., Ltd. in collaboration with Biogen Idec which are studying BACE inhibitors in clinical trials; 3) RORγt competition from potentially multiple companies which are actively assessing RORγt inhibitors in preclinical studies; and 4) LXRβ competition from Bristol-Myers Squibb which is testing an LXRβ inhibitor in cardiovascular clinical trials and Alexar Therapeutics, Inc., which is developing an LXRβ inhibitor for dermatologic conditions.

Intellectual Property Risks: Due to the nature of Vitae's business model, we consider intellectual property risks to be low as the company discovers its own product candidates and has composition-of-matter protection to 2030 and beyond.

Financial Risks: Vitae is a development-stage emerging pharmaceutical company and, despite receiving substantial partnership income from Boehringer Ingelheim, they have no product sales or royalty income and are unlikely to before late 2019. We project that the company is likely to end Q3 2014 with about $78 million in cash and equivalents which we project could last through 2016.

UPCOMING CATALYSTS

With about $78 million in cash estimated at the end of Q3, we project runway through 2016, which covers multiple clinical data releases including: phase-2 data releases from VTP-34072 in diabetes/metabolic syndrome and VTP-37948 in Alzheimer's plus phase-1 data releases from VTP-43742 in autoimmune diseases, VTP-38443 in ACS and VTP-38543 in atopic dermatitis.

FIGURE 8: ANTICIPATED MILESTONES (*OUR ESTIMATES)

YE:14	**VTP-37948/ALZ: PHASE 1 AND BIOMARKER DATA RELEASE**
H1:15	**VTP-34072/T2D-META: PHASE 2 DATA RELEASE**
YE:15*	**VTP-37948/ALZ: PHASE 2 DATA RELEASE**
YE:15	**VTP-43742/AUTOIMMUNE: PHASE 1 POC DATA RELEASE**
Q2:16	**VTP-38543/ADERM: PHASE 1 DATA RELEASE**
YE:16	**VTP-38443/ACS: PHASE 1 DATA RELEASE**
H2:17*	**VTP-38543/ADERM: PHASE 2 DATA RELEASE**
H1:18*	**VTP-38443/ACS: PHASE 2 DATA RELEASE**
H2:18*	**VTP-34072/T2D-META: PHASE 3 DATA RELEASE**
H1:19*	**VTP-37948/ALZ: PHASE 3 DATA RELEASE**
H1:19*	**VTP-43742/AUTOIMMUNE: PHASE 3 DATA RELEASE**
H2:19*	**VTP-34072/T2D-META: PDUFA/LAUNCH IN US (BI)**
YE:19*	**VTP-38543/ADERM: PHASE 3 DATA RELEASE**
Q1:20*	**VTP-37948/ALZ: PDUFA**
Q3:20*	**VTP-37948/ALZ: LAUNCH IN US (BI)**
MID:20*	**VTP-43742/AUTOIMMUNE: PDUFA**
MID:20*	**VTP-38443/ACS: PHASE 3 DATA RELEASE**
Q4:20*	**VTP-43742/AUTOIMMUNE: LAUNCH IN US**
H1:21*	**VTP-38543/ADERM: PDUFA**
Q3:21*	**VTP-38543/ADERM: LAUNCH IN US**
Q4:21*	**VTP-38443/ACS: LAUNCH IN US**

Source: Company data; Wedbush Securities, Inc.

MANAGEMENT TEAM

We believe execution risk is reduced by management's quality and experience. We are especially impressed with their dedication to constantly incorporating cutting-edge technology into their Contour® drug discovery platform in order to produce the highest quality drug candidates. Jeff Hatfield is CEO and transformed Vitae from a start-up in 2004 to a product-focused discovery and development machine which has been funded by about $152 million from big pharma since the last venture round in 2004. Prior to joining Vitae in 2004, Mr. Hatfield held various positions at Bristol-Myers Squibb and contributed to the success of Pravachol®, Plavix®, Avapro®, Abilify®, Reyataz®, and Atripla®. Given his big pharma experience and his ability to attract significant partnership revenues to drug candidates discovered using the Contour® platform, we believe he is likely to continue his success. We believe Mr. Hatfield has also surrounded himself with an impressive management team who we believe reduce execution risk.

FIGURE 9: VITAE MANAGEMENT

Name	Position and Past Experience
Jeffrey Hatfield	**Chief Executive Officer, President and Director.** Jeff Hatfield joined Vitae Pharmaceuticals as President, Chief Executive Officer and a member of the Board of Directors in March 2004. Since becoming CEO, he has successfully transitioned Vitae from a novel technology start-up to a thriving product-focused discovery and development engine with a robust pipeline that includes multiple programs advancing in human clinical trials. Mr. Hatfield has focused the company on high value

	therapeutic areas with significant global unmet medical need, including kidney disease, diabetes, Alzheimer's Disease and atherosclerosis. Notably, since joining the company Mr. Hatfield has grown shareholder value in a uniquely capital efficient way – Vitae's last venture round dates back to 2004, his first year with the company. Prior to joining Vitae Pharmaceuticals, Mr. Hatfield worked at Bristol-Myers Squibb in a variety of executive positions, including: Senior Vice President of BMS's Virology and Immunology Divisions, where he was responsible for all aspects of the $1 billion business; President and General Manager, Canada; and, Vice President, U.S. Managed Health Care. While at BMS, Mr. Hatfield was directly associated with several product successes, including Pravachol®, Plavix®, Avapro®, Abilify®, Reyataz® and Atripla®. Mr. Hatfield holds an M.B.A. from The Wharton School, University of Pennsylvania and a bachelor's degree in Pharmacy from Purdue University, where he is a Distinguished Alumni. He is a member of the Board of Directors of the Biotechnology Industry Organization (BIO), serving on the Emerging Company Section, and the Capital Formation and Intellectual Property Committees. He is also a member of the Advisory Committees for Purdue University's College of Pharmacy, Drexel University's LeBow College of Business and the Chapman-KGI School of BioPharmacy.
Richard Gregg	**Chief Scientific Officer.** Dr. Gregg spent 19 years leading various groups at Bristol-Myers Squibb Research and Development before deciding to join Vitae Pharmaceuticals. Most recently, he was Vice President of Clinical Discovery, responsible for Early Clinical Development, Clinical Pharmacology, Translational Medicine and Biomarker Technologies. Dr. Gregg developed and led the Bristol's efforts in the application of cutting edge science and analytical technologies to the clinical investigation of new drugs. He also served as Vice President of Metabolic and Cardiovascular Drug Discovery, focusing on the discovery of new drugs for diabetes, dyslipidemia, and atherosclerotic vascular disease. Before coming to Bristol, Dr. Gregg spent 10 years at the National Heart, Lung and Blood Institute, where he studied disorders of lipid and lipoprotein metabolism. He has more than 120 publications in leading medical and research journals, and has presented his research findings at national and international meetings. Dr. Gregg earned his B.S. and M.S. from Iowa State University and his M.D. from Stanford University School of Medicine. He did his internship and residency in Internal Medicine at Strong Memorial Hospital in Rochester, New York, and completed his fellowship in Endocrinology and Metabolism at the National Institutes of Health.
Richard Morris	**Chief Financial Officer.** Richard Morris has served as Chief Financial Officer since May 2014. Prior to joining Vitae, Mr. Morris worked at ViroPharma Incorporated, which he joined in 2001, and held a variety of positions including Vice President, Financial Planning and Strategic Analysis from 2012 to 2014, Vice President, Chief Accounting Officer from 2011 to 2014, Controller and Chief Accounting Officer from 2008 April 2011 and Controller from 2005 through 2008. Prior to joining ViroPharma, Mr. Morris worked for KPMG LLP in their Healthcare Assurance practice. Mr. Morris received a bachelor's degree in Accounting from Saint Joseph's University and has been a CPA since 1999.
Arthur Fratamico	**Chief Business Officer.** Arthur Fratamico, R.Ph. has served as Chief Business Officer since May 2014. Prior to joining Vitae, Mr. Fratamico served as chief business officer of Flexion Therapeutics, Inc. from June 2012 through 2014. Prior to Flexion, Mr. Fratamico led the business development efforts, including overseeing numerous licensing transactions and acquisitions, at private biotechnology companies including Trevena, Inc. from 2011 to 2012, Gemin X Pharmaceuticals, Inc. from 2008 to 2011 and MGI Pharma, Inc. from 1999 to 2008. Mr. Fratamico earned a bachelor's degree in pharmacy from the Philadelphia College of Pharmacy and an M.B.A. from Drexel University.

Source: Company data; Wedbush Securities, Inc.

INTELLECTUAL PROPERTY

Intellectual Property: Lead product candidates with composition of matter protection through 2030 and beyond in the U.S. The most advanced product candidates have composition of matter and methods of treatment in major markets worldwide. These patents and patent applications, if granted, are expected to provide Vitae with intellectual property protection for the six programs until at least 2030 and beyond.

As of June 30, 2014, Vitae had 19 issued patents and 25 pending applications in the United States, 63 issued patents and 187 pending applications in foreign jurisdictions, and 5 pending international applications filed under the Patent Cooperation Treaty (PCT). The IP covers the lead candidates and additional compounds active against chosen disease targets. For these there are 2 issued patents and 7 pending in the US, 6 issued patents and 79 pending ex-US, and 3 pending PCT cover compositions of matter or methods of use of the most advanced product candidates. All candidates were developed in-house at Vitae and thus, their IP is unencumbered as they do not owe material royalties to another party.

FIGURE 10: INTELLECTUAL PROPERTY PROTECTION THROUGH 2033 IN THE U.S.

PRODUCT / TARGET	Status	Expiration	Current Filing Scope
VTP-34072 / 11β-HSD1	Granted US; Pending RoW	2030	Broadly Filed
VTP-37948 / BACE	Pending	2033	Broadly Filed
VTP-43742 / RORγt	Pending	2035	US Provisional Application
VTP-38442 / LXRβ – Aderm	Pending	2033	PCT
VTP-38443 / LXRβ – ACS	Pending	2033	PCT
VTP-# / Immuno-oncology	Pending	2035	US Provisional Application

Source: Company data; Wedbush Securities, Inc.

VTP-34072 inhibitor of 11β-HSD1 for type 2 diabetes—patent expirations in 2030 and 2031: Vitae's most advanced 11β-HSD1 inhibitor for type 2 diabetes, VTP-34072, is partnered with BI. VTP-34072 intellectual property includes two issued US patents and two pending, one issued ex-US patent and 72 pending in Europe, Japan, Taiwan, Canada, Australia, Brazil, China and India. Composition of matter patent protection expires in November 2030, not including possible extensions. However, due to a US Patent and Trademark Office (USPTO) delay, additional patent term adjustment resulted in an extension to September 2031. (Additional extensions may be granted if there are regulatory delays.

VTP-37948 inhibitor of BACE for Alzheimer's disease—patent expiration in 2033: Vitae's most advanced BACE inhibitor compound for Alzheimer's, VTP-37948, is partnered with BI. Intellectual property protection includes one pending US patent application, one pending PCT application, and six pending patent applications in ex-US jurisdictions. Any patents that may issue from these applications will expire in August 2033, not including possible extensions due to patent office or regulatory delay(s).

VTP-43742 inhibitor of RORγt for autoimmune disease—patent expiration in 2035: Vitae's most advanced RORγt inhibitor for autoimmune diseases is VTP-43742. This candidate is covered by a U.S. provisional application filed in February 2014 and will allow filing of worldwide patent applications. Any patents that may issue from these applications could expire as late as February 2035, not including possible extensions due to patent office or regulatory delay.

VTP-38443 selective agonist of LXRβ for Acute Coronary Syndrome (ACS)—patent expiration in 2033: Vitae's most advanced LXR selective agonist for ACS is VTP-38443, which is covered by a published PCT application. The PCT application will allow for worldwide patents covering the U.S., Europe, Japan, Canada, Australia, Brazil, China and India. Any patents that may issue from these applications will expire in March 2033, not including possible extensions due to patent office or regulatory delay(s).

VTP-38543 selective agonist of LXRβ for atopic dermatitis—patent expiration in 2033: Vitae's selective agonist for atopic dermatitis is VTP-38543, which is covered by a published PCT application which will allow for worldwide patents covering the U.S., Europe, Japan, Canada, Australia, Brazil, China and India. Any patents issuing from these applications will expire in March 2033, not including possible extensions.

Contour® Drug Discovery Platform—trade secrets and know-how: With respect to Vitae's proprietary structure-based drug discovery platform called Contour, the company protects its intellectual property through trade secrets and know-how.

BUSINESS DEVELOPMENT

Vitae currently has two collaborations with Boehringer Ingelheim (BI) relating to VTP-34072/11β-HSD1 for the treatment of type 2 diabetes and VTP-37948/BACE for the treatment of Alzheimer's.

For 11β-HSD1 (VTP-34072) Vitae's collaboration with BI began with a research collaboration and license agreement to combine both of their 11β-HSD1 drug discovery programs and to provide BI with exclusive rights for type 2 diabetes and certain related metabolic disease conditions, such as dyslipidemia, obesity and hypertension. As of June 30, 2014, for this agreement Vitae has received $74.2 million including a $15 million equity investment, $22.2 million for upfront milestone and research funding and $37 million in success-based development milestone payments. Vitae may also receive up to $278 million in additional milestone payments--including up to $153 million for development and regulatory milestone payments, and up to $125 million for commercialization milestones. Vitae may also receive tiered royalty payments ranging from upper-single-digit to low-double-digit percentages based on net sales of potential future products. Vitae also may opt in to fund phase 3 clinical trials to increase royalties. The company is expected to receive $6 million from BI in Q3 2014 as a milestone payment for the first patient dosed in the phase 2 clinical trial which began in July 2014.

For BACE (VTP-37948) Vitae's second research collaboration and license agreement provides BI with an exclusive license to identify, develop and commercialize BACE inhibitors for the treatment of Alzheimer's and other conditions. As of June 30, 2014, Vitae had received $78.2 million from BI related to BACE. This includes a $15 million equity investment, $34.2 in upfront fees and research

funding and $29 million for success-based development milestones. In addition, Vitae can receive up to $326 million for additional milestones based on the first product to achieve certain pre-specified events, including up to $176 million for development and regulatory milestone payments and up to $150 million for commercialization milestone payments. Vitae may also receive tiered royalty payments from BI, ranging from upper-single-digit to the low-double-digit percentages, based on the net sales of potential future products. As with VTP-34072 for diabetes, Vitae may opt in to fund phase 3 to increase royalties.

PIPELINE

VTP-34072 FOR TYPE 2 DIABETES (11B-HSD1 INHIBITOR)

Background

VTP-34072 inhibits 11β hydroxysteroid dehydrogenase type 1 (11β-HSD1) and is being developed for type 2 diabetes. The target enzyme, 11β hydroxysteroid dehydrogenase type 1 (11β-HSD1), is responsible for the conversion of the inactive metabolite cortisone to the active metabolite cortisol. Studies have implicated the overproduction of cortisol as a precipitating factor in metabolic syndrome (MetS) (Paredes S et al 2014 Rev Assoc Med Bras 60(1):84-92; Morgan SA et al., 2014 PNAS 111(24):E2482-91). Cortisol is the primary human glucocorticoid and is critical in regulating the stress response in the metabolic, immune and cardiovascular systems. However, persistent excessive cortisol levels can lead to central obesity, dyslipidemia, hypertension and glucose dysregulation. The cortisol synthesis enzyme, 11β-HSD1, is expressed in many tissues, but is most active in metabolically active tissues including liver, adipose, skeletal muscle and brain. Despite expression in several tissues, preclinical studies suggest that inhibition of 11β-HSD1 in adipose tissue may have the most impact for treating MetS.

11B-HSD1 AS A DRUG TARGET FOR TYPE 2 DIABETES

While the activation of cortisol from cortisone is catalyzed by 11β-HSD1 in liver, adipose fat, brain tissues as well as in the periphery, the inactivation of cortisol to cortisone is catalyzed by 11β-HSD2 and is found in the kidney, sweat and salivary glands and protects the kidney from excess cortisol. 11β-HSD1 inhibition in metabolic syndrome patients may be disease-modifying as there is potential to stop progression into type 2 diabetes by reducing liver and adipose tissue cortisol levels—but not in the blood. (This profile may prevent development of Addison's disease.)

A role for 11β-HSD1 in type 2 diabetes was observed in genetic studies—including knockout mice (Kotelevtsev et al 1997 PNAS) which did not become hyperglycemic as well as mice with overexpression in adipose (Masuzaki et al 2001 Science) resulting in weight gain and metabolic syndrome symptoms. Inhibition of 11β-HSD1 reducing cortisol in adipose has been observed to significantly reduce blood glucose, lipids, and blood pressure. A therapeutic inhibitor of 11β-HSD1 should be specific as inhibition of 11 β-HSD2 could result in severe hypertension and hypokalemia due to excess cortisol activity in the kidney as seen in human genetic studies.

DESPITE NUMEROUS DIABETES TREATMENTS, NONE ARE CURES.

In 2010, approximately 17 million Americans were diagnosed with type 2 diabetes and an additional 7 million were estimated to be undiagnosed (Source: American Diabetes Association). The economic cost of only the diagnosed type 2 diabetics in the United States was estimated to be about $245 billion in 2012 with about $9.6 billion spent on prescription treatments. About 85% of type 2 diabetes patients in the US also have metabolic syndrome—a combination of elevated blood pressure, plasma glucose, lipids and weight which in combination significantly increase the risk for cardiovascular disease. Improving one or more of these risk factors may reduce the chance or degree of cardiovascular disease. There are multiple classes of drugs with different mechanisms to treat type 2 diabetes including insulins, GLP-1 analogs, sulfonylureas, biguanides, meglitinides, thiazolidinediones, alpha-glucosidase inhibitors, bile acid sequestrants, DPP-4 inhibitors, and SGLT2 Inhibitors. Despite the numerous choices and because none are curative, type 2 diabetics still progress with their disease and experience significant morbidity and mortality. Consequently, when a new class of diabetes drug is launched, especially new oral medications, they can still achieve multi-billion-dollars in sales (e.g., Januvia / DPPIV inhibitors). With a solid clinical profile, we believe that an 11β-HSD1 inhibitor, such as VTP-34072 may be differentiated due to the potential for a broader beneficial effect by not only lowering glucose, but also lowering lipids, blood pressure and weight.

FIGURE 11: SELECTED CLASSES OF TREATMENTS FOR TYPE 2 DIABETES

Class	Agent	Expected Reduction in HbA1c (%)	Advantages	Disadvantages
Biguanides	Metformin	1.0-2.0	Extensive clinical experience; hypoglycemia rare; improved lipid profile; decreased cardiovascular disease events†; some weight loss in most patients	Gastrointestinal intolerance; lactic acidosis rare (avoid in patients at increased risk, such as men with a serum creatinine level of ≥1.5 mg/dl and women with a serum creatinine level of ≥1.4 mg/dl); vitamin B12 deficiency
Sulfonylurea‡	Glyburide	1.0-1.5	Extensive clinical experience; Hypoglycemia; less durability; weight gain experience	Hypoglycemia; less durability; weight gain
Meglitinides	Nateglinide	0.5-1.0	Short duration of action, hepatic clearance, glucose-dependent postprandial action	Low efficacy, hypoglycemia in some patients, weight gain
Thiazolidinediones	Rosiglitazone	0.5-1.4	Hypoglycemia rare, more durable effect than that of metformin or sulfonylurea, improved lipid profile, some evidence of beneficial effect on coronary atherosclerosis (with pioglitazone)	Edema, heart failure, weight gain, increased risk of long-bone fractures and potential risk of bladder cancer and cardiovascular events (with rosiglitazone)□; use of rosiglitazone highly restricted
DPP-IV inhibitor	Saxagliptin	0.5-0.8	Hypoglycemia rare, infrequent side effects	Less efficacy than GLP-1–receptor agonists, angioedema, unknown long-term safety, risk of pancreatitis
Alpha-glucosidase inhibitor	Miglitol	0.5-0.9	Decreased level of postprandial glucose, hypoglycemia rare, possible decrease in risk of cardiovascular disease events**	Flatulence, diarrhea
Bile acid sequestrants	Colesevelam	0.5	Lowering of LDL cholesterol level; hypoglycemia rare	Gastrointestinal side effects, including constipation; low efficacy; only approved agent in class
D2 dopamine–receptor	Bromocriptine	0.5	Hypoglycemia rare	Low efficacy; gastrointestinal side effects, including nausea; fatigue; dizziness; rhinitis; only rapid-release agent approved
GLP-1–receptor agonist	Liraglutide Trulicity	0.5-1.5	Hypoglycemia rare, weight loss in most patients; possible protective cardiovascular effects	Nausea and vomiting; risks of pancreatitis, thyroid C-cell hyperplasia, and tumors (with liraglutide and weekly exenatide); unknown long-term safety
Amylin analogue	pramlintide	0.5-1.0	Weight loss in most patients, control of postprandial glycemia	Nausea and vomiting, modest effect, hypoglycemia with insulin use, unknown long-term safety
Insulin	Novolin	1.0-2.5	Large effect in all patients	Hypoglycemia, weight gain

Source: Wedbush Securities, Inc.

VTP-30472: A CANDIDATE FOR TREATMENT OF TYPE 2 DIABETES (T2D)

Based on the mechanism of action for VTP-30472, inhibition of 11β-HSD1 (and preclinical data), not only does it lower glucose, it also appears to shift the patient toward a normal state by having a positive impact on multiple cardiovascular and metabolic risk factors associated with "pre-diabetes" or metabolic syndrome (MetS)—a condition which is estimated to affect about 85% of type 2 diabetics and 25% of the worldwide adult population. Risk factors may include: being overweight, having elevated blood glucose, blood pressure, cholesterol and triglycerides, while having decreased levels of good or high-density lipoprotein cholesterol (HDL-C).

CLINICAL PROGRAM

Boehringer Ingelheim (BI) is managing clinical development of VTP-34072 and has conducted two phase 1 clinical trials and is conducting one ongoing phase 2 trial which began in July 2014, triggering a $6 million milestone paid in Q3 2014.

PHASE 2

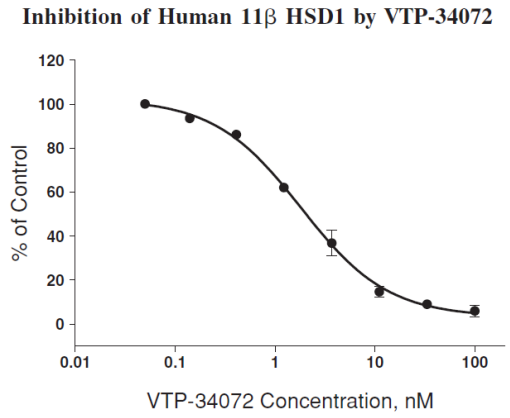
The ongoing phase 2 trial is targeting enrollment of 126 overweight type 2 diabetic patients who have discontinued all background anti-diabetic medications except metformin. The design is placebo-controlled, randomized, and double-blinded (within dose group) in which patients are dosed once daily with one of three doses or placebo for four weeks. Endpoints include blood glucose, safety and tolerability. The first patient was dosed in July 2014 and data release is anticipated in H1 2015.

PHASE 1

In the combined two phase 1 clinical trials, 142 patients were enrolled and VTP-34072 produced a desirable initial clinical profile which included highly potent and selective inhibition of 11β-HSD1 in adipose tissue and had a pharmacokinetic profile supporting once daily oral dosing.

The first (phase 1a) of two phase 1 clinical trials for VTP-34072 was designed as a single ascending dose (SAD) trial in 72 healthy, but overweight volunteers. VTP-34072 was well tolerated at all doses--there were no clinically meaningful changes in vital signs, laboratory values or electrocardiograms. The half-life for clearance from the plasma was 14-24 hours—consistent with once-daily dosing. Target activity of 11β-HSD1 was measuring using pre-dose adipose tissue biopsies compared with biopsies taken at 24 hours post-dosing. Activity in these biopsies demonstrated over 90% inhibition of 11β-HSD1 activity at 24 hours using multiple doses without changes in plasma or urinary hormone levels—suggesting no undesirable inhibition of 11β-HSD2 (the opposing enzyme which converts cortisol to cortisone).

FIGURE 12: VTP-34072 INHIBITS 11B-HSD1 IN A DOSE DEPENDENT MANNER



Source: Company data; Wedbush Securities, Inc.

The second phase 1 clinical trial (phase 1b) was designed with a once-daily multiple ascending dose (MAD) for two weeks in 70 overweight type 2 diabetes patients and VTP-34072 was well tolerated at all doses. There were no clinically meaningful changes in vital signs, laboratory values, or electrocardiograms, no dose-dependent adverse events and no serious adverse events. There were no clinically meaningful changes in undesirable plasma cortisol levels. 11β-HSD1 activity was assessed in adipose tissue biopsies taken before the first day of dosing and at 24 hours post the day 14 dose. Again, 11β-HSD1 activity was inhibited by over 90% at 24 hours in multiple dose groups.

PRECLINICAL

VTP-34072's ability to inhibit 11β-HSD1 was shown in an *in vitro* assay to have low, single-digit nM, activity and was over 1000-fold more potent in inhibiting 11β-HSD1 over the reverse enzyme 11β-HSD2. The pharmacokinetic (PK) profile was assessed in rats and cynomolgus monkeys and demonstrated high oral bioavailability and with a once-daily consistent plasma half-life.

VTP-30472 MARKET OPPORTUNITY IN TYPE 2 DIABETES: WE PROJECT MULTI-BILLION DOLLARS FOR PEAK SALES.

There are several categories of orally active drugs for the treatment of type 2 diabetes. Despite there being multiple categories of drugs to control blood sugar in diabetes patients, as none are cures, there is still a need for improved treatments. The first couple of approved drugs in a new class also have the potential to achieve multi-billion dollars in gross peak annual sales. This is evident from the relatively recent emergence of DPP-IV inhibitors as Januvia was first approved 2006 and by 2012 had sales of about $4 billion. We believe that, if approved, VTP-34072 has the potential to achieve blockbuster sales due to its first-in-class position among 11β-HSD1 inhibitors and its potential to alleviate symptoms of metabolic syndrome—and potentially to delay onset of type 2 diabetes.

VTP-30472 COULD BECOME FIRST TO MARKET AND BEST-IN-CLASS AMONG 11B-HSD1 MODULATORS.

Several 11β-HSD1 inhibitors have been developed and progressed through human clinical trials; despite this, no 11β-HSD1 inhibitor has made it into phase 3. Clinically-active 11β-HSD1 inhibitors include: Vitae's VTP-30472, Piramal/Eli Lilly's P2202, Bristol Myers program. There are over 40 U.S. and 90 European patents filed for 11β-HSD1 inhibitors and several companies (small and large) have tried to develop 11β-HSD1 inhibitors; however, the failure of these trials has provided the foundation for an emerging clinical profile for this class as well as setting the safety and efficacy standards for the successful development of an 11β-HSD1 inhibitor.

FIGURE 13: SELECTED 11β-HSD1 INHIBITORS IN DEVELOPMENT

Drug Candidate	Company(s)	Stage of Development
VTP-30472	Vitae / Boehringer Ingelheim GmbH (VTAE/BI)	phase 2
P2202	Piramal Healthcare/Eli Lilly (LLY)	phase 2 (Canada/India)
BMS-816336/BMS-770767	Bristol Myers Squibb (BMY)	phase 2 / phase 1
CNX-010-49	Connexios Life Sciences Pvt. Ltd.	Preclinical
INCB13739,INCB20817	Incyte Corporation (INCY)	Suspended in phase 2 (2012)
MK-0916,MK-0736	Merck	Presumed suspended
AMG 221/AMG 331	Amgen, Inc./Swedish Orphan Biovitrum (AMGN /SOBI:SS)	Suspended (2011)
AZD4017, AZD8329	AstraZeneca (AZN)	Suspended (2011)
LY2523199	Eli Lilly & Company (LLY)	Suspended
RG4929,RG7234	Roche Holding AG (ROG:VX)	Suspended
DIO-912	DiObex, Inc	Suspended in phase 2b
PF-00915275	Pfizer	Terminated in phase 2

Source: Company data; Wedbush Securities, Inc.

VTP-37948 FOR ALZHEIMER'S DISEASE (BACE INHIBITOR)

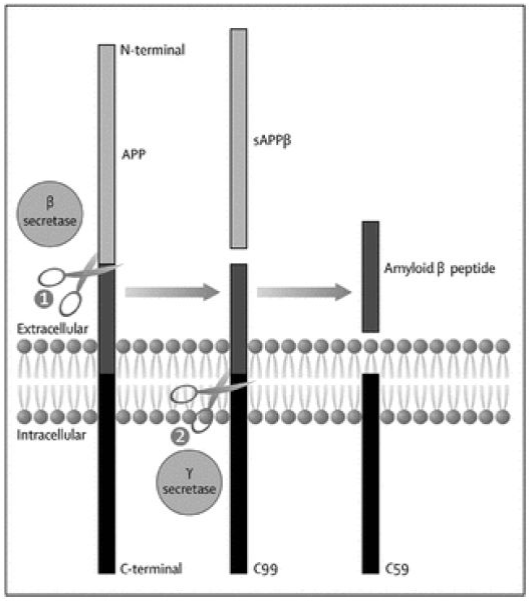
ALZHEIMER'S DISEASE

Alzheimer's disease is the most common type of dementia and involves a progressive loss of memory and increasing mental and physical disability leading to disruption in activities of daily living to the point of becoming dependent on caregivers and frequently an early death. The characteristic mark of Alzheimer's disease is the development of large amyloid β plaques in the brain parenchyma which is believed to precipitate neurofibrillary tangles, glutamatergic excitotoxicity, oxidation, inflammation, and cell apoptosis. According to Alzheimer's Association, in 2014, an estimated 5.2 million patients in the US suffer from Alzheimer's and over 500,000 die each year. Due to increasing life expectancy, the prevalence could triple by 2050.

BACE1 AS A DRUG TARGET FOR THE TREATMENT OF ALZHEIMER'S DISEASE

A brain enzyme called β-Site Amyloid Precursor Protein Cleaving Enzyme 1 (BACE1) is thought to play an important role in the neurodegenerative processes leading to Alzheimer's disease. BACE1 activity initiates a proteolytic cascade to convert amyloid precursor protein (APP) into amyloid peptide which aggregates in the brain into amyloid plaques believed to trigger a neurodegenerative cascade. Not only is BACE1 associated with amyloid production, but genetic data also supports its role in the disease process.

FIGURE 14: GENERATION OF AMYLOID B PEPTIDE



Source: Company data; Wedbush Securities, Inc.

Amyloid β is a peptide generated in and secreted by most cell types; however, neurons are the major producers. Amyloid β is produced by the sequential endoproteolytic processing of the amyloid precursor protein (APP) by sequential β and γ-secretase enzymatic activities. APP is initially cut by β-secretase which creates the membrane bound C99 APP peptide and sAPPβ fragment. C99 APP is then cleaved by γ-secretase to generate the 42 amino acid amyloid β peptide fragment.

AMYLOID AND ALZHEIMER'S DISEASE

The deposition of amyloid peptides as plaques in the brain promotes inflammation and neurodegeneration. There are over 200 autosomal dominant mutations observed in familial early-onset disease. The consistent feature across these mutations is that they increase the cleavage efficiency of β and γ secretase. For example, the double mutation in K670N and M671L (the Swedish mutation), and the single mutation A673V, both increase BACE cleavage and Amyloid β peptide production—while the A673T mutation in the B cleavage site of APP decreases cleavage efficiency by 40% and is associated with reduced incidence of Alzheimer disease and

increased cognitive faculties in older adults. Together these data support the idea that modification of BACE1 modulates amyloid β production and symptoms of Alzheimer's disease. Currently, there are no approved treatments for Alzheimer's disease that directly target the production or accumulation of amyloid β plaques.

PROSPECTIVE TREATMENTS

ANTI-AMYLOIDβ ANTIBODIES

Recently the results from phase 3 trials examining the use of two anti-amyloid β antibodies, Solanezumab (PFE) and Bapineuzumab (LLY) were published — unfortunately neither met their primary endpoint of an increase in the Alzheimer's disease cooperative study-activities of daily living scale (ADCS-ADL). However, approximately 25% of diagnosed Alzheimer disease participants were negative for amyloid deposition assessed using PET imaging; suggesting that it is likely a majority of PET negative patients did not have Alzheimer's disease. PFE/LLY hopes to exclude these individuals by requiring a positive PET image for acceptance into the third phase 3 trial. In these studies, a subset of early onset patients showed improvement. Patients who show moderate or even mild signs of Alzheimer's may be "too far gone" supporting early diagnosis and treatment in order to prevent or reduce amyloid β plaque formation. It is speculated that amyloid β peptide deposits occur over several years--possibly decades--before the initial clinical symptoms manifest.

GAMMA SECRETASE INHIBITORS

Due to the strong epidemiological association between γ secretase modulation (18-50% of all early onset) and Alzheimer disease, inhibitors of γ secretase activity were tested as a potential treatment. Initial preclinical data demonstrated efficacy in reducing the production of amyloid β peptide in mouse models; however, mutations associated with γ secretase function were lethal--possibly due to the γ secretase role in activating notch—an important factor in fetal development. Despite the adverse event profile seen in mouse models, γ secretase inhibitors were clinically tested; however, there were no signs of improvement in cognitive function and there was an increase in serious adverse events such as melanoma and colitis. We believe safety concerns make further development of γ secretase inhibitors unlikely, in our view.

BETA SECRETASE INHIBITORS (BACE)

To date, BACE knockout mice and inhibition of BACE using small molecules has not resulted in lethal effects on mouse physiology and the class is not expected to have the same adverse event profile as γ secretase inhibitors. Consequently, BACE inhibitors may provide a more desirable clinical profile—reducing Aβ without the serious adverse events—and have a larger therapeutic window.

FIGURE 15: BACE INHIBITORS IN DEVELOPMENT

Drug Candidate	Stage of Development
MK-8931 (MRK)	phase 2/3
AZD3293 (AZN/LLY)	phase 2/3 ready
VTP-37948 (VTAE/BI)	phase 2
E2609 (EISAI/BIIB)	phase 2
HPP854	phase 1
LY2886721 (LLY)	phase 2 (Discontinued June 2013 for liver enz's)
RG7129 (RHYYB)	Discontinued 2013

Source: Company data; Wedbush Securities, Inc.

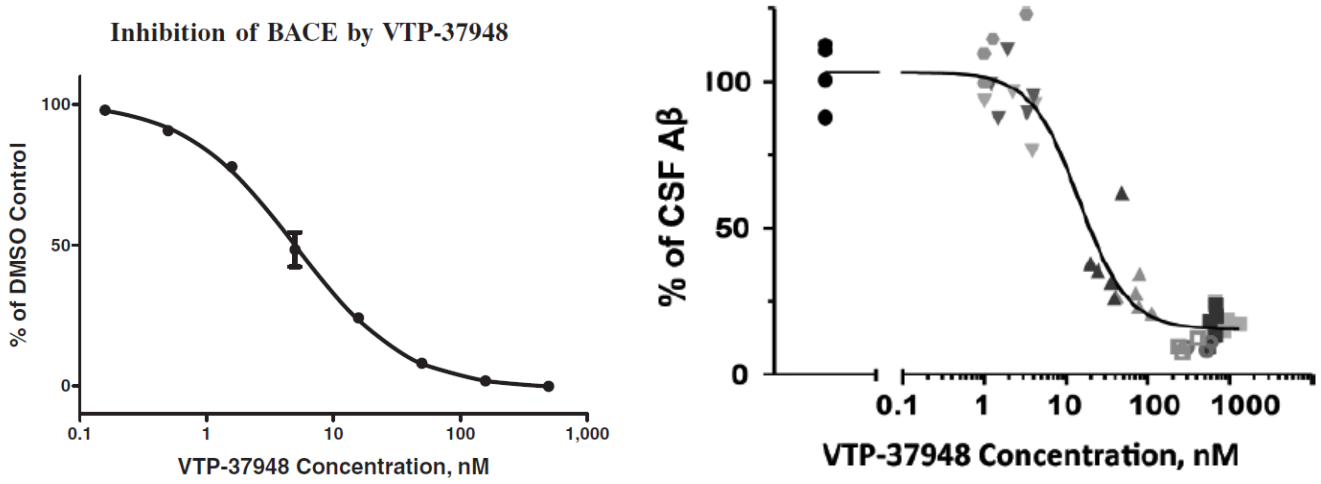
VTP-37948

Vitae discovered and is developing (in a collaboration led by Boehringer Ingelheim (BI)) an orally dosed small molecule drug candidate called VTP-37948 to inhibit BACE1 as a treatment for Alzheimer's disease. In preclinical testing, VTP-37948 demonstrated highly potent and selective inhibition of BACE1 in the brain—associated with up to a 95% reduction in brain amyloid β levels. VTP-37948 is currently in two phase 1 clinical trials involving a total of 68 healthy volunteers. The first trial is testing safety, tolerability and pharmacokinetics. The second trial is testing activity on a key biomarker--amyloid β concentration in the cerebrospinal fluid (CSF). Results from both of these phase 1 trials are expected by year-end 2014.

PRECLINICAL

In collaboration with BI, Vitae discovered, optimized and selected VTP-37948, a BACE inhibitor for the treatment of Alzheimer's disease using their Contour® platform. Preclinical studies demonstrated that VTP-37948 has a potent low nM inhibition of human BACE activity determined using a cell-free biochemical assay. In addition, VTP-37948 activity is specific in that it has shown no inhibition of pepsin and cathepsin E and a 1000 fold greater preference for BACE compared to renin and cathepsin D.

FIGURE 16: VTP-37948 INHIBITS BACE ACTIVITY AND Aβ



Source: Company data; Wedbush Securities, Inc.

In preclinical studies, orally administered VTP-37948 was shown to be effective in lowering amyloid β in brain and in the cerebral spinal fluid in rat models. Studies in rats and dogs show that VTP-37948 is well absorbed and has a high bioavailability and relatively long plasma half-life. Based on the PK profile, once-daily dosing is likely.

DEVELOPMENT PLANS

In collaboration with BI, Vitae has two ongoing phase 1 trials for VTP-37948 in a total of 68 healthy volunteers. Vitae's first phase 1 clinical trial was initiated in January 2014 and has a single dose, randomized, double blind, placebo-controlled design assessing the safety and tolerability of escalating doses of VTP-37948. The second trial is a single escalating dose study of VTP-37498 and will address the same parameters as the first phase 1 in addition to examining changes in CSF amyloid β at various times post dosing. Data readout from both phase 1 trials is anticipated around year-end 2014 and is likely to guide future development of VTP-37498.

ALZHEIMER'S MARKET

It is estimated that over 5 million Americans (majority over the age of 65 yrs.) are currently suffering with Alzheimer's disease and over 200,000 suffer from early onset disease.

FIGURE 17: APPROVED TREATMENTS FOR ALZHEIMER'S DISEASE

Drug name	Brand name	Approved For	FDA Approved
donepezil	Aricept	All stages	1996
galantamine	Razadyne	Mild to moderate	2001
memantine	Namenda	Moderate to severe	2003
rivastigmine	Exelon	All stages	2000
tacrine	Cognex	Mild to moderate	1993

Source: Company data; Wedbush Securities, Inc.

Currently there are only two classes of FDA-approved treatments for Alzheimer disease, including inhibitors of cholinesterase and N-nitrosodimethylamine (NDMA). Unfortunately, both classes of drugs do not treat the underlying cause of the disease and only address symptoms. Despite limited efficacy, Aricept, a cholinesterase inhibitor, achieved $2.1 billion in its final full year of sales before losing exclusivity. We believe that VTP-37948 as a BACE inhibitor has the potential to be disease-modifying and could achieve over $5 billion in gross peak annual sales in 2027, by our projections.

VTP-43742 FOR AUTOIMMUNE DISORDERS

VTP-43742 inhibits the retinoic acid receptor (RAR)-Related Orphan Receptor γ-t (RORγt) and is being developed to treat autoimmune disorders such as psoriasis, multiple sclerosis (MS), rheumatoid arthritis (RA), as well as more rare conditions such as Behçet's disease and autoimmune uveitis. Autoimmune disorders are characterized by an inappropriate immune response against normal, healthy tissues. A critical activity associated with many autoimmune disorders includes the activation of a specific class of lymphocytes called Th17 cells. Moreover, RORγt is an essential protein for Th17 activity.

Vitae initiated the discovery program in Q4 2012 and selected VTP-43742 as a candidate in Q1 2014. Preclinical data suggests inhibition of RORγt may provide a beneficial treatment for multiple autoimmune disorders. VTP-43742 has been shown to inhibit the secretion of Interleukin 17 (IL-17) and other Th17 cell-associated inflammatory proteins. Importantly, VTP-43742 has shown a therapeutic effect in a well-established animal model for MS. Preclinical results suggest VTP-43742 may support once a day dosing. Vitae plans to file an Investigational New Drug application (IND) with the FDA in the first half of 2015 and initiate phase 1 testing shortly thereafter with initial proof-of-concept clinical efficacy data by the end of 2015.

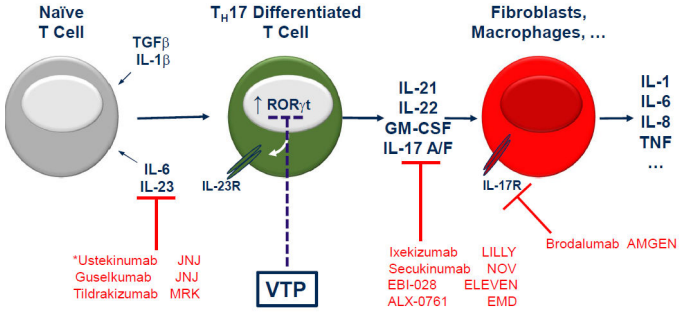
DISEASE AND TREATMENT BACKGROUND

CD4 T cells play a key role in the function of the innate and adaptive immune system. Unfortunately, many diseases, specifically autoimmune and chronic inflammatory disease have origins that can be traced to a malfunction in regulatory CD4 T cells. There are several types of regulatory CD4 T cells, most notably Th1, Th2 and Th17. Th1 and Th2 are primarily responsible for host defense against intracellular pathogens such as bacteria, viruses and protozoa through the secretion of IFN-γ, IL-4, IL-5 and IL-13. Th17 cells are responsible for host defense against extracellular bacteria and fungi at the gastrointestinal tract, lungs and skin. This occurs mainly through the secretion of potent pro-inflammatory cytokines IL-17A, IL-17F and IL-22. Several lines of evidence suggest that dysregulation of Th17 cells and overproduction of cytokines (mainly IL-17) can play an important role in several types of autoimmune disorders. Increased levels of IL-17 cytokines have been observed in patients with RA, MS, IBD and psoriasis. Additionally, deletion of IL17A in an EAE mouse model protects the mice from the disease. Studies that are more recent have shown the IL-17 antibody secukinumab to be superior to Enbrel (anti-TN**Fα)** in alleviating the disease in human psoriasis patients and preclinical models. Solid basic research and success in clinical studies have made finding targets that modulate Th17/IL-17 attractive as a potential treatment for various chronic and autoimmune diseases.

IL-17 AND TH17 CELLS IN AUTOIMMUNITY

Th17 cells are produced from naïve cells that undergo differentiation due to the presence of cytokines including Il-6, IL-21, IL-23 and TGFβ. During the differentiation process, RORγt expression is increased which drives secretion of IL-17 and the production of the IL-23 receptor. Activation of the IL-23 receptor amplifies the differentiation signal, thus stabilizing cellular differentiation and increasing the activity of Th17 cells and ultimately the production of IL-17. Differentiated and activated Th17 produce IL-17A and Il17F, Il21. Il22, and GM-CSF--all potent activators of the inflammation cascade.

FIGURE 18: T-CELL DIFFERENTIATION INTO TH17



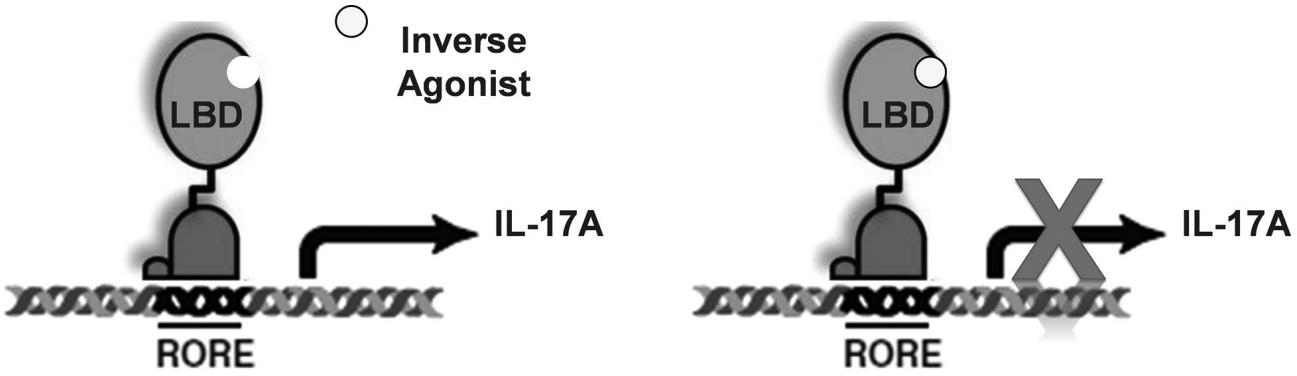
Source: Company data; Wedbush Securities, Inc.

Naive T cells differentiate into Th17 cells by action of multiple factors. Expression of RORγt in Th17 cells drives expression of inflammatory cytokines that exacerbate autoimmune disease**.**

RORγt

RORγt is a member of the ROR subfamily of nuclear hormone receptors which includes RORα, RORβ and RORγ, all of which have similar ligand binding domains (LBD). Additionally, each ROR has multiple isoforms that are expressed from the same gene loci. RORα is expressed in several tissue types including liver, skin and muscle and RORβ is expressed mostly in the brain and central nervous system, while RORγ is expressed in several tissue types. Conversely, RORγt expression is restricted to T cells and other immune cells. RORγ knockout mice have impaired Th17 differentiation and as a result are resistant to several autoimmune disorders. Other than this phenotype, the RORγ mice are healthy. This is in contrast to KO RORα mice that have a movement disorder due to the degeneration of brain cells and KO RORβ mice that are blind due to abnormal eye development. Together these data support the idea that a RORγ and RORγt inhibitors that do not inhibit RORα and RORβ could provide a novel mechanism to modulate Th17 function to treat autoimmune diseases.

FIGURE 19: RORγt BINDS TO DNA AND INITIATES TRANSCRIPTION OF INFLAMMATORY GENES



Source: Company data; Wedbush Securities, Inc.

ANTIBODY TREATMENTS

Currently there are several anti-IL-17 and anti-IL-17A antibodies under development. To date, these antibody inhibitors have shown efficacy in human trials in various autoimmune diseases including psoriasis, MS and RA. In a phase 3 clinical trial, Secukinumab demonstrated superiority compared to TNF-alpha inhibitor, Enbrel, in psoriasis patients. Compared to these therapies, we believe VTP-43742 could possibly treat a broader patient population more effectively. By directly inhibiting RORγt activity, VTP-43742 could inhibit the production of IL17 and other cytokines that can potentially modulate disease state.

FIGURE 20: IL17A, IL17RA AND IL23 INHIBITORS

Drug Candidate	Company	Target	Indication	Failed/Terminated Indications	Stage of Development
Secukinumab (AIN457)	Novartis	IL17A	Psoriasis, RA, ankylosing spondylitis, , uveitis, psoriatic arthritis, polymyalgia rheumatica, dry eye, MS, asthma	Crohn's disease, Uveitis, Behçet's uveitis,	Completed phase 3
Ixekizumab (LY2439821)	Eli Lily	IL17A	RA, psoriasis	N/A	phase 3
Brodalumab (AMG827)	Amgen	IL17RA	RA, psoriasis, Crohn's disease, asthma, psoriatic arthritis	Crohn's Disease	phase 3
Ustekinumab (CNTO 1275)	Janssen	IL12 / IL23	Psoriatic arthritis	N/A	On the Market

Source: Wedbush Securities, Inc.

RORγt ANTAGONISTS

Using classical screening methods, several RORγt inhibitors have been found and determined to be effective in preclinical studies; however, due to off-target effects, suboptimal PK/PD, and lack of selectivity, further development of these compounds was discontinued.

FIGURE 21: RORγt INHIBITORS

Licenser/Licensee (Year Announced)	Stage of Development
Orphagen /Japan Tobacco (2009)	Preclinical
Exelixis/ Bristol Myers Squibb (2010)	Preclinical
Lycera/Merck (2011)	Preclinical
Kara Bio/Pfizer (2011)	Preclinical
Phenex/Janssen Biotech (2012)	Preclinical
Teijin/Amgen (2013)	Preclinical

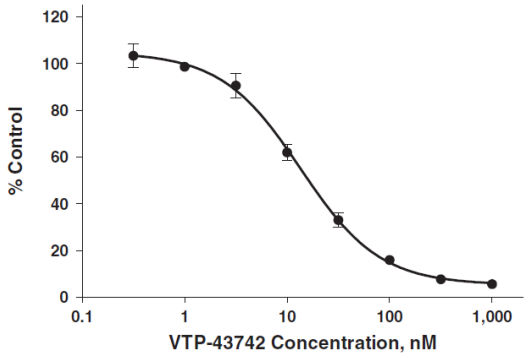
Source: Company data; Wedbush Securities, Inc.

Using the Contour® platform, Vitae has developed VTP-43742 by exploiting the variations between ROR subfamily members. Preclinical studies have indicated that VTP-43742 is 1000x more potent at binding to RORγt than to RORα or RORβ. Additionally, the ability to orally administer VTP-43742 is likely to expand its market potential.

PRECLINICAL STUDIES

VTP-43742's ability to inhibit RORγt was shown in an *in vitro* assay to have low, single digit nM (3.7nM), binding activity and was over 1000x more potent in selectively inhibiting RORγt over other ROR family members. This is important for avoiding adverse events.

FIGURE 22: VTP-43742 INHIBITION CURVE IN RORΓT T-CELL ASSAY AND BINDING AFFINITY TO ISOLATED HUMAN ROR

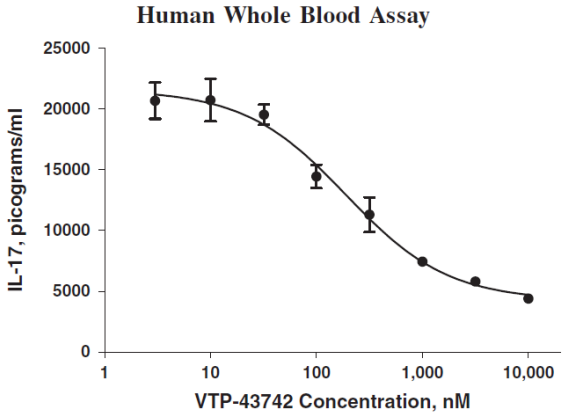


Human isotype	RORγt	RORα	RORβ
Human binding Ki (nM)	3.7	4712	3914

Source: Company data; Wedbush Securities, Inc.

In a preclinical study designed to be more representative of RORγt inhibition in humans, VTP-43742 was shown to be a potent inhibitor of IL17 production in lymphocytes in whole blood assays and was calculated to have an IC_{50} of 221 nM.

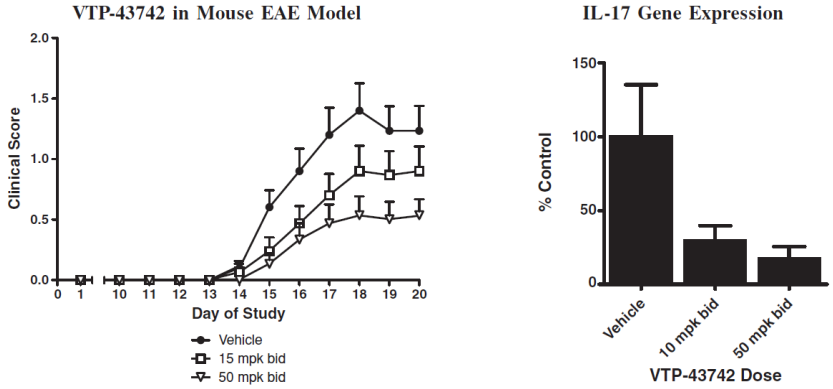
FIGURE 23: VTP-43742 INHIBITS IL-17 PRODUCED BY HUMAN LYMPHOCYTES IN WHOLE BLOOD.



Source: Company data; Wedbush Securities, Inc.

In an *in vivo* model of experimental autoimmune encephalomyelitis (EAE), VTP-43742 was shown to be effective at abating symptoms. The EAE mouse model is widely used as a model for human multiple sclerosis (MS) because of the similarities in pathogenesis and dependence on IL-17. In this experiment, twice daily, orally administered VTP-43472 was shown to reduce clinical score and IL17 gene expression in a dose-dependent manner. Most importantly, it was shown to have a maximal reduction in clinical score between 50-60%, which is similar to targeted IL-17 therapies and RORγt gene knockout mice.

FIGURE 24: VTP-43742 IS EFFICACIOUS IN REDUCING DISEASE SYMPTOMS IN EAE MODEL

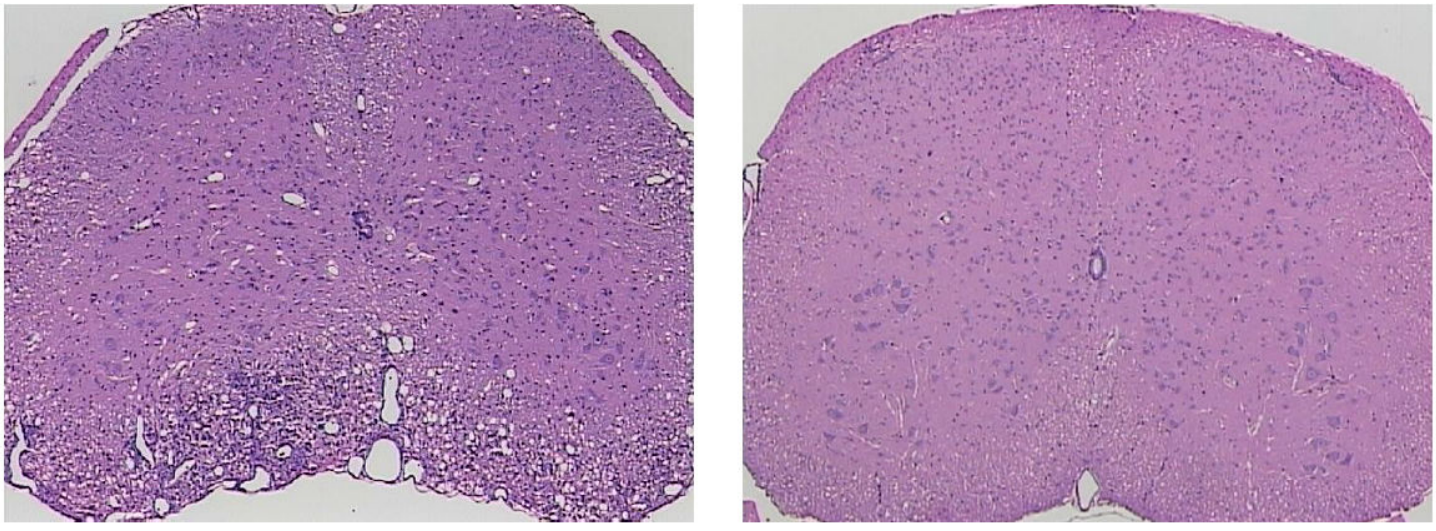


Left: VTP-43742 treatment ameliorates severity of disease in the EAE model, where mpk refers to mg/kg.
Right: VTP-43742 inhibits IL-17 gene expression within inflammatory cells in the spinal cord of treated animals.

Source: Company data; Wedbush Securities, Inc.

To assess the improvement in hallmark physical conditions in EAE mice, VTP-43742 and placebo-treated mice were sacrificed and spinal cords were assessed for the loss of myelin sheaths after disease induction. EAE is marked by the infiltration of IL17 expressing T cells into the spinal cord. These cells induce an inflammatory response which leads to a loss of myelin sheaths around nerves in the spinal cord. Histological sections from mice treated with VTP-43742 showed reduced production of vacuoles and reduced loss of axonal myelin sheaths compared with placebo. Additionally, inflammation from lymphocyte and neutrophil infiltration was reduced in VTP-43742 treated mice compared to placebo. In our view these data support the development of VTP-43742 as an inhibitor of RORγt and as a potential treatment for MS.

FIGURE 25: VTP-43742 REDUCES HISTOLOGICAL ABNORMALITIES



Source: Company data; Wedbush Securities, Inc.

DEVELOPMENT PLANS

To date, Vitae has orally and intravenously administered VTP-43742 to rats and dogs and assessed PK and oral bioavailability and plans to file an investigational new drug application (IND) and initiate phase 1 testing for VTP-43742 in the H1:2015. The first phase 1 clinical trial is likely to be a single ascending dose (SAD) study in healthy volunteers to assess the safety, tolerability and pharmacokinetics. The second phase 1 is a proof-of-concept trial planned for H2:2015. The design is a two-week, multiple ascending dose study which will assess safety, tolerability and pharmacokinetics of once daily VTP-43742 in psoriasis patients. The study will also assess the alleviation of disease symptoms including improvement in skin lesions, and changes in Th17, Il-17, and other cytokines in skin biopsies from treated and placebo controlled patients

MARKET OPPORTUNITY

We believe patients suffering from autoimmune disorders such as psoriasis, MS, RA, steroid-resistant asthma, Behçet's disease and autoimmune uveitis could potentially benefit from VTP-43742. Given the preclinical studies conducted so far, VTP-43742 could be effective in treating psoriasis and MS, each with 2 million and 1.1 million patients, respectively. Current oral treatments for MS include potential multi-billion-dollar blockbusters like Gilenya and Tecfidera. We believe with clinical success and a competitive profile, VTP-43742 could also achieve blockbuster sales.

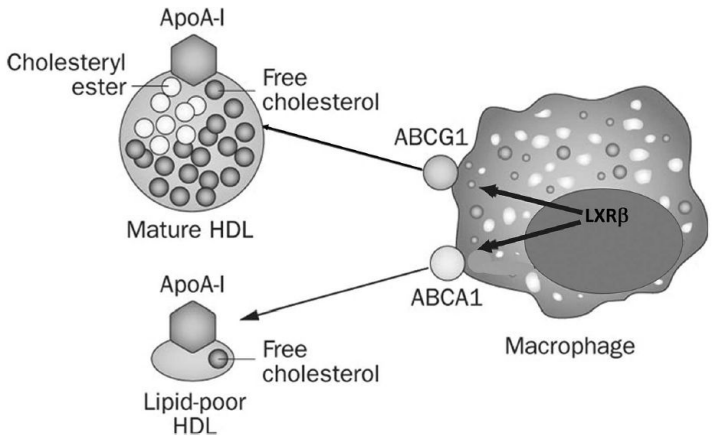
VTP-38443 TARGETING LIVER X RECEPTOR BETA (LXRB) FOR ACUTE CORONARY SYNDROME (ACS)

VTP-38443 is an agonist for liver X receptors (LXRs) and is being developed to treat acute coronary syndrome (ACS). LXRs stimulate production of proteins that transport cholesterol out of cells and inhibit production of inflammatory proteins. Several preclinical studies have demonstrated that LXR agonists can promote reverse cholesterol transport (RCT) and prevent development of atherosclerosis. VTP-38443 was discovered in-house and is being developed as an orally active LXRβ selective agonist. Its therapeutic effect includes promoting RCT, facilitating removal of cholesterol from plaques in atherosclerotic vessel walls and by inhibiting production of pro-inflammatory proteins around the plaque. These mechanisms reduce inflammation and stabilize the plaque--which may also reduce the risk of plaque rupture, blood clot formation and heart attack. Vitae envisions developing VTP-38443 to complement current ACS treatments. In preclinical testing, VTP-38443 decreased cholesteryl ester formation in plaques by over 90% and reduced plaque inflammation as well. Vitae anticipates completing preclinical studies, filing an IND and starting phase 1 clinical testing for VTP-38443 in H1:16.

DISEASE AND TREATMENT BACKGROUND

Acute coronary syndrome (ACS) is a manifestation of coronary artery disease and can result in unstable angina, acute myocardial infarction, and sudden death. ACS begins with the formation of an atherosclerotic plaque which is vulnerable to mechanical stress and/or inflammatory cell weakening which can promote the formation of blood clots. Macrophages infiltrate the atherosclerotic plaque and secrete proteases that make it susceptible to rupture that can block circulation in the artery and lead to cardiovascular events such as a heart attack or stroke.

FIGURE 26: ACTIVATION OF LXRS INCREASE SYNTHESIS OF CHOLESTEROL TRANSPORT GENES



Source: Company data; Wedbush Securities, Inc.

LIVER X RECEPTOR B (LXRβ) IN ACS

Cholesterol is tightly regulated by two distinct pathways: the systemic sterol regulatory element binding protein (SREBP) and the liver X receptors (LXR) which act on a local cellular level. Statins control the SREBP pathway and are effective at controlling cholesterol; however, there are no approved modulators of LXR activity. LXRα and LXRβ are sterol activated transcription factors that upon ligand binding initiate the transcription and repression of several genes that control cholesterol efflux. The SREBP and LXR pathways regulate each other in that most LXR-activating ligands can also inhibit SREBP. Consequently, a compound that targets the LXRβ pathway could potentially be used in combination with statins to further control cholesterol levels at both cellular and systemic levels.

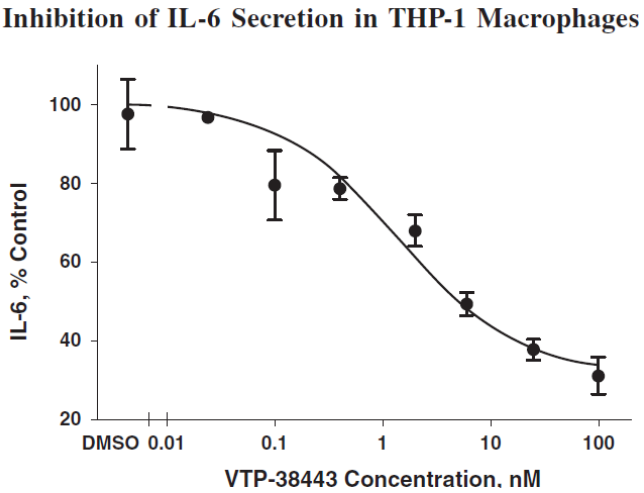
Activation of the LXR receptor has also been shown to reduce the expression of inflammatory proteins in macrophages that weaken the plaque, potentially leading rupture, blood clot formation, and cardiovascular events. By reducing inflammation, VTP-38443 could have additional benefits beyond reducing cholesterol.

PRECLINICAL

In preclinical studies, VTP-38443 was shown to be effective in reducing cholesterol and inflammation in a mouse ACS model and in human cell assays. To date, Vitae has established efficacious and maximum tolerated doses. Biochemical binding assays demonstrated that VTP-38443 is a 22x more potent agonist of LXRβ (12nM) over LXRα (262nM). In addition, in assays used to determine induction of LXRβ activity, VTP-38443 was shown to have an EC_{50} of 19nM—a 17x higher preference for LXRβ over LXRα.

FIGURE 27: VTP-38443 PREFERENTIALLY BINDS TO LXRB AND DECREASES IL-6

ASSAY	RECEPTOR	VTP-38443
Binding Ki (nM)	LXRα	262
	LXRβ	12
	LXRβ Selectivity Ratio	**22x**
Cell based reporter assay EC_{50} nM	LXRα	320
	LXRβ	19
	LXRβ Selectivity Ratio	**17x**

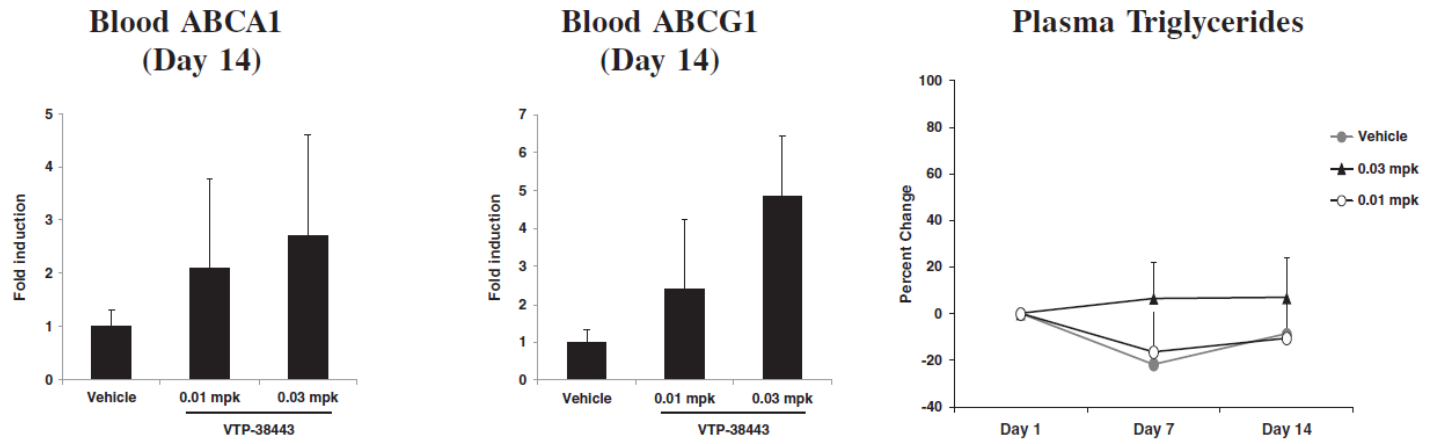


Source: Company data; Wedbush Securities, Inc.

In addition, preclinical studies have also shown VTP-38443 to effectively suppress inflammatory proteins associated with macrophages, namely IL-6. In studies using human-derived activated THP-1 macrophages, VTP-38443 was shown reduce IL-6 in a dose-dependent manner.

In a study designed to assess the effectiveness of VTP-38443 at modulating LXRβ, 0.01 or 0.03 of VTP-38443 versus vehicle control was given daily to cynomolgus macaques for 14 days. Blood samples were taken on days 0, 7 and 14. Results show that both doses the markers for RCT activation, ABCA1 and ABCG1, were elevated, while no significant changes in triglycerides were observed. Together, Vitae found the therapeutic window has a 30x difference between the dose needed to increase ABCA1 and ABG1 and the undesirable dose that increases plasma and liver triglyceride levels.

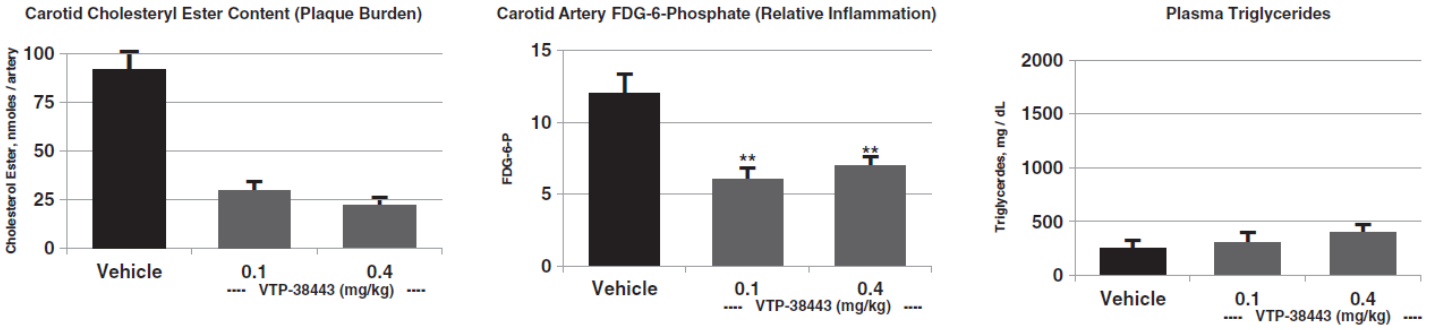
FIGURE 28: VTP-38443 INCREASES mRNA EXPRESSION OF RCT GENES IN CYNOMOLGUS MONKEYS



Source: Company data; Wedbush Securities, Inc.

In an experimental mouse model of accelerated atherosclerosis, VTP-38443 was able to prevent the accumulation of cholesterol and decrease inflammation in plaques. In this model, mice are initially fed a high cholesterol diet for two weeks then undergo surgical ligation of the left carotid artery followed by a high cholesterol diet for an additional two weeks. As a result, atherosclerotic lesions form at the ligated section marked by increase in cholesteryl and vascular inflammation. In mice the administration of VTP-38443 during the surgery produced a dose-dependent decrease in cholesterol and decrease in markers for vascular inflammation. No significant elevation in plasma triglycerides was observed.

FIGURE 29: VTP-38443 REDUCES CAROTID CHOLESTERYL ESTER CONTENT AND VASCULAR INFLAMMATION



Source: Company data; Wedbush Securities, Inc.

Across all animals tested, VTP-38443 exhibited a bioavailability of approximately 50% and demonstrated a half-life consistent with once-daily dosing.

Currently, VTP38443 is in preclinical development and has successfully completed dose ranging toxicology studies. Vitae plans to complete preclinical studies and file an IND in Q1:16. The company plans to complete a single and multiple ascending dose phase 1 study in healthy volunteers to assess safety, tolerability, and pharmacokinetics, and will also observe gene induction markers of RCT and inflammation.

MARKET OPPORTUNITY

There are more than 1 million hospital discharges (including secondary discharges) in the US which could be attributed to ACS. The cost impact of ACS is estimated to greater than $150 billion annually and direct medical costs are about $75 million. Standard-of-care treatments after an ACS event includes anticoagulants, antiplatelet agents and cholesterol lowering agents. Given its unique mechanism of action, we believe VTP-38443 can potentially be used in combination with standard-of-care to increase efficacy.

VTP-38543 FOR ATOPIC DERMATITIS (ECZEMA)

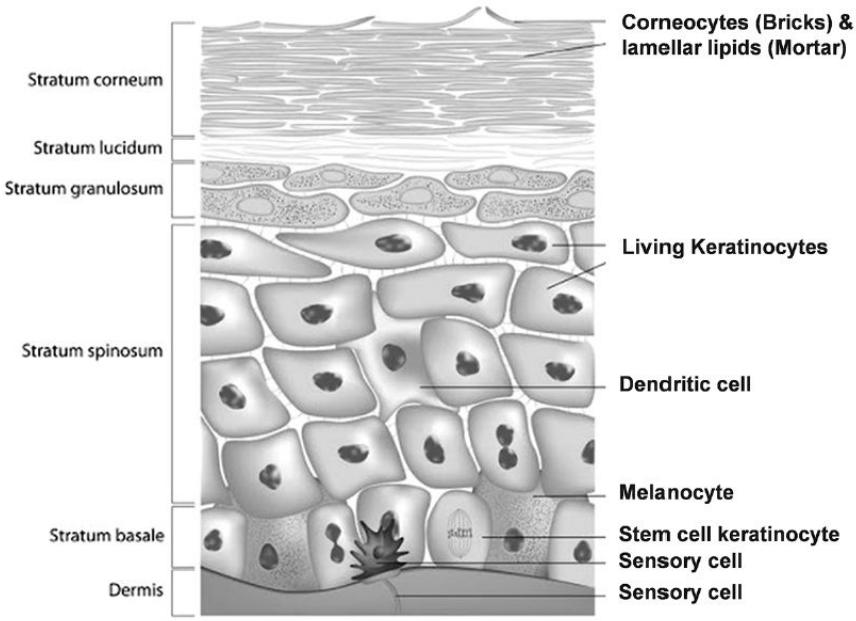
VTP-38543, also an LXRβ agonist similar to VTP-38443, transports lipids out of cells and decreases inflammation and is being developed topically for atopic dermatitis (eczema). In an animal model of skin inflammation, VTP-38543 demonstrated equal or better efficacy compared with a high potency topical corticosteroid, the current standard of care. Vitae anticipates completing preclinical studies and filing an IND for VTP-38543 by H2:15 and starting phase 1 shortly afterwards.

Atopic dermatitis is a chronic inflammatory skin disease marked by skin dryness, erythema, and itching stemming from the breakdown of skin barrier. Standard-of-care includes topical anti-inflammatory agents including glucocorticoids and calcineurin inhibitors. Although effective, each treatment has limitations and the potential for serious side effects.

BARRIER FUNCTION AND INFLAMMATION IN ATOPIC DERMATITIS

Keratinocytes differentiate into corneocytes and generate lipids that together form the stratum corneum – the outer most layer of the epidermis. The stratum corneum provides a barrier against loss of water and assaults from environmental and microbial agents. Mutations that affect barrier proteins or lipid generation have been shown to interfere with skin self-repair and induce chronic inflammation. For example, mutations in the filaggrin protein can be found in up to 55% of Europeans with atopic dermatitis.

FIGURE 30: EPIDERMIS



Source: Company data; Wedbush Securities, Inc.

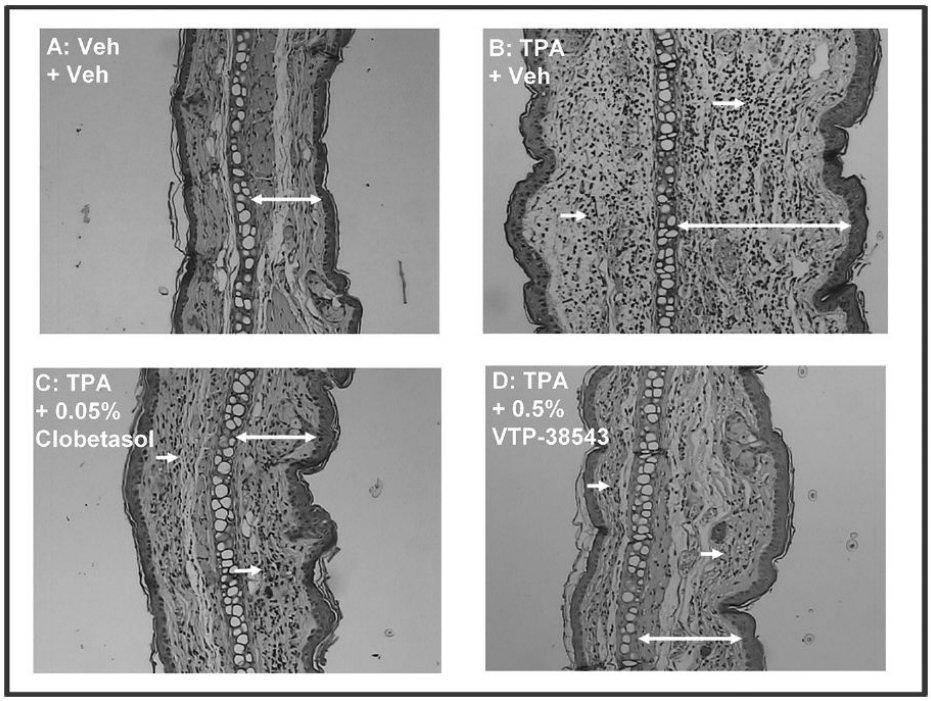
LXR stimulation induces expression of involucrin, loricrin and filaggrin genes involved in formation of the outer layer of skin. Topical application of LXR agonists improved symptoms in a mouse model of atopic dermatitis by promoting lipid synthesis and transport into the extracellular space and reducing gene expression of pro-inflammatory cytokines. Together these results suggest activating LXR pathway may increase the barrier function of the skin and provide relief to patients with atopic dermatitis.

VTP-38543

PRECLINICAL DATA

In biochemical binding assays, VTP-38543 was shown to tightly bind to LXRβ (26nM) and act as a partial agonist of LXRβ-mediated activity in a cell based assay (EC_{50} =16nM). In an inducible mouse model for skin inflammation, topically applied VTP-38543 was shown to decrease neutrophil infiltration and swelling caused by administration of tetradecanoylphorbol acetate (TPA). The effect was on par on with standard of care with the steroid clobetasol.

FIGURE 31: VTP-38543 REDUCES INFLAMMATION IN CHEMICALLY INDUCED MOUSE MODEL

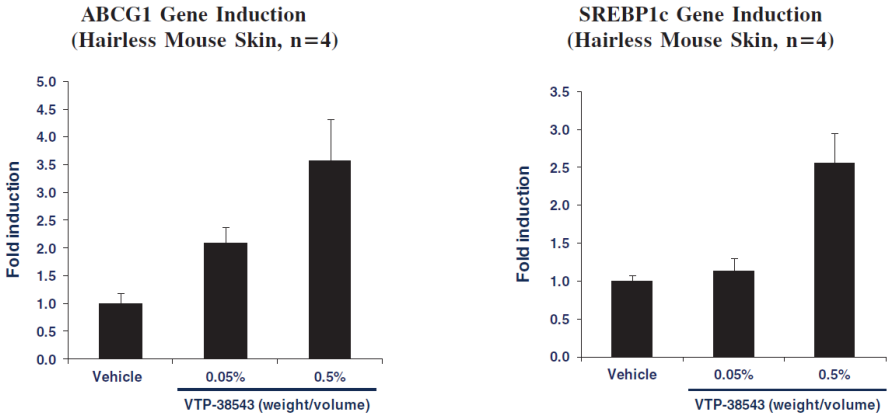


A: Vehicle treated, no chemical induction
B: Vehicle treated + chemical (TPA) induction
C: Glucocorticoid (cloβsol) + TPA induction
D: VTP-38543 + TPA induction

Source: Company data; Wedbush Securities, Inc.

ABCG1 and SREBP1c knockout mice display impaired epidermal formation due to the importance of these genes in the lipid production and extracellular transport process. Topical application of VTP-38543 was shown to increase the expression of both ABCG1 and SREBP1c in a hairless mouse model.

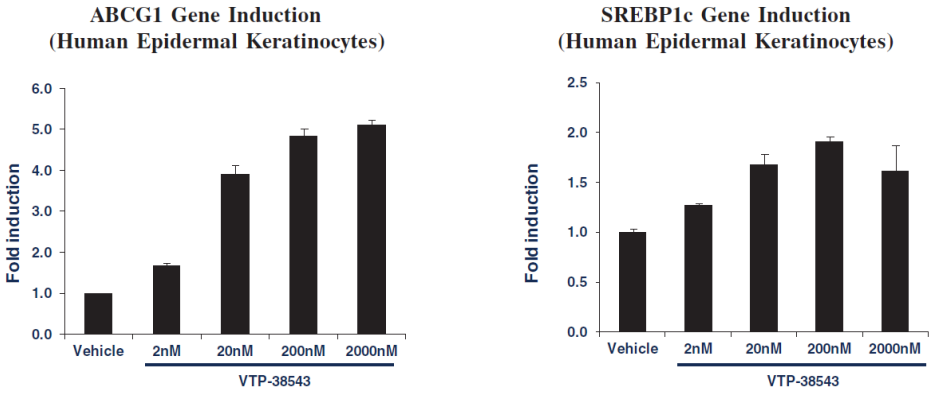
FIGURE 32: VTP-38543 INDUCED GENES CRITICAL FOR LIPID TRANSPORT AND LIPID SYNTHESIS IN SKIN



Source: Company data; Wedbush Securities, Inc.

Additionally, VTP-38543 was able to induce the expression of ABCG1 and SREBPP1c, markers for lipid metabolism and transport, in primary cultures of human epidermal keratinocytes in a dose-dependent manner.

FIGURE 33: VTP-38543 INCREASED EXPRESSION OF ABCG AND SREBP1C AFTER 24 HOURS



Source: Company data; Wedbush Securities, Inc.

DEVELOPMENT PLANS

Vitae is currently in preclinical development for VTP-38543 for atopic dermatitis. The company plans to initiate and complete two phase 1 safety and pharmacokinetic studies in H2:15. Initial plans indicate that the first trial is likely to be a single topical dose study in healthy volunteers to test safety and tolerability, as well as drug plasma pharmacokinetics and biomarkers for increased lipid and transport in skin biopsies. The second phase 1 clinical trial is likely to use topical multi-dose treatments for two weeks conducted in young adults with atopic dermatitis. The endpoints are likely to assess safety, tolerability, pharmacokinetics and clinical improvement in signs and symptoms of atopic dermatitis.

MARKET OPPORTUNITY

There are only two categories of active drugs, glucocorticoids and calcineurin inhibitors, used for the treatment of eczema. These drugs are effective; however, both have been shown to cause serious adverse events in patients. Glucocorticoids can induce skin thinning, loss of barrier function, adrenal suppression and are not useful for the face and sensitive areas. Calcineurin inhibitors can induce cancer, which triggered the black box label in 2005. The black box label severely hindered the annual sales growth of the two calcineurin inhibitors (approved in 2001) which in 2013 combined for $240 million in sales, markedly down from the total of $450 million in 2004. Given the adverse event profile of current therapies, we believe that VTP-38543 has the potential to garner a significant share of the eczema market.

Covered public companies mentioned in this report

Company	Ticker	Price (close 10/16/14)	Price Target	Rating
Lexicon Pharmaceuticals	LXRX	$1.33	$4	OUTPERFORM
Regulus Therapeutics	RGLS	$6.92	$19	OUTPERFORM
Sangamo Biosciences	SGMO	$10.95	$28	OUTPERFORM

Analyst Biography

Ms. Moussatos is a Managing Director, Equity Research responsible for the coverage of stocks in the Emerging Pharmaceuticals sector. Liana joined Wedbush from Pacific Growth Equities where she was a Senior Research Analyst. Prior to that she came from UBS Global Asset Management where she was Director and Portfolio Manager of the UBS Global Biotech Funds for five years. Previously Liana was with Bristol-Meyers Squibb where she was a manager in University and Government Licensing External Science and Technology and she also worked with Sloan-Kettering Cancer Institute in the Office of Industrial Affairs and the National Cancer Institute in the Office of Technology Development.

Liana received a B.S. in Entomology and a M.S. in Zoology and Biochemistry from Clemson University and a Ph.D. in Plant Pathology from the University of California Davis and completed a postdoctoral research fellowship in Cellular and Molecular Physiology at the Yale School of Medicine.

Liana's Edge: Liana's industry and buy-side experience provide depth in her understanding of what investors need to know along with her 13 years experience in following healthcare stocks. Her pipeline valuation includes all drug candidates / disease indications in active development and provides investors with a stock value for each program.

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

Disclosure information regarding historical ratings and price targets is available at http://www.wedbush.com/ResearchDisclosure/DisclosureQ314.pdf

Investment Rating System:
Outperform: Expect the total return of the stock to outperform relative to the median total return of the analyst's (or the analyst's team) coverage universe over the next 6-12 months.
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The Investment Ratings are based on the expected performance of a stock (based on anticipated total return to price target) relative to the other stocks in the analyst's coverage universe (or the analyst's team coverage).*

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Outperform:54% Neutral: 43% Underperform: 3%	Outperform:23% Neutral: 1% Underperform: 0%

The Distribution of Ratings is required by FINRA rules; however, WS' stock ratings of Outperform, Neutral, and Underperform most closely conform to Buy, Hold, and Sell, respectively. Please note, however, the definitions are not the same as WS' stock ratings are on a relative basis.

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Wedbush Equity Research Disclosures as of October 20, 2014

Company	Disclosure
Vitae Pharmaceuticals	1,3,5,7
Lexicon Pharmaceuticals	1
Regulus Therapeutics	1
Sangamo BioSciences	1

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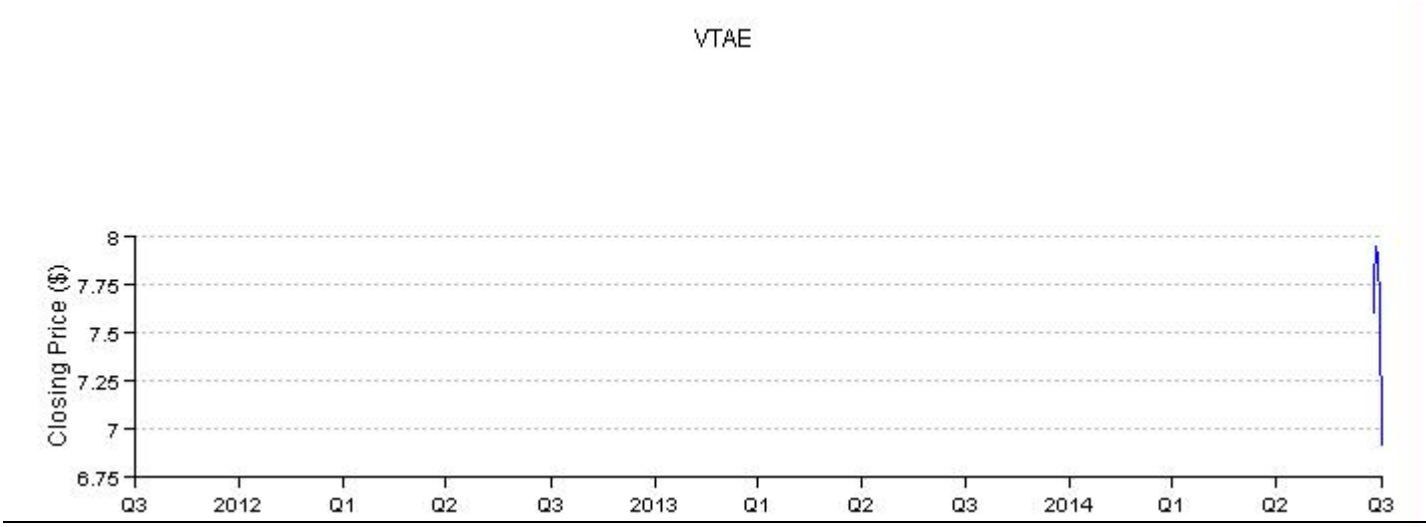

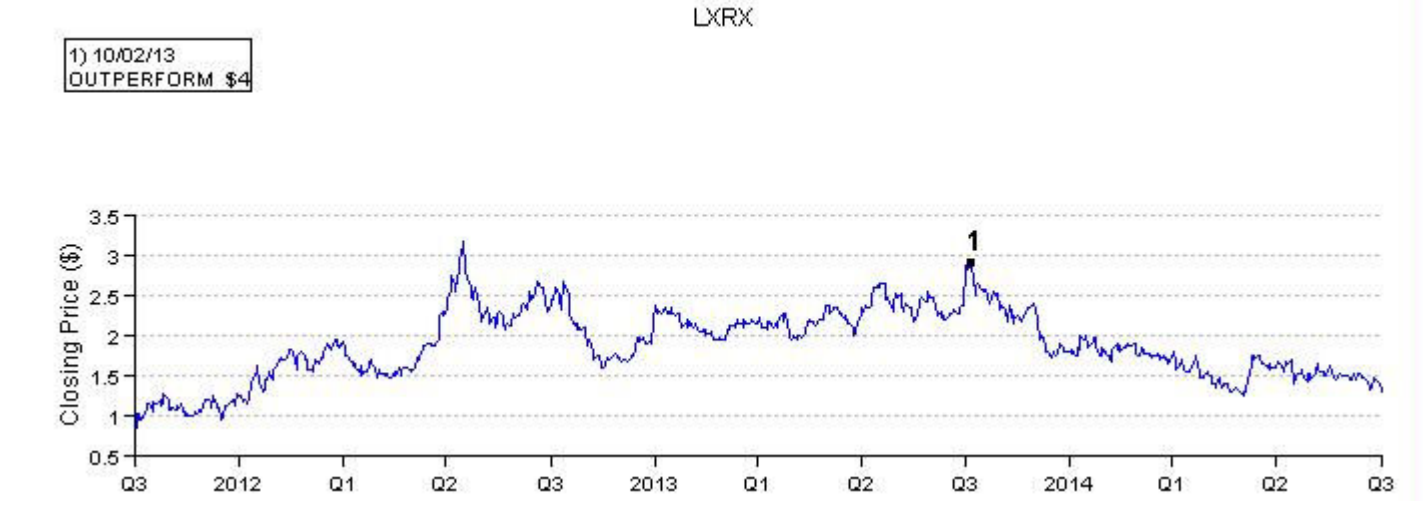

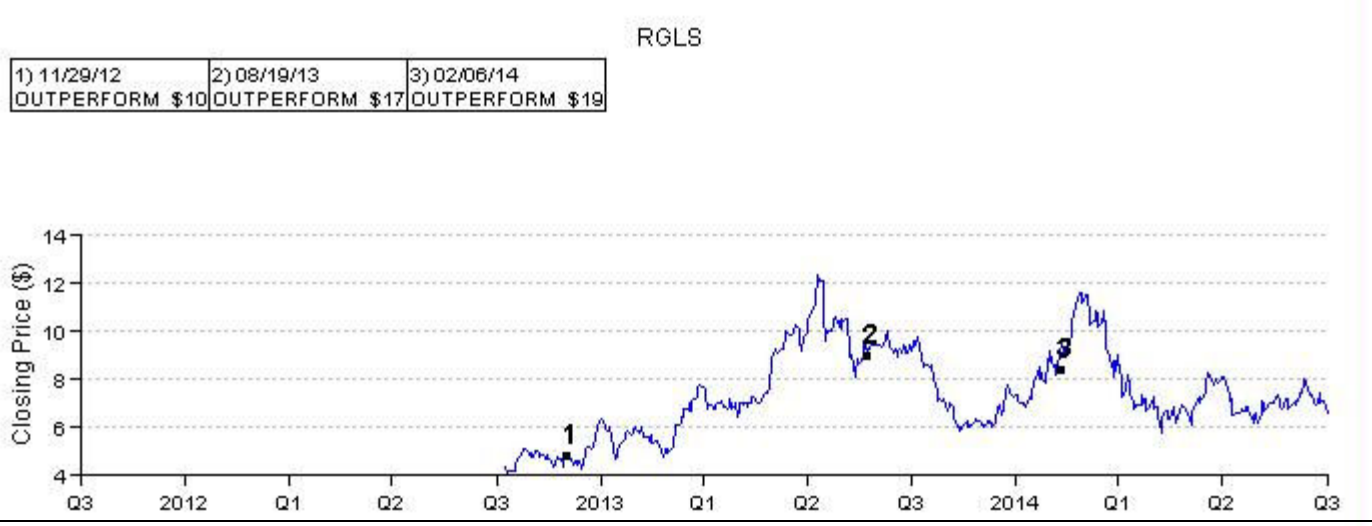

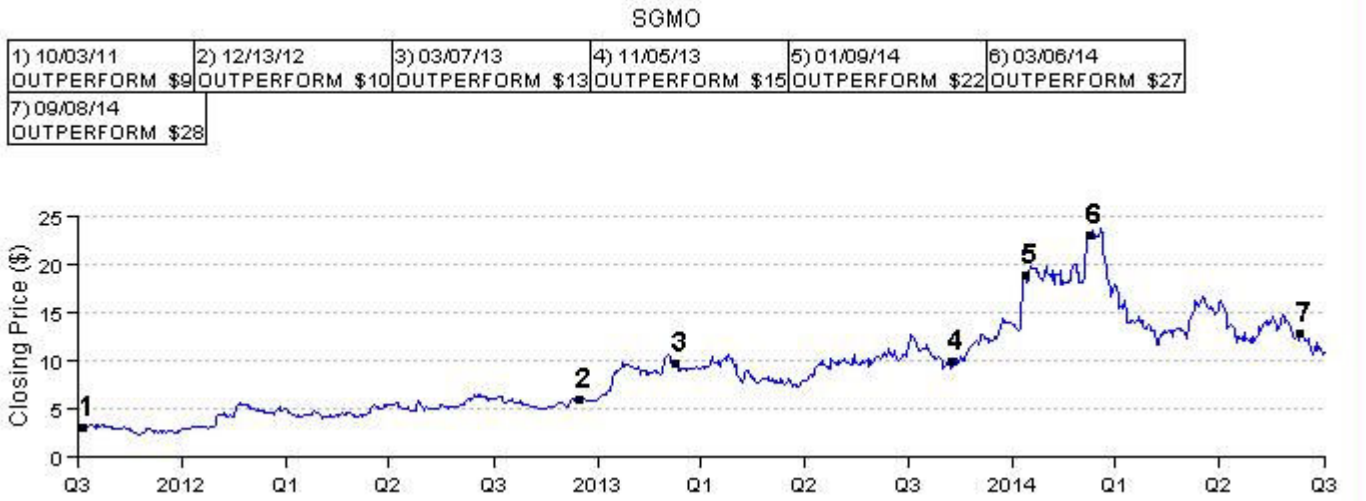

* WS changed its rating system from (Strong Buy/Buy/Hold/Sell) to (Outperform/ Neutral/Underperform) on July 14, 2009.
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