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4 February 2014

GlycoMimetics Inc.

Mimicking Biology to Combat Disease

We are initiating coverage with an Overweight rating and PT of $15: Currently GlycoMimetics has one partnered asset (GMI-1070) set to begin Phase III testing in sickle cell disease (SCD) by mid-year and a proprietary compound (GMI-1271) it hopes to enter clinical testing soon. These agents block a class of molecules on epithelial cells termed selectins, which are involved in cell mobilit y and adhesion. GlycoMimetics also has an early-stage pipeline of several pre-clinical compounds which it hopes to develop across a variety of diseases.

Proof-of-concept study de-risks Phase III program of GMI-1070: Results from a Phase II study of GMI-1070 in the setting of veno-occlusive crisis (VOC) were presented in December 2013 at the American Society of Hematology (ASH) meeting. VOC is an episode that occurs in individuals with SCD and characterized by severe pain, and in some cases organ damage, due to irregularly-shaped red blood cells. The data showed consistent improvements in the duration of crisis, use of opiods, and other clinical metrics. Partner Pfizer plans to initiate a Phase III study under a Special Protocol Assessment with FDA using time-to-hospital discharge as the primary endpoint by mid-year as well as pay for all costs going forward. Assuming an 11% royalty rate on peak sales of $666mn, we believe GlycoMimetics could receive up to $73mn in royalty. Additionally, GlycoMimetics is eligible to receive up to $340mn in milestone payments.

While early, pre-clinical data of GMI-1271 looks interesting: Preclinical work has demonstrated that if leukemic blasts can be mobilized out of the bone marrow and into the blood they become more susceptible to chemotherapy; GMI-1271 has been shown to do just that in animal models of leukemia. We expect GMI-1271 to enter the clinic in healthy volunteers over the next several months, followed by the initiation of a Phase I/II study in AML patients in the third quarter. We estimate GMI-1271 could reach global peak sales of $475mn.

GLYC: Quarterly and Annual EPS (USD)

	2012	2013			2014			Change y/y	
FY Dec	Actual	Old	New	Cons	Old	New	Cons	2013	2014
Q1	N/A	N/A	0.07A	N/A	N/A	-0.44E	N/A	N/A	-729%
Q2	N/A	N/A	-0.30A	N/A	N/A	0.43E	N/A	N/A	243%
Q3	N/A	N/A	-0.81A	N/A	N/A	0.64E	N/A	N/A	179%
Q4	N/A	N/A	-0.24E	N/A	N/A	-0.49E	N/A	N/A	-104%
Year	0.33A	N/A	-1.28E	N/A	N/A	0.27E	N/A	-488%	121%
P/E	30.2		N/A			37.8			

Source: Barclays Research.
Consensus numbers are from Thomson Reuters

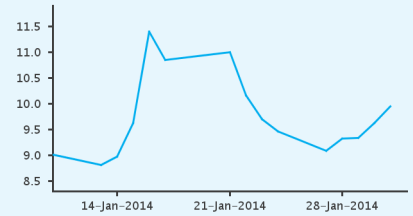
Stock Rating	OVERWEIGHT from N/A
Industry View	NEUTRAL Unchanged
Price Target	USD 15.00 from N/A

Price (03-Feb-2014)	USD 10.01
Potential Upside/Downside	+50%
Tickers	GLYC

Market Cap (USD mn)	176
Shares Outstanding (mn)	17.56
Free Float (%)	97.09
52 Wk Avg Daily Volume (mn)	0.5
Dividend Yield (%)	N/A
Return on Equity TTM (%)	N/A
Current BVPS (USD)	0.67

Source: Thomson Reuters

Price Performance — Exchange-Nasdaq
52 Week range — USD 11.99-8.40



Link to Barclays Live for interactive charting

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U.S. Biotechnology **Industry View: NEUTRAL**

GlycoMimetics Inc. (GLYC)

Stock Rating: OVERWEIGHT

Income statement ($mn)	2012A	2013E	2014E	2015E	CAGR
Revenue	15	4	35	0	-100.0%
EBITDA (adj)	4	-13	5	-40	N/A
EBIT (adj)	4	-13	5	-40	N/A
Pre-tax income (adj)	4	-13	5	-40	N/A
Net income (adj)	4	-13	5	-40	N/A
EPS (adj) ($)	0.33	-1.28	0.27	-2.15	N/A
Diluted shares (mn)	11.0	10.6	17.4	18.7	19.2%
DPS ($)	0.00	0.00	0.00	0.00	N/A

Margin and return data					Average
EBITDA (adj) margin (%)	24.7	-355.4	14.2	N/A	-105.5
EBIT (adj) margin (%)	24.0	-359.4	13.2	N/A	-107.4
Pre-tax (adj) margin (%)	24.0	-359.4	13.2	N/A	-107.4
Net (adj) margin (%)	24.0	-359.4	13.2	N/A	-107.4
ROIC (%)	-71.4	953.7	-154.6	1,488.0	553.9
ROA (%)	15.4	-141.5	12.4	-84.4	-49.5
ROE (%)	34.8	-233.0	15.3	-97.7	-70.1

Balance sheet and cash flow ($mn)					CAGR
Tangible fixed assets	0	0	0	0	-41.2%
Intangible fixed assets	0	0	-1	0	N/A
Cash and equivalents	17	0	73	21	6.9%
Total assets	18	1	74	21	4.5%
Short and long-term debt	0	0	0	0	-100.0%
Other long-term liabilities	0	0	0	0	N/A
Total liabilities	6	2	13	0	-100.0%
Net debt/(funds)	-17	0	-73	-21	N/A
Shareholders' equity	13	-1	61	21	18.8%
Change in working capital	-15	-4	11	-12	N/A
Cash flow from operations	-10	-17	16	-52	N/A
Capital expenditure	0	0	0	0	N/A
Free cash flow	-11	-17	16	-52	N/A

Valuation and leverage metrics					Average
P/E (adj) (x)	30.2	N/A	37.8	N/A	34.0
EV/EBITDA (adj) (x)	23.5	-7.9	6.6	-2.1	5.0
Equity FCF yield (%)	0.0	-16.2	8.9	-27.8	-8.8
EV/sales (x)	5.8	28.2	0.9	N/A	11.6
P/BV (x)	8.8	-110.8	2.8	8.9	-22.6
Dividend yield (%)	0.0	0.0	0.0	0.0	0.0
Total debt/capital (%)	1.6	-23.3	0.0	0.0	-5.4

Selected operating metrics					Average
SG&A/sales (%)	14.1	115.5	12.5	N/A	47.4
R&D/sales (%)	61.9	343.9	74.3	N/A	160.0
R&D growth (%)	21.0	36.6	101.6	34.6	48.5
SG&A growth (%)	2.7	100.8	1.4	15.3	30.0

Source: Company data, Barclays Research
Note: FY End Dec

Price (03-Feb-2014) **USD 10.01**
Price Target **USD 15.00**

Why Overweight? GlycoMimetics is currently in Phase III clinical testing with Pfizer-partnered GMI-1070 for the treatment of VOC. The company also has a proprietary compound, GMI-1271, which will enter clinical testing for the treatment of AML in 2014.

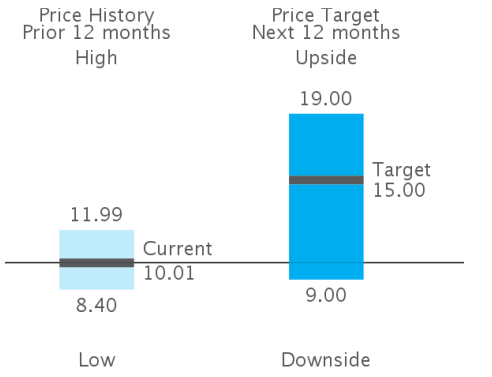
Upside case **USD 19.00**

Based on our current sales estimates for GMI-1070, assuming positive Phase III data and regulatory approval we reach a valuation of $19/share.

Downside case **USD 9.00**

Assuming clinical failure and subsequent termination of the GMI-1271, along with peak sales estimates for GMI-1070 of $500mn, we reach a valuation of $9/share.

Upside/Downside scenarios



Executive Summary: Mimicking Biology To Combat Disease

An early-stage biotechnology company, GlycoMimetics is focused on the development of molecules which mimic the structure of carbohydrates (sugars) involved in various biological processes (Figure 1). The company's initial efforts are focused in the area of rare orphan diseases. The most advanced of these compounds is GMI-1070, which recently completed Phase II testing for the treatment of vaso-occlusive crisis (VOC) in individuals with sickle-cell disease (SCD). With roughly 100,000 diagnosed individuals with SCD, there are 70,000 hospitalizations each year caused by VOC, a common event for many patients. Additionally, the company is developing GMI-1271 for the treatment of acute myeloid leukemia (AML), which is expected to enter the clinic over the next several months.

Positive Phase II data supports Phase III program with GMI-1070 in VOC. In October 2011, GlycoMimetics announced a world-wide exclusive licensing agreement with Pfizer in which the pan-selectin antagonist GMI-1070 would be fully developed by Pfizer upon success in Phase II testing. In April 2013, GlycoMimetics announced the completion of a Phase II randomized study involving 76 patients undergoing a VOC; the results showed a reduction in time to VOC resolution, length of hospital stay, and use of opioid analgesics for those treated with GMI-1070 versus placebo. Going forward, Pfizer is responsible for conducting a Phase III study and if successful, GlycoMimetics is eligible to receive up to $340mn in milestone payments along with tiered royalties (low-double digits to low-teens) on net sales. Most recently (December 2013), Pfizer had an end of Phase II (EOP2) meeting with the U.S. FDA in which proposed endpoints for a Phase III study and submission of a special protocol assessment (SPA) were discussed. We expect to hear additional details about this registration-enabling study, as well as its initiation, in the first half of 2014. ***We expect the Phase III trial to mimic the Phase II, with time-to-hospital discharge as the primary endpoint***. Based on our peak sales estimates of $666mn in 2029, we believe GlycoMimetics could receive up to $73mn in royalties from Pfizer, assuming an 11% rate (71% probability of reaching the market). Additionally, while there are several competitors on the horizon who are also looking to reduce the severity/duration of VOC, GMI-1070 is the only one in Phase III for the treatment of adults.

GMI-1271 will enter clinical testing for AML by mid-year. While much earlier in clinical development, GMI-1271 is designed to mimic the bioactive conformation of the E-selectin ligand. E-selectin is a molecule involved in endothelial cell adhesion, regulating the binding and extravasation of leukocytes from the bloodstream to tissues. Preclinical work has demonstrated that if leukemic blasts can be mobilized out of the bone marrow and in the blood they become more susceptible to chemotherapy; GMI-1271 has been shown to do just that in animal models of leukemia. We expect GlycoMimetics to enter Phase I testing with GMI-1271 in healthy volunteers over the next several months, followed by the initiation of a Phase I/II study in AML patients in 3Q. Approximately 14,590 new cases of adult AML will be diagnosed in 2013, with 10,370 dying of the disease and 36,000 patients currently living with a history of AML. Using the current standard-of-care, 60%-70% of adults with AML will achieve a complete response, with about 45% of those patients surviving for 3 years or more; the typical cure rate amongst all adults with AML is 25%. GlycoMimetics hopes to eventually test this agent in hard-to-treat patients, particularly the elderly (due to their fragile state) and those which have failed previous therapies. Using annual price of $40,000 per patient in the U.S. and $30,000 in the EU, we estimate a combined market opportunity of approximately $2.0bn, from which we expect peak sales of $475mn in 2033 (11% probability of reaching the market).

FIGURE 1
Pipeline Summary

Drug Candidate	Indication	Preclinical	Phase I	Phase II	Phase III	Worldwide Commercial Rights
GMI-1070 (Pan-selectin Antagonist)	Vaso-occlusive crisis (sickle cell disease)	→	→	→		Pfizer
GMI-1271 (E-selectin Antagonist)	Acute myeloid leukemia	→				GlycoMimetics
E-selectin and CXCR4 Antagonist	Cancer	→				GlycoMimetics
Orally-available E-selectin Antagonist	Cardiovascular disease, inflammation, cancer	→				GlycoMimetics
GMI-1051	Pseudomonas infections	→				GlycoMimetics

Source: Barclays Research, Company Reports

GMI-1070 Is the Major Near-term Value Driver

We are initiating coverage on GlycoMimetics (GLYC) with an Overweight rating and $15 price target. Our price target is based on a probability adjusted NPV of $9/share composed of royalty/milestone-derived cash flows from GMI-1070 in SCD during 2014-2029 (year of patent expiration) as well as a probability-adjusted NPV of $3/share composed of cash flows from GMI-1271 in AML during 2014-2033 (all discounted 10%). For the purpose of valuation, we only factor sales from these two products. Lastly, we attribute $3/share to the company's net cash position. A summary of our NPV model is detailed in Figure 2.

FIGURE 2
Probability-adjusted NPV Summary

Drug	Peak Sales ($ Thousand)	Stage	(Estimated) Launch	Probability of Reaching Market	GLYC Share	Probability Adjusted NPV	Per Share Value	% of Total
GMI-1070	$666,325	Phase 3	2018	71%	10-14% Royalty	$173,118	$9	61%
GMI-1271	$475,014	Phase 1	2020	11%	100%	$52,241	$3	18%
Total						**$225,359**	**$12**	**79%**
Other								
Net Cash (Cash/Equivalents - Debt)						**$60,000**	**$3**	**21%**

Discount Rate	10%
Time of Valuation	2015

Total	**$285,359**
Shares Out.	**18,500**
Per Share	**$15**

Source: Barclays Research, Company Reports

Upcoming Milestones and Catalysts

Along with the payment of a $35mn milestone by Pfizer to GlycoMimetics (in relation to the initiation of a Phase III study with GMI-1070), we believe additional clarity over the design of the study and verification of a SPA could further de-risk this program going forward. Additional catalysts over the next several years are listed below.

- 1Q 2014: Submit IND for GMI-1271.
- 2Q 2014: Begin Phase I dose-escalation study with GMI-1271 in health volunteers.

- Mid 2014: Initiate Phase I study for venous thromboembolic disease (investigator-initiated study).
- 1H 2014: GlycoMimetics receives a $15mn milestone payment by Pfizer ahead of the Phase III trial initiation with GMI-1070.
- 2H 2014: Initiation of Phase III study with GMI-1070 for the treatment of VOC (triggering a $20mn milestone payment from Pfizer to GlycoMimetics).
- 3Q 2014: Initiate Phase I/II dose-escalation study with GMI-1271 in defined patient populations with AML.
- Mid 2015: Potential top-line data readout from Phase I/II study with GMI-1271 in AML.
- 2015: Potential initiation of Phase I studies with early-stage compounds such as E-selecting and CXCR4 antagonists and GMI-1051.
- 2016: Potential top-line data readout from Phase III study with GMI-1070 (triggering a milestone payment from Pfizer to GlycoMimetics).

GMI-1070: Ameliorating VOC by Blocking Cell Adhesion

Drug Characteristics

GMI-1070 is a glycomimetic previously shown to inhibit E-, L-, and P-selectin. A family of cell adhesion molecules, selectins are single-chain transmembrane proteins expressed on the surface of leukocytes (L-selectin), platelets (P-selectin) and activated endothelial cells (E- and P-selectin). A number of studies have shown that selectins are key in mediating neutrophil, monocyte and lymphocyte rolling along the wall of blood vessels. During a VOC, red blood cells and white blood cells interact, which subsequently tethers them to the wall of blood vessels resulting in a thrombus. Pre-clinical work using GMI-1070 has demonstrated a reduction in arrested red blood cell/white blood cell aggregates leading to an improvement in blood flow.

GMI-1070 has previously been granted both Orphan Drug status and Fast Track designation for the treatment of VOC by the U.S. FDA, as well as Orphan Product status in the EU. Glycomimetics owns six issued U.S. patents which expire between 2023 and 2029 and a number of patents and patent applications for other regions of the world (mainly Europe) which are expected to expire between 2023 and 2028.

Following the recent completion of a Phase II study, Pfizer is now responsible for future development and commercialization of GMI-1070. Going forward, GlycoMimetics is eligible to receive up to $115mn in development-, $70mn in regulatory-, and $135mn in commercialization-related milestone payments along with tiered royalties (low-double digits to low-teens) on net sales. Most recently (December 2013), Pfizer had an end of Phase II (EOP2) meeting with the U.S. FDA in which proposed endpoints for a Phase III study and submission of a special protocol assessment were discussed. ***We expect this registration-enabling study to initiate in the second half of 2014, with potential for top-line data two years later.*** We expect the trial design to mimic that of the Phase II study, with time-to-hospital discharge as the primary endpoint.

Sickle cell disease is characterized by vaso-occlusive crises

An autosomal-recessive hereditary disorder, sickle cell disease (SCD) results from a single amino acid substitution in the gene which codes for the beta-globulin subunit of haemoglobin. Under low oxygen conditions, beta-globulin forms polymers, which structurally alter the shape of red blood cells and give them a crescent or sickle morphology.

Furthermore, unlike normal red blood cells which can live up to 120 days, sickle cells live for roughly 10-20 days. ***These abnormally shaped cells have trouble carrying blood and can obstruct blood vessels, leading to a vaso-occlusive crisis (VOC).*** Also referred to as sickle cell anemia, characteristic features of this disease include low level of functional red blood cells, repeat infections and periods of severe pain. In the U.S., SCD is most common in people of African descent, due to an evolutionary advantage associated with a resistance to malaria. Some common complications associated with SCD include pain, eye damage, strokes, acute chest syndrome, leg ulcers (sores), gallstones or gallbladder disease, priapism, and infections.

Vaso-Occlusive Crisis (VOC)

As a result of the abnormally-shaped cells, almost all individuals with SCD will go through an episode known as a vaso-occlusive crisis (VOC). A VOC results from a complex cascade of biological events involving multiple cell types and signalling molecules. Since the majority of individuals living with SCD have at least one VOC per year, this can result in irreversible damage to organs and lead to severe pain, chronic inflammation, pulmonary hypertension, cerebrovascular injury and kidney failure.

Research has suggested that red blood cells in the sickle state interact with white blood cells, which typically adhere to endothelial cells along blood vessels via selectins (particularly E-selectin) in order to extravasate and get to the site of an infection. Individuals with SCD typically have higher levels of cell adhesion molecules on their blood vessels than normal individuals, and these expression levels are highest during a VOC. As a result, this complex of sickle red blood cells and adherent white blood cells can aggregate and cause a blockage in blood flow. GMI-1070 has been designed to ameliorate the effects of a VOC by stopping E-selectin-mediated adhesion and sickle-cell/white blood cell interactions.

Current treatment paradigm largely consists of pain management

While there is no cure for SCD, the management of this disease typically focuses on the prevention of VOC or the treatment of symptoms.

In the case of preventing the symptoms associated with SCD, hydroxyurea is currently the only approved medicine. A crude therapy, hydroxyurea works by inducing the production of fetal haemoglobin in the blood of children and adults with SCD. Since SCD is characterized by abnormal haemoglobin, the fetal haemoglobin helps prevent red blood cells from sickling. With consistent dosing (by mouth) patients with SCD have been shown to have fewer blood transfusions, hospital visits, and survive longer.

When it comes to treating the pain associated with a VOC, different therapies are administered based on severity. In the case of mild pain, typical over the counter drugs, heating pads, rest and plenty of fluids will suffice. In the case of severe pain where hospitalization is required, patients are given fluids to prevent dehydration, NSAIDs, oxygen therapy, and in some cases opioids.

Phase II study with GMI-1070 are encouraging

GMI-1070 shows decreases in duration and severity of VOC in Phase II study

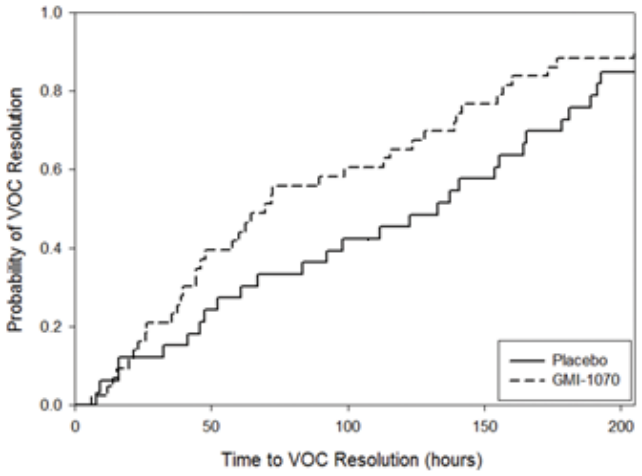
The most compelling clinical data to-date with GMI-1070 in the setting of VOC comes from a randomized, double-blind adaptive Phase II study. Overall, 76 subjects were enrolled and randomized 1:1 to receive either GMI-1070 twice-a-day or standard-of-care therapy. Overall there was a clear reduction in the time to VOC resolution (Figure 3) in the primary analysis, with a median of 69.6 hours for the GMI-1070 group versus 132.9 hours for the placebo group. Similarly, across all treatment groups an 83% reduction in the mean cumulative use of analgesic opioids was observed (Figure 4) between GMI-1070-treated and placebo-

treated patients. A comparative analysis of the study outcomes between both pediatric and adult patients is provided in Figure 5; it is important to point out that the reductions did not achieve statistical significance, which the study was not powered for. From the standpoint of safety, no subjects discontinued the study due to adverse events (AEs) and the total AE rates were similar amongst all treatment groups and ages.

While a relatively small study, we find the consistent reduction across multiple outcomes variables as reassuring. Furthermore, relative to the results from the Phase III study conducted by CytRx with MST-188, the Phase II data with GMI-1070 looks more consistent. As a result, we believe these results de-risk the future development of GMI-1070 in the setting of VOC resolution. Overall, we assign a 71% probability of this drug to reach the market in 2018.

FIGURE 3

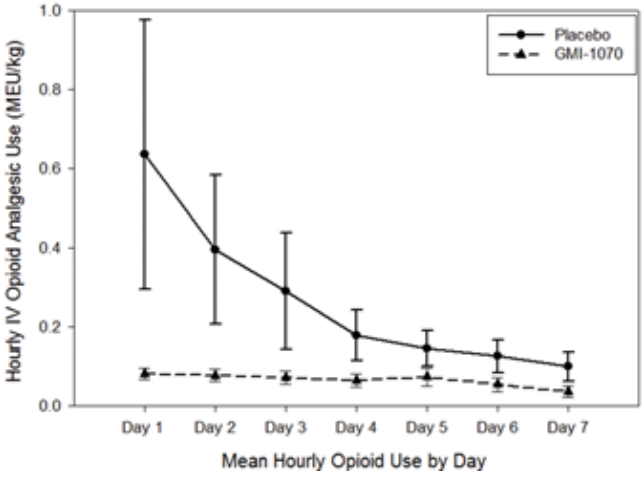
GMI-1070 Reduces Time to VOC Resolution



Source: Company Reports

FIGURE 4

GMI-1070 Reduces Use of Analgesic Opioids



Source: Company Reports

FIGURE 5
Phase II Outcomes Data with GMI-1070 in Adults and Pediatrics*

Outcomes Variables	Pediatric (N=20)			Adult (N=56)		
	Placebo	GMI-1070	% Reduction	Placebo	GMI-1070	% Reduction
Median Time to VOC Resolution (Hrs)	132.9	72.2	45.7	130.0	61.3	52.8
Median Time to Transition to Oral Opioids (Hrs)	164.2	76.4	53.5	140.8	67.0	52.4
Median Time to Discharge (Hrs)	168.2	72.2	57.1	149.9	77.5	48.2
Median Time to 1st Sustained Decrease in VAS Scores (Hrs)	146.2	101.3	30.7	125.3	43.8	65.0
Median Time to Readiness for Discharge (Hrs)	132.9	73.2	44.9	137.4	72.4	47.3

*Statistical testing comparing the % reductions between pediatrics and adults showed no significant differences.
Source: Barclays Research, Company Reports

Competition in VOC largely focused on pediatrics

While there is only one FDA approved therapy for SCD (hydroxyurea), there are a number of companies developing drugs designed to ameliorate the symptoms, particularly related to VOC. Below is a list of potential competitors on the horizon which have drugs in late-stage clinical testing.

MST-188 (Mast Therapeutics)

The most near-term competitive threat, Mast Therapeutics is currently running a Phase III study with MST-188 in children and adolescents (8-17 years of age) with SCD. MST-188 is a purified form of the copolymer surfactin poloxamer 188 (or P188). The drug is thought to be useful in the setting of SCD since it has been shown to reduce blood viscosity in large arteries and veins by reducing adhesive frictional forces. While MST-188 is in an ongoing Phase III study (termed EPIC), CytRx previously tested this compound in a 255-patient Phase III trial. Due to lessons learned from the previously failed study (see next paragraph), Mast believes that it has optimized EPIC in order to maximize the chances of success. Results from the CytRx study are detailed in Figure 6. ***While imperfect, we believe a cross-study comparison between the CytRx Phase III study and the GlycoMimetics Phase II study shows a more favourable profile with GMI-1070 versus MST-188.***

EPIC is a randomized, double-blind, placebo-controlled study which is expected to enrol 388 children and adolescents age 8-17. Based on results from the CytRx study, Mast discovered that younger patients seemed to benefit from MST-188 more than older patients. Additionally, while the CytRx study aimed to enrol 350 patients, the final number was only 255 due to problems in recruiting patients as they are undergoing a VOC (resulting in under-powering). The EPIC trial is randomizing patients in a 1:1 fashion to either MST-188 (treatment for 47 hours) or saline placebo during the time a patient is experiencing acute pain related to VOC. The primary endpoint of EPIC is duration of VOC episodes which will be

measured from the time patients are randomized to the time they receive the last dose of opioid analgesic before hospital discharge. This endpoint is different from the CytRx study, which looked at a composite score of pain, analgesic use, walking ability, and patient self-assessment. Thus, Mast believes that the new way of measuring VOC episode duration is simpler and less subjective, which the company believes was one of the short-comings of the CytRx study.

While both MST-188 and GMI-1070 are hoping to demonstrate a reduction the severity or duration of symptoms associated with VOC, the study populations are somewhat different. While Mast Therapeutics is currently conducting a study in younger patients (age 8-17), Pfizer is planning to conduct its study with GMI-1070 in adults (age 17 and older). As a result, while both drugs are being tested in the same disease the patient population is different.

FIGURE 6

Results from Phase III Study of MST-188 Conducted by CytRx

Duration of Crisis Episodes			
Groups	**MST-188**	**Placebo**	**P-value**
All randomized patients (n=255)	132.6 Hours	141.4 Hours	0.04
All treated patients (n=249)	132.3 Hours	140.4 Hours	0.07
Patients 15 years old or younger (n=73)	127.1 Hours	148.6 Hours	0.01
Patients concurrently receiving hydroxyurea (n=54)	141.4 Hours	157.2 Hours	0.02

Patients Achieving Crisis Resolution Within 168 Hours			
Groups	**MST-188**	**Placebo**	**P-value**
All randomized patients (n=255)	51.2%	35.2%	0.01
All treated patients (n=249)	51.6%	36.6%	0.02
Patients 15 years old or younger (n=73)	59.5%	27.8%	0.01
Patients concurrently receiving hydroxyurea (n=54)	46.2%	14.3%	0.02

Secondary Endpoints (all treated patients)			
Variables	**MST-188**	**Placebo**	**P-value**
Time to Discharge (hours)	148.9	152.9	0.71
Visual Analog Scale (VAS) Pain (U/Hour)	7516	7429	0.87
Total Analgesic Use (Units/Kg Morphine)	0.98	1.09	0.68
Pharmacoeconomics (total cost)	$7,859	$8,392	0.62

Source: Barclays Research, JAMA. 2001;286:2099-2106.

Effient (Lilly/Daiichi Sankyo)

An FDA approved drug (2009), Effient is an anti-platelet therapy which is currently used to reduce the rate of thrombotic cardiovascular events in patients with acute coronary syndrome. Earlier this year, results from a Phase II double-blind study were published in the *Journal of Hematology & Oncology* which tested Effient versus placebo in patients with SCD. The primary endpoint of the study was safety, measured by hemorrhagic events requiring medical intervention. Overall 62 patients were randomized, with 41 receiving 5mg Effient and 21 on placebo. Since there were no hemorrhagic events requiring medical intervention

in either arm of the study, there was no difference in the primary endpoint. The percentage of days with pain and the pain intensity was lower with Effient versus placebo; however this was not statistically significant. Lastly, there were statistically-significant decreases in biomarkers of platelet activation, particularly with surface P-selectin and plasma soluble P-selectin, when patients were treated with Effient relative to placebo.

Lilly is currently running a Phase III study, which it initiated earlier this year, in 240 children and adolescents (age 2-17) with number of VOC events per patient per year as the primary endpoint. If successful, we do not see Effient as a direct competitive threat to GMI-1070 since similar to MST-188 Lilly is running its pivotal study in children and adolescents.

HQK-1001 (HemaQuest Pharmaceuticals)

A short chain fatty acid derivative, HQK-1001 is an oral promoter-targeted agent which has previously been shown to induce the production of the fetal globulin gene. The rationale behind the use of this drug for SCD stems from the fact that babies who express high levels of fetal haemoglobin and have the autosomal recessive mutation in the beta-globulin gene do not exhibit symptoms of SCD.

In 2013, results from a Phase IIa dose-escalation study with HQK-1001 in SCD were published in the *American Journal of Hematology*. A total of 52 patients were administered HQK-1001 at multiple doses either alone or with hydroxyurea for 26 weeks. A number of patients discontinued from the trial early due to blood transfusions, adverse events or no-compliance, with only 25 patients (48%) completing the study. Of the 52 subjects, 42 (80%) had increases in their fetal haemoglobin, with 12 subjects (23%) seeing an increase of greater than or equal to 4%. The mean increase in fetal haemoglobin was 2% in the 21 patients receiving HQK-1001 alone and 2.7% in the 31 patients receiving the combination with hydroxyurea.

In June of 2013, HemaQuest announced that it had completed enrolment of a randomized, double-blind, placebo controlled Phase IIb study of HQK-1001 in patients with SCD. The trial is expected to fully enrol 77 patients, with results expected at some point this year. The primary endpoint of this study is to look at the induction of fetal haemoglobin from baseline over time, with secondary endpoints looking at frequency and intensity of pain crises.

GMI-1070 could reach $666mn in sales for treating VOC

The Centers for Disease Control (CDC) estimates that roughly 105,000 individuals are known to have SCD in the U.S.; this prevalence figure is roughly 40,000 in the EU. Based on research published by the CDC, there are roughly two emergency department visits for every individual with SCD, of which approximately 35% in the U.S. are due to a VOC. Based on wider use of hydroxyurea for prevention in the EU, we estimate that approximately 25% of ED visits are related to VOC. In total, we believe that there are approximately 73,600 VOC events in the U.S. and 20,000 in the EU which could be treated with GMI-1070. Using $9,500 in the U.S. and $6,500 in the EU for the average cost of treating a VOC with GMI-1070, we believe the current combined market opportunity is roughly $870mn. Based on our peak sales estimates of $666mn in 2029, we believe GlycoMimetics could receive up to $73mn in royalties from Pfizer assuming an 11% rate (Figure 7). As a reminder, Pfizer will be responsible for all marketing costs, as well as milestone payments through Phase III development, regulatory approval, and commercialization.

FIGURE 7
U.S. and EU Sickle Cell Disease/VOC Market

United States	2018	2029
Sickle Cell Disease Prevalence	105,663	106,714
Average ED visits per patient per year	2	2
Total number of ED visits	211,327	213,428
% ED visits due to VOC	35%	35%
Total VOC-related hospitalizations	73,964	74,700
GMI-1070 Penetration	5%	50%
Price	$9,500	$16,248
U.S. Revenue (thousands)	**$35,133**	**$606,870**
European Union		
Sickle Cell Disease Prevalence	40,253	40,653
Average ED visits per patient per year	2	2
Total number of ED visits	80,505	81,306
% ED visits due to VOC	25%	25%
Total VOC-related ED visits	20,126	20,326
GMI-1070 Penetration		45%
Price		$6,500
E.U. Revenue (thousands)		**$59,455**
Worldwide sales of GM-1070	**$35,133**	**$666,325**
Royalties to GLYC paid by PFE	**$3,865**	**$73,296**

Source: Barclays Research, www.cdc.gov.

GMI-1271: Making leukemia more susceptible to chemo

Drug Characteristics

A small molecule antagonist of E-selectin, GMI-1271 is designed to mimic the bioactive conformation of the E-selectin ligand. E-selectin is a molecule involved in endothelial cell adhesion, regulating the binding and extravasation of leukocytes from the bloodstream to tissues. The rationale behind the use of GMI-1271 in the setting of AML stems from studies done with patient samples. The ligand for E-selectin is termed HCELL (hematopoietic cell E-/L-selectin ligand) and is a functional glycoform of CD44. Previous research has demonstrated that CD44 is expressed at high levels by blasts from AML patients as well as in leukemic stem cells. CD44 has also been shown to be crucial in the homing of AML and acute lymphocytic leukemia (ALL) cells to the bone marrow. Studies using pan-selectin inhibitors show a disruption in the interaction of multiple myeloma cells to the bone marrow, making them more sensitive to the drug Velcade. Furthermore other studies also show a correlation between CD44 expression levels and leukemic relapse. GlycoMimetics has filed a number of patent applications related to GMI-1271, which if issued are expected to provide protection until 2033.

Acute myeloid leukemia is fast growing and difficult to treat

A disease of the blood and bone marrow, AML is defined by abnormal and immature cells called myeloblasts. Both white blood cells and red blood cells are derived from a common progenitor, termed a hematopoietic stem cell. Along that path to differentiation there is a divergence where lymphoid progenitors eventually become T cells and B cells while myeloid progenitors become red blood cells, platelets, and innate immune cells such as neutrophils and macrophages. Since AML cells or myeloblasts are immature and abnormal, they do not carry-out the function of typical myeloid cells, divide uncontrollably, build up in the bone

marrow and keep healthy blood cells from performing normal functions. Unlike chronic leukemias such as CML and CLL, which are slow growing, AML can rapidly progress and be very difficult to treat.

Chemotherapy and transplant continue to be the standards-of-care

Patients with newly-diagnosed AML are typically treated with the two-drug chemotherapy regimen of daunorubicin plus cytarabine; this combination typically results in a complete response (CR) rate of 65%. Older patients which cannot typically tolerate this intensive regimen have been shown to benefit from low-dose cytarabine, resulting in a relatively low CR rate of 18% versus only 1% for patients treated with hydroxyurea (Burnett et al. *Cancer* 2007).

In the setting of relapsed/refractory AML, currently there is no standard-of-care however several agents have shown activity. One combination chemotherapy regimen, mitoxantrone plus cytarabine, has previously been shown to result in a CR rate of 50%-60% in patients which have relapsed after previously obtaining a CR. Another combination, mitoxantrone, etoposide and cytarabine (MEC) has also been tested but with varying degrees of success. The immunotoxin Mylotarg (Pfizer) has also been used in relapsed/refractory disease with a reported CR rate of 30% in patients with CD33-expressing AML, however the drug was pulled from the market in 2010 due to toxicity. Lastly, bone marrow transplantation has resulted in cures following relapse. One study demonstrated a 30% rate of disease-free survival for patients in early-first relapse after receiving an allogeneic transplant from an HLA-matched donor. Nonetheless, clinical trials are still recommended for patients with recurrent AML, which leads us to believe that GMI-1271 could be well accepted if proven successful.

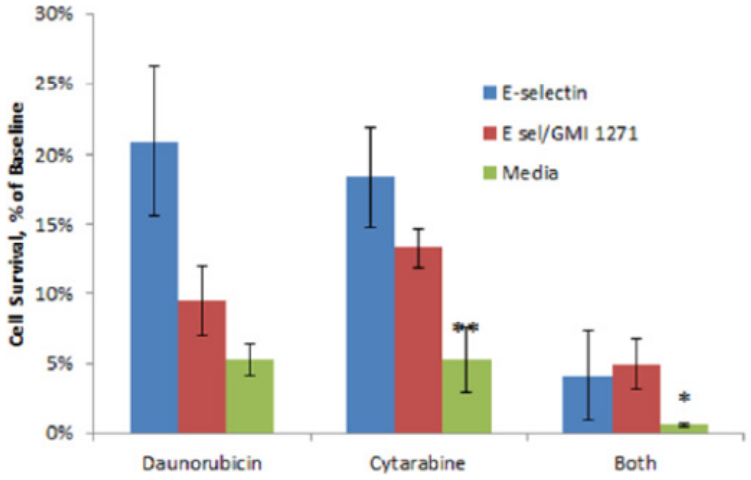
Preclinical data validates mechanism of action

The most extensive pre-clinical data presented to-date with GMI-1271 was presented at the 2012 and 2013 American Society of Hematology (ASH) annual meetings.

At the 2012 meeting, using an in vitro cell adhesion assay researchers demonstrated adhesion of leukemia cells (derived from a number of AML patient samples) to E-selectin. Subsequently, researchers showed that they could reduce the levels of cell adhesion from the majority of these same leukemia cells by adding GMI-1271. Next, using a cell survival assay, researchers first demonstrated that leukemia cells were more resistant to treatment with single-agent daunorubicin or cytarabine when bound to E-selectin coated plates; introduction of GMI-1271 was able to reverse this resistance, resulting in a decrease in cell survival (Figure 8). Lastly, using a mouse model containing human AML cells, the researchers could demonstrate a reduction in total number of leukemic cells in the bone marrow and spleen when the mice were treated with GMI-1271 versus vehicle (Figures 9 and 10). At the 2013 meeting, researchers demonstrated that the absence or therapeutic blockade of E-selectin (via GMI-1271) was able to increase survival, accelerate neutrophil recovery, and alleviate chemotherapy-induced mucositis, suggesting GMI-1271 has chemo-protective properties as well.

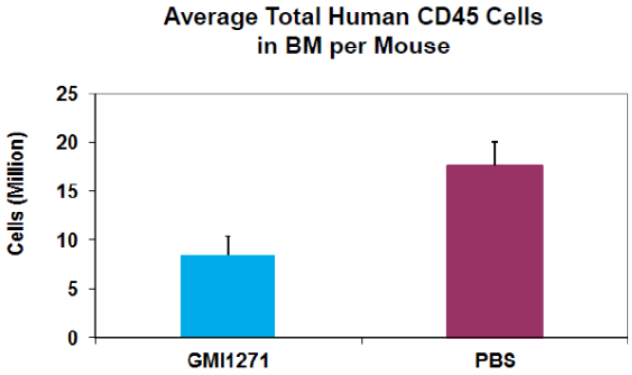
From this data, we believe there is a rationale for moving into human testing. Furthermore, based on the positive Phase I/II results with Mozobil in relapsed/refractory AML, we believe there is now clinical evidence supporting GMI-1271's mechanism-of-action in haematological cancers.

FIGURE 8
GMI-1271 Can Reverse E-Selectin-mediated Chemo-Resistance in Cancer



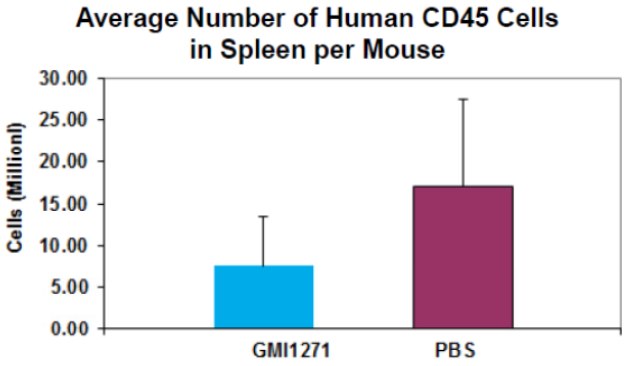
Source: Company reports

FIGURE 9
GMI-1271 Mobilizes Leukemia Cells Out of Bone Marrow



Source: Company reports

FIGURE 10
GMI-1271 Mobilize Leukemia Cells Out of Spleen



Source: Company reports

There is limited competition on the horizon for AML

Most clinical testing for the treatment of AML has either involved the use of different chemotherapy regimens or the development of targeted therapies. Below we highlight two drugs which have shown some promising results to-date.

Mozobil (Sanofi)

A CXCR4/CXCL12 inhibitor, Mozobil was initially approved in 2008 by the U.S. FDA for the mobilization of hematopoietic stem cells from the bone marrow to the blood stream for collection and subsequent autologous stem cell transplantation. Due to its mechanism of action, we believe this is the most relevant competitive threat to GMI-1271 since it is currently being tested in the setting of chemotherapy sensitization.

Results from a Phase I/II trial in relapsed/refractory AML were presented in 2012 in the journal *Blood*; the study tested the combination of Mozobil plus mitoxantrone, etoposide and cytarabine in 52 patients. After the completion of a Phase I dose-escalation portion, 46

patients were treated with 0.24mg/kg/day of Mozobil plus chemotherapy, leading to a complete remission rate of 46% and (consistent with the mechanism of action) a 2-fold increase in the level of mobilization of leukemic blasts into the circulation.

More recently, at the 2013 ASH meeting, data was presented from a combination study of Mozobil plus decitabine in newly diagnosed elderly patients with AML. Overall, 69 patients were treated with a median age of 73 years. The overall response rate was 43% with median response duration of 4.5 months. Median overall survival for the responders was 18 months, while for the non-responders was only 5 months. From the standpoint of safety, cytopenias and infections were the most common adverse events, however at the highest dose of Mozobil (810 ug/kg) there were 14 patients (56%) who experienced reversible, treatment-associated insomnia.

Currently there are a number of Phase I/II studies of Mozobil as a chemotherapy sensitizing agent in patients with relapsed/refractory AML and other haematological malignancies. Additionally, Sanofi has not provided any additional guidance with respect to a potential pivotal study.

Quizartinib (Ambit Biosciences)

A FMS-like tyrosine kinase-3 (FLT3) inhibitor, quizaritinib recently completed Phase II testing in the setting of relapsed/refractory AML and should enter Phase III in early 2014. The rationale behind the use of this drug in this disease setting is based on research showing that up to 30% of AML patients have activating mutations in the FLT3 receptor tyrosine kinase, which typically confers a more aggressive phenotype.

Results from a Phase II study were presented at the American Society of Clinical Oncology (ASCO) meeting earlier this year. A total of 110 patients with FLT3-ITD mutations and age 60 or older who had relapsed within a year of first-line thereapy were treated with single-agent quizartinib. Overall, 57% of patients achieved a complete remission, leading to a median overall survival of 25.3 weeks in the treated population and 22.7 weeks for patients age 70 and older. Additionally, 15% of patients remained alive for more than a year.

AML represents a multi-billion-dollar opportunity

The National Cancer Institute (NCI) estimates that approximately 14,590 new cases of adult AML will be diagnosed in 2013, with 10,370 dying of the disease and 36,000 patients currently living with a history of AML. Using the current standard-of-care, 60%-70% of adults with AML will achieve a complete response, with about 45% of those patients surviving for 3 years or more; the typical cure rate amongst all adults with AML is 25%.

If successful, we believe the most likely market position for GM-1271 will be in the setting of relapsed/refractory AML and in newly-diagnosed elderly patients (>75 years of age) who cannot tolerate chemotherapy. We estimate that of the people currently living with a history of AML in the U.S., 10% (3,600 patients) will relapse every year and would qualify for treatment with GMI-1271. Additionally, we estimate that 40% of newly-diagnosed patients may be too old to be treated with chemotherapy (>75 years of age) and also qualify for GMI-1271. In total, we estimate the current market to be approximately 24,000 patients in the U.S., with a slightly greater population in the EU. Using a price tag of roughly $40,000 per patient in the U.S. and $30,000 in the EU, we estimate a combined market opportunity of roughly $2.0bn dollars, from which we expect peak sales of $475mn in 2033 (Figure 11).

FIGURE 11
U.S. and EU AML Market

United States	**2020**	**2033**
AML Prevalence	37,653	40,175
Yearly Relapse Rate	10%	10%
AML Incidence	15,176	17,050
% Elderly Patients (>75 years of age)	40%	40%
Total Relapsed/Refractory and Elderly AML	24,897	26,908
GMI-1271 Penetration	2%	20%
Price	$40,000	$58,741
U.S. Revenue (thousands)	**$19,917**	**$316,118**
European Union		
AML Prevalence	31,377	33,479
% Relapsed/Refractory	10%	10%
AML Incidence	21,680	24,358
% Elderly Patients (>75 years of age)	40%	40%
Total Relapsed/Refractory and Elderly AML	24,360	26,483
GMI-1271 Penetration		20%
Price		$30,000
E.U. Revenue (thousands)		**$158,896**
Worldwide sales of GMI-1271	**$19,917**	**$475,014**

Source: Barclays Research, www.cancer.gov.

Key Risks are Largely Developmental and Typical of Biotech

Developmental/Marketing Risk

As is the case with most biotechnology companies, there is risk that GMI-1070 and GMI-1271 may not prove successful in clinical studies. With respect to GMI-1070, one of the biggest risks is the failure to recruit the desired number of patients, since it can be inherently difficult to recruit patients onto a study while they are experiencing a VOC. Consequently, if partner Pfizer decides to shrink the enrolment numbers the statistical powering of the study may be compromised. With respect to GMI-1271, the agent has yet to enter clinical testing so we do not know how this drug will perform in humans. GMI-1070 on the other hand has been through Phase II testing, however while the data was positive across multiple metrics the results were not statistically-significant. Furthermore, if clinical testing does prove successful, there are various commercialization risks associated with launching a drug, particularly with GMI-1271 since there may not be any help from an established partner such as Pfizer.

Competitive Risk

While there are several companies developing therapies for VOC and AML there are also several low-cost generic drugs currently on the market. In the setting of VOC prevention, hydroxyurea has been approved and used for decades and when taken consistently can prevent VOCs and improve survival for patients with SCD. With respect to AML, a number of chemotherapies are being used by physicians, which have associated toxicities. As a result, GMI-1070 and GMI-1271 may have issues displacing treatments which some physicians are comfortable using.

Capital/Financial Risk

As is the case with most early-stage biotechnology companies, significant amounts of capital are necessary for R&D and SG&A before a product goes on the market and generates enough revenue to generate a net gain. While the recent IPO has provided it with enough capital to last at least another 12 months (along with the potential for near-term milestone payments from Pfizer), GlycoMimetics is not likely to become profitable before it exhausts its current cash position. As a result, the company may end up raising money through an additional offering equity or debt, which may impact current or future stockholders.

Management Team Includes Several Industry Experts

Rachel K. King (CEO)

One of the co-founder of GlycoMimetics, Mrs. King brings a wealth of experience across multiple aspects of the biotechnology industry. Before GlycoMimetics, Mrs. King was an executive-in-residence at New Enterprise Associates after serving as a senior VP at Novartis. Before Novartis, Mrs. King spent 10 years with Genetic Therapy, Inc. where she was part of the executive team, helping the company successfully file an IPO and subsequently get acquired by Novartis (operated as a wholly-owned subsidiary where Mrs. King served as the CEO). After receiving her BA from Dartmouth College and MBA from Harvard Business School, Mrs. King worked at ALZA Corporation and Bain & Company.

John Magnani, PhD (VP and CSO)

An expert in glycobiology, Dr. Magnani has discovered and elucidated the function of numerous carbohydrate epitopes, selectins and tumor antigens (such as CA19-9). Prior to co-founding GlycoMimetics with Mrs. King, Dr. Magnani founded GlycoTech Corporation and served as its President and CEO. Separately, Dr. Magnani previously also co-founded the U.S. subsidiary of BioCarb, becoming its VP of Research. After receiving his PhD from Princeton University, Dr. Magnani worked at the National Institutes of Health (NIH) for 10 years as a research chemist.

Helen Thackray, MD, FAAP (VP of Clinical Development and CMO)

Joining the company in 2006, Dr. Thackray brings to the company a vast amount of experience in the design and execution of clinical trials for both small molecules and biologics. Along with her position at GlycoMimetics, where she has overseen the clinical development of GMI-1070 in SCD, Dr. Thackray is a board-certified paediatrician and an Assistant Clinical Professor of Pediatrics at the George Washington University School of Medicine. Before joining GlycoMimetics, Dr. Thackray was VP of Clinical Product Development at Biosynexus, Inc. where she oversaw the clinical development of a biologic product for hospitalized neonates. Before entering the private sector, Dr. Thackray received her BS from Stanford University, MD from George Washington University School of Medicine, and completed a paediatrics residency at Children's National Medical Center (followed by a year as a Pediatric Chief Resident and Adjunct Instructor in Pediatrics). Lastly, Dr. Thackray has served as a Medical Genetics Fellow at the National Human Genome Research Institute of the NIH.

Brian Hahn (CFO)

Joining GlycoMimetics in 2010, Mr. Hahn comes with 15 years of experience including Executive Director of Finance at MiddleBrook Pharmaceuticals, senior accountant with Bering Truck Corporation, and auditor for the Bank of Clarke County. Over the last 10 years Mr. Hahn has helped life science companies from their early stages all the way to drug launch and developed strategic plans, create business models and establish various accounting and auditing systems. Currently serving as Chair for the Financial Executive Committee (FEF) of the Tech Council of Maryland as well as a member of the Steering

Committee of the DC chapter of the Association for Bio Financial Officers, Mr. Hahn received his BBA in Accounting from Shenandoah University and MBA from the University of Maryland.

ANALYST(S) CERTIFICATION(S):

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Primary Stocks (Ticker, Date, Price)

GlycoMimetics Inc. (GLYC, 03-Feb-2014, USD 10.01), Overweight/Neutral, A/C/D/J/L

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GlycoMimetics Inc. (GLYC)	Halozyme Therapeutics Inc. (HALO)	Idenix Pharmaceuticals (IDIX)
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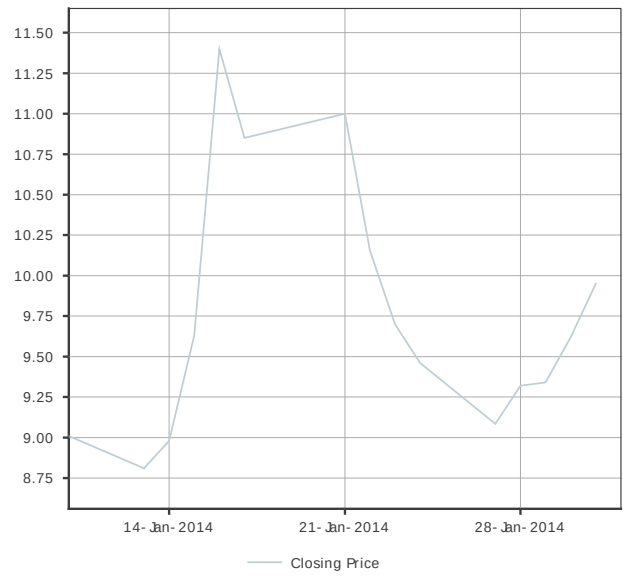
GlycoMimetics Inc. (GLYC)

	Stock Rating	Industry View
USD 10.01 (03-Feb-2014)	**OVERWEIGHT**	**NEUTRAL**

Rating and Price Target Chart - USD (as of 03-Feb-2014) **Currency=USD**



Date	Closing Price	Rating	Adjusted Price Target

Source: IDC, Barclays Research

Link to Barclays Live for interactive charting

A: Barclays Bank PLC and/or an affiliate has been lead manager or co-lead manager of a publicly disclosed offer of securities of GlycoMimetics Inc. in the previous 12 months.

C: Barclays Bank PLC and/or an affiliate is a market-maker and/or liquidity provider in equity securities issued by GlycoMimetics Inc. or one of its affiliates.

D: Barclays Bank PLC and/or an affiliate has received compensation for investment banking services from GlycoMimetics Inc. in the past 12 months.

J: Barclays Bank PLC and/or an affiliate trades regularly in the securities of GlycoMimetics Inc..

L: GlycoMimetics Inc. is, or during the past 12 months has been, an investment banking client of Barclays Bank PLC and/or an affiliate.

Valuation Methodology: We value GlycoMimetics using a probability-adjusted net present value (NPV) model. We assign $9/share for the company's GMI-1070 program (71% probability of reaching the market) and $3/share for the company's GMI-1271 program (11% probability of reaching the market), both of which are discounted 10%.Additionally, we attribute roughly $3/share in net cash. As a result, we come up with a price target of $15/share.

Risks which May Impede the Achievement of the Barclays Research Price Target: As with most developmental-stage biotechnology companies, the primary risks are 1) running out of necessary capital to fund operations and being unable to obtain capital through subsequent financings, 2) failure in demonstrating clinical benefits for their two key pipeline products, 3) competition from currently-approved or other late-stage clinical compounds.

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