COMPANY NOTE

Initiating Coverage

USA | Healthcare | Biotechnology

August 12, 2013

Jefferies

OncoMed (OMED)

Initiating With A Buy Rating And A $27 PT: Buy The Leader In Cancer Stem Cells

BUY
Price target $27.00
Price $19.84

EQUITY RESEARCH AMERICAS

Key Takeaway

We are initiating coverage on OMED with a Buy rating and a $27 price target. OMED is a leader in the science behind cancer stem cells, which are thought to drive progression and metastases, and has generated five clinical stage candidates in Phase 1 trials using its platform, creating multiple opportunities for success in major solid tumors and hematologic malignancies.

In July, Jefferies acted as lead bookrunner in the initial public offering of OMED common shares.

OMED Platform Has Been A Productive Source Of Candidates And Strong Partnerships. OncoMed has developed a strong platform of science around the discovery of cancer stem cell (CSC) targets. Cancer stem cells are thought to be the seeds of tumors, driving growth and metastases and resistant to conventional chemotherapy. We are encouraged by OMED's progress in developing a broad pipeline of clinical stage candidates based on CSC targets, with five biologics in Phase 1 trials. OMED's science has also been validated by two pharma partnerships with GSK (GSK LN, £xx, Hold) and Bayer (BAYN GR, €xx, Buy), under which OMED receives lucrative milestones and royalties given the preclinical stage of the candidates at the time each deal was signed.

Pipeline Could Yield Numerous Phase 2 Readouts By 2016. While early, we are encouraged by the efficacy signals generated to date. Single agent tumor shrinkage has been observed with demcizumab in pancreatic and lung cancer, prolonged stable disease in neuroendocrine tumors has been observed with single agent vantictumab , and there has been an encouraging preliminary response rate with OMED's anti-Notch2/3 in combination with chemotherapy in pancreatic cancer patients. Cardiovascular safety issues with demcizumab increase the risk of this program, but we are encouraged that the other candidates have not encountered major safety concerns to date. The applicability of OMED's CSC targets across a wide range of major solid and hematologic malignancies is a positive feature of the pipeline.

Valuation/Risks

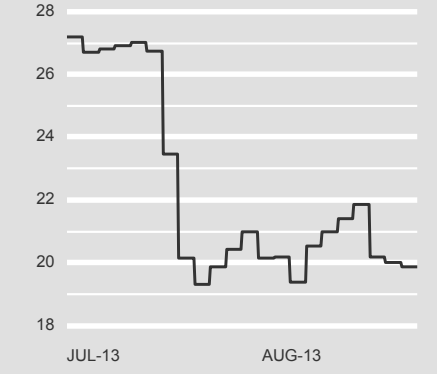
Our $27 PT = $14 demcizumab + $4 GSK + $5 Bayer collaboration + $4 cash. Risks: clinical, regulatory, commercial.

Financial Summary	
Net Debt (MM):	**($60.2)**
Cash/Share:	**$60.20**
Market Data	
52 Week Range:	**$31.00 - $17.00**
Total Entprs. Value (MM):	**$491.4**
Market Cap. (MM):	**$551.6**
Shares Out. (MM):	**27.8**
Float (MM):	**5.4**
Avg. Daily Vol.:	**NA**

Thomas Wei *
Equity Analyst
(212) 284-2326 twei@jefferies.com
Shaunak Deepak *
Equity Associate
(212) 284-2020 sdeepak@jefferies.com
* Jefferies LLC

USD	*Prev.*	**2012A**	*Prev.*	**2013E**	*Prev.*	**2014E**	*Prev.*	**2015E**
EBITDA (MM)	--	46.2	--	62.0	--	88.0	--	49.0
EV/EBITDA		10.6x		7.9x		5.6x		10.0x
EPS								
Mar	--	--	--	(0.39)A	--	--	--	--
Jun	--	--	--	(0.15)	--	--	--	--
Sep	--	--	--	(0.25)	--	--	--	--
Dec	--	--	--	(0.17)	--	--	--	--
FY Dec	--	(21.58)	--	(0.95)	--	(1.40)	--	0.49
FY P/E		NM		NM		NM		40.5x

Price Performance



OncoMed Pharmaceuticals, Inc.

BUY: $27 Price Target

THE LONG VIEW

Scenarios

Target Investment Thesis

- We believe OMED is a leader in cancer stem cell (CSC) targeting drugs
- We are encouraged by single agent activity with demcizumab in pancreatic and lung
- We see the GSK and Bayer collaborations as lucrative and validating the technology
- Target Price: $27 = $14 demcizumab + $4 GSK + $5 Bayer + $4 cash

Upside Scenario

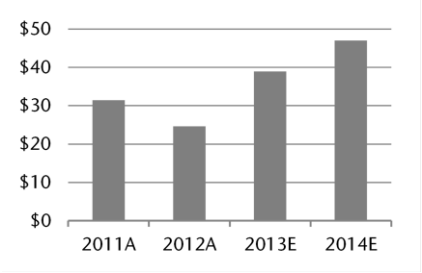
- We believe demcizumab could be a leading add-on to conventional chemotherapy
- Positive Phase 2 data and pivotal trials could increase the probability of success across programs
- No cost upside with GSK and Bayer covering costs of late stage development and commercialization
- Target Price: $49 = $27 demcizumab + $9 GSK + $9 Bayer + $4 cash

Downside Scenarios

- All programs are early stage
- Historical CV issues with demcizumab may persist or give FDA pause
- Collaborators may not opt-in to development programs
- Target Price: $11 = $7 demcizumab + $0 GSK + $0 Bayer + $4 cash

Long Term Analysis

Revenue (millions)



Source: Company data, Jefferies

Long Term Financial Model Drivers

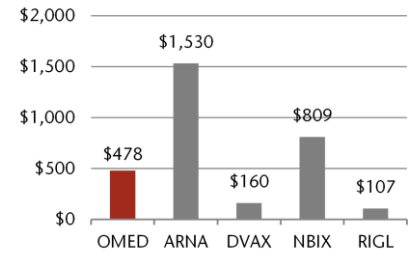
5-Year Revenue CAGR	**52%**

Other Considerations

With several eagerly anticipated product launches, anemic pipelines at large cap biotech and pharma, and an increasingly conservative FDA stance, we believe mid-cap biotech could lead sector performance in 2013. We see a premium placed on late-stage and marketed products. M&A interest could also factor into the performance of the sector, particularly among small-cap and mid-cap companies with later stage programs.

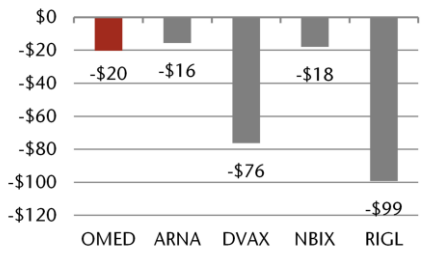
Peer Group

Group EVs



Source: FactSet

Net Income



Source: Company data

Recommendation / Price Target

Ticker	Recommendation	PT
OMED	**BUY**	**$27**
ARNA	BUY	$12
DVAX	BUY	$3
NBIX	BUY	$16
RIGL	BUY	$6

Catalysts

- 4Q13- Data from OMP-52M51 at AACR-NCI-EORTC
- 2H13/1H14 – Bayer initiates vantictumab Ph1b trials
- 2H13/1H14 – Bayer initiates OMP-54F28 Ph1b trials
- 2Q14 - Vantictumab Ph1a data at ASCO
- 2H14 – Final Ph2 data from ALPINE trial of OMP-59R5 in pancreatic

Company Description

OncoMed Pharmaceuticals (OMED) is a Redwood City, CA-based biopharmaceutical company that is a leader in the science behind cancer stem cells (CSCs), which are thought to drive cancer progression, metastasis, and chemotherapy resistance. Using proprietary technology, OMED has generated five clinical stage candidates targeting CSC pathways. Four of these compounds are being developed under two pharmaceutical partnerships with GSK and Bayer. The lead wholly owned drug is demcizumab, an antiDLL4 antibody currently in Phase 1b trials in pancreatic, lung and ovarian cancer. Close behind in development are OMP-59R5, an anti-Notch2/3 antibody, OMP-52M51, an antiNotch1 antibody, vantictumab, an anti-Fzd7 antibody, and OMP-54F28, a Fzd8-Fc fusion protein.

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

Executive Summary

OncoMed Pharmaceuticals (OMED) is a Redwood City, CA-based biopharmaceutical company that is a leader in the science behind cancer stem cells (CSCs), which are thought to drive cancer progression, metastasis, and chemotherapy resistance. Using proprietary technology, OMED has generated five clinical stage candidates targeting CSC pathways. Despite early cardiovascular safety issues with OMED's lead product candidate demcizumab, we are encouraged by single agent tumor shrinkage that has been observed with demcizumab in pancreatic and lung cancer. OMED's science has also been validated by two pharma partnerships with GSK (GSK LN, Hold) and Bayer (BAYN GR, Buy), under which OMED receives impressive milestones and royalties given the preclinical stage of the candidates at the time each deal was signed. Although these programs are still in Phase 1 trials, we note prolonged stable disease in neuroendocrine tumors has been observed with single agent vantictumab, an anti-Fzd7 being developed under the Bayer collaboration, and two pancreatic cancer partial responses observed with a combination of gemcitabine, Abraxane, and OMP-59R5, a dual Notch-2/3 inhibitor under the GSK collaboration. The applicability of OMED's CSC targets across a wide range of major solid and hematologic malignancies is a positive feature of the pipeline.

Valuation

We Derive A $27 Price Target Using A Sum-Of-The-Parts Methodology. We have constructed a discounted cash flow model for each of OncoMed's programs and partnerships and used a sum-of-the-parts valuation methodology assigning $14/share for demcizumab, $4/share for the GSK collaboration, $5/share for the Bayer collaboration, and $4/share in cash. For demcizumab, the most advanced compound in the clinic, we have modeled sales around the three lead indications: pancreatic cancer ($1.2b in 2025), lung cancer ($1.1b in 2025) and ovarian cancer ($500m in 2025). We assume $50,000 per course of therapy and peak penetration of 40% in each indication, which could prove conservative if robust overall survival benefits are demonstrated. We assume that demcizumab is re-partnered in 2015 following Phase 2 data under terms where OMED receives US royalties of 16-24% and ex-U.S. royalties of 12-20% on sales, with regulatory and sales milestones totaling $700m. We have applied a probability of success of 20% in pancreatic cancer, 20% in lung cancer, and 10% in ovarian cancer. This reflects the balance of early efficacy proof-of-concept signals with potential cardiovascular toxicity. Based on these parameters, we derive a $14/share value for demcizumab. For the GSK and Bayer collaborations, the earlier stage of development and the lack of defined indications presents a modeling challenge. We derived value for these collaborations by conducting an analysis of 28 cancer drug launches and choosing the median sales trajectory to represent a prototypical cancer drug. Based on our analysis, peak sales for the "average" cancer drug reach $700m by year 10. We applied the company's guidance on royalties for each program to this average sales trajectory and used probability of success adjustments of 20% for anti-Notch2/3, 10% for anti-Notch1, 20% for vantictumab, 10% for Fzd8-Fc, and 5% for the preclinical small molecule wnt program. We derive a $4/share value for the GSK collaboration and a $5/share value for the Bayer collaboration. We have not derived a specific value for the CSC discovery platform in our OMED valuation, although we believe investors currently place substantial value on the company's underlying technology itself.

Risks

The primary risk with the OMED story is clinical risk. While we are encouraged that OMED's platform has been a productive source of clinical stage candidates, all five programs are still in Phase 1 trials with limited patient exposure to date.

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

Efficacy Remains Uncertain. On efficacy, the only drug to have yielded a single-agent objective response is demcizumab with a single partial response in pancreatic cancer. While we are encouraged that three neuroendocrine tumor patients on single-agent vantictumab have had prolonged progression-free intervals, none of these patients qualified as an objective responder and relatively rapid progression was observed in all other tumor types tested. Even in the case of demcizumab, single agent activity as measured by response rates is encouraging but demonstrating objective responses as single agent is typically not accepted as a regulatory endpoint for approval in solid tumors and does not always translate into an overall survival benefit. The majority of OMED's supporting efficacy data comes from combination studies with chemotherapy, but these small open-label studies are difficult to interpret in the absence of a control arm. While OMED's response rates and progression-free survival (PFS) intervals in combination with chemotherapy compare favorably to chemotherapy alone, cross-trial comparisons between small Phase 1 cohorts and historical chemotherapy data is not scientifically rigorous and true claims of efficacy require a randomized Phase 2 or Phase 3 trial, the outcome of which would not be available until 2015-2016.

Safety Remains Uncertain, Particularly For Demcizumab. There has been limited safety data to date for OMED's pipeline given the early stage of development. For demcizumab, the company's lead wholly owned asset, there have been significant cardiovascular adverse events observed in Phase 1 trials to date, including hypertension, heart failure and pulmonary hypertension. These have necessitated multiple protocol interventions including close cardiac monitoring, the initiation of ACE inhibitors on first signs of BNP elevation, and multiple dosing changes from continuous to intermittent to now truncated dosing of demcizumab. Specifically, the newest dosing paradigm limits exposure to demcizumab to 70 days or less in an attempt to prevent clinical cardiovascular events. For the wnt programs, vantictumab and Fzd8 Fc, there is a theoretical risk of bone toxicity with this mechanism that will need to be monitored closely. One patient in the vantictumab Phase 1 trial suffered a Grade 2 compression fracture after a minor fall and four patients have required zoledronic acid intervention due to elevations in bone turnover as measured by beta-CTX. Other clinical stage candidates targeting the Notch pathway look cleaner, with diarrhea being the main toxicity observed to date.

New Truncated Dosing Strategy With Demcizumab Has Not Been Proven. The truncated dosing strategy with demcizumab limiting exposure to less than 70 days is novel. We are uncertain if a limited course of therapy will lead to additive efficacy on top of chemotherapy alone. The company has shared preclinical data demonstrating that truncated dosing of demcizumab with gemcitabine-Abraxane chemotherapy in a xenograft model of pancreatic cancer can lead to complete and sustained tumor eradication whereas neither demcizumab alone nor chemotherapy alone were able to achieve a sustained response. In addition, we note that partial responses and prolonged stable disease have been observed in a limited number of patients treated with truncated demcizumab dosing in the lung cancer setting. On the safety side, the cardiac safety of truncated dosing also still needs to be validated. From the safety data to date, it appears that the development of heart failure and pulmonary hypertension occurs after 120 days or more of cumulative demcizumab exposure, but we note that some patients experience modest BNP elevations even within the first 70 days, although these appear to be reversible and manageable with ACE inhibitors.

There Is Competition In The DLL4 Space. We are aware of at least two other anti-DLL4 clinical stage programs competing against demcizumab: Regeneron (REGN, $250.01, Hold) and Sanofi's (SAN FP, €78.85, Buy) enoticumab and AstraZeneca's (AZN LN, £3,235, Hold) MEDI0639. Both drugs are in Phase 1a single agent trials behind OMED, which has already advanced to Phase 1b combination studies with chemotherapy. Data from the single agent Phase 1 study of enoticumab were presented at ASCO 2013

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

from the first 53 patients. From a positive perspective, these data provide validation of OMED's efficacy signals, with two objective partial responses in non-small cell lung cancer and ovarian cancer at doses of 3mg/kg every 2 weeks. However, similar cardiovascular safety issues were observed with enoticumab including a case of Grade 3 right ventricular dysfunction, a case of Grade 3 left ventricular dysfunction and two cases of Grade 3 pulmonary hypertension. These occurred at doses of 1.5-3 mg/kg every 2 weeks. Early data are difficult to interpret and it is unclear if enoticumab will have an improved therapeutic window relative to demcizumab. REGN and SAN are currently expanding enrollment in the trial at target doses. We are not aware of any clinical data presented to date for MEDI0639.

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

Key Highlights

OncoMed Is A Leader In Targeting Cancer Stem Cells. OncoMed's clinical programs were discovered internally using proprietary technologies that enable the company to develop monoclonal antibody therapeutics targeting cancer stem cell (CSC) proteins. CSCs, known as tumor-initiating cells, are thought to be responsible for driving growth and metastasis of tumors primarily through self-renewal, a process by which CSCs divide, generating new CSCs that can subsequently differentiate and transform into bulk tumor cells. CSCs are also thought to be more resistant to standard chemotherapy. Unlike conventional oncology drugs that target bulk tumor cells with limited impact on CSCs, OncoMed's product candidates specifically target CSC pathways, blocking the process of self-renewal and driving CSC differentiation away from a bulk tumor state. OncoMed's drug candidates' targets include elements of the Notch, wnt, and other CSC pathways.

Demcizumab (Anti-DLL4)

Demcizumab Background: Mechanism of Action. Demcizumab is a humanized antibody against the Delta Like Ligand 4 (DLL4) target, which is the ligand for the members of the Notch signaling pathway. Notch is known to be involved in multiple anti-tumor pathways, with two potential mechanisms: an anti-angiogenesis mechanism (blocking the growth of new blood vessels that tumors create to feed growth) and an anti-cancer stem cell mechanism. In terms of anti-angiogenesis effects, demcizumab appears to have a differentiated mechanism of action relative to approved anti-VEGF approaches like Avastin. Specifically, demcizumab appears to cause an upregulation of VEGF expression and actually causes hypersprouting of new blood vessels, but these blood vessels do not fully mature into functional vessels with a functional lumen. This could create applications across a variety of tumors dependent on angiogenesis for survival: Avastin has demonstrated benefits of anti-angiogenesis in colorectal cancer, lung cancer, breast cancer, kidney cancer and glioblastoma. These happen to largely be the same tumor types where DLL4 expression is high. That said, the anti-cancer stem cell mechanism is perhaps what makes demcizumab unique. While there is no clinical evidence that demcizumab acts through an anti-cancer stem cell approach, the preclinical data are provocative and suggest that demcizumab can induce a reduction in the cancer stem cell population in serial transplantation experiments. Most interestingly, demcizumab treatment has resulted in significant tumor shrinkage effects in animal xenograft models despite the lack of an anti-angiogenesis effect or any effect on vascular integrity in these animals.

Demcizumab Clinical Development History. Demcizumab is currently in Phase 1b studies, with drug exposure in 114 patients to date as of May 2013. The lead indications are first-line metastatic non-small cell lung cancer in combination with carboplatin and Alimta, first-line advanced pancreatic cancer in combination with gemcitabine and Abraxane, and recurrent metastatic ovarian cancer in combination with paclitaxel. Clinical trials with demcizumab started in 2008. Demcizumab was originally part of the GSK collaboration, but the worldwide rights to demcizumab were returned to OncoMed in July 2011 following cardiovascular safety issues. The FDA placed demcizumab on partial clinical hold in April 2011. The company was able to get the partial clinical hold lifted in December 2012 through a new risk mitigation proposal: first, an effort to introduce intermittent dosing off demcizumab rather than continuous dosing, careful monitoring of cardiovascular markers and proactive intervention with cardiovascular medication, and then when that failed to prevent cardiovascular events, a new strategy of offering only a short course of therapy was introduced in early 2013 as the latest clinical development plan. Phase 1b/2 studies are underway in lung and pancreatic cancer employing this new dosing paradigm and a Phase 1b/2 study in ovarian is expected to start in 3Q13.

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

Demcizumab Phase 1a Data. A Phase 1 single agent, open-label, dose-escalating study of demcizumab was initiated in 2008 and completed in 2011 after enrollment and treatment of 55 heavily pre-treated cancer patients (median of 4 prior therapies). Patients were treated with a wide range of demcizumab doses: 0.5mg/kg once weekly (qw, n=3), 1.0mg/kg qw (n=3), 2.5 mg/kg qw (n=5), 5.0mg/kg qw (n=2), 2.5mg/kg once every two weeks (q2w, n=4), 5.0mg/kg q2w (n=5), and 10.0mg/kg q2w (n=25). For the qw dosing arms, drug was delivered once weekly for the first 9 weeks and then every other week thereafter. Tumor assessments were conducted at day 56 and every 12 weeks thereafter, and efficacy data were available on 47 patients. Eleven patients experienced some degree of tumor reduction on demcizumab monotherapy. There was an apparent dose response in monotherapy efficacy, which is encouraging because this provides support of activity. At the 10mg/kg q2w dose, there was one partial response observed in a pancreatic patient and significant tumor reductions of 20-30% in a renal cell carcinoma and a rectal cancer patient. The disease control rate (partial responses + stable disease) was 64%. There was one additional near partial response observed at the 2.5mg/kg dose qw in a non-small cell lung cancer patient, whose primary tumor shrank by more than 30% but who concurrently developed a new 0.6 cm lesion. In terms of duration of response, 31% of patients had disease control lasting longer than three months, with one platinum-refractory ovarian cancer patient of note treated with 12 prior regimens who experienced stable disease lasting over 570 days. Data on the duration of response in the pancreatic cancer patient has not been disclosed but will be released when full data are published in a peer-reviewed journal. The last public presentation of these data was in a poster at the AACR-NCI-EORTC meeting in the fall of 2010, at which time data were available on the first 39 patients. These data have subsequently been updated by the company in its prospectus and corporate presentations.

On safety, there were significant toxicities observed in this study including hypertension and rare cardiovascular events. Approximately one-third of patients developed hypertension, with the rate increasing to 40% at the highest dose and approximately 60% of these events being graded as Grade 3 hypertension. Other side effects at greater than 10% frequency including fatigue, anemia, diarrhea, headache, nausea, hypoalbuminemia, dizziness and dyspnea (shortness of breath). While hypertension itself could be a manageable side effect and is commonly observed with anti-angiogenic therapies, the primary concern was the development of clinical heart failure. In this study, three patients developed Grade 3 heart failure, one patient developed Grade 3 right ventricular failure, and one patient developed Grade 4 heart failure. For the three Grade 3 cases, the heart failure diagnosis was initially based on clinical symptoms (shortness of breath, edema) confirmed by at least a 10% decline in ejection fraction, but none of the Grade 3 cases experienced an ejection fraction below 50%. The Grade 4 case experienced a decline in ejection fraction from 52% to 37%. All five patients discontinued demcizumab and had improvement in cardiac function following initiation of medication although no follow-up echos are available to determine if ejection fractions recovered completely. Taken together, we conclude that the data support single agent activity for demcizumab, which we consider promising given the relatively low historical response rate to single agent anti-angiogenesis drugs such as Avastin, but cardiovascular toxicity raises concerns around the therapeutic window for the drug.

Demcizumab Phase 1b Studies. In parallel with the Phase 1a single agent studies, OncoMed initiated three Phase 1b studies of demcizumab with chemotherapy: first-line pancreatic cancer with gemcitabine, first-line NSCLC with carboplatin-Alimta, and first/second-line colorectal cancer with FOLFIRI chemotherapy. The colorectal cancer study was terminated due to reallocation of resources. These Phase 1b studies are being conducted effectively in three parts. The first part occurred prior to the partial clinical hold in the Phase 1a study, in which demcizumab was dosed continuously as induction and maintenance, the second part occurred after dosing protocol changes were implemented

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

to test the hypothesis of intermittent dosing of demcizumab at the beginning of each cycle of chemotherapy, and the third part is now under way to test truncated dosing of demcizumab to 70 days or less.

Safety Issues Cause New Change In Dosing Paradigm: Truncated Dosing. Despite having employed a schedule of intermittent demcizumab dosing, routine monitoring of brain natriuretic peptide (BNP), a marker of heart failure, and proactive treatment with ACE inhibitors for patients with elevations in BNP (two consecutive readings above 100 pg/mL or one reading above 200 pg/mL), two cases of pulmonary hypertension and heart failure were observed in the 5 mg/kg q3w cohort in NSCLC patients who had received extended treatment longer than 150 days with demcizumab. In addition, one patient in the pancreatic study at 5 mg/kg q4w developed pulmonary hypertension after 125 days of dosing. As a result, the Data Safety Monitoring Board (DSMB) for both studies has recommended that the protocol be revised to allow for a limited number of cycles (up to 70 days) of demcizumab with chemotherapy, followed by chemotherapy alone. In the NSCLC study, patients will be eligible to receive 4 cycles of demcizumab q3w with carboplatin-Alimta followed by Alimta maintenance. In the pancreatic study, patients will receive up to 6 cycles of demcizumab q2w with gemcitabine-Abraxane (a concurrent change to the background chemotherapy is being implemented to reflect the shifting standard of care in pancreatic cancer treatment). We believe data from the initial cohorts using the new truncated dosing schedule will be available in 2H13.

First-Line Pancreatic – Demcizumab-Gemcitabine. OncoMed initiated a Phase 1b study of demcizumab and gemcitabine in first-line pancreatic cancer at demcizumab doses of 2.5 mg/kg every 4 weeks (q4w, n=8), 2.5 mg/kg q2w (n=8), and 5 mg/kg q4w (n=8). Among patients treated at the two highest doses, 25% (4/16) achieved a partial response (PR) and 69% (11/16) experienced either a PR or stable disease (SD). With the major caveat that comparisons across studies are not rigorous, especially the comparison of a small, open-label Phase 1b study vs. a pivotal Phase 3 trial, these data compare favorably to gemcitabine alone, where only a 7% PR and 51% PR/SD were observed in the pivotal trial. Data from the Phase 1b study also compare favorably to the expected progression-free survival interval for gemcitabine alone: 164+ days at 5mg/kg q4w vs. 103d for gemcitabine alone. For 3 of the 8 patients at the highest dose of 5mg/kg q4w, dosing was truncated following the protocol revision. We expect data from the Phase 1b study for doses up to 5 mg/kg q4w to be presented at the AACR-NCI-EORTC meeting in October. The pancreatic study continues at 2.5mg/kg q2w of demcizumab with gemcitabine-Abraxane for up to 6 doses of demcizumab. Pending favorable safety, the company plans to escalate the demcizumab dose to 5 mg/kg q2w and 7.5 mg/kg q2w.

First-Line Non-Small Cell Lung Cancer – Demcizumab-Carboplatin-Alimta. OncoMed initiated a Phase 1b study of demcizumab in first-line non-squamous, non-small cell lung cancer patients with carboplatin and Alimta at demcizumab doses of 2.5 mg/kg every 3 weeks (q3w, n=6), 5 mg/kg q3w (n=20, 8 of whom received continuous demcizumab until progression or physician discretion, 8 of whom received demcizumab intermittent dosing, and 4 of whom received truncated demcizumab dosing), and 7.5 mg/kg q3w (n=3, enrollment ongoing). Among the first 22 patients with evaluable responses at the 2.5 or 5 mg/kg q3w doses, 19 experienced some degree of tumor reduction, with 41% (9/22) achieving a PR and 45% (10/22) experiencing SD. Carboplatin-Alimta was associated with only a 27% PR rate in its pivotal trial. Favorable trends were also observed on PFS: 174 days at 5 mg/kg q3w vs. 139 days for carboplatin-Alimta alone. Of note, there were also three patients who experienced very long progression-free intervals: 632+, 680+ and 710 days, respectively, despite having discontinued dosing with demcizumab early (exposure to only 1-6 cycles of demcizumab). Data from the Phase 1b study for doses up to 5 mg/kg q4w have been submitted for presentation at the AACR-NCI-EORTC meeting in October.

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

Initial Read On Truncated Demcizumab Dosing Looks Encouraging. There were three patients treated with truncated dosing of demcizumab-gemcitabine in the pancreatic cancer trial and four patients treated with truncated dosing of demcizumab-carboplatin-Alimta in the non-small cell lung cancer trial before the official protocol was revised to open new arms, and we believe these patients have been treated safely with only 1-2 patients experiencing a modest elevation in BNP and recovery in BNP levels by day 100 following treatment with ACE inhibitors. Of the four patients in the lung cancer cohort who received truncated dosing, two have achieved a partial response and two experienced stable disease. Although these data are difficult to interpret in the absence of a control arm, we are encouraged that the combination appears to have activity even with only 4 cycles of demcizumab.

Demcizumab Second-Line Ovarian Study To Start In 3Q13. As a reminder, in the Phase 1a single agent study of demcizumab, the longest progression-free interval was seen in an ovarian cancer patient, who experienced stable disease on demcizumab for 570 days prior to progressing. In addition, activity in ovarian cancer has been observed with enoticumab, a competing DLL4 antibody in Phase 1 trials. On the basis of this finding, in collaboration with MD Anderson, OncoMed will start an ovarian cancer study in platinum-resistant disease in 3Q13. Patients will be treated with demcizumab and weekly paclitaxel for up to 6 cycles of demcizumab at doses of 2.5 mg/kg q2w, 5 mg/kg q2w, and 7.5 mg/kg q2w.

Predictive Biomarkers Could Be Incorporated Into Phase 2 and 3 Studies For Demcizumab. The company continues to explore the possibility of a biomarker that could predict response to demcizumab. Tumor biopsies are being collected in ongoing Phase 1b studies to explore two possibilities: one is a 12-gene signature related to the Notch pathway and the other involves testing expression of DLL4 on tumor endothelial cells. The company plans on incorporating an analysis of response by one or both of these markers in upcoming randomized Phase 2 trials.

Our Overall Take: High Risk Bet On Efficacy And Safety Of Demcizumab. On efficacy, we are encouraged that single agent responses have been observed in heavily pre-treated patients and that a higher rate of responses with chemotherapy has been observed in both pancreatic and lung cancer relative to historical data. That said, the interpretabilty of the combination data is limited given the lack of a control arm. The critical question is whether the truncated dosing to less than 70 days will allow sufficient exposure to demcizumab to have an effect on responses, progression-free survival intervals, and ultimately, overall survival, since OS is the endpoint for regulatory approval in first-line pancreatic and NSCLC. There are instances where truncated dosing has been effective; for instance, anthracyclines are associated with a cumulative accelerating risk of heart failure, and patients are limited to a certain lifetime exposure limit to the class, but it has become a mainstay of treatment in leukemias, lymphomas, sarcomas, breast and ovarian cancer. For doxorubicin, one of the most commonly used anthracyclines, physicians will typically limit lifetime exposure to 400-450 mg/m2. At an average recommended per cycle dose of 60-75 mg/m2 q3w, this means that the typical patient can receive 6 cycles of anthracycline therapy. On safety, while the data to date would suggest truncated dosing should not lead to an increase in CV risk, there still remain relatively few patients treated with a short course of therapy and the cardiovascular safety of even 70 days of demcizumab still needs to be proven. Thus, we view demcizumab as a high risk development program and have adjusted our probability of success (10-20%) to reflect this risk.

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

OMP-59R5 (Anti-Notch2/3)

Notch Pathway: Target For Demcizumab And GSK Collaborations. In addition to demcizumab, which blocks DLL-4, an activator of Notch signaling, OncoMed and partner GSK are developing Notch pathway targeting biologics, including OMP-59R5, which binds the Notch 2 and 3 receptors, and OMP-52M51, which binds the Notch 1 receptor. Although the Notch signaling pathway is highly evolutionarily conserved and humans have four different Notch receptors, the individual Notch pathway receptors are attractive targets for OncoMed's drug candidates, as they are known to be especially important in CSCs and cancer. The Notch pathway is thought to be critical for the cell fate decision in angiogenesis, the development of new blood vessels, a process which is essential for a tumor to grow. In humans, the Jagged and Delta-like ligands (DLL) communicate with CSCs through the Notch signaling receptors. In normal function, DLL4 binds to Notch and negatively regulates sprouting and branching during tumor angiogenesis, which enables the formation of a functional vascular network. When DLL4 is blocked or inhibited, as with demcizumab, Notch creates non-productive tumor vascularization, effectively inhibiting tumor growth. This mechanism of action differs from drugs such as Avastin that interact with the Notch pathway by inhibiting vascular endothelial growth factor (VEGF), thereby inhibiting angiogenesis and suppressing tumor growth. The complementary but differentiated functions of DLL4 and VEGF in the Notch pathway have led OncoMed to consider developing an Anti-DLL4/Anti-VEGF bispecific monoclonal antibody that would inhibit both DLL4 and VEGF and ideally result in more robust CSC suppression. The molecules under development through the GSK collaboration use an alternate approach, in targeting specific Notch receptors. The lead product candidate from this collaboration, OMP-59R5, binds to a conserved epitope on both Notch 2 and Notch 3 receptors. Notch 2 and Notch 3 are thought to act together to govern vascular development and smooth muscle differentiation. Additionally, OncoMed and GSK are developing OMP-52M51, which targets Notch 1 and is thought to have efficacy in certain solid and hematological cancers.

OMP-59R5 Mechanism Of Action. OncoMed and GSK are developing OMP-59R5, a dual Notch 2/3 receptor targeting antibody derived using phage display technology licensed from MorphoSys. OMP-59R5 was originally identified by binding to Notch2 and subsequently found to also bind Notch3. OMP-59R5 is thought to have two mechanisms of action: down regulating Notch pathway signaling to have anti-CSC effects and affecting pericytes to impact stromal and tumor microenvironment. Pharmacodynamic analyses suggest that Notch pathway modulation is sustained for a week or more after dosing with clear down regulation of Notch 3 in serial tumor biopsies. Notch3 has the potential to be a predictive biomarker of response, with 70% of pancreatic and small cell lung cancers expressing Notch3. GSK retains an option to an exclusive license of OMP-59R5 through the end of Phase 2 trials. OncoMed owns the core patent family in OMP-59R5, which covers both the composition of matter and methods of use, which are expected to expire in 2029.

OMP-59R5 Phase 1. OncoMed initiated a Phase 1 dose escalation trial of OMP-59R5 in 2010 and reported interim Phase 1 data from advanced refractory solid tumor patients at ASCO in 2012. There were no objective responses with single agent treatment, but prolonged stable disease was observed in patients with several types of cancer (Kaposi's Sarcoma, adenoid cystic carcinoma, liposarcoma, triple negative breast cancer [in which JAG1, the ligand of Notch 2/3, was amplified], and rectal cancer). From a safety perspective in the Phase 1a trial, diarrhea was the most common treatment-related adverse event seen in approximately two-thirds of patients. While no Grade 4 or 5 events occurred in that trial, diarrhea was identified as a dose-limiting toxicity at 5mg/kg qw and 10mg/kg q2w. Grade 3 toxicities observed in the 36 patients evaluated included diarrhea

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(14%), anemia (3%), fatigue (3%), increased ALT (3%), and hypokalemia (3%). We are encouraged by the safety profile to date and the absence of major organ toxicities.

OMP-59R5 Phase 1b/2 ALPINE Trial In Pancreatic Cancer. In October, OMED initiated the Phase 1b/2 ALPINE trial (Antibody therapy in first-Line Pancreatic cancer Investigating anti-Notch Efficacy and safety), in which OMP-59R5 was being evaluated in combination with gemcitabine in first-line metastatic pancreatic cancer patients. Initial dosing of OMP-59R5 started at 2.5 mg/kg q2w and then 5 mg/kg q2w. In May 2013, OMED revised this protocol to replace gemcitabine with 1000mg/m2 gemcitabine + 125mg/m2 Abraxane (nab-paclitaxel) for three weeks in a four-week cycle to reflect the shifting standard of care. A third cohort of OMP-59R5 at 5 mg/kg q2w was enrolled with the new background regimen with plans to dose escalate to 7.5 mg/kg q2w. Based on the efficacy and safety data from Phase 1b, the subsequent Phase 2 part of the trial will compare the efficacy of gemcitabine and Abraxane with either OMP-59R5 or placebo and look at two primary endpoints PFS in all comers and in those patients with a particular biomarker, which we believe could be based on immunohistochemistry staining of Notch3. Key secondary endpoints include overall survival, response rate, and safety, which will be assessed in both the general population and the biomarker-specific cohort. As of June 21, OMED reported data from the 12 patients enrolled in the first three cohorts who were assessable for tumor response by RECIST criteria showing stable disease in three of four patients dosed at 2.5mg/kg q2w in combination with gemcitabine and in three of four patients dosed at 5mg/kg q2w in combination with gemcitabine. OMED observed two partial responses in the four patients dosed with 5mg/kg q2w with gemcitabine and Abraxane. From a safety standpoint, only Grade 1 or 2 events have been observed, including fatigue, nausea, rash, diarrhea, thrombocytopenia, and flu-like symptoms. OMED expects to begin Phase 2 by 2014 with final data collection for the PFS endpoint potentially in 2H14.

OMP-59R5 Phase 1b/2 PINNACLE Trial In SCLC. In May, OMED initiated the 80-patient Phase 1b/2 PINNACLE trial (Phase 1b/2 Investigation of anti-Notch Antibody therapy with Cisplatin and etoposide in small cell Lung carcinoma [SCLC] Efficacy and safety), in which OMP-59R5 is being evaluated in combination with cisplatin and etoposide in first-line extensive-stage (ES) SCLC patients. In the Phase 1b dose escalation, OMED is evaluating various doses of OMP-59R5 in combination with cisplatin and etoposide. The primary outcome will be the determination of dose-limiting toxicities (DLTs) through the end of the first 28-day cycle with the goal of determining the maximum tolerated dose (MTD). In the study, cisplatin will be dosed at 80 mg/m2 on day one and etoposide will be dosed at 100mg/m2 on days one through three, every three weeks, which is in the range of current standard of care treatment recommendations for ES SCLC of 60-80mg/m2 cisplatin and 80-120mg/m2 etoposide every 21-28 days. In the expansion phase, a randomized Phase 2 trial, patients with first-line extensive stage SCLC will receive cisplatin and etoposide with either OMP-59R5 or placebo. The primary endpoint will be PFS in the OMP-59R5 arm as compared with the placebo-containing arm. Key secondary endpoints include overall survival, response rate, and safety. Data from the Phase 1b portion of PINNACLE will be available in 2014 and OMED expects to begin the Phase 2 part of this trial in 2014 with final data collection for the PFS endpoint in 2H15.

Our Take: Good Safety Profile, But Efficacy Remains Uncertain. OMP-59R5 has one of the best safety profiles of the clinical stage candidates in the OMED pipeline, with diarrhea emerging as the dose limiting toxicity. The safety profile may even be improved in the setting of chemotherapy combination regimens paradoxically, as the chemotherapy seems to have an effect on eliminating the GI cells that are causing the diarrhea with OMP-59R5. This may allow for even higher doses of OMP-59R5 to be tolerated than the single agent data would predict. That said, efficacy in the lead indications, pancreatic and SCLC, are still unproven. There were no single agent responses in the monotherapy

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study, in which three pancreatic cancer patients were enrolled, but this would not necessarily be expected given the mechanism of action. These cancers were chosen based on OMED's screening of its proprietary tumor bank and strong preclinical data on Notch 2/3 targeting in pancreatic and SCLC. We are encouraged by the initial response rate data in ALPINE at the highest dose to date (5 mg/kg q2w with gemcitabine-Abraxane), in which 2 of 4 patients have achieved a partial response, but these data will need to be confirmed with larger patient numbers and ultimately a randomized Phase 2 trial.

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OMP-52M51 (Anti-Notch1)

OMP-52M51 Mechanism Of Action. For the second collaborative development, OncoMed and GSK are developing OMP-52M51, a humanized monoclonal antibody targeting the Notch1 receptor. The companies speculate that the compound could have utility in both hematologic malignancies and solid tumors, noting malignancies that increased Notch 1 signalizing activities may be primary drivers of tumor growth and chemotherapy resistance.

OMP-52M51 Clinical Data. The companies are currently enrolling two Phase 1a trials to evaluate OMP-52M51 in hematologic and solid tumor malignancies. OncoMed has commented on using diagnostic tests for activated Notch1, which may be used to identify patients most likely to benefit from this therapy and that the company is working to include an analysis of possible predictive biomarkers in subsequent clinical trials to identify the patient subsets in which OMP-52M51 is likely to be most efficacious. In mice, inhibiting Notch 1 has been shown to cause abnormal proliferation of endothelial tissues, leading to vascular tumors. Although the company has limited safety data to-date, in the Phase 1a trials, only diarrhea and nausea have occurred in more than one patient. GSK has an early option to obtain an exclusive license through the end of Phase 1 trials and a standard option for a license through the end of Phase 2 trials. OncoMed owns the anti-Notch1 portfolio core patent family that is expected to expire in 2029.

Phase 1 Trial Of OMP-52M51. In December 2012, OMED initiated a 54-patient open-label, single-agent, dose escalation, and expansion Phase 1 clinical trial to evaluate OMP-52M51 in hematologic cancers, and in March 2013, OMED initiated a 33-patient Phase 1 trial to evaluate OMP-52M51 in advanced refractory solid tumors. In each trial, OMED is evaluating six doses of OMP-52M51. OMED has limited efficacy data to date, and has disclosed only that drug-related adverse events of diarrhea and nausea were observed in two patients. The clinical trials will assess safety, pharmacokinetics, pharmacodynamics, and efficacy via biomarker-based patient selection, as OME believes certain malignancies have activated Notch1 may benefit disproportionately from OMP-52M51 treatment. Data from the solid tumor Phase 1a study has been submitted for presentation at the AACR-NCI-EORTC meeting in October. OMED expects that predictive biomarkers could be used to identify potential responders, thereby allowing for early proof-of-concept.

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Vantictumab (Anti-Fzd7)

Wnt Pathway. OncoMed and partner Bayer are developing several biologics and potentially some small molecules inhibitors that target the wnt signaling pathways, which are thought to be involved in breast cancer, colorectal cancer, melanoma, prostate cancer, and lung cancer. There are three wnt pathways: the canonical pathway, responsible for activation of beta-catenin signaling, the noncanonical planar cell polarity pathway, and the noncanonical Wnt/calcium pathway. OncoMed and Bayer have taken two wnt targeting compounds into the clinic: vantictumab (OMP-18R5), an anti-Fzd7 antibody, and OMP54F28, an Fzd8-Fc, both of which exploit Frizzled (Fzd) receptor signaling to inhibit the wnt pathway. There are 10 Frizzled receptors that are G-protein-coupled receptor proteins on a cell membrane that are bound by Wnt protein ligands in order to pass signals to the protein Dishevelled inside the cytoplasm. Dishevelled is thought to play a key role in cell differentiation. Vantictumab was identified for its ability to bind to Fzd7, but subsequently found to bind to 5 of the 10 Fzd receptors (Fzd1, Fzd2, Fzd5, Fzd7, Fzd8), as it binds to a discontinuous epitope that is conserved across the Fzd family. Vantictumab was found to block most beta-catenin signaling in response to the wnt ligands, thereby reducing the CSC frequency. Additionally, Bayer and OncoMed are developing OMP-54F28, a proprietary fusion protein based on a truncated form of the Frizzled8 receptor. OMP-54F28 competes with the membrane-bound Fzd8 receptor for its ligand, thereby antagonizing the Wnt signaling pathway and potentially inhibiting Wnt-driven tumor growth. In addition to the two named compounds, Bayer and OncoMed are working to develop a third, as-yet-unnamed bispecific wnt biologic and two small molecule drugs that also target the wnt pathway.

Vantictumab Mechanism Of Action. The lead wnt candidate is vantictumab (OMP-18R5), a humanized monoclonal antibody against the Fzd7 receptor that inhibits canonical wnt signaling. Antibodies to Fzd were first identified using a phage display library licensed from MorphoSys as it was shown to block most beta-catenin signaling in response to Wnt3a. Vantictumab was isolated by ability to bind Fzd7 and, in subsequent experiments, was shown to inhibit beta-catenin signaling in response to other wnt family members. Vantictumab was found to bind to five of the ten Fzd receptors: Fzd1, Fzd2, Fzd5, Fzd7, and Fzd8. Separately, the epitope on Fzd proteins bound by vantictumab was mapped through amino acid substitutions within Fzd8, leading OncoMed to conclude that it bound to a discontinuous epitope that spans a “cleft” region in the crystal structure of mouse Fz8. The residues lining the base of the cleft were seen to be highly conserved across the Fzd family, suggesting that this site is functionally important. OncoMed also conducted binding studies with cell lines from glioblastoma, lung, breast, and prostate cancers, concluding that vantictumab is effective at blocking wnt singaling from multiple wnts in cell lines derived from distinct tissues. In addition to reducing xenograft tumor volume in pancreatic, breast, lung, melanoma, hepatocellular, ovarian, colorectal, and other cancers, vantictumab reduces CSC frequency in preclinical models and induces differentiation of tumorigenic cells to less tumorigenic cell types that are more susceptible to conventional chemotherapy.

Vantictumab Phase 1a Trial. In 2011, Vantictumab was advanced into a 44-patient Phase 1 single-agent, dose-escalation trial in advanced solid tumor patients, interim data from which was reported at ASCO in June 2013. A wide range of doses have been tested to date: 0.5 mg/kg every week, 1 mg/kg every week, 0.5 mg/kg every 2 weeks, 1 mg/kg every 3 weeks, 2.5 mg/kg every 3 weeks, 5 mg/kg every 3 weeks, and 10 mg/kg every 3 weeks. In this trial, Vantictumab was shown to be tolerable and exhibited single-agent activity in patients with neuroendocrine tumors (NETs). Three of three patients with NETs (one pancreatic NET, two carcinoid tumors) have been enrolled in the Phase 1 trial and all have achieved clinical benefit, with one patient receiving study treatment for 110 days and two patients remaining on study drug for over 316 and 384 days as of May 2013. The

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

trial identified a dose-limiting toxicity of Grade 3 vomiting and diarrhea in one patient dosed at 1mg/kg once weekly. Bone toxicity is a potential on-target concern, and following a Grade 2 compression fracture in a patient with a minor fall, the trial was revised to include a safety plan to identify on-target bone toxicity and there were four patients in whom zoledronic acid was administered following a doubling of beta-CTX, a serum marker for bone turnover. As of the May 8 look, the ongoing Phase 1a trial safety database for the 24 patients included fatigue (30%), nausea (22%), vomiting (17%), increased alkaline phosphatase (13%), constipation (13%), decreased appetite (13%), and hypercalcemia (13%). OncoMed noted that vantictumab was generally well tolerated up to the current dose of 10mg/kg every three weeks. Full data from the Phase 1a study should be presented at ASCO 2014. OncoMed and partner Bayer expect to initiate three Phase 1b clinical trials in late 2013-early 2014 in distinct solid tumor indications in combination with standard-of-care therapies. We believe these may include HER2-negative breast cancer in combination with paclitaxel, pancreatic cancer with gemcitabine-Abraxane, and non-small cell lung cancer with docetaxel. Bayer retains an option to license vantictumab through the completion of the Phase 1b trials in combination with chemotherapy. OncoMed owns the core patent family covering composition of matter and methods of use of vantictumab, which are expected to expire in 2029 with additional patent applications related to non-provisinal applications that issue through 2032.

Our Take: Vantictumab Efficacy Looks Intriguing In NET, More Data Needed On Bone Effects. We are encouraged that all three patients enrolled with neuroendocrine tumors had prolonged stable disease on vanctictumab monotherapy, and we interpret this as a positive signal of single agent activity. There is the possibility of developing vantictumab in the NET setting under an accelerated pathway, but this will likely be a decision made by Bayer if they choose to trigger their option to license vantictumab in 2015. Although no responses were observed in other tumor settings as monotherapy, combination chemotherapy studies may still yield efficacy in major solid tumors. From a safety perspective, the primary concern emerging are the bone effects, which will require more patient exposure to understand the manageability of this side effect with careful monitoring and zoledronic acid intervention and how this affects the overall risk-benefit profile.

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

OMP-54F28 (Fzd8 Fc)

OMP-54F28 Mechanism Of Action. OMP-54F28 is the second product candidate from OncoMed and Bayer targeting the wnt pathway. Employing a mechanism distinct from vantictumab, OMP-54F28 is a fusion protein, or decoy receptor, based on a truncated form of the Frizzled8 (Fzd8) receptor that functions by binding Wnt ligands. To identify an appropriate decoy, OncoMed employed a standard prototype wnt assay consisting of 293 cells transfected with 8x-TCF reporter and co-incubated these cells with 293 cells transfected with expression vectors for each of the 19 Wnt family members. Those Wnts that were observed to induce singling were then studied in the presence of a range of concentrations of 10 Fzd-decoy proteins. Only Fzd4, Fzd5, and Fzd8 were able to inhibit signaling in response to Wnt3a and other wnt family members. Building from these data, OncoMed and Bayer selected OMP-54F28, a truncated Fzd8, to advance into the clinic. From a preclinical standpoint, OMP-54F28 has shown anti-tumor activity in solid tumors including pancreatic, breast, hepatocellular, ovarian, and colorectal cancers, as well as reducing CSC frequency in multiple preclinical models.

OMP-54F28 Phase 1a Trial. OMP-54F28 is in an ongoing, 36-patient Phase 1 dose-escalating single agent trial in patients with advanced solid tumors, which the companies expect to be presented at the AACR-NCI-EORTC meeting in October. In the Phase 1 trial, OncoMed has identified treatment-related adverse events including decreased appetite (23%), fatigue (23%), hypocalcemia (23%), nausea (23%), altered taste (15%), increased blood pressure (15%), peripheral edema (15%) and vomiting (15%). OncoMed and Bayer plan to initiate three Phase 1b clinical trials in late 2013 or early 2014. Target indications have not been disclosed. Bayer retains the rights to an exclusive license of OMP-54F28 through completion of certain Phase 1 testing. OncoMed owns the patent family covering the composition of matter and methods of use of OMP-54F28 that are expected to expire in 2031and has related patent applications pending that are expected to issue and could extend coverage through 2032.

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

Preclinical Pipeline And Collaborations

Additional Wnt-Targeting Compounds. Although the number of compounds developed through their collaboration is technically uncapped in number, OncoMed and Bayer have announced their intention to develop three biologics and two small molecules that target the wnt pathway. OncoMed and Bayer have commented on their plans to advance a third wnt biologic in preclinical studies, namely a bispecific biological product. Bayer and OncoMed have been sparing in their commentary around the small molecule wnt pathway inhibitors in development, but we expect further details as these product candidates evolve through preclinical testing.

RSPO-LGR Pathway. OncoMed identified the R-spondin (RSPO) ligands signal through the LGR receptor family, which is thought to be an important CSC pathway. The RSPO-LGR pathway consists of four R-spondins (RSPO1-4) that function in cell signaling through receptor proteins LGR4-6. Recent studies have shown that LGR receptors are specifically distributed on adult stem cells and that LGR-expressing cells are linked to cancer development. OncoMed is currently conducting preclinical studies of RSPO-LGR pathway-modulating antibodies and plans to enter clinical trials with an RSPO-LGR targeting drug candidate in 2014, enabling a Phase 1b trial in 2016.

GSK Collaboration. In December 2007, OncoMed entered into a partnership with SmithKline Beecham Corporation (now GSK) to develop anti-CSC drug candidates targeting the Notch signaling pathway. At the time, OncoMed received $35m in cash, consisting of a $17.5m upfront payment and a $17.5m equity investment and was eligible to receive up to $1.4b in conjunction with development of four product candidates. In 2010 and 2012, OncoMed received $9m and $14m in development milestone payments. In July 2011, OncoMed and GSK modified the development agreement to focus entirely on two product candidates, OMP-59R5 and OMP-52M51, as GSK had agreed to terminate its option to obtain exclusive rights to demcizumab and the DLL4/VEGF bispecific. Under this revised agreement, GSK has the right to exercise an option to obtain an exclusive license to develop and commercialize OMP-59R5 and OMP-52M51through the completion of proof-of-concept or through the completion of Phase 1 trials of OMP-52M51. Separately, under certain circumstances, OncoMed may owe GSK single-digit royalties on net product sales of demcizumab. OncoMed is eligible to receive aggregate payments of up to $344.5m on OMP-59R5 and $349.5m on OMP-52M51, including option exercise fee and development, regulatory, and commercial milestones, in addition to up to $2m in funding to support development activities conducted in conjunction with one of these product candidates and $15m in bonus payments on clinical success. In June 2013, OncoMed received an $8m payment from GSK for the OMP-59R5 program, in addition to the $17m earned through March 30 and the $14m earned on OMP-52M51. On both product candidates, OncoMed is eligible to a percentage of royalties in the low-to-high teens on net product sales. If GSK does not exercise its options to either program during the relevant option periods, OncoMed would have worldwide rights to these programs. In July 2012, OncoMed revised the structure of the milestone payments to reflect its decision to initiate a Phase 1b/2 trial for OMP-59R5. Under the amended agreement, there are several committees that meet to discuss the collaboration and make development decisions jointly, prior to GSK's decision to exercise its development option. Although obligated to make commercially reasonable efforts to progress the two compounds in clinical development, OncoMed is not subject to a committee determination on its allocation of resources and there are no explicit expenditure requirements.

Bayer Collaboration. In June 2010, OncoMed entered a partnership with Bayer Schering Pharma AG (now Bayer Pharma AG) to discover and develop novel anti-CSC biologics and small molecules that target the Wnt signaling pathway. OncoMed received $40m in an upfront cash payment and Bayer acquired rights to exercise an option to

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

obtain an exclusive license to develop and commercialize biologic therapeutics through the completion of Phase 1 trials. Both OncoMed and Bayer agreed to jointly conduct research to discover potential new small molecule therapeutics targeting the wnt pathway with OncoMed leading the discovery and development of the therapeutics prior to Bayer exercising its option. OncoMed is eligible to receive payments of up to $387.5m for each biologic therapeutic, including option fees and research, development, regulatory, and commercial milestones. Royalty percentages differs for each candidate: for vantictumab, royalties range from the low-to-high teens, and for the Fzd8-Fc, royalties range from the high single digits to mid-teens. For the small molecule candidates under development, Bayer could pay up to $112m per program, including research, development, regulatory, and commercial milestones and advancement fees. OncoMed is eligible to earn single-digit percentage royalties payments on net product sales for the small molecule compounds. If Bayer elects not to exercise its options for any class of biologic products, OncoMed will regain worldwide rights to the program. In August 2012, OncoMed amended its agreement with Bayer to reallocate certain amounts between two milestone payments applicable to the biologic product candidate development and redefine when payments applicable to certain biologic product candidates are due. OncoMed received $20m in development milestones from Bayer in 2011 and $5m related to the OMP-54F28 in 2012. While the number of potential therapeutics developed through this collaboration remains uncapped, the parties intend to advance three biologics and two small molecules. Under this agreement, decisions are generally made jointly, but OncoMed has final decision making authority on the biologic product candidates through Phase 1 of development and Bayer has final decision making authority for biologic products in later stages of development assuming it exercises its option to license.

University of Michigan. In January 2001, OncoMed entered into a license agreement with the University of Michigan granting exclusive, royalty-bearing, worldwide license under certain patent rights to certain technologies. Under the terms of this agreement, OncoMed will pay the University of Michigan an annual license maintenance fee and reimburse for prosecution and maintenance of patents. OncoMed is required to pay the University of Michigan royalties in the low-single digits based on net sales of products protected under the patents including demcizumab. After paying $10m in royalties to the University of Michigan, OncoMed can convert the license to a fully paid-up license by transferring 0.25% of their non-voting capital stock.

MorphoSys. In June 2006, OncoMed entered into a license agreement with MorphoSys to obtain access to phage display technologies and the licenses to related patents, which were used to identify antibodies such as OMP-59R5 and OMP-18R5. OncoMed obtained a worldwide, non-exclusive, royalty-free extended research license to use antibodies identified during the subscription term, for which OncoMed pays an annual license maintenance fee of EUR20,000 through 2015. For commercial therapeutic licenses, OncoMed will make milestone payments for certain events of up to EUR5.8m per product, and pay tiered single-digit royalties on net sales of licensed products on a country-by-country basis. GSK will reimburse 50% of such payments for OMP-59R5.

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Management

Paul Hastings - President and CEO. Prior to joining OncoMed in 2006, Mr. Hastings was President and CEO of QLT, Inc. Prior to that, Mr. Hastings served as President and CEO of Axys Pharmaceuticals, which was acquired by Celera Corporation in 2001. Prior to Celera, Mr. Hastings served as President of Chiron BioPharmaceuticals, a division of Chiron Corporation. Prior to Chiron, Mr. Hastings was President and CEO of LXR Biotechnology. Mr. Hastings received a B.S. in pharmacy from the University of Rhode Island.

John Lewicki, PhD - EVP and CSO. Prior to joining OncoMed, Dr. Lewicki was SVP, R&D of an early-stage biotechnology company working in the infectious disease field. From 1983-2000, Dr. Lewicki served in various capacities at Scios, Inc. where he managed the company research across diverse therapeutic areas. Dr. Lewicki received his PhD in Physiology/pharmacology from U.C. San Diego.

Jakob Dumont, M.D. - SVP, CMO. Prior to joining OncoMed, Dr. Dumont was at Genentech, a member of the Roche Group, where he was Clobal Medical Director for Avastin, having held leadership positions at stages of oncology drug development. Prior to Genentech, Dr. Dumont held leadership positions on the Avastin franchise for the Breast and Ovarian cancer indication. Dr. Dumont received his M.D. from Cornell and continues to teach as an adjunct clinical professor at Stanford.

William Waddill - SVP and CFO. Prior to joining OncoMed, Mr. Waddill was SVP and CFO at Ilypsa where he was responsible for corporate financial matters. Prior to Ilypsa, Mr. Waddill was founder and principal at Square One Finance. Previously, Mr. Waddill, held positions at Exelixis as Senior Director, Finance and Administration. Mr. Waddill received a BS in accounting from the University of Illinois, Chicago and certification as a public accountant.

Initial Public Offering

In July, Jefferies acted as lead book-runner agent in an initial public offering of 5.52m OMED common shares at $17 per share.

Company Overview

OncoMed Pharmaceuticals (OMED) is a Redwood City, CA-based biopharmaceutical company that is a leader in the science behind cancer stem cells (CSCs), which are thought to drive cancer progression, metastasis, and chemotherapy resistance. Using proprietary technology, OMED has generated five clinical stage candidates targeting CSC pathways. Four of these compounds are being developed under two pharmaceutical partnerships with GSK and Bayer. The lead wholly owned drug is demcizumab, an anti-DLL4 antibody currently in Phase 1b trials in pancreatic, lung and ovarian cancer. Close behind in development are OMP-59R5, an anti-Notch2/3 antibody, OMP-52M51, an anti-Notch1 antibody, vantictumab, an anti-Fzd7 antibody, and OMP-54F28, a Fzd8-Fc fusion protein.

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OMED: Historical and Projected Revenue and Earningss

December 31 Fiscal Year ($000s)	2012A	1Q13A	2Q13E	3Q13E	4Q13E	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E
Demcizumab Sales															
U.S. Demcizumab Sales	0	0	0	0	0	0	0	0	0	0	101,592	431,341	752,003	1,331,096	1,732,391
International Demcizumab Sales	0	0	0	0	0	0	0	0	0	0	0	40,637	258,804	601,603	1,064,877
WW Demcizumab Sales	0	0	0	0	0	0	0	0	0	0	101,592	471,977	1,010,808	1,932,699	2,797,268
Y/Y Change	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	365%	114%	91%	45%
Demcizumab Royalties to Oncomed	0	0	0	0	0	0	0	0	0	0	16,255	73,891	161,457	335,720	508,749
% Of Sales	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	16%	16%	16%	17%	18%
GSK/Bayer Royalties to Oncomed	0	0	0	0	0	0	0	0	0	0	29,232	98,250	152,203	195,561	268,508
Demcizumab Upfront/Milestone Payments	0	0	0	0	0	0	0	25,000	65,000	75,000	110,000	100,000	85,000	75,000	45,000
GSK/Bayer Upfront/Milestone Payments	24,659	2,932	11,000	10,000	15,000	38,932	47,000	48,000	50,000	92,797	158,676	223,173	164,026	132,542	199,367
Other Revenues	22	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total Revenue	**24,681**	**2,932**	**11,000**	**10,000**	**15,000**	**38,932**	**47,000**	**73,000**	**115,000**	**167,797**	**314,162**	**495,314**	**562,687**	**738,823**	**1,021,624**
Y/Y Change	NM	17%	120%	100%	23%	58%	21%	55%	58%	46%	87%	58%	14%	31%	38%
COGS	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
% Product sales	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Research and Development	39,396	9,436	11,479	13,521	15,564	50,000	75,000	35,000	45,500	59,150	76,895	99,964	129,953	168,938	219,620
Y/Y Change	NM	-16%	28%	93%	28%	27%	50%	-53%	30%	30%	30%	30%	30%	30%	30%
% Total Revenue	NM	NM	NM	NM	NM	NM	160%	48%	40%	35%	24%	20%	23%	23%	21%
Selling, General and Administrative	6,818	1,900	2,633	3,367	4,100	12,000	12,960	13,997	15,396	16,936	18,630	20,493	22,542	24,796	27,276
Y/Y Change	NM	13%	NM	NM	-20%	76%	8%	8%	10%	10%	10%	10%	10%	10%	10%
% Total Revenue	28%	65%	24%	34%	27%	31%	28%	19%	13%	10%	6%	4%	4%	3%	3%
Total Operating Expenses	**46,214**	**11,336**	**14,112**	**16,888**	**19,664**	**62,000**	**87,960**	**48,997**	**60,896**	**76,086**	**95,525**	**120,456**	**152,495**	**193,735**	**246,896**
Income From Operations	**(21,533)**	**(8,404)**	**(3,112)**	**(6,888)**	**(4,664)**	**(23,068)**	**(40,960)**	**24,003**	**54,104**	**91,711**	**218,638**	**374,858**	**410,192**	**545,088**	**774,728**
Operating margin	NM	NM	NM	NM	NM	NM	NM	33%	47%	55%	70%	76%	73%	74%	76%
Total Other Income (Expense), Net	134	31	73	171	164	439	751	1,007	1,246	1,459	1,912	2,748	3,932	5,413	7,415
Interest income	140	31	73	171	164	439	751	1,007	1,246	1,459	1,912	2,748	3,932	5,413	7,415
Interest expense	-6	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Pretax-Income	(21,399)	(8,373)	(3,039)	(6,717)	(4,500)	(22,629)	(40,209)	25,010	55,350	93,170	220,549	377,606	414,124	550,502	782,143
Income Tax Expense	0	0	0	0	0	0	0	9,254	20,479	34,473	81,603	139,714	153,226	203,686	289,393
Tax Rate	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	37.0%	37.0%	37.0%	37.0%	37.0%	37.0%	37.0%	37.0%
Non-GAAP Net Income	**(21,399)**	**(8,373)**	**(3,039)**	**(6,717)**	**(4,500)**	**(22,629)**	**(40,209)**	**15,757**	**34,870**	**58,697**	**138,946**	**237,892**	**260,898**	**346,816**	**492,750**
Y/Y Change	NM	-19%	-24%	236%	-11%	6%	78%	-139%	121%	68%	137%	71%	10%	33%	42%
Shares Outstanding	1,028	22,265	22,315	27,835	27,885	25,075	29,783	30,302	33,689	30,752	34,202	31,302	34,815	31,952	35,515
Non-GAAP EPS	**($20.81)**	**($0.38)**	**($0.14)**	**($0.24)**	**($0.16)**	**($0.90)**	**($1.35)**	**$0.52**	**$1.04**	**$1.91**	**$4.06**	**$7.60**	**$7.49**	**$10.85**	**$13.87**
Y/Y Change	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	84%	43%	85%
Options Expense	786	225	250	300	350	1,125	1,350	1,620	1,944	2,333	2,799	3,359	4,031	4,837	5,805
% Operating Income	NM	NM	NM	NM	NM	NM	NM	6.7%	3.6%	2.5%	1.3%	0.9%	1.0%	0.9%	0.7%
GAAP EPS	**($21.58)**	**($0.39)**	**($0.15)**	**($0.25)**	**($0.17)**	**($0.95)**	**($1.40)**	**$0.49**	**$1.00**	**$1.86**	**$4.01**	**$7.53**	**$7.42**	**$10.76**	**$13.77**

Source: Company data, Jefferies LLC estimates
August 9, 2013

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

OMED: Historical and Projected Changes in Financial Position

December 31 Fiscal Year ($000s)	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022
Net income	**(21,399)**	**(22,629)**	**(40,209)**	**15,757**	**34,870**	**58,697**	**138,946**	**237,892**	**260,898**	**346,816**	**492,750**
Adjustments to reconcile net cash flows from operating activities:											
Depreciation and amortization	1,295	1,395	1,595	1,695	1,995	2,195	2,495	2,695	2,995	3,195	3,495
Deferred rent and other long-term liabilities	(12)										
Utilization of NOL carryforwards	-	-	-	9,254	20,479	34,473	594	-	-	-	-
Upfront/milestone payments	-	-	-	50,000	(25,000)	(25,000)	-	-	-	-	-
Total Adjustments	**1,283**	**1,395**	**1,595**	**60,949**	**(2,526)**	**11,668**	**3,089**	**2,695**	**2,995**	**3,195**	**3,495**
Changes in operating assets and liabilities:											
Accounts receivable	(4,023)	(2,375)	(1,345)	(4,333)	(7,000)	(8,800)	(24,394)	(30,192)	(11,229)	(29,356)	(47,134)
Prepaid expenses and other current	(3,411)										
Accounts payable and accrued liabilities	(3,184)										
Deferred revenue	(2,165)										
Other	(446)										
Net cash flows provided by operating activities	**(33,345)**	**(23,609)**	**(39,958)**	**72,372**	**25,345**	**61,566**	**117,641**	**210,395**	**252,665**	**320,655**	**449,111**
Cash flows from investing activities:											
Acquisitions, net of cash acquired	-										
Capital Expenditure	(714)	(1,000)	(2,000)	(3,000)	(4,000)	(5,000)	(5,000)	(5,000)	(5,000)	(5,000)	(5,000)
Net cash flows used in investing activities	**(714)**	**(1,000)**	**(2,000)**	**(3,000)**	**(4,000)**	**(5,000)**	**(5,000)**	**(5,000)**	**(5,000)**	**(5,000)**	**(5,000)**
Cash flows from financing activities:											
Proceeds (repurchases) from common stock	156	89,103	72,912	2,012	2,943	4,251	7,661	8,622	12,142	16,999	23,677
Proceeds (repayments) from borrowings	(346)										
Other	-										
Net cash flows used in financing activities	**(190)**	**89,103**	**72,912**	**2,012**	**2,943**	**4,251**	**7,661**	**8,622**	**12,142**	**16,999**	**23,677**
Net increase (decrease) in cash and cash equivalents	**(34,249)**	**64,494**	**30,953**	**71,385**	**24,288**	**60,817**	**120,303**	**214,016**	**259,807**	**332,654**	**467,789**
Exchange rate changes			-	-	-	-	-	-	-	-	-
Cash and cash equivalents, beginning of the year	**104,554**	**70,305**	**134,799**	**165,752**	**237,137**	**261,425**	**322,242**	**442,545**	**656,561**	**916,368**	**1,249,022**
Cash and cash equivalents, end of the year	**70,305**	**134,799**	**165,752**	**237,137**	**261,425**	**322,242**	**442,545**	**656,561**	**916,368**	**1,249,022**	**1,716,811**

Source: Company data, Jefferies LLC estimates
August 9, 2013

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

OMED: Historical Condensed Balance Sheets

	12/31/2012	3/31/2013
Current assets:		
Cash and cash equivalents	16,263	9,937
Short-term investments	49,976	50,282
Receivables – related parties	4,023	23
Prepaid and other current assets	1,123	1,222
Total current assets	71,385	61,464
Property and equipment, net	5,462	5,190
Other assets	2,921	3,170
Total assets	79,768	69,824
Current liabilities:		
Accounts payable	849	809
Accrued liabilities	3,798	5,348
Current portion of deferred revenue	14,726	14,726
Current portion of deferred rent	560	579
Liability for shares issued with repurchase rights	14	12
Convertible preferred stock warrant liability	182	161
Total current liabilities	20,129	21,635
Deferred revenue, less current portion	17,320	14,388
Deferred rent, less current portion	3,750	3,598
Liability for shares issued with repurchase rights, less current portion	23	21
Total liabilities	41,222	39,642
Stockholder equity		30,182
Total liabilities and stockholder equity		69,824

Source: Company data, Jefferies LLC estimates
August 9, 2013

Source: Company data, Jefferies LLC estimates

Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

Company Description

OncoMed Pharmaceuticals (OMED) is a Redwood City, CA-based biopharmaceutical company that is a leader in the science behind cancer stem cells (CSCs), which are thought to drive cancer progression, metastasis, and chemotherapy resistance. Using proprietary technology, OMED has generated five clinical stage candidates targeting CSC pathways. Four of these compounds are being developed under two pharmaceutical partnerships with GSK and Bayer. The lead wholly owned drug is demcizumab, an anti-DLL4 antibody currently in Phase 1b trials in pancreatic, lung and ovarian cancer. Close behind in development are OMP-59R5, an anti-Notch2/3 antibody, OMP-52M51, an anti-Notch1 antibody, vantictumab, an anti-Fzd7 antibody, and OMP-54F28, a Fzd8-Fc fusion protein.

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Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

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4. Stocks are sorted to a maximum of 30 stocks with the maximum country exposure at around 50%. Limits are also imposed on a sector basis.
5. Once a month, analysts are invited to recommend their best ideas. Analysts' stock selection can be based on one or more of the following: non-Consensus investment view, difference in earnings relative to Consensus, valuation methodology, target upside/downside % relative to the current stock price. These are then assessed against existing holdings to ensure consistency. Stocks that have either reached their target price, been downgraded over the course of the month or where a more suitable candidate has been found are removed.
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9. Transaction fees are not included.
10. All corporate actions are taken into account.

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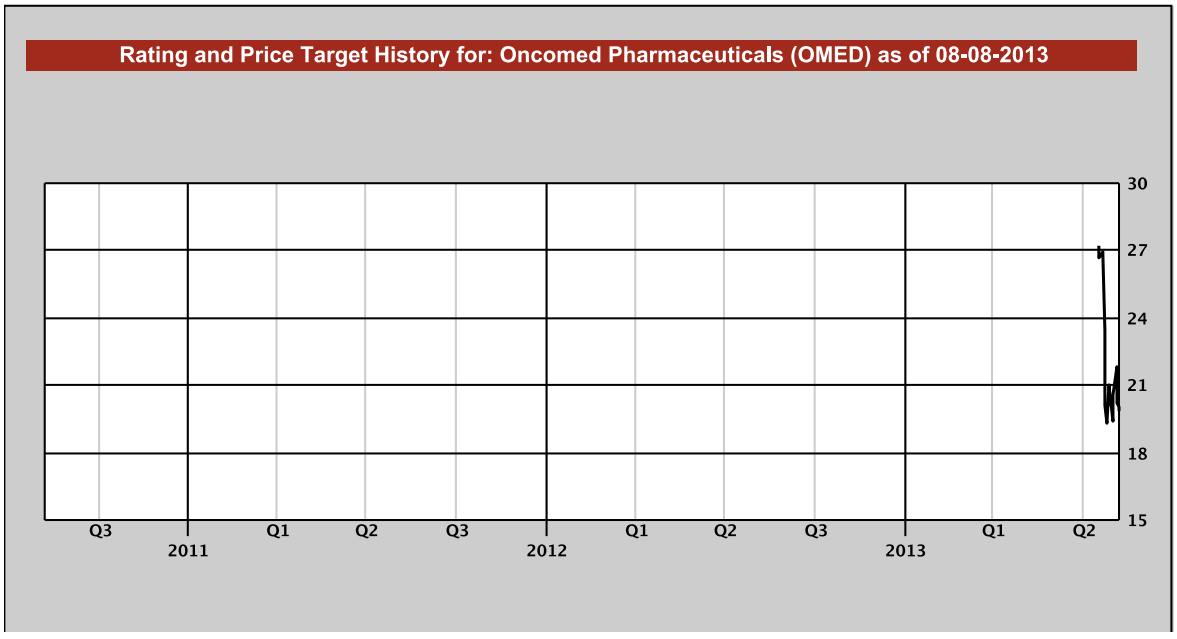
- Arena Pharmaceuticals, Inc. (ARNA: $7.51, BUY)
- AstraZeneca PLC (AZN LN: p3,235.00, HOLD)
- Dynavax Technologies Inc. (DVAX: $1.36, BUY)
- GlaxoSmithKline Plc (GSK LN: p1,663.00, HOLD)
- Neurocrine Biosciences (NBIX: $14.43, BUY)
- Regeneron Pharmaceuticals, Inc. (REGN: $252.49, HOLD)
- Rigel Pharmaceuticals, Inc. (RIGL: $4.12, BUY)
- Sanofi (SAN FP: €78.85, BUY)



Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

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Rating	Count	Percent	IB Serv./Past 12 Mos. Count	IB Serv./Past 12 Mos. Percent
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HOLD	752	44.84%	116	15.43%
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Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com

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Thomas Wei, Equity Analyst, (212) 284-2326, twei@jefferies.com