AEGIS CAPITAL CORP

Biotechnology
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Company Update

March 18, 2013

Stemline Therapeutics, Inc.

Rating: Buy

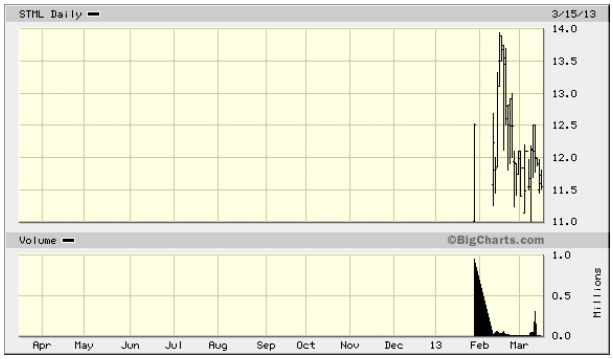
Cracking The Cancer Stem Cell Conundrum

Key Metrics

STML - NASDAQ	$11.55
Pricing Date	Mar 15 2013
Price Target	$35.00
52-Week Range	$13.95 - $10.33
Shares Outstanding (mm)	7.0
Market Capitalization ($mm)	$80.9
3-Mo Average Daily Volume	79,379
Institutional Ownership	NM
Debt/Total Capital	NM
ROE	NM
Book Value/Share	$(0.44)
Price/Book	(26.3)x
Dividend Yield	NM
LTM EBITDA Margin	NM

EPS ($) FY: December

	2012E	Prior 2013E	Curr. 2013E	Prior 2014E	Curr. 2014E
1Q-Mar	(0.39)E	--	(0.42)E	--	(0.50)E
2Q-Jun	(0.50)E	--	(0.36)E	--	(0.47)E
3Q-Sep	(0.55)E	--	(0.40)E	--	(0.46)E
4Q-Dec	(0.60)E	--	(0.45)E	--	(0.51)E
FY	(2.04)E	--	(1.61)E	--	(1.94)E
P/E	NM		NM		NM



Company Description:
Stemline Therapeutics, Inc. (http://www.stemline.com/) is a biotechnology firm headquartered in New York, NY.

Investment Highlights:

- **Initiating Coverage.** We are starting coverage of Stemline Therapeutics, Inc., an emerging biotechnology company focusing on the development of novel anti-cancer therapeutics, with a Buy rating and an 18-month price target of $35.00 per share. In our view, Stemline Therapeutics represents a unique investment opportunity in the small-cap oncology arena, due to its proprietary knowledge base in the domain of cancer stem cells (CSCs), its rapidly advancing clinical-stage pipeline, and its extremely capital-efficient business model. As such, we consider the firm an undervalued entity and would recommend that investors focusing on the oncology sector consider this a key long-term holding. We expect the company to have a catalyst-rich next 18 months, and note that this was the first biotech IPO of 2013.
- **Risk-Mitigated Clinical-Stage Pipeline.** One of the most appealing aspects of the Stemline story is the fact that the company's lead drug candidate, SL-401, is risk-mitigated from a regulatory as well as a clinical perspective. The drug is a fusion protein that utilizes the interleukin-3 (IL-3) protein linked to an exotoxin from the bacterium that causes the infectious disease diphtheria. The drug is internalized into cells expressing high levels of the IL-3 receptor and once inside, the diphtheria toxin kills the cells. Since many cancer cells expressed in hematological malignancies express the IL-3 receptor, this drug selectively ablates both tumor bulk and CSCs without injuring healthy tissue. Stemline's second candidate, SL-701, is a peptide vaccine aimed at brain cancers. Both SL-401 and SL-701 have demonstrated strong survival-enhancing effects in their respective target indications.
- **Accelerated Path To Market.** SL-401 appears to have found a niche in an ultra-rare disease condition called blastic plasmacytoid dendritic cell neoplasm (BPDCN), which only afflicts 2,000 people a year. We believe that the firm could obtain approval for SL-401 on the basis of a single, randomized, open-label study enrolling 40 patients. As such, the drug could be on the market in late 2015.
- **Capital-Efficient Operating Model; Attractive Valuation.** We note that Stemline currently trades at an enterprise value of roughly $60mm, whereas other companies with mid- to late-stage oncology assets typically trade in the $200mm - $500mm range. The firm was founded in 2003 and to date has spent under $20mm.

The Disclosure section may be found on page 49 of this report.

Investment Thesis

Stemline Therapeutics, Inc. (STML/NASDAQ) is an emerging biotechnology company focusing on the development of novel anti-cancer therapeutics. Founded in 2003, the company went public as the first official biotechnology Initial Public Offering (IPO) of 2013. The offering was underwritten by Aegis Capital Corp., which was the sole bookrunner, and co-managers Feltl & Co. and Sunrise Securities. Stemline Therapeutics raised over $38 million in gross proceeds in this offering, which included substantial participation by major healthcare-focused funds, who are committed to the long-term potential of the company. In our view, Stemline is a highly differentiated firm in the cancer therapeutics sector because it has a lead program – SL-401 – that may benefit from an accelerated path to market in an ultra-orphan hematological malignancy, blastic plasmacytoid dendritic cell neoplasm (BPDCN), and also has a rapidly advancing technology platform that is focused on identifying promising candidate molecules that specifically target cancer stem cells (CSCs). We believe that Stemline could potentially achieve market entry with SL-401 in BPDCN in late 2014 / early 2015, while its second pipeline candidate, SL-701, could potentially receive licensure in another rare cancer type – pediatric glioma – in the late 2016 / early 2017 time frame. We also consider Stemline a very efficiently-managed firm, which managed to conduct operations and advance its pipeline from 2003 to 2012 on a total of roughly $15 million in capital raised through common stock financings as a privately-held entity.

We are initiating coverage of STML with a Buy rating and an 18-month price target of $35.00 per share, which assumes a total firm value of roughly $420mm based on a composite discounted cash flow analysis and about 12mm shares outstanding (fully-diluted) as of mid-2014. In our view, Stemline Therapeutics is one of the most attractive development-stage oncology-focused investment opportunities currently available to healthcare-focused investors, given its potential for an accelerated path to market for its lead candidate, SL-401, and its diversified early-stage pipeline targeting CSCs. Based on a comparables-based valuation approach, we establish a projected firm value of $420mm in mid-2014. This also translates into a price target of roughly $35.00 per share, assuming roughly 12mm fully-diluted shares outstanding at end-2013. We believe an investment in Stemline Therapeutics shares may entail above-average risk and volatility.

Investment Positives

Accelerated Approval Pathway. Unlike many other biotechnology firms, which typically must take a tortuous and lengthy path towards commercialization, Stemline Therapeutics may be able to benefit from an accelerated pathway to market for its lead drug candidate, SL-401. This is because the drug has been shown to exert interesting clinical impact in an ultra-rare blood cancer known as BPDCN. If Stemline can successfully show that, in BPDCN patients, the drug provokes a highly significant impact on disease, SL-401 could be approved on the basis of a single small pivotal study.

Cancer Stem Cell Focus. We believe that the firm's knowledge of the cancer stem cell space sets it apart from other companies trying to develop chemotherapeutic agents or targeted drugs. If Stemline can successfully achieve the simultaneous targeting of both cancer stem cells and tumor bulk, we believe that its therapeutic agents could demonstrate clinical superiority in terms of both achieving remission in treated subjects and keeping such individuals cancer-free because of their ability to eliminate the cancer stem cells, which are known to play a central role in mediating cancer recurrence.

Attractive Valuation. In our opinion, Stemline Therapeutics currently trades at an interesting level for investors because of the relatively advanced maturity of its clinical-stage pipeline – SL-401 is on the cusp of Phase 3 development in BPDCN and acute myeloid leukemia, while SL-701 is in Phase 2b development in pediatric glioma and

adult glioblastoma – and because of the well-characterized mechanism of action underlying SL-401. The firm currently trades at a modest enterprise value of about $60 million, while other mid-stage cancer-focused firms trade at substantially higher valuations. Another firm that is focusing on cancer stem cells, Verastem, Inc., currently trades at a roughly $150 million enterprise value. Verastem, in contrast to Stemline, is unlikely to be able to negotiate an accelerated path to market for its lead candidates, in our view. We would also note that antibody- and vaccine-focused mid-stage oncology companies like Merrimack Pharmaceuticals and Celldex Therapeutics currently trade at enterprise values in the $500 million – $700 million range, without being substantially closer to commercialization at this point than Stemline Therapeutics.

Investment Risks

Financial Outlook. Stemline Therapeutics has been unprofitable since inception and may require additional capital in order to drive the future clinical development of its pipeline and finance the acquisition of other products and pipeline candidates. Thus, the company's stock could experience above-average risk and volatility, in our opinion.

FDA Unpredictability. New therapeutics development is a multi-year process that requires human clinical trials prior to FDA approval. The amount of additional clinical data that may be required to support regulatory filings on Stemline Therapeutics' drug candidates is unclear at this juncture, making it impossible to predict the precise timing of market entry and revenue generation. The FDA could also ask for additional data on Stemline's experimental candidates prior to granting formal licensure. Also, review times at the FDA may take longer than originally expected.

Competitive Landscape. Stemline Therapeutics is likely to face competitors with greater financial resources and larger organizations for marketing, sales, distribution, and service, assuming that the firm's candidates successfully obtain regulatory approval. Many of Stemline's peers may have stronger links to reimbursement agencies. This may allow them to establish more favorable relationships with payers than Stemline.

Partnership Risk. Thus far, Stemline lacks commercial experience as an entity and could eventually find itself having to rely upon partners to establish sales and marketing support for its products if they reach the market. Accordingly, therefore, the company is likely to be dependent upon such sub-licensees to execute on the commercialization of Stemline's proprietary drugs. In addition, certain elements of Stemline's intellectual property and drug candidate ownership rights are licensed from third parties. Should these third parties revoke the rights that they originally provided to Stemline, the company may be unable to further develop its candidate drugs or realize profits from their commercialization.

Intellectual Property. Stemline Therapeutics relies on patents and trade secrets to protect its products from competition. The pharmaceutical industry is litigious, and lawsuits are considered to be a normal part of doing business. A court might not uphold Stemline's intellectual property rights, or it could find that Stemline infringed upon another party's property rights. The company is also dependent in part upon the continue validity of intellectual property in-licensed from third parties.

Industry risks. The securities of emerging biotechnology and specialty pharmaceuticals companies are inherently volatile and increasingly subject to development and regulatory risk. Meeting or missing commercial milestones may result in changes in the perception of the firm and the stock price. We do not anticipate volatility to subside near-term.

For additional risk considerations, please refer to the company's SEC filings.

Valuation

Risk-Adjusted Net Present Value Analysis

We have projected the total firm value for Stemline Therapeutics based upon a sum-of-the-parts valuation. We are forecasting peak revenues for the firm's two clinical-stage drug candidates — SL-401 and SL-701 — of roughly $800 million and $240 million, respectively. This calculation yields a risk-adjusted NPV of roughly $345 million for these two candidates (see Table 1, below). Our estimates factor in a 40% tax rate and a 15% – 20% discount rate on future cash flows. We also project that the preclinical candidates in Stemline's pipeline could provide a $20 million rNPV contribution. Finally, we ascribe a $10 million rNPV contribution to the firm's proprietary StemScreen® technology platform, which could be leveraged through research agreements or licensing deals to garner additional revenue for the firm.

Thus, the total calculated firm value should, in our view, approximate $420 million. In this way, we believe, investors should note that the current market cap of Stemline neither appropriately values the company based on its existing clinical-stage pipeline and its potential for an accelerated pathway to commercialization, nor does it give the company any credit for an early – yet highly diversified – preclinical portfolio or the StemScreen® technology platform. Thus, we believe that Stemline Therapeutics may be an undervalued investment proposition with substantial risk-mitigation at this juncture.

Table 1: Composite Net Present Value (rNPV) Analysis

Stemline Therapeutics	Product	Launch Year	Patent Expiry	Peak Sales	Royalty Rate	Probability To Launch	NPV	Amount Per Share
Phase 2 / 3								
Hematological Malignancies	**SL-401**	2015	2027	$800MM	NA	70%	$305MM	$25.30
Brain Cancer	**SL-701**	2016	2025	$240MM	NA	50%	$40MM	$3.35
Preclinical								
Pipeline Candidates	**Various**	2020	2032	NA	NA	20%	$20MM	$1.70
Platform	**StemScreen™**	NA	NA	NA	NA	NA	$10MM	$0.90
Total							$375MM	$31.25
Debt at end-2013							$MM	$0.00
Cash at end-2013							$45MM	$3.75
Firm Value							$420MM	$35.00

Source: Company reports; Aegis Capital Corp. estimates

In our view, the main factors distinguishing Stemline Therapeutics from its peers are the solid scientific underpinnings of the mechanism of action for its lead candidate, particularly in BPDCN, and the management team's well-established track record of accomplishing clinical proof-of-concept and diversification even on a limited budget.

It is also appropriate, in our view, to examine the relative valuations of Verastem, Inc. and Stemline Therapeutics at this juncture. Another CSC-focused company, Verastem was the subject of a highly-publicized IPO in 2012. Currently, Verastem trades at a market capitalization of approximately $200 million, while Stemline trades at a market capitalization of roughly $85 million. On an enterprise value basis, Verastem is currently trading at nearly three times the value being attributed by the market to Stemline's pipeline and technology platform. We do not consider this a warranted valuation discrepancy. Stemline possesses a lead candidate in SL-401 that could reach the market faster than any of Verastem's candidates given the accelerated development path possible in BPDCN. Further, we believe that Stemline's CSC-focused early-stage pipeline may be superior in value to Verastem's portfolio because some of Stemline's candidates are monoclonal antibodies, which are difficult to genericize, whereas Verastem is developing small molecules that are simpler for generics firms to copy.

Comparables Analysis

Based on a comparable company analysis, it appears to us that the stock is worth approximately $35.00 per share (see Table 2, below). This assumes that the shares trade in line with the comp group average enterprise value of roughly $375 million and that the firm has approximately 12 million shares outstanding as of mid-2014. We believe that a comparison to a broad-based group of firms across the oncology domain is warranted, since Stemline has a diversified pipeline and competes across multiple cancer types.

Table 2: Comparable Company Analysis
(Millions, Except Per-Share Data)

Development	Therapeutic Area	Company	Ticker	Rating	Closing price 3/15/2013	Shares (MM)	Market cap ($MM)	Cash ($MM)	Debt ($MM)	Enterprise value ($MM)
Phase 2	Oncology / Hematology / Infections	ArQule, Inc.	ARQL	Not Rated	$2.55	62	159	83	2	78
Phase 3	Oncology	AVEO Pharmaceuticals	AVEO	Not Rated	$7.46	43	324	161	26	190
Phase 2	Oncology	Celldex Therapeutics	CLDX	Not Rated	$12.06	76	914	160	13	767
Phase 2 / 3	Oncology	Cyclacel Pharmaceuticals	CYCC	Not Rated	$5.77	9	54	18	0	36
Phase 2 / 3	Oncology	CytRx Corporation	CYTR	Buy	$2.51	30	76	22	0	54
Phase 2 / 3	Oncology	Galena Biopharma	GALE	Buy	$1.92	68	130	34	0	96
Phase 2 / 3	Oncology	ImmunoCellular Therapeutics	IMUC	Not Rated	$2.75	51	140	10	0	130
Phase 1 / 2	Oncology	Infinity Pharmaceuticals	INFI	Not Rated	$49.37	48	2352	326	0	2026
Phase 1 / 2	Oncology	Merrimack Pharmaceuticals	MACK	Not Rated	$6.03	94	568	87	0	481
Phase 3	Oncology	Northwest Biotherapeutics	NWBO	Not Rated	$3.60	26	92	10	0	82
Phase 3	Oncology	OncoGenex Pharmaceuticals	OGXI	Not Rated	$12.19	15	179	75	0	103
Phase 3	Oncology	Oncolytics Biotech	ONCY	Not Rated	$2.99	77	229	29	0	201
Phase 2 / 3	Oncology	Puma Biotechnology, Inc.	PBYI	Not Rated	$29.12	35	1019	160	0	859
Phase 2	Oncology	Verastem, Inc.	VSTM	Not Rated	$9.20	21	196	47	0	149
		Average					**459**			**375**
								Discrepancy		
Current valuation	**Oncology**	**Stemline Therapeutics, Inc.**	**STML**	**Buy**	**$11.55**	**7**	**86**	**29**	**0**	**57**
					Derived 12-month compa-month comparable value					
										Projected
Target valuation (18-month)	**Oncology**	**Stemline Therapeutics, Inc.**	**STML**	**Buy**	**$35.00**	**12**	**420**	**45**	**0**	**375**

Source: First Call and Aegis Capital Corp. estimates

Free Cash Flow: We believe that Stemline Therapeutics is likely to remain unprofitable for the foreseeable future. We define free cash flow as operating cash flow minus capital expenditures and dividend payments. We utilize a discounted cash flow analysis supporting a risk-adjusted Net Present Value (rNPV) framework to derive our $35.00 price target. This approach is described further in the next section of the report.

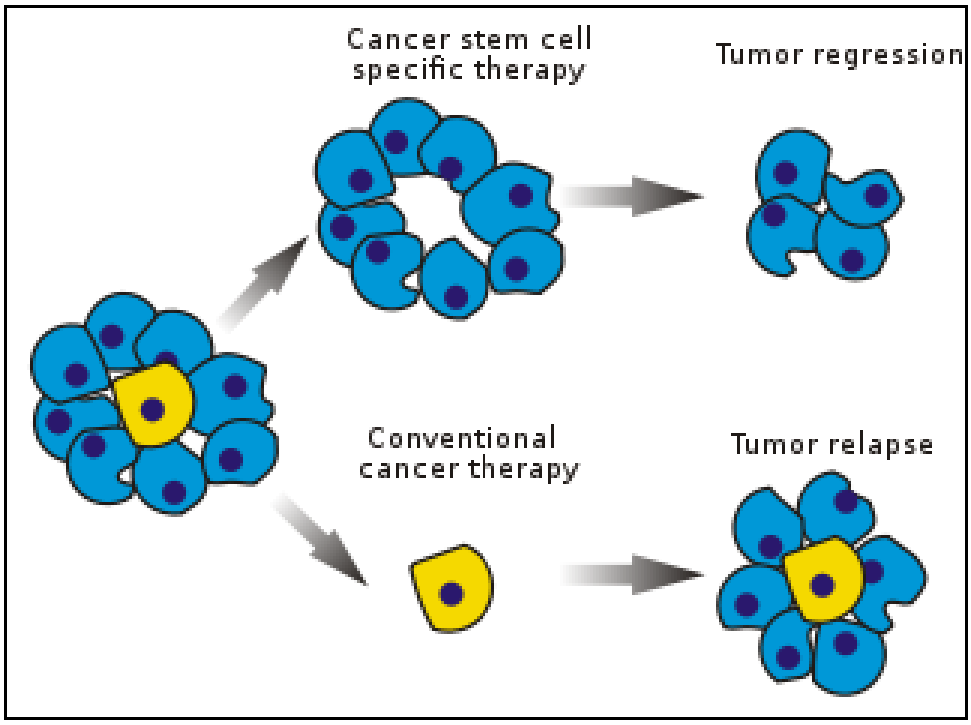
Our detailed analysis is split into three principal components: our discounted cash flow model, including the rNPV assessment of Stemline Therapeutics' clinical-stage development pipeline (presented in the preceding section); our assessment of the markets for Stemline's principal pipeline candidates, and the associated sales model for these drugs; and the near-term financial outlook for the company. Our historical income statement and financial projections are presented toward the back of this report.

Taxes: Stemline Therapeutics, Inc. has guided towards the expectation that the company is likely to continue to report net operating losses for the next several years, as the development of SL-401 and SL-701 through proof-of-concept and registration-quality clinical trials continues. Accordingly, therefore, we do not anticipate substantial tax liability for the foreseeable future. While the firm has not – unlike the majority of biotechnology firms that have been in existence for similar periods of time – accumulated a massive amount of net operating loss carry-forwards, we believe that by the time SL-401 and SL-701 reach the market, the net operating loss carry-forwards that would have been accumulated should offset taxes in the initial launch years of both products. Eventually, however, we would expect that the effective tax rate to be applied in the case of Stemline Therapeutics is likely to approach the federal U.S. statutory corporate tax rate of 35% plus the appropriate state-based tax supplement. Using these assumptions, we apply a roughly 40% effective tax rate to future cash flows.

Company Overview

Stemline Therapeutics was originally founded in 2003 as a privately-held firm funded by institutional and angel investors. The company was established with the specific aim of identifying and developing differentiated therapeutic approaches to oncology that are primarily aimed at targeting cancer stem cells (CSCs), the malignant cells within tumors that have been characterized over the course of the past decade as a principal driver of cancer recurrence and tumor re-growth in patients who had previously achieved substantial and durable responses to standard treatment methods, such as chemotherapy, radical surgery and radiation therapy. As shown below, CSC-targeting therapy allows more efficient and permanent tumor regression to be achieved.

Figure 1: Principle Underlying Cancer Stem Cell-Targeting Therapy



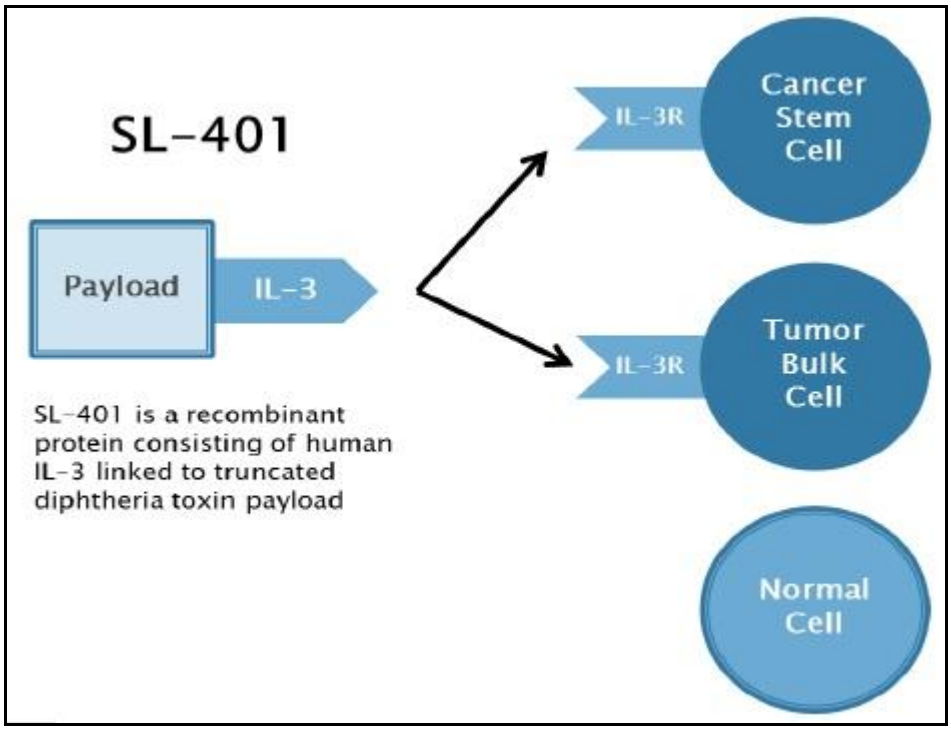
Source: Gupta et al., Nature Medicine 15: 1010 – 1012 (2009)

Today, Stemline Therapeutics is a clinical-stage biopharmaceutical company focused on discovering, acquiring, developing and commercializing proprietary therapeutics that target both CSCs and tumor bulk. The firm believes that it is developing the most clinically advanced pipeline of anti-CSC therapeutics and that it possesses the broadest intellectual property portfolio relating to CSCs in the biotechnology industry. These properties, in our view, clearly establish the company as a leader in the CSC field.

Stemline is developing two clinical-stage product candidates, SL-401 and SL-701, for which the company holds global marketing rights. The lead indications for SL-401, a biologic targeted therapy, are hematological malignancies including blastic plasmacytoid dendritic cell neoplasm (BPDCN) and acute myeloid leukemia (AML). The lead indications for SL-701, a subcutaneously delivered synthetic peptide vaccine, are pediatric and adult brain cancer. Specifically, Stemline is developing SL-701 in pediatric glioma and adult glioblastoma multiforme (GBM). In completed Phase 1/2 clinical trials, both SL-401 and SL-701 have demonstrated potent single-agent activity – including durable complete responses (CRs) – and longer overall survival (OS) in patients vs. that achieved in the past with traditional therapies.

In the near-term, Stemline Therapeutics plans to advance SL-401 into a single-arm, non-randomized trial in up to 40 – 50 patients with BPDCN, an ultra-orphan disease that is estimated to afflict approximately 2,000 patients worldwide, with roughly 700 patients presenting each year in the U.S. We believe that this trial could be initiated in late 2013 and yield sufficient data to justify a formal regulatory submission for approval of the drug in mid-2014. Assuming a six-month Priority Review period, SL-401 could enter the market for treatment of BPDCN in late 2015 / early 2016. Stemline is also planning to conduct a randomized Phase 2b trial in up to 240 adult subjects with relapsed or refractory AML patients who have failed two previous treatments (i.e. third-line AML) with overall survival (OS) as the primary endpoint. According to the firm's preclinical and clinical findings, SL-401 may also be useful in other blood cancers like myelodysplastic syndrome (MDS), lymphomas and myelomas. The strategy is to establish SL-401 in niches within the broader scope of hematological malignancies and then expand its use. The figure below depicts SL-401's basic mechanism of action.

Figure 2: SL-401 Mechanism Of Action



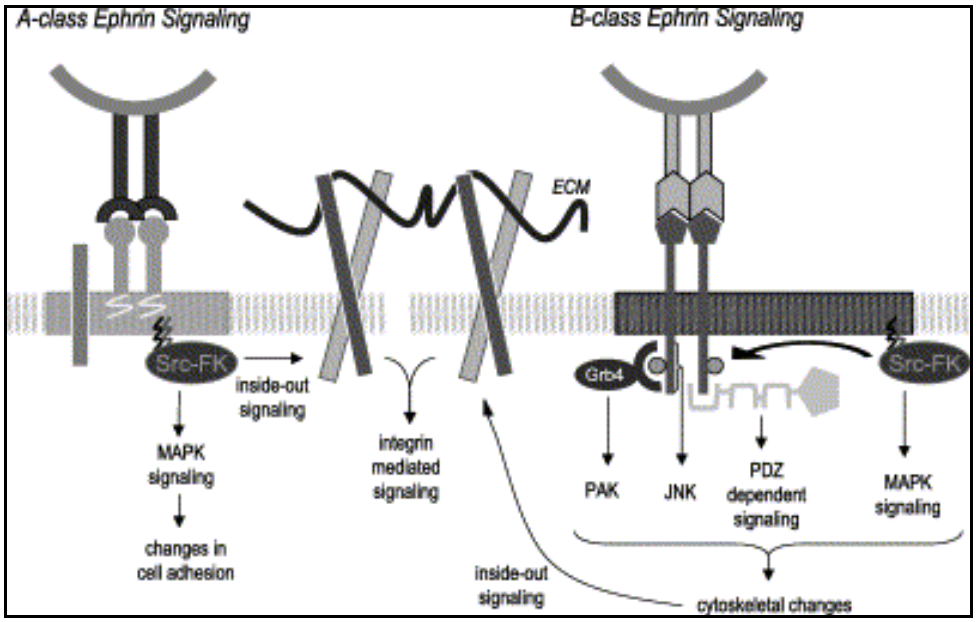
Source: Stemline Therapeutics, Inc.

One of the most interesting aspects of Stemline Therapeutics as an oncology play is the data that has been generated thus far in proof-of-concept clinical trials with SL-401. The ultra-orphan disease opportunity, BPDCN, constitutes a particularly promising area for application of this drug because it can easily be covered by a firm of Stemline's size, represents potential for orphan drug pricing, and is wholly unaddressed by currently-available anti-cancer therapies routinely used in other blood cancers. Median survival for BPDCN patients is only about 14 months. BPDCN is considered an aggressive and intractable disease. Thus far, in three patients treated with SL-401, Stemline has seen two durable complete responses (CRs), meaning that these patients were rendered completely free of disease and have remained in remission for over three months and over five months, respectively. In addition, the third treated patient achieved a partial response (PR). Although this is a very small data set, if it can be replicated in a broader population, we believe that Stemline could be well-positioned to achieve accelerated approval for SL-401 in BPDCN. This could occur within the next 24 – 30 months.

SL-701, the firm's second pipeline candidate, is a novel brain cancer vaccine consisting of several synthetic peptides specifically designed to elicit a defined anti-cancer immunological response against targets present on brain cancer tumor bulk and CSCs. Unlike SL-401, which is constructed to directly kill tumor cells and CSCs, SL-701 is aimed at "training" the patient's own immune system to recognize and eliminate the cancer. The target antigens that SL-701 is believed to sensitize the immune system against – the interleukin-13 receptor (IL-13Rα2) and the ephrin A2 (EphA2) protein – are known to be highly expressed in glioblastoma. Thus, in theory an immune response activated against these targets should eliminate both CSCs and tumor bulk in these indications. Stemline aims to advance SL-701 into a Phase 2b clinical trial to treat pediatric patients with newly diagnosed brain stem glioma (BSG) as well as non-brain stem glioma. The firm also plans to conduct a proof-of-concept study in roughly 30 patients and then advance SL-701 into a randomized Phase 2b trial in adult patients with glioblastoma (GBM) who have failed one previous treatment (i.e. second-line GBM).

The diagram below depicts ephrin signaling pathways. The A-class ephrins are glycosyl-phosphatidylinositol (GPI)-anchored receptor tyrosine kinases lacking a cytoplasmic domain, whereas the B-class ephrins have a single transmembrane domain with a short cytoplasmic motif. Ephrin A2, which is one of the antigenic targets presented by SL-701, plays a role in the guidance of neuronal growth cones to their targets in the CNS. Various publications have attested to the role of Ephrin A2 in cancer[1], and, as indicated earlier, it is expressed by both CSCs and tumor bulk in brain malignancies.

Figure 3: Ephrin Signaling Pathways



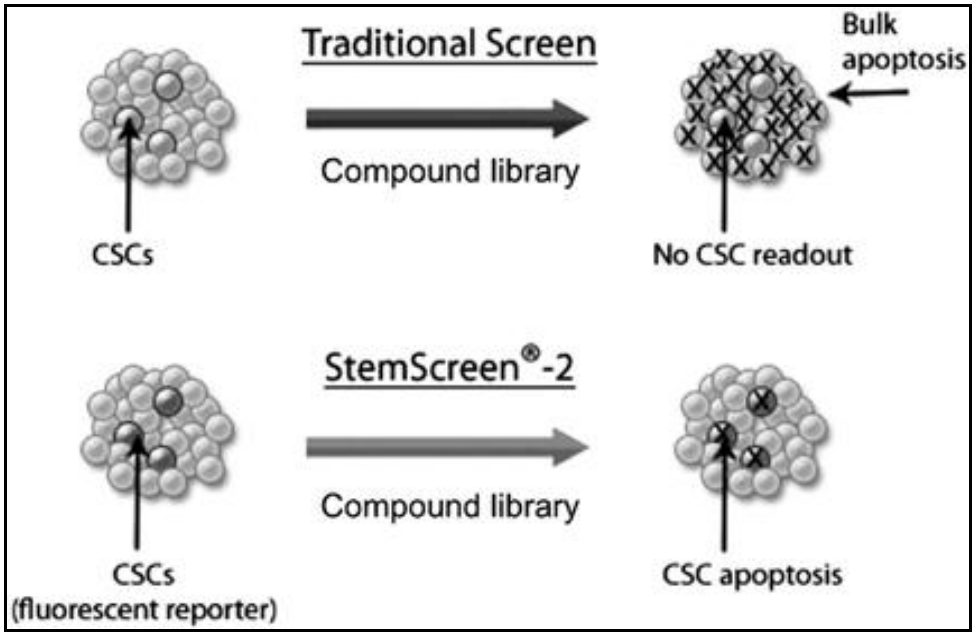
Source: Alberts, Molecular Biology of the Cell (2005)

Like the lead opportunities for SL-401 in blood cancers, the lead indications for SL-701 in brain cancers represent a relatively narrow niche within oncology overall. However, in our view they are strategically well-suited to targeting by a firm like Stemline because collectively they represent a small enough market to be covered by a company of Stemline's size, and because they are poorly served by currently-approved therapies. Accordingly, therefore, we believe that Stemline made an intelligent strategic choice to focus on these malignancies. Adult GBM represents a market of roughly 25,000 cases annually worldwide, and pediatric glioma represents fewer than 10,000 cases globally.

[1] Genander and Frisén, Current Opinion in Cell Biology 22: 611-616 (2010)

Beyond the clinical-stage candidates SL-401 and SL-701, Stemline also has a proprietary discovery platform, StemScreen®, for the discovery of novel CSC-targeted compounds, via which the firm has already discovered or validated several of its candidates and which may be instrumental in the discovery of new therapies targeting a wide range of cancer types. This platform provides Stemline with its own endogenous and proprietary discovery engine, and may also form the basis for licensing agreements and drug discovery and development campaigns conducted in partnership with other entities that could yield additional sources of revenue.

Figure 4: StemScreen® Technology Platform



Source: Stemline Therapeutics, Inc.

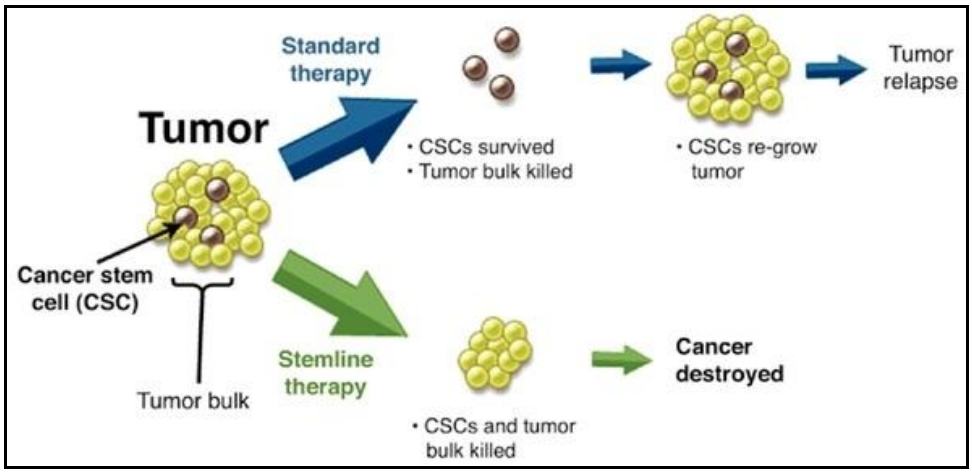
The CSC-targeting approach could fundamentally alter the approach to oncology drug development. CSCs have been identified in virtually all major tumor types, including leukemia and cancers of the brain, breast, colon, prostate and pancreas. These cells are the highly malignant "seeds" of a tumor that self-renew and generate more mature cells that comprise tumor bulk. As such, CSCs may be directly responsible for tumor initiation, propagation, and metastasis. Many of the characteristics of CSCs – such as slow growth, presence of multi-drug resistance proteins, anti-cell death mechanisms, and over-activated DNA repair machinery – may enable CSCs to resist therapeutic agents traditionally used to treat cancer. Further, while standard therapies may initially shrink tumors by targeting the tumor bulk, which excludes CSCs, there is a large body of evidence indicating that treatment failure, tumor relapse and poor survival are largely the result of the failure of conventional cancer treatments to eradicate CSCs.

Stemline holds fundamental intellectual property (IP) in the CSC-targeting field. One of its core patents covers a method to treat cancer through use of monoclonal antibodies and other antibody-based compounds that target CSCs, and related pending applications that cover methods to treat cancer through use of small molecule or oligonucleotide-based compounds that target CSCs. Additional patents and pending applications cover specific CSC-targeting agents aimed at discrete molecular targets; the use of CSC-targeting diagnostics in combination with CSC-targeting therapeutics; application of oligonucleotide-based drugs targeting CSCs in treatment of cancer; and novel approaches to CSC-targeted drug discovery, including the StemScreen® platform to discover compounds that target CSCs.

Stemline Therapeutics Pipeline

Unlike the vast majority of other oncology-focused companies developing drug candidates to treat various forms of cancer, Stemline Therapeutics has judiciously and determinedly built a broad-based pipeline of highly innovative candidate agents that preferentially target and eliminate cancer stem cells (CSCs). The firm was built upon the concept that the optimal way to treat cancer is to pair the ability to target tumor bulk with the ability to effectively ablate CSCs, which would, if left untouched, eventually regenerate the tumor over time. The schematic below illustrates this principle.

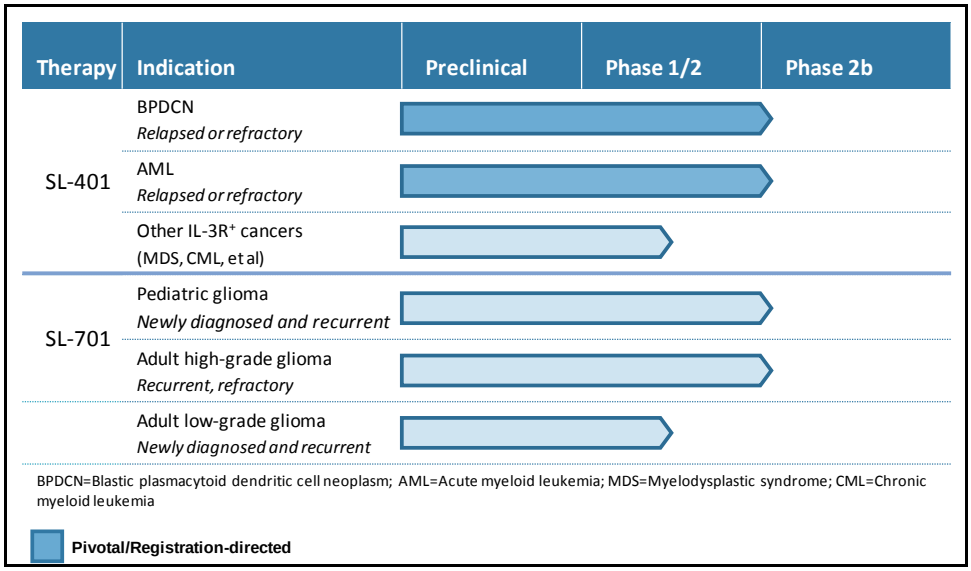
Figure 5: Cancer Stem Cell Targeting Principle



Source: Stemline Therapeutics, Inc.

Simultaneously, displaying a sophisticated understanding of the demands being leveled at publicly-traded biotech firms, Stemline has also established an advanced clinical-stage pipeline with two programs – SL-401 and SL-701 – centered on agents known to both target CSCs as well as tumor bulk. What we like about this portfolio is the fact that it focuses on niche opportunities within oncology that Stemline could tackle on its own.

Figure 6: Stemline Therapeutics Clinical-Stage Pipeline



Source: Stemline Therapeutics, Inc.

The firm's lead drug candidate, SL-401, is aimed at various hematological malignancies (blood cancers) and utilizes a tried-and-tested anti-cancer approach that was first proved with the commercialized drug Ontak® (denileukin diftitox). This agent was designed as a fusion protein and one of the first successful examples of a toxic payload-associated targeted therapy for use in cancer. Ontak® is composed of the interleukin-2 (IL-2) receptor-binding domain fused to the exotoxin secreted by *Corynebacterium diphtheriae*, the pathogen that causes the infectious disease diphtheria. The toxin exerts a cell-killing (cytotoxic) effect by inhibiting protein synthesis in mammalian cells through selective inactivation of eukaryotic elongation factor-2 (eEF-2). Ontak®, which was first approved in 1999 for treatment of cutaneous T cell lymphoma (CTCL), showed broad potency against a wide range of hematological malignancies.

Nevertheless, Ontak® did not achieve broad usage in blood cancers because IL-2 receptor expression – necessary to permit the drug to achieve ingress into cancer cells – is not uniform across different cancer types[2]. Wherever IL-2 receptor expression was high in cancer types targeted by Ontak®, responses to the drug appeared to be significant and robust. One recent example of this was observed in patients with fludarabine-refractory chronic lymphocytic leukemia (CLL), a cancer type widely considered incurable[3]. Ontak® provided substantial benefit in this difficult-to-treat patient population, with roughly half the treated patients in this study showing >50% reductions in the CLL cells in their blood. Two treated patients had 95% reductions in CLL cell counts. Two partial responders in this 18-patient trial had prolonged responses lasting over a year (14 and 19 months). However, in cancer types where the IL-2 receptor was less widely expressed and at lower levels on cancer cell surfaces, cytotoxicity specific to tumor cells exerted by Ontak® proved to be less efficient. Therefore, we believe that the issue with Ontak® may have lain primarily with the drug's targeting mechanism, not with the principle underlying its original design. Investors should note that antibody-drug conjugates, armed antibodies, and armed fusion proteins are now considered *de rigueur* in the cancer treatment continuum, and indeed are fast-becoming the vanguard of specialized therapy.

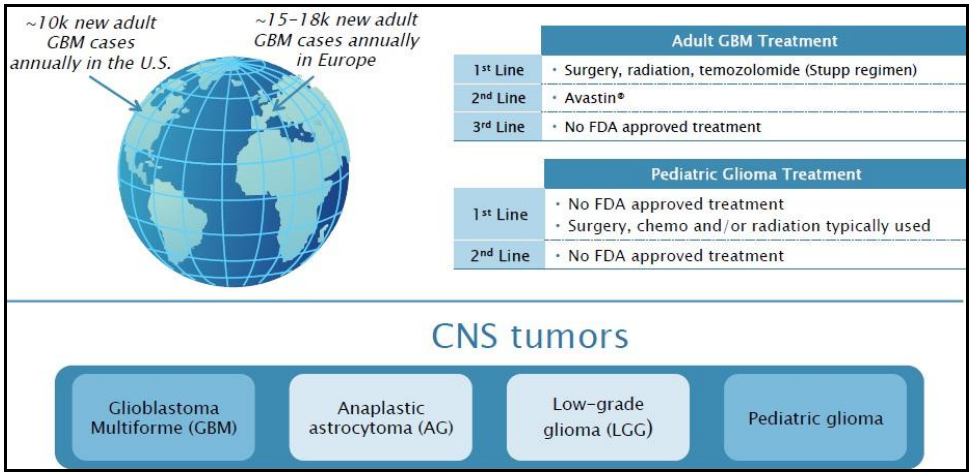
However, we believe that the fact that Ontak® received FDA approval and utilized the same mechanism of action and therapeutic principles that underlie SL-401 should be considered promising for SL-401, which uses the same diphtheria toxin component present in Ontak® as the toxic payload of the fusion protein that – when delivered intracellularly – selectively kills cancer cells. However, unlike Ontak®, which used the interleukin-2 receptor (the alpha chain of which is generally referred to as CD25) to home in on cancer cells, SL-401 uses the interleukin-3 (IL-3) protein to selectively target and ablate cancer cells. Interleukin-3 binds to the interleukin-3 receptor, low affinity (IL-3RA), which is designated CD123.

We believe that one of the most interesting aspects of SL-401 biology is the fact that CD123 appears to be highly expressed in various hematodermic neoplasms (in which there is abnormal proliferation of immune cells in the blood that leads to the formation of skin lesions)[4] and, indeed, across many hematological malignancies. Importantly, traditional therapies – including those currently deployed to treat blood cancers, target the genetic material (DNA and RNA) of the cancer cells during replication. However, CSCs are often quiescent and thus do not replicate regularly, making these genetic material-targeted therapies ineffective. Diphtheria toxin (DT) is ideally suited, in our view, to the targeting of CSCs because it interferes with protein synthesis, which constantly occurs in all cells including CSCs, even if they are quiescent. In addition, DT is not subject to multi-drug resistance efflux pumps expressed by cancer cells, which typically allow these cells to exhibit resistance to traditional chemotherapeutic agents.

[2] Robak. Leukemia & Lymphoma 45: 205-219 (2004)
[3] Frankel *et al*., Clinical Cancer Research 9: 3555-3561 (2003)
[4] Piña-Oviedo *et al*., Applied Immunohistochemistry & Morphology 15: 481-486 (2007)

Stemline Therapeutics' second clinical-stage pipeline candidate, SL-701, is an agent consisting of several synthetic peptides corresponding to several epitopes known to be expressed on both the CSCs and tumor bulk in various brain cancers. As shown below, the brain cancer market is a niche opportunity that can feasibly be targeted by a company Stemline's size, and existing therapeutic options are few and far between[5]. Accordingly, we believe that SL-701 could potentially become a lucrative product opportunity.

Figure 7: Brain Cancer Market Overview



Source: Stemline Therapeutics, Inc.; National Cancer Institute

In two completed Phase 1/2 clinical trials, SL-701 demonstrated single agent anti-tumor activity in pediatric patients with newly diagnosed brainstem glioma (BSG), and other high-grade gliomas (HGGs), and in adult patients with refractory or recurrent GBM, and other HGGs. SL-701 induced tumor shrinkage or disease stabilization in 84% (16/19) of patients in the pediatric study, and 59% (13/22) of patients in the adult study. This includes two CRs and five PRs. Seven of ten pediatric patients with newly diagnosed BSG treated with SL-701 survived past the historical median of 9.6 months, including three children who have survived for periods >50% greater than the historical median survival. Additionally, the OS of adult patients with recurrent or refractory GBM and other HGGs who were treated with SL-701 was increased vs. historical results for similar patients treated with a wide range of therapies.

Immune system cells utilize human leukocyte antigen (HLA) molecules to bind and present antigenic peptides to T cells to initiate a specific immune response. The SL-701 peptides were designed to bind to HLA-A2, the most common Class I HLA molecule (approximately 45-50% of the North American population). Accordingly, in the completed Phase 1/2 trials, HLA-A2+ patients were specifically enrolled. Based on the clinical responses and survival signal seen in these studies, Stemline aims to continue to select HLA-A2+ patients for both the pivotal Phase 2b pediatric trial and the randomized Phase 2b adult second-line GBM trial. The company also plans to test SL-701 peptide binding to other Class I HLA molecules to potentially expand the target population.

Stemline intends to test SL-701 in a Phase 2b trial in pediatric patients with newly-diagnosed BSG and non-brain stem glioma, which could start late in 2013 or early in 2014. The firm also aims to initiate a Phase 2b trial in adult second-line GBM. There are also open INDs for patients with low-grade glioma, or LGG. We believe that the insertion of a product like SL-701 into the brain cancer treatment continuum could have substantial benefit for patients who currently have limited treatment options. It is important to note that second-line GBM patients have very limited survival prognoses.

[5] Caffo *et al*., International Journal of Molecular Sciences 14: 2135-2174 (2013)

In addition to SL-401 and SL-701, Stemline Therapeutics has also since inception been building a preclinical pipeline of drug candidates that leverage the firm's proprietary knowledge base in the domain of cancer stem cells (CSCs). This pipeline is listed in the table below. Currently, Stemline has several small molecule and monoclonal antibody candidates in early-stage development. Pending the generation of positive *in vivo* data with these agents, we expect that the company could be in position to file 1 – 2 Investigational New Drug (IND) applications per year – starting in 2014 and continuing for the next two to three years – with the existing preclinical pipeline as a foundation, and is in position to announce the discovery of new targets and therapeutic molecules over the course of the coming years.

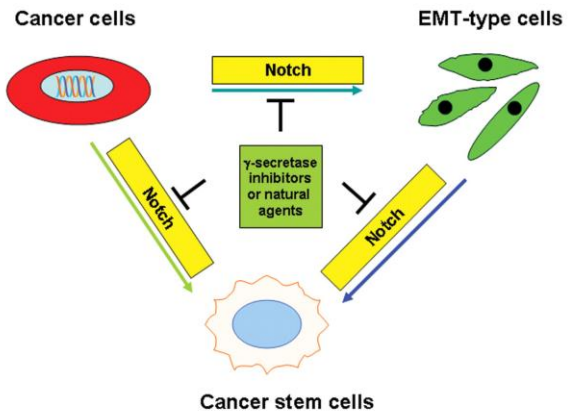
Table 3: Stemline Therapeutics Preclinical Pipeline

Target	Compound	Class	In Vitro Efficacy	In Vivo Efficacy
Notch	SL-301	Small molecule	+	+
CD123	SL-101	mAb-based	+	
T-201	SL-201	Small molecule	+	
---		3 small molecule hits	+	
T-601	SL-601	mAb-based		

Source: Stemline Therapeutics, Inc.

The above preclinical pipeline is intriguing from our perspective because it spans both well-known targets, such as Notch and CD123 (the IL-3 receptor, which is also the target of SL-401), and undisclosed proprietary targets, which were unearthed by Stemline using its StemScreen® technology platform. This approach allows Stemline to preferentially screen for new targets that have specific relevance to CSCs.

Figure 8: Notch Pathway Involvement In Cancer



Source: Wang et al., Anticancer Research 31: 1105-1113 (2011)

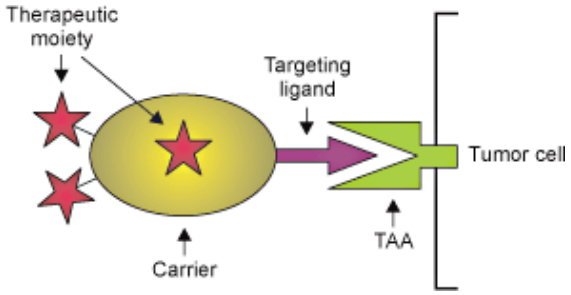
As shown above, the involvement of Notch in cancer has been studied with reference to its role in regulating CSCs. The Notch pathway is one of the central pathways in mammalian cell development, regulating proliferation and cell fate specification (differentiation). The above figure depicts how Notch stimulates the proliferation of epithelial-mesenchymal transition (EMT) type cells – which are cells that can switch back and forth between epithelial (skin and neural tissue) and mesenchymal (connective tissue and muscle) cell fates – and also drives the proliferation of CSCs, which have been found to have various characteristics in common with EMT-type cells[6].

[6] Karamboulas & Ailles, Biochimica et Biophysica Acta 1830: 2481-2495 (2013)

SL-401 Overview

Stemline Therapeutics' most advanced clinical candidate, SL-401, is based on a widely-accepted principle in cancer therapy – that of targeting a cytotoxic payload specifically into cancer cells. In this case, the drug was developed to concomitantly ablate both tumor bulk as well as cancer stem cells (CSCs). We are encouraged by the fact that the agent is based on the same fundamental design principles that informed the development of Ontak® (denileukin diftitox), because we consider Ontak® a validating precedent from both a regulatory standpoint – this drug was approved by the FDA in 1999 for treatment of hematological malignancies, most notably cutaneous T-cell lymphoma – as well as because we believe that the strong efficacy data generated with Ontak® provides risk-mitigation when assessing SL-401's clinical prospects.

Figure 9: SL-401 Design Principle



Source: Principles in Cancer Drug Design (2005)

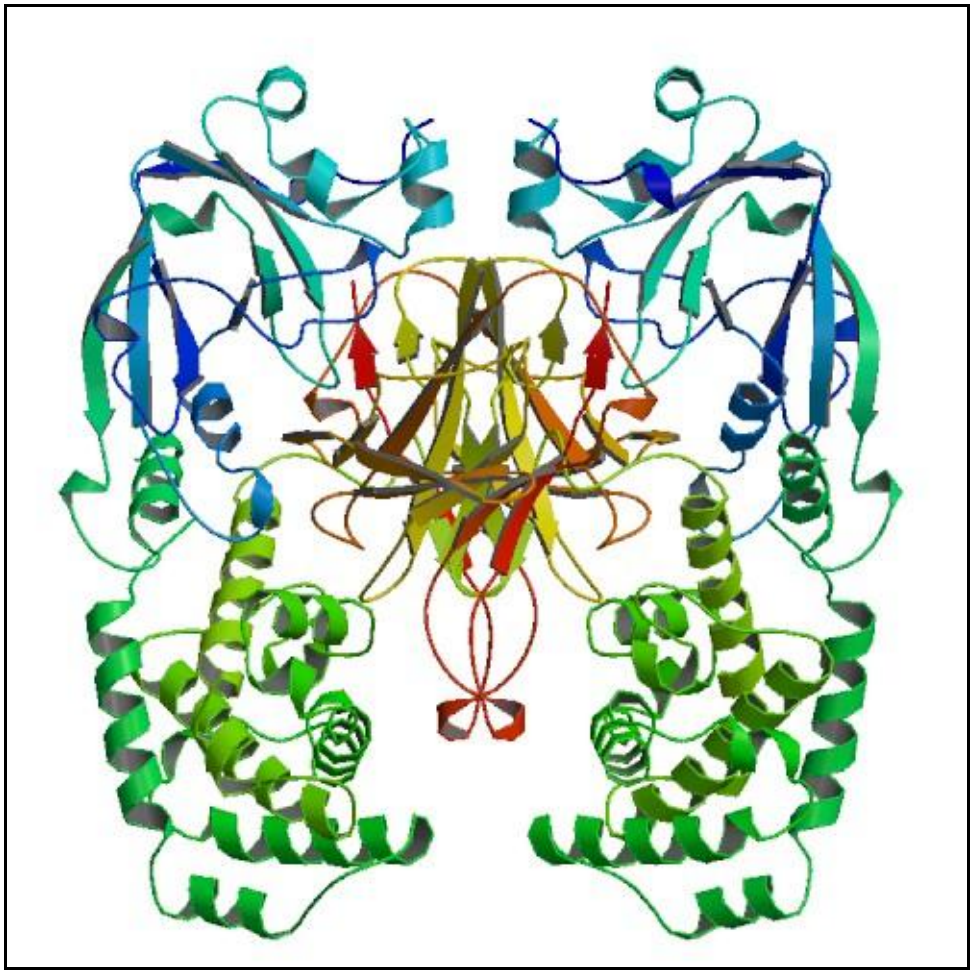
As shown in the figure above, the basic concept behind the design of cytotoxic payload-carrying anti-cancer agents is the use of a targeting ligand – which binds to receptors selectively expressed on the surfaces of cancer cells and, in the case of SL-401, CSCs – to deliver the cytotoxic payload specifically into tumor bulk cells and CSCs while leaving normal cells unscathed. In the case of both Ontak® and SL-401, the selection of the diphtheria toxin as the payload of choice is informed by the fact that it is a very potent cell-killing agent and the fact that – since it is a biologic molecule that interferes with multiple protein synthesis pathways – the evolution of resistance is very difficult. Unlike small molecules targeting – for example – receptor tyrosine kinase proteins on the surfaces of cancer cells, diphtheria toxin cannot be resisted through single mutations in protein structure. This makes it, in our view, a solid choice as a cytotoxic payload.

We recognize the advantages to Stemline Therapeutics of working with a payload that has already received the green light from the FDA and that has demonstrated efficacy in various hematological malignancies – particularly those affecting the skin. These characteristics make SL-401 a substantially more risk-mitigated clinical-stage asset than many other anti-cancer agents currently in development, in our view.

It is useful, from our perspective, for investors to be aware of the clinical development history and commercial trajectory of Ontak®. We believe that this drug represents the most direct precedent-setting product for SL-401, although – for reasons that we elaborate further in our market model section for the drug – we are reasonably optimistic that the commercial experience with Ontak® is not fully indicative of the opportunity that SL-401 may represent. Initially developed by Ligand Pharmaceuticals, Ontak® was originally approved in 1999 under the accelerated approval program for cutaneous T-cell lymphoma based on durable objective responses in an open-label study comparing two different doses. The sole manufacturer of Ontak® in the U.S. is now the oncology division of the Japanese pharmaceutical firm Eisai Inc., which as of January 2013 has been experiencing a shortage of the drug due to manufacturing issues. On October 15, 2008, the FDA converted the approval of Ontak® solution for intravenous use for the

treatment of persistent or recurrent CD-25 positive cutaneous T-cell lymphoma from accelerated approval to regular approval following confirmation of an improvement in progression-free survival (PFS) and overall response rate (ORR). Efficacy was demonstrated in a placebo-controlled multinational dose-ranging study that enrolled 144 patients with CD-25 positive stages Ia-III cutaneous T-cell lymphoma. Patients who had three or fewer prior therapies were randomly assigned to receive an IV infusion of either denileukin diftitox at 0.018mg/kg or 0.009mg/kg on days 1-5 of a 21-day cycle (maximum 8 cycles) or placebo (saline). The three study arms were balanced for baseline demographic characteristics. Patients were required to have CD-25 expression in tumor specimens on central laboratory testing. The median age was 59 years; 34 percent were age 65 years or older. Roughly 65% of the study population had less advanced (≤ stage IIa) disease; the median number of prior therapies was two.

Figure 10: Ontak® (Denileukin Diftitox) Protein Structure



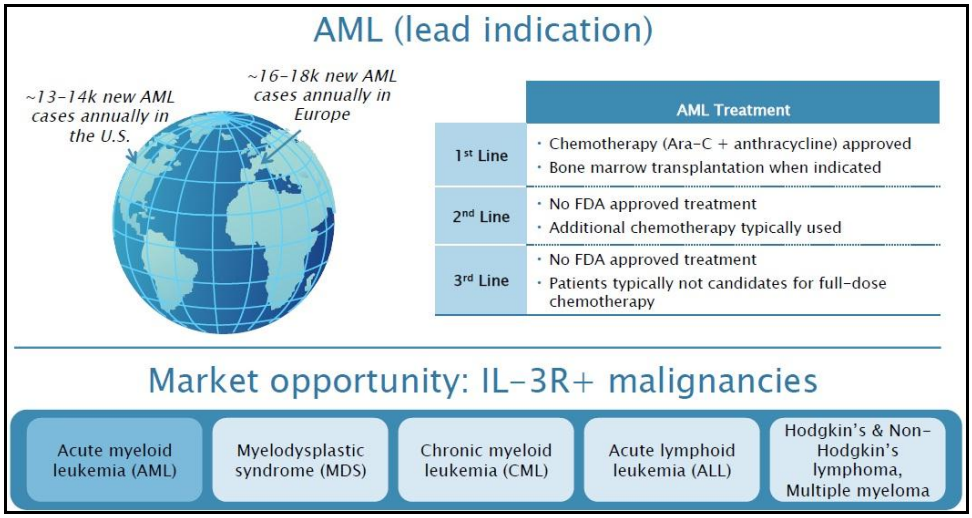
Source: Protein Data Bank

ORR for patients receiving the 0.018mg/kg dose was 46% and median response duration of 220 days. The ORR for patients on receiving the 0.009mg/kg dose was 37% and median response duration of 277 days. Patients on the saline placebo had an ORR of 16% and median response duration of 81 days. Significant improvements in PFS at both doses of denileukin diftitox were noted (HR=0.27 comparing 0.018 mg/kg vs. placebo, p=0.0002; HR=0.42 comparing the 0.009 mg/kg vs. placebo, p=0.02). Treatment-emergent adverse events that occurred in at least 20% of patients in the 0.018mg/kg group, and more frequently than in the placebo arm, were: fever, nausea, rigors, fatigue,

vomiting, headache, peripheral edema, diarrhea, anorexia, rash, and myalgia. Serious adverse events in patients receiving denileukin diftitox included infusion reactions, capillary leak syndrome, and loss of visual acuity, including loss of color vision. Laboratory abnormalities reported included hypoalbuminemia and hepatic transaminitis.

The figure below provides an overview of the market opportunity associated with SL-401 in the indication of acute myeloid leukemia (AML) alone; it does not include blastic plasmacytoid dendritic cell neoplasm (BPDCN), an even more interesting indication because it is an ultra-orphan opportunity. Although BPDCN is only estimated to afflict 2,000 patients in the U.S. and Europe, it could represent the first approved indication for SL-401 and thus could allow Stemline to establish a high per-patient price for the drug. This would be justified based on its rarity and the fact that existing therapies are ineffective – BPDCN is aggressive and intractable, with a 14-month median survival.

Figure 11: SL-401 Market Opportunity



Source: Stemline Therapeutics, Inc.

The table below provides an overview of the clinical activity data that has thus far been reported with SL-401. Clinical experience has largely been restricted due to Stemline's capital-constrained condition prior to its successful IPO – nevertheless, as shown below, even single-cycle regimens have elicited potentially encouraging responses. We would draw investors' attention to the high blast reduction and disease stabilization rates achieved after only limited exposure to SL-401 (in the table below, patients were only given a single cycle of therapy).

Table 4: SL-401 Hematological Malignancies Clinical Data

	AML *Relapsed, refractory (n=59)*	AML *≥3rd line (n=35*)*	AML *Not chemo candidate (n=11)*	MDS *High risk (n=7)*	BPDCN *(n=3)*
Blast reductions / disease stabilization	46%	43%	55%	43%	67%
Blast reductions	25% 2 Durable CRs	23% 1 Durable CR	27%	29%	67% 2 CRs

AML = Acute myeloid leukemia; MDS = Myelodysplastic syndrome; BPDCN = Blastic plasmacytoid dendritic cell neoplasm
CR = Complete response
*Subpopulation of relapsed, refractory

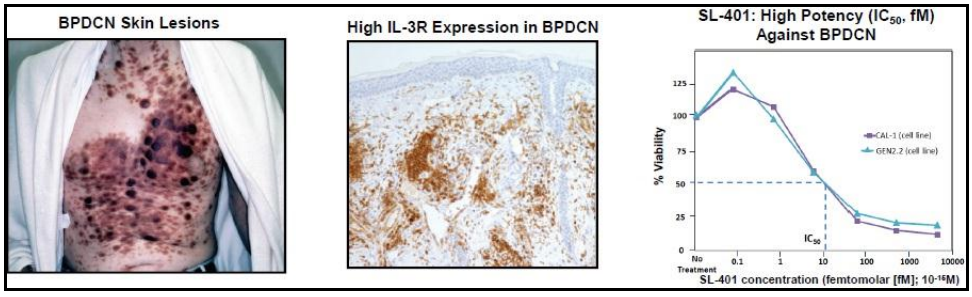
Source: Stemline Therapeutics, Inc.

In our view, two things are abundantly clear from the data in Table 4 – the patient data sets were relatively small, but not substantially smaller than those normally seen in proof-of-concept studies done in oncology; and the activity of the drug is quite striking, particularly when one notes the context that all of these studies involved late-stage patients, generally with relapsed/refractory or aggressive disease. Finally, we would recommend that investors note the fact that all of this activity data was achieved using only a single cycle of therapy. Clearly, in our view, the data reported thus far should only be a modest indication of the true optimal efficacy profile of SL-401.

What is also encouraging to us is the fact that – as reported in the literature – the safety profile of Ontak®, at least with respect to injection site reactions, hypoalbuminemia, edema and liver enzyme elevation, is known to improve over time. Accordingly, the safety profile of SL-401, which is closely related to Ontak®, should also improve over time. Unlike the vast majority of other anti-cancer agents, which are fundamentally toxic and which therefore can only be given for limited periods of time and in limited amounts, SL-401 and Ontak® actually become better-tolerated the more they are given. In our view, many of the issues associated with Ontak® are likely to be absent from SL-401. Stemline's drug has never been linked to vision problems, unlike Ontak®, and we believe there is stronger evidence in favor of the therapeutic impact associated with usage of IL-3 as a targeting ligand vs. IL-2 in the indications that Stemline is focusing on with SL-401.

The lead indication for SL-401 is currently blastic plasmacytoid dendritic cell neoplasm (BPDCN). An ultra-orphan hematological malignancy, BPDCN was unknown prior to 1994 and has previously been described as blastic natural killer cell lymphoma or $CD4^+/CD56^+$ hematodermic neoplasm, given the characteristics of the disease and the principal markers expressed by the tumor cells. The figure below shows the pathological manifestations of BPDCN, which is principally characterized by the large, dark and diffuse skin-based lesions (left panel) seen in patients. The disorder is a highly aggressive neoplasm associated with cutaneous manifestations, followed by dissemination to blood, bone marrow, and other tissues. Neoplastic cells exhibit a lineage-negative $CD4^+/CD56^+/CD43^+/HLA\text{-}DR^+$ immunophenotype, initially suggesting an NK-cell derivation[7]. The recent discovery of CD123 antigen (the IL-3 receptor) expression by tumor cells indicates a relationship to immature dendritic cells (DCs). Plasmacytoid DCs express high CD123 levels and produce interferon (IFN)-alpha when triggered by antigens. In all cases, neoplastic cells were reactive for CD123, BDCA-2, and MxA protein, providing strong evidence for an immature plasmacytoid DC derivation for this rare neoplasm. As shown in the central panel, the advantage of using SL-401 in BPDCN is the fact that IL-3 receptor expression is essentially a hallmark of this disease. All patients diagnosed with BPDCN express this receptor, which makes them candidates for treatment with SL-401. The far right panel below depicts the extremely potent activity of SL-401 against cell lines expressing high levels of CD123.

Figure 12: SL-401 Hematological Malignancies Clinical Data



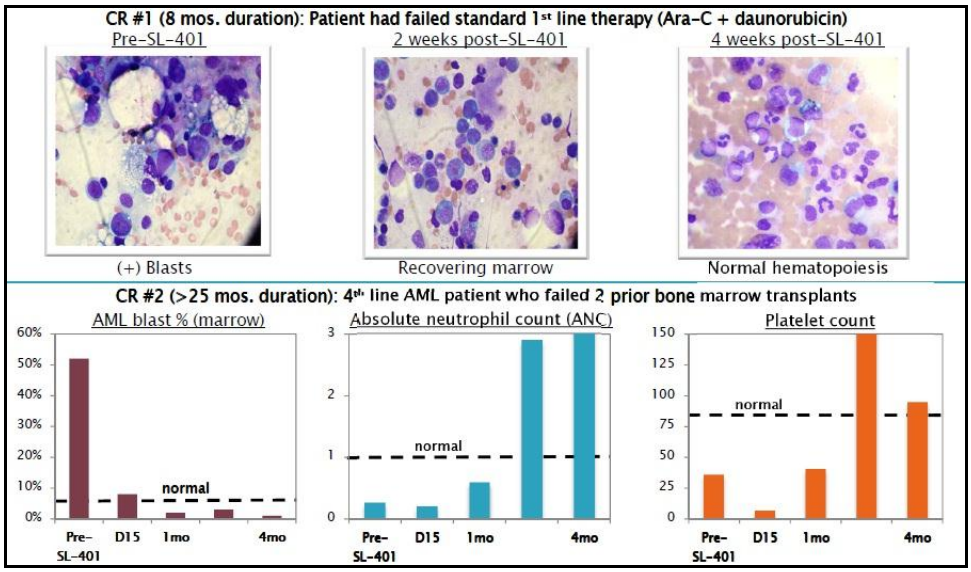
Source: Stemline Therapeutics, Inc.

[7] Pilichowska *et al.*, American Journal of Clinical Pathology 128: 445-453 (2007)

In all patients with BPDCN who were treated with a single cycle of SL-401, drug activity was readily observed. All these patients had been heavily pre-treated with standard of care therapeutic regimens (primarily multiple lines of chemotherapy) and were relapsed or refractory. Two patients who received the higher dose of SL-401 – which Stemline proposes to be the optimal therapeutically effective dose – exhibited complete responses, both of which have now persisted for over three months (one of them for over five months). The first patient – treated third-line with SL-401 – presented with malignant blasts in bone marrow and blood, low peripheral blood counts, and enlarged lymph nodes and spleen. All of these signs and symptoms were eliminated post-treatment with SL-401. The second patient was treated fourth-line and presented with multiple skin lesions and a history of blasts in bone marrow and blood. Similarly, all signs and symptoms were eliminated using SL-401. Given the low historical median survival rate of 14 months in BPDCN, we consider this initial data early but extremely encouraging.

The second major indication in which Stemline Therapeutics plans to investigate SL-401 is acute myeloid leukemia (AML). While this disease can be amenable to traditional treatment, patients still often relapse or become refractory to chemotherapeutic regimens against which they initially showed responses. As shown below, SL-401 administered to patients on a single-cycle basis was able to elicit complete responses that were extremely durable (eight months in one case and >25 months in another) in subjects who had previously failed at least one prior line of chemotherapy. In some cases, SL-401 demonstrated efficacy even in the fourth-line setting. The figure below shows how SL-401 restored normalcy to the bone marrow cytology in these patients, returning them to normal hematopoiesis, and that the drug also restored blood cell counts to normal levels.

Figure 13: SL-401 Acute Myeloid Leukemia Individual Patient Data

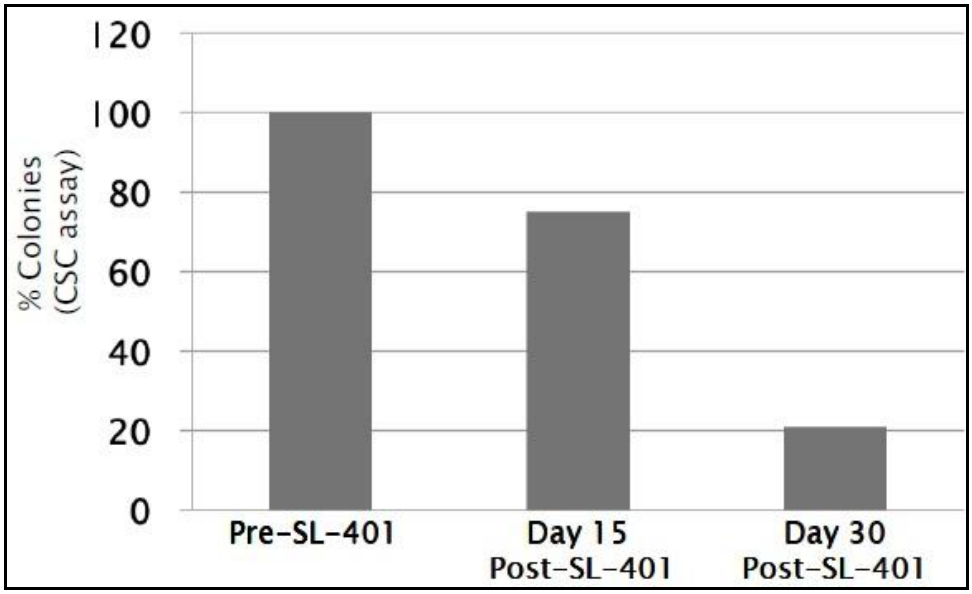


Source: Stemline Therapeutics, Inc.

In our view, the AML indication could essentially be treated as a free call option at this point by investors entering Stemline stock at current levels. This is because the BPDCN commercial opportunity could be lucrative on its own, without SL-401 ever seeing substantial use in AML. However, if Stemline were able to prove in a controlled manner that SL-401 can also work in AML patients – whether post-third-line or not – the market potential for the drug would be substantially increased. We would note that Stemline does possess some proof-of-concept survival data in the AML setting as well; however, AML patients may not uniformly express the IL-3 receptor although it is generally present on CSCs in patients with these types of cancers.

There is some preliminary clinical evidence supporting the hypothesis that SL-401 can impact both cancer stem cells (CSCs) and tumor bulk. In three AML patients who were treated with the drug, CSC levels declined over time, reaching their lowest levels 30 days post-dosing (see below). In our view, repeated cycle administration with SL-401 could eventually lead to complete ablation of the CSC populations in treated patients.

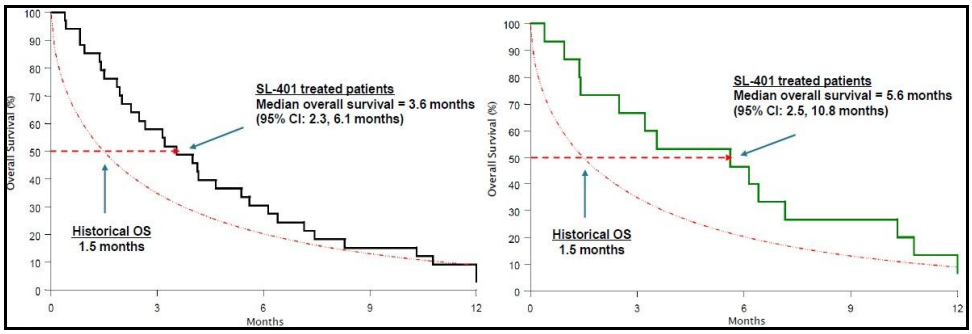
Figure 14: SL-401 Clinical Anti-CSC Activity



Source: Stemline Therapeutics, Inc.

We believe that it is important for investors to note the fact that some of the clinical data generated thus far with SL-401 involved doses of the drug that are below what Stemline considers the optimal effective dose range of 9 – 16.6µg/kg/day, and therefore much of this clinical data may under-represent the effectiveness of the drug. This is best illustrated below, via the overall survival (OS) curves of third-line or greater AML patients treated with a single cycle of SL-401. The pair of Kaplan-Meier plots on the left shows patients treated with all doses of SL-401 (n=35), while the pair of plots on the right shows those given the optimal effective doses (n=16). There appears to be a clear dose-response: while, in the overall group, SL-401 more than doubled median OS of third-line or greater AML patients vs. historical controls (1.5 months → 3.6 months), in the patients given the optimal effective doses, median OS more than tripled (1.5 months → 5.6 months). In our view, this data at the very least indicates that SL-401 warrants further study in post-third-line AML at the maximum tolerated dose (16.6µg/kg/day).

Figure 15: SL-401 Acute Myeloid Leukemia Kaplan-Meier Curves

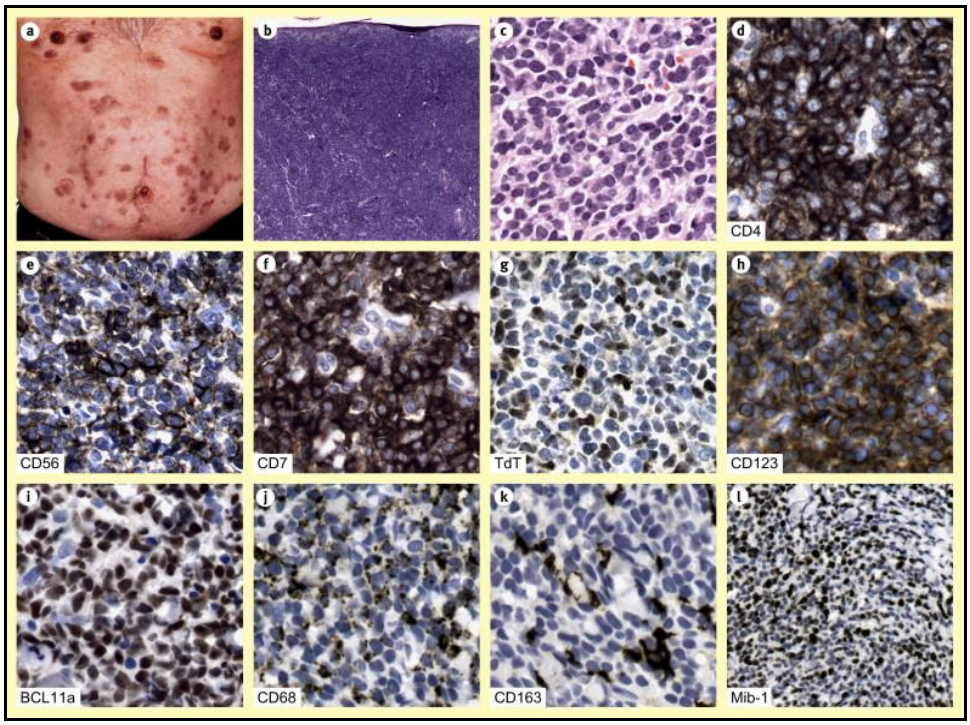


Source: Stemline Therapeutics, Inc.

Blastic Plasmacytoid Dendritic Cell Neoplasm

The background of BPDCN is somewhat confusing, as the disease has only been formally recognized since 1994 and has previously been referred to by different names. Initially, it was called hematodermic neoplasm – referring to the particularity of the skin lesions formed by abnormal cells that originated in the blood. Characterization of the cells with natural killer cell surface markers led researchers to refer to the disease as blastic natural killer cell lymphoma; however, in recent years the World Health Organization has renamed the disorder blastic plasmacytoid dendritic cell neoplasm in recognition of the fact that the abnormal cells (as shown in the figure below) express various markers associated with immature dendritic cells, particularly CD123 (IL-3 receptor).

Figure 16: BPDCN Histopathological Characteristics



Source: Kevin Gatter, Oxford University, U.K.

An estimated annual incidence for BPDCN of 700 patients in the U.S. and roughly 1,000 patients in Europe is derived from a projection made in a recently published review of epidemiological data[8]. This paper indicates that the estimated incidence of BPDCN should be 0.44% of hematological malignancies. Since the etiology of BPDCN remains unclear and it has been classified as both lymphoma and leukemia, the actual incidence is difficult to estimate accurately. However, using the 0.44% percentage number, and assuming approximately 160,000 hematological malignancies occurring each year in the U.S. and 230,000 in continental Europe, this translates into approximately 700 cases in the U.S. and 1,000 in Europe occurring annually.

The 0.44% incidence rate reflects cases of BPDCN misdiagnosed as NHL or AML, so it does not include cases diagnosed correctly. It also does not include cases misdiagnosed as cancers other than NHL or AML such as cutaneous lymphomas, leukemias involving the skin, and others. This may be a further reason that the 700 and 1,000 numbers represent a conservative assumption.

[8] Wang *et al*., *Acta Haematologica* 127: 124-127 (2012)

SL-401 Licensing Agreement Background

In June 2006, Stemline Therapeutics entered into a research and license agreement with Scott and White Memorial Hospital for SL-401, the firm's biologic targeted therapy invoking the interleukin-3 receptor (IL-3R). Under the agreement, Scott and White granted Stemline Therapeutics an exclusive, royalty-bearing, worldwide license for research, development and commercialization of SL-401, and any products containing or comprising this compound in finished dosage pharmaceutical form, for the diagnosis, prophylaxis and/or treatment of any disease or condition in humans or animals.

Under the terms of the agreement, Stemline must pay Scott and White royalties based on adjusted gross sales, by either the company or its sub-licensees, of products containing the licensed compound for a period of ten years following the first commercial sale of each product in each country. The royalty rates for each product range from the low- to mid-single digits and are tiered based on the total annual sales achieved by the product.

The company holds sub-licensing rights under the agreement, subject to the payment to Scott and White of a percentage of the up-front payments received from a sub-licensee. If Stemline fails to exert what under the agreement constitute materially reasonable efforts to secure the regulatory approval of SL-401 and commercialize the drug, Scott and White has the option, according to the terms of the arrangement, to convert the exclusive license to Stemline into a non-exclusive license.

The agreement provides for Scott and White to conduct a research program with SL-401. In March 2010, the agreement was amended to further the regulatory advancement of SL-401. Stemline has thus far made certain payments to Scott and White for such research services pursuant to the agreement, which to date total $700,000 in total. Additionally, upon Stemline's request, the agreement requires Scott and White to either assign to the firm its IND for SL-401 or grant Stemline the exclusive right to reference its IND in the event that Stemline decides to file its own IND for SL-401. Stemline may assign the agreement to one of its affiliates, one of its purchasers of all or substantially all of its assets or in connection with a merger, a change in control or a similar transaction.

We note that Scott and White – while perhaps less familiar to investors than institutions like Memorial Sloan-Kettering Cancer Center, the University of Texas' M.D. Anderson Cancer Center, or Johns Hopkins University's Sidney Kimmel Comprehensive Cancer Center – is nonetheless an old and well-respected hospital system. Located in Bell County, TX, Scott & White Memorial Hospital was founded in 1897, when Dr. Arthur C. Scott and Dr. Raleigh R. White, Jr., opened the Temple Sanitarium in Temple, TX. Caring for the heart of Texas between Dallas and Austin, Scott & White, with more than 800 physicians and scientists, is a large multi-specialty group practice. The primary clinical teaching campus of Texas A&M Health Science Center College of Medicine, Scott & White is ranked as one of the top 100 hospitals and one of the top 15 teaching hospitals in the U.S. by Thomson Reuters.

Since the licensing agreement was effected between Scott and White and Stemline Therapeutics, Scott and White has played a key role in adding value to the clinical data package for SL-401. It was at Scott and White that both of the two patients with BPDCN who achieved durable complete responses upon administration of SL-401 were treated. We anticipate that Scott and White may treat more BPDCN patients with SL-401 in the future, and that the hospital is also likely to play a key role in the development of the drug for the treatment of other hematological malignancies.

SL-401 Market Model Overview

We have modeled sales of SL-401 in BPDCN and in post-third-line AML, since we believe that these are the indications in which the vast majority of the clinical proof-of-concept data has been generated. While some investors may be skeptical of ascribing value to the deployment of SL-401 in a disease where only three patients have been treated with the drug – which, we acknowledge, is the case with BPDCN – we would simply point out that there appears to be a very solid mechanistic rationale for the effectiveness of the drug in this disease and that patients with the disorder all express the CD123 antigen on the abnormal cells. Accordingly, therefore, we believe that there is a relatively high likelihood that SL-401 could achieve regulatory approval in the BPDCN indication. We have assumed that Stemline Therapeutics would conduct a single, randomized pivotal study with a single-arm design in roughly 40 patients. This trial could start in late 2013, reach full enrollment in mid-2014, and report sufficient data to permit formal regulatory submission of the drug in late 2014 or early 2015.

Assuming Orphan Drug status (which we regard as a given because of the ultra-rare nature of BPDCN), we expect that a six-month review period could put the drug on the market in the U.S. for BPDCN in late 2015 / early 2016. In our view, European approval could be secured within 12 months of the drug's introduction into the U.S. market. Since this is an ultra-rare disease and median survival is currently only 14 months with existing treatment, we believe that if SL-401 is as effective as the initial patient data showed, it could command pricing of roughly $150,000 per patient annually. This is comparable to pricing for other orphan drugs used to treat other ultra-rare diseases, but it is by no means at the upper end of the scale. For comparison, Soliris (eculizumab), a monoclonal antibody for the treatment of paroxysmal nocturnal hemoglobulinuria (PNH) that is marketed by Alexion Pharmaceuticals, is priced at roughly $500,000 per patient annually.

Stemline has indicated that it plans to advance SL-401 into a 200-patient randomized Phase 2b clinical trial to treat adult relapsed or refractory AML patients who have failed two previous treatments (i.e., third-line AML) with OS as the primary endpoint. Assuming that this trial begins in late 2013, we believe that top-line data could be obtained in early- to mid-2015. If these results turn out to be positive, we expect that Stemline could directly pursue regulatory approval in the U.S. We would expect that the drug could be deployed in post-third-line AML in late 2016 or early 2017. While we believe that it may be difficult to sustain the same pricing in post-third-line AML as may already have been established for BPDCN, we would note that the post-third-line AML setting is typically a difficult indication for drugs and therefore SL-401 would have the advantage of being one of the few therapies available if it is demonstrated to be effective.

In our market model, we have assumed incidence and prevalence of BPDCN based on previously cited estimates, which may under-estimate the frequency with which this disease occurs. In our view, the disorder may exhibit the same characteristics as many other ultra-rare diseases when an effective therapy becomes available – ultra-rare disorders such as Gaucher's disease, Fabry disease and the like all showed a substantial increase in diagnosis frequency after effective therapeutics were introduced, because patients became much more proactive about coming forward and physicians became more active in pinpointing potential cases of these disorders. We do not expect the incidence ever to reach more than a few thousand cases per year, but even these numbers yield substantial market potential. We have utilized 3% year-on-year pricing increases to account for inflation, and we assume that Stemline Therapeutics would market this drug independently for both BPDCN and post-third-line AML because these indications are small enough to be handled by a relatively small sales force. Our assumptions are that Stemline would hire 30 – 40 people in the U.S. and could use distributors in Europe.

Table 5: SL-401 Hematological Malignancies Estimated Sales Model

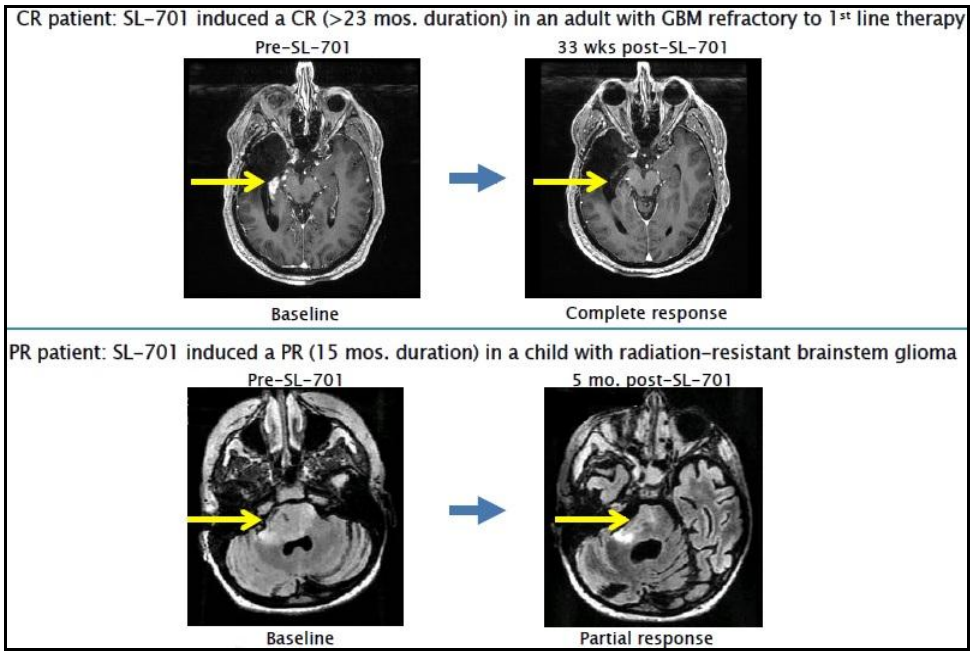
U.S. market	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	Comments
Total patients with hematological malignancies	160,000	164,800	169,744	174,836	180,081	185,484	190,121	194,874	199,746	204,739	209,858	215,104	219,944	224,893	229,953	235,127	240,417	245,226	Total number of patients with blood cancers in the U.S.
Blastic plasmacytoid dendritic cell neoplasm (0.44%)	*704*	*725*	*764*	*804*	*846*	*890*	*932*	*974*	*999*	*1,024*	*1,049*	*1,076*	*1,100*	*1,124*	*1,150*	*1,176*	*1,202*	*1,226*	Number of patients with BPDCN (0.044% of total)
Acute myeloid leukemia (8.7%)	*13,920*	*14,338*	*14,937*	*15,560*	*16,207*	*16,694*	*17,111*	*17,539*	*17,977*	*18,427*	*18,887*	*19,359*	*19,795*	*20,240*	*20,696*	*21,161*	*21,638*	*22,070*	Number of patients diagnosed with AML (8.7% of total)
Post-third line AML	*4,176*	*4,301*	*4,481*	*4,668*	*4,862*	*5,008*	*5,133*	*5,262*	*5,393*	*5,528*	*5,666*	*5,808*	*5,938*	*6,072*	*6,209*	*6,348*	*6,491*	*6,621*	Number of post-third line AML patients (30% of AML patients)
Penetration % (BPDCN)	0%	0%	15%	24%	36%	42%	55%	62%	67%	75%	77%	81%	76%	71%	66%	61%	54%	45%	Peak market penetration rate - ~80% of eligible cases
Patients treated (BPDCN)	0	0	115	193	305	374	512	604	669	768	808	871	836	798	759	717	649	552	Total eligible cases treated
Penetration % (post-third line AML)	0%	0%	0%	0%	5%	15%	21%	29%	34%	36%	38%	40%	42%	37%	34%	28%	23%	16%	Peak market penetration rate - ~40% of eligible cases
Patients treated (post-third line AML)	0	0	0	0	0	751	1,078	1,526	1,834	1,990	2,153	2,323	2,494	2,247	2,111	1,778	1,493	1,059	Total eligible cases treated
Revenue per patient annually	$0	$0	$120,000	$123,600	$127,308	$131,127	$135,061	$139,113	$143,286	$147,585	$152,012	$156,573	$161,270	$166,108	$171,091	$176,224	$181,511	$186,956	Pricing at wholesale acquisition cost: ~$150,000 per patient annually (ref. ultra-rare orphan drug pricing)
Annual sales ($MM)	**0**	**0**	**14**	**24**	**39**	**148**	**215**	**296**	**359**	**407**	**450**	**500**	**537**	**506**	**491**	**440**	**389**	**301**	
RoW (developed countries - mainly Europe)																			
	2013	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021**	**2022**	**2023**	**2024**	**2025**	**2026**	**2027**	**2028**	**2029**	**2030**	
Total patients with hematological malignancies	220,000	226,600	233,398	240,400	247,612	255,040	261,416	267,952	274,651	281,517	288,555	295,769	302,423	309,228	316,185	323,300	330,574	337,185	Total number of patients with blood cancers in Europe
Blastic plasmacytoid dendritic cell neoplasm (0.44%)	*968*	*997*	*1,050*	*1,106*	*1,164*	*1,224*	*1,281*	*1,340*	*1,373*	*1,408*	*1,443*	*1,479*	*1,512*	*1,546*	*1,581*	*1,616*	*1,653*	*1,686*	Number of patients with BPDCN (0.044% of total)
Acute myeloid leukemia (8%)	*17,600*	*18,128*	*18,905*	*19,713*	*20,552*	*21,423*	*22,220*	*22,776*	*23,345*	*23,929*	*24,527*	*25,140*	*25,706*	*26,284*	*26,876*	*27,480*	*28,099*	*28,661*	Number of patients diagnosed with AML (8% of total)
Post-third line AML	*5,280*	*5,438*	*5,672*	*5,914*	*6,166*	*6,427*	*6,666*	*6,833*	*7,004*	*7,179*	*7,358*	*7,542*	*7,712*	*7,885*	*8,063*	*8,244*	*8,430*	*8,598*	Number of post-third line AML patients (30% of AML patients)
Penetration % (BPDCN)	0%	0%	0%	11%	21%	34%	45%	55%	61%	66%	70%	67%	63%	58%	49%	42%	37%	28%	Peak market penetration rate - ~70% of eligible cases
Patients treated (BPDCN)	0	0	0	122	244	416	576	737	838	929	1,010	991	953	897	775	679	612	472	Total eligible cases treated
Penetration % (post-third line AML)	0%	0%	0%	0%	0%	3%	11%	19%	27%	32%	35%	38%	40%	37%	34%	28%	23%	16%	Peak market penetration rate - ~40% of eligible cases
Patients treated (post-third line AML)	0	0	0	0	0	193	733	1,298	1,891	2,297	2,575	2,866	3,085	2,918	2,741	2,308	1,939	1,376	Total eligible cases treated
Revenue per patient annually	$0	$0	$0	$50,000	$51,500	$53,045	$54,636	$56,275	$57,964	$59,703	$61,494	$63,339	$65,239	$67,196	$69,212	$71,288	$73,427	$75,629	Pricing at wholesale acquisition cost: ~$50,000 per patient annually (lower drug pricing ex-U.S.)
Annual sales ($MM)	**0**	**0**	**0**	**6**	**13**	**32**	**72**	**115**	**158**	**193**	**220**	**244**	**263**	**256**	**243**	**213**	**187**	**140**	
Total WW Sales ($MM)	**0**	**0**	**14**	**30**	**51**	**180**	**286**	**411**	**517**	**600**	**671**	**744**	**800**	**762**	**734**	**653**	**576**	**441**	

Source: Company Reports and Aegis Capital Corp. estimates

SL-701 Overview

Originally designed by researchers at the University of Pittsburgh Medical Center, SL-701 is a synthetic peptide-based vaccine aimed at eliciting a potent endogenous immune response against brain cancers, particularly adult glioblastoma multiforme (GBM) and pediatric glioma. SL-701 consists of peptides corresponding to sequences in IL-3Rα2 and EphA2, which are antigens known to be expressed by both tumor bulk and cancer stem cells (CSCs) in brain tumors. The drug was developed with the goal of activating cytotoxic T lymphocytes against the brain tumors and associated CSCs[9]. Thus far, SL-701 has shown durable complete responses (CRs) and multiple partial responses (PRs) along with encouraging survival benefit vs. historical data. SL-701 also appears to be safe and well-tolerated, with negligible side effects including mild injection site reactions and transient fever. We note that the CSC-targeting approach has been posited to have particular relevance in glioma therapy, because these tumors typically regenerate driven by residual tumor stem cell proliferation even if the tumor bulk is completely removed[10].

Figure 17: SL-701 Clinical Proof-of-Concept Activity Data



Source: Stemline Therapeutics, Inc.

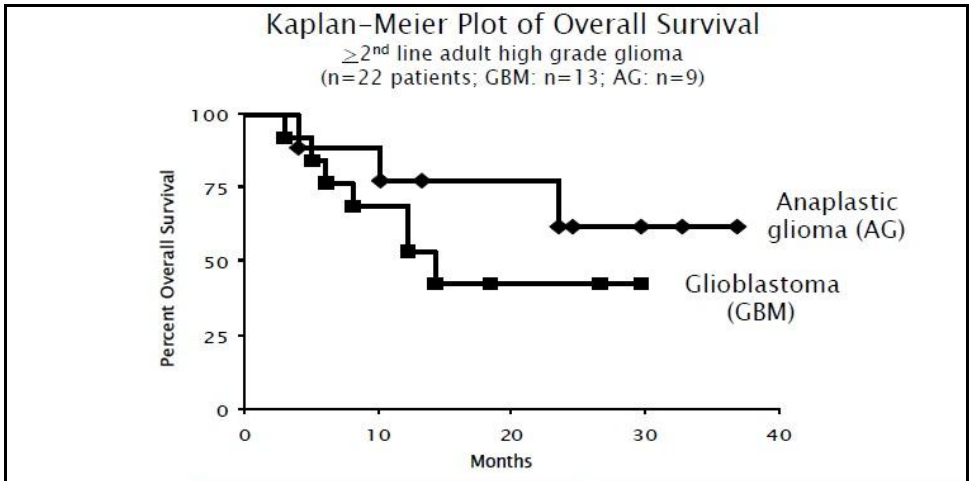
The above radiographic images show two examples of the tumor responses generated by treatment with the vaccine. In the upper set of images, an adult patient's tumor is clearly shown to have regressed to the point where it is no longer visible. In the lower set of images, a pediatric patient with radiation-resistant brainstem glioma is shown to have experienced a partial response of lengthy duration after treatment with SL-701.

In adult high-grade recurrent or refractory glioma, 59% of patients dosed (n=22) showed overall response or disease stabilization. There were two CRs in this population, one of which involved the refractory GBM patient who has remained disease-free for nearly two years (>23 months) and whose tumor response is shown in Figure 17 above. Median overall survival for GBM patients is roughly 5 – 7 months. In comparison to SL-701, patients with refractory GBM who receive Avastin® – the last line of defense for such cases – only show survival prolongation of 1 – 2 months.

[9] Zhu *et al*., Journal of Translational Medicine 12: 5-10 (2007)
[10] Ghebeh *et al*., Hematology / Oncology & Stem Cell Therapy 1: 1-2 (2008)

SL-701 has also shown impact in pediatric glioma patients (n=22), with 86% exhibiting overall response or disease stabilization. There were three PRs, with two showing >14 months' duration. One patient showed prolonged (20-month) disease-free survival on SL-701 post-surgery. Finally, there were four patients with stable disease who survived >13 months after receiving SL-701. Thus, while we believe that the data seen thus far with SL-701 is still comparatively early-stage, we consider it encouraging, indicative of the activity of the vaccine and validation of the overall approach. The Kaplan-Meier plots shown below indicate the magnitude of the survival benefit in adult high-grade glioma, and the table beneath the chart indicates how substantial the difference was between SL-701-treated patients and historical controls. Median OS was doubled for patients with GBM and anaplastic glioma; in our view, this is very encouraging.

Figure 18: SL-701 Survival Benefit Comparison Data



	GBM		AG	
	SL-701	Historical	SL-701	Historical
Median OS	13 months	5-7 months	>22 months	9-11 months
6 month OS	80%	38-55%	85%	66%
12 month OS	55%	14-25%	76%	44-47%

Source: Stemline Therapeutics, Inc.; National Cancer Institute

Stemline Therapeutics has stated that it plans to manufacture and formulate SL-701 as a mixture of IL-13Rα2, EphA2 and a helper peptide. In the 701 Ped-G and 701 Adult-RHGG Studies, SL-701 (which is comprised of IL-13Rα2 and EphA2 synthetic peptides) was mixed with additional peptides, including YKL-40 and GP-100 peptides in the adult study, and survivin peptide in the pediatric study. Given the clinical anti-tumor activity observed in both trials, Stemline's research team believe that the IL-13Ra2 and EphA2 peptides – the common feature of both trials – represent the active components of the product. Thus, the firm thinks that SL-701 need not be mixed with any other peptides for clinical activity. Accordingly, while Stemline plans continued evaluation of additional peptides, the firm plans to advance SL-701 into future trials without additional peptides.

In addition, Stemline is working on modification of the administration regimen of SL-701 to include a more commercially available and viable adjuvant. An adjuvant is a substance administered to a patient to potentially help enhance the patient's immune response to a vaccine. These changes may impact the effectiveness of the vaccine and we cannot predict what this impact may be in future clinical trials. In addition, we would note that – as was the case with SL-401 – Stemline Therapeutics is planning in future to take control of the clinical development of SL-701 and file its own INDs. Previously, both SL-401 and SL-701 were being studied under investigator-initiated INDs.

SL-701 Market Model Overview

We have modeled sales of SL-701 in adult glioblastoma (GBM) and pediatric glioma, since these are the indications that Stemline has stated it aims to target with this drug. Since SL-701 was designed specifically to address brain cancers, we do not anticipate that it is likely to see usage beyond these niche indications. Furthermore, since brain cancers in general are very difficult to address, we are assuming that benefits likely to be seen with the drug are not going to be similar to those that could be achieved by SL-401. As per company guidance, we have assumed that Stemline Therapeutics would conduct additional proof-of-concept development work with SL-701 prior to petitioning for regulatory approval of the drug in the U.S. and Europe. In the pediatric indication, the firm aims to initiate a multi-center NIH-funded trial in pediatric malignant non-brainstem and brainstem glioma, while seeking a Special Protocol Assessment (SPA) for a pivotal Phase 2b trial, which would probably be a 100-patient, randomized study. In the adult GBM indication, Stemline anticipates initially performing an open-label proof-of-concept trial in roughly 30 – 40 patients. If this data proves to be positive, the firm contemplates conducting a Phase 2b trial in adult patients with 2nd-line GBM, which would compare SL-701 + Avastin® to Avastin® alone. This study is likely to be a 200-patient trial. The firm also aims to continue all ongoing investigator-sponsored grant-funded trials in both newly diagnosed and recurrent adult low-grade gliomas. Such studies, if positive, could pave the way for the expansion of use for SL-701 into broader niche populations within the brain cancer domain. We believe Stemline could start both the pediatric glioma Phase 2b study and the adult GBM proof-of-concept study in 2014, reach full enrollment in mid- to late 2015, and report sufficient data to permit formal regulatory submission of the drug in early- to mid-2016 for pediatric glioma and in early to mid-2018 for adult GBM. Given filing of a Biologics License Application (BLA) and Priority Review, we would anticipate U.S. approval of the drug in late 2016 or early 2017 for pediatric glioma and in late 2018 / early 2019 for adult GBM.

In our view, European approval could be secured within 12 months of the drug's introduction into the U.S. market. Since these are rare diseases and existing therapeutic agents do not provide substantial survival benefit, we believe that SL-701 could command pricing of roughly $25,000 per patient annually in the U.S. and $15,000 per patient annually in Europe. This is comparable to pricing for various other anti-cancer agents, but still cheaper than Avastin®, which typically costs $40,000 – $50,000 annually. In our market model, we have assumed incidence and prevalence of brain cancer based on estimates that quote incidence of approximately 50,000 cases per year in the U.S. and roughly 65,000 per year in Europe. Within this context, it is estimated that adult glioblastoma afflicts roughly 10,000 individuals per year in the U.S. and 16,000 – 18,000 in Europe. We have utilized 3% year-on-year pricing increases to account for inflation, and we assume that Stemline Therapeutics would market this drug independently in the U.S. market for both adult GBM and pediatric glioma because these indications are small enough to be handled by a relatively minimal sales force.

Our assumptions are that Stemline would utilize 10 – 15 people in the U.S. and use distributors in Europe in order to commercialize the drug. We have assumed a 20% discount rate, reflecting what we perceive as higher risk to commercialization in brain cancer than would be the case for SL-401. Furthermore, since there is less data supporting the efficacy of SL-701 and less mechanistic evidence, we have ascribed only a 50% probability of eventual success to this candidate. We have applied a 40% tax rate to all future cash flows, and also project net margins of roughly 50%, which we consider conservative given the relatively limited marketing resources that we believe Stemline would require. While we do not consider SL-701 as substantial a value driver as SL-401, at this juncture we believe that the market is not ascribing any value to this agent and thus investors can treat it as a free call option.

Table 6: SL-701 Brain Cancer Estimated Sales Model

U.S. market	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	Comments
Total patients with brain cancer	48,000	49,440	50,923	52,451	54,024	55,645	57,036	58,462	59,924	61,422	62,957	64,531	65,983	67,468	68,986	70,538	72,125	73,568	Total number of patients with brain cancer in the U.S.
Adult glioblastoma (21%)	*10,032*	*10,382*	*10,694*	*11,015*	*11,345*	*11,685*	*11,978*	*12,277*	*12,584*	*12,899*	*13,221*	*13,552*	*13,856*	*14,168*	*14,487*	*14,813*	*15,146*	*15,449*	Number of patients with glioblastoma (21% of total)
Pediatric glioma (5%)	*2,400*	*2,472*	*2,546*	*2,623*	*2,701*	*2,782*	*2,852*	*2,923*	*2,996*	*3,071*	*3,148*	*3,227*	*3,299*	*3,373*	*3,449*	*3,527*	*3,606*	*3,678*	Number of pediatric glioma patients (5% of total)
Penetration % (adult glioblastoma)	0%	0%	0%	0%	0%	3%	14%	21%	27%	30%	27%	21%	19%	16%	12%	9%	7%	3%	Peak market penetration rate - ~30% of eligible cases
Patients treated (adult glioblastoma)	0	0	0	0	0	351	1,677	2,578	3,398	3,870	3,504	2,846	2,633	2,267	1,738	1,333	1,060	463	Total eligible cases treated
Penetration % (pediatric glioma)	0%	0%	0%	4%	16%	21%	24%	27%	32%	37%	40%	38%	35%	27%	23%	16%	11%	8%	Peak market penetration rate - ~40% of eligible cases
Patients treated (pediatric glioma)	0	0	0	105	432	584	684	789	959	1,136	1,259	1,226	1,155	911	793	564	397	294	Total eligible cases treated
Revenue per patient annually	$0	$0	$0	$25,000	$25,750	$26,523	$27,318	$28,138	$28,982	$29,851	$30,747	$31,669	$32,619	$33,598	$34,606	$35,644	$36,713	$37,815	Pricing at wholesale acquisition cost: ~$25,000 per patient annually (ref. late-stage cancer drug pricing)
Annual sales ($MM)	**0**	**0**	**0**	**3**	**11**	**25**	**65**	**95**	**126**	**149**	**146**	**129**	**124**	**107**	**88**	**68**	**53**	**29**	
RoW (developed countries - mainly Europe)																			
	2013	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**	**2021**	**2022**	**2023**	**2024**	**2025**	**2026**	**2027**	**2028**	**2029**	**2030**	
Total patients with brain cancer	65,000	66,950	68,959	71,027	73,158	75,353	77,237	79,168	81,147	83,175	85,255	87,386	89,352	91,363	93,418	95,520	97,670	99,623	Total number of patients with brain cancer in Europe
Adult glioblastoma (25%)	*16,250*	*16,738*	*17,240*	*17,757*	*18,290*	*18,838*	*19,309*	*19,792*	*20,287*	*20,794*	*21,314*	*21,847*	*22,338*	*22,841*	*23,355*	*23,880*	*24,417*	*24,906*	Number of patients with glioblastoma (25% of total)
Pediatric glioma (4%)	*2,600*	*2,678*	*2,758*	*2,841*	*2,926*	*3,014*	*3,089*	*3,167*	*3,246*	*3,327*	*3,410*	*3,495*	*3,574*	*3,655*	*3,737*	*3,821*	*3,907*	*3,985*	Number of pediatric glioma patients (4% of total)
Penetration % (adult glioblastoma)	0%	0%	0%	0%	0%	0%	4%	11%	16%	18%	20%	17%	14%	12%	9%	7%	5%	3%	Peak market penetration rate - ~20% of eligible cases
Patients treated (adult glioblastoma)	0	0	0	0	0	0	772	2,177	3,246	3,743	4,263	3,714	3,127	2,741	2,102	1,672	1,221	747	Total eligible cases treated
Penetration % (pediatric glioma)	0%	0%	0%	0%	5%	11%	14%	17%	21%	25%	28%	30%	25%	19%	15%	8%	6%	4%	Peak market penetration rate - ~30% of eligible cases
Patients treated (pediatric glioma)	0	0	0	0	146	332	433	538	682	832	955	1,049	894	694	561	306	234	159	Total eligible cases treated
Revenue per patient annually	$0	$0	$0	$0	$15,000	$15,450	$15,914	$16,391	$16,883	$17,389	$17,911	$18,448	$19,002	$19,572	$20,159	$20,764	$21,386	$22,028	Pricing at wholesale acquisition cost: ~$15,000 per patient annually (lower drug pricing ex-U.S.)
Annual sales ($MM)	**0**	**0**	**0**	**0**	**2**	**5**	**19**	**45**	**66**	**80**	**93**	**88**	**76**	**67**	**54**	**41**	**31**	**20**	
Total WW Sales ($MM)	**0**	**0**	**0**	**3**	**13**	**30**	**84**	**139**	**193**	**229**	**240**	**217**	**200**	**174**	**141**	**109**	**85**	**49**	

Source: Company Reports and Aegis Capital Corp. estimates

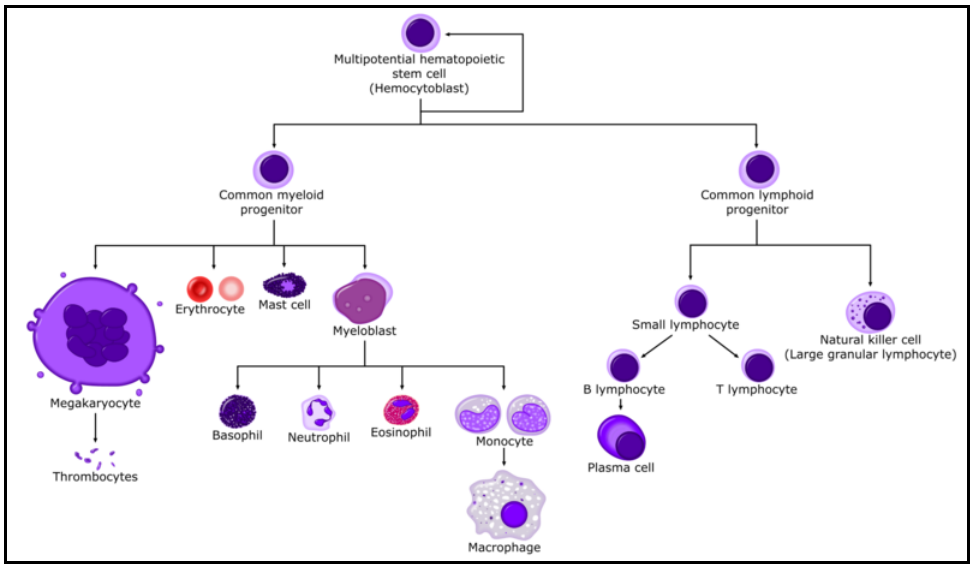
Competitive Landscape

In this section, we assess the competitive landscape across the three major fronts on which we believe that Stemline Therapeutics is likely to be competing for the foreseeable future. These comprise: a) hematological malignancies, largely targeted by SL-401; b) brain cancers, exclusively targeted by SL-701; and c) cancer stem cell-focused approaches, wherein the company is likely to come up against firms like Verastem, which has a competing platform aimed at identifying and developing anti-CSC therapeutics.

Hematological Malignancies – Overview

Collectively, these types of cancers are termed "liquid tumors" because they all afflict cells that migrate around the body in the bloodstream. Hematological malignancies affect all the major immune cell types – T lymphocytes, B lymphocytes and plasma cells. In the immune system, the different cell types mature according to the lineage schematic shown below. T cells directly attack invading pathogens and can kill them through the release of pro-inflammatory cytokines (cytotoxic T cells). Other T cell types, such as the regulatory and memory T cells, perform support roles and facilitate communication between different immune system components. B cells play a key role in antigen presentation – displaying foreign protein sequences to T cells so that the T cells can recognize pathogens and distinguish them from "self" antigens – proteins normally expressed by the body's own tissues. Plasma cells represent the most mature form of B cell, which produces large volumes of antibodies. These cell types coordinate to provide immunological protection against bacteria, viruses and other harmful agents.

Figure 19: Hematopoietic Cell Lineages



Source: Scientific American

The lineage schematic shows how all blood cells – whether red blood cells, which carry oxygen, or white blood cells, which perform immune system functions – arise from a common multipotent progenitor cell (hemocytoblast). Myeloid cell progenitors that differentiate from the hemocytoblast progenitors give rise to both red blood cells and monocytes and macrophages. Lymphoid progenitors give rise to B and T lymphocytes and natural killer cells. Hematological malignancies are generally classified into three main types – leukemias, which affect T cells; lymphomas, which affect B cells; and myelomas, which affect plasma cells. Leukemias are so called because the cells that they affect are trafficked in the blood stream (leukocytes). Lymphomas are so called because

they affect cells in the lymph nodes (lymphocytes). These blood cancers collectively constitute approximately 10% of all cancers in the U.S. and may represent slightly higher or lower proportions of all cancers in other geographies, depending upon epidemiological and environmental factors. The table shown below provides a basic breakdown of the classification of the major blood cancer types.

Table 7: Blood Cancer Classification

Cancer Type	Indication Name	Cell Type Affected	Prevalence
Leukemia	Acute lymphocytic leukemia (ALL)	T lymphocytes	4.0%
Leukemia	Acute myeloid leukemia (AML)	T lymphocytes	8.7%
Leukemia	Chronic lymphocytic leukemia (CLL)	T lymphocytes	10.2%
Leukemia	Chronic myelogenous leukemia (CML)	T lymphocytes	3.7%
Leukemia	Other leukemias	T lymphocytes	2.8%
Lymphoma	Hodgkin's lymphoma (HL)	B lymphocytes	7.0%
Lymphoma	Non-Hodgkin's lymphoma (NHL)	B lymphocytes	55.6%
Myeloma	Multiple myeloma	Plasma cells	8%

Source: National Cancer Institute

These cancers primarily occur in adults – for example, over 90% of all cases of leukemia affect adult patients. Leukemias are mainly characterized by a massive increase in the numbers of immature white blood cells, which are known as "blasts." These cells can over-proliferate to such an extent that the excess in their numbers can cause the blood of the patient to appear whitish in color. Thus, their presence can easily be seen in a blood sample. Common symptoms of leukemias include loss of appetite, weight loss, fever, night sweats, chills, frequent infections, muscle weakness, skin discoloration, and liver and spleen enlargement. Acute forms of leukemia are often treatable using many different types of strategies, but chronic lymphocytic leukemia is generally considered untreatable using existing widely-deployed drugs.

Unlike leukemias, which primarily involve mobile cells in the bloodstream, lymphomas typically manifest as solid tumors in the lymph nodes. They occur when certain types of immune cells begin behaving abnormally, usually by exhibiting substantially heightened proliferation rates. While lymphomas can often be localized to individual lymph nodes, they can also metastasize very easily and spread into other organs.

Although some forms of lymphoma are characterized as indolent – i.e. slow-growing – others are highly invasive, rapidly growing, and aggressively malignant. Most forms of lymphoma, however, are typically treatable and can often be cured if detected at an appropriate stage. Symptoms are similar to those seen in leukemias, with the addition of lymph node swelling.

Finally, myelomas involve plasma cells, which are found primarily concentrated in bone marrow. The abnormal cells in patients with myeloma typically aggregate in the bone marrow and the principal symptoms include elevated calcium, renal function impairment or failure, anemia, and bone lesions – which can be excruciatingly painful. Although myelomas are considered treatable, the average life expectancy for a patient with myeloma is roughly five to seven years with standard of care.

The main treatment modality for myelomas involves an autologous stem cell transplant, preceded by very high-dose aggressive chemotherapy to kill as many of the abnormal cells in the bone marrow as possible. The autologous cell transplant is designed to replenish the depleted immune system with normal blood cell progenitors, which would ideally repopulate the plasma cell population. Nevertheless, this treatment approach seldom offers a permanent solution, and recurrence rates after bone marrow transplants are often associated with accelerated disease progression, poor prognosis and death.

Hematological Malignancies – Drug Therapy

We note that there are currently many drugs on the market to treat hematological malignancies, and there has been a spate of approvals in this arena over the course of the past decade. Targeted therapies became extremely popular in the wake of the success of Gleevec (imatinib mesylate), a drug introduced by Novartis in 2003. Now the standard of care for patients with chronic myelogenous leukemia (CML), Gleevec – subsequent to its initial approval – has broadened its reach into other cancer types such as gastrointestinal stromal tumors (GIST) and myelodysplastic syndromes involving Philadelphia chromosome translocations. Other agents similar to Gleevec include Tasigna (nilotinib), also made by Novartis, and Sprycel (dasatinib), introduced by Bristol-Myers Squibb.

Myelodysplastic syndrome (MDS) is often treated with hypomethylating agents like Vidaza (azacitidine) and Dacogen (decitabine); multiple myeloma is being addressed with proteasome inhibitors like Velcade (bortezomib) and Kyprolis (carfilzomib), along with the thalidomide family of drugs – Thalomid (thalidomide), Revlimid (lenalidomide), and Pomalyst (pomalidomide). Thalomid, Revlimid and Pomalyst are all made by Celgene, which is arguably the world's most successful blood cancer company.

The older-generation drugs – nitrogen mustard alkylating agents, anti-folates, topoisomerase inhibitors, nucleoside analogs, anthracycline DNA intercalating agents, and tubulin-binding compounds – remain popular. These drugs are often deployed as the first line of defense and are typically applied in combination. During the early years of chemotherapy application, it became *de rigueur* to apply different chemotherapeutic drugs with different mechanisms of action simultaneously in the same patient. Many of the drugs now used to treat hematological malignancies are listed overleaf in Table 8.

Some of the more popular chemotherapy drug "cocktails" used today in the hematological malignancy setting include ABVD (Adriamycin, bleomycin, vinblastine and dacarbazine), CBV (cyclophosphamide, carmustine and etoposide), CHOP (cyclophosphamide, doxorubicin, vincristine – under the name Oncovin – and prednisone), R-CHOP (CHOP plus the antibody agent Rituxan), COPP (cyclophosphamide, vincristine, procarbazine and prednisone), CVAD (cyclophosphamide, vincristine, Adriamycin and dexamethasone), EPOCH (etoposide, prednisone, vincristine, cyclophosphamide and doxorubicin), MACOP-B (methotrexate, leucovorin, Adriamycin, cyclophosphamide, vincristine, prednisone and bleomycin), POMP (mercaptopurine, vincristine, methotrexate and prednisone), Thal/Dex (thalidomide and dexamethasone), and VAD (vincristine, Adriamycin and dexamethasone). The combination of older-generation chemotherapeutic drugs and next-generation targeted therapies has also begun gaining in popularity.

As targeted therapies have become more popular, many of which seek to avoid the toxic side effects of broad-spectrum chemotherapy, some of these chemotherapy regimens have seen their usage decline. The main drawback of the simultaneous application of multiple chemotherapeutic drugs in the same patient is the fact that the side effect profiles of these drugs are toxic enough when they are applied as single agent therapies, and in combination the side effect burdens of some of these cocktails can be horrendous. As such, therefore, we believe the advent of agents like SL-401 may potentially be advantageous in the context of these older-generation regimens.

We would note that the field of hematological malignancies is very crowded from the viewpoint of drug development. Thus, SL-401 may have a challenging road ahead when it comes to broadening usage beyond AML and BPDCN. For this reason, we believe that the strategy being employed by Stemline Therapeutics – focusing on niche indications wherein there is less competition – is a productive one. On Table 9, we have listed many of the currently-active drug development projects ongoing in various blood cancers.

Table 8: Hematological Malignancies – Current Therapies

Drug Name	Generic Name	Company	Patent Expiry	Primary Target Indication	Drug Class	Mechanism of Action
Velsar	vinblastine	various	Expired	Leukemia / lymphoma	Tubulin-binding agent	Microtubule disruption
Vincasar	vincristine	various	Expired	Leukemia / lymphoma	Tubulin-binding agent	Microtubule disruption
Cerubidine / Rubidomycin	daunorubicin	various	Expired	Acute lymphoblastic leukemia	Anthracycline	DNA intercalation
DepoCyt	cytarabine	various	Expired	Leukemia / lymphoma	Nucleoside analog	DNA synthesis interference
Folex	methotrexate	various	Expired	Leukemia / lymphoma	Antifolate agent	Anti-metabolic agent
Clafen / Cytoxan / Neosar	cyclophosphamide	various	Expired	Leukemia / lymphoma	Alkylating agent	DNA synthesis interference
Etopophos / Toposar / VePesid	etoposide	various	Expired	Acute lymphoblastic leukemia	Topoisomerase inhibitor	DNA synthesis interference
Lanvis	thioguanine	various	Expired	Acute lymphoblastic leukemia	Thiopurine agent	Cytostatic agent
NA	mercaptopurine	various	Expired	Acute lymphoblastic leukemia	Thiopurine agent	Immunosuppression
Adriamycin	doxorubicin HCl	various	Expired	Non-Hodgkin's lymphoma	Anthracycline	DNA intercalation
Idamycin	idarubicin	various	Expired	Acute lymphoblastic leukemia	Anthracycline	DNA intercalation
Trisenox	arsenic trioxide	various	Expired	Acute lymphoblastic leukemia	Cytostatic agent	Blockade of cell growth
Ambochlorin	chlorambucil	various	Expired	Acute lymphoblastic leukemia	Alkylating agent	DNA synthesis interference
Leucovorin	folinic acid	various	Expired	Leukemia / lymphoma	Folic acid derivative (adjuvant)	Bone marrow protection
Fusilev	levoleucovorin	Spectrum Pharmaceuticals	December 2019	Leukemia / lymphoma	Folic acid derivative (adjuvant)	Bone marrow protection
Rituxan	rituximab	Biogen Idec / Roche	May 2015	Non-Hodgkin's lymphoma	Anti-CD20 antibody	B cell ablating antibody
Adcetris	brentuximab vedotin	Seattle Genetics	2026	Hodgkin's lymphoma	Anti-CD30 armed antibody	Selective antibody-drug conjugate
Arzerra	ofatumumab	GlaxoSmithKline / Genmab	2023	Chronic lymphocytic leukemia	Anti-CD20 antibody	B cell ablating antibody
Treanda	bendamustine HCl	Teva Pharmaceutical Industries	September 2015	Non-Hodgkin's lymphoma	Alkylating agent	DNA synthesis interference
Fludara	fludarabine	various	Expired	Leukemia / lymphoma	Nucleoside analog	DNA synthesis interference
Bosulif	bosutinib	Pfizer	November 2026	Chronic myelogenous leukemia	Kinase inhibitor	Cell proliferation blocker
Sprycel	dasatinib	Bristol-Myers Squibb	June 2020	Chronic myelogenous leukemia	Kinase inhibitor	Cell proliferation blocker
Gleevec	imatinib mesylate	Novartis AG	July 2015	Chronic myelogenous leukemia	Kinase inhibitor	Cell proliferation blocker
Tasigna	nilotinib	Novartis AG	July 2023	Chronic myelogenous leukemia	Kinase inhibitor	Cell proliferation blocker
Synribo	omacetaxine mepesuccinate	Teva Pharmaceutical Industries	June 2023	Chronic myelogenous leukemia	Kinase inhibitor	Cell proliferation blocker
Iclusig	ponatinib HCl	Ariad Pharmaceuticals	NA	Chronic myelogenous leukemia	Kinase inhibitor	Cell proliferation blocker
Velcade	bortezomib	Takeda Pharmaceutical Co. Ltd.	January 2022	Multiple myeloma	Proteasome inhibitor	Protein breakdown blockade
Kyprolis	carfilzomib	Onyx Pharmaceuticals	December 2027	Multiple myeloma	Proteasome inhibitor	Protein breakdown blockade
Zolinza	vorinostat	Merck & Co.	March 2027	Peripheral T cell lymphoma	Hydroxamate	Histone deacetylase inhibitor
Folotyn	pralatrexate	Spectrum Pharmaceuticals	July 2022	Peripheral T cell lymphoma	Antifolate agent	DNA synthesis interference
Zevalin	ibritumomab tiuxetan	Spectrum Pharmaceuticals	February 2016	Non-Hodgkin's lymphoma	Armed antibody	Selective radioablation
Istodax	romidepsin	Celgene Corporation	August 2021	Peripheral T cell lymphoma	Depsipeptide agent	Histone deacetylase inhibitor
Revlimid	lenalidomide	Celgene Corporation	April 2027	Multiple myeloma	Thalidomide analog	Cell proliferation / growth inhibitor
Thalomid	thalidomide	Celgene Corporation	2018	Multiple myeloma	Thalidomide agent	Cell proliferation / growth inhibitor
Pomalyst	pomalidomide	Celgene Corporation	August 2024	Multiple myeloma	Thalidomide analog	Cell proliferation / growth inhibitor
Vidaza	azacitidine	Celgene Corporation	Expired	Myelodysplastic syndrome	Hypomethylating agent	Gene expression regulator (epigenetic)
Dacogen	decitabine	Eisai / Astex Pharmaceuticals	May 2013	Myelodysplastic syndrome	Hypomethylating agent	Gene expression regulator (epigenetic)

Source: Company Reports and Aegis Capital Corp. estimates

Table 9: Hematological Malignancies – Experimental Drug Candidates

Drug Name	Generic Name	Company	Target Indication	Drug Class	Mechanism of Action	Status
Zaltrap / Eylea	aflibercept	Regeneron Pharmaceuticals	Acute lymphoblastic leukemia	Fusion protein	VEGF-binding fusion trap	Phase 3
HuLuc63 / BMS901608	elotuzumab	Bristol-Myers Squibb	Multiple myeloma	Antibody	Anti-CS1 monoclonal antibody	Phase 3
PCI-32765	ibrutinib	Pharmacyclics / Johnson & Johnson	Chronic lymphocytic leukemia	Bruton's tyrosine kinase blocker	Cell division signal blocker	Phase 3
GS-1101 / CAL-101	idelalisib	Gilead Sciences	Chronic lymphocytic leukemia	Kinase inhibitor	Cell division signal blocker	Phase 3
LBH-589	panobinostat	Novartis AG	Multiple myeloma	Hydroxamate	Histone deacetylase blocker	Phase 3
MLN9708	ixazomib citrate	Takeda Pharmaceutical Co. Ltd.	Multiple myeloma	Proteasome inhibitor	Protein breakdown pathway blocker	Phase 3
CNTO 328	siltuximab	Johnson & Johnson	Multiple myeloma	Antibody	Anti-interleukin 6 (anti-IL-6) antibody	Phase 3
MLN9708	alisertib	Takeda Pharmaceutical Co. Ltd.	Diffuse large B cell lymphoma / T cell lymphoma	Aurora kinase inhibitor	Cell cycle inhibitor (Aurora A kinase)	Phase 3
BI 6727	volasertib	Boehringer Ingelheim	Acute myeloid leukemia	Polo-like kinase 1 inhibitor	Dihydropteridinone derivative	Phase 3
CYC682	sapacitabine	Cyclacel Pharmaceuticals	Acute myeloid leukemia	Nucleoside analog prodrug	DNA synthesis inhibitor	Phase 3
SL-401	NA	Stemline Therapeutics	Blastic plasmacytoid dendritic cell neoplasm (BPDCN) Acute myeloid leukemia (AML)	Armed antibody	Anti-interleukin 3 receptor (anti-IL3R) diphtheria toxin-armed antibody	Phase 2/3
ARQ197	tivantinib	ArQule / Daiichi Sankyo	Multiple myeloma	Kinase inhibitor	c-Met inhibitor	Phase 2
IPH2102 / BMS986015	lirilumab	Innate Pharma / Bristol-Myers Squibb	Acute myeloid leukemia	Anti-KIR antibody	Natural killer cell function regulation	Phase 2
MK2206	NA	Merck & Co.	Diffuse B cell lymphoma	Kinase inhibitor	Akt inhibitor	Phase 2
Kinevet	masitinib	AB Science	Multiple myeloma	Kinase inhibitor	c-Kit / PDGFαR / FGFR inhibitor	Phase 2
Targretin	bexarotene	Valeant Pharmaceuticals International	Acute myeloid leukemia	Synthetic retinoid	Cell growth inhibitor / cell death inducer	Phase 2
TH-302	nitroazole prodrug	Threshold Pharmaceuticals	Multiple myeloma	Alkylating agent	Hypoxia-activated cytotoxic prodrug	Phase 2
ABT-888	veliparib	Abbott Laboratories	Multiple myeloma	PARP inhibitor	PARP1 / PARP2 blockade	Phase 2
CHR-2797	tosedostat	Cell Therapeutics	Acute myeloid leukemia	Chemotherapy agent	Aminopeptidase inhibitor	Phase 2
Zarnestra	tipifarnib	Johnson & Johnson	Acute myeloid leukemia	Farnesyltransferase inhibitor	Ras kinase pathway blocker	Phase 2
SNDX-275	entinostat	Syndax	Acute myeloid leukemia	Multi-HDAC inhibitor	Histone deacetylase blocker	Phase 2
CMC-544	inotuzumab ozogamicin	Pfizer	Acute lymphocytic leukemia	Armed antibody	Anti-CD22 calicheamicin-linked antibody	Phase 2
E7070	chloro-indolyl sulfonamide	Eisai Inc.	Acute myeloid leukemia / Myelodysplastic syndrome	Cell cycle inhibitor	Cell cycle / amino acid transport blocker	Phase 2
CP-868-596	crenolanib	Arog Pharmaceuticals	Acute myeloid leukemia	Kinase inhibitor	PDGFαR / PDGFβR inhibitor	Phase 2
ACE-011	sotatercept	Acceleron	Myelodysplastic syndrome	Activin receptor antagonist	Chimeric activin sequestrator	Phase 2
PF-04449913	NA	Pfizer	Acute myeloid leukemia	Hedgehog pathway blocker	Smoothened protein antagonist	Phase 1/2
TL-32711	birinapant	TetraLogic Pharmaceuticals	Acute myeloid leukemia	Smac peptidomimetic	Apoptosis-blocking protein inhibitor	Phase 1/2
ARRY-520	NA	Array Biopharma	Multiple myeloma	Mitosis blocker	Kinesin spindle protein (KSP) inhibitor	Phase 1/2
HMR-1275	alvocidib / flavopiridol	Sanofi S.A.	Chronic lymphocytic leukemia	Cell cycle inhibitor	Cyclin-dependent kinase blocker	Phase 1/2
Vargatef™ / BIBF 1120	NA	Boehringer Ingelheim	Acute myeloid leukemia	Kinase inhibitor	Angiokinase blocker	Phase 1/2
HuMax-CD38	daratumumab	Genmab / Johnson & Johnson	Multiple myeloma	Antibody	Anti-CD38 antibody	Phase 1/2
PLX3397	NA	Plexxikon (Daiichi Sankyo)	Acute myeloid leukemia	Kinase inhibitor	FMS / c-Kit / FLT3 kinase inhibitor	Phase 1/2
AC220	quizartinib	Ambit Biosciences	Acute myeloid leukemia / Myelodysplastic syndrome	Kinase inhibitor	FLT3 tyrosine kinase inhibitor	Phase 1/2
ALT-801	NA	Altor Bioscience	Multiple myeloma	Fusion protein	IL-2 / T cell receptor fusion protein	Phase 1/2
CC-223	NA	Celgene Corporation	Multiple myeloma	Dual mTOR inhibitor	Selective ATP-competitive mTOR blocker	Phase 1/2
ACY-1215	rocilinostat	Acetylon Pharmaceuticals	Multiple myeloma	HDAC6 inhibitor	Histone deacetylase blocker	Phase 1/2
PRI-724	crenolanib	Prism Pharma Co. Ltd	Acute myeloid leukemia / Chronic myeloid leukemia	CBP / β-catenin inhibitor	Wnt cell division signal pathway blocker	Phase 1/2
BT-062	NA	Biotest AG	Multiple myeloma	Armed antibody	Anti-CD138 antibody-drug conjugate	Phase 1/2
SGI-110	decitabine prodrug	Astex Pharmaceuticals	Acute myeloid leukemia / Myelodysplastic syndrome	Hypomethylating agent	Gene methylation regulator (epigenetic)	Phase 1/2
AT7519	NA	Astex Pharmaceuticals / Novartis AG	Multiple myeloma	Cell cycle inhibitor	Cyclin-dependent kinase blocker	Phase 1/2
MOR03087	NA	MorphoSys	Multiple myeloma	Antibody	Anti-CD38 antibody	Phase 1/2
AKN-028	NA	Akinion Pharmaceuticals	Acute myeloid leukemia	Kinase inhibitor	FLT3 / c-Kit tyrosine kinase inhibitor	Phase 1/2
Actimab-A™	lintuzumab-Ac225	Actinium Pharmaceuticals	Acute myeloid leukemia	Armed antibody	Anti-CD33 radioactively armed antibody	Phase 1/2

Source: Company Reports and Aegis Capital Corp. estimates

Brain Cancers – Overview

Tumors occurring in the central nervous system (CNS) are typically classified as brain cancers. While some brain cancers originate within the brain tissue itself, they can also originate initially in lymphatic tissue, in blood vessels, in the cranial nerves, in the brain envelopes (meninges), skull, pituitary gland, or pineal gland, and migrate deeper into brain tissue. Within the brain itself, the involved cells may be neurons or glial cells (which include astrocytes, oligodendrocytes, and ependymal cells). Brain tumors may also spread from cancers primarily located in other organs (metastatic tumors).

Any brain tumor is inherently serious and life-threatening because of its invasive and infiltrative character in the limited space of the intracranial cavity. These types of cancers are often difficult to diagnose because of the extent of brain protection afforded by the skull, and in many cases they are diagnosed at a relatively late stage when the tumor is so entrenched that surgery cannot be attempted. Early-stage brain cancer can be successfully addressed through surgery to remove the tumors, as long as these are in a relatively accessible part of the brain; proximity to any regions that regulate vital functions can preclude removal.

Table 10: Brain Cancer Classification

Cancer Type	Indication Name	Cell Type Affected	Prevalence
Glioma	Ependymomas	Ependymal cells	52.4%
Glioma	Astrocytomas	Astrocytes	
Glioma	Oligodendrogliomas	Oligodendrocytes	
Glioma	Mixed gliomas	Multiple types	
Meningiomas	Meningotheliomatous tumors	Epithelial	20.8%
Meningiomas	Mixed meningiomas	Epithelial	
Meningiomas	Fibrous meningiomas	Epithelial	
Meningiomas	Psammomatous	Epithelial	
Adenomas	Pituitary adenomas	Pituitary gland	17.2%
Nerve sheath tumors	Malignant schwannoma	Schwann cells	9.6%
Lymphoma	Neurofibrosarcoma	Multiple types	
Myeloma	Neurosarcoma	Neurons	

Source: National Cancer Institute

Brain Cancers – Drug Therapy

There are relatively few drugs available to treat brain cancer. Due to its often highly aggressive nature and recalcitrance to treatment with systemic doses of chemotherapeutic drugs, malignant brain cancer is generally considered a death sentence. A standardized chemotherapy regimen is available, known as PCV (procarbazine, CeeNu, and vincristine). CeeNu, or lomustine, is a nitrogen mustard alkylating agent. Finally, Temodar (temozolomide), a newer alkylating agent, has been used for the past several years based on data released in 2007 showing that in Grade IV astrocytoma patients, the drug quadrupled the number of patients alive after four years when combined with radiation therapy vs. radiation therapy alone. Temodar, marketed by Merck & Co. (formerly Schering-Plough), is slated to lose patent protection in the summer of 2013.

Other drugs that have recently been approved to treat brain cancer include the Novartis product Afinitor (everolimus), an mTOR inhibitor, and the Roche antibody Avastin (bevacizumab), which is widely used in solid tumors because of its potent anti-angiogenic effect. However, the impact of Avastin on overall survival in glioblastoma multiforme (GBM) is extremely small – the drug was initially approved in recurrent GBM based on an overall survival enhancement from 8.7 months to 9.2 months. Data from the Phase 3 AVAglio study of Avastin for treatment of newly diagnosed GBM showed that Avastin plus radiation and temozolomide enhanced median progression-free survival (PFS) by 36% vs. radiation and temozolomide plus placebo in people with

newly-diagnosed glioblastoma. Specifically, Roche noted that a 4.4-month improvement in median PFS was observed compared to those who received radiation and chemotherapy plus placebo (10.6 months vs. 6.2 months). No new safety findings were observed in the study and adverse events were consistent with those seen in previous trials of Avastin for approved indications. However, the interim results for overall survival, the other co-primary endpoint, did not reach statistical significance. Final OS data from this trial are expected later this year but may not show any additional benefit.

Table 11: Brain Cancer Current Drugs

Drug Name	Generic Name	Company	Patent Expiry	Target Indication	Drug Class	Mechanism of Action
Afinitor	everolimus	Novartis AG	June 2020	Sub-ependymal giant cell astrocytoma (SEGA)	mTOR inhibitor	Cell growth inhibition
Avastin	bevacizumab	Roche	February 2019	GBM	Anti-VEGF antibody	Angiogenesis blocker
CeeNu	lomustine	various	Expired	GBM	Alkylating agent	DNA strand disruption
Matulane	procarbazine	various	Expired	GBM	Alkylating agent	DNA strand disruption
Temodar	temozolomide	various	August 2013	GBM	Nucleoside analog	DNA synthesis interference

Source: National Cancer Institute

There are a number of experimental drug candidates currently in development for the treatment of brain cancer. Most of these agents are aimed at glioblastoma; there are significantly fewer clinical trials currently being conducted in pediatric glioma. While most of the agents we have observed being tested in brain cancer are relatively new drugs, there are several development-stage programs in brain cancer involving drugs that have already been approved in other indications. Interestingly, a few of these existing approved drugs are also currently used to treat hematological malignancies. Examples would include Celgene Corporation's Revlimid – primarily a treatment for multiple myeloma and myelodysplastic syndrome – as well as Bristol-Myers Squibb's Sprycel (dasatinib), utilized principally in the treatment of chronic myelogenous leukemia. A table of the candidates currently being tested in brain cancer is shown below.

Table 12: Brain Cancer Experimental Candidates

Drug Name	Generic Name	Company	Target Indication	Drug Class	Mechanism of Action	Status
CDX-110	rindopepimut	Celldex Therapeutics	Glioblastoma	EGFRvIII vaccine	Peptide stimulatory vaccine	Phase 3
Inlyta	axitinib	Pfizer	Glioblastoma	Tyrosine kinase inhibitor	Cell proliferation blocker	Phase 2
Sprycel	dasatinib	Bristol-Myers Squibb	Glioblastoma	Kinase inhibitor	Cell proliferation blocker	Phase 2
PD 0332991	NA	Pfizer	Glioblastoma	Cyclin-dependent kinase 4 / 6 inhibitor	Cell cycle interruption	Phase 2
MetMab	onartuzumab	Roche	Glioblastoma	Monoclonal anti-Met antibody	Cell proliferation blocker	Phase 2
TKI258	dovitinib	Novartis AG	Glioblastoma	Kinase inhibitor	FGFR3 inhibitor - blocks proliferation	Phase 2
PF-299804	dacomitinib	Pfizer	Glioblastoma	Kinase inhibitor	Pan-HER irreversible inhibitor	Phase 2
PLX3397	NA	Plexxikon (Daiichi Sankyo)	Glioblastoma	Kinase inhibitor	FMS / c-Kit / FLT3 kinase inhibitor	Phase 2
TRC105	NA	Tracon Pharma	Glioblastoma	Anti-CD105 (endoglin) antibody	Angiogenesis blocker	Phase 2
BKM 120	NA	Memorial Sloan-Kettering	Glioblastoma	PI3K inhibitor	Cell proliferation inhibitor	Phase 2
Tykerb	lapatinib	GlaxoSmithKline	Pediatric glioma	Tyrosine kinase inhibitor	Cell proliferation / growth inhibitor	Phase 2
Revlimid	lenalidomide	Celgene Corporation	Pediatric glioma	Thalidomide analog	Cell proliferation / growth inhibitor	Phase 2
Theracim	nimotuzumab	Eurofarma Laboratorios	Pediatric glioma	Anti-EGFR monoclonal antibody	Cell proliferation inhibitor	Phase 2
SL-701	NA	Stemline Therapeutics	Glioblastoma Pediatric glioma	Synthetic IL-13Rα2 / Eph A2 peptide vaccine	Peptide stimulatory vaccine	Phase 2
AMG 102	rilotumumab	Amgen	Glioblastoma	Anti-hepatocyte growth factor antibody	Cell proliferation inhibitor	Phase 2
AMG 386	NA	Amgen	Glioblastoma	peptide-Fc fusion protein	Angiogenesis blocker	Phase 1 / 2
Zolinza	vorinostat	Merck & Co.	Glioblastoma	HDAC inhibitor	Hypomethylating agent	Phase 1 / 2
TPI 287	third-generation taxane	Tapestry Pharmaceuticals	Glioblastoma	Taxane (microtubule binder)	Cell division blocker	Phase 1 / 2
AXL 1717	picropodophyllin	Axelar AB	Glioblastoma	Insulin-like growth factor 1 receptor blocker	Cell proliferation blocker	Phase 1 / 2
R04929097	NA	Roche	Glioblastoma	Notch signaling pathway inhibitor	Cell proliferation blocker	Phase 1 / 2

Source: National Cancer Institute; ClinicalTrials.gov

Cancer Stem Cell-Targeting Approaches

While the principle of cancer stem cell targeting has been investigated avidly over the course of the past decade, relatively few drugs have been developed specifically with the aim of eliminating CSCs. Accordingly, therefore, we believe that Stemline Therapeutics is operating in a relatively open environment with a sparsely populated competitive landscape. There are several other CSC-focused companies currently in operation. Oncomed Pharmaceuticals, founded in August 2004, has raised approximately $300 million in total since inception, with $187 million coming in the form of equity financings and $112 million from payments made by its pharmaceutical partners.

While Oncomed remains privately-held, its lead investors include well-known venture capital firms such as De Novo Ventures, Phase4 Ventures, Morgenthaler Ventures and others, along with the strategic participation of GlaxoSmithKline. The company has an intriguing early-stage clinical pipeline that includes an anti-Notch1 antibody designated OMP52M51 – interestingly, one of the targets that Stemline Therapeutics is working on as well – that recently began Phase 1 testing in solid tumors.

The company is also developing a second antibody targeting Notch2 / 3 and a third antibody called demcizumab, designated OMP-21M18, which targets Delta-like ligand 4 (DLL4), a component of the Notch signaling pathway. Given this background, we consider Oncomed to be Stemline's clearest competitor in the Notch signaling domain as it pertains to development of anti-CSC therapeutics.

Demcizumab, which was originally placed on partial clinical hold by the FDA, is being tested in two Phase 1b combination trials currently. One study is assessing the drug in combination with standard-of-care gemcitabine (Gemzar) in first-line advanced pancreatic cancer patients, while the second is examining demcizumab's activity in combination with Eli Lilly & Co.'s Alimta (pemetrexed) in first-line advanced non-small cell lung cancer (NSCLC) patients.

Following the lifting of the partial clinical hold by the FDA in January 2013, Oncomed plans to initiate a Phase 1b / 2 trial with demcizumab in epithelial ovarian cancer in collaboration with researchers at the M.D. Anderson Cancer Center and, later in 2013, additional Phase 2 trials with the drug in NSCLC and pancreatic cancer.

We would also draw investors' attention to the fact that there is a significant precedent acquisition transaction in the cancer stem cell-targeting arena. The drug discovery company Boston Biomedical Inc. announced in March 2012 that it would be acquired by Japan-based Dainippon Sumitomo Pharma Co., Ltd. for up to $2.63 billion. The transaction closed in April 2012. Boston Biomedical Inc. was originally spun out of another Boston-based company, ArQule, Inc., in 2007. Dainippon Sumitomo paid $200 million upfront and is slated to pay up to $540 million in development-based milestone payments and up to $1.89 billion in sales-based milestone payments, according to the terms of the deal. Norwood-based Boston Biomedical had a pipeline of drug candidates targeting cancer stem cells, including BBI608 and BBI503.

The orally-administered BBI608 is ready for Phase 3 trials for colorectal cancer in North America and Phase 1b and Phase 2 trials for multiple solid tumors. BBI608 targets both highly malignant cancer stem cells as well as heterogeneous cancer cells. The acquisition transaction was consummated less than a year after Boston Biomedical signed a $15 million licensing deal with Dainippon Sumitomo Pharma for BBI608. In our view, this transaction is particularly encouraging for Stemline Therapeutics from a valuation perspective, as it would suggest that Stemline may not need to advance many of its currently-preclinical candidates from its anti-CSC platform very far into the clinic in order to attract interest from more established pharmaceutical companies.

In our view, however, Verastem is Stemline's most direct publicly-traded competitor at present. Verastem's scientific co-founders, Piyush Gupta and Bob Weinberg, pioneered novel assay platform technology that allowed them to screen for CSC-directed agents. This assay relies on the patent protected methodology for stably inducing epithelial-to-mesenchymal transition (EMT) in an epithelial tumor cell population that was first described in a *Cell* publication in 2009[11].

As Stemline Therapeutics has also discovered, the principal challenges associated with the identification and development of CSC-directed drugs using conventional high-throughput screening techniques are that a) the CSCs are relatively scarce compared to the bulk tumor and b) enriched CSC populations are invariably unstable, and their defining characteristics are rapidly lost when cultured *in vitro*.

In order to address these issues, Verastem's assay platform utilizes a non-tumorigenic mammary epithelial cell line (HMLE) experimentally transduced with short hairpin RNA to downregulate the expression of E-cadherin. Depleting the cells of E-cadherin expression induces EMT and adoption of a mesenchymal phenotype.

Retention of CSC properties by these so-called $HMLE^{shEcad}$ cells has been validated by analyses showing: (i) enrichment for the cell surface marker profile of CSCs with high CD44 and low CD24 expression (CD44high/CD24low); (ii) increased ability to form tumor spheres, characteristic of CSCs cultured *in vitro*; and (iii) a 100-fold increase in the ability to seed tumors in mice relative to control HMLE cells. In addition, these EMT-induced $HMLE^{shECad}$ cells are more resistant to standard chemotherapies like paclitaxel and doxorubicin, consistent with prior findings showing that such drugs permit enrichment for CSCs.

In our view, however, the main drawback to the Verastem system is the fact that it relies upon artificial manipulation to create CSC-like cells. While this may not constitute a major problem initially – since Verastem is only using this platform to screen compounds and identify new molecular targets – it may result in the identification of targets and compounds that may not in fact have a true impact on actual human CSCs *in vivo*.

Furthermore, Verastem's screening methodology could fail to identify true anti-CSC agents due to the artificially-induced nature of the CSC-like characteristics of Verastem's cells. In contrast, Stemline Therapeutics purposefully designed its StemScreen® technology platform to avoid any artificial manipulation. There is no gene expression regulation that is imposed on the cells using the StemScreen® approach, unlike the requirement for artificial E-cadherin down-modulation that is an integral component of Verastem's methodology. Furthermore, we would note that Stemline's system has led to the identification of both monoclonal antibody lead candidates as well as small molecules, whereas Verastem's early-stage pipeline consists solely of small molecules.

StemScreen® Technology Overview

Stemline Therapeutics has developed a bifurcated approach to facilitate the identification and development of anti-CSC therapeutics. StemScreen®-1 is a technology developed to discover CSC-targeted compounds and involves the isolation of CSCs, the discovery of potential CSC targets through CSC gene expression analysis, and the identification and validation of compounds that impact candidate CSC targets. StemScreen®-2 utilizes a cell-based assay developed to track and follow CSCs in their natural state during high throughput screening. Stemline has deployed StemScreen® to discover several of its product candidates, and believes that this platform could be instrumental in the discovery of additional new therapies targeting a wide range of cancer types.

[11] Gupta *et al.*, Cell 138: 645-659 (2009)

StemScreen®-1 Platform

StemScreen®-1 is a validated, proprietary drug discovery platform designed to identify CSC-targeted compounds based on the isolation of CSCs and evaluation of CSC gene expression profiles. CSCs are isolated from primary tumor tissue or cell lines, and then subjected to gene expression analysis using a variety of technologies, including microarray. A control tissue, such as normal bone marrow is analyzed as a comparator against the gene expression profile of the isolated CSCs. These data are then interfaced with an information base of compounds and their mechanisms of action (i.e. which gene products and pathways they impact). Compound classes are then identified as likely to impact CSC-specific pathways discovered by the gene expression analyses.

Select compounds within these classes are then tested in Stemline's proprietary anti-CSC functional *in vitro* and *in vivo* assays. Compounds that demonstrate anti-CSC activity are then considered for further development, which may include lead optimization. The StemScreen®-1 platform has been used to discover several of Stemline's preclinical drug candidates, including SL-201, SL-301, and SL-601. In addition, SL-401 demonstrated activity against CSCs as determined by both an *in vitro* colony formation and *in vivo* animal implantation assay, thereby validating certain StemScreen®-1 anti-CSC assays.

StemScreen®-2 Platform

StemScreen®-2 is a proprietary high throughput drug discovery platform that Stemline is developing in order to discover novel anti-CSC compounds. Traditional oncology drug discovery screens have largely relied upon readouts that measure activity against tumor bulk, and have not been specifically designed to identify compounds with activity against CSCs. StemScreen®-2 is based on a key discovery, covered by intellectual property controlled by Stemline, that immortal cancer cell lines harbor not only tumor bulk but also CSCs. This discovery enables compounds to be screened, in a high throughput manner, for activity against CSCs in their natural state.

The StemScreen®-2 approach utilizes a cell-based assay that can track and follow CSCs in their natural state during high throughput screening. In particular, StemScreen®-2 utilizes a CSC-specific promoter linked to a reporter in order to identify and follow CSCs in their native environment of surrounding tumor bulk. In this way, StemScreen®-2 enables the high-throughput identification of compound "hits" with anti-CSC activity.

Early-Stage Pipeline Assets

SL-301 is a small molecule gamma-secretase inhibitor that inhibits Notch, a pathway expressed by CSCs and tumor bulk of multiple cancer types. SL-301 has demonstrated activity against brain and pancreatic CSCs and tumor bulk *in vitro*, and against glioblastoma and medulloblastoma CSCs in several *in vivo* animal models. SL-101 is a monoclonal antibody targeting CD123 that has shown *in vitro* activity against certain hematologic cancers. SL-201 is a small molecule active against certain hematologic and solid tumors. Finally, SL-601 is a monoclonal antibody that targets a cell surface marker on bladder CSCs, which is also expressed on a variety of other solid tumors.

Stemline Therapeutics has also in-licensed certain intellectual property directed to mAb-based therapeutics to validated oncology targets including Glypican-3, Tie-1, CD133, Frizzled, Smoothened and Patched. Some of these antibody targets are also being pursued by other biopharmaceutical companies. Stemline Therapeutics has stated that it may independently develop – or partner with third parties to develop – any or all of these monoclonal antibodies. Thus, from our perspective, the company may have more strategic flexibility than investors currently give it credit for and it may have a superior CSC-focused drug discovery engine to that of Verastem.

Financing History / Capital Structure

Over the course of its history as a privately-held biopharmaceutical company, Stemline Therapeutics has raised roughly $17 million to support its research activities (see table below). In our view, the firm has demonstrated a capital-efficient operating history.

Table 13: Financing History

		Net Proceeds	Shares	Price	Notes
Private Company					
	Common Stock	$ 16,451,786	3,643,270	$ 4.52	Average price per share (2003-2012)
	Convertible Preferred				
Public Company					
	IPO	$ 38,152,910	3,815,291	$ 10.00	
	Secondary				
Total Amount		$ 54,604,696	7,458,561		

Source: Stemline Therapeutics, Inc.

Stemline Therapeutics, Inc. was originally incorporated in Delaware in August 2003. The firm has had operational headquarters in New York City since inception. On January 28, 2013, the firm successfully completed an Initial Public Offering (IPO), after which its shares began trading on the NASDAQ Capital Market under the ticker "STML."

The most recent capital structure indicates that Stemline Therapeutics had about 7.5 million shares outstanding and issued following its IPO, in which roughly 3.8 million shares of common stock were offered at a price of $10.00 per share. The fully-diluted share count stands at 9.4 million, factoring in all outstanding options and warrants (as shown in the table below). We estimate that Stemline currently has sufficient capital to fund operations through the end of 2014, following the IPO completion.

Table 14: Capital Structure

	Number of Shares	Weighted Average Exercise Price	Expiration Date	Total Cash
Cash, cash equivalents and marketable securities				**$34,500,000**
Common Stock	7,458,561			
Options	1,819,839	$2.76	various	$5,022,756
Warrants	99,529	$15.00	1/29/2018	$1,492,935
Fully Diluted Shares	**9,377,929**			**$41,015,691**

Source: Stemline Therapeutics, Inc.

Intellectual Property Portfolio

Stemline Therapeutics, Inc. has systematically built an IP estate since inception. The most relevant elements of the firm's IP portfolio are listed below.

Table 15: Patent Estate

Program	Patent #	Application #	Title	Priority Date	Issue Date	Expiry Date	Jurisdiction	Description
SL-401	7,763,242	11/899,747	Methods for treating myelodysplastic syndrome with a human interleukin-3-diphtheria toxin conjugate	9/7/2006	7/27/2010	2027	United States	SL-401
SL-401		12/368,048	Methods for treating acute myeloid leukemia with diphtheria toxin-interleukin-3 conjugates	9/7/2006	Pending	2027	United States	SL-401
SL-701	7,612,162	11/231,618	Peptide analogs capable of enhancing stimulation of a glioma-specific CTL response	9/21/2004	11/3/2009	2025	United States	SL-701 (IL-13Ra2 mutant peptide composition)
Therapeutics targeting CSCs	8,038,998	11/271,381	Novel methods of cancer therapy targeted against a cancer stem line	9/18/1997	10/18/2011	2017	United States	Broad coverage of mAb therapeutics targeting CSCs
Therapeutics targeting CSCs		11/900,029	Cancer stem cell-targeted cancer therapy	9/7/2006	Pending	2027	United States	Broad coverage of therapeutics targeting CSCs
Therapeutics targeting CSCs		11/899,690	Monitoring cancer stem cells	9/7/2006	Pending	2027	United States	Broad coverage of therapeutics targeting CSCs (with companion diagnostics)
Therapeutics targeting CSCs	7,361,336	09/468,286	Methods of cancer therapy targeted against a cancer stemline	9/18/1997	4/22/2008	2017	United States	mAb therapeutics targeting Frizzled
Therapeutics targeting microRNA		10/849,037	Methods of manipulating the fate of cells	6/21/2001	Pending	2022	United States	Broad coverage of therapeutics targeting microRNA
Diagnostics targeting CSCs	6,004,528	08/933,330	Methods of cancer diagnosis and therapy targeted against the cancer stemline	9/18/1997	12/21/1999	2017	United States	Broad coverage of CSC-directed Diagnostics
Therapeutics targeting IL-3R	6,733,743	09/799,100	Methods to impair hematologic cancer progenitor cells and compounds related thereto	3/6/2000	5/11/2004	2021	United States	mAb-based compounds targeting IL-3R
CSC-targeted Drug Discovery Platform		11/667,819	Methods and means related to cancer stem cells	11/12/2004	Pending	2025	United States	CSC-targeted Drug Discovery Platform (including high throughput screen)

Source: Company filings

Stemline's current IP portfolio includes over a dozen issued patents and more than 30 pending patent applications in the U.S. and abroad. This portfolio includes owned and exclusively in-licensed intellectual property that appears to be both early and broad with respect to the use of CSC-directed therapeutics and diagnostics (including companion diagnostics), as well as drug discovery. One of the main assets in Stemline Therapeutics' portfolio is the company's proprietary discovery platform, StemScreen®, designed specifically for the identification of novel cancer stem cell (CSC)-targeted compounds. StemScreen® contrasts with traditional drug discovery methods that have been designed to identify compounds that target tumor bulk, not CSCs. The StemScreen® platform includes an assay that utilizes live cells to track CSCs in their natural state during high throughput screening, which is a method that permits the rapid testing of many compounds on a small scale for enhanced efficiency. Stemline believes that this approach represents a major technological advance because it not only combines the highly desired properties of high-throughput capacity and CSC-targeting capability, but it also does not require artificial manipulation to create CSC-like cells as other systems do. Stemline Therapeutics researchers have utilized StemScreen® to discover a number of the firm's preclinical candidates, and believe that this platform may be instrumental in novel compound discovery relevant to a wide range of cancers.

From our perspective, the patent estate protecting Stemline Therapeutics' marketed product portfolio represents a reasonable asset for a company of Stemline's size. We believe the firm has successfully accomplished the task of assembling a relatively long-lived patent estate. Furthermore, we note that the company is advantageously positioned from a competitive standpoint with respect to its lead drug candidate, SL-401, which would enjoy a minimum of seven years of market exclusivity in the blastic plasmacytoid dendritic cell neoplasm (BPDCN) indication because this is clearly an ultra-orphan disease, and which would also potentially benefit from classification as a branded biologic drug, which as of today would imply at least 12 years of market exclusivity post-launch. Accordingly, therefore, we believe that Stemline Therapeutics possesses sufficient exclusivity-enabling protection to justify the value that we see in its portfolio.

Financial Review and Outlook

Revenue: We do not forecast any net revenue in 2012, 2013 or 2014. Management does not provide specific revenue or earnings guidance.

- **SL-401 Open IND BPDCN Clinical Data:** We anticipate that additional clinical data is likely to become available over the course of the next four to six months with SL-401 in more patients diagnosed with BPDCN. It may be possible for Stemline to enroll up to five more patients with BPDCN under the open IND that is currently in effect. If these other patients were to exhibit similar response data to those previously treated with the drug, we believe this could be a substantial catalyst for the stock.

- **FDA Sanction of SL-401 BPDCN Pivotal Pathway:** We expect that Stemline is likely to finalize the scope and nature of the pivotal trial necessary to secure approval of SL-401 in BPDCN with the FDA by the second half of 2013. If the trial design is indeed as small as previously indicated by the firm, we believe it could be initiated before the end of 2013 and fully enrolled by mid- to late-2014. Data could be available in early 2015 or earlier.

Gross Margins: We project that the gross margins on Stemline's late-stage agents are likely to approximate >80%, which are in line with margins achieved on typical biotech drugs and which should enable healthy cash flow generation. Investors should note that Stemline Therapeutics could conceivably market both of its clinical-stage candidates independently, because these agents – at least initially – are targeted towards niche markets representing small patient populations. The overall sales and marketing expenses associated with the limited sales force needed to commercialize these drugs means that Stemline's net margins should also be relatively high vs. its peers.

Operating Expenses: For 2012 and 2013, we estimate operating expense levels that are slightly higher than those in 2010 and 2011. We estimate R&D expenses to continue to rise gradually, from a projected $6.8 million in 2012 to $11.8 million in 2013.

Taxes: As previously indicated, Stemline Therapeutics has not accumulated substantial net operating loss carry-forwards since inception, with the accumulated deficit for the company standing at roughly $15 million as of the end of 2012. Accordingly, while we do not anticipate the firm attaining profitability in the foreseeable future, we would expect that the statutory U.S. corporate tax rate would apply relatively early in the product commercialization timeline. We have assumed an effective tax rate of 40% in our risk-adjusted Net Present Value (rNPV) calculations.

Share Count: The outstanding *pro forma* fully-diluted share count stands at roughly 9.4 million shares post-IPO. The fully-diluted shares account for the conversion of roughly 2 million outstanding warrants and options. Given the company's cash position, strategic goals, and capital structure, a share repurchase program is unlikely, in our view.

EPS: We forecast a net loss of approximately $2.04 per share in 2012, and a net loss of $1.61 for 2013. We do not anticipate that Stemline could attain profitability near-term.

Balance Sheet: Following the recently-consummated IPO, Stemline Therapeutics has approximately $29 million in cash and equivalents (*pro forma*). We do not expect the firm to require additional capital for at least 15 months.

Cash Flow: We project that Stemline will remain unprofitable for the foreseeable future.

Guidance: The firm does not provide financial guidance.

Management Team

The firm seeks to align the management team's interests with those of shareholders by using equity-based incentive awards, which generally consist of either stock options or shares of restricted stock that vest upon achievement of milestones directly related to achievement of specific sales levels. Many of Stemline Therapeutics' management team members have substantial prior experience in the biotechnology and healthcare arenas.

Ivan Bergstein, M.D.
Chief Executive Officer
Dr. Bergstein founded Stemline in August 2003 and has served as Chief Executive Officer since the company's inception. Prior to founding Stemline, Dr. Bergstein was Medical Director of Access Oncology, Inc., a private clinical stage oncology-focused biotechnology company. Previously, he was a senior biopharmaceuticals analyst at Cancer Advisors, Inc., a Wall Street-based firm that advised investment funds on public oncology focused companies. Dr. Bergstein received a B.A. in Mathematics from the University of Pennsylvania and an M.D. from the Mount Sinai Medical Center, where he completed a general surgery internship. Subsequently, he was named the Jerome A. Urban post-doctoral fellow at Cornell University Medical College. Dr. Bergstein then went on to complete a residency in internal medicine and a clinical Fellowship in hematology-medical oncology at the New York Presbyterian Hospital-Weill Medical College of Cornell University, where he is currently a voluntary faculty member.

Eric K. Rowinsky, M.D.
Chief Medical Officer, Head of Research & Development
Dr. Rowinsky has served as Stemline's Chief Medical Officer and Head of R&D since 2011. A widely-respected expert in the oncology sector, Dr. Rowinsky has more than 25 years of experience managing clinical trials and developing drugs from preclinical stages through to regulatory approval. He was previously Executive Vice President and Chief Medical Officer for ImClone Systems, Inc., which was subsequently acquired by Eli Lilly & Co., where he led the FDA approval of Erbitux® for head and neck and colorectal cancer, and advanced eight other monoclonal antibodies through clinical development. Dr. Rowinsky was previously Director of the Institute of Drug Development ("IDD") at the Cancer Therapy and Research Center. In addition, he held the SBC Endowed Chair for Early Drug Development at the IDD and was a Clinical Professor of Medicine at the University of Texas Health Science Center at San Antonio. Dr. Rowinsky was also an Associate Professor of Oncology at the Johns Hopkins University School of Medicine. He was a longstanding National Cancer Institute (NCI) principal investigator on U01 anticancer drug development grants, and was integrally involved in pivotal clinical and preclinical investigations, which led to the development of paclitaxel, docetaxel, topotecan, irinotecan, erlotinib, gefitinib, and temsirolimus, among others. Dr. Rowinsky is currently an Adjunct Professor of Medicine at the New York University School of Medicine and sits on the Board of Directors of a number of public and private biopharmaceutical companies, including Biogen Idec, Inc., one of the world's foremost and largest biotechnology firms. Dr. Rowinsky received his M.D. from Vanderbilt University School of Medicine. He completed his residency in internal medicine at the University of California at San Diego and his fellowship in medical oncology at Johns Hopkins Oncology Center.

Kenneth Hoberman
Vice President, Operations
Kenneth Hoberman has served as Vice President of Operations at Stemline Therapeutics since February 2012. From 2004 to 2012, Mr. Hoberman was Vice President of Corporate and Business Development of Keryx Biopharmaceuticals, Inc., where he was instrumental in securing multiple sources of capital including over $200 million in equity investments through public and private offerings. He also initiated and executed a $100

million strategic alliance and originated, negotiated and closed dozens of licensing and operational contracts, and helped grow the company to a $900 million market capitalization at peak. Previously, he was Managing Director at Hawkins BioVentures, a healthcare advisory firm and has served as a consultant to various healthcare-related companies. Mr. Hoberman received a B.S. / B.A. in Finance from Boston University and completed post-baccalaureate studies at Columbia University.

Stephen P. Hall, M.B.A.
Vice President, Finance, Chief Accounting Officer
Mr. Hall joined Stemline Therapeutics as a full time employee in October 2012 and serves as the company's Vice President of Finance and Chief Accounting Officer. Prior to joining Stemline on a full-time basis, Mr. Hall had previously acted as an advisor to Stemline since March 2012 on the company's accounting, finance, human resources, and information technology matters. Previously, Mr. Hall was founder and managing director of Deimos Consulting, LLC, a management consulting firm specializing in life sciences. Mr. Hall has also served as Senior Vice President, CFO, Chief Compliance Officer and Treasurer of Orthocon, Inc. a New York-based medical products company, from October 2009 to October 2010. Prior to this, Mr. Hall served as Senior Vice President, CFO and Treasurer of Helicos BioSciences, a public life science company, from May 2008 until August 2009. He previously served as Senior Vice President and Chief Financial Officer of TriPath Imaging, Inc., a public cancer diagnostics company, from September 2001 to December 2006, when it was acquired by Becton Dickinson & Co., at which time Mr. Hall continued to serve as Senior Advisor to Becton Dickinson & Co., from December 2006 to June 2007. He served as CFO and President of the Imaging and Power System Division of Colorado Medtech, Inc., public a medical products and services company, from September 1999 until August 2001. From September 1993 to January 1999, he served as CFO for BioTechnica International, Inc., a publicly-held agricultural products company. Mr. Hall spent four years with the accounting firm of Peat, Marwick, Mitchell & Co. He earned an A.B. degree from Harvard College and an M.B.A. from the Stanford Graduate School of Business.

Thomas P. Cirrito, Ph.D.
Vice President, R&D / Director, Business Development
Dr. Cirrito is currently VP of R&D and Director of Business Development for Stemline. Prior to joining Stemline in 2005, Dr. Cirrito was a biopharmaceuticals equities analyst at Piper Jaffray, where he covered large and small cap biotechnology firms. Previously he was a life sciences consultant for A.G. Edwards Capital Partners, a venture capital group. He received a B.A. in Biological Sciences and a Ph.D. in Immunology from Washington University (St. Louis, MO). Dr. Cirrito currently serves on the Scientific and Business Advisory Board of the Alzheimer's Drug Discovery Foundation.

Michael Szarek, Ph.D.
Vice President, Clinical & Regulatory Affairs
Dr. Szarek has over 15 years of experience in clinical and regulatory affairs in multiple therapeutic areas. He previously led a regulatory affairs team at ImClone Systems, Inc. that was responsible for development strategies for Erbitux® (cetuximab) and multiple other monoclonal antibody therapeutics. Prior to his tenure at ImClone, he was a director and biotechnology analyst and CIBC World Markets (later Oppenheimer and Co.). Previously, he was the worldwide statistical lead for Lipitor® (atorvastatin) at Pfizer, Inc. Dr. Szarek has published over 30 articles in leading medical and statistical journals and has served on numerous independent academic Steering Committees and Data Monitoring Committees for multiple Phase 3 trials. He earned an M.S. degree in biostatistics from the School of Public Health at Harvard University in Boston, MA, and a Ph.D. in biostatistics from New York University.

Joan Shankle
Regulatory Affairs
Joan Shankle has over 30 years of experience in the biopharmaceutical industry. She specializes in quality and regulatory development and has filed multiple BLAs and NDAs. She has led teams involved in oncology drug development, including that responsible for Ontak® (denileukin diftitox). Most recently, Ms. Shankle led the regulatory program for Istodax® (FK228, also known as romidepsin or depsipeptide), a novel histone deacetylase inhibitor drug derived from the bacterium *Chromobacterium violaceum* that was developed for the treatment of various hematological malignancies, at Gloucester Pharmaceuticals. The successful development of Istodax® by Ms. Shankle's group led to the agent's formal FDA approval in November 2009 and the subsequent acquisition of Gloucester by Celgene Corporation, one of the world's leading oncology-focused biotechnology firms, in January 2010.

John O'Loughlin, Ph.D.
Chemistry, Manufacturing and Controls (CMC)
Dr. O'Loughlin has over 27 years of experience with biopharmaceutical industry-based processes governing chemistry, manufacturing, and controls (CMC). He has held senior positions at Seragen Inc., Imreg Inc. and Interferon Sciences. Earlier in his career, Dr. O'Loughlin led the process development and manufacturing of Ontak® (denileukin diftitox) at Marathon Biopharmaceuticals, the first dedicated contract manufacturer approved for the commercial production of a recombinant biologic. This background makes him amply qualified to oversee the CMC portion of the development of SL-401, a recombinant biologic drug with various structural similarities to Ontak®. As an executive consultant, he has also worked extensively on process/product development, analytical biochemistry, CMC and regulatory strategy with multiple companies including Ligand Pharmaceuticals, Cambrex Inc., Adnexus Therapeutics, and GTC Biotherapeutics.

Chris Brooks, Ph.D.
Director of Preclinical Development
Dr. Brooks currently serves as Stemline's head of preclinical development. Prior to joining Stemline Therapeutics in 2010, Dr. Brooks was a senior staff scientist at Progenics Pharmaceuticals, Inc., where he led an R&D team that developed novel antibody therapeutics. He has published numerous papers, including peer-reviewed articles and book chapters, on molecular mechanisms of cancer development. His research has been featured in journals such as *Science*, *Current Opinions in Cell Biology*, *Cancer Cell*, and *Nature Reviews Cancer*. He received a B.S. in Biological Sciences *cum laude* from Binghamton University in upstate New York and a Ph.D. in Pathology and Cell Biology from the College of Physicians and Surgeons at Columbia University.

Mark Jacobson, M.A.
Director of Corporate Development
Mr. Jacobson currently serves as the company's director of corporate development. Prior to joining Stemline in 2009, Mr. Jacobson was as a healthcare analyst for Publicis Healthcare Communications Group, a life sciences marketing and communications company, where he developed research reports on the healthcare industry as a whole and on targeted companies within the industry. He received a B.S. in biology with honors from Iowa State University and obtained a Master's degree in Biotechnology from Columbia University.

Board of Directors

The firm's Board of Directors includes several senior-level individuals with substantial experience at the executive management level in the biotechnology and pharmaceuticals sectors. In our view, Stemline's board possesses the necessary knowledge base and experience to provide appropriate guidance to the company's senior executives as the firm seeks to build shareholder value. Several members of the firm's Board of Directors have particularly substantial experience buying and selling companies and creating value for shareholders through M&A transactions.

J. Kevin Buchi, C.P.A.
Chairman of the Board
Mr. Buchi served as the CEO of Cephalon, Inc. starting in December 2010, following the departure of longtime CEO Frank Baldino, Jr., and led Cephalon up to the point at which the firm was acquired in a $6.8 billion transaction by Teva Pharmaceutical Industries in October 2011. Mr. Buchi joined Cephalon in March 1991. From January 2010 through December 2010, Mr. Buchi was Chief Operating Officer. In this role, he managed the company's global sales and marketing functions, as well as product manufacturing, business development and investor relations. From February 2006 through January 2010, Mr. Buchi served as CFO. At various times in his career at Cephalon, he had oversight of corporate finance, accounting, information systems, facilities, human resources and administration. Mr. Buchi graduated from Cornell University with a B.A. degree in chemistry. He was a synthetic organic chemist for the Eastman Kodak Company before going on to obtain a master's degree in management from the J.L. Kellogg Graduate School of Management at Northwestern University. Mr. Buchi worked for a large public accounting firm before beginning his career in the pharmaceutical industry with E.I. du Pont de Nemours & Co. in 1983. Mr. Buchi is a certified public accountant and has previously served on the boards of various other public and private firms.

Ivan Bergstein, M.D.
Director
See bio above.

Eric Dobmeier, J.D.
Director
Mr. Dobmeier is currently the Chief Operating Officer of Seattle Genetics, Inc., a leading oncology-focused therapeutic antibody company. In his role at Seattle Genetics, he is responsible for the company's business development, manufacturing, corporate communications, legal, market research and program and alliance management functions. Mr. Dobmeier joined Seattle Genetics in March 2002 and has served in positions of increasing responsibility since then, most recently as Chief Business Officer from May 2007 to June 2011. Prior to joining Seattle Genetics, Mr. Dobmeier was with the law firms of Venture Law Group and Heller Ehrman LLP where he represented technology companies in connection with public and private financings, mergers and acquisitions and corporate partnering transactions. He received a J.D. from the School of Law at the University of California at Berkeley and an A.B. in History from Princeton University.

Kenneth Zuerblis, C.P.A.
Director
Mr. Zuerblis is currently Executive Vice President and CFO of Savient Pharmaceuticals, Inc., a publicly-traded commercial-stage biotechnology company. Prior to joining Savient, Mr. Zuerblis served as CFO and Senior Vice President at ImClone Systems from 2008 through 2009. In that role, he was responsible for the strategic planning and leadership of finance and related operations and helped lead all aspects of the 2008 sale of the company to Eli Lilly & Co. From 1994 through 2005, Mr. Zuerblis served as Chief Financial Officer of Enzon Pharmaceuticals Inc., and held the position of Corporate

Controller from 1991 through 1994. Enzon developed the first three FDA approved products using PEGylation technology. Most notably, during Mr. Zuerblis' 14-year tenure, Enzon transformed from an early stage biotechnology company into a fully integrated biopharmaceutical company with five marketed products. He began his career at KPMG, LLP in 1982 where he held management positions of increasing responsibility over a 10-year period. Mr. Zuerblis brings nearly 30 years of proven leadership expertise in building fully integrated biopharmaceutical organizations to the Stemline Board of Directors. He has an established and extensive track record of managing complex commercial and research organizations, raising capital, overseeing multifaceted merger and acquisition transactions, and directing all investor and shareholder relations. Mr. Zuerblis earned his Bachelor of Science in Accounting from Seton Hall University and is a certified public accountant in the State of New Jersey.

Ron Bentsur, M.B.A.
Director
Mr. Bentsur is CEO of Keryx Biopharmaceuticals, Inc., a publicly-traded, late-stage biopharmaceuticals company that is currently developing a therapeutic agent for the treatment of hyperphosphatemia in patients with end-stage renal disease. Prior to joining Keryx, Mr. Bentsur served as CEO of XTL Biopharmaceuticals, Inc. from 2006 to 2009. From 2000 to 2006, Mr. Bentsur was employed by Keryx, where he served as Vice President Finance and CFO from 2003 until 2006. From 1998 to 2000, Mr. Bentsur served as Director of Technology Investment Banking at Leumi Underwriters, where he was responsible for all technology and biotechnology private placement and advisory transactions. From 1994 to 1998, Mr. Bentsur was a New York-based investment banker, primarily at ING Barings Furman Selz. He holds a B.A. in Economics and Business Administration with distinction from the Hebrew University of Jerusalem and an M.B.A., *magna cum laude*, from New York University's Stern Graduate School of Business.

Scientific Advisory Board

The firm's Scientific Advisory Board, which is particularly important for a company at Stemline's stage of development, includes several world-renowned and respected physicians who are considered among the leading authorities in their respective fields.

Hagop Kantarjian, M.D.
Chairman, Department of Leukemia, M.D. Anderson Cancer Center, Houston, TX
One of the world's foremost experts in the domain of blood cancers – particularly diseases such as acute myeloid leukemia (AML) and myelodysplastic syndrome (MDS), Dr. Kantarjian is a professor of medicine and Chairman of the Department of Leukemia at The University of Texas M. D. Anderson Cancer Center, and holds the Kelcie Margaret Kana Research Chair. He has been integrally involved with the design, development and registration of a wide range of chemotherapy agents, targeted therapies, and biological therapies for the treatment of acute and chronic leukemias and myelodysplastic syndrome. Dr. Kantarjian has taken an active role in the medical community, participating in numerous editorial boards and medical societies and holding administrative positions. He has authored or co-authored more than 750 medical publications and abstracts and serves on editorial boards for four scientific journals. In 1997, he received the first Emil J Freireich Award for Outstanding Clinical Research at M. D. Anderson. Dr. Kantarjian was named a Scholar of the Leukemia Society of America from 1989 to 1994 and a Special Fellow of the Leukemia Society of America from 1982 to 1983. He received his Bachelor of Science degree and medical degree from The American University of Beirut in 1975 and 1979, respectively. Dr. Kantarjian completed his residency in internal medicine at The American University of Beirut and a fellowship in medical oncology at M. D. Anderson.

Patrick Wen, M.D.
Director, Neuro-Oncology, Dana-Farber / Harvard Cancer Center
Professor, Harvard Medical School
Dr. Wen is professor of neurology at the Harvard Medical School and director of neuro-oncology at the Dana-Farber/Brigham and Women's Cancer Center in Boston, MA. He is chair of the Dana-Farber/Harvard Cancer Center Neuro-Oncology Clinical Trials Committee and principal or co-investigator of many ongoing clinical protocols. Dr. Wen's clinical research focuses on targeted molecular therapies and anti-angiogenic therapies for gliomas, meningiomas, and brain metastases. He serves on numerous national and international committees, including the NCI Developmental Therapeutics and the Cancer Diagnostic and Treatment SBIR Study Sections and co-chairs the American Brain Tumor Consortium New Agents Committees. He is Associate Editor for the *Journal of Neuro-Oncology* and on the editorial board of *Neuro-Oncology*. Dr. Wen is Vice President of the Society of Neuro-Oncology. He has authored or coauthored numerous peer-reviewed manuscripts, book chapters, reviews, editorials, and abstracts and is actively involved in a number of professional societies. He has received numerous awards, including the Society of Neuro-Oncology Research Excellence Award and the George Cannellos Award for Excellence in Clinical Care and Research from the Dana-Farber Cancer Institute. Dr. Wen earned his medical degree from the Medical College of St. Bartholomew's Hospital at the University of London. He completed his residency at the Harvard Longwood Neurology Training Program, followed by a fellowship in neurology at Brigham and Women's Hospital in Boston, MA.

Owen O'Connor, M.D., Ph.D.
Leader, Lymphoid Development and Malignancy Program
Associate Professor of Medicine, Columbia University Medical Center
Dr. O'Connor's research and clinical efforts have led to numerous innovations and patents on novel small molecules, and have produced one of the largest portfolios of new drugs for lymphoma in the world. Over the past decade, his work has contributed to the FDA approval of drugs for relapsed and refractory mantle cell lymphoma, cutaneous T-cell lymphoma, and relapsed or refractory peripheral T-cell lymphoma. He co-invented and developed Folotyn® (pralatrexate) at Memorial Sloan-Kettering Cancer Center, which became the first drug ever approved by the FDA for the treatment of patients with relapsed or refractory peripheral T-cell lymphoma, and which was originally commercialized by Allos Therapeutics, acquired by Spectrum Pharmaceuticals in 2012. Dr. O'Connor received his B.S. in biology, *magna cum laude*, from Manhattan College in 1982, his Ph.D. from the New York University School of Medicine, and his M.D. from Robert Wood Johnson Medical School at the University of Medicine & Dentistry of New Jersey in 1994. Dr. O'Connor was previously deputy director of clinical research and cancer treatment at the NYU Cancer Institute, chief of the division of hematologic malignancies and medical oncology in the Department of Medicine, and professor of medicine and pharmacology at the NYU Langone Medical Center.

David Reardon, M.D.
Clinical Director, Neuro-Oncology, Dana-Farber / Harvard Cancer Center
Associate Professor of Medicine, Harvard Medical School
Dr. David Reardon is clinical director at Dana-Farber Cancer Institute's Center for Neuro-Oncology and associate professor at Harvard Medical School. Prior to joining the Dana-Farber Cancer Institute, Dr. Reardon served as Associate Deputy Director of The Preston Robert Tisch Brain Tumor Center at Duke University Medical Center. In 2007, Dr. Reardon was the recipient of the R. Wayne Rundles Award for Excellence in Cancer Research. He has co-authored approximately 140 peer-reviewed manuscripts focused on neuro-oncology and 15 additional articles and book chapters. Dr. Reardon received his M.D. from Tufts Medical School in 1986 and completed a pediatrics residency at Johns Hopkins Hospital. He also completed a fellowship in pediatric hematology/oncology at the University of Michigan Hospital, MOTT Children's Hospital in 1992.

Table 16: Stemline Therapeutics, Inc. (STML) – Historical Income Statements, Financial Projections

FY end December 31
$ in thousands, except per share data

			2013E					2014E				
	2011A	2012E	1QE	2QE	3QE	4QE	2013E	1QE	2QE	3QE	4QE	2014E
Revenue												
Product revenue	-	-	-	-	-	-	-	-	-	-	-	-
Contract research	-	-	-	-	-	-	-	-	-	-	-	-
Total revenue	-	-	-	-	-	-	-	-	-	-	-	-
Expenses												
Research & development	1,629	4,035	1,300	1,600	1,800	2,100	6,800	2,400	2,700	3,100	3,600	11,800
General and administrative	1,088	3,318	1,000	1,100	1,200	1,300	4,600	1,400	1,500	1,600	1,700	6,200
Total expenses	2,717	7,353	2,300	2,700	3,000	3,400	11,400	3,800	4,200	4,700	5,300	18,000
Gain (loss) from operations	(2,717)	(7,353)	(2,300)	(2,700)	(3,000)	(3,400)	(11,400)	(3,800)	(4,200)	(4,700)	(5,300)	(18,000)
Other income/expense												
Interest income	24	12	26	29	23	18	96	15	6	14	17	52
Interest expense	(99)	(109)	-	-	-	-	-	-	-	-	-	-
Other income	47	417.678	-	-	-	-	-	-	-	-	-	-
Other expense	(10)	-	-	-	-	-	-	-	-	-	-	-
Total investment income and other	(38)	320	26	29	23	18	96	15	6	14	17	52
Loss before provision for income taxes	(2,755)	(7,033)	(2,274)	(2,671)	(2,977)	(3,382)	(11,304)	(3,785)	(4,194)	(4,686)	(5,283)	(17,948)
Income tax benefit (loss)	-	-	-	-	-	-	-	-	-	-	-	-
Net loss/income	(2,755)	(7,033)	(2,274)	(2,671)	(2,977)	(3,382)	(11,304)	(3,785)	(4,194)	(4,686)	(5,283)	(17,948)
Net loss per share (basic)	(0.80)	(2.04)	(0.42)	(0.36)	(0.40)	(0.45)	(1.61)	(0.50)	(0.47)	(0.46)	(0.51)	(1.94)
Net loss per share (diluted)	(0.80)	(2.04)	(0.42)	(0.36)	(0.40)	(0.45)	(1.61)	(0.50)	(0.47)	(0.46)	(0.51)	(1.94)
Weighted average number of shares outstanding (basic)	3,442	3,446	5,468	7,484	7,534	7,584	7,017	7,634	8,934	10,234	10,284	9,271
Weighted average number of shares outstanding (diluted)	3,442	3,446	5,468	7,484	7,534	7,584	7,017	7,634	8,934	10,234	10,284	9,271

Source: Company Reports and Aegis Capital Corp. estimates

Public companies mentioned in this report:

Ariad Pharmaceuticals (ARIA/NASDAQ – $21.81)

ArQule, Inc. (ARQL/NASDAQ – $2.55)

AVEO Pharmaceuticals (AVEO/NASDAQ – $7.46)

Bristol-Myers Squibb (BMY/NYSE – $38.96)

Celgene Corporation (CELG/NASDAQ – $112.40)

Celldex Therapeutics (CLDX/NASDAQ – $12.06)

Cyclacel Pharmaceuticals (CYCC/NASDAQ – $5.77)

CytRx Corporation (CYTR/NASDAQ – $2.51 – Buy)

Dainippon Sumitomo Pharma Co., Ltd. (DNPUF/OTCBB – $16.73)

Galena Biopharma (GALE/NASDAQ – $1.92 – Buy)

GlaxoSmithKline (GSK/NYSE – $45.30)

ImmunoCellular Therapeutics (IMUC/NASDAQ – $2.75)

Infinity Pharmaceuticals (INFI/NASDAQ – $49.37)

Keryx Biopharmaceuticals (KERX/NASDAQ – $7.32)

Merck & Co. (MRK/NYSE – $44.09)

Merrimack Pharmaceuticals (MACK/NASDAQ – $6.03)

Northwest Biotherapeutics (NWBO/NASDAQ – $3.60)

Novartis AG (NVS/NYSE – $174.88)

OncoGenex Pharmaceuticals (OGXI/NASDAQ – $12.19)

Oncolytics Biotech (ONCY/NASDAQ – $2.99)

Onyx Pharmaceuticals (ONXX/NASDAQ – $87.71)

Pfizer Inc. (PFE/NYSE – $28.02)

Puma Biotechnology (PBYI/NASDAQ – $29.12)

Seattle Genetics (SGEN/NASDAQ – $33.55)

Spectrum Pharmaceuticals (SPPI/NASDAQ – $7.76)

Verastem (VSTM/NASDAQ – $9.20)

Required Disclosures

Price Target

Our 18-month price target is $35.00 per share.

Valuation Methodology

We derive our price target using a discounted cash flow-based sum-of-the-parts analysis approach, which derives a $375 million total enterprise value for the company's clinical-stage assets, SL-401 and SL-701, as well as the early-stage pipeline and the proprietary cancer stem cell-targeting drug discovery platform. Our total firm valuation of $420 million assumes $45 million in cash as of mid-2014; this translates into a price target of $35.00 per share based on 12 million shares (fully-diluted) and no debt as of mid-2014.

Risk Factors

Various factors may impede or prevent achievement of the price target by the company's shares. Such risk factors may include, but are not limited to, clinical, regulatory, competitive, financial, and reimbursement issues. Products that have yet to be submitted to regulatory agencies for review may not reach the market due to regulatory concerns, which could preclude approval. The company may require financing to sustain and grow its pipeline, which could be dilutive to current shareholders. We expect competition from existing entities against the company's products.

For important disclosures go to www.aegiscap.com.

Research analyst compensation is dependent, in part, upon investment banking revenues received by Aegis Capital Corp.

Aegis Capital Corp. intends to seek or expects to receive compensation for investment banking services from the subject company within the next three months.

Rating	Percent	Investment Banking Services/Past 12 Mos. Percent
BUY [BUY]	82.35	25.00
HOLD [HOLD]	17.65	16.67
SELL [SELL]	0.00	0.00

Meaning of Ratings

A) A Buy rating is assigned when we do not believe the stock price adequately reflects a company's prospects over 12-18 months.
B) A Hold rating is assigned when we believe the stock price adequately reflects a company's prospects over 12-18 months.
C) A Sell rating is assigned when we believe the stock price more than adequately reflects a company's prospects over 12-18 months.

Other Disclosures

The information contained herein is based upon sources believed to be reliable but is not guaranteed by us and is not considered to be all inclusive. It is not to be construed as an offer or the solicitation of an offer to sell or buy the securities mentioned herein. Aegis Capital Corp., its affiliates, shareholders, officers, staff, and/or members of their families, may have a position in the securities mentioned herein, and, before or after your receipt of this report, may make or recommend purchases and/or sales for their own accounts or for the accounts of other customers of the Firm from time to time in the open market or otherwise. Opinions expressed are our present opinions only and are subject to change without notice. Aegis Capital is under no obligation to provide updates to the opinions or information provided herein. Additional information is available upon request.

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