March 7, 2012

HEALTHCARE/BIOTECHNOLOGY

Stock Rating:

OUTPERFORM

12-18 mo. Price Target	$16.00
VSTM - NASDAQ	$11.10

3-5 Yr. EPS Gr. Rate	NM
52-Wk Range	$11.85-$10.00
Shares Outstanding	21.1M
Float	7.5M
Market Capitalization	$233.8M
Avg. Daily Trading Volume	7,050
Dividend/Div Yield	NM/NM
Fiscal Year Ends	Dec
Book Value	NM
2011E ROE	NM
LT Debt	$0.0M
Preferred	NA
Common Equity	NA
Convertible Available	No

52 Wk High: Range since January 2012 IPO.

EPS Diluted	Q1	Q2	Q3	Q4	Year	Mult.
2010E	--	--	--	--	(0.59)	NM
2011E	(0.29)A	(0.46)A	(0.56)A	(0.24)	(1.39)	NM
2012E	(0.17)	(0.18)	(0.19)	(0.31)	(0.85)	NM
2013E	--	--	--	--	(1.31)	NM

Verastem, Inc.

Initiating at Outperform; Novel Approach to Targeting Cancer Stem Cells

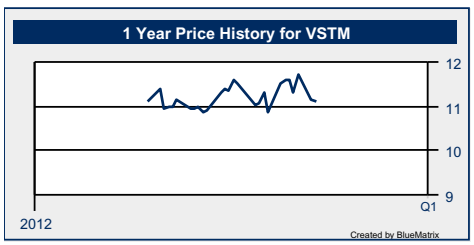
SUMMARY

We are initiating coverage of VSTM at Outperform, with a $16 PT. Using a unique discovery platform, VSTM is developing cancer stem cell (CSC)-targeted therapies. We believe there is growing evidence CSCs have a key role in tumor initiation/metastasis. VSTM expects to advance Wnt pathway inhibitor VS-507 and a FAK inhibitor (VS-4718/VS-5095) into ph.I by 1Q13. Based on preclinical results and compelling biological rationale, we believe these compounds have broad anticancer potential. VSTM first plans to pursue triple negative breast cancer (TNBC), which we believe is a substantially underserved, multi-billion dollar market. We believe VSTM is an attractive long-term investment and expect the stock to appreciate as clinical progress and high-profile scientific publications further underscore the importance of CSCs.

KEY POINTS

- **VS-507, a novel CSC-targeted therapy.** We see strong potential in Wnt pathway inhibition, as aberrant activation of this pathway is associated with tumor formation and CSC self-renewal. Based on preclinical results, we believe VS-507 will complement chemotherapy and expect a value inflection in VSTM when ph.I combo results become available, potentially in 2H13.
- **VS-4718/VS-5095 should be competitive FAK inhibitors.** We view FAK as a high-value target and note Pfizer's/Glaxo's FAK inhibitors are in ph.I testing. We believe PFE/GSK's near-term clinical results could provide positive read-through for VS-4718/VS-5095. Importantly, we believe VSTM's compounds have differentiated CSC-selectivity and expect growing focus on VS-4718/VS-5095 into ph.I results in '13.
- **TNBC is a logical first indication for CSC-targeted therapies.** There are high recurrence rates and poor survival in TNBC with current regimens, likely at least in part due to treatment-resistant CSCs. Based on this substantial unmet need, we believe VSTM's candidates have blockbuster potential in TNBC.
- **VSTM's platform has significant long-term value**. Since VSTM's proprietary technology overcomes key hurdles to developing CSC-targeted therapies, we expect the company to continue to deliver highly differentiated drug candidates. At current levels, we believe VSTM does not fully reflect the value of the company's platform.

Stock Price Performance



Company Description

Verastem, Inc. is a biopharmaceutical company focused on discovering and developing novel drugs that selectively target cancer stem cells.

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Investment Thesis

We are initiating on Verastem, Inc. with an Outperform rating and a $16 price target. Verastem is developing therapies that target cancer stem cells (CSCs), which are believed to be critical for tumor initiation, metastasis and recurrence. Based on a growing body of evidence, we believe current cancer therapies often fail because they generally do not eliminate CSCs. Verastem's unique discovery platform is designed to identify compounds that specifically target CSCs. We believe these candidates will likely complement chemotherapeutics and, in certain cases, targeted therapies, improving many current cancer treatment regimens. Led by a proven management team, the company will advance two CSC-targeting therapies into the clinic in the next 12 months, with initial proof of concept results expected in 2013. We view Verastem as an attractive long-term investment. We expect the stock to appreciate in the next 12-18 months as clinical progress and high-profile scientific publications further validate the company's drug candidates and platform.

VS-507 is a novel CSC-targeting therapy. VS-507, a formulation of the antibiotic salinomycin, inhibits the Wnt signaling pathway and kills CSCs with a high degree of selectivity. Aberrant activation of the Wnt signaling pathway is common in a range of cancers, including breast cancer, colorectal cancer, and lung cancer, and has been associated with tumor formation and recurrence. Consistent with this, Verastem's preclinical VS-507 results have shown substantially greater suppression of tumor initiation and metastasis compared to paclitaxel in mouse breast cancer models. The company plans to advance VS-507 into the clinic in late 2012, and we expect early phase I safety and biomarker results in 2013. We believe VS-507 will ultimately have the greatest potential when combined with potent existing therapies that are not specifically targeted at CSCs, and we expect the first combination data for the compound later in 2013. We believe these results will likely lead to a positive valuation inflection point for Verastem.

VS-4718 and VS-5095 inhibit FAK, a target with substantial anticancer potential. VS-4718 and VS-5095 are highly potent inhibitors of focal adhesion kinase (FAK), a target that has generated significant interest in the pharmaceutical industry. FAK overexpression is found in many solid cancers and, importantly, FAK dysregulation been associated with poorer prognosis in breast cancer. Using proprietary preclinical assays, Verastem has shown that VS-4718 and VS-5095 have high CSC selectivity, and both compounds suppress mammary tumor growth and lung metastasis in mouse models. Verastem plans to advance one of these FAK inhibitors into the clinic in early 2013, only a few months behind VS-507, and we expect phase I safety and biomarker results in late 2013. Although both Pfizer and GlaxoSmithKline have FAK inhibitors already in phase I testing (PF-04554878 and GSK2256098, respectively), we believe positive results for these candidates would provide validation for Verastem's FAK inhibitors, leading Verastem stock higher. Further, we believe Verastem's FAK inhibitors will be competitive with PF-04554878/GSK2256098, given the high degree of FAK specificity and selective activity vs. CSCs of these compounds.

Rapid clinical path in triple negative breast cancer (TNBC). Following phase I results from the VS-507 and FAK inhibitor program, Verastem expects to select one compound for phase II testing with chemotherapy in neoadjuvant TNBC. The company currently intends to use pathologic complete response (pCR) as the phase II endpoint for an advanced candidate. We believe this endpoint will enable the company to quickly produce meaningful combination data, as pCR in the neoadjuvant setting can be reached with a very short treatment duration. Achieving primary overall survival (OS) and progression-free survival (PFS) endpoints in the breast cancer treatment/adjuvant settings would require much longer timelines. Additionally, we believe pCR may ultimately be an approvable endpoint in phase III trials for the neoadjuvant setting, as this measure of benefit is an emerging surrogate marker for longer-term clinical outcomes. Importantly, a recent *JAMA* editorial by a current FDA director included positive commentary on pCR, and we expect further Agency guidance on the acceptability of this endpoint over the next several months.

Substantial market opportunity in TNBC. TNBC (HER2-, ER-, PR-) represents 10-20% of all breast cancers, and there is a growing body of evidence that suggests that CSCs have a central role in increasing TNBC treatment resistance. There are currently no targeted therapies for TNBC, and patients are most often treated with standard cytotoxic

agents. Despite aggressive treatment, patients with TNBC have reduced survival and higher recurrence rates compared to other types of breast cancer. Based on clear unmet need, we currently estimate either VS-507 or one of Verastem's FAK inhibitors achieving peak sales of $3.4B in TNBC. However, we probability-adjust these sales by 45%, given the compounds' early stage of development. Importantly, we believe both programs have significant sales potential in a broad range of additional cancers, which represents upside to our current valuation.

Verastem's proprietary discovery platform has substantial intrinsic value. The company's discovery platform for CSC-targeting therapies is based on unique EMT technology, which allows Verastem to reliably produce pure populations of CSCs in vitro. This is critical, as it allows the company to screen a large number of compounds for activity against CSCs and also identify CSC biomarkers for target patient populations. The EMT technology provides a competitive edge, as other companies in the CSC space will likely have great difficulty in isolating CSCs and growing large numbers of these cells for effective drug screening. Further, Verastem's discovery platform is covered by extensive patent applications licensed from the Whitehead Institute. With this unique drug discovery platform and a solid patent application estate, we believe Verastem has the potential to develop a new class of cancer therapeutics over the next several years. As a result, we believe Verastem's technology should be a primary component to any sum-of-the-parts assessment of the company's value.

A proven leadership team. CEO Dr. Christoph Westphal and members of the company's management team and board have a proven track record of success with a number of different biotechnology companies. Notably, Dr. Westphal was instrumental in building companies such as Sitris, which was acquired by GSK; Alnylam; and Momenta Pharmaceuticals. Importantly, the company's scientific advisory board is co-chaired by leading cancer biology innovators, Robert Weinberg, Eric Lander and Piyush Gupta, who have made substantial contributions in cancer stem cell research at MIT and are responsible for the development of Verastem's EMT technology.

Valuation

Our $16 price target is based on a probability-adjusted, forward discounted cash flow valuation. In our valuation, we model either VS-507 or one of Verastem's FAK inhibitors achieving peak royalties of approximately $510M in TNBC and $250M in total partnership milestones (Exhibit 1). However, we probability-adjust TNBC royalties and milestone revenues by 45% and 50%, respectively. Although Verastem's drug candidates are early stage, we believe an aggressive 45% probability of success is appropriate, given the company's multiple shots on goal with the Wnt and FAK inhibitor programs. We also apply a conservative 15% discount rate to reflect the early stage of Verastem's pipeline, but utilize a 3% terminal growth rate to reflect the likely productivity of the company's discovery platform. In addition to cash flows, we also include $200M in technology value, which we believe is conservative based on the valuation of comparable platform companies (Exhibit 2).

Exhibit 1: Verastem Forward DCF Valuation

(thousands USD)	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	TV
TNBC royalty	-	-	-	-	-	-	1,833	18,569	40,773	70,442	108,526	153,437	184,263	210,601	229,753	
probability-adj.	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	*45%*	
Milestones	-	-	-	15,000	-	-	20,000	40,000	-	-	25,000	-	25,000	-	-	
probability-adj.	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	
Revenue	-	-	-	15,000	-	-	21,833	58,569	40,773	70,442	133,526	153,437	209,263	210,601	229,753	
growth	*NA*	*NA*	*NA*	*NA*	*NA*	*NA*	*NA*	*168%*	*-30%*	*73%*	*90%*	*15%*	*36%*	*1%*	*9%*	
EBIT	(28,061)	(34,139)	(34,428)	(20,296)	(36,870)	(38,516)	(18,407)	16,526	(3,784)	18,856	73,560	83,956	130,811	123,196	134,505	
margin ex-milestones	NA	NA	NA	NA	NA	NA	NA	NA	-9%	27%	45%	55%	57%	58%	59%	
Tax Paid	-	-	-	-	-	-	-	826	-	943	3,678	8,396	13,081	18,479	40,351	
tax rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*5%*	*0%*	*5%*	*5%*	*10%*	*10%*	*15%*	*30%*	
EBIT after tax	(28,061)	(34,139)	(34,428)	(20,296)	(36,870)	(38,516)	(18,407)	15,700	(3,784)	17,913	69,882	75,561	117,730	104,716	94,153	
Gross Investment	(40)	(40)	(32)	(25)	(20)	(16)	(13)	(293)	(143)	(141)	(200)	(153)	(105)	(211)	(345)	
Free Cash Flow	(28,101)	(34,179)	(34,460)	(20,321)	(36,889)	(38,532)	(18,420)	15,407	(3,926)	17,772	69,682	75,407	117,625	104,506	93,809	805,190
Period	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Discounted FCF	(24,436)	(25,844)	(22,658)	(11,619)	(18,341)	(16,659)	(6,925)	5,037	(1,116)	4,393	14,978	14,094	19,117	14,770	11,529	86,046
Total FCF	42,367															
Net cash, YE2012	100,213															
Technology value	200,000															
Total value	342,579															
Shares, YE2012	21,209															
Value per share	**$16**															

Source: Oppenheimer estimates.

Exhibit 2: Acquisition Value of Early Platform-based Companies

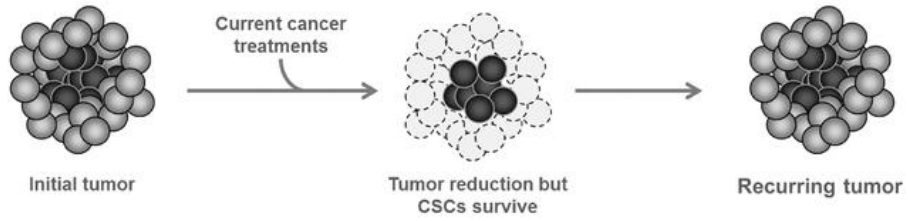
Buyer	Target	Platform	Value ($M)
Roche	Glycart	Antibody	$180
Merck	Sirna	RNAi	$865
GSK	Domantis	Antibody	$454
Esai	Morphotek	Antibody	$325
GSK	Sirtris	Sirtuins modulators/patents	$720
		Average	**$509**

Source: Company reports.

Discovery Platform

Targeting cancer stem cells (CSCs) has clear potential to lead to improved therapeutic regimens. Research has emerged over the last decade suggesting CSCs may be the underlying cause of tumor initiation, metastasis, and recurrence. Current cancer therapies may ultimately fail because they only eradicate non-CSCs, leaving CSCs behind to self-renew or produce non-CSCs. Importantly, CSCs have been isolated across many different cancer types, including certain aggressive cancers. We believe Verastem is well positioned to take advantage of the growing understanding of the importance of CSCs in cancer biology, given the company's proprietary screening technologies and leading-edge, CSC-targeting drug candidates.

Exhibit 3: CSCs Can Survive Treatment with Current Therapies



Source: Verastem.

Verastem has a unique discovery platform for CSC-targeting therapies. The company's platform is based on epithelial-to-mesenchymal transition (EMT) technology licensed from the Whitehead Institute at MIT. This technology allows for the creation of an isolated CSC population in vitro, which has allowed Verastem to screen a library of over 300,000 compounds for activity against CSCs. Verastem's library includes both new chemical entities (NCEs) and approved drugs. Additionally, the EMT technology allows Verastem to identify biomarkers for CSCs. With these biomarkers, the company can identify optimal cancer subgroups for specific CSC-targeting therapies and develop companion diagnostics. We note Verastem's EMT technology is unique, as other companies attempting to develop CSC-targeting therapies likely face significant difficulties isolating sufficient quantities of purified CSCs for effective drug screening.

Verastem has licensed a broad estate of patent applications covering the discovery platform. The company owns several families of patent applications from the Whitehead Institute, which could provide patent protection until 2025-2031. These applications include methods of identifying CSCs and CSC-targeting compounds; methods of creating CSCs such as through the EMT process; and methods of treating cancer such as through the use of CSC biomarkers. These patent applications appear very extensive, covering many aspects of the discovery process. Verastem also has an agreement with the Broad Institute for first right to negotiate for patent and patent applications covering use of biomarkers in the EMT process and also compounds identified in the screening process with EMT technology. Additionally, the discovery platform is further protected by proprietary knowledge and trade secrets.

Pipeline

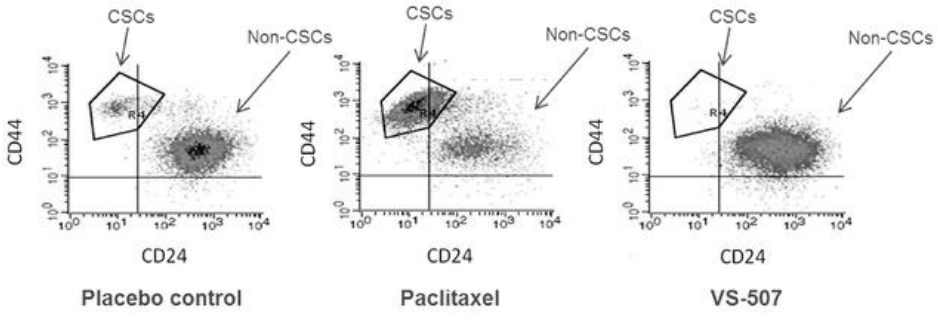
VS-507: Wnt Inhibitor Program

After screening 15,000 compounds, Verastem found salinomycin, an antibiotic, to have optimal CSC-specific toxicity with 10x selectivity for CSCs vs. non-CSCs. VS-507 is a proprietary formulation of salinomycin. Pending patent issuances, Verastem will have patent protection for method of use and formulation in the U.S. and E.U. until 2032-2033. Although Verastem does not have a composition of matter patent, the company believes its current patent applications are highly defensible given the profound novelty of the discovery.

VS-507 inhibits the Wnt pathway, which plays a key role in cancer. In vitro work suggests salinomycin inhibits the Wnt/β-catenin signaling cascade likely via suppressing Wnt1-induced LRP6 phosphorylation. Wnt/β-catenin signaling regulates cell proliferation/migration/differentiation and can also promote stem cell self-renewal. As a result, aberrant activation of this pathway has been associated with formation of tumors. Although a range of cancers carry mutations in at least one component of this pathway, dysregulation of this pathway is very common in breast cancer.

In vitro results show promising activity against CSCs. These results were based on biomarkers associated with CSCs. For example, breast CSCs express a $CD44^{High}CD24^{Low}$ profile compared to non-CSCs. Based on this profile, VS-507 exposure resulted in a decrease in CSCs in breast cancer cell lines, while exposure with a common breast cancer chemotherapy, paclitaxel, resulted a decrease in non-CSCs only (Exhibit 4). Additionally, a separate study in breast cancer cell lines showed that VS-507 exposure resulted in the loss of CSC-associated gene expression, while paclitaxel did not. Overall, we believe these results provide an initial rationale for VS-507 in paclitaxel-resistant patients or in combination with paclitaxel in first-line patients.

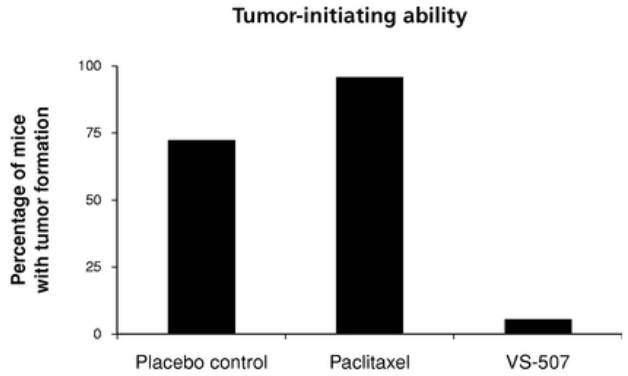
Exhibit 4: CD44/CD22 Expression in Treated Breast Cancer Cell Lines



Source: Verastem S-1 filing.

Animal studies support VS-507's anti-tumor activity vs. paclitaxel. VS-507 inhibited tumor initiation in mouse studies. Breast cancer cell lines were treated with VS-507, paclitaxel, or placebo. The cell lines were then expanded in culture and injected into mice. Substantially less tumor formation occurred in the VS-507 mouse group (Exhibit 5). In another study, VS-507 was able to reduce metastasis formation. Breast cancer cell lines were treated in vitro with VS-507, paclitaxel, or placebo prior to being injected into the tail vein of mice. After three weeks, the VS-507 and paclitaxel mouse groups showed 4x and 2x fold reduction, respectively, in metastatic burden compared to placebo. Although it is uncertain how these results will translate in the clinic, we believe the preclinical data is in-line with the hypothesis that CSC-targeting therapies can inhibit tumor initiation and metastasis.

Exhibit 5: Mouse Model of Breast Cancer



Source: Verastem S-1 filing.

TNCB is an attractive first indication for VS-507, for a number of reasons. First, TNBC is clearly a significant market opportunity, as the subpopulation's poorer prognosis and lack of effective therapies could support accelerated regulatory status and premium pricing. Second, a CSC-targeting therapy is an ideal fit for TNBC, given the high incidence of CSCs in breast cancer and the high recurrence rate with TNBC. Third, only approximately 10-20% of breast cancer has triple negative status, narrowing the target population and allowing for potentially more rapid clinical development. Further, Verastem may pursue TNBC patients with tumors classified as claudin-low, further narrowing the target population. Tumors with low claudin protein levels have a higher percentage of CSCs, are more resistant to treatment, and result in poorer prognosis.

Phase I data to provide first read on safety and potentially early efficacy in 2013. Verastem plans to file an IND and start a phase I dose escalation study in solid tumors in late 2012. We expect safety results and early CSC biomarker data in 2013. Based on animal toxicity results, there is limited visibility on VS-507's safety profile. However, management hypothesizes the drug's clinical profile could possibly include muscle weakness or gastrointestinal side effects. Once the phase I study reaches a maximum tolerated dose, Verastem will likely begin evaluating VS-507 in combination with chemotherapy by either 1) enrolling a breast cancer expansion cohort at the selected target dose, or 2) immediately transitioning VS-507 to a phase II study. Choosing to test a phase I expansion cohort would provide a first look at combination data by year=end 2013.

Phase II combination results in neoadjuvant TNBC would provide proof-of-concept. Based on phase I results from VS-507 and the company's lead FAK inhibitor, Verastem will select only one of these compounds to ultimately advance into a phase II combination study in neoadjuvant TNBC. Although clinical plans are still under discussion, management believes one possible design could be a controlled trial on a taxane-based background therapy. Verastem is pursuing neoadjuvant TNBC as the initial indication, as this setting provides a rapid path to proof-of-concept. The primary endpoint of pathologic complete response (pCR) in the neoadjuvant setting can be achieved with only 5-6 months of treatment. The path to approval may also be rapid in the neoadjuvant setting as pCR is regarded as an emerging surrogate endpoint for longer-term clinical outcomes such as recurrence free survival and could potentially be an approvable endpoint. In a recent *JAMA* editorial, Janet Woodcock, director at The Center for Drug Evaluation and Research (CDER), provided some positive commentary on pCR and noted FDA draft guidance on pCR for the neoadjuvant setting was forthcoming.

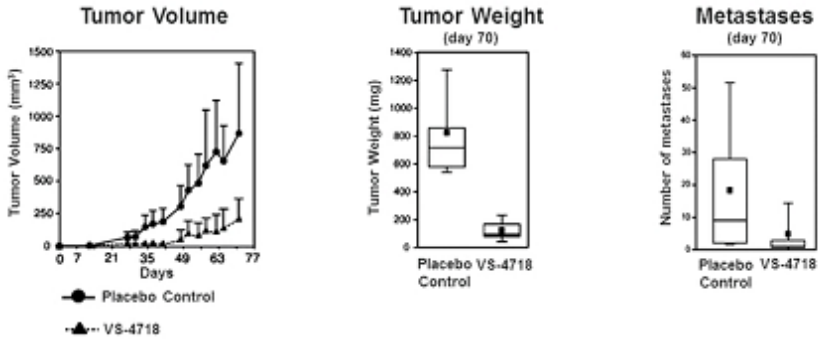
VS-4718 and VS-5095: FAK Inhibitor Program

In November 2011, Verastem licensed patent applications from Poniard Pharmaceuticals covering compounds that primarily inhibit non-receptor tyrosine focal adhesion kinase (FAK). Among these compounds, Verastem has screened two compounds, VS-4718 and VS-5095, which are approximately 25x more selective for CSCs compared to non-CSCs. Verastem paid $250,000 up-front and will potentially pay up to $13.25M in developmental and commercial milestones along with low to middle single digit royalties on net sales. The composition of matter patent applications could provide exclusivity for VS-4718 and VS-5095 in the US and Europe until 2030-2031.

FAK is involved with breast and other cancers. VS-4718 and VS-5095 have shown high in vitro selectivity for FAK, which plays a significant role in regulating cell proliferation, migration, and survival. Overexpression of FAK occurs in many solid cancers, particularly in breast cancer. In a Memorial Sloan-Kettering retrospective study of a random sampling of human breast cancer tumors, FAK was amplified (>3 copies) in as many as 50% of cases. Additionally, FAK has been implicated in tumor progression and metastasis. An analysis of breast cancer patient databases showed that elevated FAK expression correlated with shorter metastasis-free survival and was a predictor of poor survival. Further, animal studies suggest deletion of FAK in mouse models inhibits mammary tumorgenesis and lung metastasis.

Preclinical results support anti-tumor activity. VS-4718 has shown tumor growth inhibition and reduction in metastatic burden in mouse models of breast cancer. In one study, TNBC cells were implanted into mice and upon tumor formation at around 12 days, the mice were fed VS-4718 or placebo in drinking water. Mice fed VS-4718 had substantially smaller primary tumors and less lung metastases (Exhibit 6). VS-5095 has shown similar preclinical results as VS-4718.

Exhibit 6: Mouse Model of Triple Negative Breast Cancer



Source: Verastem S-1 filing.

FAK program offers additional shots on goal. Based on FAK overexpression in breast cancer and encouraging preclinical results, we believe both FAK inhibitors have potential in TNBC. Verastem will only select one FAK inhibitor to enter the clinic and notes VS-4718 and VS-5095 have shown very similar preclinical profiles. The selected FAK inhibitor will be compared against VS-507 to determine which compound will be best for phase II development in TNBC. If the FAK inhibitor is not selected for TNBC, we believe the compound can be robustly developed in many other solid cancers with high numbers of CSCs and FAK overexpression such as pancreatic, colon, brain, and lung cancers. Verastem is still developing clinical plans for the company's candidates in indications outside of TNBC.

FAK inhibitor program to be developed in parallel with VS-507. Verastem plans to advance one of the FAK inhibitors into a phase I dose escalation study in solid tumors in early 2013. The FAK program will only lag the VS-507 program by roughly 3 months allowing Verastem to evaluate the compounds generally in parallel. We would expect safety and biomarker results for the FAK inhibitor program in late 2013. Like VS-507, there is limited visibility on the safety profile of Verastem's FAK inhibitors. However, other FAK inhibitors discussed below have shown GI side effects. Once the phase I study reaches a maximum tolerated dose, Verastem will likely evaluate the FAK inhibitor in combination with chemotherapy by either 1) enrolling a phase I dose expansion cohort in breast or other cancer, or 2) immediately initiating a phase II study. We note if a phase I expansion cohort is evaluated prior to phase II development, we could see initial combination data by early 2014.

There is strong interest in FAK inhibitors, but few competitors in development. We are aware of only a few early FAK inhibitors in the clinic. Pfizer completed a phase I dose escalation study in solid tumors for PF-00562271, but now appears to be focusing on a next-generation FAK inhibitor, PF-04554878. This second compound is in a phase I dose-escalation study in solid tumors and Pfizer has reported initial results. GSK is developing GSK2256098 in a phase I dose escalation solid tumor study, which has an expansion cohort focused on glioblastoma. Results appear to be very limited. Additionally, a private

company, CureFAKtor, will move CFAK-C4 into the clinic and begin a phase I study with gemcitabine in pancreatic cancer in early 2012. We believe interest in FAK inhibition, from GSK and particularly Pfizer, highlights the potential of this target. Although Verastem may be behind Pfizer and GSK, we believe clinical success with other FAK inhibitors provides some validation for VS-4718 and VS-5095. Additionally, we believe Verastem could have a competitive edge, as VS-4718 and VS-5095's were specifically screened for their high selectivity for CSCs. Additionally, Verastem's knowledge of CSC biomarkers may allow the company to target the optimal cancer subpopulations.

Pfizer's FAK inhibitors showed early efficacy and manageable safety. Pfizer reported final phase I dose escalation results (n=99) for PF-00562271 in solid tumors at ASCO 2010. Adverse events were largely grade 1/2 and reversible. They included GI events (nausea, vomiting, diarrhea), peripheral edema, anorexia, fatigue, headache, dizziness, and hypotension. Dose limiting toxicities were headache, nausea, vomiting and facial swelling. Although 75% of patients had 3 or more prior treatments, the drug demonstrated some signs of efficacy. For example, 7 of 20 colorectal cancer patients demonstrated stable disease. For PF-04554878, interim phase I dose-escalation results (n=36) were reported at ASCO 2011. Similar to the first compound's, adverse events were also largely grade 1/2 and reversible. PF-04554878 also had similar side effects such as nausea (33%), vomiting (31%), diarrhea (19%), decreased appetite (17%), headache (19%), and fatigue (25%), with the exception of unconjugated hyperbilirubenemia (28%). Dose limiting toxicities were grade 3 headache (n=1) and unconjugated hyperbilirubenemia (n=2). PF-04554878 also showed early efficacy with 33% of patients achieving stable disease at two cycles of treatment and 11% maintaining stable disease beyond this point. Overall, it is difficult to draw any meaningful conclusions regarding VS-4718 and VS-5095 based on these early results. Notably, breast cancer comprised a very small portion of patients in both of Pfizer's trials.

Market Opportunity

Breast cancer is a large market that is still underserved. Breast cancer is the second most common cancer with an annual incidence of approximately 230,000 in the U.S. alone. Over the last 15 years, new therapies have emerged primarily for two breast cancer subgroups: 1) patients overexpressing human epidermal growth factor receptor 2 (HER2+) and 2) patients expressing hormone receptors for estrogen (ER+) or progesterone (PR+). Approximately 20-25% of breast cancer patients are HER2+ and can be treated with HER2 targeted therapies such as Herceptin and Tykerb. Approximately 65% of breast cancer patients are ER+ and PR+ and are largely responsive to hormonal therapies such as generic tamoxifen, Aromasin, and Arimidex. Verastem is pursuing TNBC (HER2-/ER-/PR-) which does not respond to the previously mentioned therapies. TNBC represents 10-20% of all breast cancer, slightly smaller than the HER2+ population targeted by Herceptin and Tykerb.

Triple negative breast cancer represents a significant unmet medical need. Compared to other breast cancer types, TNBC has occurred in higher frequency in women who are younger, are of African descent, or exhibit the BRCA1 mutation. Based on very large population based studies, TNBC patients have poorer prognosis with one study showing worse survival compared to other breast cancer types at 5 years (77% vs. 93%). These studies also show TNBC patients have greater relapse rates and worse time to relapse particularly within the first three years of diagnosis. In addition to a poor prognosis, there are no targeted therapies for TNBC, which consists of a fairly heterogeneous group of cancers. As a result, there are no standard treatments for TNBC and physicians utilize common systemic chemotherapies. Encouragingly, ER- cancers have shown chemosensitivity and notably, taxanes and anthracyclines have shown higher pathologic complete responses in the neoadjuvant setting for TNBC compared to other types of breast cancers.

The Wnt and FAK programs have blockbuster potential in TNBC. We model either VS-507 or one of Verastem's FAK inhibitors achieving initial approval in neoadjuvant TNBC, followed by expansion into the primary care and adjuvant settings. We estimate peak sales of $3.4B, with a 15% royalty stream to Verastem (Exhibit 7). Our estimates are based on $60,000 US annual pricing per patient, a premium to current breast cancer therapies such as Herceptin. We believe this is justified given the unmet need in TNBC

and US pricing appreciation while Verastem's candidates are in development. Importantly, we probability-adjust TNBC royalties by 45% in our valuation.

Exhibit 7: Oppenheimer TNBC Revenue Model

Triple Negative Breast Cancer	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027
UNITED STATES											
Neoadjuvant treatment											
Addressable population	195,921	200,506	205,197	209,999	214,913	219,942	225,089	230,356	235,746	241,263	246,908
Pts receiving neoadjuvant chemo	97,961	100,253	102,599	105,000	107,457	109,971	112,544	115,178	117,873	120,631	123,454
HER2-/ER-/PR- patients	19,592	20,051	20,520	21,000	21,491	21,994	22,509	23,036	23,575	24,126	24,691
Penetration	*0%*	*0%*	*3%*	*9%*	*17%*	*23%*	*28%*	*36%*	*39%*	*41%*	*42%*
Neoadjuvant patients treated	0	0	616	1,890	3,654	5,059	6,302	8,293	9,194	9,892	10,370
Treatment duration, months	6	6	6	6	6	6	6	6	6	6	6
Average price/patient	$ 30,000	$ 30,000	$ 30,000	$ 31,500	$ 33,075	$ 34,729	$ 36,465	$ 37,924	$ 39,441	$ 41,018	$ 42,454
U.S. Neoadjuvant Sales (000's)	**$ -**	**$ -**	**$ 18,468**	**$ 59,535**	**$ 120,840**	**$ 175,681**	**$ 229,821**	**$ 314,495**	**$ 362,622**	**$ 405,744**	**$ 440,254**
Primary treatment											
Addressable population	98,142	100,438	102,789	105,194	107,655	110,174	112,753	115,391	118,091	120,854	123,682
HER2-/ER-/PR- patients	19,628	20,088	20,558	21,039	21,531	22,035	22,551	23,078	23,618	24,171	24,736
1st-line HER2-/ER-/PR- patients	6,477	6,629	6,784	6,943	7,105	7,272	7,442	7,616	7,794	7,976	8,163
Penetration	*0%*	*0%*	*0%*	*3%*	*10%*	*18%*	*23%*	*29%*	*35%*	*39%*	*41%*
1st-line patients treated	0	0	0	208	711	1,309	1,712	2,209	2,728	3,111	3,347
Treatment duration, months	10	10	10	10	10	10	10	10	10	10	10
Average price/patient	$ 50,000	$ 50,000	$ 50,000	$ 52,500	$ 55,125	$ 57,881	$ 60,775	$ 63,206	$ 65,735	$ 68,364	$ 70,757
1st-line U.S. Sales	**$ -**	**$ -**	**$ -**	**$ 10,935**	**$ 39,168**	**$ 75,759**	**$ 104,022**	**$ 139,596**	**$ 179,318**	**$ 212,666**	**$ 236,812**
Refractory HER2-/ER-/PR- patients	13,151	13,459	13,774	14,096	14,426	14,763	15,109	15,462	15,824	16,194	16,573
Penetration	*0%*	*0%*	*0%*	*3%*	*5%*	*10%*	*15%*	*23%*	*29%*	*34%*	*36%*
Refractory patients treated	0	0	0	352	721	1,476	2,266	3,479	4,589	5,506	5,966
Treatment duration, months	6	6	6	6	6	6	6	6	6	6	6
Average price/patient	$ 30,000	$ 30,000	$ 30,000	$ 31,500	$ 33,075	$ 34,729	$ 36,465	$ 37,924	$ 39,441	$ 41,018	$ 42,454
Refractory U.S. Sales	**$ -**	**$ -**	**$ -**	**$ 11,101**	**$ 23,857**	**$ 51,271**	**$ 82,642**	**$ 131,938**	**$ 180,994**	**$ 225,852**	**$ 253,299**
U.S. Primary Treatment Sales (000's)	**$ -**	**$ -**	**$ -**	**$ 22,035**	**$ 63,024**	**$ 127,031**	**$ 186,664**	**$ 271,535**	**$ 360,312**	**$ 438,519**	**$ 490,111**
Adjuvant treatment											
Addressable population	195,921	200,506	205,197	209,999	214,913	219,942	225,089	230,356	235,746	241,263	246,908
Pts receiving adjuvant chemo	97,961	100,253	102,599	105,000	107,457	109,971	112,544	115,178	117,873	120,631	123,454
HER2-/ER-/PR- patients	19,592	20,051	20,520	21,000	21,491	21,994	22,509	23,036	23,575	24,126	24,691
Penetration	*0%*	*0%*	*0%*	*3%*	*6%*	*11%*	*20%*	*28%*	*32%*	*35%*	*37%*
Adjuvant patients treated	0	0	0	630	1,289	2,419	4,502	6,450	7,544	8,444	9,136
Treatment duration, months	12	12	12	12	12	12	12	12	12	12	12
Average price/patient	$ 60,000	$ 60,000	$ 60,000	$ 63,000	$ 66,150	$ 69,458	$ 72,930	$ 75,848	$ 78,881	$ 82,037	$ 84,908
U.S. Adjuvant Sales (000's)	**$ -**	**$ -**	**$ -**	**$ 39,690**	**$ 85,299**	**$ 168,043**	**$ 328,316**	**$ 489,214**	**$ 595,072**	**$ 692,734**	**$ 775,686**
Total U.S. Sales (000's)	**$ -**	**$ -**	**$ 18,468**	**$ 121,260**	**$ 269,164**	**$ 470,755**	**$ 744,801**	**$ 1,075,244**	**$ 1,318,006**	**$ 1,536,997**	**$ 1,706,051**
Annual U.S. price/patient	$ 60,000	$ 60,000	$ 60,000	$ 63,000	$ 66,150	$ 69,458	$ 72,930	$ 75,848	$ 78,881	$ 82,037	$ 84,908
YoY increase	*NA*	*NA*	*NA*	*5.0%*	*5.0%*	*5.0%*	*5.0%*	*4.0%*	*4.0%*	*4.0%*	*3.5%*
EUROPE											
Neoadjuvant treatment											
Addressable population	315,951	323,344	330,910	338,654	346,578	354,688	362,988	371,482	380,174	389,070	398,175
Pts receiving neoadjuvant chemo	157,975	161,672	165,455	169,327	173,289	177,344	181,494	185,741	190,087	194,535	199,087
HER2-/ER-/PR- patients	31,595	32,334	33,091	33,865	34,658	35,469	36,299	37,148	38,017	38,907	39,817
Penetration	*0%*	*0%*	*1%*	*8%*	*16%*	*23%*	*28%*	*36%*	*39%*	*41%*	*42%*
Neoadjuvant patients treated	0	0	331	2,709	5,545	8,158	10,164	13,373	14,827	15,952	16,723
Treatment duration, months	6	6	6	6	6	6	6	6	6	6	6
Average price/patient	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250
EU Neoadjuvant Sales (000's)	**$ -**	**$ -**	**$ 8,686**	**$ 71,117**	**$ 145,563**	**$ 214,143**	**$ 266,796**	**$ 351,050**	**$ 389,203**	**$ 418,737**	**$ 438,987**
Primary treatment											
Addressable population	157,835	161,528	165,308	169,176	173,135	177,186	181,332	185,576	189,918	194,362	198,910
HER2-/ER-/PR- patients	31,567	32,306	33,062	33,835	34,627	35,437	36,266	37,115	37,984	38,872	39,782
1st-line HER2-/ER-/PR- patients	10,417	10,661	10,910	11,166	11,427	11,694	11,968	12,248	12,535	12,828	13,128
Penetration	*0%*	*0%*	*0%*	*3%*	*10%*	*18%*	*23%*	*29%*	*35%*	*39%*	*41%*
1st-line patients treated	0	0	0	335	1,143	2,105	2,753	3,552	4,387	5,003	5,383
Treatment duration, months	10	10	10	10	10	10	10	10	10	10	10
Average price/patient	$ 43,750	$ 43,750	$ 43,750	$ 43,750	$ 43,750	$ 43,750	$ 43,750	$ 43,750	$ 43,750	$ 43,750	$ 43,750
1st-line E.U. Sales	**$ -**	**$ -**	**$ -**	**$ 14,655**	**$ 49,993**	**$ 92,093**	**$ 120,427**	**$ 155,396**	**$ 191,936**	**$ 218,876**	**$ 235,485**
Refractory HER2-/ER-/PR- patients	21,150	21,645	22,151	22,670	23,200	23,743	24,299	24,867	25,449	26,045	26,654
Penetration	*0%*	*0%*	*0%*	*3%*	*5.0%*	*10%*	*15%*	*23%*	*29%*	*34%*	*36%*
Refractory patients treated	0	0	0	567	1,160	2,374	3,645	5,595	7,380	8,855	9,595
Treatment duration, months	6	6	6	6	6	6	6	6	6	6	6
Average price/patient	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250	$ 26,250
Refractory E.U. Sales	**$ -**	**$ -**	**$ -**	**$ 14,877**	**$ 30,450**	**$ 62,325**	**$ 95,675**	**$ 146,871**	**$ 193,731**	**$ 232,447**	**$ 251,880**
EU Primary Treatment Sales (000's)	**$ -**	**$ -**	**$ -**	**$ 29,532**	**$ 80,443**	**$ 154,418**	**$ 216,103**	**$ 302,268**	**$ 385,666**	**$ 451,323**	**$ 487,365**
Adjuvant treatment											
Addressable population	315,087	322,460	330,005	337,727	345,630	353,718	361,995	370,466	379,135	388,006	397,086
Pts receiving adjuvant chemo	157,543	161,230	165,003	168,864	172,815	176,859	180,997	185,233	189,567	194,003	198,543
HER2-/ER-/PR- patients	31,509	32,246	33,001	33,773	34,563	35,372	36,199	37,047	37,913	38,801	39,709
Penetration	*0%*	*0%*	*0%*	*3%*	*6%*	*11%*	*20%*	*28%*	*32%*	*35%*	*37%*
Adjuvant patients treated	0	0	0	1,013	2,074	3,891	7,240	10,373	12,132	13,580	14,692
Treatment duration, months	12	12	12	12	12	12	12	12	12	12	12
Average price/patient	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500
EU Adjuvant Sales (000's)	**$ -**	**$ -**	**$ -**	**$ 53,192**	**$ 108,874**	**$ 204,272**	**$ 380,095**	**$ 544,585**	**$ 636,946**	**$ 712,962**	**$ 771,339**
Total EU Sales (000's)	**$ -**	**$ -**	**$ 8,686**	**$ 153,841**	**$ 334,879**	**$ 572,833**	**$ 862,994**	**$ 1,197,902**	**$ 1,411,816**	**$ 1,583,022**	**$ 1,697,691**
Annual E.U. price/patient	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500	$ 52,500
YoY increase	*NA*	*NA*	*NA*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
WW Sales (000's)	**$ -**	**$ -**	**$ 27,154**	**$ 275,101**	**$ 604,043**	**$ 1,043,587**	**$ 1,607,795**	**$ 2,273,146**	**$ 2,729,822**	**$ 3,120,019**	**$ 3,403,742**
WW Royalties (000's)	**$ -**	**$ -**	**$ 4,073**	**$ 41,265**	**$ 90,606**	**$ 156,538**	**$ 241,169**	**$ 340,972**	**$ 409,473**	**$ 468,003**	**$ 510,561**
royalty rate	*15.0%*	*15.0%*	*15.0%*	*15.0%*	*15.0%*	*15.0%*	*15.0%*	*15.0%*	*15.0%*	*15.0%*	*15.0%*

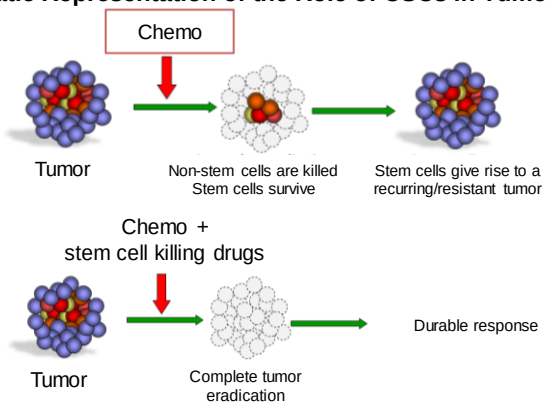
Source: Oppenheimer estimates.

We see substantial upside in indications outside breast cancer. CSCs have been identified in many types of cancer including prostate, pancreatic, colon, brain, lung and leukemia. FAK overexpression and Wnt signaling pathway dysregulation are also found in a wide range of cancer. Further, with strong knowledge of CSC biomarkers, Verastem should be able to identify optimal cancer subgroups for the company's drug candidates. We do not include sales for cancers types outside of TNBC in our valuation.

Cancer Stem Cell Background

Introduction
Cytotoxic chemotherapeutic agents were developed based on the idea that agents that will preferentially kill rapidly dividing cells should be effective anticancer therapies. However, despite advances that have led to the development of new therapies, tumor recurrence and metastasis following successful initial remission continue to be a challenge. The cancer stem cell theory attempts to answer why chemotherapeutic agents, even though they can be successful at killing rapidly dividing tumor cells, do not prevent metastasis and rumor recurrence. A major challenge now is to discover agents and strategies that target cancer and tumor relapse at their apparent source.

Exhibit 8: Schematic Representation of the Role of CSCs in Tumor Recurrence



Source: Verastem, Inc.

Stem Cells and Cancer Stem Cells
A stem cell is by definition any cell that has the ability for self-renewal and to differentiate into a different cell type. For instance, hematopoietic stem cells, found in the bone marrow, have the ability for self-renewal and can give rise to all of the different types of cells that form our blood. Similarly, by definition, a cancer stem cell also has the ability to self-renew and to regrow the tumor from which it was isolated or identified. While a normal stem cell ultimately gives rise to a differentiated tissue with no proliferative potential, a CSC gives rise to progeny that do not undergo terminal differentiation and exhibit uncontrolled proliferation. The stem cell theory maintains that a fraction of the cells found in the tumor are such CSCs. The main issue has been that the definition of a CSC has been, until recently, a functional one. A CSC is a cancer cell that can regrow the original tumor, which implies that every time potential CSCs were identified, their tumor-forming ability had to be confirmed. With the establishment of CSC markers, the identification of these cells has become less of a challenge. However, it has still been difficult to purify the rare CSCs away from the rest of the tumor, to obtain enough of them for drug-screening, and, finally, to understand why these cells are resistant to drugs that kill the tumor they were isolated from. Verastem's technology allows for the transformation of cancer cells into CSCs *in vitro*, which allows for abundant and pure fraction of CSCs to be used to screen novel therapies.

Research in the mid-1990s showed that tumor-initiating cells were present in acute myeloid leukemia (AML). Briefly, purified populations of leukemia-initiating cells were isolated that contained a genetic abnormality identical to that found in the blast cells. These AML-initiating cells, and not the more abundant blast cells, were able to regrow tumors when implanted in mouse tumor models.

Limitations of current CSC research
The main challenge of CSC research rests on the fact that only cells that could get implanted in mice and form tumors could be confirmed as CSCs. Instead, other tumor-initiating cells from humans that were unable to implant in the mouse model, for reasons unrelated to their "stemness," would not be confirmed as CSCs. This meant that there was

an underestimation of how many different types of CSCs really existed. In addition, liquid tumors were much easier to isolate potential stem cells from, since cancer cells are not held together like they are in a solid tumor. Solid tumors instead contain both tumor cells and various stromal cells that support the growth of the tumor. Breaking the interactions between the cancer and stromal cells might change the properties of the tumor cells. In addition, preclinical models may not capture the original tumor microenvironment that solid tumors are a lot more dependent on than liquid tumors. Nevertheless, advances in research have led to the identification of CSCs from several solid tumors using appropriate cell surface markers. Most importantly, a number of markers used in isolating cancer stem cells have been shown to be predictive of disease progression, indicating that they identify clinically important cell populations. Despite the challenges, the CSC theory explains the differences in the cells that make up the tumor, and offers a new path for cancer drug development. Finally, unlike the cells that form the bulk of the tumor that are genetically unstable and rapidly accumulate new mutations, since tumor-initiating cells rely on self-renewal and maintenance of a primitive/non-differentiated state, signaling pathways that would be required for self-renewal are more likely to be conserved, consequently making them potentially good drug targets. Importantly, Verastem's technology to induce the formation of CSCs *in vitro* allows circumvents the prior issues of cell purification and *in vitro* growth in the absence of stromal cells.

Stem Cells, Cancer Stem Cells and Breast Cancer

The mammary gland is unique since most of its development occurs after birth, and a considerable number of adult breast stem cells are required. One tempting extrapolation from this is that the mammary gland is particularly prone to carcinogenesis and that adult breast stem cells play a very important role in breast cancer. In breast cancer patients, a recent study has also demonstrated the presence of $CD44^{high}CD24^{low}$ stem-like cells in metastases, suggesting a role for breast CSCs in the metastatic process of this disease. Importantly, Verastem's initial target indication for its compounds is breast cancer.

Chemoresistance of Cancer Stem Cells

CSCs are likely to share many of the properties of normal stem cells, including relative quiescence, resistance to drugs and toxins through the expression of several drug pumps, active DNA repair that fixes the damage of chemotherapy and resistance to apoptosis. Therefore, tumors might have a built-in population of drug-resistant pluripotent cells that can survive chemotherapy and repopulate the tumor. Stem cells are different from more mature differentiated cells in that they divide infrequently. Thus, antimitotic chemotherapies are less effective in stem cells than in the cells of the bulk of the tumor. Scientists from the Weinberg Lab at MIT showed that when cancer cells are induced to form CSCs, they acquire chemoresistance to common chemotherapies. This would allow for the screening of compounds that could kill CSCs normally not killed by cytotoxics, that, when used with chemotherapy, would lead to eradication of the bulk of the tumor, as well as the CSC core. Since TNBC has a high recurrence rate, presumably due to chemoresistant CSCs left behind the initial chemo regimens, targeting TNBC as an initial target indication for Verastem's compounds makes sense scientifically.

Cancer Stem Cell Renewal

Non-stem cells divide into two daughter cells that have the same properties as the original mother cell. However, stem cells, and consequently CSCs, can also divide into one daughter that is an exact copy of the original and retains the ability to divide and initiate additional tumors, and another daughter cell that differentiates to a rapidly dividing cancer cell. This asymmetric division is controlled by various pathways that govern stem cell self-renewal and differentiation. Self-renewal pathways are tightly controlled in normal stem cells and they tend to be constitutively activated or unregulated in CSCs. In normal stem cells, the three major signaling pathways regulating these processes are Wnt, Notch and Hedgehog. Not surprisingly, these pathways frequently go unregulated in many types of cancers, and specifically within subpopulations of these cancers that possess stem-like properties. Consequently, targeting these pathways allows for new classes of targeted agents, in addition to the promise of killing the cells responsible for tumor initiation, recurrence, and even metastasis.

Epithelial-Mesenchymal Transition (EMT) and Cancer

Epithelial cells provide cell-cell cohesion essential to maintaining the integrity of the multicellular organism and form a barrier from the external environment. Mesenchymal cells provide support and structure to the epithelial cells through the production of an extracellular matrix (ECM) and, and unlike the rather confined and immobile epithelial cell,

are highly motile and invasive. Epithelial and mesenchymal cells are not necessarily confined to stay that way early in development. Epithelial-mesenchymal transition (EMT) and the reverse mesenchymal-epithelial transition (MET) provide flexibility in the development of different tissues during embryogenesis, and allow for dynamic tissue repair during fibrosis and after injury. EMT is characterized by down-regulation of epithelial markers, such as E-cadherin, and an up-regulation of mesenchymal markers, particularly vimentin or fibronectin, accompanied by an increase in cell migration and invasion.

Oncogenic EMT is associated with loss of adherence, cytoskeletal changes and the acquisition of a motile and invasive phenotype similar to developmental EMT. In fact, clinical evidence suggests that activation of EMT regulators in cancer cells correlates with poor patient outcomes and tumor aggressiveness.

Verastem's technology to induce EMT and screen for anti-CSC compounds.
The Weinberg Lab at MIT has shown that induction of an EMT in immortalized human mammary epithelial cells results in the down-regulation of epithelial markers (E-cadherin), up-regulation of mesenchymal markers, and $CD44^{high}/CD24^{low}$ markers, precisely the antigenic phenotype that has been ascribed to neoplastic mammary stem cells. These cells were also increasingly more resistant than their non-CSC precursors to chemotherapeutic agents (paclitaxel and doxorubicin). Furthermore, these CSCs were also capable of growing unattached to a surface, similar to CSCs isolated from patients and unlike their precursors which would normally undergo apoptosis. Weinberg's group went on to screen libraries of compounds that would reduce the viability of these newly induced CSCs. Importantly, compounds that targeted the Wnt and FAK pathways showed cytotoxic activity against the induced CSCs. Wnt is one of the core developmental pathways preserved in stem cells, and has been extensively studied in cancer. The FAK pathway is crucial for the controlled migration of cells from their original site, as one would expect from metastatic CSCs. Finally, it makes scientific sense that these induced CSCs would be sensitive to FAK inhibitors, since up-regulation of FAK is required for the survival of cancer cells that can grow unattached to a surface. Below, we discuss the roles of the Wnt and FAK pathways in CSCs, in particular, their role in breast CSCs.

The Wnt Pathway and CSCs
The Wnt pathway is aberrantly activated across numerous cancers. The best known role for Wnt/β-catenin signaling is in colon cancer, where nearly 90% of these tumors harbor mutations that result in Wnt/β-catenin pathway activation. The most common type of mutation in colon cancer results in the inactivation of APC, thus driving constitutive activation of β-catenin. Activating mutations within β-catenin itself are also found in colon cancer. Furthermore, β-catenin has a critical role in CSCs, as illustrated by the fact that stem-like colon cells with a high level of β-catenin signaling have greater tumorigenic potential than counterpart cells with low β-catenin signaling.

Wnt ligands binding to their receptors Frizzled (Fz) and low-density lipoprotein receptor-related protein 5 (LRP5) and LRP6 activate the Wnt pathway, leading to the release of β-catenin from the "degradation complex," which consists of adenomatous polyposis coli protein (APC), axis inhibition protein (axin), glycogen synthase kinase 3 (GSK3) and casein kinase 1 (CK1). This facilitates the entry of β-catenin into the nucleus, where it regulates target gene transcription through association with the transcription factor TCF–LEF (lymphoid enhancer binding factor). Soluble Frizzled-related protein (SFRP) and Dickkopf protein (DKK) are endogenous secreted antagonists of Wnt signaling.

Wnt and CSC Self-renewal
Prior research has shown that the Wnt pathway regulates the self-renewal of leukemia-initiating cells. Mutations downstream of Wnt receptors, such as those found in APC or β-catenin, were the first examples of aberrant Wnt signaling in human cancers. Some cancers, however, demonstrate hallmarks of constitutive Wnt signaling in the absence of downstream mutations.

Triple negative breast cancers and non-small cell lung cancers (NSCLCs) have been known to harbor high levels of uncomplexed cytosolic β-catenin and exhibit a high basal level of Wnt/β-catenin transcriptional activation, suggesting an autocrine mechanism of Wnt activation. A role for autocrine Wnt signaling has been described in various breast cancer cell lines. Certain triple negative breast cancer lines have been shown to express Wnt ligands, and harbor hallmarks of aberrant Wnt/β-catenin signaling in the absence of common mutations in the pathway. Wnt signaling in these cells is inhibited by

overexpression of endogenous inhibitors such as Dkk1, thus validating an autocrine Wnt loop that is amenable to pharmacologic inhibition.

Wnt/ β-catenin and EMT in Breast Cancer

The Wnt pathway is activated during multiple stages of mammary development from the earliest stage during embryogenesis, where Wnt signaling is necessary for mammary bud formation, to the alveolar differentiation and side branching observed during pregnancy. Activation of the Wnt pathway in breast cancer cells induces EMT in numerous models. Importantly, markers indicating active Wnt/β-catenin signaling, including nuclear β-catenin, also correlate clinically in breast cancer patients with poor prognosis. Furthermore, the Wnt pathway regulates EMT. Finally, cells undergoing EMT possess important properties normally found in stem cells, including the acquisition of the stem cell related $CD44^{high}CD24^{low}$ marker profile. Taken together, these data implicate the Wnt/β-catenin pathway in epithelial plasticity and EMT in both development and cancer.

Strategies for Targeting the Wnt Pathway

While agents targeting the Hedgehog and Notch pathways have been successful at showing antitumor activity, Wnt pathway antagonists have been more challenging to develop, for two reasons. First, since there are 19 Wnt ligands and 10 Fz receptors, it has been challenging to target Wnt/Fz successfully. Second, β-catenin is an intracellular signaling protein with no known enzymatic function, which is why it has been considered to be “undruggable.” The only approach to target the Wnt pathway that has reached the clinic is targeting the epithelial cell adhesion molecule (EpCAM). However, since EpCAM is also expressed on most normal epithelial cells, tumor-specific targeting has been somewhat of a challenge.

Exhibit 9: Select Anti-Cancer Agents Targeting the Hedgehog, Notch and Wnt Pathways

Agent	Target	Cancer indication	Sponsor	Stage
Vismodegib	Hedgehog	Basal cell carcinoma	Roche/Curis	Market
Saridegib	Hedgehog	Chondrosarcoma	Infinity	Phase II
MK-0752	Notch	Breast cancer	Merck	Phase I/II
Adecatumumab	EPCAM	Breast cancer	Amgen	Phase II
Catumaxomab	EPCAM/CD3	Ascites, ovarian, gastric	Fresenius / Trion	Phase II/III
MT110	EPCAM/CD3	Lung, gastrointestinal	Amgen	Phase I
OMP-18R5	Wnt/Frizzled	Solid tumors	Bayer/Oncomed	Preclinical

Source: Oppenheimer & Co. Inc.

Instead, a more suitable approach is targeting the required co-receptors LRP5 and LRP6. LRP5 and LRP6 are required for breast tissue development during embryogenesis and after, where it is found on cells that express mammary stem cell markers. In humans, increased Lrp6 expression is associated with basal-like breast cancer.

FAK and EMT

In the context of epithelial cancer, EMT provides a mechanism for tumor cells to leave the primary tumor, setting the stage for metastatic spread. Full detachment from the basal surface leads to anoikis, a form of apoptosis induced by cell detachment from the extracellular matrix (ECM). Cells are attached to the ECM via transmembrane proteins called integrins, which, in turn, cause cytoskeleton rearrangements as part of cell migration through the focal adhesion kinase (FAK). Importantly, mammary stem cells (MaSCs) and mammary cancer stem cells (MaCSCs) detach from the ECM and form mammospheres in serum-free suspension culture without undergoing apoptosis. Expression of constitutively activated FAK has been shown to render cells resistant to anoikis. In addition, specifically ablating FAK mammary stem cells during early embryonic stage reduced the pool of cancer stem/progenitor cells in primary tumors, decreased their self-renewal and migration, and compromised their tumorigenicity and maintenance.

FAK and Cell Migration

Cell migration requires coordinated and dynamic regulation of the integrin-mediated focal adhesions and the cytoskeleton networks they connect with. FAK is the key protein in the regulation of focal adhesion dynamics as cells devoid of FAK exhibit impaired migration. FAK has also been shown to promote invasion of breast cancer cells through an endothelial monolayer. Upon activation by integrin-mediated cell adhesion, FAK undergoes autophosphorylation at Y397, which serves as a binding site for Src and PI3K.

Importantly, expression of FAK and phosphorylation of Y397, but not necessarily by FAK itself, is required for EGF-and PDGF-stimulated cell motility, suggesting an important role of FAK protein scaffolding function in mediating its regulation in cell migration. The role of FAK in promoting cell migration is beyond its association with two major signaling molecules Src and PI3K.

Of relevance to breast cancer cell migration, breast cancer cells express the chemokine receptor CXCR4, which binds the chemokine CXCL12 (also known as stromal cell differentiation factor 1, SDF-1). CXCL12 is found abundantly in the bone marrow, where it promotes the growth of hematopoietic stem cells. Circulating breast CSCs migrate to the CXCL12-rich bone marrow, where they establish bone metastases. In fact, bone is the most common site for breast cancer metastasis. Importantly, CXCR4-mediated chemotaxis is dependent on FAK. However, targeting CXCR4 to prevent breast cancer metastasis is not practical, since plerixafor (Mozobil), a CXCR4 antagonist, mobilizes hematopoietic stem cells, and is indicated for peripheral blood stem cell mobilization.

Inhibition of FAK

Several orally bioavailable ATP-competitive FAK inhibitors have been developed and have entered early clinical testing (Exhibit 10). Several small-molecule tyrosine kinase inhibitors such as dasatinib or bosutinib, which were originally developed as bcr/abl inhibitors for the treatment of CML, have been found to block additional targets including src and FAK. Phase I and II studies with these compounds have been conducted in patients with solid tumors, and stable disease for prolonged periods has been described in subgroups of patients. Whether or not inhibition of FAK contributed to treatment outcome remains to be determined.

Exhibit 10: Select Anti-Cancer Agents Targeting FAK

Agent	Type of cancer	Sponsor	Stage
PF00562271	Head and neck, prostate, pancreas	Pfizer	Phase I
PF-04554878	Advanced non-hem. cancers	Pfizer	Phase I
GSK2256098	Solid tumor	GSK	Phase I

Source: Oppenheimer & Co. Inc.

Management Team

Exhibit 11: Verastem Leadership

Personnel	Position	Description
Christoph Westphal, M.D., Ph.D.	Chairman and CEO	Dr. Westphal is a partner at Longwood Founders Fund, and he was a former founder and CEO of Sirtris Pharmaceuticals acquired by GlaxoSmithKline, Alnylam Pharmaceuticals, and Momenta Pharmaceuticals. He is also a cofounder of Alnara Pharmaceuticals acquired by Eli Lilly and Ovascience. He holds an M.D. from Harvard Medical School, Ph.D. in genetics from Harvard University, and a B.A. from Columbia University.
Robert Forrester, LL.B.	Chief Operating Officer	Mr. Forrester has 10 years of experience as CEO, COO or CFO of private and public life science companies with Forma Therapeutics, CombinatoRx, and Coley acquired by Pfizer. He was a former managing director of the Proprietary Investment Group at MeesPierson, part of the Fortis Group, and previously worked in corporate finance investment banking groups at BZW (now Barclays Capital) and UBS. He holds a LL.B. from Bristol University.
Jonathan Pachter, Ph.D.	Head of Research	Dr. Pachter has over 20 years of experience in leading discovery of small molecule and monoclonal antibody therapeutics for cancer. Most recently, he was former Head of Cancer Biology at OSI Pharmaceuticals acquired by Astellas Pharma in 2010. Dr. Pachter completed postdoctoral work in Pharmacology at Yale University School of Medicine and holds a Ph.D. from Baylor College of Medicine.
Richard Aldrich	Board of Directors	Mr. Aldrich is a founder and partner of Longwood Founders Fund. He cofounded and helped build several successful biotech companies including Sirtris Pharmaceuticals, Concert Pharmaceuticals, and Alnara Pharmaceuticals acquired by Eli Lilly. Previously, he was a general partner of RA Capital and Chief Business Officer of Vertex Pharmaceuticals; and held management positions at Biogen Inc.
Henri Termeer	Board of Directors	Mr. Termeer is the former CEO and chairman of the board of Genzyme Corporation acquired by Sanofi in 2011.
Robert Weinberg, Ph.D.	Co-Chair Scientific Advisory Board	Dr. Weinberg is a founding member of the Whitehead Institute and the Daniel K. Ludwig Professor for Cancer Research in the Department of Biology at MIT. He is an internationally recognized authority on the genetic basis of human cancer development. Dr. Weinberg is a member of the National Academy of Sciences, the Institute of Medicine and a Fellow of the American Academy of Arts and Sciences. He holds a Ph.D. in biology from MIT.
Eric Lander, Ph.D.	Co-Chair Scientific Advisory Board	Dr. Lander is founding director of the Broad Institute and director of its Genome Biology Program. As a principal leader of the Human Genome Project, Dr. Lander and colleagues are using these findings to explore the molecular mechanisms underlying the basis of human disease. He is a professor of biology at MIT and professor of systems biology at Harvard Medical School. In 1990 he founded the Whitehead Institute/MIT Center for Genome Research, which became part of the Broad Institute in 2003. Dr. Lander holds a B.A. in mathematics from Princeton University and a Ph.D. in mathematics from Oxford University as a Rhodes Scholar.
Piyush Gupta, Ph.D.	Co-Chair Scientific Advisory Board	Dr. Gupta is Assistant Professor at the Whitehead Institute. He is an expert in the application of genomic methods to cancer and has made many contributions to the understanding of cancer metastasis and cancer stem cells. Dr. Gupta holds a B.S. in mathematics from the University of Chicago and a Ph.D. in biology at MIT. Dr. Gupta has done doctoral research with Dr. Robert Weinberg and post-doctoral research with Dr. Eric Lander in cancer biology. Dr. Gupta has developed methods that enable the systematic identification of compounds that selectively kill cancer stem cells. His work has led to the identification of novel anti-cancer stem cell chemicals of potential therapeutic utility.

Source: Verastem, Inc.

Stock prices of other companies mentioned in this report (as of March 6, 2012):

Alnylam (ALNY-NASD, $12.65, Not Rated)
Bayer (BAYRY-OTC, $69.35, Not Rated)
Fresenius (FMS-NYSE, $69.70, Not Rated)
GlaxoSmithKline (GSK-NYSE, $44.77, Not Rated)
Infinity Pharmaceuticals (INFI-NASD, $8.01, Not Rated)
Merck (MRK-NYSE, $38.49, Not Rated)
Pfizer (PFE-NYSE, $21.49, Not Rated)
Poniard (PARD-OTC, $1.02, Not Rated)
Roche (RHHBY-OTC, $43.55, Not Rated)

Verastem Income Statement 2010A-2016E

Amounts in thousands, except per-share figures

		2011					2012								
	2010A	1QA	2QA	3QA	4QE	2011E	1QE	2QE	3QE	4QE	2012E	2013E	2014E	2015E	2016E
Revenues:															
TNBC royalty	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other revenue	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total operating revenue	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**
Operating expenses:															
Cost of goods	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Research & development	400	1,200	2,000	2,283	2,500	7,983	2,575	2,650	2,725	5,300	13,250	21,750	26,250	25,750	25,750
Selling, general & administrative	384	500	700	995	1,010	3,205	1,075	1,150	1,200	1,250	4,675	6,311	7,889	8,678	9,546
Total operating expenses	**784**	**1,700**	**2,700**	**3,278**	**3,510**	**11,188**	**3,650**	**3,800**	**3,925**	**6,550**	**17,925**	**28,061**	**34,139**	**34,428**	**35,296**
Income (Loss) from operations	**(784)**	**(1,700)**	**(2,700)**	**(3,278)**	**(3,510)**	**(11,188)**	**(3,650)**	**(3,800)**	**(3,925)**	**(6,550)**	**(17,925)**	**(28,061)**	**(34,139)**	**(34,428)**	**(35,296)**
Other income (expense)	(2)	(5)	(6)	(7)	(7)	(25)	(4)	5	3	1	5	13	123	19	195
Pretax income (loss)	**(786)**	**(1,705)**	**(2,706)**	**(3,285)**	**(3,517)**	**(11,213)**	**(3,654)**	**(3,795)**	**(3,922)**	**(6,549)**	**(17,920)**	**(28,048)**	**(34,016)**	**(34,409)**	**(35,101)**
Income tax provision (benefit)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net income (loss)	**(786)**	**(1,705)**	**(2,706)**	**(3,285)**	**(3,517)**	**(11,213)**	**(3,654)**	**(3,795)**	**(3,922)**	**(6,549)**	**(17,920)**	**(28,048)**	**(34,016)**	**(34,409)**	**(35,101)**
Basic & diluted net loss per share	**($0.59)**	**($0.29)**	**($0.46)**	**($0.56)**	**($0.24)**	**($1.39)**	**($0.17)**	**($0.18)**	**($0.19)**	**($0.31)**	**($0.85)**	**($1.31)**	**($1.56)**	**($1.55)**	**($1.23)**
Basic and diluted common shares outstanding	1,325	5,850	5,850	5,850	14,734	8,071	21,059	21,109	21,159	21,209	21,134	21,484	21,834	22,184	28,534

Source: Company documents and Oppenheimer & Co. Inc.

Verastem Abbreviated Balance Sheet 2010A-2016E

Amounts in thousands, except per-share figures

	2010A	2011E	2012E	2013E	2014E	2015E	2016E
Cash and cash equivalents	3,584	58,307	100,213	73,007	40,356	7,324	63,635
PPE	8	899	1,258	2,139	2,781	3,337	3,671
Other Assets	12	280	980	3,626	9,065	14,504	17,405
Total Assets	**3,604**	**59,486**	**102,451**	**78,772**	**52,202**	**25,165**	**84,711**
Accounts Payable	279	1,558	1,869	1,962	2,061	2,164	2,272
Accrued Expenses	89	953	1,619	3,724	6,704	8,044	9,653
Other Liabilities	-	146	512	1,894	4,545	7,727	9,273
Total Liabilities	**368**	**2,656**	**4,000**	**7,581**	**13,310**	**17,935**	**21,198**
Total Shareholders Equity	**3,236**	**56,830**	**98,451**	**71,191**	**38,892**	**7,230**	**63,513**

Source: Company documents and Oppenheimer & Co. Inc.

Investment Thesis

Based on a unique discovery platform, VSTM is developing cancer stem cell (CSC)-targeted therapies. We believe there is growing evidence CSCs have a key role in tumor initiation/metastasis. VSTM expects to advance Wnt pathway inhibitor VS-507 and a FAK inhibitor (VS-4718/VS-5095) into ph.I by 1Q13. Based on preclinical results and compelling biological rationale, we believe these compounds have broad anticancer potential. VSTM first plans to pursue triple negative breast cancer (TNBC), which we believe is a substantially underserved, multi-billion dollar market. We believe VSTM is an attractive long-term investment and expect the stock to appreciate as clinical progress and high profile scientific publications further underscore the importance of CSCs.

Price Target Calculation

Our price target of $16 is based on a forward DCF valuation, which includes probability-adjusted product royalties and milestones in triple negative breast cancer. We model a ~58% EBIT margin and ~30% tax rate in the out years, resulting a 2027 free cash flow of $93M and terminal value of cash flow of $805M. We utilize a 15% discount rate to reflect the early stage of VSTM's drug candidates, but utilize a 3% terminal growth rate to reflect the productivity of the company's discovery platform. Our valuation also includes $200M in technology value, which we believe is conservative based on platform acquisition comps.

Key Risks to Price Target

Key risks to our price target include, but are not limited to, clinical failure of VSTM's drug candidates, success of competing drugs in the clinic, and failure to secure funding for operations. There is liquidity risk with the shares, based on the company's float.

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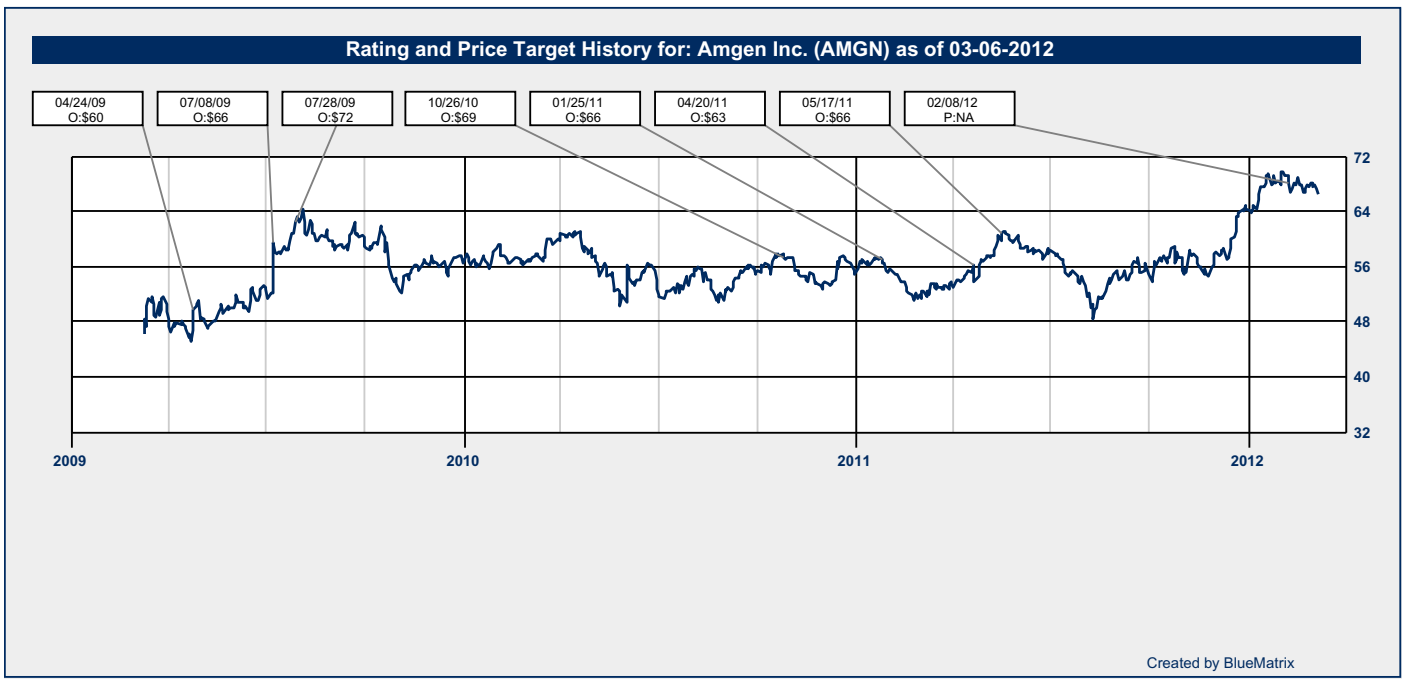
Stock Prices as of March 7, 2012

Amgen Inc. (AMGN - Nasdaq, 66.54, PERFORM)
Curis Inc. (CRIS - Nasdaq, 4.24, OUTPERFORM)


Rating and Price Target History for: Amgen Inc. (AMGN) as of 03-06-2012
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07/08/09 O:$66
07/28/09 O:$72
10/26/10 O:$69
01/25/11 O:$66
04/20/11 O:$63
05/17/11 O:$66
02/08/12 P:NA
72
64
56
48
40
32
2009
2010
2011
2012
Created by BlueMatrix


Rating and Price Target History for: Curis Inc. (CRIS) as of 03-06-2012
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01/30/12 O:$7.5
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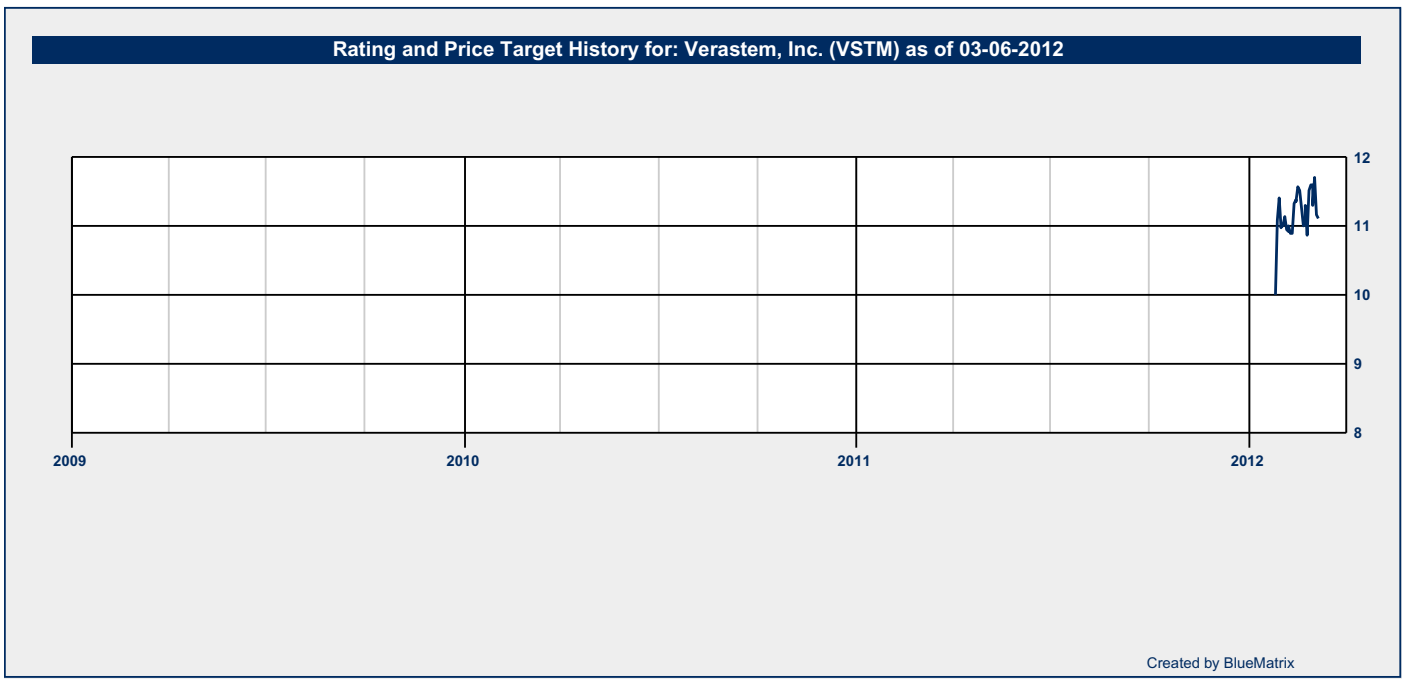
5
4
3
2
1
0
2009
2010
2011
2012
Created by BlueMatrix



All price targets displayed in the chart above are for a 12- to- 18-month period. Prior to March 30, 2004, Oppenheimer & Co. Inc. used 6-, 12-, 12- to 18-, and 12- to 24-month price targets and ranges. For more information about target price histories, please write to Oppenheimer & Co. Inc., 85 Broad Street, New York, NY 10004, Attention: Equity Research Department, Business Manager.

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			IB Serv/Past 12 Mos.	
Rating	Count	Percent	Count	Percent
OUTPERFORM [O]	334	56.10	145	43.41
PERFORM [P]	254	42.70	85	33.46
UNDERPERFORM [U]	7	1.20	3	42.86

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