

COMPANY NOTE

Initiating Coverage

USA | Healthcare | Biotechnology

February 4, 2014

Jefferies

GlycoMimetics, Inc. (GLYC) Initiate With A Buy - Two Clinical Programs Move Forward In 2014

Key Takeaway

We believe GLYC shares are attractive with meaningful upside as its clinical programs become de-risked over the next 12-24 months. GLYC's partner Pfizer is planning to initiate Phase III trials testing GMI-1070 in sickle cell disease patients hospitalized due to vaso-occlusive crises. We believe the Phase III has 75% odds of success. Furthermore, GLYC will initiate a Phase I study in Q2 '14 with GMI-1271 in AML and other hematological malignancies.

JEF was joint book-running manager for GLYC's IPO on Jan 9, 2014.

GMI-1070 Starts Phase III Trials In Q3: The company's partner, Pfizer (PFE, \$30.60, Hold), is planning to initiate Phase III trials for GMI-1070 in patients with vaso-occlusive crises. We currently estimate 75% odds of success and estimate at a minimum the Phase III would need to be sized between 300-400 patients to achieve statistical significance with a more conservative 30 hour improvement in resolution of vaso-occlusive crises. The Phase II data showed clinically meaningful improvements in resolution of vaso-occlusive crises by a mean of 40 hours, time to hospital discharge by more than 2 days, and 80% reduction in the need for opioids.

GMI-1070 Timelines To Market: With Phase III trials expected to start in Q3, we anticipate it would take 2 years to complete patient enrollment, and expect data in 2H 2016 and potential market approval in late '17. We estimate approximately 66K U.S. hospitalizations annually due to vaso-occlusive crises, and estimate peak risk-adjusted U.S. sales of \$563M, of which GLYC would receive low teen royalties from Pfizer.

GMI-1271 Offers Pathway To Tackle Chemoresistance In AML: GMI-1271, an E-selectin inhibitor, will initiate Phase I trials in Q2 '14. Preclinical data reported reductions in tumor volume by 50-67% when combined with chemotherapy vs chemotherapy alone. E-selectin are found to be overexpressed in both solid and hematopoietic malignancies including acute myeloid leukemia (AML). E-selectin has been suggested as one of the factors responsible for metastasis of cancers. We currently estimate risk-adjusted peak sales of \$78M for GMI-1271.

Valuation/Risks

We arrive at our \$13 PT by using a DCF methodology. Risks to our estimates include clinical trial failure, regulatory approval risks, and commercial launch risks.

USD	Prev.	2013E	Prev.	2014E	Prev.	2015E	Prev.	2016E
Rev. (MM)	--	5.2	--	35.0	--	3.1	--	25.0
EV/Rev		24.1x		3.6x		40.4x		5.0x
Cons. EPS	--	--	--	--	--	--	--	--
EPS								
Mar	--	(2.18)A	--	(0.24)	--	--	--	--
Jun	--	(2.16)A	--	(0.28)	--	--	--	--
Sep	--	(2.09)	--	1.57	--	--	--	--
Dec	--	(0.22)	--	(0.31)	--	--	--	--
FY Dec	--	(6.65)	--	0.74	--	(1.41)	--	(0.02)
FY P/E		NM		13.5x		NM		NM

BUY

Price target \$13.00

Price \$10.01

Financial Summary

Net Debt (MM):	(\$60.9)
Long-Term Debt (MM):	\$0.0
Cash & ST Invest. (MM):	\$60.9
Cash/Share:	\$3.27
Cash (MM):	\$60.9

Market Data

52 Week Range:	\$11.99 - \$8.40
Total Entprs. Value (MM):	\$125.3
Market Cap. (MM):	\$186.2
Insider Ownership:	57.0%
Institutional Ownership:	43.0%
Shares Out. (MM):	18.6
Float (MM):	7.1
Avg. Daily Vol.:	NA

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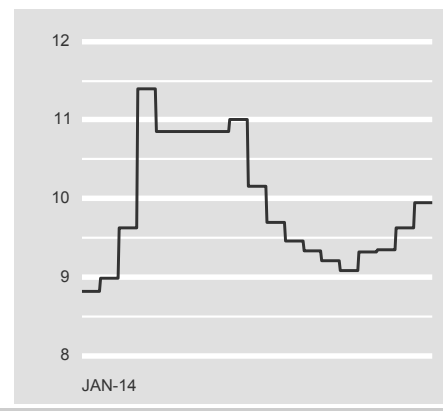
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Price Performance



GLYC

Initiating Coverage

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GlycoMimetics, Inc.**Buy: \$13 Price Target****Scenarios****Target Investment Thesis**

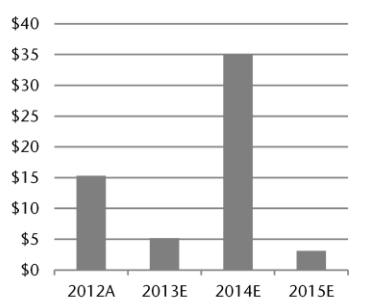
- Positive outcome in GMI-1070's PIII study for sickle cell pain crisis, with US approval in H2 2017 and EU approval in early 2018
- GMI-1070 expected to have peak sales of \$563M (risk-adjusted) in US and \$74M (risk-adjusted) in EU.
- U.S. regulatory approval of GMI-1271 in AML in 2020 with peak sales of \$78M (risk-adjusted)
- DCF-based PT: \$13

Upside Scenario

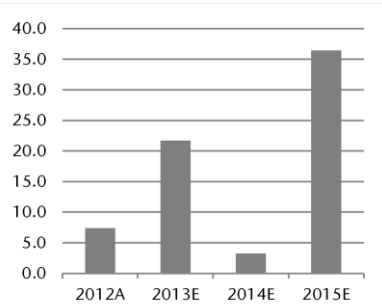
- Positive expansion of GMI-1271 and GMI-1070 into other blood disorders.
- DCF-based PT: \$24

Downside Scenario

- Negative outcome in GMI-1070 PIII study, DCF-based PT: \$2
- Negative outcome in GMI-1271 clinical program, DCF-based PT: \$6

Long Term Analysis**Revenue (millions)**

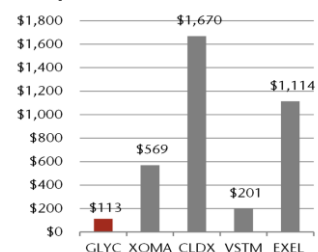
Source: Company data; Jefferies estimates

Enterprise Value (EV)/Sales

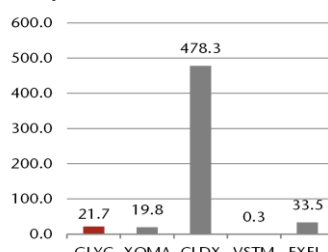
Source: Company data; Jefferies estimates

Other Considerations

We consider small-cap and mid-cap biotech companies with late-stage programs to continue to be attractive targets for partnering or M&A partnering with large-cap biotech and pharma companies, which we believe will be a driving factor for performance in the biotech sector 2013-2014.

Peer Group**Group EV**

Source: Factset, Jefferies estimates

Group EV/2013E Sales

Source: Factset, Jefferies estimates

Recommendation / Price Target

Ticker	Rec.	PT
GLYC	Buy	\$13
XOMA	Buy	\$11
CLDX	Buy	\$29
VSTM	Buy	\$21
EXEL	Hold	\$5

Catalysts

- Initiation of GMI-1070 PIII in H1 2014
- IND filing for GMI-1271 in Q1' 14 and initiation of PI in AML in H1' 2014
- Preliminary data from GMI-1271 PI in AML in H2 2014
- Initiation of Plb/II of GMI-1271 in AML in H2 2014

Company Description

GlycoMimetics is a biotechnology company that is developing inhibitors that target the complex carbohydrates involved in multiple human diseases. GlycoMimetics develops its inhibitors by mimicking the interaction of these complex carbohydrates with proteins. GlycoMimetics's lead product is GMI-1070, which is a pan-selectin inhibitor that will soon undergo critical phase III evaluation in sickle cell pain crisis. GlycoMimetics's pipeline also includes GMI-1271; an E-selectin inhibitor targeting acute myeloid leukemia, GMI-1050; a lectin inhibitor targeting pseudomonas infections and a dual E-selectin & CXCR4 antagonist targeting multiple cancers.

Executive Summary

GlycoMimetics is a biotech company founded in 2003 and based in Gaithersburg, MD. The company focuses on the discovery and development of glycomimetic therapeutics to address potential unmet medical needs. The company has partnered with Pfizer (PFE, \$30.60, Hold) for its lead pipeline program, GMI-1070. GMI-1070, a potential treatment for vaso-occlusive crises in sickle cell disease patients, will enter Phase III trial in Q3'14. Based on the Phase II data, we believe GMI-1070 has 75% odds of success. Furthermore, our statistical analysis suggests the Phase III could be adequately powered to achieve a $p < 0.05$ if the trial is sized between 300-400 patients. Upon initiation of the Phase III, GLYC will receive a \$35M milestone from Pfizer. We estimate the trial could complete in H2 2016 with potential U.S market entry in late 2017. Furthermore, GLYC will embark in a Phase I trial testing GMI-1271, its E-selectin inhibitor, in a Phase I standard 3+3 dose-escalation trial in haematological malignancies in Q2 '14 with preliminary data expected by YE '14, and with a more expanded trial potentially in AML in 1H '15. GLYC has other preclinical programs targeting selectin inhibition for potentially other indications including prophylaxis treatment of vaso-occlusive crises, other agents targeting oncology indications, and treatments for *Pseudomonas* respiratory infections. While the focus of the company is on moving GMI-1271 through clinical development, these other preclinical programs may augment the clinical pipeline longer-term, and represent upside to our estimates.

- **GMI-1070 Could Be The First Novel Therapeutic For Treatment of Vaso-Occlusive Crises.** We believe promising Phase II data for GMI-1070 as a treatment for resolution of vaso-occlusive crises (VOC) in hospitalized sickle cell disease patients supports a favorable outlook for the Phase III trial which should commence in Q3 '14 with data available in 2H '16 and approval/launch in late '17. While the Phase III trial design has not been disclosed, our analysis estimates the trial could be sized between 300-400 patients to achieve statistical significance. The Phase II data reports patients treated with GMI-1070 showed a mean reduction of 40 hours vs. placebo in time to resolution of VOC. The Phase II trial also reported a mean reduction of 50 hours in the time to hospital discharge, and time to transition off IV analgesics by 45 hours. The cumulative amount of opioid analgesic administered during hospitalization was also reduced by 80%. Furthermore, the trial enrolled patients between 12-60 years of age, and showed consistent efficacy regardless of the age group, and this clearly differentiates GMI-1070 from a competitive program (MST-188) which has shown through age subgroup analysis that its efficacy is primarily in those younger than 18 years. The difference in the efficacy between the treatment arm and placebo arm was maintained when patients were stratified by age groups, and gives us additional confidence in the PII data especially as it compares to prior trials with other treatments where at least one other treatment (MST-188) has observed activity in patients who are 18 years and younger.
- **GMI-1271 Represents Significant Upside.** While we attribute modest value for GMI-1271, we believe as the program delivers clinical data could drive GLYC shares meaningfully higher in the next 12-18 months. The company plans to initiate Phase I 3+3 dose-escalation trial in Q2 '14 with preliminary data available in late 2014. Based on preclinical data generated, it appears GMI-1271 reduces tumor volume when combined with Ara-C (chemotherapy) vs Ara-C alone in animal models of AML. Mouse xenograft models treated with GMI-1271 plus chemotherapy was able to

reduce the tumor burden by 50% and 67% in bone marrow and spleen respectively vs. chemotherapy.

Valuation

We arrive at our \$13 PT based on a DCF valuation model which assumes a WACC of 12%, terminal growth rate of 0% and outstanding shares of 18.6 million, driven by potential U.S. sales of GMI-1070 and GMI-1271. We estimate unadjusted peak sales of \$1.4B (\$151.7M adjusted), and is derived from GMI-1070 (\$637M unadjusted; 77M risk-adjusted), and GMI-1271 (\$752 M unadjusted; 75M risk-adjusted). We estimate U.S. approval and launch of GMI-1070 in H2 2017 and early 2020 for GMI-1271. We believe GMI-1070 to be approved in EU in early 2018. We have not have included EU sales of GMI-1271 in our model.

Exhibit 1: DCF sensitivity analysis

Discount rate	Price/Share
8.0%	\$17.88
10.0%	\$15.21
12.0%	\$13.11
14.0%	\$11.44
16.0%	\$10.11

Source: Jefferies estimates

Risks

Clinical Failure: As with all companies in biotechnology and pharmaceuticals developing treatments of the future, a clinical failure can lead to delays in approval or possibly discontinuation of programs.

Regulatory Failure: The FDA could determine the new drug application is inadequate for GMI1070 and GMI-1271 and could delay approval. Any delays in approval timelines could impact our earnings estimates, price target, and/or rating.

Commercial Failure: We currently project \$152 million (risk-adjusted) in U.S. sales for GMI-1070 and GMI-1271. Our estimates may rely on the success of the company/partners to receive drug reimbursement from private/public payors.

Financing Risks: We expect the company to have adequate cash to 1H 2016. It may need additional financing(s) in 2016 and beyond to fund a potential U.S. launch of GMI-1070 and GMI-1271, and to fund R&D in additional indications.

Company Overview

GlycoMimetics is a biotechnology company that is developing inhibitors that target the complex carbohydrates involved in multiple human diseases. GlycoMimetics develops its inhibitors by mimicking the interaction of these complex carbohydrates interactions with proteins. GlycoMimetics' lead product is GMI-1070, which is a pan-selectin inhibitor that will soon undergo critical Phase III evaluation in sickle cell pain crisis. GlycoMimetics' pipeline also includes GMI-1271; an E-selectin inhibitor targeting acute myeloid leukemia, GMI-1050; a lectin inhibitor targeting pseudomonas infections, and a dual E-selectin & CXCR4 antagonist targeting multiple cancers.

Exhibit 2: GlycoMimetics' pipeline

Product	Mechanism of Action	Indication	Development Phase
GMI-1070	Pan selectin inhibitor	Sickle cell pain crisis (VOC)	Phase II
GMI-1271	E-selectin inhibitor	Acute myeloid leukemia	Preclinical
Dual E-selectin & CXCR4 antagonist	E-selectin & CXCR4 inhibitor	Multiple cancers	Preclinical
GMI-1050	Lectin inhibitor	Pseudomonas infection	Preclinical

Source: Company reports.

Exhibit 3: Key Upcoming Milestones

Product	Indication	Event	Date
GMI-1070	Sickle cell pain crisis	End of PII meeting with FDA and development of PIII design	Q1 2014
		Initiation of PIII trial for vaso occlusion pain crisis in sickle cell disease	H1 2014
		Completion of enrollment for PIII trial	H1 2016
		Data readout from the PIII study	H2 2016
		US approval	H2 2017
		EU approval	H1 2018
GMI-1271	Acute myeloid leukemia	IND filing	Q1 2014
		Initiation of Phase I	H1 2014
		Phase I data read-out from healthy volunteers	H2 2014
		Initiation of PIIb/II	YE 2014

Source: Company estimates, Jefferies.

GMI-1070

Alleviating the Sickle Cell Pain Crisis

GLYC's lead candidate GMI-1070 is a novel synthetic pan-selectin inhibitor with inhibitory activity targeting all three selectins (E-selectin, L-selectin and P-selectin), and is about to initiate Phase III trials in Q3 '14 as a potential treatment of vaso-occlusive crises (VOC) in sickle cell disease (SCD) patients. Selectins are glycoprotein cell adhesion molecules implicated in inflammatory processes responsible for VOC. Therefore, targeting selectins in inflammatory responses may represent a novel approach and potentially addressing a significant unmet medical need. Under GLYC's licensing agreement with Pfizer (PFE, \$30.60, Hold), GMI-1070's Phase III development and worldwide commercialization will be conducted by Pfizer and GlycoMimetics will receive milestone payments and royalties upon marketing approval from the regulatory authorities. GMI-1070 has received both orphan drug and fast track status for VOC from the FDA. GMI-1070 has been granted orphan drug status by European Medical Agency (EMA).

We estimate ~75% odds of a positive outcome for GMI-1070 in resolution of VOC in SCD and estimate GMI-1070 could launch in late 2017. We assume ~\$2,500 per treatment per day for ~4 days in the hospital, totalling ~\$10,000 per patient. We believe it can achieve peak market penetration of ~36% by 2030, driving ~\$751 million by 2030 (unadjusted). In the U.S., on a risk-adjusted basis using a 25% discount rate to account for the potential risk associated with the Phase III trial, we estimate peak sales of \$530 million. We estimate GLYC would receive \$68M in peak royalties in the U.S.

U.S. Market Opportunity

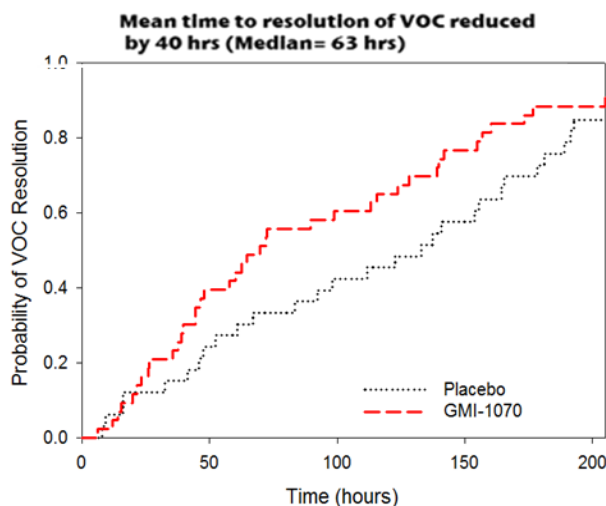
Sickle Cell Anemia Pain Market. Sickle cell disease is a blood disorder that affects 70,000-80,000 people in the U.S. (Source: Genetics Home Reference). The prevalence tends to be higher in African-Americans (1 in 600) and Hispanics than Caucasians. GMI-1070 will target SCD patients who are hospitalized as a result of sharp, intensive pain caused by a VOC crisis. The average rate of VOC crises is 0.8 episodes per patient-year in sickle cell anemia, 0.4 episodes per patient-year in sickle haemoglobin disease and sickle β^+ -thalassemia, and 1.0 episode per patient-year in sickle β^0 -thalassemia (Platt, O. S. et al., *NEJM* 1991, 325(1), 11). However, the rate within these subgroups varies widely — 5.2% of patients with 3-10 crises events per year accounted for 32.9% of all VOC crises. The genotype prevalence is broken down according to the following: ~65% for sickle cell anemia (SS), ~25% for sickle hemoglobin C disease (SC), ~8% for sickle β^+ -thalassemia, and 2% for sickle β^0 -thalassemia. Based on the prevalence of each genotype, we estimate ~41,600 pain episodes per year for sickle cell anemia, ~8,000 episodes per year for sickle haemoglobin disease, ~2,560 episodes per year for sickle β^+ -thalassemia, and 1,600 episodes per year for sickle β^0 -thalassemia. Altogether, we estimate a total of ~53,760 VOC crises events occur in the U.S., or approximately ~67% of the total SCD prevalence. Also, up to a third of all patients hospitalized for VOC crises are at risk of re-hospitalization within 30 days from hospital discharge.

Phase II De-Risks GMI-1070 Phase III Trial

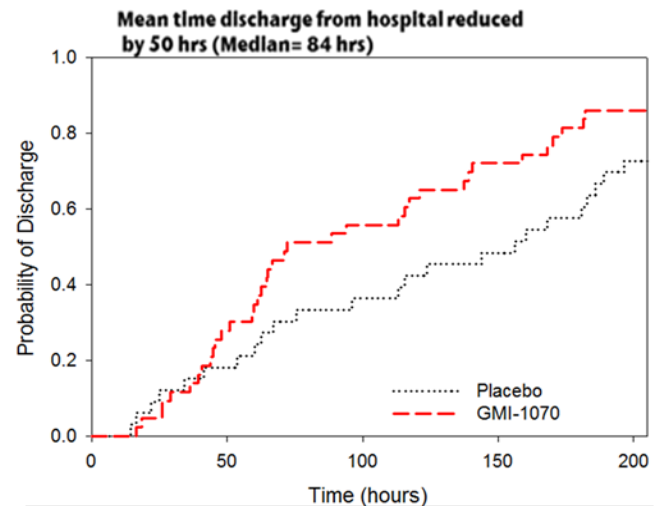
Mean time to Resolution of VOC Reduced by 40hrs in PII with Favourable Safety Profile: In April 2013, GLYC reported top line data from the placebo-controlled PII trial testing GMI-1070 in VOC in SCD. In the 76 patient randomized, double-blind, placebo-controlled phase II clinical trial evaluating the safety, efficacy and pharmacokinetics of GMI-1070, patients treated with GMI-1070 showed a mean reduction of 40 hours vs. placebo (median=63 hours; $p=0.192$) in time to resolution of VOC. In the primary efficacy analysis, GMI-1070 showed a mean time to VOC resolution of 103.6 h + 20.9 (standard error), whereas placebo showed a mean time to VOC

resolution of 144 h + 23.5 (standard error). The Phase II trial also reported a mean reduction of 50 hours in the time to hospital discharge (median = 84 hours; $p = 0.092$), and time to transition off IV analgesics by 45 hours (median=76 hours; $p=0.089$). The cumulative amount of opioid analgesic administered during hospitalization was also reduced by 80%. In the PII trial, 43 patients were administered GMI-1070 and 33 patients were given placebo. 20/76 pts were pediatric (13 GMI-1070 arm; 7 placebo arm). The difference in the efficacy between the treatment arm and placebo arm was maintained when patients were stratified by age groups, and gives us additional confidence in the PII data especially as it compares to prior trials with other treatments where at least one other treatment (MST-188) has observed activity in patients who are 18 years and younger.

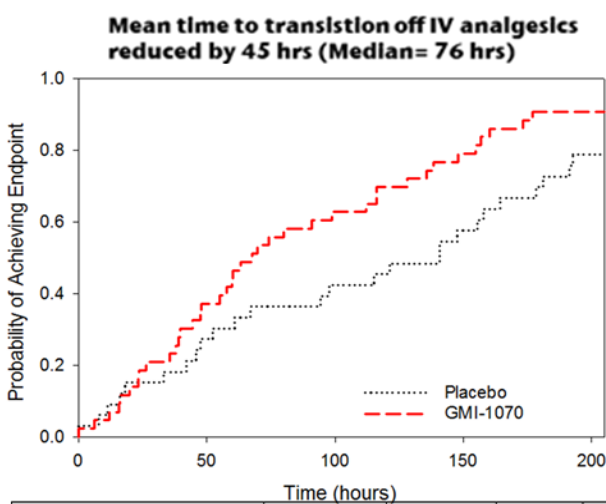
Exhibit 4: Summary of multiple measure of VOC experience observed in GMI-1070 PII trial



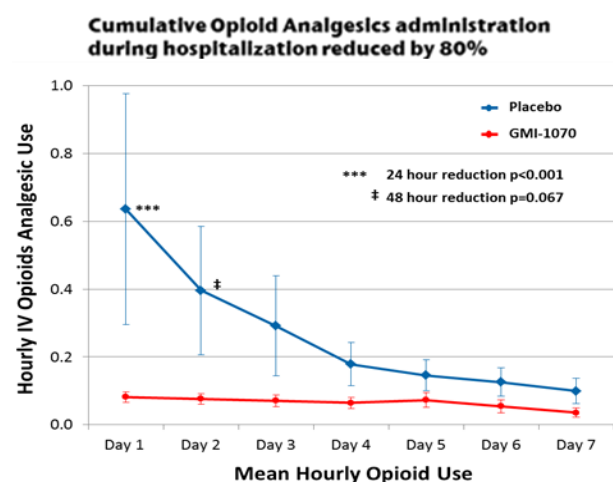
VOC resolution	GMI-1070	Placebo	Reduction	P-value
Mean \pm SE (hrs)	103.6 \pm 20.9	144.6 \pm 23.5	28%	0.19
median (95% CI), (hrs)	69.6 (44.3, 115.5)	132.9 (67.0, 164.2)	48%	0.19



Time to discharge from hospital	GMI-1070	Placebo	Reduction	P-value
Mean \pm SE (hrs)	118.8 \pm 21.7	173.5 \pm 24.5	32%	0.1
median (95% CI), (hrs)	72.2 (59.9, 121.0)	156.1 (75.4, 185.8)	54%	0.09



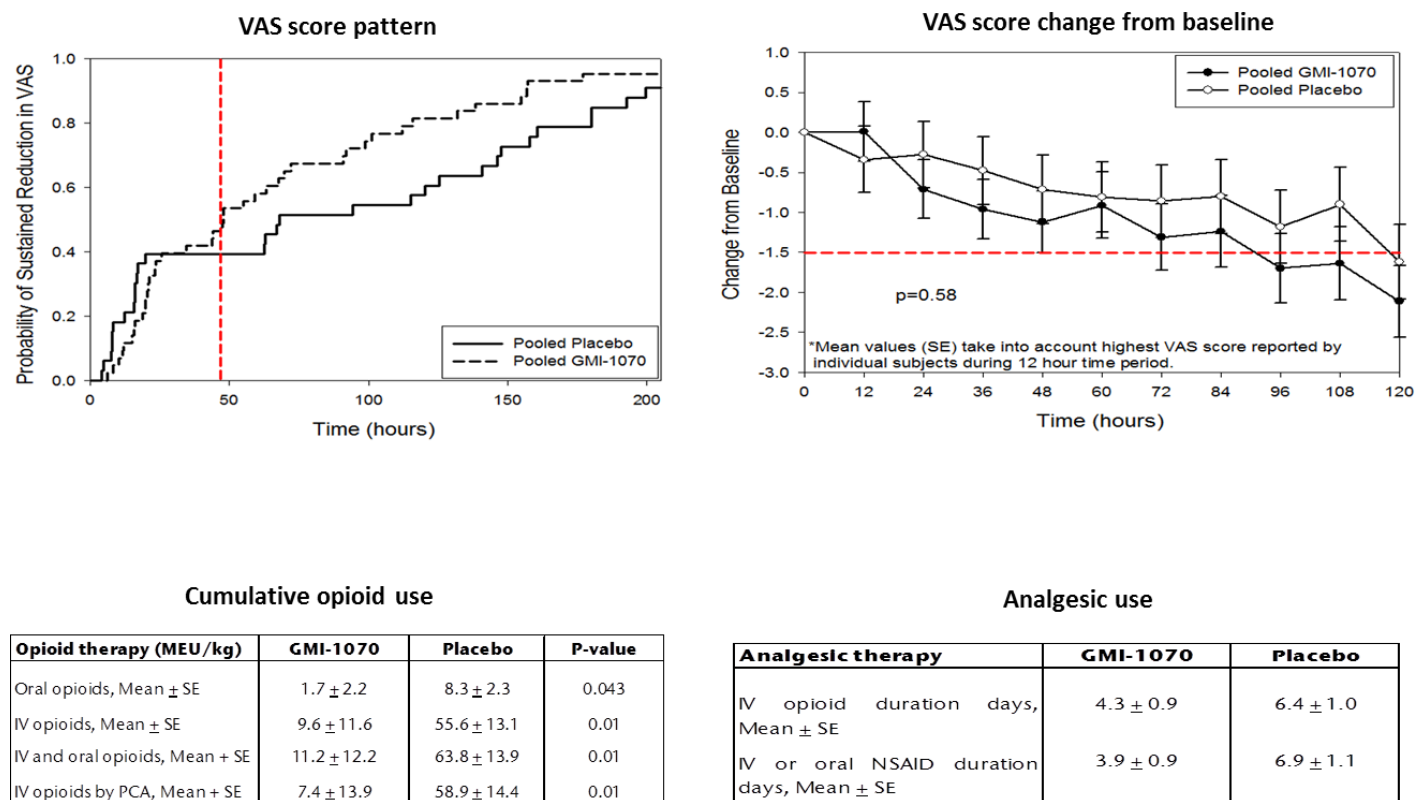
Transition of IV analgesics	GMI-1070	Placebo	Reduction	P-value
Mean \pm SE (hrs)	87.8 \pm 15.9	135.2 \pm 17.5	35%	0.05
median (95% CI), (hrs)	128.0 (57.7, 156.9)	181.0 (97.7, 217.0)	29%	0.2



Source: Telen M, et al, ASH 2013, oral session, abstract 776.

The trial was designed to show a 40% mean reduction in the time to resolution of VOC, however a 28% mean reduction in the time to resolution of VOC was observed with no statistical significance. VOC resolution outcome was defined as a reduction in pain measured on VAS pain scale (1-10; 1 no pain, 10- highest) and time to transition of IV analgesics. The mean VAS pain score at first evaluation was 8.3 (± 1.6) and 9.0 (± 1.6) for patients randomised to GMI-1070 and placebo, respectively. At the time of discharge from hospital the mean VAS pain score was 3.0 (± 2.8) and 4.0 (± 3.0) for patients treated with GMI-1070 and placebo respectively. Compared to placebo, the GMI-1070 treated patients had quicker reduction in VAS score starting 48 hours from study start while pain reduction of ~1.5cm or greater was more rapid within the first 100 hours of study start. There was significant reduction in the opioid use (includes oral, IV and patient controlled analgesic route) within the first 24 hours of study start and also during the complete hospitalization duration. The cumulative use of oral, IV, both (IV and oral) and PCA opioids was 1.7, 9.6, 11.2 and 7.4 MEU/kg, respectively for GMI-1070 treated patients v. 8.3, 55.6, 63.8, and 58.9 MEU/kg, respectively for placebo. There was also reduction of 2-3 days in the use of analgesic depending on the type of analgesic used in the GMI-1070 treated patients v. placebo.

Exhibit 5: Summary of multiple measures of pain and opioid use patterns observed in GMI-1070 PII trial



Source: Telen M, et al, ASH 2013, oral session, abstract 776.

Though the primary and secondary endpoint didn't meet statistical significance ($P=0.192$), the reduction in the VOC resolution and secondary end points was clinically relevant and bodes well for the Phase III design. We believe that a small sample size and large variations (standard deviation) observed in the time to resolution of VOC were reasons for not achieving a statistical significance in the PII trial. The standard deviation

observed in both treatment and placebo arm was ~135h (95%-130% variability). In addition, we believe that higher variability in the treatment arm could be a result of two different dosages tested in the trial. Earlier clinical studies have reported huge variations (standard deviations) in smaller SCD trials. In a Phase III clinical trial using poloxamer 188 to treat VOC in SCD, the trial showed a variation of ~60% in both the treatment and control arms and failed to achieve statistical significance due to less than adequate sample size. Drawing on the experience of drugs tested in earlier SCD clinical trials and the GMI-1070 PII trial, we believe a sufficiently powered design for PIII design would yield statistical significance if GMI-1070 observes similar efficacy v. a placebo control as reported in the PII trial.

There were no dose limiting toxicities in the PII trial. There were 28 events of serious adverse events (n = 23). The AEs and rehospitalisation rates were similar across treatment groups. There was no change in leucocytosis between the two treatment groups. The most common AE observed in both treatment arm and placebo arm were gastrointestinal disorders (40%), and pyrexia (18%). Exanthematous pustulosis was observed in one patient from the treatment group after discharge which resolved without any intervention.

Exhibit 6: Summary of AEs observed in GMI-1070 PII trial

Treatment related AEs

Treatment group	Gastrointestinal disorders	Rash	Hepatobiliary	Renal/ Urinary	Pyrexia	Headache
GMI-1070 (N=43), %	42%	14%	5%	7%	19%	19%
Placebo (N=33), %	36%	6%	6%	6%	18%	12%

Sickle cell disease related AEs

Treatment group	Acute Chest Syndrome	RBC transfusion	ICU stay	Death	Readmission VOC (14 days)	Readmission VOC (30 days)
GMI-1070 (N=43), %	14%	35%	0%	0%	9%	21%
Placebo (N=33), %	9%	52%	3%	0%	9%	21%

Source: Telen M, et al, ASH 2013, oral session, abstract 776.

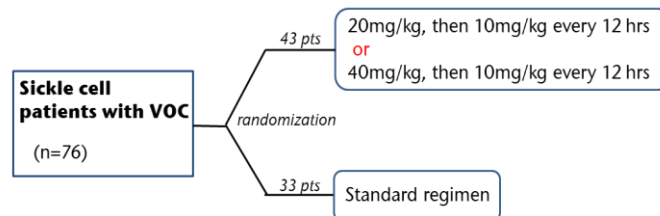
Patient baseline characteristics: The Phase II trial enrolled a total of 76 patients of which 43 were randomised to GMI-1070 arm and 33 to placebo arm. The median age was 25 years with an equal split across both genders. Both arms enrolled SCD patients belonging to the homozygous HbSS subset (91%). When comparing the baseline characteristics, more number of patients in the placebo arm reported daily outpatient meds (58%), ≥ 3 VOC admissions (42%) and ACS (18%) in previous 12 months v. 42% with daily outpatient med, 30% with ≥ 3 VOC admissions and 12% with ACS in the GMI-1070 arm. However, these imbalances were not significant to influence trial outcomes, in our view.

Exhibit 7: Patient baseline characteristics in GMI-1070 PII study

Characteristics	GMI-1070	Placebo
<i>N</i>	43	33
Gender		
Male	42%	39%
Female	58%	61%
Age, mean (SD)	25.4 (10.8)	25.0 (10.20)
Patients receiving hydrourea	51%	70%
Genotype		
HbS S	91%	91%
Hb S ⁰ thalassemia	2%	9%
HbS C	7%	0%
Baseline data		
Daily out-patient meds (% n)	42%	58%
≥3 VOC admissions in previous 12 months (% n)	30%	42%
Acute chest syndrome in previous 12 months (% n)	12%	18%
Visual analog scale (VAS) at presentation, mean (SD)	8.3 (1.6)	9.0 (1.5)
Hemoglobin g/dL, mean (SD)	8.3 (1.4)	8.2 (2.1)
WBC X 10 ³ , mean (SD)	12.8 (5.0)	13.6 (5.6)
Absolute neutrophil count (ANC) X 10 ³ , mean (SD)	7.3 (3.9)	8.3 (5.1)

Source: Telen M, et al, ASH 2013, oral session, abstract 776.

Phase II Trial Design: The PII trial tested the efficacy and safety of GMI-1070 in a randomized, double-blind, placebo-controlled Phase II trial in patients with sickle cell pain crisis. The study enrolled 76 patients (ages 12-60) who have been hospitalized for vaso-occlusive crisis across 22 clinical sites in U.S. and Canada. Twenty of the patients were pediatric. Of the 76 patients, 43 patients were randomized to the GMI-1070 arm and 33 patients into the placebo arm. The trial compared two different doses (low and high dose) of GMI-1070 given intravenously twice daily during the hospital stay for sickle cell pain crisis versus placebo. The low dose patients were given a loading dose of 20 mg/kg followed by maintenance dose of 10mg/kg of GMI-1070 every 12 hours, whereas the high dose patients were given 40mg/kg followed by 20mg/kg GMI-1070 every 12 hours. The primary endpoint was reduction in time to resolution of vaso-occlusive crisis (up to 7 days), and included pain score, feeling ready to leave the hospital, and actual time of leaving the hospital. Pain score was categorised on VAS pain scale of 1-10 and the patients in the GMI-1070 should show a sustained reduction of VAS ≥1.5 cm v. placebo to achieve statistical significance. Secondary endpoints include safety, pharmacokinetics (PK), and markers of inflammation and cell stickiness in the blood up through 28 days post-last dose. The trial was powered to show 40% reduction in the time to resolution of a VOC crisis.

Exhibit 8: Summary of GMI-1070 Phase II design**Endpoints****Primary:**

- Reduction in vaso occlusive crisis
- Reduction in pain score
- Readiness to leave the hospital
- Actual time of hospital discharge

Secondary:

- Pharmacokinetics
- Safety and tolerability

Inclusion Criteria:

- 12-60 yrs
- Confirmed sickle cell disease with VOC and hospitalization
- Able to receive drug within 24 hrs post medical evaluation

Exclusion Criteria:

- Fever >39°C
- Acute chest syndrome

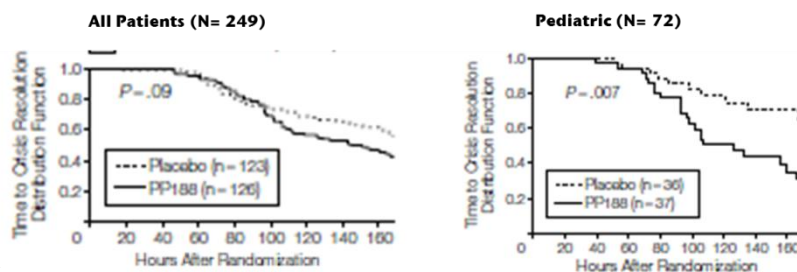
Source: www.clinicaltrials.gov

GMI-1070 Phase III Trial Design Could Draw Upon Previous SCD Clinical Trial Experiences.

MST-188's EPIC PIII Is An Ideal Proxy: Mast Therapeutics' (MSTX, Not Covered, \$0.83) EPIC trial is a randomized, double-blind, two-arm, placebo-controlled PIII trial that is evaluating the efficacy of MST-188 (formerly called p188 or poloxamer) in SCD pain crisis. It will enroll a total of 388 pediatric patients (age: 8-17) who have SCD and are experiencing acute pain typical of vaso-occlusive crisis, and who require parenteral opioid analgesic treatment. Patients are equally randomized to MST-188 or placebo. The trial is 90% powered with an alpha of 0.05 to detect a 16 hour difference (20% reduction) between the two arms. The design accounts for a standard deviation of 50% in individual responses in both arms. The EPIC trial design was formulated based on a failed PIII study using Poloxamer-188 (unpurified MST-188) in which the patients showed a mean reduction of 8 hours between the treatment and placebo arm in resolution of VOC with a standard deviation of 60%. The failed clinical trial involved both adult and pediatric patients, where the adult patients showed no benefit, but pediatric patients on MST-188 observed a mean reduction of 22 hours v. placebo. The failed PIII trial was 80% powered with 0.05 alpha to detect a 26% reduction. In the EPIC trial, MSTX has taken a conservative estimate of 16 hours as treatment difference between the active and placebo arm and initially was targeting pediatric patients for the study. In order to address the complications of subjective element of endpoints studied in the failed MST-188 PIII study, MSTX has modified its primary endpoint analysis and will now measure the reduction in duration of VOC as primary endpoint (from start of the treatment after hospitalization to discharge). The failed PIII trial utilized pain relief (pain scores ≤ 40 maintained during 2 consecutive readings obtained 4 hrs apart v. placebo; Scale: 1-100: 1-no pain, 100-highest), freedom from analgesic use, ability to walk and patients belief of relief from pain episodes as co-primary endpoints. The EPIC trial is using the same dosage as the failed Poloxamer-188 PIII trial, which is continuous IV infusion of MST-188 at 100 mg/kg for 1 hour followed by 30 mg/kg/hour for up to 48 hours. The average duration of VOC crisis for placebo group is 96 hours and the drop-out rate expected is 3%. Therefore, we believe that this trial design template of EPIC trial could be an ideal proxy template for phase III design of GMI-1070.

Exhibit 9: Patient characteristics and results of failed Poloxamer 188 (MST-188) Phase III study

Characteristics	Poloxamer 188 (MST-188)	Placebo
N	126	123
Gender		
Male	41%	41%
Female	59%	59%
Race		
African-American	98%	98%
Other	2%	2%
Age, mean (SD)	21.1 (9.0)	20.9 (9.0)
Weight, mean (SD)	58.3 (17.0)	58.7 (16.9)
Patients receiving hydrourea	21%	23%
Genotype		
SS/S β^0 thalassemia	77%	79%
Sickle cell disease	14%	14%
S β^+ thalassemia	9%	7%
Baseline data		
Time from onset of crisis to randomization (days), mean (SD)	2.3 (2.1)	1.9 (1.8)
Time from admission to randomization (hrs.), mean (SD)	8.84 (5.7)	8.4 (5.0)
Time from randomization to drug infusion (hrs.), mean (SD)	2.2 (1.3)	2.3 (1.5)
Length of study drug infusion (hrs.), mean (SD)	47.8 (6.1)	47.3 (6.7)
No. of pain locations, mean (SD)	3.8 (1.8)	3.8 (1.8)
Pain intensity (range 1-100), mean (SD)	73.4 (17.8)	73.8 (20.8)



Source: Orringer E, et al, JAMA, 2001, 286, 2099.

GMI-1070 PIII May Enroll ~300-400 patients: We believe Pfizer will use a 90% power (β) with alpha (α) of 0.05 to show reduction in duration of VOC crisis by 20% (treatment difference = 30 hours) with a standard deviation of 90%. Though the PII trial observed a reduction in VOC crisis duration by 28% (40 hours), we believe in a larger clinical trial there will be a regression to the mean and a conservative 20% reduction could provide increased odds of success in achieving statistical significance and at the same time would be a clinically relevant outcome to drive market adoption. The regression to the mean due to larger population will also reduce the variability or standard deviation and therefore we estimate a 90% standard deviation which is conservative to MST-188's EPIC trial which assumes a 50% standard deviation. We also take a conservative drop-out rate of 5% v. 3% for EPIC trial. Based on the above parameters mentioned we expect the PIII trial of the GMI-10170 to enroll somewhere between 300-400 pts. GLYC management has suggested it could take up to 2 years to complete enrollment for the Phase III and may use a fixed dose treatment regimen vs. a weight based dose used in the Phase II trial. The Phase III will have similar primary end points to the Phase II trial.

Exhibit 10: Possible patient numbers required for GMI-1070 Phase III study

Assumptions	GMI-1070 PIII	MST-188 (EPIC trial)
Treatment mean difference (hrs)	30	16
Reduction in mean VOC (%)	20	20
Drop-out rate (%)	5	3
Standard deviation (%)	80	50
Statistical power (β)(%)	90	90
Statistical significance (two sided- α)	0.05	0.05

		Treatment difference (mean hrs)						
		15	16	20	25	30	35	40
Standard Deviation (%)	45	398	350	224	143	100	73	56
	48	453	398	255	163	113	83	64
	50	492	432	277	177	123	90	69
	55	595	523	335	214	149	109	84
	60	708	622	398	255	177	130	100
	65	831	730	467	299	208	153	117
	70	964	847	542	347	241	177	136
	75	1106	972	622	398	277	203	156
	80	1259	1106	708	453	315	231	177
	85	1421	1249	799	512	355	261	200
	90	1593	1400	896	574	398	293	224

Source: JEF Est.

Phase II clinical data validates the efficacy and safety of GMI-1070 in SCD

Pfizer Agreement. In 2011, GlycoMimetics entered into a worldwide license agreement with Pfizer for the Phase III clinical development, approval and potential commercialization of GMI-1070. GLYC received an upfront payment of \$22.5M and can receive up to \$320M in development, regulatory and commercial milestone payments. Upon approval, GLYC will receive tiered royalties which can range from low double digits to low teens depending on the net sales of GMI-1070. Upon the initiation of PIII, GLYC will receive a milestone payment of \$35M. According to the agreement if PFE does initiate the GMI-1070 PIII trial by April 2014, it will have to make advance payment of \$15M against the first payment.

Other Selectin Inhibitors for VOC in Clinical Trials

SelG1. SelG1 from Selexys/Novartis pharmaceuticals (NOVN, CHF 71.40, Hold) is a P-selectin inhibitor that is undergoing phase II (SUSTAIN) trial in 174 adult and pediatric patients for treatment of VOC in SCD. The SUSTAIN trial is a randomized study and will evaluate high dose SelG1, low dose SelG1 or placebo in the presence or absence of hydroxyurea therapy. The study will administer the active dose and placebo once every 4 weeks intravenously through week 50 and examine the effectiveness of SelG1 in reducing the rate of sickle cell-related pain crisis in each active dose level v. placebo (www.clinicaltrials.gov). No details have been provided as to how resolution of pain crisis will be measured.

Vaso-Occlusive Crisis (VOC) in Sickle Cell Disease

Vaso-occlusive crisis is a common painful complication of SCD which occurs when the circulation of blood vessels is obstructed by sickled red blood cells, causing ischemic injuries. The most common complaint is of pain, and recurrent episodes may cause

irreversible organ damage. Vaso-occlusion may cause a variety of clinical complications as pain syndromes, stroke, leg ulcers, spontaneous abortion and renal insufficiency. Symptoms of sickle cell crisis include severe pain, anemia, chest pain and difficulty breathing, strokes, joint pain and arthritis, blockage of blood flow in spleen or liver and severe infections. Severe pain develops in the chest, back, arms, legs, and abdomen. Factors that can cause sickle cell crises include infections, low oxygen tension, concomitant medical conditions, dehydration, acidosis, extreme physical exercise, physical or psychological stress, alcohol, pregnancy and cold weather.

Pain – Acute or Chronic. SCD pain can either be acute or chronic, or some combination of the two. Acute pain of tissue infarction can be sudden and unpredictable in onset and intensity. Sickling deformity of red cells causes vaso-occlusion (blocking off of blood supply to tissue and organs as sickled cells stick together inside blood vessels) and tissue infarction. When attacks occur repeatedly, there can be long-term damage to organs, joints and bones.

Treatment: Treatment is limited to opioid pain medications, anti-inflammatory medications, and antibiotics for infection. Opioids are commonly prescribed to treat SCD pain, however the specific treatment can depend on whether the patient is opioid-naïve or -tolerant. Long-term opioid use, however, presents a problem of physiologic tolerance that ultimately reduces efficacy. Furthermore, opioid therapy can lead to dependence and addiction, and interruption of regular opioid use can lead to severe withdrawal symptoms.

Exhibit 11: Treatment of acute pain

Severity	Opioid-naïve	Opioid-tolerant
Mild/moderate	Dihydrocodeine tablets (30 mg/4h)	Immediate-release morphine sulphate (10-40 mg/4h)
	Co-codamol (2 tablets/4h)	Hydromorphone (1.3-3.9 mg/4h)
	Co-proxamol (2 tablets/4h)	
Severe	Diamorphine (2.5-5 mg/4h SC)	Diamorphine (10-20 mg/2-4h SC)

Source: Okpala, I. Management of Pain in Sickle-Cell Disease, *J. Royal Soc. Of Med.*, 2002, 95, 456.

Droxia (hydroxyurea) is the only FDA-approved medication to prevent painful episodes in sickle cell disease. Droxia decreases the frequency and severity of sickle cell crisis and reduces the number of blood transfusions and hospitalizations. It is recommended as first-line therapy to treat adults and adolescents with moderate-to-severe recurrent pain (~2-3 times a year). It is given as a single oral dose and is dependent on weight (starting dose is 15 mg/kg/d).

Droxia was investigated in a large clinical study (Multicenter Study of Hydroxyurea in Sickle Cell Anemia) of 299 adult patients (≥ 18 years) with moderate-to-severe disease (≥ 3 painful crises yearly). In this study, compared to placebo, Droxia was able to significantly reduce the annual rate of painful crises (2.5 v. 4.6, -46%, $p=0.001$), the annual rate of painful crises requiring hospitalization (1.0 v. 2.5, -60%, $p=0.0027$), the incidence of chest syndrome (56 v. 101, -45%, $p=0.003$), the number of patients transfused (55 v. 79, -30%, $p=0.002$), and the number of units of blood transfused (423 v. 670, -37%, $p=0.003$).

Treatment failure is a prevalent issue with Droxia due to a host of causes including non-compliance, or also due to decreased marrow reserve and genetic factors (Platt, O. S. et al, *N Engl J Med*, 2008, 358, 1362). Interestingly, a 2006 study suggested that ~70% of

eligible patients were non-compliant on Droxia with adverse events cited as the most likely factor (Lanzkron, S. et al. *Am J Hematol* 2006, 81, 927).

The most common adverse events were hematologic, with neutropenia and low reticulocyte and platelet levels occurring in nearly all patients. Hydroxyurea increases fetal hemoglobin (HbF) production and total hemoglobin concentration in the body. The precise mechanism by which Droxia exerts its effects is unknown. Various studies propose that hydroxyurea acts as a ribonucleotide reductase inhibitor which causes an immediate inhibition of DNA synthesis without interfering with the synthesis of ribonucleic acid or protein.

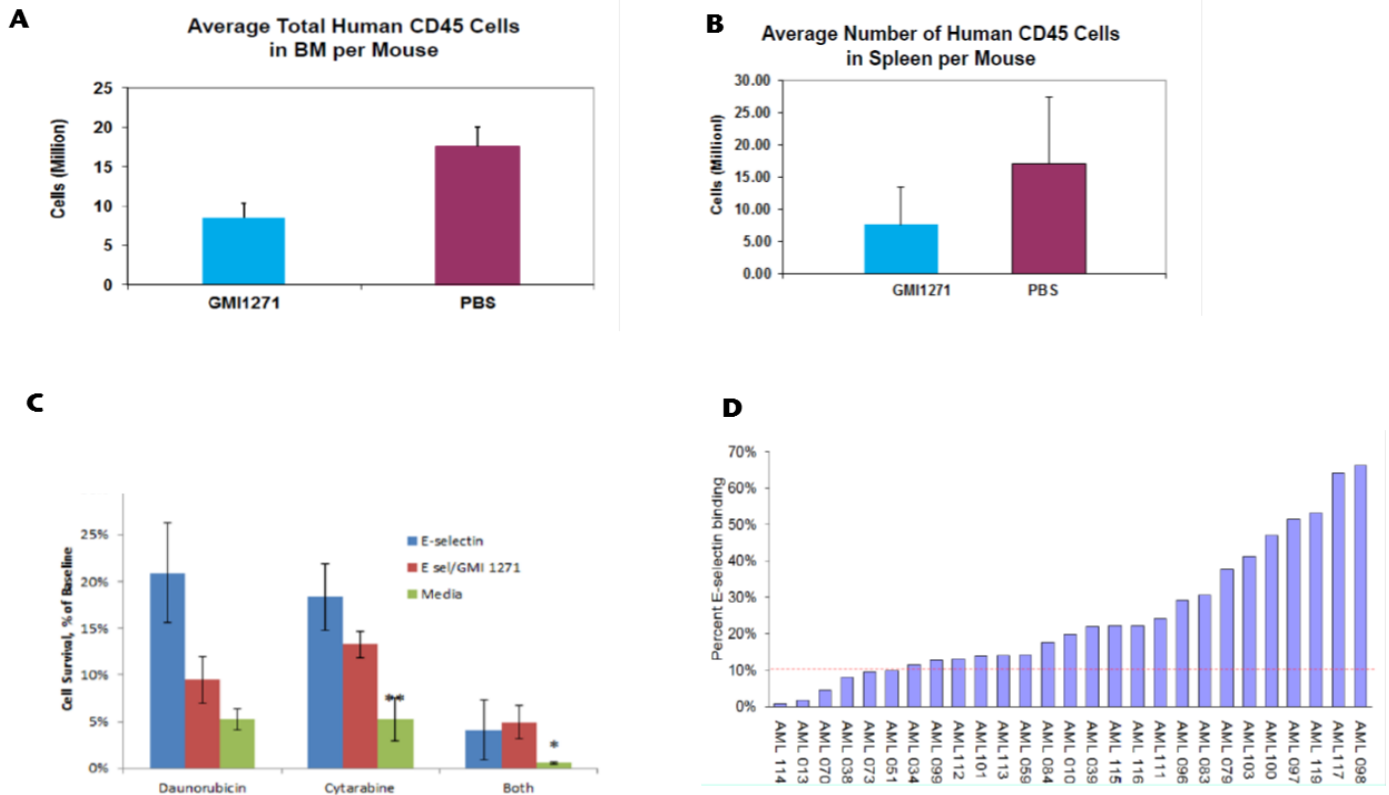
GMI-1271 Development Could Provide Significant Upside

GMI-1271 is an inhibitor of E-selectin molecule that is found to be overexpressed in both solid and hematopoietic malignancies including acute myeloid leukemia (AML). E-selectin has been suggested as one of the factors responsible for metastasis of cancers through hematogenous route. In xenograft models, GMI-1271 has shown encouraging efficacy in reducing the tumor burden in AML and GLYC is planning to file an IND in Q1 '14 and initiate a phase I trial in Q2 '14 with preliminary data by late 2014. Going forward, GLYC may expand GMI-1271 into multiple myeloma and other blood disorders.

If successful we estimate GMI-1271 could launch in the US in 2020. Due to its early stage of clinical development, we currently estimate peak sales of \$78M which is risk-adjusted using a 90% discount rate to account for potential development risk. We currently do not model EU sales for GMI-1271.

GMI-1271 Shows Encouraging Activity in Pre-Clinical Models.

GMI-1271 Blocks AML Blast Adhesion to E-Selectin and Reduces the Tumor Burden: Expression of E-selectins has been shown to enhance of survival of AML blasts and confer resistance to chemoradiation. Using selectin binding assays, GLYC has shown that 85% of human AML blasts express E-selectin. In preclinical studies using mouse xenograft models, GMI-1271 has observed reductions in the number of AML cells when treated with GMI-1271 plus chemotherapy compared to chemotherapy alone. Mouse xenograft models treated with GMI-127 plus chemotherapy was able to reduce the tumor burden by 50% and 67% in bone marrow and spleen respectively vs. chemotherapy. These studies were backed by in vitro results which showed E-selectin expressing human AML blasts when treated with GMI-1271 reduced the chemoresistance to danarubicin or cytarabine. Up-regulation of wnt and hedgehog pathways by E-selectin have been attributed to the chemo-resistance caused by E-selectin overexpression and blocking by GMI-1271 reduced the activity of wnt suggesting the role of E-selectin in wnt upregulation.

Exhibit 12: GMI-1271 reduces the AML tumor burden in pre-clinical models

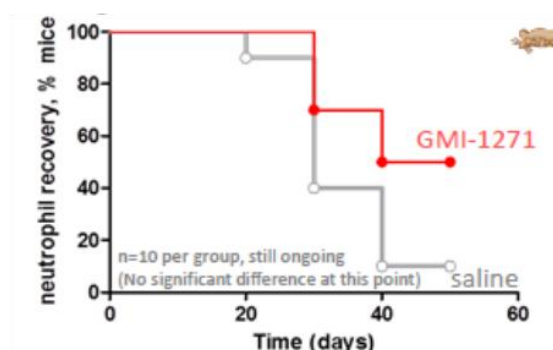
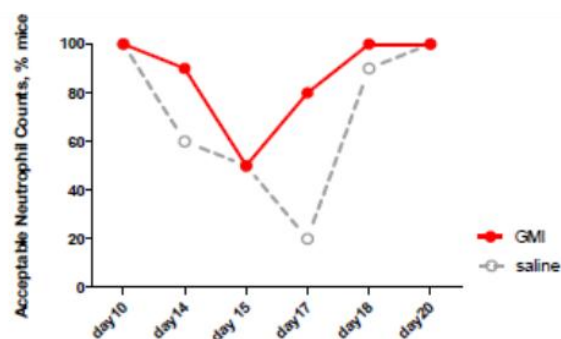
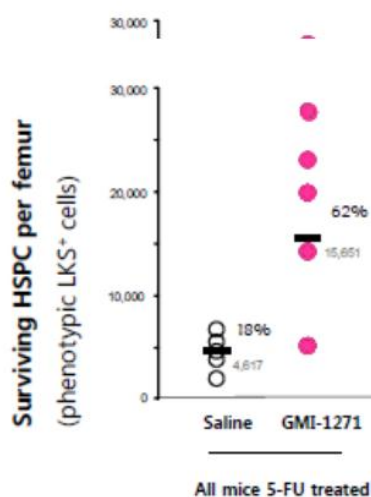
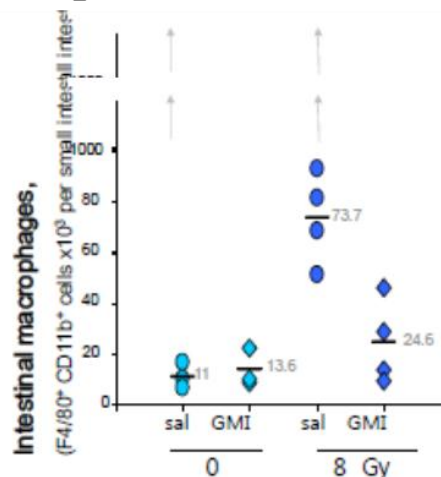
A & B: GMI-1271 reduces the tumor burden in mice treated with chemotherapy v. placebo

C: In Vitro studies show that GMI-1271 reduces the chemoresistance of AML cells

D: 85% of primary human AML blasts show expression of E-selectin

Source: Chien S, et al, ASH 2011, oral session, abstract 579

In addition, blocking of E-selectin through GMI-1271 showed recovery of mice from repeated doses of chemotherapy and increased its survival. Some of the severe side effects of chemotherapy is neutropenia (lack of enough neutrophils), and intestinal mucositis (damaged intestine) that leads to death of patients. Blocking of E-selectin by GMI-1271 in mouse models lead to quiescence of hemotopoietic stem cells upon chemoradiation and leading to quick neutrophil recovery once chemoradiation was completed, thereby increasing survival of GMI-1271 treated mice v. placebo. Similarly it has been shown that E-selectin is highly expressed in damaged intestine and blocking of E-selectin through GMI-1271 alleviates the intestinal mucositis by blocking the recruitment of inflammatory macrophages.

Exhibit 13: GMI-1271 helps in neutrophil recovery and reduces intestinal mucositis**A****B****C****D****A & B:** GMI-1271 helps in the neutrophil recovery and survival of mice**C:** GMI-1271 leads to more surviving hemopoietic stem cells**D:** GMI-1271 reduces the intestinal mucositis by reducing the recruitment of intestinal macrophages

Source: Ingrid W, et al, ASH 2013, poster session, abstract 2266.

Acute Myeloid Leukemia

Acute myeloid leukemia (AML) is the rapid growth of abnormal myeloid blast cells that accumulate in the bone marrow and interfere with the production of normal blood cells. AML is the most common acute leukemia affecting adults, and accounts for approximately 1.2% of cancer deaths in the US (Jemal A et al, Cancer j, 52, 23) and its incidence is expected to increase as the population ages. AML progresses rapidly and is typically fatal within weeks or months if left untreated. Symptoms of AML include replacement of normal bone marrow with leukemic cells, fatigue, and shortness of breath, easy bruising and bleeding, and increased risk of infection. AML has several subtypes; treatment and survival rate varies among subtypes. Five-year survival varies from 15–70%, and relapse rate varies from 33–78%, depending on subtype.

Treatment: AML is treated initially with chemotherapy aimed at inducing a remission; patients may go on to receive additional chemotherapy or a hematopoietic stem cell transplant. The treatment of first line AML includes induction chemotherapy followed by a consolidation therapy. In the first line AML, treatment of AML other than M3 subtype includes 7+3 regimen therapy which includes 7 consecutive days of infusion therapy with cytarabine while anthracycline is given for 3 continuous days. AML M3 subtype is treated with all-trans retinoic acid (ATRA). If the patient in the induction phase achieves remission in induction consolidation therapy with additional chemotherapy with or without radiation therapy or stem cell transplant is suggested. For patients who have failed on prior therapies and have relapsed or refractory AML, the most common treatment is stem cell transplant.

Exhibit 14: Treatment of AML

	AML subtypes	Treatment
1st line AML	AML other than M3 subtype	
	Induction phase	7 days IV of cytarabine and 3 days IV of anthracycline
	Consolidation	3-5 rounds of chemotherapy ± radiation or Bone marrow transplant
	AML M3 subtype	All-trans retinoic acid (ATRA)
Refractory/Relapsed AML	All subtypes	Bone marrow/Stem cell transplantation

Source: American Cancer Society

Other Pipeline Products

Dual E-Selectin and CXCR4 Antagonist: GlycoMimetic has developed two molecules, GMI-1257 and GMI-1215, that are dual inhibitors of E-selectin and CXCR4. E-selectin and CXCR4 overexpression has been shown to be responsible for progression and metastases of cancers. In preclinical models both these inhibitors have shown an IC50 range of 3.6-4.1 µM and 0.31-1.1µM for inhibition of E-selectin and CXCR4 respectively. In mouse AML models both these inhibitors along with chemotherapy have shown to eradicate or reduce the tumor burden increasing the survival of mice when compared to chemotherapy alone (Chien S, et al, ASH 2011, abstract 615). GLYC management has not suggested which molecule it wants to proceed further with for clinical evaluation in cancers.

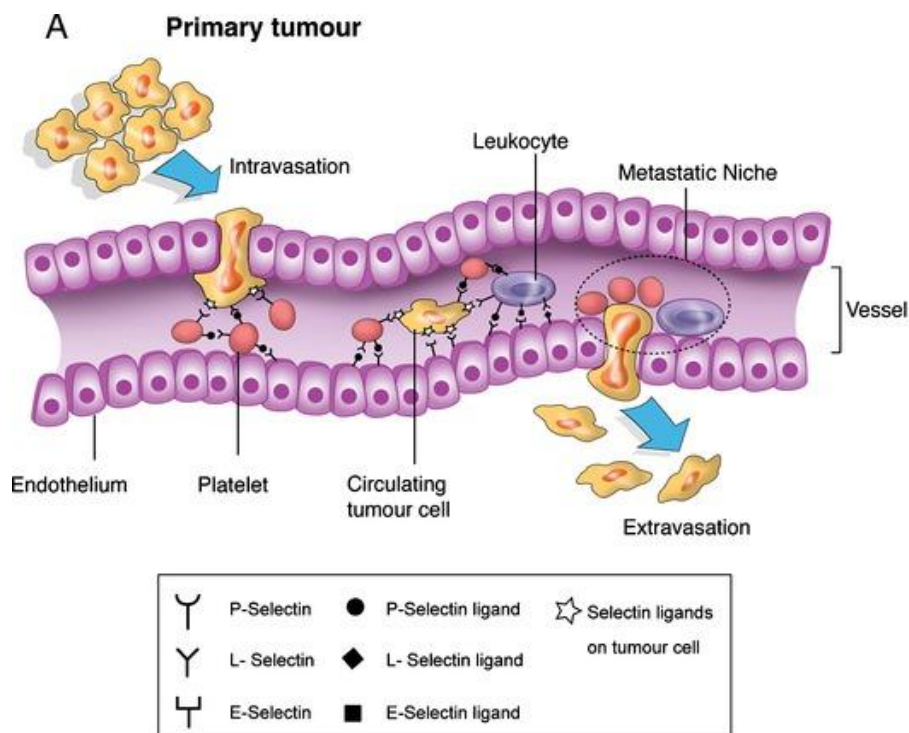
GMI-1050: GMI-1050 is an inhibitor of carbohydrate molecules called lectins for treatment of *Pseudomonas aeruginosa* respiratory infections. GMI-1050 is in early stage pre-clinical studies and inhibits the lectins PA-IL and PA-III, synthesised by *P. aeruginosa* to adhere to host respiratory epithelial cells. GLYC has suggested that GMI-1050 has shown encouraging efficacy in inhibiting the adhesion of *P. aeruginosa* to cell in both in vitro and in vivo preclinical assays.

Why Target Selectins?

Background: Selectins are a family of transmembrane molecules, expressed on the surface of leukocytes and activated endothelial cells. There are three known members in the selectin family; L, E, and P-selectin. All three selectins share similar cassette structure: N-terminal extracellular domain with structural homology to calcium-dependent lectins,

followed by a domain homologous to epidermal growth factor, and two to nine consensus repeats (CR) similar to sequences found in complement regulatory proteins, a hydrophobic transmembrane domain and possesses a short cytoplasmic tail. The initial attachment of leukocytes, during inflammation, from the blood stream is afforded by the selectin family, and causes a slow downstream movement of leukocytes along the endothelium via transient, reversible, adhesive interactions called leukocyte rolling. Mouse knock studies have shown that all the three selectins are equally important in leukocyte rolling and deficiency of any one selectin affects the role of other selectins. This leukocyte rolling is aggravated in the sickle cell disease where the abnormal shaped cells bind to the epithelium of the blood vessels through selectins and block the blood flow causing vaso-occlusive pain.

It is becoming evident that selectins may play a major role in inflammation and progression of cancer (Ley K, *Trends Mol Med*, 2003, 6, 263). Tumor cells exploit the selectin-dependent mechanisms mediating cell tethering and rolling interactions through recognition of carbohydrate ligands on tumor cell to enhance distant organ metastasis, showing 'leukocyte mimicry' (Barthel S, et al. *Exp. Opin. Ther. Targets*, 2007, 11, 1473). A number of studies have shown increased expression of carbohydrate ligands on metastatic tumor enhanced E-selectin expression on the surface of endothelial vessels at the site at tumor metastasis, and the capacity of metastatic tumor cells to roll and adhere to endothelial cells, indicating the role of selectins in metastasis (Gout, S. et al. *Cancer Res*, 2006, 66, 9117). In addition to E-selectin, the role of P-selectin (expressed on platelets) and L-selectin (on leukocytes) in cancer dissemination has been suggested in the way that they interact with circulating cancer cells at an early stage of metastasis.

Exhibit 15: Role of selectins in cancer metastasis

Source: http://www.springerimages.com/Images/RSS/1-10.1007_s10555-010-9263-y-0

Capital Structure

After its IPO in January, in which GlycoMimetics raised net proceeds of \$57.4 million from a public equity offering of 8.6 million shares of common stock (includes an underwriter option of 1.05 million) at \$8/share, the company has cash of \$60.9M, which is sufficient to fund operations to 1H 2016. Lock-up expiration from the IPO occur 180 days post-IPO. GlycoMimetics plans to use the net proceeds to fund research and development of preclinical pipeline, including drug discovery, and remainder for working capital and other general corporate purposes.

Management Team

Rachel King, CEO

Rachel King is the co-founder and CEO of GlycoMimetics. Ms. King brings over a decade of management experience through her previous roles as a SVP of Novartis Corporation and CEO of Genetic Therapy, Inc. and holds a M.B.A. from Harvard Business School and a bachelor's degree from Dartmouth College. Ms. King currently serves as chair of the Board of the Biotechnology Industry Association and Maryland Life Sciences Advisory Board.

John Magnani, VP and Chief Scientific Officer

Dr. Magnani is the co-founder and CSO of GlycoMimetics. Dr. Magnani is an expert in glycobiology and has been credited with discovery of Sialyl Lewis x carbohydrate molecule involved in cell-cell recognition. Dr. Magnani holds a Ph.D. from Princeton University and has previously worked as a scientist NIH for ten years.

Helen Thackray, VP of Clinical Development and Chief Medical Officer

Dr. Thackray is an expert in clinical trial design and execution and joined GlycoMimetics in 2006. Previously Dr. Thackray spent five years as VP of Clinical Development at Biosynexus managing its clinical portfolio. Dr. Thackray holds an M.D. from George Washington University School of Medicine and a B.S. from Stanford University.

Brian Hahn, Chief Financial Officer

Prior to joining GlycoMimetics in 2010, Mr. Hahn held several senior financial roles at MiddleBrook Pharmaceuticals and Bering Truck Corporation. Mr. Hahn holds an M.B.A. degree from the University of Maryland and a B.B.A. degree in accounting from Shenandoah University.

Exhibit 16: GlycoMimetics Income Statement

Glycomimetics

Quarterly Income Statement

(All values in \$MM except EPS and average shares)

(All values in \$MM except EPS and average shares)																																
	2011A		2012A		2013E						2014E				2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E
	FY	FY	1Q4	2Q4	3Q4	4Q4	FY	1Q4	2Q4	3Q4	4Q4	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	
Revenue:																																
GM1-K70 US Royalties	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	12	6.7	12.1	16.8	22.9	28.9	35.7	42.2	47.5	53.3	58.3	61.2	64.3	67.6	14	15		
GM1-K70 EU Royalties	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.4	1.1	1.9	2.9	4.0	4.9	5.6	6.3	7.1	7.5	7.8	8.2	8.9	0.5	0.2			
GM1-K27 Revenues	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	20.6	36.1	47.1	55.3	60.8	63.6	66.4	68.3	72.3	73.7	75.2	76.7	78.2		
License and collaboration revenues	3.8	15.3	13	13	13	13	5.2	0.0	0.0	35.0	0.0	35.0	3.1	25.0	50.0	50.0	0.0	0.0	0.0	0.0	40.0	0.0	0.0	0.0	0.0	0.0	0.0	40.0	0.0	0.0	0.0	
Total revenue, net	3.8	15.3	13	13	13	13	5.2	0.0	0.0	35.0	0.0	35.0	3.1	25.0	51.2	57.0	13.3	39.3	65.0	80.0	135.8	199.6	117.4	126.7	135.0	141.4	186.3	151.6	78.6	80.0		
Costs and expenses:																																
Cost of goods sold	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6	0.0	0.0	0.0	0.0	10	2.0	2.4	2.8	3.0	3.2	3.3	3.5	3.6	3.7	3.8	3.8	3.9		
Research & development	7.8	9.4	2.9	2.9	2.9	3.0	11.6	3.9	4.5	5.1	5.2	16.7	26.8	22.5	21.9	10.3	20.3	20.9	21.5	22.1	22.8	23.5	24.0	24.4	24.9	25.4	25.9	26.2	26.5	26.7		
Selling, general & administrative	2.1	2.2	0.7	0.7	0.6	0.6	2.6	0.7	0.7	0.7	0.7	2.6	2.6	2.8	3.0	3.3	3.4	8.6	8.9	9.1	9.4	9.7	10.0	10.3	10.6	10.9	11.2	11.6	11.9	12.3		
Total operating expenses	9.9	11.6	3.6	3.6	3.5	3.6	14.2	4.6	5.2	5.8	5.9	21.3	30.0	25.3	24.9	22.6	23.7	30.5	32.3	33.6	35.0	36.2	37.1	38.0	39.0	40.0	40.9	41.5	42.2	42.9		
Income (loss) from operations	(6.1)	3.7	(2.3)	(2.3)	(2.2)	(2.3)	(9.0)	(4.6)	(5.2)	29.3	(5.9)	13.7	(26.9)	(0.3)	26.3	34.5	(10.5)	8.8	32.7	46.3	100.8	72.4	80.3	88.7	96.1	101.4	145.4	110.1	36.4	37.1		
Other income (expense):																																
Miscellaneous (expense) income	0.0	0.0	0.0	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0		
Interest income	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0		
Interest expense	(0.0)	(0.0)	0.0	0.0	0.0	0.0	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0		
Net profit (loss) before income taxes	(6.1)	3.7	(2.3)	(2.2)	(2.2)	(2.3)	(9.0)	(4.5)	(5.1)	29.3	(5.9)	13.8	(26.9)	(0.3)	26.3	34.5	(10.5)	8.8	32.7	46.3	100.8	72.4	80.3	88.7	96.1	101.4	145.4	110.1	36.4	37.1		
Income tax expense (benefit)															0.0	0.0	0.0	0.0	0.0	6.2	35.3	25.3	28.1	310	33.6	35.5	50.9	38.5	12.7	13.0		
Income tax rate (%)															0.0%	0.0%	0.0%	0.0%	0.0%	35.0%	35.0%	35.0%	35.0%	35.0%	35.0%	35.0%	35.0%	35.0%	35.0%	35.0%		
Net income (GAAP)	(6.1)	3.7	(2.3)	(2.2)	(2.2)	(2.3)	(9.0)	(4.5)	(5.1)	29.3	(5.9)	13.8	(26.9)	(0.3)	26.3	34.5	(10.5)	8.8	32.7	30.1	65.5	47.1	52.2	57.7	62.4	65.9	94.5	71.6	23.6	24.1		
EPS, GAAP																																
Basic	(6.78)	4.06	(2.18)	(2.16)	(2.09)	(2.22)	(6.65)	(0.24)	(0.28)	157	(0.31)	0.74	(141)	(0.02)	127	164	(0.49)	0.40	149	136	213	208	220	220	220	220	220	220	220	220	220	
Diluted	\$ (6.78)	\$ 0.33	\$ (2.18)	\$ (2.16)	\$ (2.09)	\$ (2.22)	\$ (6.65)	\$ (0.24)	\$ (0.28)	\$ 157	\$ (0.31)	\$ 0.74	\$ (141)	\$ (0.02)	\$ 127	\$ 164	\$ (0.49)	\$ 0.40	\$ 146	\$ 132	\$ 2.82	\$ 1.98	\$ 2.16	\$ 2.34	\$ 2.48	\$ 2.57	\$ 3.61	\$ 2.68	\$ 0.87	\$ 0.87		
Weighted average share- Basic	0.9	0.9	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	
Weighted average share- Diluted	0.9	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	

Source: Jefferies estimates, company data

Exhibit 17: GlycoMimetics Balance Sheet

Glycomimetics

Balance Sheet

(All values in \$MM)

	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E
	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY
Current assets:												
Cash and cash equivalents	17.4	10.8	74.5	44.8	42.3	63.1	95.0	80.0	84.1	111.7	136.7	197.2
Certificants of deposit	0	0	0	0	0	0	0	0	0	0	0	0
Cash and investments	17.4	10.8	74.5	44.8	42.3	63.1	95.0	80.0	84.1	111.7	136.7	197.2
Prepaid expenses	0.6	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Total current assets	18.0	11.1	74.8	45.1	42.6	63.4	95.4	80.3	84.5	112.1	137.1	197.5
Property and equipment, net	0.5	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Total assets	18.4	11.6	75.3	45.5	43.0	63.8	95.8	80.7	84.9	112.5	137.5	197.9
Current liabilities:												
Accounts payable	0.8	0.5	17	2.1	3.6	2.3	4.5	5.1	6.2	7.3	8.4	9.5
Accrued expenses	0.5	0.7	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0
Deferred rent	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Accrued bonuses	0.3	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Current portion of deferred revenue	4.0	0.1	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total current liabilities	5.7	1.7	3.8	4.2	5.7	4.3	6.5	7.1	8.2	9.3	10.4	11.5
Deferred rent, excluding current portion	0.2	0.1	14	14	14	14	14	14	14	14	14	14
Total Liability	5.9	1.8	5.2	5.6	7.1	5.7	7.9	8.5	9.6	10.7	11.8	12.9
Total stockholders' equity	12.5	9.7	70.1	39.9	35.9	58.1	87.9	72.2	75.3	101.8	125.7	185.0
Total liabilities and stockholders' equity	18.4	11.6	75.3	45.5	43.0	63.8	95.8	80.7	84.9	112.5	137.5	197.9

Source: Jefferies estimates, company data

Exhibit 18: GlycoMimetics Cash Flow Statement

Glycomimetics

Cash Flow Statement

(All values in \$MM)

	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E
	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY	FY
Cash flows from operating activities:												
Net income	3.7	(9.0)	13.8	(26.9)	(0.3)	26.3	34.5	(10.5)	8.8	32.7	30.1	65.5
Adjustments to reconcile cash by operating activities:												
Depreciation and amortization expense	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Compensation expense	0.2	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Changes in operating assets and liabilities:												
Prepaid expenses	(0.2)	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Deferred rent	(0.1)	(0.0)	12	0.4	15	(13)	2.2	0.6	11	11	11	11
Deferred revenue	(13.8)	(3.9)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Accounts payable	(0.0)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)	(0.2)
Accrued expenses and deferred rent	(0.3)	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Net cash provided by operating activities	(10.5)	(12.6)	14.8	(26.7)	1.0	24.8	36.5	(10.0)	9.7	33.6	31.0	66.5
Cash flows from investing activities:												
Restricted cash	(0.0)	0.0	(2.5)	(3.0)	(3.5)	(4.0)	(4.5)	(5.0)	(5.5)	(6.0)	(6.0)	(6.0)
Purchase of fixed assets	(0.2)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)
Net cash (used in) provided by investing activities	(0.2)	(0.0)	(2.5)	(3.0)	(3.5)	(4.0)	(4.5)	(5.0)	(5.5)	(6.0)	(6.0)	(6.0)
Cash flows from financing activities:												
Cash received from noncontrolling interest investment	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Issuance of common stock, net of offering costs	0.0	0.0	57.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Issuance of common stock from exercise of stock options	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Repurchase from preferred stock	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Proceeds from preferred stock	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Proceeds from notes payable	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Principal payments on debt	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Payments under capital lease obligations	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net cash (used in) provided by financing activities	0.0	0.0	57.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Effect if exchange rate changes on cash/equivalents												
Increase (decrease) in cash and cash equivalents	(10.7)	(12.6)	69.6	(29.7)	(2.5)	20.8	32.0	(15.0)	4.2	27.6	25.0	60.4
Cash and cash equivalents at beginning of period	28.2	17.4	4.8	74.5	44.8	42.3	63.1	95.0	80.0	84.1	111.7	136.7
Cash and cash equivalents at end of period	17.4	4.8	74.5	44.8	42.3	63.1	95.0	80.0	84.1	111.7	136.7	197.2

Source: Jefferies estimates, company data

Exhibit 19: GlycoMimetics DCF Analysis

Glycomimetics
Discounted Cash Flow Analysis

(All values in \$MM)	2012A	2013E	2014E	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E
Sales	15.3	5.2	35.0	3.1	25.0	51.2	57.0	13.3	39.3	65.0	80.0	135.8	108.6	117.4	126.7	135.0	141.4	186.3	151.6	78.6	80.0
Operating Expenses	11.6	14.2	21.3	30.0	25.3	24.9	22.6	23.7	30.5	32.3	33.6	35.0	36.2	37.1	38.0	39.0	40.0	40.9	41.5	42.2	42.9
EBIT	3.7	(9.0)	13.7	(26.9)	(0.3)	26.3	34.5	(10.5)	8.8	32.7	46.3	100.8	72.4	80.3	88.7	96.1	101.4	145.4	110.1	36.4	37.1
(-): Taxes	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	16.2	35.3	25.3	28.1	31.0	33.6	35.5	50.9	38.5	12.7	13.0
EBIAT	3.7	(9.0)	13.7	(26.9)	(0.3)	26.3	34.5	(10.5)	8.8	32.7	30.1	65.5	47.1	52.2	57.7	62.4	65.9	94.5	71.6	23.6	24.1
(+): Depreciation	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
(+): FAS-123 Options	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
(-): Capital expenditures	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
(-): Changes in working capital	1.9	(3.8)	1.0	0.2	1.3	(1.5)	2.0	0.4	0.9	0.9	0.9	0.9	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Unlevered free cash flow	1.7	(5.2)	12.7	(27.2)	(1.7)	27.8	32.4	(10.9)	7.8	31.7	29.2	64.6	47.1	52.2	57.7	62.4	65.9	94.5	71.6	23.6	24.1

Source: Jefferies estimates, company data

Exhibit 20: GMI-1070 Revenue Build for Sickle Cell Disease

GMI-1070	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032																
U.S.																																		
Total Net Sales of GMI-1070 in sickle cell crisis (\$M)				\$13.8	\$74.0	456%	\$134.8	82%	\$186.8	39%	\$254.6	36%	\$321.4	26%	\$396.3	23%	\$469.1	18%	\$528.1	13%	\$591.8	12%	\$647.6	9%	\$680.4	5%	\$714.8	5%	\$751.0	5%	\$15.8	-98%	\$16.6	5%
Risk discount				23%	23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%	
Total Net Sales of GMI-1070 in sickle cell crisis - risk adj. (\$M)				\$10.4	\$55.5		\$101.1		\$140.1		\$190.9		\$241.0		\$297.2		\$351.9		\$396.1		\$443.8		\$485.7		\$510.3		\$536.1		\$563.3		\$11.8		\$12.4	
Total Net Sales of GMI-1070 in sickle cell crisis - risk adj. (\$M)				\$10.4	\$55.5		\$101.1	82%	\$140.1	39%	\$190.9	36%	\$241.0	26%	\$297.2	23%	\$351.9	18%	\$396.1	13%	\$443.8	12%	\$485.7	9%	\$510.3	5%	\$536.1	5%	\$563.3	5%	\$11.8	-98%	\$12.4	5%
Royalty rate				12%	12%		12%		12%		12%		12%		12%		12%		12%		12%		12%		12%		12%		12%		12%		12%	
U.S. royalties to GMI-1070 - risk adj. (\$M)				\$1.24	\$6.66		\$12.13	82%	\$16.81	39%	\$22.91	36%	\$28.92	26%	\$35.67	23%	\$42.22	18%	\$47.53	13%	\$53.26	12%	\$58.29	9%	\$61.24	5%	\$64.34	5%	\$67.59	5%	\$14.2	-98%	\$14.9	5%
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032																
U.S. Sickle Cell Crisis Market Opportunity																																		
Total no. of sickle cell disease patients (000's)	82,824	84,480	86,170	87,893	89,651	91,444	93,273	95,139	97,042	98,982	100,962	102,981	105,041	107,142	109,285	111,470	113,700	115,974																
Total no. of pain episodes in sickle cell patients (000's)	66,425	67,753	69,108	70,491	71,900	73,338	74,805	76,301	77,827	79,384	80,972	82,591	84,243	85,928	87,646	89,399	91,187	93,011																
% of total no. of sickle cell disease patients	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%	80.2%																
Sickle cell anemia (SS)	53,836	54,912	56,011	57,131	58,273	59,439	60,628	61,840	63,077	64,339	65,625	66,938	68,277	69,642	71,035	72,456	73,905	75,383																
Genotype prevalence	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%	65%																
Average no. of pain episodes per year	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0																
Sickle hemoglobin disease (SC)	8,282	8,448	8,617	8,789	8,965	9,144	9,327	9,514	9,704	9,898	10,096	10,298	10,504	10,714	10,928	11,147	11,370	11,597																
Genotype prevalence	23%	23%	23%	23%	23%	23%	23%	23%	23%	23%	23%	23%	23%	23%	23%	23%	23%	23%																
Average no. of pain episodes per year	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4																
Sickle (β+) thalassemia	2,650	2,703	2,757	2,813	2,869	2,926	2,985	3,044	3,105	3,167	3,231	3,295	3,361	3,429	3,497	3,567	3,638	3,711																
Genotype prevalence	8%	8%	8%	8%	8%	8%	8%	8%	8%	8%	8%	8%	8%	8%	8%	8%	8%	8%																
Average no. of pain episodes per year	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4	0.4																
Sickle (β0) thalassemia	1,656	1,690	1,723	1,758	1,793	1,829	1,865	1,903	1,941	1,980	2,019	2,060	2,101	2,143	2,186	2,229	2,274	2,319																
Genotype prevalence	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%	2%																
Average no. of pain episodes per year	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0																
Patients on GMI-1070 (000's)			1,382	7,049	12,223	16,134	20,945	25,179	29,574	33,341	36,437	39,644	42,121	42,964	43,823	44,700	912	930																
% of total no. of pain episodes in sickle cell patients			2.0%	10.0%	17.0%	22.0%	28.0%	33.0%	38.0%	42.0%	45.0%	48.0%	50.0%	50.0%	50.0%	50.0%	1.0%	1.0%																
U.S. Net Sales of GMI-1070 in sickle cell crisis (\$M)				\$13.8	\$74.0	456%	\$134.8	82%	\$186.8	39%	\$254.6	36%	\$321.4	26%	\$396.3	23%	\$469.1	18%	\$528.1	13%	\$591.8	12%	\$647.6	9%	\$680.4	5%	\$714.8	5%	\$751.0	5%	\$15.8	-98%	\$16.6	5%
Risk discount				23%	23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%		23%	
U.S. Net Sales of GMI-1070 in sickle cell crisis - risk adj. (\$M)				\$10.4	\$55.5		\$101.1		\$140.1		\$190.9		\$241.0		\$297.2		\$351.9		\$396.1		\$443.8		\$485.7		\$510.3		\$536.1		\$563.3		\$11.8		\$12.4	

Source: Jefferies estimates, company data

Exhibit 21: GMI-1271 Revenue Build for Acute Myeloid Leukemia

GMI-1271	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032															
U.S.																																	
Total Net Sales of GMI-11271 in AML (\$M)			\$0.0	\$0.0	####	\$0.0	####	\$205.6	####	\$391.5	90%	\$470.6	20%	\$552.8	17%	\$608.3	10%	\$635.6	4%	\$663.8	4%	\$692.8	4%	\$722.7	4%	\$737.2	2%	\$751.9	2%	\$766.9	2%	\$782.3	2%
Risk discount								90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	
Total Net Sales of GMI-1271 in AML - risk adj. (\$M)			\$0.0	\$0.0	\$0.0	\$20.6	\$39.1	\$47.1	\$55.3	\$60.8	\$63.6	\$66.4	\$69.3	\$72.3	\$73.7	\$75.2	\$76.7	\$78.2															
Total Net Sales of GMI-1271 in sickle cell crisis - risk adj. (\$M)			\$0.0	\$0.0	\$0.0	####	\$20.6	####	\$39.1	90%	\$47.1	20%	\$55.3	17%	\$60.8	10%	\$63.6	4%	\$66.4	4%	\$69.3	4%	\$72.3	4%	\$73.7	2%	\$75.2	2%	\$76.7	2%	\$78.2	2%	
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032															
U.S. AML Market Opportunity																																	
Total no. of sickle cell disease patients (000's)	19,100	19,482	19,872	20,269	20,674	21,088	21,510	21,940	22,379	22,826	23,283	23,748	24,223	24,708	25,202	25,706	26,220	26,745															
Relapsed/refractory prevalence	6,017 32%	6,137 32%	6,260 32%	6,385 32%	6,512 32%	6,643 32%	6,776 32%	6,911 32%	7,049 32%	7,190 32%	7,334 32%	7,481 32%	7,630 32%	7,783 32%	7,939 32%	8,097 32%	8,259 32%	8,425 32%															
Elderly 1st line eligible prevalence	5,291 28%	5,455 28%	5,624 28%	5,777 29%	5,934 29%	6,116 29%	6,238 29%	6,363 29%	6,490 29%	6,620 29%	6,752 29%	6,887 29%	7,025 29%	7,165 29%	7,309 29%	7,455 29%	7,604 29%	7,756 29%															
Elderly 1st line ineligible prevalence	3,495 18%	3,565 18%	3,637 18%	3,709 18%	3,783 18%	3,859 18%	3,936 18%	4,015 18%	4,095 18%	4,177 18%	4,261 18%	4,346 18%	4,433 18%	4,522 18%	4,612 18%	4,704 18%	4,798 18%	4,894 18%															
Non-elderly 1st line prevalence	4,202 22%	4,286 22%	4,372 22%	4,459 22%	4,548 22%	4,639 22%	4,732 22%	4,827 22%	4,923 22%	5,022 22%	5,122 22%	5,225 22%	5,329 22%	5,436 22%	5,544 22%	5,655 22%	5,768 22%	5,884 22%															
Patients on GMI-1271 (000's)						3,163	6,023	7,240	8,504	9,359	10,658	10,212	11,119	11,341	11,568	11,799	12,035																
% market share						15.0%	28.0%	33.0%	38.0%	41.0%	42.0%	43.0%	44.0%	45.0%	45.0%	45.0%	45.0%																
U.S. Net Sales of GMI-1271 (\$M)																																	
Risk discount						90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%															
U.S. Net Sales of GMI-1271 - risk adj. (\$M)						\$205.6	\$391.5	\$470.6	\$552.8	\$608.3	\$635.6	\$663.8	\$692.8	\$722.7	\$737.2	\$751.9	\$766.9	\$782.3															

Source: Jefferies estimates, company data

Company Description

GlycoMimetics, Inc., a clinical stage biotechnology company, focuses on the discovery and development of glycomimetic drugs to address unmet medical needs resulting from diseases. Its lead product includes GMI-1070, a pan-selectin antagonist that has completed Phase II clinical trials for the treatment of vaso-occlusive crisis. The company is partnered with Pfizer on the development of GMI-1070. The company is also developing GMI-1271, a specific E-selectin antagonist, which it plans to initiate a Phase I clinical trials patients with acute myeloid leukemia and potentially other hematologic cancers. GlycoMimetics, Inc. was incorporated in 2003 and is headquartered in Gaithersburg, Maryland.

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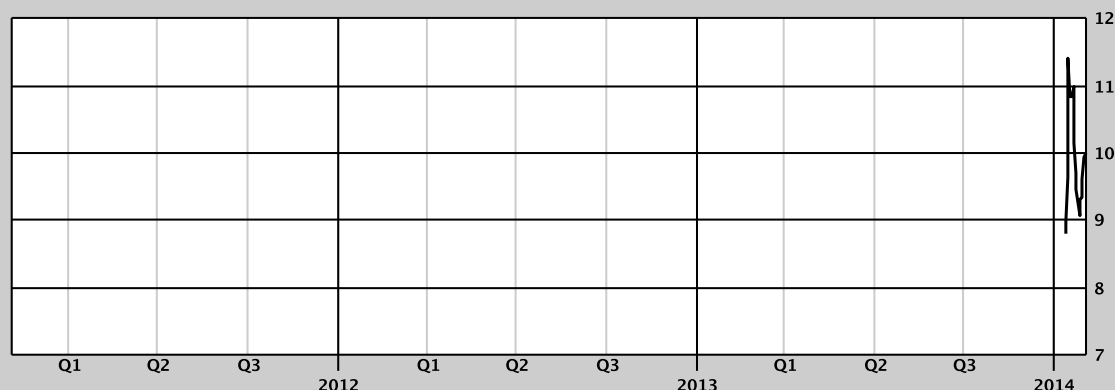
Risk which may impede the achievement of our Price Target

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- Celldex Therapeutics, Inc. (CLDX: \$24.10, BUY)
- Exelixis, Inc. (EXEL: \$6.53, HOLD)
- Novartis AG (NOVN VX: CHF71.45, HOLD)
- Pfizer, Inc. (PFE: \$30.60, BUY)
- Verastem Inc. (VSTM: \$11.97, BUY)
- XOMA Ltd. (XOMA: \$7.12, BUY)

Rating and Price Target History for: GlycoMimetics, Inc. (GLYC) as of 02-03-2014



Distribution of Ratings

Rating	Count	Percent	IB Serv./Past 12 Mos.	
			Count	Percent
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HOLD	783	43.09%	122	15.58%
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