Forward-Looking Statements

To the extent that statements contained in this presentation are not descriptions of historical facts regarding GlycoMimetics, Inc. ("GlycoMimetics," "we," "us," or "our"), they are forward-looking statements reflecting management’s current beliefs and expectations. Forward-looking statements are subject to known and unknown risks, uncertainties, and other factors that may cause our or our industry’s actual results, levels of activity, performance, or achievements to be materially different from those anticipated by such statements. You can identify forward-looking statements by terminology such as “may,” “will,” “should,” “expects,” “plans,” “anticipates,” “believes,” “estimates,” “predicts,” “potential,” “intends,” or “continue,” or the negative of these terms or other comparable terminology. Forward-looking statements contained in this presentation include, but are not limited to, statements regarding: (i) the expected timing of completion and data readout of the ongoing Phase 3 clinical trial of Rivipansel by Pfizer Inc. (ii) the timing of receipt of clinical data for our drug candidates; (iii) our expectations regarding the potential safety, efficacy, or clinical utility of our drug candidates; (iv) the size of patient populations targeted by drug candidates we or our collaborators develop and market adoption of our potential drugs by physicians and patients; (v) the likelihood and timing of regulatory filings and approvals; and (vi) our cash needs and expected cash runway, as well as potential royalties and milestone payments under license and collaboration agreements.

Various factors may cause differences between our expectations and actual results, including unexpected safety or efficacy data, unexpected side effects observed during preclinical studies or in clinical trials, lower than expected enrollment rates in clinical trials, changes in expected or existing competition, changes in the regulatory environment for our drug candidates, failure of our collaborators to support or advance our collaborations or drug candidates, our need for future capital, the inability to protect our intellectual property, and the risk that we become a party to unexpected litigation or other disputes. For a further description of the risks associated with forward-looking statements, as well as other risks facing GlycoMimetics, please see the risk factors described in the Company’s Annual Report on Form 10-K filed with the U.S. Securities and Exchange Commission on March 6, 2019, as well as other reports we file with the U.S. Securities and Exchange Commission from time to time, including those factors discussed under the caption “Risk Factors” in such filings. Forward-looking statements speak only as of the date of this presentation, and GlycoMimetics undertakes no obligation to update or revise these statements, except as may be required by law.
Late-Stage Clinical Pipeline with Upcoming Catalysts

- **Enrolling uproleselan Phase 3: R/R acute myeloid leukemia**
  - Breakthrough Therapy Designation granted in May 2017
  - Targeting top-line data 2021
  - IP through 2032 in US, EU and Japan
  - ASH abstracts support evolving biomarker data; strongly supports targeting this mechanism

- **Uproleselan market expansion via consortium-funded trial: NCI**
  - Strong, independent KOL support in newly-diagnosed AML setting
  - First patient dosed 2Q19; active enrollment and significant engagement of sites

- **Rivipansel Phase 3; Pfizer partnership; Disappointment in vaso-occlusive crisis**
  - Evaluating underlying data; presentation at future scientific meeting planned

- **Strong balance sheet; funded through multiple catalysts / milestones: Cash Q3 ’19 $170.9 million**

- **Well positioned to drive value creation**
  - Pipeline of potentially ‘game-changing’ therapeutic opportunities
  - Novel glycobiology/chemistry platform
Uproleselan (GMI-1271)

Breakthrough Therapy Designation

Significant Market Opportunity
Significant Unmet Need in AML
Highest Incidence, Lowest 5-yr Survival of all Leukemias

Estimated New Cases (2019)

21,450 New Cases

All Other Leukemias

5-Year Survival (2008 – 2014)

Survival Rate %

0 10 20 30 40 50 60 70 80 90

CML CLL ALL AML

67.6 84.2 68.1 27.4

SEER 2019 Statistics

GlycoMimetics, Inc.
E-selectin:
- Is constitutively expressed in the bone marrow microvasculature, levels up-regulated in AML
- Binds to the E-selectin ligand expressed on AML cells to activate pathways for chemoresistance

In preclinical models:
- Prevents trafficking of tumor cells to the bone marrow
- Disrupts cell adhesion-mediated drug resistance (CAMDR) within bone marrow microenvironment
- Inhibits activation of cancer survival pathways (e.g. NF-κB)
- Protects normal HSCs by enhancing quiescence and ability for self-renewal
- Reduces chemotherapy-associated toxicity (e.g. severe mucositis)

Uproleselan disrupts the interaction between AML cells and the bone marrow microenvironment
Uproleselan Product Positioning in AML

Position uproleselan as potential foundational backbone treatment that:
- Deepens achievement / depth of remission
- Extends overall survival
- Mitigates chemotherapy-related toxicity

~21,000 Patients\(^1\)
(Estimated New Cases in USA)

"Fit" patients eligible for intensive therapy

- GMI-Sponsored Phase 3
  Relapsed / Refractory AML
  Combination of Uproleselan + MEC/FAI
  ~8,500 Patients/Year

- NCI-Sponsored Phase 2/3
  Newly Diagnosed, Elderly AML
  Combination of Uproleselan + 7&3

"Unfit"

- HOVON
  Discontinued

\(^1\) SEER 2019
Final Efficacy/Correlative Results: Uproleselan Phase 1/2 Oral Presentation at ASH 2018

- **R/R AML Cohort**: 41% CR/CRi; 8.8 mos. Median Overall Survival
- **Newly Diagnosed AML Cohort**: 72% CR/CRi; 9.2 mos. Event Free Survival
- >50% of evaluable patients archived a stringent MRD-negativity
  - Appears to enhance depth of response
- **E-selectin ligand expression**
  - Detectable in every patient tested; target biologically relevant
  - Higher in those R/R patients achieving CR/CRi, MRD- and prolonged median OS

Data supports biological/clinical activity and late-stage registration program
Sialyl Le$^x$ (E-Selectin Ligand) Expression is Associated with Aggressive Tumors and Poor Clinical Outcomes

“In conclusion, our meta-analysis showed that a high level of sLeX [E-selectin ligand] expression was significantly associated with lymphatic invasion, venous invasion, deep invasion, lymph node metastasis, distant metastasis, tumor stage, tumor recurrence, and OS in cancer”

Twenty nine (29) cancer studies published between 1993 and 2003 were used for meta-analysis

OncoTargets and Therapy 9: 3113-3125 (2016)
E-Selectin Ligand Expression On Leukemic Blasts Associated with Poor Prognosis in Patients with AML

Independent Data from 89 Serially Acquired AML Patient Samples

- Mean fluorescence intensity of E-selectin-Fc binding
  - 4-fold higher for relapsed/refractory patients than for newly diagnosed patients (p=0.0026)

- Percent E-selectin-Fc binding
  - higher in patients with unfavorable than favorable/intermediate risk (p=0.019)

- Expression of E-selectin ligands by leukemic stem cells
  - tightly correlated with expression in leukemic blasts in the same patient

Higher E-selectin ligand expression associated with chemo resistance / AML persistence
R/R AML Patients with High E-Selectin Ligand Expression Had Improved Clinical Outcomes When Treated with Uproleselan

<table>
<thead>
<tr>
<th>High E-selectin ligand (≥10%)</th>
<th>Low E-selectin ligand (&lt;10%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong># of Patients</strong></td>
<td><strong>Outcomes</strong></td>
</tr>
<tr>
<td>22</td>
<td>CR/CRi rate</td>
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<tr>
<td>14</td>
<td>45%</td>
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<tr>
<td></td>
<td>Median overall survival</td>
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<tr>
<td></td>
<td>12.7 months</td>
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<td>5.2 months</td>
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</tbody>
</table>

Improved overall survival, in patients who would otherwise be expected to do worse, suggests that uproleselan is exerting intended biologic activity.

* Data on E-sel ligand was available for 36 patients at study entry.
Breadth and Depth of Portfolio Showcased at ASH 2019

Publication Number: 2690
TITLE: High E-Selectin Ligand Expression Contributes to Chemotherapy-Resistance in Poor Risk Relapsed and Refractory (R/R) Acute Myeloid Leukemia (AML) Patients and Can be Overcome with the Addition of Uproleselan
Session Name: 617. Acute Myeloid Leukemia: Biology, Cytogenetics, and Molecular Markers in Diagnosis and Prognosis

Publication Number: 2650
TITLE: A Double-Blind, Placebo-Controlled, Phase 3 Registration Trial to Evaluate the Efficacy of Uproleselan (GMI-1271) with Standard Salvage Chemotherapy in Patients with Relapsed/Refractory (R/R) Acute Myeloid Leukemia
Session Name: 616. Acute Myeloid Leukemia: Novel Therapy, excluding Transplantation

Publication Number: 1366
TITLE: A Randomized Phase 2/3 Study of Conventional Chemotherapy +/- Uproleselan (GMI-1271) in Older Adults with Acute Myeloid Leukemia Receiving Intensive Induction Chemotherapy
Session Name: 616. Acute Myeloid Leukemia: Novel Therapy, excluding Transplantation

Publication Number: 3802
TITLE: Synergistic Targeting of BTK and E-Selectin/CXCR4 in the Microenvironment of Mantle Cell Lymphomas
Session Name: 605. Molecular Pharmacology, Drug Resistance—Lymphoid and Other Diseases

Publication Number: 907
TITLE: CD162 Is a Key E-Selectin Receptor Promoting Acute Myeloid Leukemia Chemo-Resistance in the Bone Marrow Niche
Session Name: 604. Molecular Pharmacology and Drug Resistance in Myeloid Diseases: The Impact of Cell-Cell Interactions, Surface Antigens, and Mitochondria

Publication Number: 2657
TITLE: Blocking Vascular Niche E-Selectin Dampens AML Stem Cell Regeneration/Survival Potential In Vivo By Inhibiting MAPK/ERK and PI3K/AKT Signaling Pathways
Session Name: 616. Acute Myeloid Leukemia: Novel Therapy, excluding Transplantation

Publication Number: 3772
TITLE: Transcriptome Profiling of Glycosylation Genes Defines Correlation with E-selectin Ligand Expression and Clinical Outcome
Session Name: 602. Disordered Gene Expression in Hematologic Malignancy, including Disordered Epigenetic Regulation
Uproleselan Relapsed / Refractory AML Phase 3 Study Design

Key Eligibility Criteria
- ≥18 and ≤75 years in age
- Either primary refractory or relapsed (first or second relapse) AML
- Eligible for intensive salvage treatment
- ≤1 prior HSCT

Randomize 1:1

Induction (1 Cycle)
- Upro plus MEC or FAI (n=190)
- Placebo plus MEC or FAI (n=190)

Consolidation (Up to 3 Cycles)
- Upro plus HiDAC or IDAC
- Placebo plus HiDAC or IDAC

Follow-Up for Overall Survival

1:1 Randomization (stratified by age, disease status and backbone chemo)

MEC: Mitoxantrone, etoposide and cytarabine
FAI: Fludarabine, cytarabine and idarubicin
HiDAC/IDAC: High-dose or Intermediate-dose cytarabine

Phase III Primary Endpoint: Overall Survival, defined as the time of randomization until death from any cause – analysis of OS will not be censored for transplant
Key Eligibility Criteria
- ≥ 60 years in age
- AML and fit for 7+3
  - Includes sAML
  - Excludes FLT3+

NCI Phase 2/3 Study Design – Frontline “Fit” AML

**Induction**
(1 Cycle)
- Upro plus 7+3 (n=125)
- 7+3 (n=125)

**Consolidation**
(Up to 3 Cycles)
- Upro plus IDAC
- IDAC

**Randomize 1:1**

**Interim Analysis:** 250 patients (shown in diagram)
- Interim Go/No-Go: Event-free survival (EFS) - defined as the time from the date of registration/randomization to the first of failure to achieve a remission during induction, relapse, or death due to any cause

**Phase 3 Primary Endpoint:** Overall survival (OS) – measured from the date of registration/randomization to death from any cause
- 90% power to detect median OS HR 0.75
# Historical Benchmarks - What Are We Trying to Beat?

<table>
<thead>
<tr>
<th>Population</th>
<th>Registration Program Primary Outcome Measure</th>
<th>Uproleselan Phase 1/2 Results</th>
<th>Historical Comparator's</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relapsed / Refractory AML</td>
<td>Overall Survival (months)</td>
<td>8.8 months</td>
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<td>5.4 months (MEC)</td>
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<td></td>
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<td>Feldman et al (2005)</td>
<td>Lintuzumab + MEC vs. MEC</td>
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<td></td>
<td></td>
<td>5.2 months (MEC)</td>
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<td>Roboz et al (2014)</td>
<td>Elcytarabine vs. Inv. choice</td>
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<td></td>
<td></td>
<td>3.4 months (Inv. choice)</td>
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<tr>
<td>Newly Dx “Fit” for Intensive Chemo AML</td>
<td>Event-Free Survival (months)</td>
<td>9.2 months</td>
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<td></td>
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<td>Lowenberg et al (2009)</td>
<td>7+3</td>
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<td>~6.5 months</td>
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<td>Lancet et al (2014)</td>
<td>Vyxeos vs. 7+3</td>
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<td>2.0 months (7+3)</td>
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</table>
GMI-1359
E-Selectin / CXCR4 Antagonist
Solid Tumor Indications
GMI-1359: Small molecule, dual inhibitor against E-selectin and CXCR4

- Disrupts tumor–stromal interactions
- Inhibits cell survival/activation pathways
- Prevent trafficking / mobilizes dormant cancer cells from protective niches to make them more susceptible to lysis by chemotherapy

Complementary pathways relevant for tumors that originate/metastasize to bone

### Table 1: Biomarkers used to date for the detection of circulating cancer stem cells in different cancer types

<table>
<thead>
<tr>
<th>Tumor type (Reference)</th>
<th>Cell surface markers on circulating cancer stem cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancreatic (39,71,72)</td>
<td>CD133, CD44, CD26, CXCR4, c-Met, ALDH1, ABCG2</td>
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<tr>
<td>Breast (28,30,73-77)</td>
<td>CD44, ANTXR1, ALDH1, CXCR4, ALDH1</td>
</tr>
<tr>
<td>Colorectal (43,78,79)</td>
<td>CD133, CD44, CD44v6, CXCR4, CD26</td>
</tr>
<tr>
<td>Gastric cancer (80)</td>
<td>CD133, MMP-13</td>
</tr>
<tr>
<td>Glioblastoma (6,81)</td>
<td>CXCR4, ABCG2, CD133, ALDH1</td>
</tr>
<tr>
<td>Lung (82,83)</td>
<td>CD133</td>
</tr>
<tr>
<td>Osteosarcoma (84,85)</td>
<td>ABCG2</td>
</tr>
<tr>
<td>Retinoblastoma (86)</td>
<td>c-Met</td>
</tr>
<tr>
<td>Head and neck cancer (87)</td>
<td>CD133</td>
</tr>
<tr>
<td>Ovarian (88)</td>
<td></td>
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</tbody>
</table>

**Validated Biomarkers for Detection of Circulating Cancer Cells**

- **Major E-selectin ligands**
  - CD133
  - CD44
  - CD26
  - CXCR4
  - c-Met
  - ALDH1
  - ABCG2

- **CXCR4 ligand**
  - ANTXR1
The E-selectin / CXCR4 Axis Plays a Critical Role in the Progression of Breast Cancer

1. Cancer cells use a specific molecule to enter the bone marrow. Disabling that molecule, E-selectin, researchers were able to block cancer cells from getting inside.

2. Another molecule anchored the cancer cell inside the bone marrow.

3. Scientists also discovered a way to eject those cancer cells, sending them back into circulation where they may be more vulnerable to the immune system or cancer treatment.

Region of bone marrow where breast cancer cells hide.

E-selectin antagonist – blocks tumor cells from entering niches

CXRC4 antagonist – ejects cells from protective niches

Adapted from Duke Health
GMI-1359 Phase 1b Dose Escalation/
Proof-of-Principle Program

- Lead Investigative Site - Duke University Medical Center
- Single/multiple ascending dose within each patient – 3.5, 5.0 & 7.0 mg/kg
- Range 6-12 patients with metastatic, HR+, stable/minimally progressive breast cancer
- Endpoints – Safety, PK & PD
- Data by YE 2020

Possible Clinical Relevance
- Mobilization of Circulating Tumor Cells
  - High-risk breast cancer, including inflammatory breast cancer
  - Other solid tumors (Osteosarcoma)
- Mobilization of Primitive HSCs
  - Transplant (Auto, Allo)
  - Ex-vivo gene editing
- Mobilization of Marrow Infiltrating Lymphocytes
  - Combinations with checkpoint inhibitors

Data read-out expected in second half 2020
Positioned for Success
Pipeline, Progress, Catalysts
A Portfolio of Exciting Product Candidates

### Wholly Owned Proprietary Programs

<table>
<thead>
<tr>
<th>Compound</th>
<th>Therapeutic Area</th>
<th>Discovery</th>
<th>Pre-Clin</th>
<th>Ph 1</th>
<th>Ph 2</th>
<th>Ph 3</th>
<th>Registration</th>
<th>Partner</th>
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<tbody>
<tr>
<td><strong>Selectins</strong></td>
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<tr>
<td>Uproleselan (GMI-1271) and GMI-1687</td>
<td>Relapsed / Refractory AML</td>
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<td>Newly Diagnosed “Fit” AML</td>
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<td>Hem-Onco &amp; Inflammation</td>
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<tr>
<td>GMI-1359</td>
<td>Various Tumor Types</td>
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<td><strong>Galactins</strong></td>
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<tr>
<td>GMI-1757</td>
<td>Hem-Onco &amp; Inflammation</td>
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<tr>
<td>[Galactin-3/E-selectin]</td>
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<tr>
<td>Galectin-3 Inhibitors</td>
<td>Fibrosis &amp; Oncology</td>
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### Partnered Programs

<table>
<thead>
<tr>
<th>Compound</th>
<th>Therapeutic Area</th>
<th>Discovery</th>
<th>Pre-Clin</th>
<th>Ph 1</th>
<th>Ph 2</th>
<th>Ph 3</th>
<th>Registration</th>
<th>Partner</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rivipansel</td>
<td>SCD Vaso-occlusive Crisis</td>
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<td>Pfizer</td>
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</tbody>
</table>
## Recent/Upcoming News Flow

- First patient enrolled, Uproleselan R/R AML pivotal trial
- First patient enrolled, Uproleselan Newly Diagnosed pivotal trial
- Clinical trial planning for GMI-1359, dual function inhibitor
- Rivipansel Phase 3 top-line readout
- ASH abstracts released/presented
- GMI-1359 first patient enrolled in breast cancer P1b trial 4Q ‘19
- GMI-1359 data read-out from breast cancer trial YE ‘20
- Topline data, R/R pivotal trial 2021
## Investment Opportunity – Nasdaq: GLYC

### Advancing Pipeline
- Uproleselan: BTD from FDA for R/R AML; Phase 3 read-out targeted 2021
- GMI-1359: Simultaneous blockade of CXCR4 & E-Selectin targets enhancing anti-tumor immune response, Proof-of-principle trial to be initiated in 2019

### Significant Revenue Opportunities
- Uproleselan: > 44,000 AML patients in 7 major markets; expansion potential into other hematologic malignancies
- GMI-1359: Targeting solid tumors with high propensity to metastasize to the bone (e.g. breast, osteosarcoma)

### Strong Investment Base
- Top-tier biotech investors
- Cash balance of ~$170.9 million as of September 30, 2019; runway into ‘22

### Experienced Team
- Pioneers in the field of glycobiology and small-molecule, therapeutic “mimetics”
- Relationships with leading KOLs and oncology networks