OncoMed Pharmaceuticals

(OMED-NASDAQ)

Stock Rating: **Outperform**
Industry Rating: **Outperform**

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Biotechnology

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Initiating With Outperform on Cancer Stem Cell Leadership

Investment Thesis

We are initiating coverage of OncoMed (OMED) with an Outperform rating and $32 price target. Our positive outlook is based on OMED's leadership position in the cancer stem cell (CSC) therapeutic space and on prospects for success across multiple large cancer indications. We believe that biologic rationale, preclinical data and early clinical data provide substantial proof-of-concept for the cancer stem cell (CSC) approach and OMED therapeutics in particular. While OMED is expected to generate phase 2 proof-of-concept data across 11 different studies by 2016 we believe that upside potential from current valuation levels exists on prospects for lead anti-DLL4 antibody demcizumab in non small-cell lung (NSCLC) and pancreatic cancer alone. Early response rate data for demcizumab + GEMZAR in pancreatic Ca and demcizumab + carboplatin/ALIMTA in NSCLC suggest potential synergy with chemotherapy that could be more broadly leveragable.

Forecasts

We forecast a loss per share of ($0.77) in 2013 and expect continued losses through 2018. We expect initial profitability in 2019 with GAAP EPS of $3.71 forecast, growing to $6.65 in 2020. Estimates are driven by expected royalties from demcizumab in pancreatic Ca and NSCLC.

Valuation

We arrive at our $32 price target by applying a 20x multiple on 2020 EPS estimate of $6.65 and discounting at 30%.

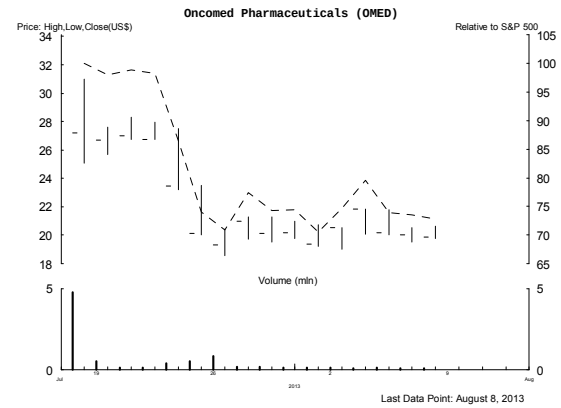
Recommendation

We rate OMED shares Outperform.

Securities Info

Price (9-Aug)	**$19.84**	**Target Price**	**$32**
52-Wk High/Low	$31/$19	Dividend	--
Mkt Cap (mm)	$553	Yield	--
Shs O/S (mm, BASIC)	27.8	Float O/S (mm)	5.4
Options O/S (mm)	NA	ADVol (30-day, 000s)	553

Price Performance



Valuation/Financial Data

(FY-Dec.)	2011A	2012A	2013E	2014E
EPS GAAP	-$15.40	-$1.00	-$0.77	-$1.43
P/E			nm	nm
First Call Cons.				
FCF	na	na	na	na
P/FCF			na	na
EBITDA ($mm)	-$15	-$22	-$25	-$44
EV/EBITDA			nm	nm
Rev. ($mm)	$31	$25	$33	$47
EV/Rev			12.4x	8.7x

Quarterly EPS	**1Q**	**2Q**	**3Q**	**4Q**
2012A	na	na	na	na
2013E	-$0.39A	$0.52	-$0.33	-$0.57

Balance Sheet Data (31-Mar)

Net Debt ($mm)	-$144	TotalDebt/EBITDA	nm
Total Debt ($mm)	$0	EBITDA/IntExp	na
Net Debt/Cap.	nm	Price/Book	5.2x

Notes: Quarterly EPS may not sum due to share count. All values in US$.

Source: BMO Capital Markets estimates, Bloomberg, Thomson Reuters, and IHS Global Insight.

Please refer to pages 52 to 56 for Important Disclosures, including the Analyst's Certification.

Investment Thesis

We believe that OMED is best-positioned in the rapidly evolving area of cancer stem cell (CSC) therapeutics with potential proof-of-concept data emerging across 11 separate phase 2 programs through 2016. We believe that opportunity for lead Notch pathway targeting anti-DLL4 antibody demcizumab alone in pancreatic cancer and NSCLC could support significant upside from current valuation levels alone and see further option value from nine other less advanced programs.

Key Investment Highlights

- OncoMed (OMED) is a development stage biotechnology company focused on identification, isolation, and targeting of the cancer stem cell (CSC).
- Evidence is emerging that cancer stem cells are the key drivers of cancer proliferation and metastatic spread and are not responsive to traditional chemotherapy.
- Expert feedback has suggested that profound benefits of certain high-value biologics, like HERCEPTIN in Her2+ breast cancer, can be linked to effects on key cancer stem cell pathways.
- OncoMed (OMED) is well positioned in the cancer stem cell (CSC) therapeutics space with data from 11 separate phase 2 trials expected to report by 2016.
- Upside to valuation could be achieved alone by the company's lead Notch targeting anti-DLL4 antibody demcizumab in pancreatic cancer and non small-cell lung cancer (NSCLC) where response rates in combination with standard of care chemotherapy are higher than that seen historically for standard of care alone.
- Additional upside potential could emerge from the company's unpartnered DLL4/VEGF bispecific antibody, anti-Notch1 program, Notch 2/3 targeting OMP-59R5, Wnt pathway targeting anti-Fzd7 drug OMP-18R5, anti-Fzd8-Fc targeting drug OMP-54F28 and first-in-class RSPO-LGR targeting biologics, all across multiple cancer indications.
- OncoMed could also benefit from validation of other cancer stem cell (CSC) therapeutics from companies like Verastem and Stemline, and from higher visibility of cancer stem cell (CSC) therapeutic progress at high profile conferences such as the International Society for Stem Cell Research (ISSCR) meeting in Vancouver June 18-21, 2014.
- Risks to consider in an investment in OMED include the potential for toxicity from targeting normal stem cells, prior history of clinical hold on demcizumab, apparent sensitivity of cancer stem cell (CSC) therapeutics to dose scheduling, complexity of cancer stem cell pathways, and difficulty in extrapolating preclinical and early clinical response rate data into more definitive benefit in later stage studies.

Valuation

We arrive at our $32 price target by applying a 20x multiple to 2020 EPS estimate of $6.65 and discounting at 30%.

Our sum-of-the-parts NPV analysis suggests a present value of $26 per share for the product pipeline and $29 per share including cash. Our product NPV is driven solely by estimates for demcizumab in pancreatic Ca and NSCLC and assumes a 15% royalty, 30% likelihood of success and additional 10% discount rate.

In evaluating development stage oncology comparables we believe that Clovis (CLVS), Array BioPharma (ARRY) and Epizyme (EPZM) are most suitable comparables with enterprise values (EV) of $2 billion, $734 million and $924 million, respectively. At an EV of $450 million OMED trades at steep discount to these oncology comparables, albeit at a premium to other cancer stem cell companies, like Verastem (VSTM) at $253 million and Stemline (STML) at $386 million. We believe that this premium is warranted given the broader portfolio, greater patient experience and larger end markets targeted by OMED.

Exhibit 1: OncoMed Comps

ONCOMED COMPARABLES

Company	Ticker	Price	Shares (M)	Market Cap (M)	Cash (M)	EV (M)	Therapeutic Focus	Stage of Development
ArQule	ARQL	$2.54	62.6	$159.1	$87.1	$73.7	Oncology	Phase 3
Array BioPharma	**ARRY**	**6.21**	**116.8**	**725.2**	**86.6**	**733.9**	**Oncology**	**Phase 3**
Aveo Oncology	AVEO	2.16	51.9	112.1	191.6	-53.4	Oncology	Phase 2
Celldex Therapeutics	CLDX	18.90	80.9	1,528.4	84.0	1,455.8	Oncology	Phase 2
Clovis Oncology	**CLVS**	**72.20**	**29.7**	**2,143.2**	**129.6**	**2,013.6**	**Oncology**	**Phase 2**
Curis	CRIS	3.86	80.5	310.9	50.5	290.6	Oncology	Marketed
Cyclacel Pharmaceuticals	CYCC	3.00	17.7	53.1	17.8	35.3	Oncology	Phase 3
Epizyme	**EPZM**	**35.50**	**28.4**	**1,009.4**	**85.0**	**924.3**	**Oncology**	**Phase 1**
Geron	GERN	1.55	130.4	202.2	79.8	122.3	Oncology	Phase 2
Infinity Pharmaceuticals	INFI	19.04	49.2	937.6	302.6	635.1	Oncology	Phase 2
NewLink Genetics	NLNK	17.74	25.6	454.2	64.0	397.3	Oncology	Phase 3
Stemline Therapeutics	STML	33.46	12.4	416.0	30.7	385.9	Oncology	Phase 2
Synta Pharmaceuticals	SNTA	6.79	69.1	469.4	90.4	402.7	Oncology	Phase 3
Telik	TELK	1.42	4.6	6.5	5.6	0.9	Oncology	Phase 2
Threshold Pharmaceuticals	THLD	4.96	56.5	280.5	104.2	176.2	Oncology	Phase 2
Verastem	VSTM	15.00	21.3	319.3	66.7	252.6	Oncology	Phase 1
ZIOPHARM Oncology	ZIOP	3.10	83.6	259.0	55.7	203.4	Oncology	Phase 3
Mean				$552.1		$473.5		
Median				319.3		290.6		
OncoMed	OMED	$19.93	29.8	$593.9	$143.6	$450.3	Oncology	Phase 2

ONCOMED SUM-OF-THE-PARTS

Target	Indication	Price	Shares (M)	Launch Year	Peak Sales ($M)	Probability	NPV/share ($M)
Demcizumab - U.S.	NSCLC			2018	$1,158.3	30%	$7.22
Demcizumab - EX-U.S.	NSCLC			2018	1,158.3	30%	7.22
Demcizumab - U.S.	Pancreatic Cancer			2018	918.0	30%	5.78
Demcizumab - EX-U.S.	Pancreatic Cancer			2018	918.0	30%	5.78
Total							**$25.99**

Source: Company reports, Thomson Reuters, and BMO Capital Markets.

Overview

OncoMed Pharmaceuticals is a clinical development-stage biopharmaceutical company focused on discovery and development of monoclonal antibody therapeutics targeting cancer stem cells (CSCs). OncoMed utilizes proprietary technologies to identify, validate, and develop antibodies or other protein-based biologics that target molecular pathways involved in CSC self-renewal, differentiation, and growth. These CSC-directed agents hold potential promise of improving cancer treatment. Five internally discovered anti-CSC product candidates, targeted at the Notch and Wnt pathways, are in clinical development, and two additional antibodies are in preclinical development with IND filings planned for as early as 2014. (See Exhibit 2.)

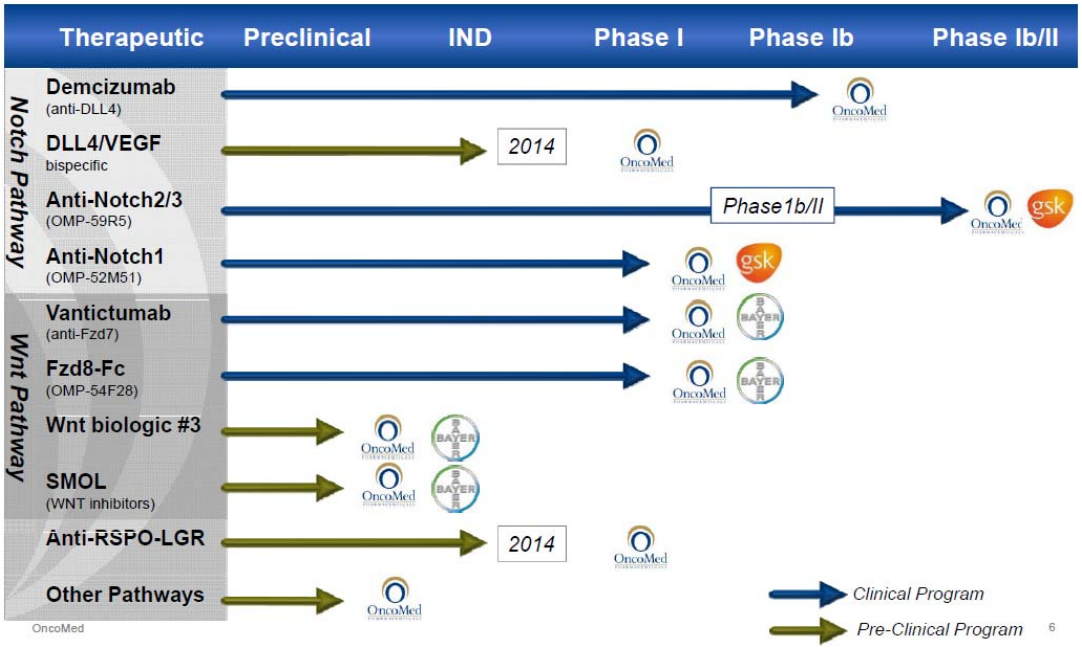
The lead product candidate, demcizumab (OMP-21M18), is a humanized monoclonal antibody (mAb) that inhibits Delta-like ligand 4 (DLL4) in the Notch signaling pathway. Preclinical studies have demonstrated demcizumab's anti-tumor and anti-CSC activities in multiple solid tumor types, including pancreatic, lung, breast, colon, melanoma, and ovarian cancers. In phase 1 studies, demcizumab demonstrated single-agent activity in advanced solid tumor patients, with a disease control rate (DCR) of 64% in the cohort receiving the highest dose and one partial response in recurrent pancreatic adenocarcinoma.

A partial clinical hold was placed on demcizumab's phase 1a program after the emergence of Grade 3 and 4 cardiovascular adverse events in that program. FDA lifted this clinical hold in January 2013 after reviewing data from more recent phase1b trials of demcizumab that included a risk mitigation plan and modifications in dosing schedules.

Two phase 1b trials of demcizumab are currently ongoing: one in advanced pancreatic cancer as a first-line therapy in combination with standard-of-care gemcitabine, and the other one in non-small lung cancer (NSCLC) as a first-line therapy in combination with standard-of-care carboplatin and ALIMTA. Initial data from the ongoing phase 1b trials suggest encouraging anti-tumor activity and manageable hypertension.

Another important pipeline asset for OncoMed is OMP-59R5, a fully human anti-Notch2/3 mAb that blocks Notch receptor signaling. OMP-59R5 binds to an epitope that is conserved between Notch2 and Notch3 receptors. A single-agent phase 1a trial of OMP-59R5 in advanced solid tumor patients has been completed, and two phase 1b/2 trials are ongoing: the ALPINE trial is in pancreatic cancer and the PINNACLE trial is in small cell lung cancer. Both the ALPINE trial and the PINNACLE trial include an analysis of a predicative biomarker to indentify patients who may derive the greatest benefit from OMP-59R5. The estimated primary completion dates are January 2014 for the ALPINE trial and October 2015 for the PINNACLE trial. OMP-59R5 is partnered with GlaxoSmithKline (GSK), and GSK retains an option through the end of certain phase 2 trials to obtain an exclusive license to OMP-59R5.

Exhibit 2: OncoMed Pipeline



Source: OncoMed Pharmaceuticals.

OncoMed also partners with GSK on OMP-52M51, a mAb against Notch1. OMP-52M51 is currently in two separate phase 1 trials in hematologic malignancies and advanced solid tumors, and the primary completion dates for both trials are May 2014.

OncoMed's Wnt pathway programs include two pipeline candidates in phase 1 development: vantictumab (anti-Fzd7) and OMP-54F28 (Fzd8-Fc). These programs are partnered with Bayer, and Bayer has an option to license vantictumab or OMP-54F28 at any point through completion of certain phase 1b trials.

Phase 1 trial of vantictumab in advanced solid tumors demonstrated single-agent activity in neuroendocrine tumors (NETs), with all three NET patients enrolled in the trial achieving clinical benefit. OncoMed described vantictumab as well-tolerated. Bone-related adverse events were observed but could be effectively managed through a protection strategy. OncoMed plans to initiate three phase 1b combination trials of vantictumab + standard chemotherapy in three solid tumor indications in 2H13.

OMP-54F28 (Fzd8-Fc) is a decoy receptor of Wnt that demonstrated strong synergy with chemotherapy in preclinical studies. Data from an ongoing phase 1 trial of OMP-54F28 in advanced solid tumors are expected at year-end 2013 or early 2014. OncoMed also plans to initiate three phase 1b clinical trials in late 2013 or early 2014 in distinct tumor types in combination with standard-of-care therapies.

Rationale for Targeting Cancer Stem Cells

Cancer stem cells refer to a subpopulation of cancer cells that are capable of continuous proliferation to sustain the growth of the tumor and have certain properties of normal stem cells. Malignant tumors are typically composed of morphologically and functionally heterogenous cell populations, and while the majority of tumor cells are destined to differentiate, a small number of cells, referred to as cancer stem cells, remain undifferentiated and possess the ability to self-renew and to differentiate to some degree into more mature non-stem-cell cancer lineages. When transplanted into immunodeficient mice, such as the non-obese diabetic/severe combined immunodeficient (NOD/SCID) mice, CSCs can regenerate a phenocopy of the original tumor and drive neoplastic proliferation, whereas the non-stem-cell tumor cells do not have tumorigenic activity.

CSCs may arise from normal tissue stem cells that have lost certain ability to limit growth after differentiation, or may arise from differentiated tumor cells that have acquired the capacity to self-renew. Irrespective of their origin, CSCs possess a number of fundamental properties that promote the growth, proliferation, and metastasis of tumors.

Description of the cancer stem cell concept appeared in the literature as early as in the 1970s and the term “cancer stem cells” was coined in the early 1980s. Research was initially hampered by the low frequency of CSCs, and this hurdle was eventually overcome with advances in cell sorting technologies and the refinement of the NOD/SCID xenotransplantation model.

The first isolation of CSC was achieved in acute myelogenous leukemia (AML), a form of blood cancer, in 1994. For solid tumors, CSC was first isolated from biopsies from patients with breast cancer in 2003. The scientific founders of OncoMed were involved in the studies that defined the existence of breast cancer stem cells. In these studies, two cell-surface markers, CD44+ and CD24-, were used to fractionate cells from breast cancer biopsies into distinct subpopulations, and it was demonstrated that only the small subpopulation of CD44+/CD24- cells was capable of initiating tumor growth when implanted into host mice, whereas the bulk of tumor cells was non-tumorigenic. As a classic case of the CSC paradigm, the tumors harvested from CSCs-injected animals recapitulated the cellular heterogeneity of the original tumor biopsy. Since then, CSCs have been identified in most of the common solid tumor types, including cancers of the colon, lung, pancreas, brain, and skin. The surface markers and frequency of CSCs from various types of cancers are summarized in Exhibit 3.

Exhibit 3: Characteristics of CSCs in Various Tumor Types

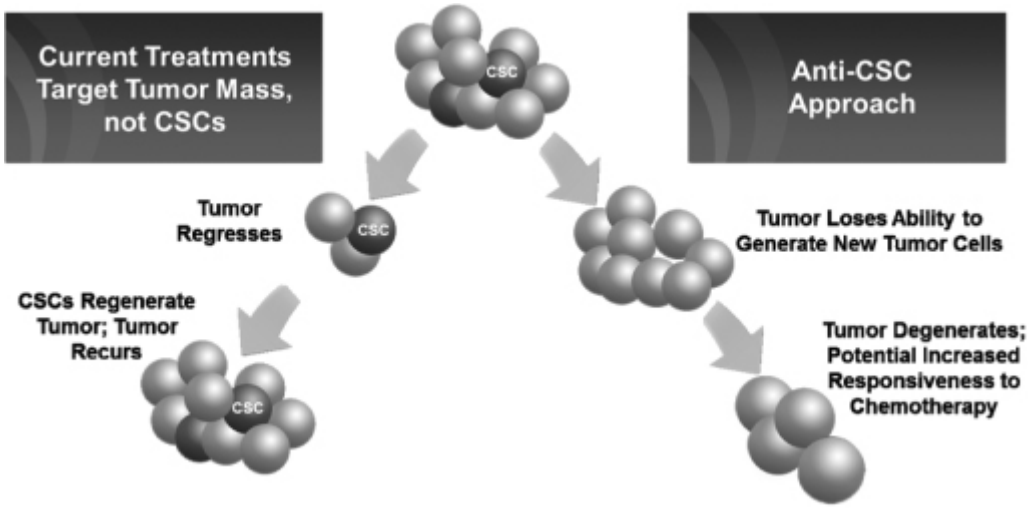
Cancer	Animal	Type of injection	CSC Frequency	Minimal number of biomarker +ve cells to obtain a tumor	Biomarkers
AML	NOD/SCID	Intravenous	0.75%	2×10^5	$CD34^+/CD38^-$
AML	NOD/SCID, NOD/SCID/$\beta_2m^{-/-}$ and NOD/SCID/IL2r$\gamma c^{-/-}$	Intravenous and intrabone	0.076%	7.5×10^3 or 10^6	$CD34^+/CD38^-$ or $CD34^+/CD38^+$
AML	NOD/SCID	Intrafemoral	0.06–0.00009% of bulk	NA	NA
Bladder	Rag2γcDKO	Intradermal	3–36.3%	100	CD44
Breast	NOD/SCID	Mammary fat pad	11–35%	200	$ESA^+/CD44^{high}/CD24^{low-neg}$
Breast	NOD/SCID	Humanized mammary fat pad	3–10%	500	$ALDH\text{-}1^+$
Brain	NOD/SCID	Intracranial	6–29%	100	$CD133^+$
Colorectal	NOD/SCID	Renal capsule	1.8–24.5%	100	$CD133^+$
Colorectal	NOD/SCID	Subcutaneous	2.60%	200	$ESA^{high}/CD44^+$
Colorectal	NOD/SCID	Subcutaneous	3.50%	25 serially passaged	$ALDH\text{-}1^+$
Head and neck squamous cell carcinoma	NOD/SCID and Rag2γcDKO	Subcutaneous	10–12%	5000	$CD44^+$
Liver	SCID	Intrahepatic	2.50%	103	$CD45^-/CD90^+$
Lung	SCID and NUDE	Subcutaneous, after in vitro expansion	0.4–1.5%	104	$CD133^+$
Lung	NOD/SCID/IL2r$\gamma c^{-/-}$	Subcutaneous	Median 15%	≤500	$lin\text{-}/CD166^+$
Melanoma	NOD/SCID	Subcutaneous	1.6-20.4%	105	$ABCB5^+$
Melanoma	Rag2γcDKO	Intradermal	2.5–41%	100	$CD271^+$
Melanoma	NOD/SCID/IL2r$\gamma c^{-/-}$	Subcutaneous	NA	1 (in 28% of cases)	NA
Melanoma	NUDE, (NOD/SCID, NOD/SCID/IL2r$\gamma c^{-/-}$)	Subcutaneous	8–11%	1000	$CD271^+$
Pancreatic	NOD/SCID	Subcutaneous and intrapancreatic	0.2-0.8%	100	$ESA^+/CD44^+/CD24^+$
Pancreatic	NUDE	Intrapancreatic	3.6 cells per high-power field	500	$ESA^+/CD133^+$

Source: BMO Capital Markets, adapted from Baccelli I and Trumpp A, *Journal of Cell biology* 2012*;* 198:281.

The characterization of CSCs has provided a new framework for therapeutic strategy for attacking cancer. CSCs have been shown to be resistant to cytotoxic chemotherapy, radiotherapy, and some targeted therapies. This is probably because CSCs are slow-cycling or quiescent cells, or because CSCs express efflux pumps that eject the drugs. In a study published in 2008, breast cancer biopsies taken from patients at the time of initial diagnosis were compared with biopsies taken after 12 weeks of treatment with docetaxel, a standard cancer chemotherapy for breast cancer. The authors of the study found that a greater percentage of cells expressed CD44, a breast cancer stem cell marker, in samples collected after 12 weeks of conventional chemotherapy. The post-treatment samples also contained an increased number of chemoresistant cells compared to pre-treatment samples.

It is postulated CSCs may be responsible for cancer recurrence by evading conventional therapy and seeding tumor growth after treatment. Thus, therapies that target non-stem-cell cancer cells but spare CSCs may have limited effectiveness, because even if the bulk of tumor cells are eliminated, cancer recurrence could ultimately occur. Conversely, therapies aimed at eliminating CSCs, either alone or in addition to bulk tumor cells, offer the promise of a durable response and a potential cure. In addition, targeting CSCs may also have impact on cancer metastasis, because CSCs have the ability to move freely and proliferate without attachment to other cells or surfaces.

Exhibit 4: Differential Outcomes of Strategies that Target CSCs vs. Tumor Bulk



Source: OncoMed Pharmaceuticals.

Although the CSC paradigm is widely accepted by now, it is certainly not without controversy. Some researchers have argued that CSC is merely an experimental artifact reflecting the limitations of using NOD/SCID mice for assessing tumorigenic potential. A key support for this argument is a study published in 2008 that compared the growth of primary melanoma cells in NOD/SCID mice verus the more immunocompromised NOD/SCID IL2R gamma null mice. The study demonstrated that although only 1 in 100,000 melanoma cells was tumorigenic in NOD/SCID mice, as many as 1 in 4 melanoma cells was tumorigenic in the NOD/SCID IL2R gamma null mice.

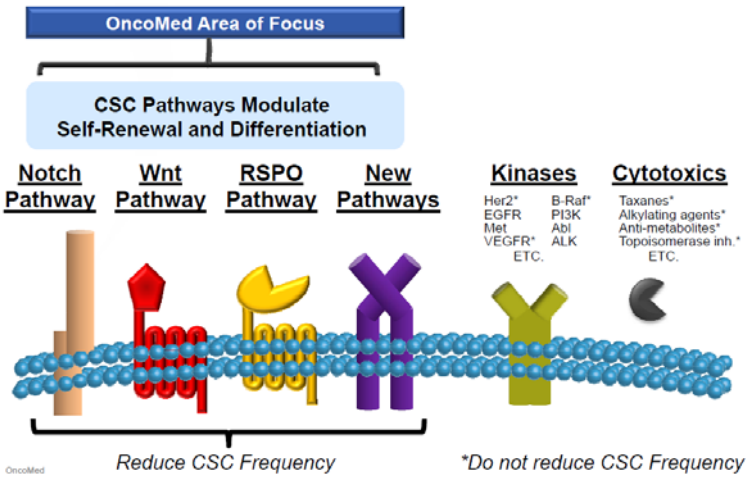
OncoMed Approach to Identifying/Targeting Cancer Stem Cells

OncoMed has developed a number of proprietary technologies for characterizing CSCs, identifying novel drug targets, and evaluating the effects of therapeutic product candidates. These proprietary technologies include (1) flow cytometry and the use of specific surface markers for identification and isolation of CSCs; (2) antibody development platforms for antibody humanization, phage display, proprietary mammalian display and bispecific antibody generation; and (3) proprietary xenograft models with freshly resected human tumors propagated in mice. OncoMed believes patient-derived models are superior to cell line-based models as used in traditional cancer research, because patient-based models are more representative of the clinical features of human tumors and offer the ability to test the therapeutic candidates on human tumors with varied genetic backgrounds.

OncoMed's overall goal in CSC therapeutics development is to specifically target key biologic pathways required for the maintenance, proliferation and survival of CSCs, such as the Notch and Wnt pathways. (See Exhibit 5.) The basic approach has been to develop antibodies and other protein-based therapeutics targeting the extracellular and cell surface proteins in these pathways. OncoMed has developed specific antibodies targeting key extracellular proteins in the Notch and Wnt pathways and is among the first companies to initiate clinical trials with antibodies blocking these pathways. In addition, OncoMed is also developing antibodies that target another pathway of interest, the RSPO-LGR pathway, in preclinical studies.

OncoMed is also exploring the approach of combining antibodies targeting the CSC pathways with chemotherapeutic agents to achieve synergy and has shown that inhibition of the Notch and Wnt pathways drives differentiation of CSCs toward a non-tumorigenic state.

Exhibit 5: Biologic Pathways in Which OncoMed Focuses Its Development Efforts



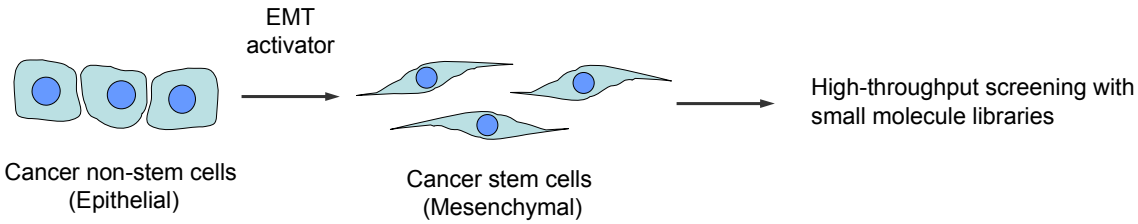
Source: OncoMed Biosciences.

Competitor Approach to Identifying/Targeting Cancer Stem Cells

Verastem

Verastem (VSTM) is a biotechnology company that develops small molecule drugs targeting CSCs. Verastem uses high-throughput screening of compound libraries to identify compounds that target cancer stem cells. A major challenge for high-throughput screening in CSCs is the limited number of CSCs, which makes it necessary to expand CSCs in culture. However, CSCs typically do not remain stable in culture and convert into other types of cancer cells over time. To overcome this problem, Verastem utilizes a technology based on the discovery of its scientific cofounders that linked the natural emergence of CSCs with a process called epithelial-to-mesenchymal transition (EMT). Using this technology, cancer *non-stem* cells can be turned into a stable population of cancer stem cells. The stable CSCs are similar to natural CSCs in that they are resistant to drugs and capable of forming new tumors. Using stable CSCs generated with this technology, Verastem has screened over 300,000 compounds for their ability to kill CSCs. Verastem has generated a pipeline of small molecule compounds using this process.

Exhibit 6: Verastem's EMT Technology for Generating CSCs for High-throughput Screening



Source: BMO Capital Markets, adapted from Verastem's SEC filing document.

Verastem's most advanced product candidate is VS-6063, or defactinib, which is a potent inhibitor of focal adhesion kinase (FAK). FAK plays a critical role in the growth and survival of CSCs. Verastem is developing VS-6063 in two indications, mesothelioma and ovarian cancer. In a phase 1 dose-escalation trial of VS-6063 in combination with paclitaxel, one patient with platinum-resistant ovarian cancer achieved a complete response (n=6). Verastem described VS-6063 at all dose levels in combination with paclitaxel as being well-tolerated. The phase 1b expansion portion of the same trial is currently ongoing and expects to enroll an additional 15 patients. The primary endpoints of the phase 1b expansion portion of the trial are safety, tolerability, and efficacy as determined by RECIST and proprietary CSC biomarkers.

Verastem's next most advanced product candidates are VS-4718, another FAK inhibitor, and VS-5584, a PI3K/mTOR inhibitor. Both VS-4718 and VS-5584 are expected to enter phase 1 development in patients with advanced cancers in 2H13.

Stemline Therapeutics

Stemline Therapeutics (STML) is a biotechnology company developing CSC therapeutics with a focus in targeting both CSCs and the tumor bulk. Stemline utilizes a set of platform technologies, StemScreen-1 and StemScreen-2, to identify novel CSC-directed compounds. StemScreen-1 involves isolating CSCs from primary tumor tissue or cell lines, and subjecting CSCs to gene expression analysis using a variety of technologies including microarray. The gene expression profile data are then interfaced with information database of compounds to identify compound classes that are likely to have an impact on CSC-specific pathways. Select compounds are then tested in functional assays. StemScreen-1 was responsible for the discovery of several of Stemline's preclinical candidates, including SL-201, SL-301, and SL-601.

StemScreen-2 is a high-throughput drug discovery platform that allows the rapid testing of many compounds on a small scale of live, labeled CSCs in their natural state. CSCs are labeled through a fluorescent reporter, the expression of which is controlled by a CSC-specific promoter. As a result, CSCs can be tracked and followed in their native environment, surrounded by tumor bulk. This could represent an advantage over screening methods that require purification of CSCs away from the rest of the tumor, which could alter the phenotype of CSCs. An initial screen of a moderately sized chemical compound library has identified several "hits," some of which were further validated using secondary functional assays to confirm anti-CSC activity. Stemline plans to further optimize Stemline-2 for larger-scale screenings.

Stemline's lead product candidates include SL-401 and SL-701, both of which are in phase 1/2 development. SL-401 is a targeted therapy that specifically recognizes and binds to cells expressing IL-3R, which is overexpressed on leukemia cells. SL-401 consists of IL-3 genetically linked to a truncated diphtheria toxin. Upon binding to IL-3R, SL-401 is internalized by target cells and the diphtheria toxin payload is released, which leads to inhibition of protein synthesis and apoptosis. IL-3R is not normally expressed on hematopoietic stem cells, but is expressed on CSCs of multiple hematologic cancers including acute myeloid leukemia (AML), chronic myeloid leukemia (CML), myelodysplastic syndrome (MDS), and T-cell acute lymphoid leukemia. IL-3R is also expressed by tumor bulk in AML, CML, MDS, and B-cell acute lymphoid leukemia. SL-401 is currently being developed for the orphan indications of blastic plasmacytoid dendritic cell neoplasm (BPDCN), a rare hematologic cancer, third-line AML, and other hematologic cancers. SL-401 demonstrated single agent anti-tumor activity in a phase 1/2 trial in patients with advanced hematologic cancers. A single cycle of SL-401 induced 3 complete responses (CRs) in relapsed/refractory BPDCN patients (n=5) and 2 CRs in relapsed/refractory AML patients (n=59). In addition, a single cycle of SL-401 at therapeutically relevant doses improved the median overall survival (OS) by more than 3x compared with the historical data for similar patients receiving traditional treatments.

SL-701 is a subcutaneously-delivered brain cancer vaccine comprising synthetic peptides that correspond to epitopes of brain cancer targets IL-13R alpha2 and EphA2. A mutation was introduced into the synthetic peptide for IL-13R alpha2 to increase immunogenicity. Both IL13-R alpha2 and EphA2 were overexpressed on brain cancer cells, and Stemline has determined that EphA2 is also expressed on brain CSCs. SL-701 demonstrated single agent clinical activity

in pediatric patients with glioma in a phase 1/2 trial, in which 86% (19/22) of evaluable patients achieved sustained durable tumor reductions or disease stabilizations, including three patients who achieved durable partial responses (PRs).

Review of OncoMed Pipeline

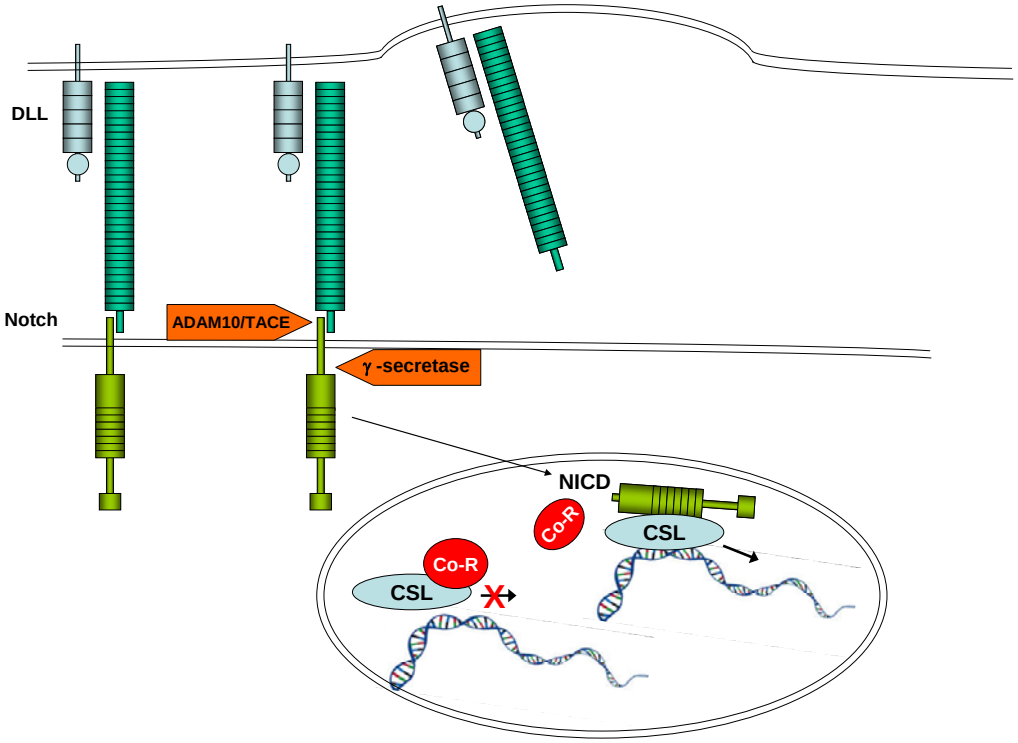
Targeting DLL4 With Demcizumab

Demcizumab (OMP-21M18) is a humanized monoclonal antibody (mAb) that targets the Notch signaling pathway by binding to and inhibiting Delta-like ligand 4 (DLL4).

The evolutionarily conserved Notch pathway controls cell fate decisions and critically regulates key functions during embryonic development as well as stem cell maintenance and differentiation in adult tissues. The mechanism of Notch signaling is quite distinctive. Both Notch receptors and ligands are transmembrane proteins, thus the interaction between Notch receptors and ligands enables short-range communication between adjacent cells.

In mammals, there are four Notch receptors, named Notch1, Notch2, Notch3, and Notch4, and five canonical ligands, named DLL1 (Delta-like ligand 1), DLL3, DLL4, Jagged1, and Jagged2. Notch receptors are heterodimers consisting of an extracellular subunit non-covalently bound to a transmembrane subunit, with both subunits derived from a single precursor after its cleavage by a protease. Ligand binding to the extracellular subunit of Notch promotes two proteolytic cleavage events in the Notch receptor. The first cleavage, catalysed by ADAM-family metalloproteases, occurs just outside the membrane, and this cleavage releases the extracellular portion of Notch, which continues to interact with the ligand. (See Exhibit 7.) The second cleavage by gamma-secretase occurs just inside the inner leaflet of the cell membrane, and this cleavage releases the active Notch intracellular domain (NICD). The NICD translocates to the nucleus and interacts with the transcription factor complex CSL (consisting of CBF-1, Supporessor of Hariless, Lag-1), and helps to convert the transcriptional co-repressor (CoR) complex into an activator complex, thereby inducing the expression of a panel of Notch target genes. (See Exhibit 7.)

Exhibit 7: Schematic of the DLL/Notch Pathway



Source: BMO Capital Markets, adapted from Thurston et al. Nat Rev Cancer 2007, 7:327.

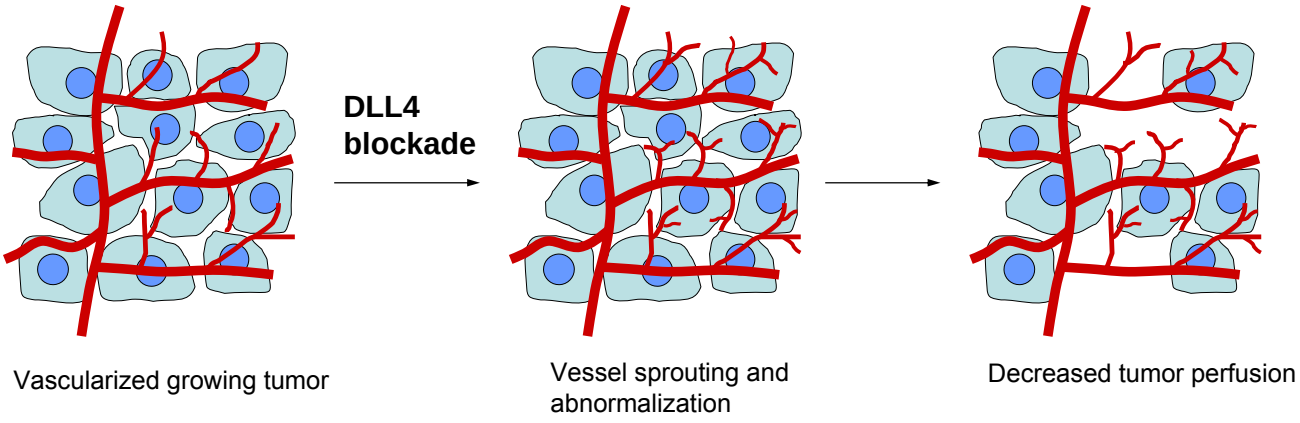
Dysregulation of the Notch pathway is linked to genetic disorders and cancer. Genetic mutations in Notch or those that upregulate Notch have been associated with a wide range of cancers, including cancers of breast, prostate, pancreatic, lung, cervical, and colon. Most notably, activating mutations in Notch are believed to be responsible for more than 50% of T-cell acute lymphoblastic leukemia (T-ALL) cases.

DLL4 is an important component of Notch-mediated stem cell renewal and vascular development, and inhibition of DLL4 has been shown to result in broad spectrum of anti-tumor activity. To date, two mechanisms have been proposed to explain the effects of DLL4 inhibition on tumor growth. The first mechanism involves DLL4's biological role in angiogenic sprouting, or branching of blood vessels.

In normal embryonic development, DLL4/Notch mediates lateral inhibition to coordinate the behavior of endothelial cells to form branches. Central to this process is the decision of the endothelial cell to become a tip cell or a stalk cell. To form new blood vessels, endothelial cells need to migrate away from established blood vessels. Endothelial cells that lead this migration, known as tip cells, are highly invasive and migratory. The endothelial cells that follow the tip cells, known as the stalk cells, are less migratory but highly proliferative. VEGF signaling induces single endothelial cells to become tip cells of emerging angiogenic sprouts, and these tip cells then suppress tip-cell features in adjacent stalk cells via DLL4/Notch-mediated lateral inhibition.

The expression of DLL4 has been found to be higher in tumor blood vessels than in the tumor cells or the surrounding normal tissue. Consistent with DLL4's role in embryonic development, inhibition of DLL4 in tumor blood vessels has been demonstrated to cause increased sprouting, resulting in an increased number of small vessel branches. These highly branched and sprouted vessels, however, are poorly functional, as demonstrated by poor perfusion using intravascular tracers. As a result, the disorganized vascular structures in tumors in which DLL4 has been blocked are inadequate to support tumor growth.

Exhibit 8: Blocking DLL4 Leads to Increased Sprouting and Formation of Poorly Functional Vessel Branches



Source: BMO Capital Markets, adapted from Thurston et al. *Nat Rev Cancer* 2007, 7:327.

A second mechanism through which inhibition of DLL4 has a negative impact on tumor growth was proposed by OncoMed scientists (Hoey T. et al., *Cell Stem Cell* 2009, 5:168). Using a xenograft tumor model of human colon tumor in NOD-SCID mice, the authors demonstrated that treatment of established tumor with an anti-human DLL4 mAb, demcizumab (OMP-21M18), caused a decrease in tumor growth, and that the combination of anti-human DLL4 mAb + chemotherapeutic agent irinotecan had an augmented effect than either agent alone. Because the anti-human DLL4 antibody (demcizumab) did not cross-react with mouse DLL4, it was unlikely that the anti-tumor effect of anti-DLL4 was mediated through a dysregulation of angiogenesis. The authors therefore examined the effect of DLL4 inhibition on the tumor, specifically on the colon cancer stem cells (CSCs), which were identified as ESA+/ CD44+/CD166+. The authors demonstrated that treatment with anti-human DLL4 reduced the frequency of CSCs in recipient mice compared with control, and that treatment with anti-human DLL4 mAb + irinotecan further decreased the frequency of CSCs (treatment with irinotecan alone increased the frequency of CSCs compared with control). The authors then demonstrated an augmented anti-tumor effect could be achieved by inhibiting DLL4/Notch signaling in both CSCs and host blood vessels, using anti-human DLL4 mAb + anti-mouse DLL4 mAb. This finding confirmed prior findings of DLL4's effect on angiogenesis and demonstrated that the two anti-tumor mechanisms of DLL4-inhibiotion could operate simultaneously. Obviously, when a human cancer patient is treated with anti-human DLL4 mAb, both anti-tumor

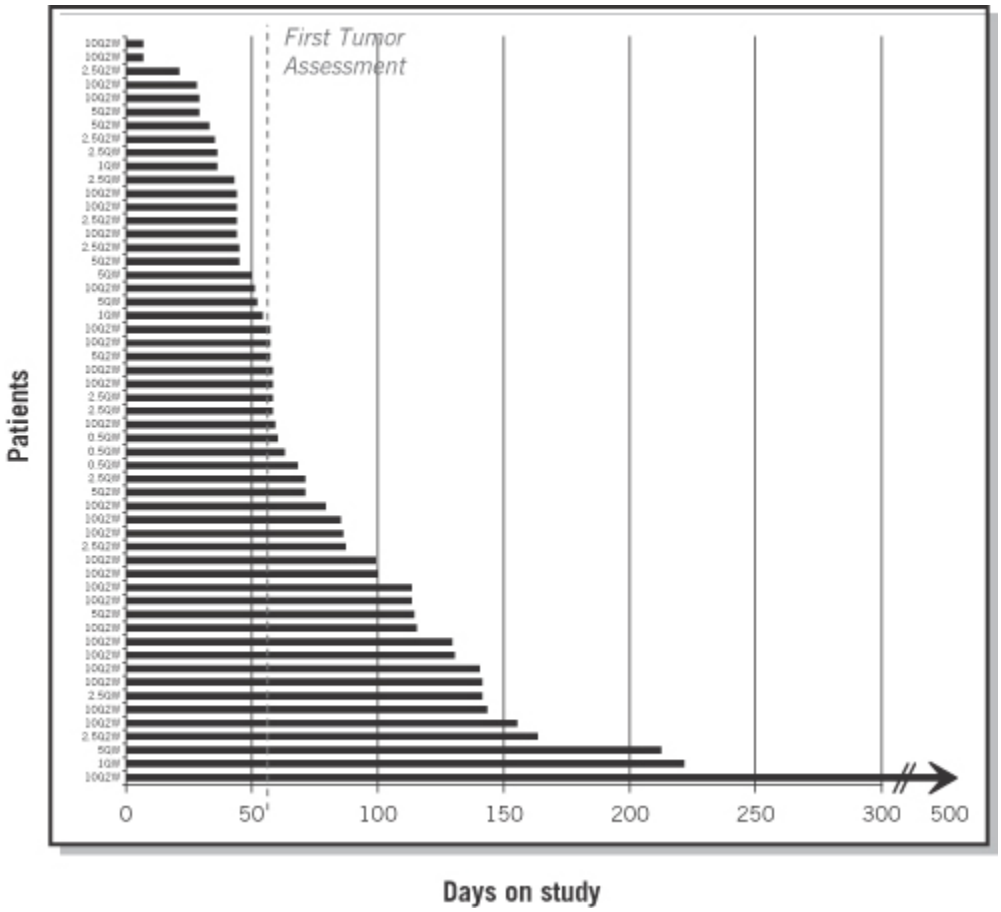
mechanisms will be triggered. Lastly, the authors showed that adding irinotecan to the dual mAb treatment further increased the anti-tumor effect.

From 2008 to 2011, OncoMed conducted a phase 1a trial of demcizumab in 55 patients with advanced solid tumors. These patients had been heavily pretreated, with a mean of four prior therapies. In this phase 1a trial, one refractory pancreatic cancer patient achieved partial response (PR), and stable disease (SD) was observed in patients with refractory NSCLC, colorectal cancer, head and neck cancer, sarcoma, melanoma, renal cell carcinoma, and other solid tumors. In the cohort receiving the highest dose (10 mg/kg every other week), the disease control rate (DCR, defined as the proportion of patients with RECIST PR and SD) was 64% out of 25 evaluable patients.

PR and SD were defined according to the Response Evaluation Criteria in Solid Tumors (RECIST) criteria: PR means that there has been at least a 30% decrease in the sum of the diameters of measured tumor lesions compared to baseline, and that there has been no growth of new or non-measurable tumor lesions; SD means that there has been less than a 30% decrease and no more than a 20% increase in the sum of the diameters of measured tumor lesions compared to baseline, and that there has been no growth of new target or non-measurable tumors.

Furthermore, 31% of patients (17/55) treated in the phase 1a trial had disease control of ≥3 months on study, including a patient with refractory ovarian cancer who had progressed previously on 12 chemotherapy regimens, but achieved SD lasting more than 570 days with demcizumab treatment. Exhibit 9 summarizes the number of days each patient remained on the study.

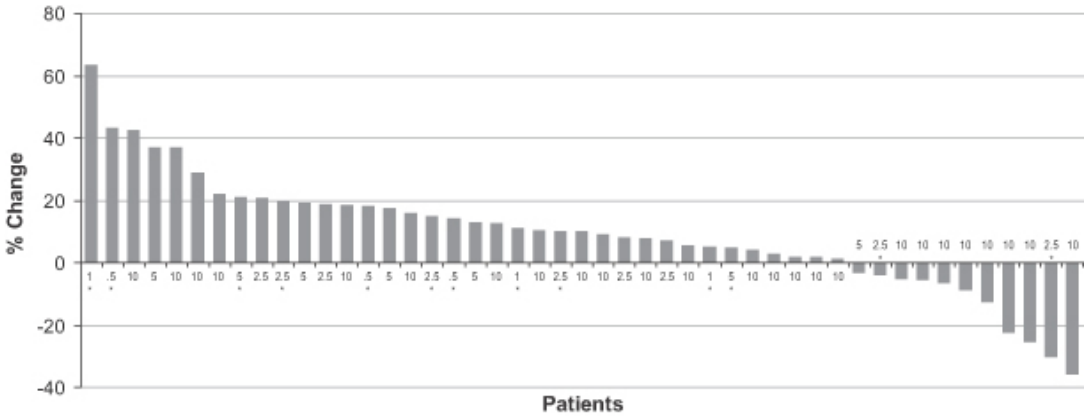
Exhibit 9: Number of Days Each Patient Remained on the Phase 1a Study of Demcizumab



Source: OncoMed Pharmaceuticals.

A commonly used and widely accepted efficacy measure in early oncology clinical trials is best percent change. In the phase 1a trial, radiographic assessments of the tumor were performed prior to start of treatment start (baseline), around day 56 of the trial, and then every 12 weeks while on the trial. At each assessment, the sum of the size of the target lesions (cm) was compared to that of the baseline, and a percent change was calculated. The "best percent change in target lesions size" is the lowest percentage change value among all of the assessed time points. Note that the best percent change measure is distinct from RECIST response, which takes into consideration non-target (non-measurable) lesions, the occurrence of any new lesions (target or non-target lesions) and clinical data. Exhibit 10 depicts in a waterfall format the values of best percent change in measured tumor lesions for the 47 patients enrolled in the phase 1a trial who had an assessment at baseline and at a subsequent tumor assessment time point. Notably, several of the patients in the phase 1a trial had decreases in their tumor measurements.

Exhibit 10: Best Percent Change in Measured Target Tumor Lesions in the Phase 1a Trial of Demcizumab



In phase 1a experience, the most common treatment-related adverse event was hypertension, which was experienced by approximately a third of the patients. Other treatment-related adverse events across the dose levels tested that occurred in ≥10% of patients were: fatigue, anemia, diarrhea, headache, nausea, hypoalbuminemia, blood pressure increase, dizziness, and dyspnea. The number of patients who discontinued the phase 1a study and the reasons for discontinuation are summarized in Exhibit 11. Approximately 36% of patients (20/55) discontinued because of adverse events and 56% of patients (31/55) discontinued because of progressive disease.

Exhibit 11: Study Discontinuation in the Phase 1a Trial of Demcizumab

Reasons for Discontinuation	n (N=55)	%	Time of Discontinuation
Progressive Disease	31	56%	Between days 28 and 219 on study
Adverse Event	20	36%	Between days 3 and 139 on study
Investigator Decision	2	4%	Day 211 and day 518
Declining Performance Status	1	2%	Day 42
Withdrawal of Consent	1	2%	Day 85

Source: BMO Capital Markets and OncoMed Pharmaceuticals.

Clinical congestive heart failure was observed in the phase 1a trial, particularly in patients who received high doses of demcizumab with more frequent administration for prolonged periods of time (≥100 days). Three patients developed Grade 3 clinical congestive heart failure, one patient developed Grade 4 clinical congestive heart failure, and one patient developed Grade 3 right ventricular failure (Grade definition followed the Common Terminology Criteria for Adverse Events, or CTCAE). The cardiac function of all of these patients improved after discontinuation of demcizumab and initiation of cardiac medications.

As a result of the cardiac adverse events, FDA placed the demcizumab phase 1a program on a partial clinical hold, which meant that new patients could not be started in the phase 1a trial in the US but patients who had already been treated could continue to receive treatment. The partial clinical hold was lifted in December 2012 after FDA reviewed new data from other ongoing trials and risk mitigation strategies proposed by OncoMed (discussed in details later in this section). The phase 1a trial was completed in November 2011.

Three phase 1b clinical trials of demcizumab in combination with chemotherapy were initiated in 2010. One of these phase 1b trials, which was evaluating demcizumab + FOLFIRI as a first- or second-line therapy for colorectal cancer, was closed owing to resource prioritization. The other two phase 1b trials are ongoing: one in first-line advanced pancreatic cancer evaluating demcizumab in combination with standard-of-care gemcitabine, and the other in first-line advanced non-small lung cancer (NSCLC) evaluating demcizumab in combination with standard-of-care carboplatin and ALIMTA (pemetrexed).

After the cardiac adverse events occurred in the phase 1a trial, OncoMed paused the ongoing phase 1b trials and amended the trials in New Zealand and Australia to include a risk mitigation plan to enhance the therapeutic index of demcizumab in an effort to maximize efficacy and manage tolerability. The risk mitigation plan included intermittent dosing of the drug, blood testing and echocardiography, cardiac monitoring using B-type natriuretic peptide (BNP), and intervention with cardioprotective medication like angiotensin-converting enzyme (ACE) inhibitors. OncoMed noted increases in BNP levels in patients who developed declines in left ventricular ejection fraction (LVEF), and believes that BNP represents a biomarker that allows for early intervention with cardioprotective medication, such as ACE-inhibitors or carvedilol. OncoMed also believes that intermittent dosing of demcizumab may enhance its therapeutic index in combination with chemotherapy. OncoMed has obtained support for its risk mitigation plan from phase 1b investigators and Independent Ethics Committees in Australia, New Zealand, and Europe, and notified the relevant clinical authorities in these regions.

As of May 15, 2013, the phase 1b trials combining demcizumab with chemotherapy have enrolled and treated a total of 59 patients. Across all dose levels tested, fatigue was the most common treatment-related adverse event in the phase 1b trials and was experienced in approximately 42% of the patients. Other treatment-related demcizumab adverse events (including all CTCAE Grades) occurring in ≥10% of patients were: nausea, vomiting, hypertension, neutropenia, decreased appetite, increased BNP levels, pulmonary hypertension, thrombocytopenia, diarrhea, peripheral edema, anemia, dyspnea, constipation, increased alanine aminotransferase, and rash.

Twenty one patients in the pancreatic cancer trial discontinued the study as of May 15, 2013, and the reasons for discontinuation are as follows: progressive disease (8 patients, between day 11 and day 168 on study); adverse event (6 patients, between day 23 and day 168 on study); discontinued when the phase 1b trial was paused (3 patients, between day 32 and day 58 on study); withdrew consent (2 patients, between day 16 and day 19 on study); clinical deterioration (1 patient, on day 21 on study); and death (1 patient, on day 50 on study).

Twenty five NSCLC patients discontinued the study as of May 15, 2013, and the reasons for discontinuation are as follows: progressive disease (12 patients, between days 35 and 224 on

study); discontinued when the phase 1b trial was paused (5 patients, between days 21 and 133 on study); adverse event (7 patients, between days 21 and 189 on study); and clinical deterioration (1 patient, day 140 on study).

Seven colorectal cancer patients discontinued, including 4 patients who discontinued when the phase 1b trial was paused (between days 56 and 140 on study) and 3 patients who discontinued because of progressive disease (between days 27 and 113 on study).

Exhibit 12: Study Discontinuation Across Three Phase 1b Trials of Demcizumab

	Pancreatic Cancer Trial	NSCLC Trial	Colorectal Cancer Trial
Progressive Disease	8	12	3
Adverse Event	6	7	
Discontinuation when phase 1a	3	5	4
Withdrawal of Consent	2		
Clinical Deterioration	1	1	
Death	1		
Total discontinuation	21	25	7

Source: BMO Capital Markets and OncoMed Pharmaceuticals.

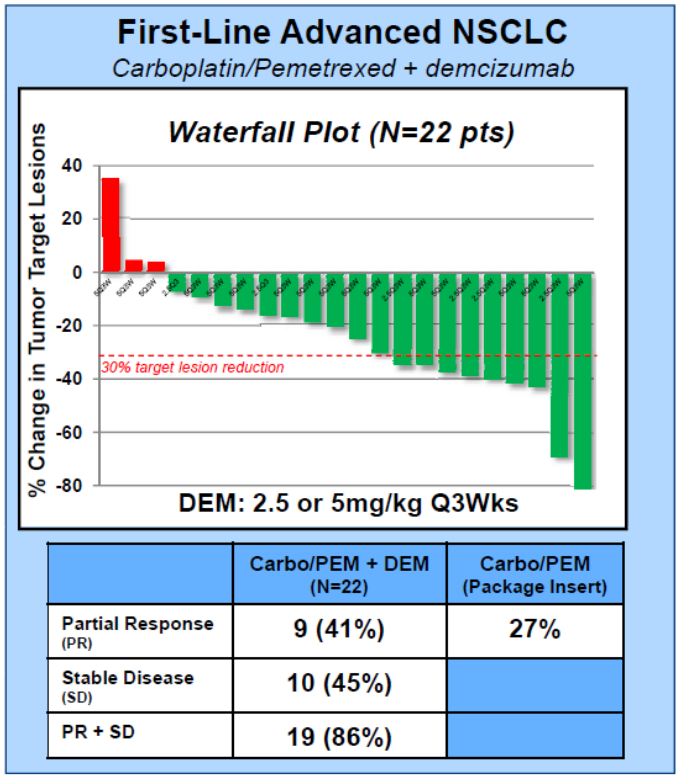
The two phase 1b trials, in NSCLC and in pancreatic cancer, are enrolling new cohorts of patients. According to clinicaltrials.gov, the phase 1b NSCLC trial plans to enroll 32 patients and the phase 1b pancreatic cancer trial plans to enroll 40 patients. As of May 15, 2013, OncoMed noted multiple early, objective responses and stable disease (SD) in these ongoing trials.

In the first-line NSCLC trial, patients with unresectable, locally advanced, recurrent, or metastatic non-squamous NSCLC received demcizumab + carboplatin + ALIMTA. Some tumor shrinkage were noted in 19 of 22 patients whose tumor responses were assessable at around day 56, with best percent reduction in the sums of measured tumors ranging from 7% to 81%. Of all assessable patients, 9 patients (41%) achieved a RECIST partial response (PR), 10 patients (45%) had SD and 3 patients (14%) had progressive disease. OncoMed referenced an expected PR rate of 27% for platinum + ALIMTA chemotherapy alone in this patient population. Some of these patients remain on the trial and continue to have clinical benefit.

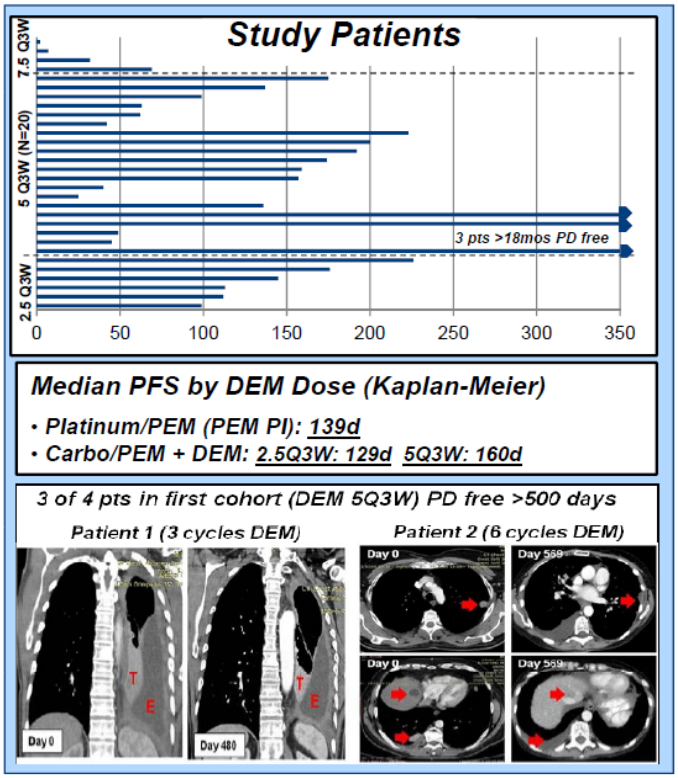
In addition, as reported by the investigators, three patients who had their therapy stopped when the trial was paused (in the first dose cohort) remained progression-free for more than 18 months. Of these three patients, one patient received three cycles of demcizumab + carboplatin + ALIMTA on study followed by three cycles of carboplatin + ALIMTA off study. Another patient received six cycles of demcizumab + carboplatin + ALIMTA on study followed by additional carboplatin + ALIMTA off-study. The third patient received only one cycle of demcizumab + carboplatin + ALIMTA on study followed by continued carboplatin + ALIMTA induction therapy and then ALIMTA maintenance therapy off study, and this patient achieved a complete response (CR) with resolution of all tumor lesions.

The median progression free survival (PFS) for patients in demcizumab's phase 1b NSCLC trial were 129 and 160 days for the 2.5 and 5 mg/kg groups, respectively. (See Exhibit 13.) OncoMed referenced an expected PFS of 139 days for platinum + ALIMTA chemotherapy alone in this patient population according to historical data.

Exhibit 13: Results of the Phase 1b First-line NSCLC Trial of Demcizumab + carboplatin + ALIMTA



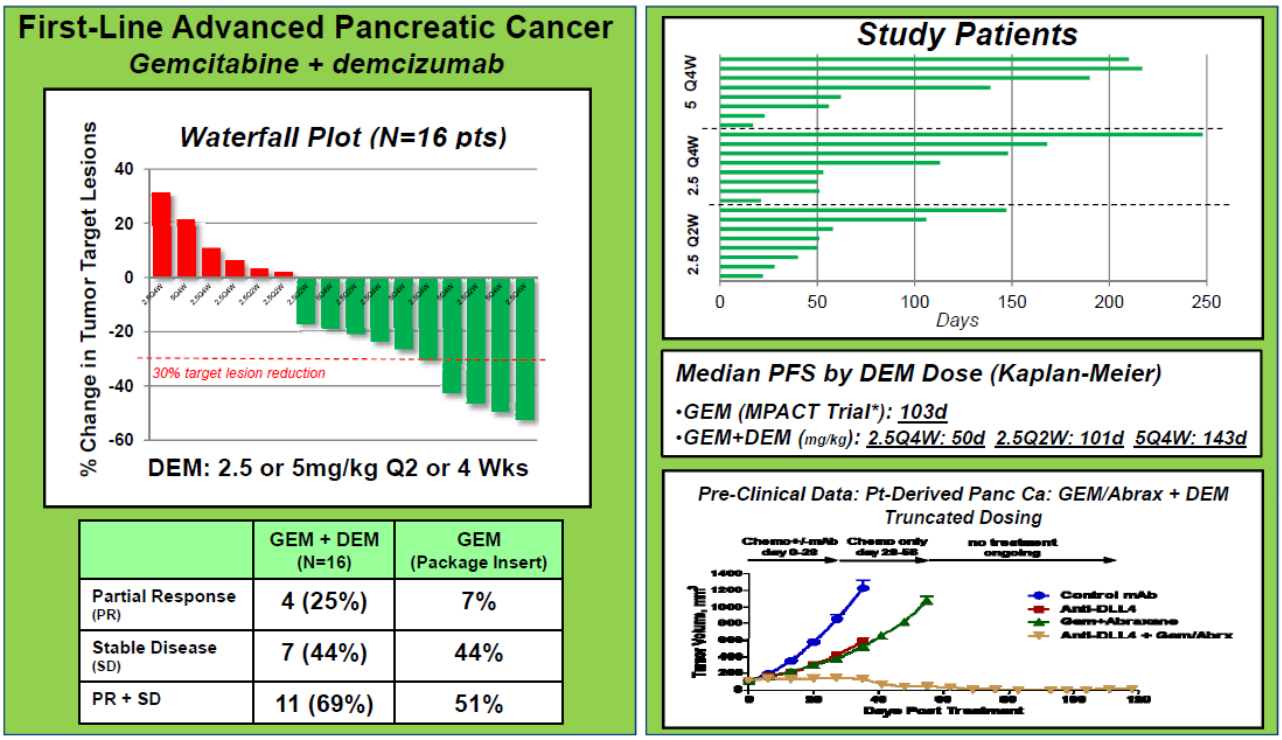
	Carbo/PEM + DEM (N=22)	Carbo/PEM (Package Insert)
Partial Response (PR)	9 (41%)	27%
Stable Disease (SD)	10 (45%)	
PR + SD	19 (86%)	



Source: OncoMed Phamaceuticals.

In the first-line pancreatic cancer trial, patients with locally advanced or metastatic pancreatic cancer (with tumor ≥1 cm and radiographically apparent on CT or MRI) are administered gemcitabine + demcizumab at doses of 2.5 mg/kg once every 14 days, 2.5 mg/kg once every 28 days, or 5 mg/kg once every 28 days. Notable tumor shrinkage was observed in 10 of the 16 patients whose tumor responses were assessable around day 56, with best percent reduction in the sums of measured tumors ranging from 17% to 53%. Of all assessable patients, four patients (25%) achieved a RECIST PR, seven patients (44%) achieved SD, and five patients (31%) had progressive disease. OncoMed referenced an expected PR rate of 7% and an expected clinical benefit (PR and SD) of 44% to 51% for gemcitabine chemotherapy alone in this patient population according to historical data. As of May 15, 2013, the median PFS for patients in the phase 1b pancreatic cancer trial were 101, 50, and 143 days for the 2.5 mg/kg every two weeks, 2.5 mg/kg every four weeks and 5 mg/kg every four weeks groups, respectively. OncoMed referenced a PFS of 104 days for gemcitabine chemotherapy alone in this patient population.

Exhibit 14: Results of the First-Line Pancreatic Cancer Trial of Demcizumab + Gemcitabine



	GEM + DEM (N=16)	GEM (Package Insert)
Partial Response (PR)	4 (25%)	7%
Stable Disease (SD)	7 (44%)	44%
PR + SD	11 (69%)	51%

Source: OncoMed Pharmaceuticals.

A Data Safety Monitoring Board (DSMB), consisting of six academic thoracic oncologists who oversee the NSCLC phase 1b trial's safety and efficacy, reviewed data from the second and third dose cohorts, and based on data from the third cohort, unanimously approved proceeding with enrollment of an expansion cohort. While the enrollment of the expansion cohort was ongoing, two patients in the third cohort who had been treated for ≥150 days developed pulmonary hypertension and heart failure. The DSMB reviewed the data and recommended the addition of two new cohorts to evaluate higher dose therapy for a more limited duration. Enrollment in the first of these two new cohorts began in 1H13.

Similarly, after reviewing patient data from the first three dose cohorts of the pancreatic cancer phase 1b trial, and as one patient in the third dose cohort with ≥125 days of treatment developed pulmonary hypertension and heart failure, the DSMB for that trial recommended adding an additional cohort to evaluate a more limited duration of treatment.

In November 2012, OncoMed submitted to FDA the interim data from these phase 1b trials, including a description of its risk mitigation strategy to minimize cardiac toxicity. As a result, FDA lifted the partial clinical hold on demcizumab in the US in December 2012.

OncoMed is also evaluating demcizumab in other solid tumor settings. OncoMed plans to initiate a phase 1b/2 trial of demcizumab + paclitaxel in platinum-resistant ovarian cancer at the MD Anderson Cancer Center (MDACC) in 2H13. OncoMed recently presented preclinical studies of demcizumab in patient-derived ovarian cancer xenografts at the American

Association for Cancer Research (AACR) Annual Meeting in April 2013. The preclinical study demonstrated that demcizumab inhibited tumor growth and reduced CSC frequency in a panel of patient-derived ovarian cancer xenograft models. Similar to the strategy used in the preclinical study of demcizumab in colon cancer, the ovarian cancer preclinical study utilized anti-human DLL4 (demcizumab) and anti-murine DLL4 to block Notch signaling in both the tumor and stromal/vascular cells in the xenografts. OncoMed found that anti-DLL4 treatment significantly inhibited tumor growth both as a single agent and in combination with paclitaxel. Serial transplantation studies demonstrated that anti-DLL4 + paclitaxel greatly reduced CSC frequency, whereas treatment with paclitaxel alone had the opposite effect and increased ovarian CSC frequency. These preclinical data suggest that anti-DLL4 treatment may sensitize chemoresistant ovarian cancer stem cells to therapy.

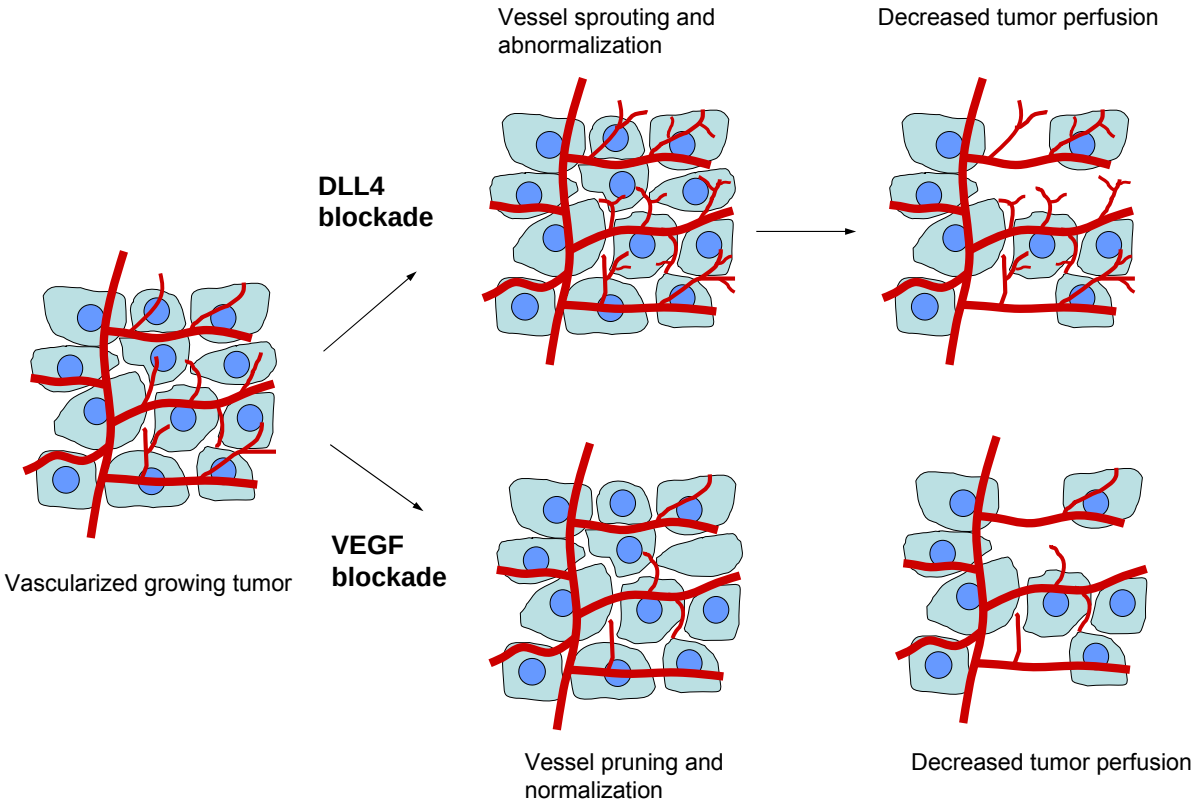
OncoMed's demcizumab patent portfolio includes an issued US composition-of-matter patent that expires in 2028, seven issued foreign patents or allowed foreign patent applications, two pending US patent applications and approximately 15 additional pending foreign applications.

Potential Synergy With Bispecific DLL4/VEGF Targeting

Using proprietary bispecific antibody technology, OncoMed has developed a bispecific monoclonal antibody that targets both human DLL4 and human VEGF. This bispecfic antibody is currently in preclinical development.

Inhibition of vascular endothelial growth factor (VEGF) signaling has become a well-established strategy for anti-cancer therapies. The anti-VEGF mAb developed by Genentech (part of Roche), AVASTIN, has been approved in treating a number of solid tumors including colorectal, NSCLC, renal cell, brain and ovarian cancers. Although both VEGF and Notch/DLL4 pathways play a critical role in angiogenesis and tumor growth, these two pathways have differential effects on the vasculature: anti-DLL4 treatment induces an abnormal increase of poorly perfused blood vessels unable to support tumor growth, whereas the anti-VEGF therapy significantly decreases vasculature reducing the blood supply to tumors. (See Exhibit 15.) Therefore, a synergistic effect could be achieved in targeting both the DLL4/Notch and VEGF pathways.

Exhibit 15: Differential Treatment Effects of anti-DLL4 vs. anti-VEGF

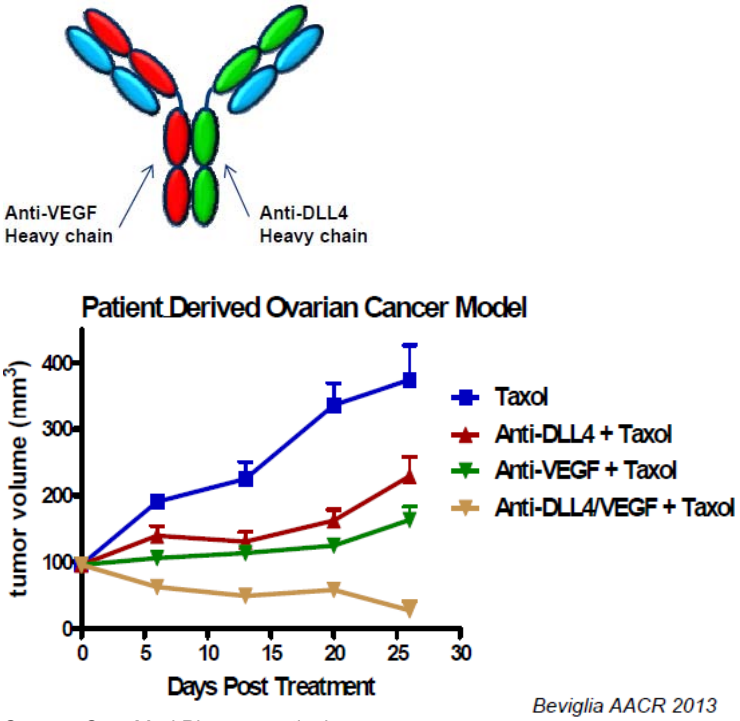


Source: BMO Capital Markets, adapted from Thurston et al. *Nat Rev Cancer* 2007, 7:327.

OncoMed demonstrated in preclinical studies that simultaneous targeting of DLL4 and VEGF with a dual specific antibody resulted in substantially improved anti-tumor activity compared to either anti-DLL4 or anti-VEGF alone. These preclinical studies involved OncoMed's DLL4/VEGF bispecific antibody, which has nanomolar affinity to human VEGF and human DLL4, and a tumor model in which human colon cancer cells derived from tumor specimens were implanted intradermally into human skin graft in NOD/SCID mice. The tumor model was selected based on its sensitivity to both the anti-human DLL4 antibody (demcizumab) and to the human VEGF inhibitor AVASTIN (bevacizumab). In these preclinical studies, demcizumab, AVASTIN, or the bispecific mAb was administered to mice intraperitoneally at a dose of 25 mg/kg weekly. The bispecific antibody caused an 87% inhibition of tumor growth, whereas demcizumab and AVASTIN caused 45% and 70% inhibition of tumor growth, respectively. As expected, AVASTIN caused a significant decrease of blood vessels, down-regulated VEGFR2, and increased hypoxia. Treatment with the bispecific antibody led to increased blood vessels, up-regulated VEGFA and VEGFR2, and enhanced hypoxia, and these effects were more pronounced compared with demcizumab. In separate experiments with mice bearing subcutaneous human colon tumors, the bispecific antibody delayed tumor recurrence following termination of chemotherapy and decreased the frequency of CSCs. These results suggested that the DLL4/VEGF bispecific antibody could have higher anti-tumor activity than

therapeutic agents that target DLL4 or VEGF alone. OncoMed plans to file an IND for this antibody in 2014.

Exhibit 16: Structure and Preclinical Data of the VEGF/DLL4 Bispecific Antibody



Source: OncoMed Pharmaceuticals.

Targeting Notch 2/3 with OMP-59R5

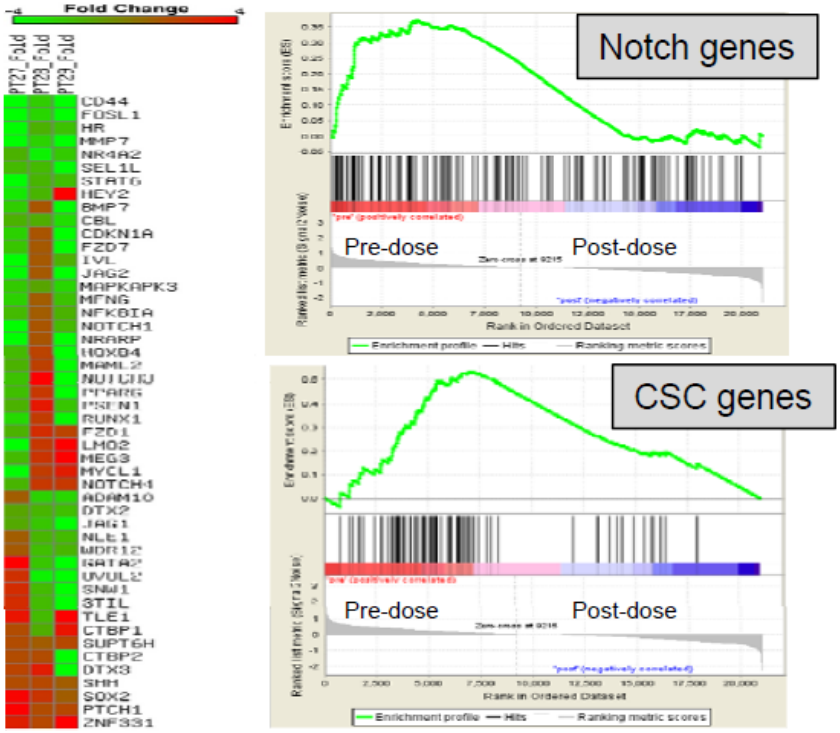
OMP-59R5 is a human antibody that binds both the Notch2 and Notch3 receptors through recognizing a conserved epitope. OMP-59R5 is originally derived from phage display (a technology licensed from MorphoSys AG). OncoMed is currently conducting two phase 1b/2 trials of OMP-59R5, one in advanced pancreatic cancer and one in small cell lung cancer.

The rationale for targeting Notch 2/3 for the treatment of cancer is two-fold. First, downregulating Notch pathway signaling could have anti-CSC effects, through the same mechanism as proposed for demcizumab's anti-CSC effect (described in the earlier section); second, targeting Notch 2/3 receptors could have an effect on the tumor microenvironment by affecting pericytes, the contractile cells that wrap around the endothelial cells of capillaries and venules. Studies have shown that Notch3, through its interaction with Jagged1, is an important player in pericyte function. Both mechanisms of actions have been confirmed by OncoMed in preclinical studies.

In December 2010, OncoMed initiated a phase 1a dose escalation trial of OMP-59R5 in patients with advanced refractory solid tumors. This trial was completed and results were presented at the ASCO Annual Meeting in June 2012 and in a plenary session at the EORTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics in November 2012. In this trial, 42 patients in eight dose-escalation cohorts received OMP-59R5 at doses of 0.5 mg/kg, 1 mg/kg, 2.5 mg/kg, and 5mg/kg administered weekly, and 5 mg/kg, 7.5 mg/kg, and 10mg/kg administered every other week and 7.5mg/kg administered every three weeks. Several patients with Kaposi's Sarcoma, adenoid cystic carcinoma, liposarcoma, triple negative breast cancer, and rectal cancer achieved prolonged stable disease for 56 or more days. OMP-59R5 was described as being generally well tolerated and three MTDs were established: 2.5mg kg weekly, 7.5mg/kg every other week and 7.5mg/kg administered every three weeks.

The dose limiting toxicity in the phase 1a trial of OMP-59R5 was diarrhea, which was less pronounced with every-other and every-three-week dosing schedules. Across the dose levels tested, diarrhea was the most common treatment-related adverse event and was observed in approximately two-thirds of patients. Other treatment-related adverse events that occurred in ≥10% of patients included fatigue, anemia, nausea, decreased appetite, hypokalemia, and vomiting, all of which were CTCAE Grade 1, 2, or 3 events. No Grade 4 or 5 events were observed on the trial. Pharmacokinetics (PK) analysis of OMP-59R5 demonstrated fast and dose-dependent clearance, whereas pharmacodynamic analyses on surrogate and tumor tissue suggested sustained Notch pathway modulation over a period of a week or more after dosing. Downregulation of Notch3 receptor was also observed in serial tumor biopsies during treatment with OMP-59R5.

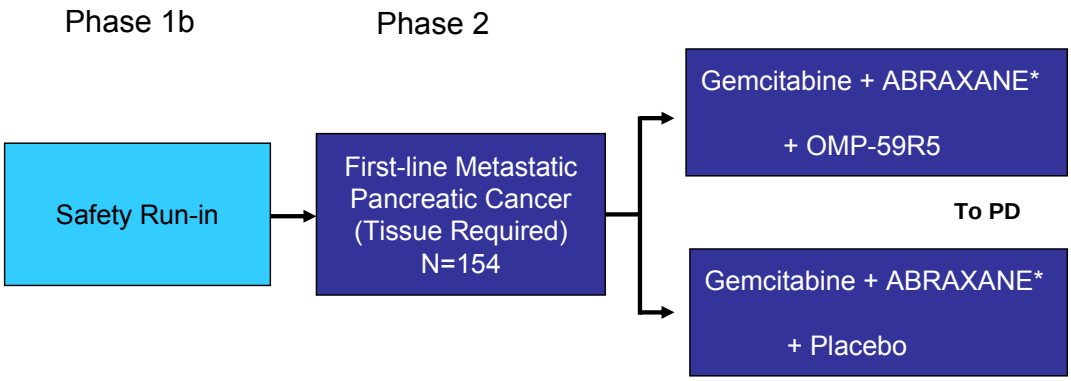
EXHIBIT 17: Decrease in Notch and CSC Pathways As Demonstrated by Serial Tumor Biopsies in the Phase 1a Study of OMP-59R5



Source: OncoMed Pharmaceuticals.

In October 2012, OncoMed initiated ALPINE, a phase 1b/2 trial of OMP-59R5 in first-line advanced pancreatic cancer patients. The trial consists of a phase 1 safety run-in to determine the maximum tolerated dose (MTD) of OMP-59R5 + gemcitabine + ABRAXANE, followed by a randomized, placebo-controlled phase 2 portion to evaluate the efficacy and safety of OMP-59R5 + gemcitabine + ABRAXANE in patients with previously untreated stage IV pancreatic cancer. The initial study design was to compare the efficacy of OMP-59R5 + standard-of-care gemcitabine vs. placebo + gemcitabine, but has since been amended as a result of the changing standard of care in pancreatic cancer, based on landmark studies presented at the ASCO Gastrointestinal Cancer Symposium in 2013 illustrating a survival benefit to the combination of gemcitabine and ABRAXANE over gemcitabine alone. The primary endpoints of the ALPINE trial are dose limiting toxicities (DLT) and MTD of OMP-59R5 + gemcitabine + ABRAXANE, as well as progression-free survival (PFS). The ALPINE trial plans to enroll 154 patients and the enrollment is ongoing. The estimated primary completion date for the ALPINE trial is January 2014, according to clincialtrials.gov.

Exhibit 18: Study Design of the ALPINE Trial

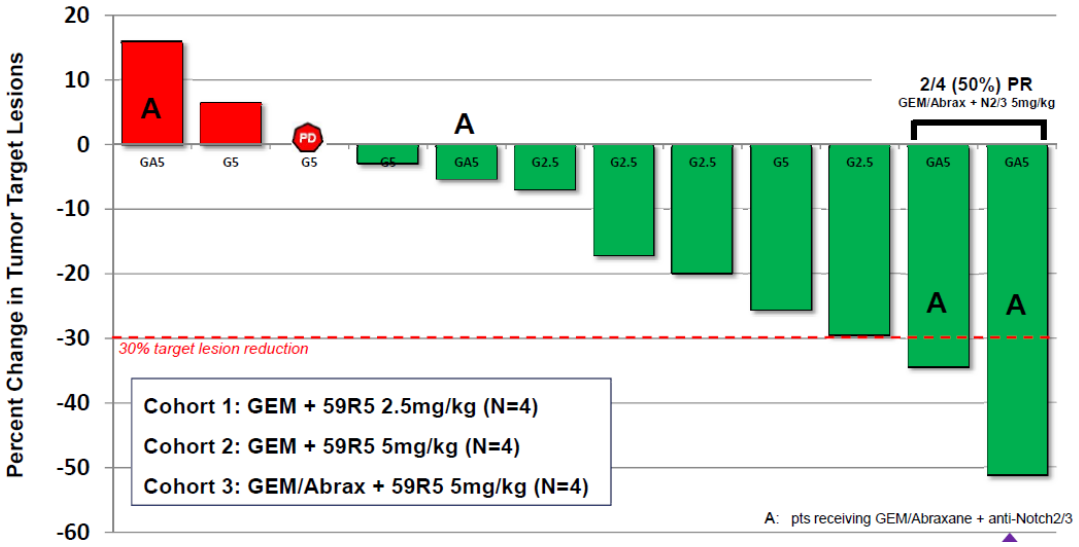


***ABRAXANE 225 mg/m2 followed by gemcitabine 1000 mg/m2 weekly for 5 weeks, 3 weeks rest**

Source: OncoMed Pharmaceuticals.

To date, clinical data are available for 12 patients who had been assessable for tumor response by RECIST criteria in the first three dose escalations cohorts (n=4 for each cohort) of the ALPINE trial. The first two cohorts were treated according to the original study protocol. The first cohort received gemcitabine + OMP-59R5 at 2.5 mg/kg every two weeks, and the best RECIST response was SD in three out of four patients as of June 21, 2013. The second cohort received gemcitabine + OMP-59R5 at 5 mg/kg every two weeks, and the best RECIST response was SD in three out of four patients. Finally, the third cohort received gemcitabine + ABRAXANE + OMP-59R5 at 5 mg/kg every two weeks, and the best RECIST response was PR in two of four patients, with SD and progressive disease in the other two patients. Exhibit 19 summarizes the best tumor response by radiographic means for target tumor lesions for the 12 patients across the first three dose escalation cohorts in the ALPINE trial. As of June 11, 2013, drug-related adverse events encountered were limited to Grade 1 or 2, including fatigue, nausea, rash, diarrhea, thrombocytopenia, and flu-like symptoms.

Exhibit 19: Best Tumor Response in the First Three Dose Escalation Cohorts in the Phase 1b ALPINE Trial in Pancreatic Cancer

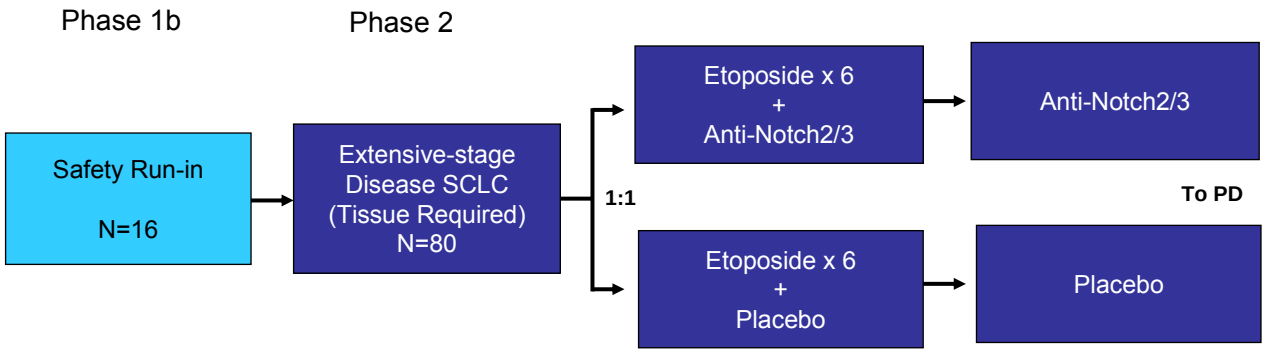


Source: OncoMed Pharmaceuticals.

In May 2013, OncoMed initiated a second phase 1b/2 trial, known as PINNACLE, in small cell lung cancer (SCLC). The PINNACLE trial evaluates OMP-59R5 + cisplatin + etoposide in first-line extensive-stage small cell lung cancer patients. Following a phase 1b dose escalation and expansion phase, a randomized, placebo-controlled phase 2 portion will proceed in the same patients to compare the efficacy of OMP-59R5 + cisplatin + etoposide for six cycles followed by single agent OMP-59R5 vs. placebo + cisplatin + etoposide for six cycles. The combination of cisplatin and etoposide is the standard-of-care for this indication. The primary endpoints of the PINNACLE trial are DLT and MTD of OMP-59R5 + cisplatin + etoposide, as well as PFS. The PINNACLE trial plans to enroll 80 patients and the enrollment is ongoing. The estimated primary completion date for the PINNACLE trial is October 2015 according to clinicaltrials.gov.

Both ALPINE and PINNACLE will include an analysis of a predicative biomarker to identify patients that might derive the greatest benefit from OMP-59R5.

Exhibit 20: Study Design of the PINNACLE Trial



*** Etoposide 100 mg/m2 on days 1-3, cisplatin 80 mg/m2 on day1, every 3 weeks**

Source: OncoMed Pharmaceuticals.

OMP-59R5 is part of OncoMed's collaboration with GSK, and GSK has the option through the completion of certain phase 2 trials to obtain an exclusive license to OMP-59R5.

OncoMed's OMP-59R5 patent portfolio includes two issued US patents, including a composition-of-matter patent expiring in 2030 and a methods of use patent expiring in 2029, and five issued foreign patents. OncoMed also has an issued US patent broadly covering OMP-59R5 expiring in 2028, an issued US patent broadly covering uses of OMP-59R5 in the treatment of cancer that expires in 2027, and an issued patent in 13 European countries that also broadly covers OMP-59R5 and its use expiring in 2027.

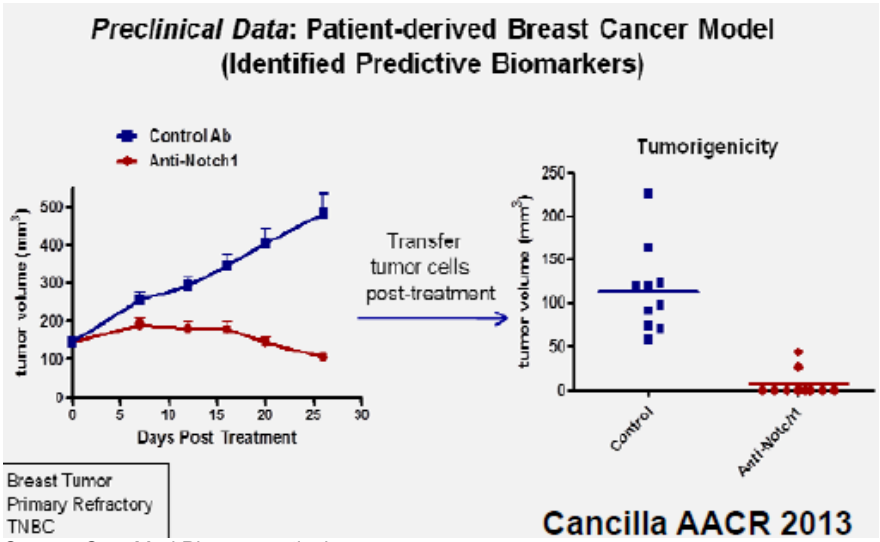
Targeting Notch1 With OMP-52M51

OncoMed is currently developing OMP-52M51, a humanized anti-Notch1 mAb that binds with high affinity and selectivity to human Notch1 and blocks Notch1 signaling. Certain types of cancers harbor mutations that lead to increased Notch1 signaling, which could be a primary driver of tumor growth as well as resistance to chemotherapy. Such activating Notch1 mutations have been characterized in T cell leukemia, chronic lymphocytic leukemia, and non-small cell lung cancer.

In preclinical studies, OncoMed developed a primary human xenograft model derived from breast tumor cells bearing an activating Notch1 mutation, and demonstrated that blocking Notch signaling with OMP-52M51 impeded tumor growth and reduced CSC frequency. (See Exhibit 21.) Interestingly, the OMP-52M51-sensitive tumor cells were derived from a patient who failed to respond to pre-operative chemotherapy and developed metastatic disease following surgery. Additionally, using an immunohistochemistry assay that detects the activated form of Notch1 by staining the Notch1 intracellular domain (ICD) and a large panel of human tumors (>600), OncoMed found elevated Notch1 ICD levels in ~30% of chemo-resistant breast cancer, a frequency that was significantly higher than in unselected breast cancer patients. Other cancer

types that OncoMed found to have elevated Notch1 levels (in 7-29% of tumor cells) include chemo-resistant gastric cancer, cholangiocarcinoma, esophageal cancer, hepatocellular carcinoma (HCC), and small cell lung cancer (SCLC). This finding suggested that Notch1 signaling may play a significant role in cancer chemoresistance, and that anti-Notch1 may hold potential as an effective therapy for chemo-resistant breast cancer and other tumors. The Notch ICD assay could also be used as a predictive biomarker to identify patients most likely to benefit from this therapy in certain hematologic malignancies and solid tumors.

Exhibit 21: Preclinical Data of OMP-52M51 (Anti-Notch1) in Human Breast Cancer Xenograft Tumor Model



Source: OncoMed Pharmaceuticals.

OncoMed is currently developing its humanized anti-Notch1 mAb, OMP-52M51, in two separate phase 1 trials in lymphoid malignancies and advanced solid tumors. The phase 1 trial in lymphoid malignancies was initiated in October 2012 and has an expected enrollment of 53 patients. The phase 1 trial in relapsed or refractory solid tumors was initiated in February 2013, and has an expected enrollment of 33 patents. The primary completion dates for both phase 1 trials are May 2014. OncoMed currently has limited clinical data on these phase 1a trials, and noted drug-related adverse events occurring in at least two patients thus far have included diarrhea and nausea. OncoMed plans to include the active Notch1 biomarker analysis in future clinical trials of OMP-52M51.

OMP-52M51 is part of OncoMed's collaboration with GSK, which has a standard option during certain time periods through the completion of specified phase 2 trials to obtain an exclusive license to OMP-52M51 or an early option during certain time periods through completion of certain phase 1 trials to obtain an exclusive license.

OncoMed's OMP-52M51 patent portfolio includes 1 issued US composition-of-matter patent expiring in 2029, 3 pending US patent applications, 5 issued foreign patents, and approximately

23 pending foreign applications. Patents that issue from the pending patent applications are expected to expire in 2029.

Targeting Wnt Pathway With Vantictumab and Fzd8-Fc

The Wnt pathway, which signals through the Frizzled (Fzd) receptors family and other co-receptors, play a key role in directing cell fate during embryogenesis and in adult stem cells. In addition, inappropriate activation of the Wnt pathway underlies the oncogenesis of numerous tumor types.

The Wnt pathway involves a large number of ligands, receptors, and coreceptors. There are 19 human Wnts, 10 Fzd receptors, and several co-receptors including low-density lipoprotein receptor-related protein 5/6 (LRP5/6). Several distinct downstream signaling cascades can be triggered by Wnt, including activation of β-catenin, known as the canonical pathway, as well as the planar cell-polarity pathway and the Ca2+ pathway.

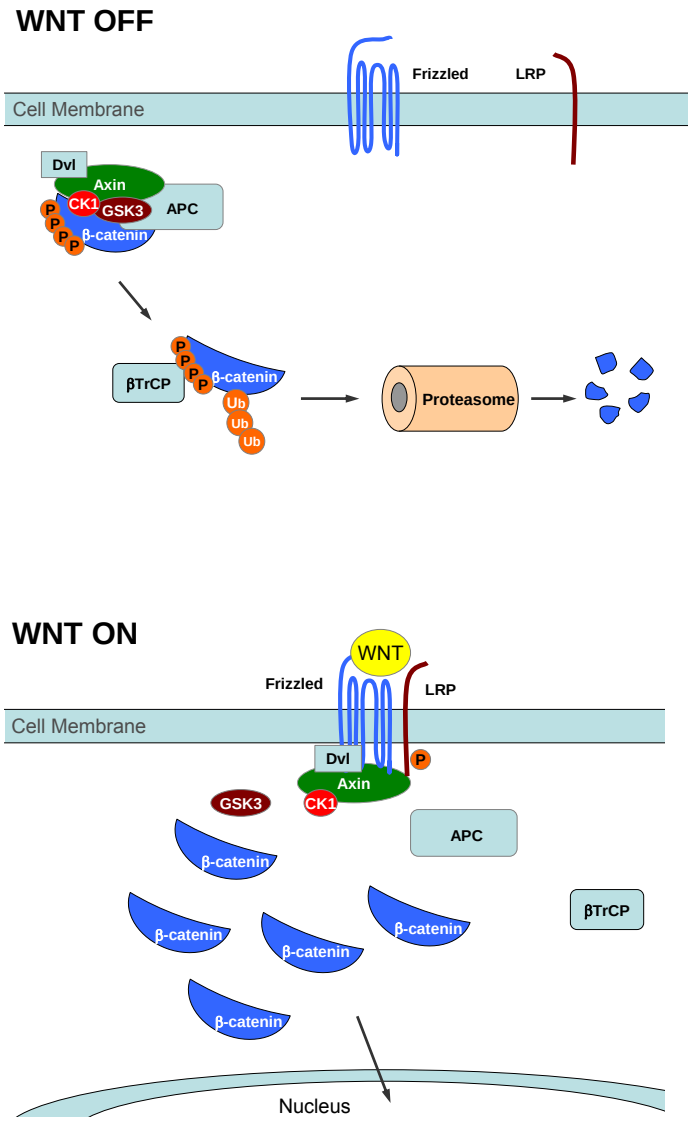
Wnt proteins are ~40kD secreted glycoproteins with many conserved cysteines. Wnt proteins are lipid modified, and the lipids on Wnts are required for receptor binding and efficient signaling. The receptor for Wnt is a heterodimer consisting of Frizzled (Fzd) and an LRP5/6 protein. The mammalian Fzd proteins are seven-transmembrane proteins with large extracellular cysteine-rich domains (CRD) that provide a primary platform for Wnt binding, including a lipid-binding hydrophobic groove. The interaction between Wnt and Fzd is promiscuous: a single Wnt can bind multiple Fzd proteins, and a single Fzd can bind multiple Wnt proteins. The LRP5 and LRP6 proteins are single transmembrane coreceptors that associate with the Fzd proteins.

In the canonical pathway, Wnt signaling regulates the protein levels of β-catenin, an integral E-cadherin cell-cell adhesion adaptor protein and transcriptional co-regulator. As shown in Exhibit 22, under normal conditions, β-catenin binds to a cytosolic scaffold known as the destruction complex, consisting of the adenomatous polyposis coli protein (APC), Axin, and glycogen synthase kinase-3 (GSK3). The destruction complex phosphorylates β-catenin, and phosphorylated β-catenin then undergoes ubiquitination and is subsequently degraded by the proteasome. When Wnt ligand binds to Fzd, the coreceptor LRP5/6 is recruited to form a complex with Wnt-bound Fzd. LPR proteins becomes phosphorylated in the cytoplasmic tail, and resulting in association with Axin. The cytoplasmic part of Fzd interacts with Dishevelled (Dvl), which in turn interacts with Axin and facilitates the binding between the LRP tail and Axin. The sequestration of Axin leads to the disintegration of the destruction complex, resulting in accumulation of β-catenin in the cytosol. Stabilized β-catenin then traffics into in the nucleus and promotes transcription of genes related to proliferation and survival by acting as a coactivator for the TCF/Lef family of transcription factors, upon replacement of the transcriptional Groucho repressors. The complexes of TCF/Lef and β-catenin are the ultimate effectors of the canonical signaling pathway.

The interaction between Wnt ligands and Wnt receptors is regulated by a series of extracellular antagonists, which antagonize the Wnt ligand in various ways. Secreted Frizzled-related proteins (sFRPs) and Wnt inhibitory protein (WIF) bind to Wnt ligands and block their

interaction with Wnt receptors, whereas proteins of the Dickkopf (DKK) and the WISE/SOST families antagonize signaling by binding to LRP5/6.

Exhibit 22: Schematic Diagram of the Canonical Pathway of Wnt Signaling



Source: BMO Capital Markets, adapted from Clevers H and Nusse R, Cell 149:1192, 2012.

The Wnt pathway is required for the self-renewal of adult stem cells in a variety of tissues. For example, inhibition of Wnt signaling eliminates hair follicles and other skin appendages such as the mammary gland, and over-expression of Axin reduces the number of transplantable stem cells. Conversely, activation of the Wnt pathway can lead to expansion of stem cells in hair follicle and hematopoietic systems.

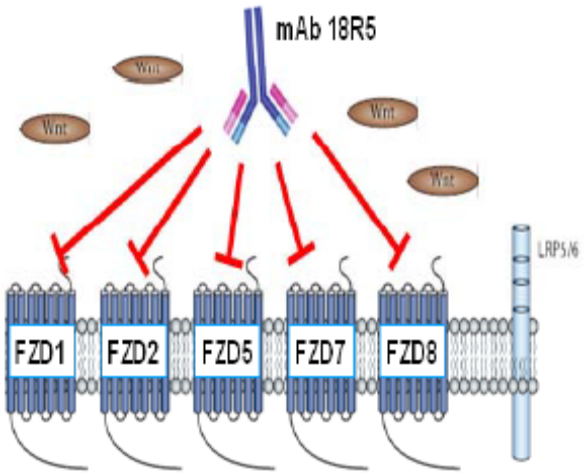
Given the involvement of Wnt signaling in stem cell self-renewal, it is perhaps not surprising that inappropriate activation of the Wnt pathway leads to cancers in various tissue types, particularly in those that normally depend on Wnt for self-renewal or repair. Germline mutations in the APC gene are associated with familiar adenomatous polyposis, a hereditary cancer syndrome, and patients with hereditary Axin2 mutations are predisposed to colon cancer. Furthermore, inactivating APC mutations are associated with an overwhelming majority of colorectal cancers, whereas loss-of-function mutations in Axin cause hepatocellular carcinomas and oncogenic β-catenin mutations lead to colon cancer, melanoma, as well as a wide variety of other solid tumors.

The Wnt pathway also plays a role in bone biology, and Wnt signaling is known to activate osteoblasts. Mutations in the LRP5 extracellular domain are associated with hereditary high bone mass diseases, whereas a loss of function mutation in LRP6 is associated with a hereditary disorder with one of its characteristics being osteoporosis. In multiple myeloma, local DKK1 production by malignant plasma cells induces osteolytic bone lesions and fractures.

Vantictumab (OMP-18R5)

Vantictumab (OMP-18R5) is a fully human mAb that interacts with the extracellular domain of 5 out the 10 Frizzled receptors, including Fzd1, Fzd2, Fzd5, Fzd7, Fzd8 (Exhibit 23). Vantictumab blocks the canonical pathway of Wnt signaling and is currently in phase 1 development. Vantictumab was identified by phage display screening of antibodies against Fzd7.

Exhibit 23: Schematic of Vantictumab's Mechanism of Action

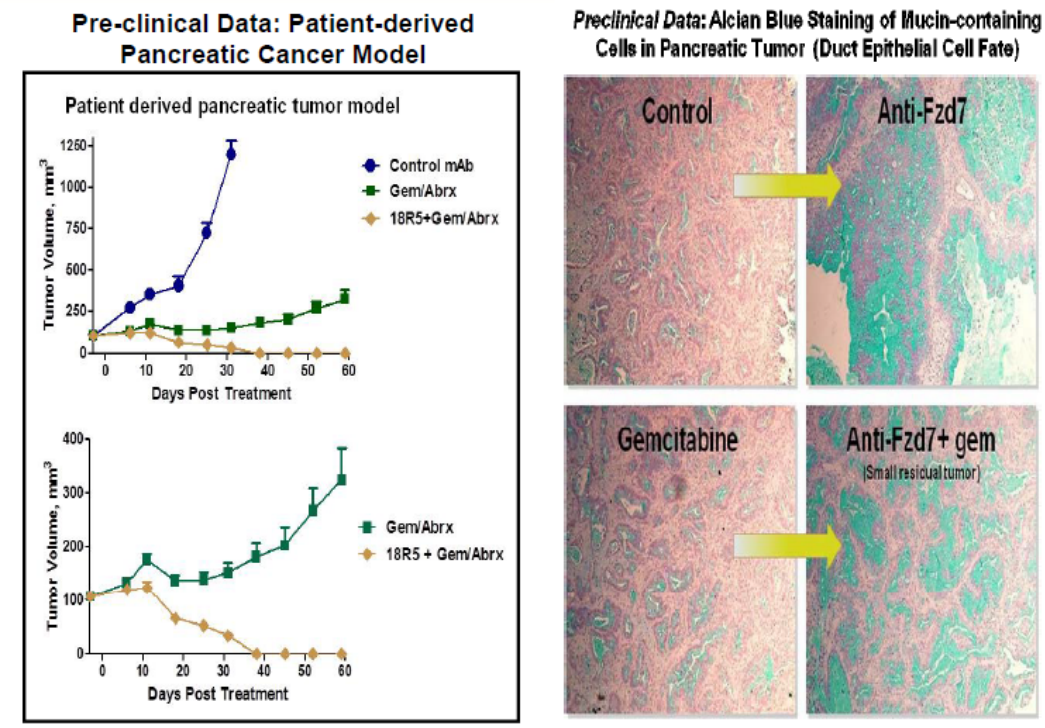


Source: OncoMed Pharmaceuticals.

In human tumor xenograft models, vantictumab treatment inhibited growth of several types of human tumors, including breast, pancreatic, and lung tumors. Vantictumab demonstrated activity in 6 of 11 pancreatic tumors, 3 of 6 breast tumors, and 7 of 8 non-small cell lung tumors tested. Consistent with its mechanism of action, vantictumab did not inhibit the growth of colon

tumors harboring APC and β-catenin mutations, but demonstrated strong activity against colon tumors with wild-type APC and β-catenin. OncoMed also reported significant synergy between vantictumab and several standard-of-care chemotherapies, including TAXOL in NSCLC and breast cancer models, irinotecan in colon cancer models, and gemcitabine in pancreatic cancer models (Exhibit 24).

Exhibit 24: Preclinical Data for Vantictumab Demonstrated Synergy with Chemotherapy Agents



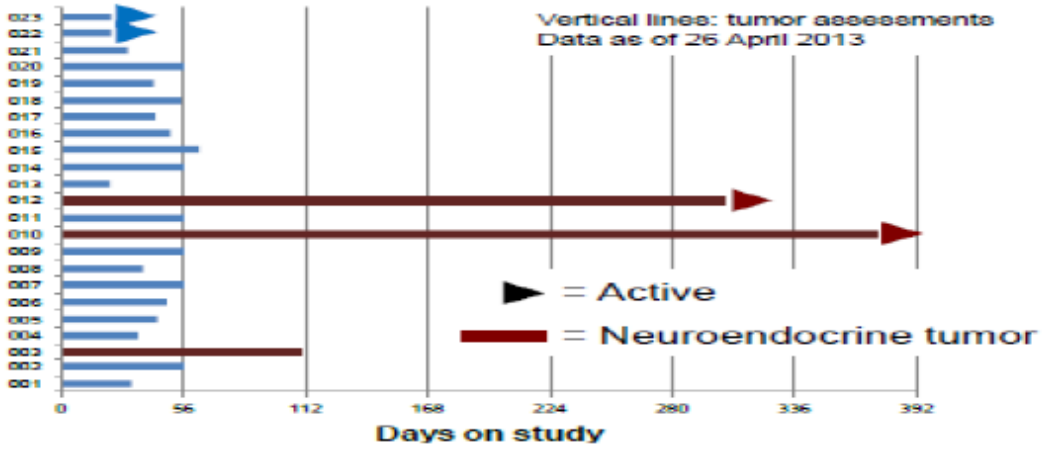
Source: OncoMed Pharmaceuticals.

Additional preclinical results demonstrated that vantictumab reduced the cancer stem cell (CSC) frequency by 3x in two tumor xenograft models (one pancreatic tumor and one breast tumor). Chemotherapy treatment alone resulted in an increase of CSC frequency in these models, whereas the combination of chemotherapy and vantictumab reduced CSC frequencies by 10x in both tumor models. Vantictumab also induced differentiation of CSCs to less tumorigenic cell types, which could be more susceptible to chemotherapy.

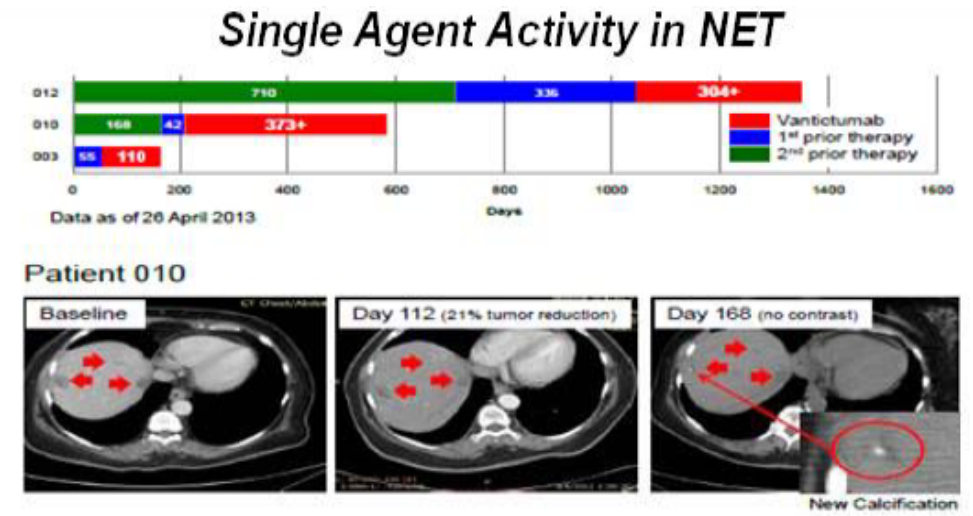
OncoMed is currently conducting a phase 1 single-agent dose escalation trial of vantictumab in patients with advanced solid tumors. This trial follows a 3+3 design. Patients receive intravenous (IV) infusions of vantictumab at assigned dosing schedules with dose levels of 0.5 mg/kg to 10 mg/kg for 56 days. After 56 days, if the tumor is smaller or if there is no evidence of disease progression, patients may continue to receive vantictumab every week until disease progression. Initiated in May 2011, this phase 1 trial plans to enroll 44 patients. Data of the

phase 1 trial from 18 patients were presented at the 2013 ASCO meeting. These data demonstrated that vantictumab had a half-life ranging from 1.5 days (0.5 mg/kg) to 3 days (2.5 mg/kg), and that its clearance was dose-dependent, consistent with target-mediated drug disposition. In cohorts with the highest dose exposures, efficacy correlated with preclinical tumor models. PD biomarker analysis demonstrated that the WNT pathway in tumors and surrogate tissues was modulated after the treatment. Three patients enrolled in the trial had neuroendocrine tumors (NETs) and all three patients achieved clinical benefit (Exhibit 25). Of the three patients, one patient had pancreatic neuroendocrine tumors and two patients had carcinoid tumors. These three patients had received study treatment for 110, 316+, and 384+ days based on a May 8, 2013 cut-off date for data analysis. The most common (>10%) treatment-related adverse events in a total of 23 patients across the dose levels tested included fatigue (30%), nausea (22%), vomiting (17%), increased alkaline phosphatase (13%), constipation (13%), decreased appetite (13%), and hypercalcemia (13%). Two treatment-related Grade 3 adverse events were observed, which included dose-limiting toxicities of Grade 3 diarrhea and vomiting in one patient. One patient at the lowest dose cohort (0.5 mg/kg q1w) had a bone fracture on day 110. This patient had a ~4x increase in a marker for bone degradation, β-C-terminal telopeptide (β-CTX), by day 28. Less frequent dosing and safety plan revision enabled further dose escalation. Two patients received zoledronic acid upon doubling of β-CTX, and their β-CTX returned to baseline. The primary completion date for the phase 1 trial is December 2013.

Exhibit 25: Single Agent Activity of Vantictumab in Neuroendocrine Tumors (NETs)



Source: OncoMed Pharmaceuticals.



Source: OncoMed Pharmaceuticals.

OncoMed plans to initiate three phase 1b clinical trials in 2H13 in three solid tumor indications in combination with standard-of-care therapies. Vantictumab is part of OncoMed's collaboration with Bayer, which has an option to license vantictumab at any point through completion of certain phase 1b trials.

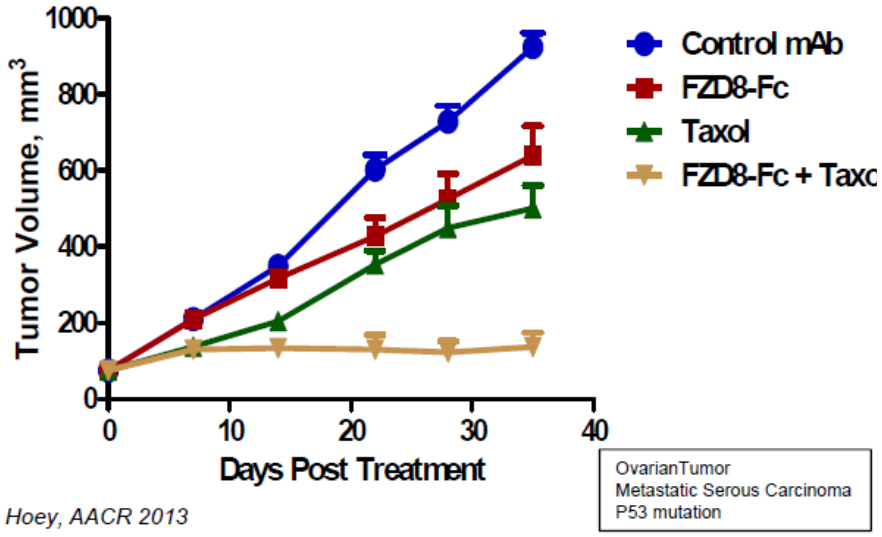
OncoMed has an issued US composition-of-matter patent for vantictumab that expires in 2029. OncoMed also has 4 pending US patent applications, 2 allowed foreign patent applications or foreign patents, and approximately 15 additional pending foreign applications, and to the extent that the patent applications in this family issue, they are expected to expire in 2029.

Fzd8-Fc (OMP-54F28)

OncoMed's second Wnt pathway modulator is Fzd8-Fc (OMP-54F28), which is a fusion protein containing part of the Fzd8 receptor fused to the Fc portion of a human immunoglobulin protein. Fzd8-Fc serves as a decoy receptor. Compared with vantictumab, which binds Frizzled receptors, OMP-54F28 has a distinct mechanism of action of binding Wnt ligands, thus serving as a decoy receptor.

In preclinical studies, OncoMed has established that OMP-54F28 has strong anti-tumor activity in solid tumors including pancreatic, breast, hepatocellular, ovarian, colorectal and other cancers, and that OMP-54F28 reduced CSC frequency in multiple preclinical models, either as a single agent or when combined with approved therapies.

Exhibit 26: Preclinical Data Demonstrating Synergy of OMP-54F28 With Taxol in a Patient-Derived Ovarian Tumor Model



Source: OncoMed Pharmaceuticals.

OncoMed initiated a dose escalation phase 1 study in July 2012 to evaluate OMP-54F28 in patients with advanced solid tumors. Patients are dosed at 0.5, 1, 2.5, 5 and 10 mg/kg OMP-54F28 administered IV once every 3 weeks, and patients are assessed for DLTs from days 0-28. All patients receive vitamin D3 QD and calcium carbonate BID from day 0 through 30 days after discontinuation of OMP-54F28. Patients with a >2x increase in fasting β-CTX or a >3% decrease in bone mineral density (BMD) from screening or a T-score decline to <-2.5 in the total femur or L1-L4 DEXA scan measurement will be started on zoledronic acid. Subjects with stable disease or a response at day 56 are allowed to continue to receive OMP-54F28 until disease progression. As of a data cut-off date of May 28, 2013, the most common (>10% of patients) treatment-related adverse events in 13 patients across the dose levels tested included decreased appetite (23%), fatigue (23%), hypocalcemia (23%), nausea (23%), altered taste (15%), increased blood pressure (15%), peripheral edema (15%), and vomiting (15%). The only treatment-related Grade 3 event was one Grade 3 anemia. Enrollment is still ongoing for the phase 1 study, which has an expected enrollment of 36 patients and a primary completion date of December 2013 according to clinicaltrials.gov. OncoMed expects to present data for this phase 1 study by YE13 or early 2014.

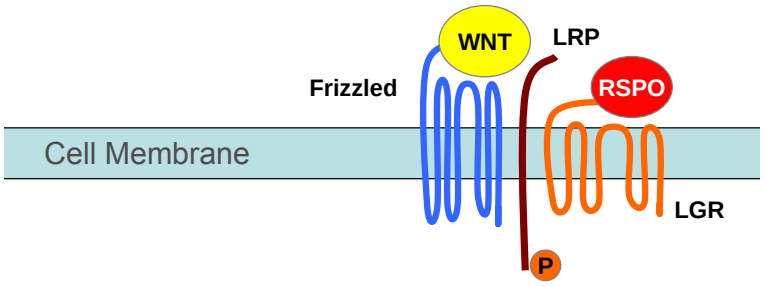
OncoMed plans to initiate three phase 1b clinical trials in late 2013 or early 2014 in distinct tumor types in combination with standard-of-care therapies. OMP-54F28 is part of OncoMed's collaboration with Bayer, which retains an option to license OMP-54F28 at any point through the completion of certain phase 1b clinical trials.

OncoMed has 1 pending US patent application and 17 foreign national patent applications for OMP-54F28, and patents that issue from these applications are expected to expire in 2031. OncoMed also owns a broad-issued US patent relating to certain Fzd-Fc biologics and uses of Fzd-Fc biologics in the treatment of cancer expiring in 2027.

Prospects for Anti-RSPO-LGR

The R-spondin-LGR signaling axis has recently been appreciated as an important pathway that potentiates Wnt/β-catenin signaling in normal stem cells. R-spondins (RSPOs) are secreted proteins function as Wnt agonists because they potently enhance β-catenin signaling but require the presence of Wnt for this effect. However, the exact mechanism of action of RSPOs and the identity of their receptors had remained elusive until recently. In 2011, several groups identified the leucine-rich repeat-containing G-protein coupled receptor 4/5/6 (LGR4/5/6), an adult stem cell marker, as receptors for RSPOs. The LGR proteins are seven-transmembrane proteins and they physically reside within the Fzd/LRP receptor complexes. (See Exhibit 27.)

Exhibit 27: Schematic of RSPO-LGR Complexed with Wnt-Fzd-LRP

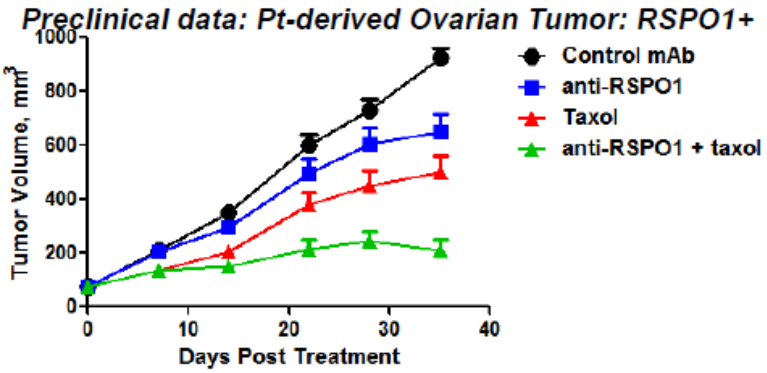


Source: BMO Capital Markets, adapted from Lau et al. *Genome Biol.* 2012; 13(3): 242

As early as in 2007, OncoMed identified that the RSPOs signal through the LGR receptor family and filed patents applications on therapeutic techniques based on this discovery. OncoMed has identified antibodies to proteins in the family that modulate RPO-LGR signaling, and has generated preclinical data. Using a reporter-based screen for secreted β-catenin signaling activities produced by a panel of human tumors, OncoMed discovered RSPO activity produced by numerous human tumors of multiple tumor types including ovarian, pancreatic, colon, breast, and non-small cell lung cancer. Using human tumor xenograft models, OncoMed demonstrated that anti-RSPO treatment markedly inhibited tumor growth in several tumor types, and that RPSO blockade promoted tumor differentiation and reduced the frequency of tumor initiating cells. Additional preclinical data demonstrated synergistic effects between anti-RSPO and chemotherapy in a ovarian tumor xenograft model. (See Exhibit 28.) OncoMed plans to file its first IND on an antibody targeting the RSPO-LGR pathway in 2014.

OncoMed has emphasized its significant intellectual property position on antibodies that disrupt RSPO-LGR pathway signaling, which includes two issued US patents broadly covering human or humanized monoclonal antibodies that disrupt binding of RSPO to LGR or disrupt RSPO activation of LGR. These patents expire in 2028.

Exhibit 28: Preclinical Data of Anti-RSPO in Human Tumor Xenograft Models



Source: OncoMed Pharmaceuticals.

Commercial Opportunity for Initial Target Markets

Non-Small Cell Lung Cancer for Demcizumab

National Cancer Institute (NCI) estimates that approximately 228,000 new cases of lung cancer will be diagnosed in the US in 2013, representing approximately 14% of all cancer diagnosis. Approximately two-thirds of people diagnosed with lung cancer in the US are 65 years or older and the average age at the time of diagnosis is about 70.

Lung cancer is classified as small cell lung cancer (SCLC), which accounts for 15% of lung cancer cases, and non-small cell lung cancer (NSCLC), which accounts for 84%. NSCLC is consisted of three major histological subtypes: squamous cell carcinoma, adenocarcinoma, and large cell carcinoma. The distinction between SCLC and NSCLC came into prominence in the 1970s and was based on differential clinical features and sensitivities to cytotoxic therapies. SCLC was characterized by widespread metastatic spread at diagnosis and demonstrated partial or complete response to conventional cytotoxic therapies, whereas NSCLC, including all major forms, was less likely to spread at diagnosis and typically failed to demonstrate objective responses to cytotoxic agents.

Squamous cell cancer accounts for approximately 30% of NSCLC. Squamous cell cancer often develops from epithelial cells near the center of the lung in one of the main airways (the left or right bronchus). Often a result of smoking, squamous cell cancer used to be the most common form of lung cancer, but its incidence has decreased dramatically in the past four decades. Adenocarcinoma is a cancer of epithelial cells that make fluids (mucus) to keep the lung moist and is often found in the peripheral compartments of the lung, near respiratory bronchioles and the alveoli. Adenocarcinoma is the most common form of lung cancer in North America. Large-cell lung carcinomas lack features to be classified as any other carcinoma, and this type of lung cancer tends to grow quickly.

Lung cancer is the leading cause of cancer death; NCI estimates that 160,000 lung cancer deaths will occur in 2013, more than colon, breast, and prostate cancer deaths combined and accounting for approximately 27% of all cancer deaths. The one-year relative survival for lung cancer is 44%, and the five-year survival for lung cancer is 16%. The five-year survival for small cell lung cancer (6%) is significantly lower than that for NSCLC (18%). Exhibit 29 provides detailed five-year survival rates for NSCLC by stage.

Exhibit 29: Five-Year Survival Rate of NSCLC by Stage

Stage	5-Year Survival Rate
IA	49%
IB	45%
IIA	30%
IIB	31%
IIIA	14%
IIIB	5%
IV	1%

Source: National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database.

A stage IA lung tumor is 3cm or smaller, and a stage IB tumor is 5cm or smaller or may have grown into the bronchus or visceral pleura. A stage IIA tumor is 3cm or smaller with cancer in the lung's lymph nodes, or between 5cm and 7cm with no cancer in lymph nodes, whereas a stage IIB tumor can be 7cm or smaller with cancer in the nodes, or those larger than 7cm without cancer in nodes, or those that have invaded chest wall or bronchus, or those with secondary tumors in the same lobe. Stage III tumor may be of any size; stage IIIA is often defined by cancer that has spread to mediastinal nodes, presence of secondary tumors, or tumor growth into the mediastinum, neck or spine; stage IIIB is often defined by cancer that has spread to lymph nodes in or near the other lung or above the collarbone. In Stage IV lung cancer, malignant tumors are found in both lungs, or the cancer has spread to other parts of the body, such as the brain, bones, liver, or in the fluid between the two layers of pleura.

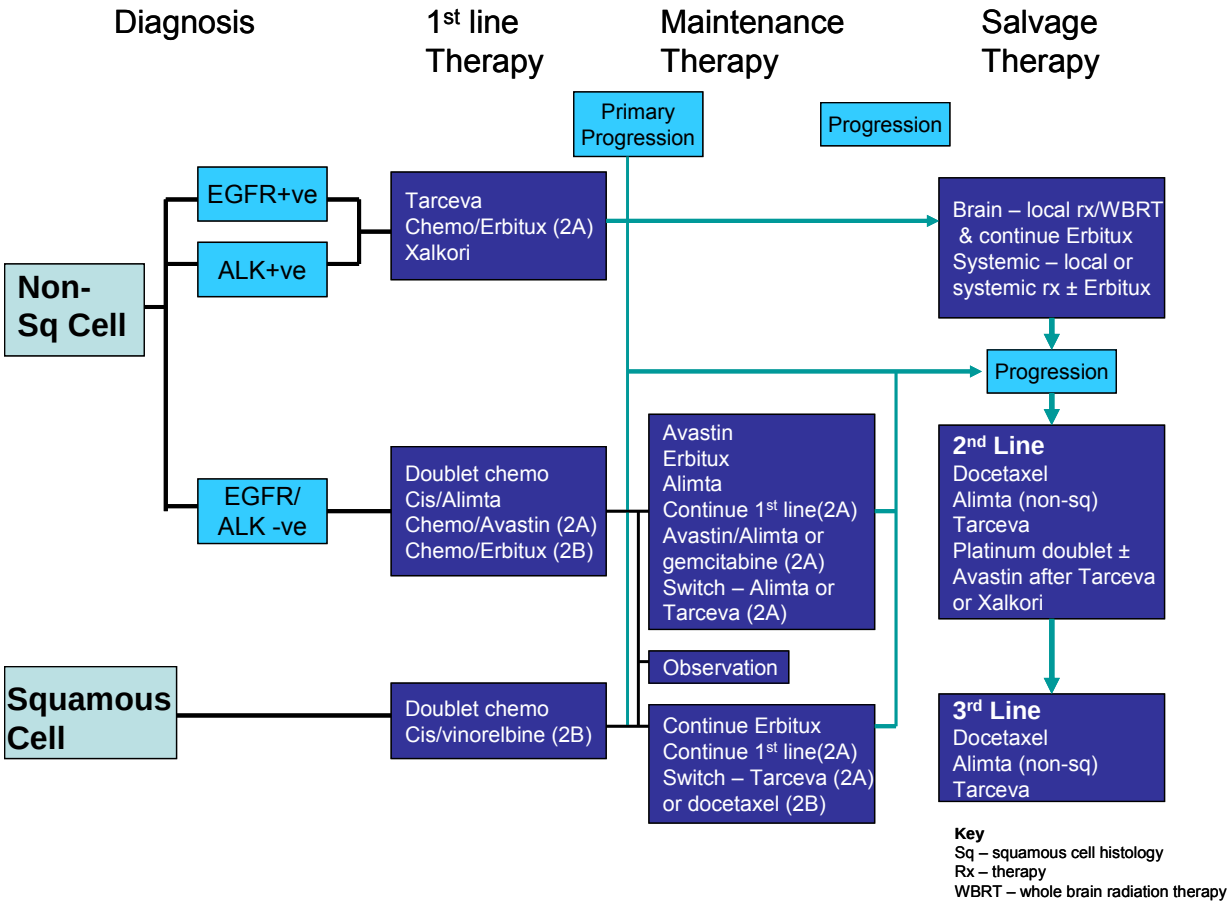
Current treatment recommendations for NSCLC have been defined in the National Comprehensive Cancer Network (NCCN) guidelines and categorized according to the stages of the disease. The treatment guidelines most relevant to OncoMed's development of demcizumab are those for locally advanced and metastatic NSCLC. (See Exhibit 30.)

Treatment of NSCLC is dependent on a number of criteria including histology, the presence of an oncogenic mutation, and patient performance status. Subjects who do not have an oncogenic driver mutation and have an ECOG performance status of 3 at baseline, or in whom PS decreases on treatment are not good candidates for the rigors of chemotherapy, are likely to be offered TARCEVA or best supportive care.

The diagram in Exhibit 30 summarizes the NCCN treatment guideline options for patients with an oncogenic driver or for those without an oncogenic driver and an ECOG PS of 0 or 1. At

diagnosis of metastatic disease, the first decision point for therapy is based on tumor histology, as large randomized clinical trials have shown that inclusion of both the chemotherapy agent ALIMTA and VEGF inhibitor AVASTIN led to inferior outcomes, leading to FDA contra indication of use in squamous histology. Since EGFR or ALK mutations are not observed in subjects with squamous cell histology, first line therapy is doublet chemotherapy (with or without ERBITUX), including a 2B recommendation for cisplatin/vinorelbine.

Exhibit 30: NCCN Treatment Overview for PS 0-1 Locally Advanced and Metastatic NSCLC



Source: NCCN and BMO Capital Markets.

For subjects with non-squamous histology, namely adenocarcinoma or less commonly large cell carcinoma, NCCN follows the American College of Pathologist recommendation for universal EGFR and ALK mutation testing. If a patient's status is known at diagnosis or before the need to start treatment, NCCN recommends single agent TARCEVA for EGFR+ve NSCLC and XALKORI for ALK+ve NSCLC. When the status of EGFR or ALK is not know at the time of treatment initiation, standard chemotherapy is started with an option at the time of mutation diagnosis to add TARCEVA or XALKORI, or switch to the targeted agent at the end of

induction. In subjects who have non-squamous disease and do not have an activating EGFR or ALK mutation, doublet chemotherapy is recommended. The addition of a biologic agent is considered an option by NCCN.

Traditionally, all subjects completing 4-6 cycles of chemotherapy were observed until the development of symptomatic disease, and while observation remains an option, maintenance therapy is becoming more common. Numerous trials have evaluated continuation of targeted or well-tolerated chemotherapeutic agents such as ALIMTA or switching to one of these therapies, as a maintenance strategy and for non-squamous cell disease. AVASTIN, ERBITUX or ALIMTA are considered category 1 options for maintenance while continuation of first-line therapy, AVASTIN combined with ALIMTA or gemcitabine or switching to ALIMTA or TARCEVA are considered as alternative options. Maintenance options for subjects with squamous disease are more limited and include continuation of ERBITUX if it was part of the front-line regimen, continuation of the front-line regimen, switch to TARCEVA, or early use of docetaxel, which is indicated for use in the second line.

Non-squamous ALK or EGFR mutation +ve subjects who start TARCEVA or XALKORI, continue therapy until progression and depending on the location and tenor of the metastases may continue TARCEVA in the context of progressive disease. In the case of a metastasis limited to the brain, local therapy for a solitary lesion, or whole brain radiation for numerous lesions are indicated with the continuation of TARCEVA, which is assumed to still be controlling the primary disease. Similarly for local or systemic progression outside of the brain, consideration of continuing TARCEVA on the background of additional therapy can be considered to prevent rebound of disease that is still being controlled by TARCEVA.

Not all patients respond to initial therapy, demonstrating primary resistance, and these patients along with those progressing after maintenance therapy or showing acquired resistance to a targeted therapy, may be eligible for salvage treatment. Agents approved for use in the second line setting include docetaxel, ALIMTA, and TARCEVA. Obviously, ALIMTA use is limited to the non-squamous cell setting and in patients who have not progressed on ALIMTA. TARCEVA's original approval was based on the BR21 trial of TARCEVA in the salvage setting where benefits were shown irrespective of EGFR mutation status. Given that TARCEVA has subsequently found a role in the front-line setting for EGFR mutation positive patients, the standard front-line chemotherapy regimens, with or without AVASTIN, are recommended for use after the development of acquired resistance to TARCEVA or in the case of ALK+ve disease, XALKORI. Following failure of second-line therapy, third-line options become a matter of using what has not been used before and given poor outcomes for all metastatic NSCLC, especially in the absence of an identified oncogene, clinical trials remain a valid option.

Pancreatic Cancer for Demcizumab

Pancreatic tumor could develop from exocrine or endocrine cells. About 95% of the pancreatic cancer cases are exocrine tumors.

National Cancer Institute (NCI) estimates that approximately 45,220 new cases of pancreatic cancer will be diagnosed in the US in 2013. Pancreatic cancer is the 10th most commonly diagnosed cancer in men and the 9th most commonly diagnosed cancer in women in the US. Pancreatic cancer ranks 4th as a cause of cancer death in both men and women. In 2013, approximately 38,460 people are expected to die from pancreatic cancer in the US. The mean age at the time of diagnosis of pancreatic cancer is 71 years.

The prognosis of pancreatic cancer is dependent on the stage of the disease at diagnosis. At Stage I, the pancreatic tumor is found only in the pancreas. In Stage II, the tumor has invaded nearby tissue but not nearby blood vessels, and the tumor may or may not have spread to the lymph nodes. In Stage III, the tumor has invaded nearby blood vessels. In Stage IV, the cancer has spread to distant organ, such as the liver or lungs. The median survival ranges from 24.1 months for Stage IA to just 4.5 months for the most advanced stage. The five-year survival rate for pancreatic cancer, including all stages, is 6%. Even for a Stage I or Stage II patient whose tumor has been surgically removed, the five-year survival rate is only about 20% to 25%.

Exhibit 31: Median Survival of Pancreatic Cancer

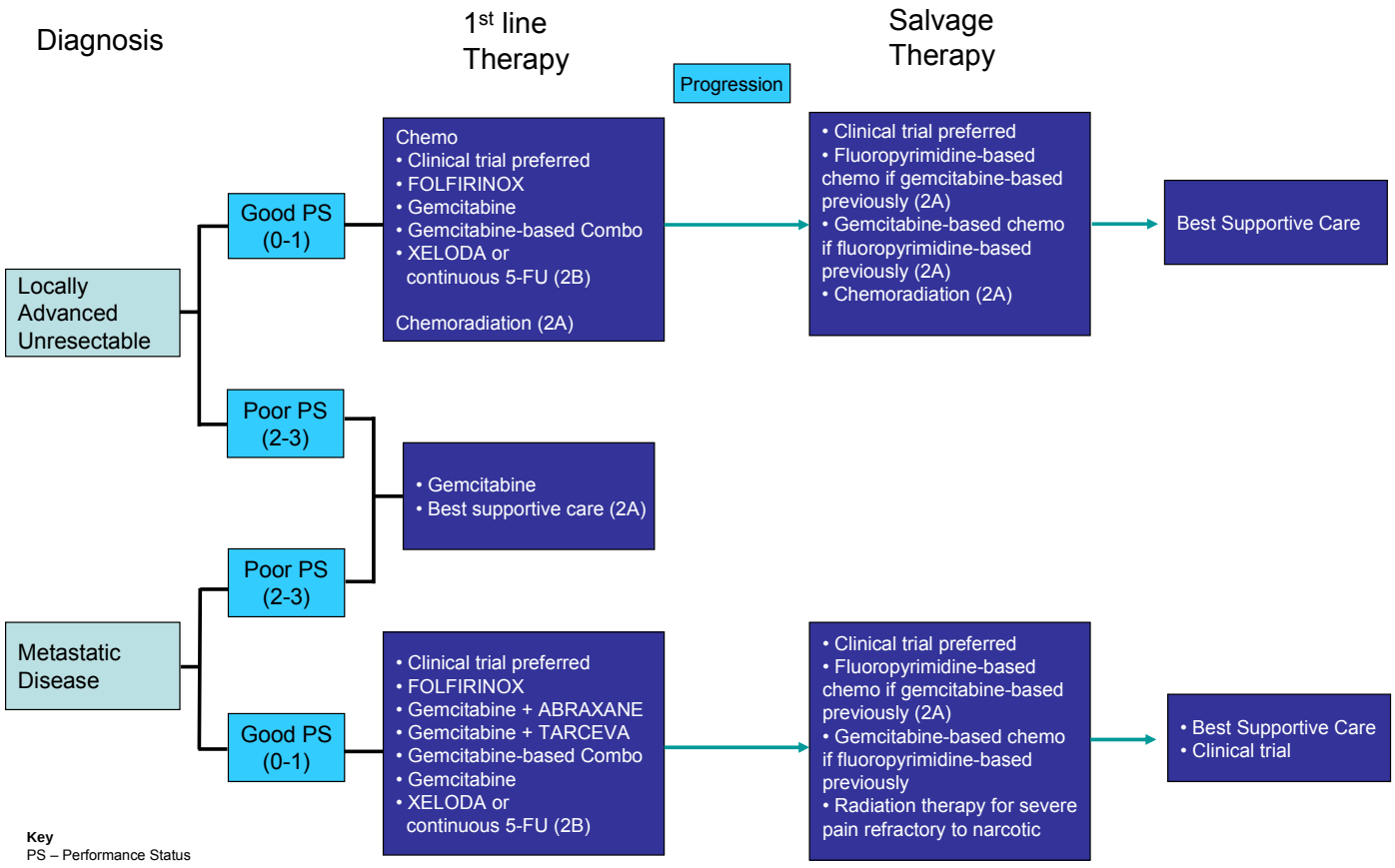
Stage	Median Survival (Months)
IA	24.1
IB	20.6
IIA	15.4
IIB	12.7
III	10.6
IV	4.5

Source: Bilimoria et al. *Cancer* 2007; 110:738.

Current treatment recommendations for pancreatic cancer have been defined in NCCN guidelines and categorized according to the stages of the disease. The treatment guidelines most relevant to OncoMed's development of demcizumab are those for locally advanced and metastatic pancreatic cancer. (See Exhibit 32.)

Treatment of locally advanced unresectable and metastatic pancreatic cancer is dependent on the patient performance status. Subjects who do not have an ECOG performance status of 0-1 at baseline are recommended to receive gemcitabine or best supportive care. For patients with metastatic pancreatic cancer and good performance status, acceptable chemotherapy combinations include FOLFIRINOX, gemcitabine + ABRAXANE, gemcitabine + TARCEVA, gemcitabine + XELODA, gemcitabine + cisplatin, or fixed-dose rate gemcitabine, docetaxel, XELODA (GTX regimen). For patients with locally advanced unresectable pancreatic cancer, mono- or combination systemic chemotherapy as noted for metastatic disease, may be considered as initial therapy prior to chemoradiation. Patients who progress with metastatic disease are not candidates for chemoradiation unless required for palliative purposes.

Exhibit 32: NCCN Treatment Overview for Locally Advanced Unresectable and Metastatic Pancreatic Cancer



Source: NCCN version 1.2013 and BMO Capital Markets.

Market Model

Our market model for OncoMed is composed of US and ex-US markets for its lead product, demcizumab. We model this product in two settings, non-small cell lung cancer (NSCLC) and pancreatic cancer.

In terms of the US market for NSCLC, we assume an annual incidence of 135,000 NSCLC patients, of whom the non-squamous portion is 65%, resulting in 87,750 new NSCLC patients annually. We believe that 80% of those patients, or 70,200, will receive platinum doublet chemotherapy. Half of those patients also receive Avastin and represent the VEGFR share, resulting in 35,100 patients who are eligible for demcizumab. We assume that Avastin will have 100% penetration into that market through 2017, at which time we expect that demcizumab will

enter the market. We expect that demcizumab market share will steadily increase until reaching its apex of 55% by the end of 2021. We forecast that market share will remain roughly at that level through 2025, the outer limit of our model. We expect that demcizumab will cost roughly $10,000 per month per patient with the average duration of treatment being six months. We expect that OncoMed will partner this product and receive roughly a 15% royalty on US sales.

In terms of the ex-US market for NSCLC, we expect the market size and dynamics to be similar to that of the US market. Additionally, we expect that OncoMed will partner demcizumab internationally and likely receive a 15% royalty on ex-US sales.

For the US pancreatic cancer market, we assume an incidence of 30,000 patients annually, of which 90% are eligible for demcizumab. In our estimation, demcizumab will enter this market in early 2018 and increase its market share rapidly reaching its peak at 85% in 2020 and plateauing through 2025, the extent of our model. We expect the pricing to be roughly $10,000 per month with an average duration of treatment of four months. We believe that OncoMed will partner demcizumab in pancreatic cancer and receive a royalty rate of 15% on US sales.

For the ex-US pancreatic cancer market, we believe the market size and dynamics will be similar to that in the US. We believe that OncoMed will also partner demcizumab international sales for pancreatic cancer and will receive roughly a 15% royalty on those sales.

Forecasts

We estimate 2013-2018 per share losses of $(0.77), $(1.43), $(2.52), $(2.70), $(3.01), and $(1.25), respectively. Our forecast calls for OMED to become profitable in 2019 with EPS of $3.71, increasing to $6.65 in 2020, our valuation year.

Exhibit 33: OMED Income Statement 2012A–2020E

INCOME STATEMENT ($M)	2007A	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
REVENUES										
Product Revenues	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 112.2	$ 364.2	$ 520.2
Collaborative Revenues/Milestones	-	24.7	32.9	47.0	48.0	50.0	30.0	-	-	-
TOTAL REVENUES	**$ -**	**$ 24.7**	**$ 32.9**	**$ 47.0**	**$ 48.0**	**$ 50.0**	**$ 30.0**	**$ 112.2**	**$ 364.2**	**$ 520.2**
EXPENSES (GAAP)										
Cost of Goods Sold (COGS)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ 0.2	$ 0.7	$ 1.0
R&D Expense	-	39.9	46.6	72.0	108.0	120.0	120.0	120.0	120.0	120.0
SG&A Expense	-	7.2	11.0	19.0	27.0	35.0	43.0	51.0	59.0	67.0
TOTAL EXPENSES	-	47.1	57.6	91.0	135.0	155.0	163.0	171.2	179.7	188.0
Operating Income	**-**	**(22.4)**	**(24.6)**	**(44.0)**	**(87.0)**	**(105.0)**	**(133.0)**	**(59.0)**	**184.5**	**332.2**
Depreciation and Amortization	-	-	-	-	-	-	-	-	-	-
EBIT	-	(22.4)	(24.6)	(44.0)	(87.0)	(105.0)	(133.0)	(59.0)	184.5	332.2
Interest Income	-	0.1	0.6	1.0	0.8	0.6	0.4	0.2	0.7	3.0
Interest Expense		(0.0)								
Other Expense	-	-	-	-	(0.1)	-	-	(0.1)	-	(0.3)
Interest and Other (net)	$ -	$ 0.1	$ 0.6	$ 1.0	$ 0.7	$ 0.6	$ 0.4	$ 0.1	$ 0.7	$ 2.7
Pre-Tax Income	-	(22.2)	(24.0)	(43.0)	(86.3)	(104.4)	(132.6)	(59.0)	185.3	334.9
Income Taxes	-	-	-	-	-	-	-	-	-	-
Net Income (GAAP)	**-**	**(22.2)**	**(24.0)**	**(43.0)**	**(86.3)**	**(104.4)**	**(132.6)**	**(59.0)**	**185.3**	**334.9**
EPS (GAAP) (basic)	#DIV/0!	$ (1.00)	$ (0.83)	$ (1.53)	$ (2.69)	$ (2.86)	$ (3.17)	$ (1.31)	$ 3.88	$ 6.95
EPS (GAAP) (diluted)	**#DIV/0!**	**$ (1.00)**	**$ (0.77)**	**$ (1.43)**	**$ (2.52)**	**$ (2.70)**	**$ (3.01)**	**$ (1.25)**	**$ 3.71**	**$ 6.65**
Total of Reconciliation Items	-	6.2	8.0	8.0	8.0	8.0	8.0	8.0	8.0	8.0
Net Income (Non-GAAP)	$ -	$ (40.4)	$ (16.0)	$ (35.0)	$ (78.3)	$ (96.4)	$ (124.6)	$ (51.0)	$ 193.3	$ 342.9
Impact of Adjustments to EPS	#DIV/0!	1.01	0.31	0.27	0.24	0.21	0.18	0.16	0.16	0.16
EPS (Non-GAAP) (basic)	#DIV/0!	$ (4.56)	$ (0.51)	$ (1.25)	$ (2.44)	$ (2.64)	$ (2.98)	$ (1.13)	$ 4.05	$ 7.12
EPS (Non-GAAP) (diluted)		$ (4.56)	$ (0.45)	$ (1.16)	$ (2.29)	$ (2.49)	$ (2.83)	$ (1.08)	$ 3.87	$ 6.81
Weighted average shares outstanding (basic)		22.2	25.0	28.0	32.1	36.7	42.4	46.5	47.7	48.2
Weighted average shares outstanding (diluted)		22.2	26.1	30.1	34.1	38.8	44.5	48.6	49.9	50.4

Source: Company reports and BMO Capital Markets.

Exhibit 34: OMED Balance Sheet 2012A–2020E

BALANCE SHEET ($M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
ASSETS									
Cash and cash equivalents	$ 16.3	$ 76.0	$ 32.4	$ 20.5	$ (4.5)	$ (17.7)	$ (17.3)	$ 167.4	$ 501.7
Marketable securities	50.0	50.3	50.3	50.3	50.3	50.3	50.3	50.3	50.3
Restricted cash	-	-	-	-	-	-	-	-	-
Total cash, cash equivalents, and short-term investments	$ 66.2	$ 126.3	$ 82.7	$ 70.8	$ 45.8	$ 32.6	$ 33.0	$ 217.7	$ 552.0
Accounts receivable	4.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Inventory	-	-	-	-	-	-	-	-	-
Prepaid expenses and other current assets	1.1	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2
Total Current Assets	$ 71.4	$ 127.5	$ 83.9	$ 72.0	$ 47.0	$ 33.8	$ 34.3	$ 218.9	$ 553.3
Plant, property, and equipment, net	5.5	5.6	6.2	6.8	7.4	8.0	8.6	9.2	9.8
Other assets	2.9	3.17	3.17	3.17	3.17	3.17	3.17	3.17	3.17
TOTAL ASSETS	**$ 79.8**	**$ 136.3**	**$ 93.3**	**$ 82.0**	**$ 57.6**	**$ 45.0**	**$ 46.1**	**$ 231.3**	**$ 566.3**
Current Liabilities									
Accounts payable	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8
Accrued expenses	3.8	5.3	5.3	5.3	5.3	5.3	5.3	5.3	5.3
Deferred revenue	14.7	14.7	14.7	14.7	14.7	14.7	14.7	14.7	14.7
Current portion of long-term debt	-	-	-	-	-	-	-	-	-
Collaboration payable	-	-	-	-	-	-	-	-	-
Other current liabilities	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8
Total Current Liabilities	$ 20.1	$ 21.6	$ 21.6	$ 21.6	$ 21.6	$ 21.6	$ 21.6	$ 21.6	$ 21.6
Notes payable	-	-	-	-	-	-	-	-	-
Convertible preferred stock warrant liability	-	-	-	-	-	-	-	-	-
Convertible preferred stock	182.8	182.8	182.8	182.8	182.8	182.8	182.8	182.8	182.8
Other liabilities	21.1	18.0	18.0	18.0	18.0	18.0	18.0	18.0	18.0
TOTAL LIABILITIES	**$ 224.0**	**$ 222.4**	**$ 222.4**	**$ 222.4**	**$ 222.4**	**$ 222.4**	**$ 222.4**	**$ 222.4**	**$ 222.4**
Shareholder's Equity									
Common stock	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Additional paid-in capital	4.1	86.2	86.2	161.2	241.2	361.2	421.2	421.2	421.2
Accumulated other comprehensive income	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Accumulated deficit	(148.4)	(172.3)	(215.3)	(301.7)	(406.0)	(538.6)	(597.6)	(412.3)	(77.4)
TOTAL SHAREHOLDER'S EQUITY (DEFICIT)	$ (144.2)	$ (86.1)	$ (129.1)	$ (140.4)	$ (164.8)	$ (177.4)	$ (176.3)	$ 8.9	$ 343.8
TOTAL LIABILITIES AND SHAREHOLDER'S EQUITY	**$ 79.8**	**$ 136.3**	**$ 93.3**	**$ 82.0**	**$ 57.6**	**$ 45.0**	**$ 46.1**	**$ 231.3**	**$ 566.3**

Source: Company reports and BMO Capital Markets.

Exhibit 35: OMED Cash Flow Statement 2012A–2020E

CASH FLOW STATEMENT ($M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
Cash Flow From Operating Activities									
Net Income	$ (22.2)	$ (17.2)	$ (15.3)	$ (13.4)	$ (13.4)	$ (11.4)	$ 8.4	$ 66.7	$ 89.1
Depreciation & Amortization	1.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Stock-based compensation	0.8	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Loss on disposal of equipment	-	-	-	-	-	-	-	-	-
Issuance of stock options for services	-	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)
Deferred Taxes and Other	(0.1)	-	-	-	-	-	-	-	-
Working Capital Adjustments									
Accounts receivable	(4.0)	-	-	-	-	-	-	-	-
Inventory	-	-	-	-	-	-	-	-	-
Prepaid expenses and other current assets	(0.5)	-	-	-	-	-	-	-	-
Accounts payable	(3.2)	-	-	-	-	-	-	-	-
Accrued expenses	-	-	-	-	-	-	-	-	-
Deferred rent	(0.4)	-	-	-	-	-	-	-	-
Deferred revenue	-	-	-	-	-	-	-	-	-
Collaboration payable	(2.2)	-	-	-	-	-	-	-	-
Other assets/liabilities	(2.9)	-	-	-	-	-	-	-	-
Total Working Capital (Decrease)	$ (13.23)	$ -	$ -	$ -	$ -	$ -	$ -	$ -	$ -
TOTAL CASH FROM OPERATIONS	**$ (33.3)**	**$ (16.8)**	**$ (14.9)**	**$ (13.1)**	**$ (13.0)**	**$ (11.1)**	**$ 8.7**	**$ 67.1**	**$ 89.4**
Cash From Investing Activities									
Purchases of marketable securities	(73.9)	-	-	-	-	-	-	-	-
Sales and maturities of marketable securities	112.7	-	-	-	-	-	-	-	-
Capital expenditures	(0.7)	(0.5)	(0.5)	(0.5)	(0.5)	(0.5)	(0.5)	(0.5)	(0.5)
Other	-	-	-	-	-	-	-	-	-
TOTAL CASH FROM INVESTING	38.0	(0.5)	(0.5)	(0.5)	(0.5)	(0.5)	(0.5)	(0.5)	(0.5)
Cash From Financing Activities									
Proceeds from issuance of common stock	0.2	-	-	-	-	-	-	-	-
Proceeds from exercise of stock options	-	-	-	-	-	-	-	-	-
Proceeds from exercise of warrants	-	-	-	-	-	-	-	-	-
Proceeds from sale - leaseback of property and equipment	-	-	-	-	-	-	-	-	-
Payment of capital lease obligations	-	-	-	-	-	-	-	-	-
Proceeds from (payments on) long-term debt	(0.3)	-	-	-	-	-	-	-	-
TOTAL CASH FROM FINANCING	(0.2)	-	-	-	-	-	-	-	-
Increase (decrease) in cash and cash equivalents	4.5	(17.3)	(15.4)	(13.6)	(13.5)	(11.6)	8.2	66.6	88.9
Cash and cash equivalents at beginning of quarter	11.8	93.3	47.8	34.0	9.0	(6.2)	(25.5)	100.8	412.8
Cash and cash equivalents at end of quarter	$ 16.3	$ 76.0	$ 32.4	$ 20.5	$ (4.5)	$ (17.7)	$ (17.3)	$ 167.4	$ 501.7

Source: Company reports and BMO Capital Markets.

Exhibit 36: OMED Demcizumab Market Model—NSCLC, US

DEMCIZUMAB (M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
U.S. MARKET									
Non-Small Cell Lung Cancer (NSCLC)									
Incidence	135,000	135,000	135,000	135,000	135,000	135,000	135,000	135,000	135,000
Non-Squamous Incidence	65%	65%	65%	65%	65%	65%	65%	65%	65%
Non-Squamous NSCLC Patients	87,750	87,750	87,750	87,750	87,750	87,750	87,750	87,750	87,750
Patients Receiving Platinats	80%	80%	80%	80%	80%	80%	80%	80%	80%
Eligible Front-Line Patients	70,200	70,200	70,200	70,200	70,200	70,200	70,200	70,200	70,200
VEGFR Share									
Eligible for Demcizumab									
Demcizumab Penetration	0%	0%	0%	0%	0%	0%	15%	33%	43%
Avastin Penetration									
Demcizumab Patients	-	-	-	-	-	-	2,633	9,433	13,601
Avastin Patients									
Price per Month									
Duration of Treatment (months)									
Cost per Patient	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000
Demcizumab NSCLC Sales (M)	$ -	$ -	$ -	$ -	$ -	$ -	$ 158.0	$ 566.0	$ 816.1
Royalty Rate	15%	15%	15%	15%	15%	15%	15%	15%	15%
DEMCIZUMAB NSCLC U.S. ROYALTIES (M)	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 23.7**	**$ 84.9**	**$ 122.4**

Source: Company reports and BMO Capital Markets.

Exhibit 37: OMED Demcizumab Market Model—NSCLC, Ex-US

DEMCIZUMAB (M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
EX-U.S. MARKET									
Non-Small Cell Lung Cancer (NSCLC)									
Incidence	135,000	135,000	135,000	135,000	135,000	135,000	135,000	135,000	135,000
Non-Squamous Incidence	65%	65%	65%	65%	65%	65%	65%	65%	65%
Non-Squamous NSCLC Patients	87,750	87,750	87,750	87,750	87,750	87,750	87,750	87,750	87,750
Patients Receiving Platinats	80%	80%	80%	80%	80%	80%	80%	80%	80%
Eligible Front-Line Patients	70,200	70,200	70,200	70,200	70,200	70,200	70,200	70,200	70,200
VEGFR Share									
Eligible for Demcizumab									
Demcizumab Penetration	0%	0%	0%	0%	0%	0%	15%	33%	43%
Avastin Penetration									
Demcizumab Patients	-	-	-	-	-	-	2,633	9,433	13,601
Avastin Patients									
Price per Month									
Duration of Treatment (months)									
Cost per Patient	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000	$ 60,000
Demcizumab NSCLC Sales (M)	$ -	$ -	$ -	$ -	$ -	$ -	$ 158.0	$ 566.0	$ 816.1
Royalty Rate	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%
DEMCIZUMAB NSCLC EX-U.S. ROYALTIES (M)	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 23.7**	**$ 84.9**	**$ 122.4**

Source: Company reports and BMO Capital Markets.

Exhibit 38: OMED Demcizumab Market Model—Pancreatic Cancer, US

DEMCIZUMAB (M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
U.S. MARKET									
Pancreatic Cancer									
Incidence	30,000	30,000	30,000	30,000	30,000	30,000	30,000	30,000	30,000
Eligible for Demcizumab	90%	90%	90%	90%	90%	90%	90%	90%	90%
Patients Eligible for Demcizumab	27,000	27,000	27,000	27,000	27,000	27,000	27,000	27,000	27,000
Demcizumab Penetration	0%	0%	0%	0%	0%	0%	35%	75%	85%
Demcizumab Patients	-	-	-	-	-	-	5,400	16,200	22,950
Price per Month									
Duration of Treatment (months)									
Cost per Patient	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000
Demcizumab Pancreatic Cancer Sales (M)	$ -	$ -	$ -	$ -	$ -	$ -	$ 216.0	$ 648.0	$ 918.0
Royalty Rate	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%
TOTAL DEMCIZUMAB PANCREATIC CANCER U.S. ROYALTIES (M)	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 32.4**	**$ 97.2**	**$ 137.7**

Source: Company reports and BMO Capital Markets.

Exhibit 39: OMED Demcizumab Market Model—Pancreatic Cancer, Ex-US

DEMCIZUMAB (M)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E
EX-U.S. MARKET									
Pancreatic Cancer									
Incidence	30,000	30,000	30,000	30,000	30,000	30,000	30,000	30,000	30,000
Eligible for Demcizumab	90%	90%	90%	90%	90%	90%	90%	90%	90%
Patients Eligible for Demcizumab	27,000	27,000	27,000	27,000	27,000	27,000	27,000	27,000	27,000
Demcizumab Penetration	0%	0%	0%	0%	0%	0%	35%	75%	85%
Demcizumab Patients	-	-	-	-	-	-	5,400	16,200	22,950
Price per Month									
Duration of Treatment (months)									
Cost per Patient	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000	$ 40,000
Demcizumab Pancreatic Cancer Sales (M)	$ -	$ -	$ -	$ -	$ -	$ -	$ 216.0	$ 648.0	$ 918.0
Royalty Rate	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%	15.0%
TOTAL DEMCIZUMAB PANCREATIC CANCER EX-U.S. ROYALTIES (M)	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ -**	**$ 32.4**	**$ 97.2**	**$ 137.7**

Source: Company reports and BMO Capital Markets

Management

Paul J. Hastings has served as president and chief executive officer and a member of the board of directors since 2006. Prior to joining OncoMed, Mr. Hastings was president and chief executive officer of QLT, a publicly traded biotech company, from 2002 to 2005. From 2001 to 2002, Mr. Hastings served as president and chief executive officer of Axys Pharmaceuticals, acquired by Celera in 2001. From 1999 to 2001, Mr. Hastings served as President of Chiron BioPharmaceuticals, a division of Chiron Corporation. From 1998 to 1999, Mr. Hastings was president and chief executive officer of LXR Biotechnology. From 1994 to 1998, Mr. Hastings held positions of increasing responsibility at Genzyme, including vice-president, global marketing, Genzyme Corporation; Vice-President, general manager of Genzyme Therapeutics Europe; president, Genzyme Therapeutics Europe; and president, Genzyme Therapeutics Worldwide. Since June 2011, Mr. Hastings has served on the board of directors of Pacira Pharmaceuticals, Inc., a publicly traded pharmaceutical company, and Relypsa, a privately held biotechnology company. From 2008 to 2009, Mr. Hastings served as chairman of the board of directors of Proteolix, Inc, acquired by Onyx Pharmaceuticals in 2010. From 2000 to 2007, Mr. Hastings served on the board of directors of ViaCell, Inc., a publicly traded biotechnology company that was sold to Perkin Elmer in 2007. Mr. Hastings currently serves as the chairman emeritus of BayBio, a non-profit trade association serving the life science industry in Northern California, and as chairman, emerging companies section of the Biotechnology Industry Organization. Mr. Hastings received a B.S. in Pharmacy from the University of Rhode Island.

John A. Lewicki, Ph.D. has served as executive vice president and chief scientific officer since December 2009, and was senior vice president, research and development from 2004 to 2009. From 1983 to 2000, Dr. Lewicki served in various capacities at Scios, a public biopharmaceutical company, including 12 years as vice president of research, managing the company's diverse research areas. Dr. Lewicki has authored or coauthored more than 70 publications and book chapters and is listed as an inventor on over 30 issued US patents. Dr.

Lewicki holds a Ph.D. in Physiology/Pharmacology from the University of California, San Diego.

Jakob Dupont, M.D. has served as senior vice president and chief medical officer since January 2012 and served as vice president, clinical research since October 2011. From 2006 to 2011, Dr. Dupont served in various capacities at Genentech, including global medical director, AVASTIN from January 2011, in which capacity he oversaw the global medical strategy and late-stage medical program for AVASTIN; group and associate group director and global clinical leader for AVASTIN Breast and GYN Cancers from September 2009 to January 2011; associate group director and medical director in charge of clinical development for the angiogenesis pipeline at Genentech from June 2008 to October 2009; medical director for AVASTIN breast cancer, GYN cancer and melanoma development from March 2008 to June 2008; and Associate Medical Director for AVASTIN breast cancer, GYN cancers and melanoma development from September 2006 to March 2008. Since February 2009, Dr. Dupont has also served as an adjunct clinical assistant professor at the Stanford University School of Medicine. From January 2002 to September 2006, Dr. Dupont was a faculty member and laboratory researcher at Memorial Sloan-Kettering Cancer Center. Dr. Dupont received an A.B. in Philosophy from Vassar College, an M.A. in Philosophy from New York University, and an M.D. from the Joan & Sanford I. Weill Medical College of Cornell University. Dr. Dupont completed his medical oncology fellowship at Memorial Sloan-Kettering Cancer Center, his internal medicine residency at the New York Presbyterian Hospital–Cornell Campus, and his internal medicine internship at The University of Michigan Medical Center in Ann Arbor, Michigan.

Sunil Patel has served as senior vice president, chief business officer since December 2012 and previously served as senior vice president, corporate development since July 2009. From September 2008 to June 2009, Mr. Patel served as the vice president of corporate development & marketing at BiPar Sciences, a privately held biotechnology company focused on the development of cancer therapies and was acquired by Sanofi-Aventis S.A. in 2009. From May 2007 to August 2008, Mr. Patel served as the vice president of corporate development at Allos Therapeutics, a publicly traded biopharmaceutical company focused on the development and commercialization of cancer therapeutics. Previously, Mr. Patel held corporate development, marketing, and strategy positions with Connetics Corporation, Abgenix, and Gilead Sciences. From 1998 to 2003, Mr. Patel worked as a consultant with McKinsey & Company. Since October 2010, Mr. Patel has served on the board of directors of Ligand Pharmaceuticals, a publicly traded biotechnology company. Mr. Patel received a B.S. in Chemistry from the University of California Berkeley and an M.S. in Molecular Bioengineering/Biotechnology from the University of Washington.

William D. Waddill has served as senior vice president, chief financial officer, treasurer and assistant secretary since October 2007. From October 2006 to September 2007, Mr. Waddill served as the senior vice president, chief financial officer of Ilypsa, a privately held biotechnology company that developed drugs for the treatment of renal disease and was acquired by Amgen in 2007. From February 2000 to September 2006, Mr. Waddill served as a principal at Square One Finance, where he provided financial consulting and outside chief financial officer services to venture-backed companies. From December 1996 to February 2000, Mr. Waddill served as senior director of finance and administration at Exelixis, a biotechnology

company focused on development of drug therapies for cancer and other proliferative diseases. He received a B.S. in Accounting from the University of Illinois, Chicago and certification as a public accountant (inactive) after working at PriceWaterhouseCoopers and Deloitte.

Austin Gurney, Ph.D. has served as senior vice president, molecular and cellular biology since December 2009, and previously served as vice president of molecular and cellular biology from 2004. Prior to joining OncoMed, Dr. Gurney worked at Genentech, where his research contributed to the discovery of numerous growth factors and cytokines. Dr. Gurney has authored or co-authored more than 60 published scientific papers and is listed as an inventor on over 600 patents related to therapeutic applications in immunology and cancer. Dr. Gurney received a B.S. in Biology from Rensselaer Polytechnic Institute and a Ph.D. in Molecular and Cellular Biology from the Case Western Reserve University School of Medicine.

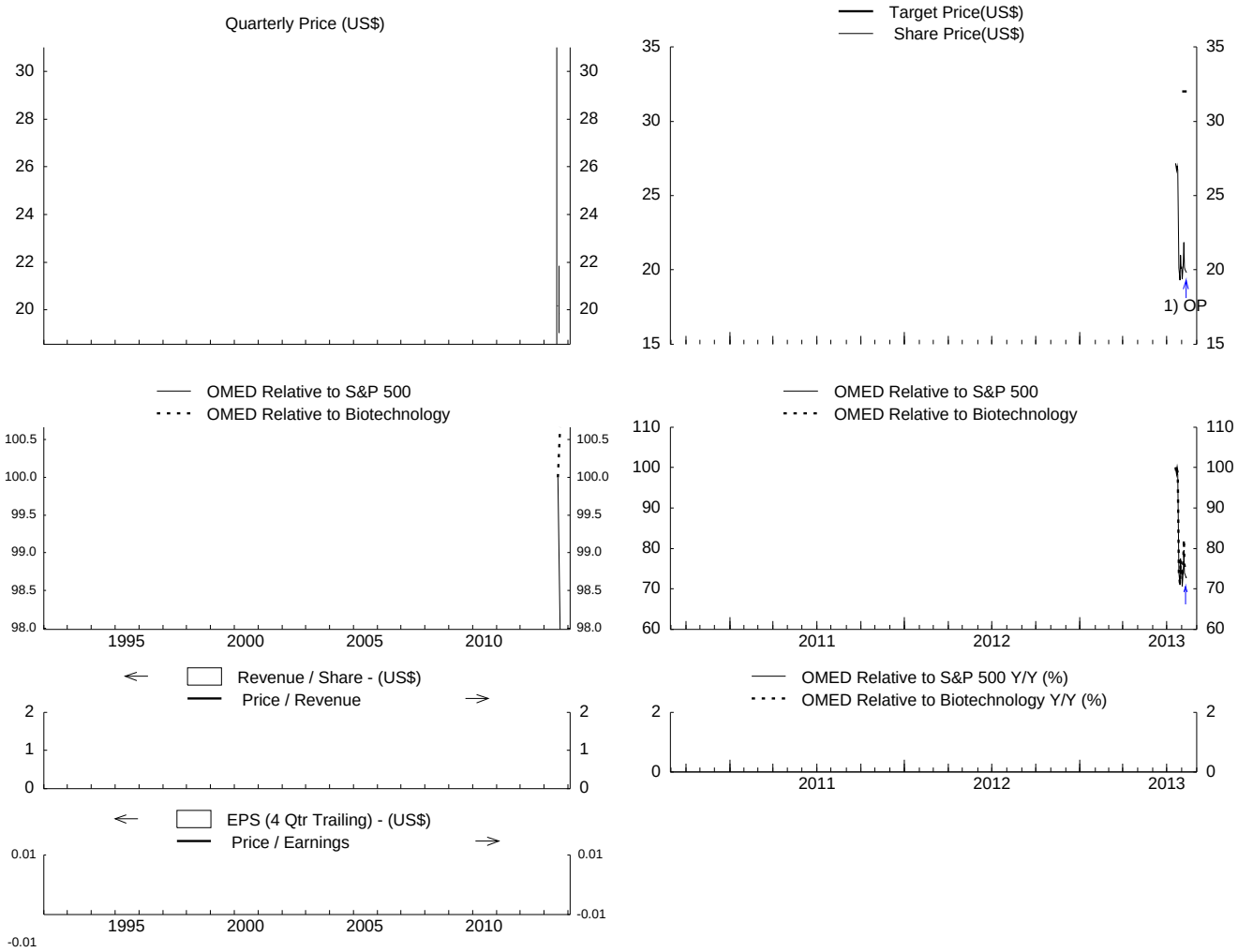
Timothy Hoey, Ph.D. has served as senior vice president, cancer biology since January 2009, and previously served as vice president, cancer biology from 2005 to January 2010. Prior to joining OncoMed, Dr. Hoey served as director, biology department at Amgen, where he was responsible for characterization of oncogenes and development of drugs to target oncogene products. Dr. Hoey previously served as director, biology department at Tularik, a public biopharmaceutical company acquired by Amgen in 2004. Dr. Hoey has authored or co-authored more than 50 published scientific papers and is listed as an inventor on several patents. Dr. Hoey received a B.S. in Biology from the University of Michigan and a Ph.D. in Biological Sciences from Columbia University.

Alicia J. Hager, J.D., Ph.D. has served as vice president and general counsel since December 2012 and previously served as vice president, legal affairs since July 2010 and chief patent counsel since November 2008. From June 2008 to October 2008, Dr. Hager served as senior patent counsel. From October 2002 to May 2008, Dr. Hager was an associate at the law firm of Morrison & Foerster LLP, where she served as intellectual property counsel for biotech and pharmaceutical clients. Prior to Morrison & Foerster, Dr. Hager was a patent agent at the law firm of Heller Ehrman White & McAuliffe LLP. Dr. Hager received an A.B. in Chemistry from Occidental College, an A.M. and Ph.D. in Chemistry from Harvard University, and a J.D. from Stanford Law School.

Other companies mentioned (priced as of the close on August 9, 2013):

Roche (RHHBY, $64.20, Not Rated)
Stemline Therapeutics (STML, $33.20, Not Rated)
Verastem (VSTM, $14.72, Not Rated)

Oncomed Pharmaceuticals (OMED)



FYE (Dec.)	EPS US$	P/E Hi - Lo	DPS US$	Yield% Hi - Lo	Payout %	BV US$	P/B Hi - Lo	ROE %
Range*:		na na		NC			>15 >15	
Current*	0.00	na	0.00	0.0	na	3.8	5.3	0

OMED - Rating as of 17-Jul-13 = NR

	Date	Rating Change	Share Price
1	8-Aug-13	NR to OP	$19.88

* Current EPS is the 4 Quarter Trailing to Q1/2013.
* Valuation metrics are based on high and low for the fiscal year.
* Range indicates the valuation range for the period presented above.

Last Price (August 9, 2013): $19.84
Sources: IHS Global Insight, Thomson Reuters, BMO Capital Markets.

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Sell	Underperform	5.3%	3.2%	1.4%	6.5%	3.5%	5.6%

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