Rodman &Renshaw®

Verastem, Inc. (VSTM)

COMPANY UPDATE

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LIFE SCIENCES

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Market Outperform / Speculative Risk

VSTM FY1Q12 Earnings; Board Additions Bolster Credentials in the CSC Drug Space

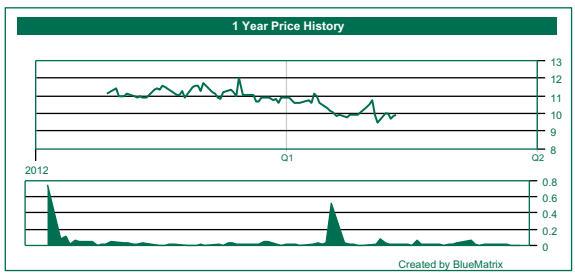
MARKET DATA	5/14/2012
Price	$9.53
Exchange	NASDAQ
Target Price	$19.00
52 Wk Hi - Low	$12.24 - $8.89
Market Cap(MM)	$200.7
EV(MM)	$221.0
Shares Out (MM)	21.1
Public Mkt Float (MM)	21.1
Avg. Daily Vol	26,431
Short Interest	365,618

BALANCE SHEET METRICS	
Cash (MM)	$47.8
LTD (MM)	$0.0
Total Debt/Capital	NA
Cash/Share	$30.67
Book Value(MM)	NA
Book Value/Share	$(8.19)

Cash (MM): Includes cash and equivalents, short-term and long-term securities.

EARNINGS DATA ($)			
FY - Dec	**2011E**	**2012E**	**2013E**
Q1 (Mar)	--	(0.47)*A*	--
Q2 (Jun)	--	(0.49)	--
Q3 (Sep)	--	(0.52)	--
Q4 (Dec)	(4.01)	(0.54)	--
Full Year EPS	(10.59)	(2.04)	(1.64)

INDICES	
DJIA	12,695.4
SP-500	1,338.4
NASDAQ	2,590.3
NBI	1,310.8



Verastem, Inc. (VSTM) reported FY1Q12 earnings before market open this morning, recording EPS of ($0.47) on $0mm revenues, greater than both our ($0.18) EPS estimate and the Street consensus of ($0.28). We remind investors that, as an early stage biotech, VSTM share price is driven mainly by its ability to execute against milestones with its pipeline candidates rather than earnings estimates. The Company reported operating expenses of $6.93mm, higher than our estimate of $3.75mm and the Street consensus of $4.50mm. Specifically, R&D spending of $4.80mm was substantially higher than our $2.50mm estimate, as was actual G&A expense ($2.13mm vs. $1.25mm). VSTM recorded a weighted average of 14.69mm shares outstanding and finished 1Q12 with $109.3mm cash, equivalents, short- and long-term investments. A comparison of 1Q12 results vs. our estimates appears on Table 1.

Incremental updates since FY2011. Unsurprisingly, little new news was provided in today's release. The next milestone for VSTM - the results of an ongoing IND tox assessment of the Wnt inhibitor VS-507 - is not anticipated until early 2013. That said, VSTM noted incremental updates to its development programs and advisory board composition. Preclinical data presentations on the Wnt VS-507 program, the FAK VS-4718/5095 program, and a cancer stem cell biomarker discovery project were presented at AACR in early April (please see our VSTM note from April 3rd). Further backfilling its pipeline portfolio, VSTM announced the acquisition of a novel mTOR/PI3K program from S*Bio, which builds on a 300,000-compound in-licensing deal with the Broad Institute announced earlier in the quarter. VSTM also announced the naming of Henri Termeer, former Genzyme Chair and CEO, as Lead Director of its Board of Directors, as well as the addition of Dr. Max Wicha, Distinguished Professor of Oncology and Director of the University of Michigan Comprehensive Cancer Center, to its scientific advisory board. We view both of these arrivals has high value additions to the company's advisory team with the ability to bolster the company's competitive advantage in the cancer stem cell therapeutic space as the field matures. Details on the S*Bio acquisition and Dr. Wicha's accomplishments are provided on Page 2.

Changes to our model. We have made a number of changes to our model in light of today's reporting. 1Q12 operating expenses, shares outstanding and EPS have been updated to reflect actual results. R&D and G&A expense have been raised for 2Q-3Q12 to reflect positive QoQ growth for the remainder of the year. Full year R&D expense for 2013 through 2015 has also been raised to maintain previous assumptions for YoY growth rates. Finally, share count estimates have been lowered going forward to be brought in-line with the current actual weighted average of shares outstanding. A summary of these changes appears on Table 3.

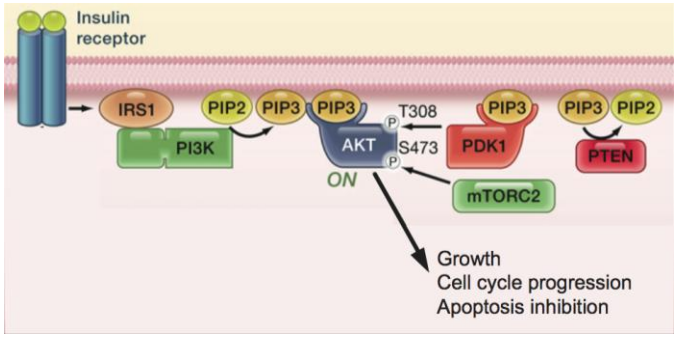
We maintain our Market Outperform rating for VSTM shares and $19 price target. We derive our price target through a combination of methodologies, including a DCF-based valuation of $17 and a CAGR valuation of $22. Risks to our valuation include the risk of clinical failure with VS-507 and/or VS-4718/5095 for reasons of safety or inactivity. In our view, the value of VSTM shares will be primarily milestone-driven over the next six to twelve months, as VS-507 completes IND tox assessment.

Additional Thoughts on the S*Bio PI3K/mTOR acquisition by VSTM.

Specifics of the deal. On May 11, 2012 VSTM completed its acquisition of S*Bio's preclinical stage dual PI3K/mTOR inhibitor program, and related patent rights, for an upfront fee of $350,000, with the potential for up to $21mm in development and regulatory milestone payments, along with a low to mid single digit royalty on net sales. Based on available information from the S*Bio website, we assume the company has acquired the SB2343 PI3K/mTOR program, a asset composed of a library of ~500 compounds, however the exact number of compounds in VSTM acquisition have not been disclosed. Prior data presentations of SB2343 at an AACR conference in 2011 describe the molecule class as novel tetra-substituted purine with favorable pharmacokinetic and pharmacodynamic properties, along with oral bioavailability.

Backgrounder on PI3K/mTOR signaling in Cancer. The mammalian target of rapamycin, or mTOR, is a serine/threonine kinase that integrates various signaling inputs to exert control over protein and lipid biosynthesis as well as growth factor driven cell cycle progression. mTOR activity is closely tied to the PI3K/Akt pathway, which is a key regulator of cell proliferation, survival and growth. Growth factor activation of receptor tyrosine kinases IGF1R, MET or EGFR stimulate phosphoinositol-3 kinase (PI3K) activity, which induces the accumulation of the lipid second-messenger molecule PIP3 in the plasma membrane. This in turn activates phosphoinositide dependent protein kinase-1 (PDK1) and facilitates the recruitment of the AGC kinase AKT to the plasma membrane via a plekstrin homology (PH) domain. Secondary phosphorylating actions by PDK1 and mTORC2 fully activate AKT, which in turn phosphorylates a number of effectors to inhibit apoptosis and promote cell cycle progression (Figure 1).

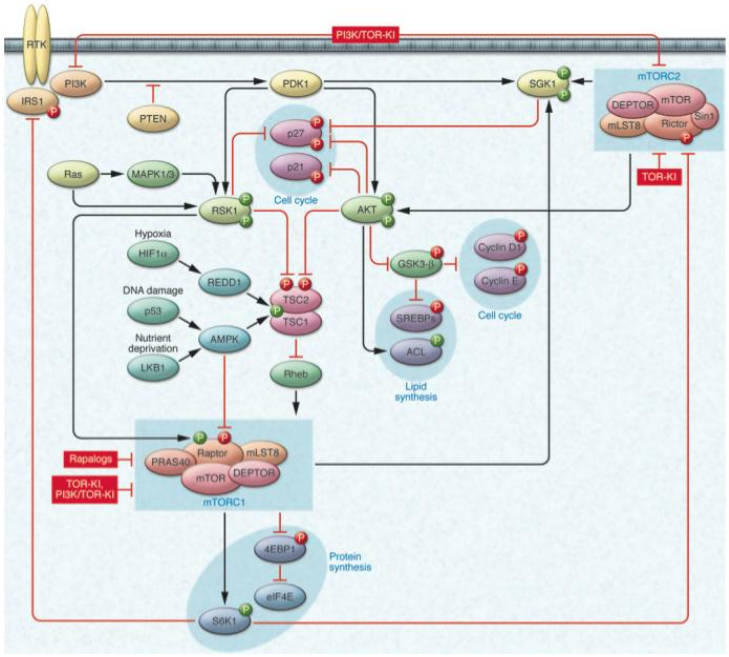
Figure 1. AKT Activation Pathway



Source: Manning BD, Cell, 2010

mTORC1 is a major effector of AKT activation. In addition to PI3K/AKT signaling, mTORC1 integrates many different signaling inputs, including growth factor signaling, DNA damage checkpoint activation and reaction to hypoxic stress, to regulate protein biosynthesis. mTORC1 achieves this by activating ribosomal biogenesis by phosphorylating ribosomal S6 kinase 1 (S6K1) and promoting protein translation via inhibitory phosphorylation of the eukaryotic initiating factor 4E-binding protein 1(4EBP1) (Figure 2). mTORC1 also activates serine/glucocorticoid kinase 1 (SGK1), a negative regulator of p21 and p27 cell cycle inhibitory proteins, to promote cell cycle progression. Interestingly, mTORC2 is a negatively regulated substrate of mTORC1. mTORC1 activation is thus governed by a negative feedback loop in which mTROC2-dependent activation of AKT, and in turn mTORC1, is attenuated by mTORC1-mediated inactivation of mTORC2.

Figure 2. Overview of the mTOR Signaling Network



Source: Wander SA, Hennessy BT, Slingerland JM, Journal of Clinical Investigation, 2011.

Unsurprisingly, given its control over biosynthetic and proliferative mechanisms, the PI3K/mTOR network is frequently deregulated in cancer cells resulting from activating genetic lesions of RTKs feeding into the pathway or of pathway intermediates. *PIK3Ca* encodes the p110α catalytic subunit of PI3K and is one of most frequently mutated kinases in human cancer. PI3K activity is naturally opposed by the dephosphorylating activity of the phosphatase and tensin homolog, PTEN. *PTEN* is frequently deleted or inactivated in cancer, leaving aberrant pathway activation in its absence.

Our take. VSTM's acquisition of S*Bio's PI3K/mTOR inhibitor program is another example (like the FAK inhibitor acquisition from Poniard) of the company's commitment to exploring and exploiting the role of different signaling pathways in the maintenance and survival of cancer stems cells and doing it under favorable economic terms. While the specific importance of either PI3K, mTOR or in tamdem to the cancer stem cell phenotype have yet to concretely established, we would anticipate that inhibition of the pathway has some negative impact on stem cell fate given PI3K/AKT's stimulating effect on GSK3β (a common effector molecule with Wnt signaling). We remain cautious on the clinical prospects for this line of therapy as a single-agent however, based on the performance competing drugs like BEZ235 (Novartis, NVS, Not Rated) which achieved a best response of stable disease in ~ 25% of patients with advanced solid tumors in Phase I clinical testing.

Dr. Max Wicha Lends VSTM Formidable Bona Fides in Cancer Stem Cell Research. As mentioned above, we regard Dr. Max Wicha's membership on the VSTM scientific advisory board as welcome addition to the team. Dr. Wicha is widely held as a leading expert in cancer stem cell biology, particularly in breast cancer. As a clinician, he also carries several years of experience in the conduct of clinical trials which should prove invaluable to VSTM's development going forward. A quick look at select publications by Dr. Wicha, enclosed below, illustrates his contribution to the field.

Select publications:

PSA Lo and Behold: Prostate Cancer Stem Cells. Wicha MS. Cell Stem Cell. 2012 May 4;10(5):482-3.

Role of microRNAs in the regulation of breast cancer stem cells. Liu S, Clouthier SG, Wicha MS. J Mammary Gland Biol Neoplasia. 2012 Mar;17(1):15-21.

Antiangiogenic agents increase breast cancer stem cells via the generation of tumor hypoxia. Conley SJ, et al. PNAS. 2012 Feb 21;109(8):2784-9. Epub 2012 Jan 23.

Breast cancer stem cells, cytokine networks, and the tumor microenvironment. Korkaya H, Liu S, Wicha MS. J Clin Invest. 2011 Oct;121(10):3804-9. Review.

Regulation of cancer stem cells by cytokine networks: attacking cancer's inflammatory roots. Korkaya H, Liu S, Wicha MS. Clin Cancer Res. 2011 Oct 1;17(19):6125-9.

Breast cancer stem cells are regulated by mesenchymal stem cells through cytokine networks. Liu S, et al. Cancer Res. 2011 Jan 15;71(2):614-24.

Targeting breast cancer stem cells. McDermott SP, Wicha MS. Mol Oncol. 2010 Oct;4(5):404-19. Review.

CXCR1 blockade selectively targets human breast cancer stem cells in vitro and in xenografts. Ginestier C, et al. J Clin Invest. 2010 Feb;120(2):485-97.

Aldehyde dehydrogenase 1-positive cancer stem cells mediate metastasis and poor clinical outcome in inflammatory breast cancer. Charafe-Jauffret E, et al. Clin Cancer Res. 2010 Jan 1;16(1):45-55.

Table 1. Rodman and Consensus Estimates versus Actual Results

Verastem, Inc. (VSTM) Forecasted Results vs. Actual ($MM)	R&R Estimates	Consensus	Actual	Variance
Total Revneues	0.00	0.00	0.00	0.00
R&D Expense	2.50	3.10	4.80	2.30
SG&A Expense	1.25	1.30	2.13	0.88
Operating Expenses	3.75	4.40	6.93	3.18
Operating Income (loss)	(3.75)	(4.50)	(6.93)	(3.18)
Other Income	0.00		0.00	0.00
Net Income (loss)	(3.75)	(4.50)	(6.93)	(3.18)
Accretion of preferred stock	0.00		(0.01)	(0.01)
Net Income Spplicable to Shareholders	(3.75)	(4.30)	(6.93)	(3.18)
Basic Shares Outstanding	21.06		14.69	(6.37)
Diluted Shares Outstanding	21.06		14.69	(6.37)
Basic EPS	(0.18)	(0.28)	(0.47)	(0.29)
Diluted EPS	(0.18)	(0.28)	(0.47)	(0.29)

Table 2. Upcoming Milestones

Milestone	Date
• Peer-review scientific journal publication	2H 2012
• IND filing of VS-507 with FDA and initiation of Phase I trial	2H 2012
• IND filing of VS-4718/VS-5095 with the FDA and initiation of Phase I trial	1H 2013

Table 3. Changes to Our Model

Verastem, Inc (VSTM) New vs. Old Income Statement ($MM)	Old 2Q12 FY 2012E	New 2Q12 FY2012E	Old 3Q12 FY2012E	New 3Q12 FY2012E	Old 4Q12 FY2012E	New 4Q12 FY2012E	Old 2012 FY2012E	New 2012 FY2012E	Old 2013 FY2013E	New 2013 FY2013E	Old 2014 FY2014E	New 2014 FY2014E	Old 2015 FY2015	New 2015 FY2015
Total Revenues	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
R&D Expense	2.75	5.10	3.25	5.40	3.70	5.70	12.20	21.00	15.66	23.00	25.84	37.95	62.01	91.08
SG&A Expense	1.30	2.25	1.35	2.40	1.40	2.60	5.30	9.38	6.25	10.00	8.00	12.00	15.95	15.95
Operating Expenses	4.05	7.35	4.60	7.80	5.10	8.30	17.50	30.38	21.91	33.00	33.84	49.95	77.96	107.03
Operating Income (loss)	(4.05)	(7.35)	(4.60)	(7.80)	(5.10)	(8.30)	(17.50)	(30.38)	(21.91)	(33.00)	(33.84)	(49.95)	(77.96)	(107.03)
Other Income	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Net Income	(4.05)	(7.35)	(4.60)	(7.80)	(5.10)	(8.30)	(17.50)	(30.38)	(21.91)	(33.00)	(33.84)	(49.95)	(77.96)	(107.03)
Basic Shares Outstanding	21.37	14.91	21.70	15.14	22.02	15.36	21.38	14.92	26.94	20.08	31.21	24.28	31.52	24.52
Dilute Shares Outstanding	21.37	14.91	21.70	15.14	22.02	15.36	21.38	14.92	26.94	20.08	31.21	24.28	31.52	24.52
Basic EPS (GAAP)	(0.19)	(0.49)	(0.21)	(0.52)	(0.23)	(0.54)	(0.82)	(2.04)	(0.81)	(1.64)	(1.08)	(2.06)	(2.47)	(4.36)
Diluted EPS (GAAP)	(0.19)	(0.49)	(0.21)	(0.52)	(0.23)	(0.54)	(0.82)	(2.04)	(0.81)	(1.64)	(1.08)	(2.06)	(2.47)	(4.36)

Table 4. Updated Income Statement

Verastem, Inc. (VSTM)										
Income Statement ($MM)	**2010A**	**2011E**	**1Q12A**	**2Q12E**	**3Q12E**	**4Q12E**	**2012E**	**2013E**	**2014E**	**2015E**
Product Sales and Royalties										
VS-507, US Sales										0.0
VS-507, Ex-US Royalties										0.0
FAK Inhibitor, US Sales										0.0
Fak Inhibitors, Ex-US Royalties										0.0
Total Product Sales and Royalties										**0.0**
License and Milestone Revenue										
Collaboration Revenue										
Total Revenue										
Cost of Goods Sold										
Gross Profit										0.0
Operating expenses:										
R&D	0.4	9.9	4.8	5.1	5.4	5.7	21.0	23.0	38.0	91.1
SG&A	0.4	3.8	2.1	2.3	2.4	2.6	9.4	10.0	12.0	15.9
Total operating expenses	**0.8**	**13.7**	**6.9**	**7.4**	**7.8**	**8.3**	**30.4**	**33.0**	**50.0**	**107.0**
		0.0								
Operating income (loss)	(0.8)	(13.7)	(6.9)	(7.4)	(7.8)	(8.3)	(30.4)	(33.0)	(50.0)	(107.0)
Other income (expense)										
Interest income		0.0								
Interest (expense)										
Total other income	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Pretax Income	(0.8)	(13.7)	(6.9)	(7.4)	(7.8)	(8.3)	(16.1)	(33.0)	(50.0)	(107.0)
Provision for Income Tax										
Effective Tax Rate										
Net income	(0.8)	(13.7)	(6.93)	(7.35)	(7.80)	(8.30)	(16.1)	(33.0)	(50.0)	(107.0)
Accretion of preferred stock	(0.0)	(0.0)	(0.0)							
Net income applicable to common shareholders	(0.8)	(13.7)	(6.9)	(7.4)	(7.8)	(8.3)	(30.4)	(33.0)	(50.0)	(107.0)
Basic EPS to common shareholders	$ (0.92)	$ (10.59)	$ (0.47)	$ (0.49)	$ (0.52)	$ (0.54)	$ (1.08)	$ (1.64)	$ (2.06)	$ (4.36)
Diluted EPS to common shareholders	$ (0.92)	$ (10.59)	$ (0.47)	$ (0.49)	$ (0.52)	$ (0.54)	$ (1.08)	$ (1.64)	$ (2.06)	$ (4.36)
Basic Shares Outstanding	0.850	1.295	14.693	14.913	15.137	15.364	14.915	20.080	24.281	24.524
Diluted Shares Outstanding	0.850	1.295	14.693	14.913	15.137	15.364	14.915	20.080	24.281	24.524
% change in diluted shares outstanding				*1.5%*	*1.5%*	*1.5%*	1052%	*6.1%*	*1.0%*	*1.0%*

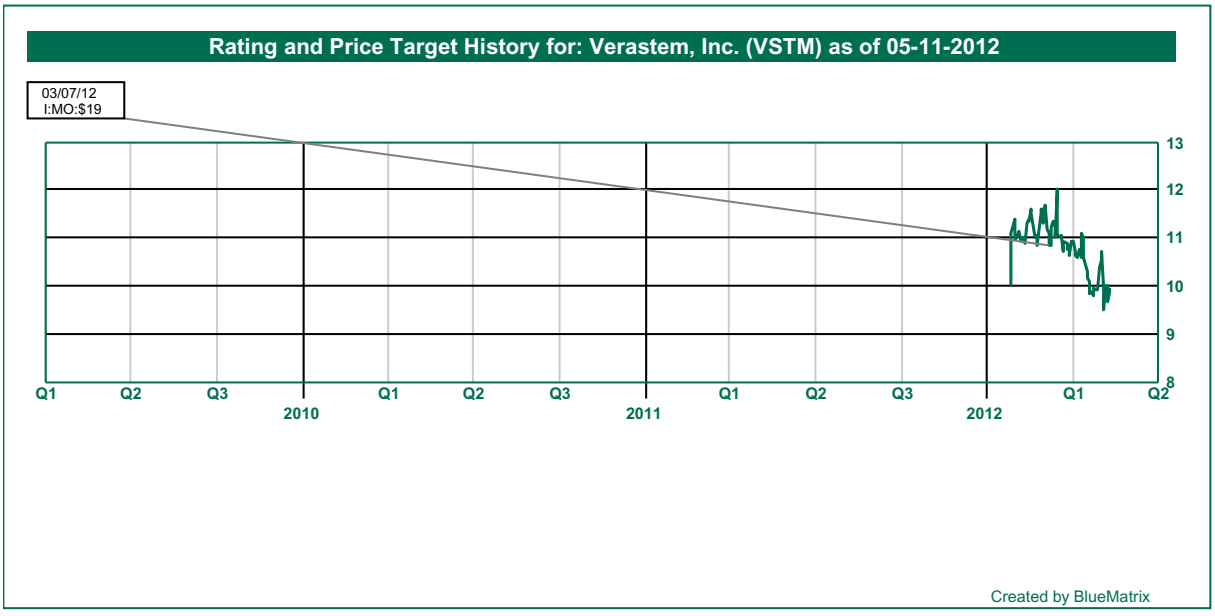
RODMAN & RENSHAW RATING SYSTEM: Rodman & Renshaw employs a three tier rating system for evaluating both the potential return and risk associated with owning common equity shares of rated firms. The expected return of any given equity is measured on a RELATIVE basis of other companies in the same sector, as defined by First Call. The price objective is calculated to estimate the potential movement in price a given equity could achieve given certain targets are met over a defined time horizon. Price objectives are subject to exogenous factors including industry events and market volatility. The risk assessment evaluates the company specific risk and accounts for the following factors, maturity of market, maturity of technology, maturity of firm, cash utilization, and valuation considerations. Potential factors contributing to risk: relatively undefined market, new technologies, immature firm, high cash burn rates, intrinsic value weighted toward future earnings or events.

RETURN ASSESSMENT

- Market Outperform (Buy): The common stock of the company is expected to outperform a passive index comprised of all the common stock of companies within the same sector, as defined by First Call.
- Market Perform (Hold): The common stock of the company is expected to mimic the performance of a passive index comprised of all the common stock of companies within the same sector, as defined by First Call.
- Market Underperform (Sell): The common stock of the company is expected to underperform a passive index comprised of all the common stock of companies within the same sector, as defined by First Call.

RISK ASSESSMENT

- Speculative - The common stock risk level is significantly greater than market risk. The stock price of these equities is exceptionally volatile.
- Aggressive - The common stock risk level is materially higher than market level risk. The stock price is typically more volatile than the general market.
- Moderate - The common stock is moderately risky, or equivalent to stock market risk. The stock price volatility is typically in-line with movements in the general market.



RATING SUMMARY

Distribution of Ratings Table

			IB Serv./Past 12 Mos	
Rating	**Count**	**Percent**	**Count**	**Percent**
Market Outperform(MO)	73	62.39%	10	13.70%
Market Perform(MP)	28	23.93%	3	10.71%
Market Underperform(MU)	6	5.13%	0	0.00%
Under Review(UR)	10	8.55%	3	30.00%
Total	117	100%	16	100%

Investment Banking Services include, but are not limited to, acting as a manager/co-manager in the underwriting or placement of securities, acting as financial advisor, and/or providing corporate finance or capital markets-related services to a company or one of its

affiliates or subsidiaries within the past 12 months.

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