PIONEERING NEW MEDICINES
for the treatment of malignant and life-threatening diseases of the bone marrow
August 2021
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Investment Highlights

<table>
<thead>
<tr>
<th><strong>LSD1 Inhibition</strong></th>
<th>Novel mechanism of action and potential first-in-class treatment for heme malignancies, potential to achieve disease modification</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bomedemstat</strong></td>
<td>Internally-discovered small molecule product candidate to inhibit LSD1; promising Phase 1 and 2 safety and tolerability with &gt;150 patients treated to date</td>
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<tr>
<td><strong>Robust Pipeline</strong></td>
<td>Phase 2 in Essential Thrombocythemia (ET) and Myelofibrosis (MF) Preclinical programs in hemoglobinopathies and solid tumors</td>
</tr>
<tr>
<td><strong>Untapped Market</strong></td>
<td>ET: Poorly tolerated existing treatments, large addressable market MF: No disease modifying therapy, high unmet need</td>
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<tr>
<td><strong>Upcoming News Flow</strong></td>
<td>2021: Phase 2 data updates in both ET and MF</td>
</tr>
<tr>
<td><strong>Strong Investors</strong></td>
<td>Raised $340M to date from leading financial and strategic investors</td>
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<tr>
<td><strong>Leadership Team</strong></td>
<td>Experienced in drug development and successful exits</td>
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</table>
# Leadership

<table>
<thead>
<tr>
<th>Name</th>
<th>Title</th>
<th>Company</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hugh Y. Rienhoff, Jr., MD</td>
<td>Founder, CEO</td>
<td>FerroKin BioSciences, DNA Sciences, WebMD, Wilson Therapeutics, Aurora Biosciences, NEA</td>
</tr>
<tr>
<td>Laura Eichorn</td>
<td>Interim CFO, COO</td>
<td>FerroKin BioSciences, DNA Sciences, NEA</td>
</tr>
<tr>
<td>Wan-Jen Hong, MD</td>
<td>CMO</td>
<td>Genentech, Stanford Medicine</td>
</tr>
<tr>
<td>Jennifer Peppe</td>
<td>SVP, Clinical Operations</td>
<td>FerroKin BioSciences, Genzyme, Dana-Farber Cancer Institute</td>
</tr>
<tr>
<td>Amy Tapper, PhD</td>
<td>SVP, Non-clinical and CMC</td>
<td>FerroKin BioSciences, Peptimmune, Genzyme, Momenta</td>
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</table>
### Our Pipeline

<table>
<thead>
<tr>
<th></th>
<th>DISCOVERY</th>
<th>IND ENABLING</th>
<th>PHASE 1</th>
<th>PHASE 2</th>
<th>PHASE 3</th>
<th>NEXT ANTICIPATED MILESTONE(S)</th>
</tr>
</thead>
</table>
| Essential Thrombocythemia  
(bomedemstat)       | Enrollment | Enrollment   | Enrollment | Enrolling | Data updates 2021 and 2022; Phase 3 FPI |
| Myelofibrosis     
(bomedemstat)       | Enrollment | Enrollment   | Enrollment | Enrollment | Data updates 2021 and 2022; FPI 2021; Data updates 2022 |
| Myelofibrosis*    
(bomedemstat + ruxolitinib) | Enrollment | Enrollment   | Enrollment | Enrolling | Enrolling |
| Polycythemia Vera* 
(bomedemstat)       | Enrollment | Enrollment   | Enrollment | Enrolling | Enrolling |
| Hemoglobinopathies  
(NCE)                | Lead Optimization | Enrollment | Enrollment | Enrolling | Nominate IND candidate |
| Solid Tumors       
(NCE, combination)  | Lead Discovery | Enrollment | Enrollment | Enrolling | Nominate IND candidate |

*Investigator Sponsored Trial

FPI: First patient dosed. NCE: New chemical entity.
Myeloproliferative Neoplasms and Bomedemstat
A new mutation generally in JAK2 (a kinase), MPL (a receptor) or CALR (an MPL chaperone). Results in constitutive activation of the JAK-STAT hematopoietic growth signals.

Myelofibrosis (MF)
Inflammation and progressive bone marrow scarring
Strong Rationale for LSD1 Inhibition in MPNs

- LSD1 regulates the proliferation of blood stem cells and is also essential for their differentiation into mature megakaryocytes and granulocytes.
- Inhibiting LSD1 has been shown to reduce the hallmark symptoms of MPNs as well as lower the number of cells with the mutations that drive these diseases.
Bomedemstat: A Novel LSD1 Inhibitor for the Treatment of MPNs

- Discovered by Imago; patent life until at least 2034
- Addresses limitations of other LSD1 inhibitors, e.g., minimize crossing the blood-brain barrier
- In 3 mouse models of MPNs, LSD1 inhibition reduces key hallmarks of disease; is disease modifying
- Demonstrated activity and well-tolerated in >150 patients treated to date
- Platelet count serves as a biomarker of bomedemstat activity on megakaryocytes; allows for personalized dosing
- FDA Orphan & Fast Track designation for ET and MF
- EMA Orphan & PRIME designation for MF
- EMA Orphan designation for ET

Survival benefit for \( \text{Jak}^{2\,\text{V617F}} \, \text{mice} \) treated with bomedemstat

![Survival benefit](image-url)
Bomedemstat in ET
Essential Thrombocythemia (ET)

ET Overview

• Elevated platelets (>450 x 10^9/L)
• US prevalence: ~80-100K
• Significant morbidity and mortality due to thrombotic events and progression to AML

Current Treatments

• Cytoreductive therapy, most often with hydroxyurea (HU), is indicated for all high-risk and many intermediate risk patients (~50% of total)
• We believe there is a significant unmet need for ~20% of treated patients who become intolerant or resistant to HU
• Anagrelide is approved in the US and EU but not widely used due to cardiotoxicities

Bomedemstat initial addressable market: ET patients intolerant of or resistant to HU
Bomedemstat in ET: Phase 2 Trial in High-risk Patients

International trial (~60 patients)

- Open-label, once-daily bomedemstat
- High-risk patients who are intolerant of, or resistant to standard-of-care, most often hydroxyurea

Objectives

- Safety and tolerability
- Reduction of platelet count to ≤400 x 10⁹/L in the absence of thromboembolic or hemorrhagic events and progression
- Reduction in mutant allele frequency (MAF)
Bomedemstat in ET: Patients Achieve Normal Platelet Counts

Proof-of-Principle achieved in Phase 2 Trial

- 10/12 (83%) of patients dosed for >6 weeks achieved a platelet count of <400 x 10^9/L
- Mean reduction of 547 x 10^9/L at Week 12 (N=10)
- 160 adverse events (AEs) of which 78 attributed to bomedemstat by the Investigator
- One serious adverse event (SAE) but deemed unrelated by the investigator

Phase 2: Platelet Counts

Platelet Count (10^9/L)

Weeks on Treatment

Scr = Screening

Source Imago interim, unaudited and ongoing Phase 2 data of Investigational Product
**Bomedemstat in ET: Controls WBCs and Maintains Hb levels**

### White Blood Cells (WBC)

**At Week 12 (N=10)**
- Mean WBC reduction of $3.3 \times 10^9$/L
- 80% (4/5) with elevated WBCs fell to normal ($<10^{10}$/L)

### Hemoglobin (Hb)

*Pt removed due to in/exclusion violation*

Source: Imago interim, unaudited and ongoing Phase 2 data of Investigational Product
ET: epidemiology, treatment guidelines and addressable market

- Limited competition in late-stage clinical development
- Potential upside with 1L use if data demonstrate a greater reduction in thrombotic events compared to hydroxyurea and/or lower rates of progression to MF or AML or superior safety over standards of care

US Prevalence: 80,000 – 100,000

IPSET or Revised IPSET risk stratification

High-risk and many intermediate risk patients (40,000 – 50,000) receive aspirin and a cytoreductive therapy, generally hydroxyurea (HU)

8,000 – 10,000 patients become resistant or intolerant to HU
Bomedemstat in ET: Plans for Registrational Phase 3 Trial

FDA discussion in May (Type C) about Phase 3 design and endpoints

• Phase 3 trial -- a randomized controlled study assessing the superiority ofomedemstat compared to BAT in second-line treatment of ET

• Primary endpoint is the proportion of patients who achieve a normal platelet ($\leq 400 \times 10^9$/L) and WBC ($\leq 10 \times 10^9$/L) count in the absence of hemorrhagic and thromboembolic events and disease progression (MF and AML)

• Phase 2 results suggest Imago can achieve this primary endpoint in a Phase 3 trial

• Positive Phase 3 protocol review by FDA for the registrational trial expected in 2022
Bomedemstat in MF
Myelofibrosis (MF)

**MF Overview**

- Chronic inflammation and progressive bone marrow failure
- US prevalence: ~18-20K
- Median survival from diagnosis: ~5 years
- Significant risk of transformation to AML
- Quality of Life (QOL) degraded by severe constitutional symptoms and splenomegaly

**Ruxolitinib/Fedratinib**

- JAK1/2 inhibitors with QOL benefit
- Toxicities include anemia, thrombocytopenia, neutropenia, immunosuppression, tumors
- We estimate only ~1/3 of MF patients in US receive ruxolitinib therapy – 40% discontinue after 3 years
- The unmet need for all MF patients is sustained improvement of QOL and survival

**Bomedemstat initial addressable market: patients with MF whose disease is not adequately managed by JAK inhibitors**
**Bomedemstat in MF: Phase 2 Trial in Advanced Disease**

**International trial (89 patients, enrollment complete)**
- Open-label, once-daily bomedemstat
- Patients with platelets $\geq 100 \times 10^9/L$ who are resistant to an approved therapy

**Objectives**
- Safety and tolerability
- Symptom reduction (MPN10 TSS)
- Spleen volume reduction (MRI or CT)

**Demographics**
- 50% have received 2+ prior therapies
- 58% high risk by IPSS
- 66% have 2+ mutations associated with MF
- 47% have mutations with high risk for AML transformation
## Reducions in Mutant Allele Frequency (MAF)

<table>
<thead>
<tr>
<th>Pt</th>
<th>MPN driver</th>
<th>Other somatic mutations (of 262 genes sequenced)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>JAK2_V617F (75%)</td>
<td>U2AF1_Q157R (32%)</td>
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<tr>
<td>2</td>
<td>JAK2_V617F (45%)</td>
<td>ZBTB33_Y56S (93%)</td>
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<tr>
<td>3</td>
<td>JAK2_V617F (87%)</td>
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<tr>
<td>4</td>
<td>CALR_52b_del (33%)</td>
<td>ASXL1_1_642X (38%)</td>
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<tr>
<td>5</td>
<td>MPL_W515K (94%)</td>
<td>ASXL1_Q780* (18%)</td>
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<td>6</td>
<td>JAK2_V617F (96%)</td>
<td>TET2_NRN1890 (41%)</td>
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<td>7</td>
<td>CALR_K385NCX (34%)</td>
<td>ASXL1_R693* (22%)</td>
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<td>8</td>
<td>MPL_W515K (50%)</td>
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<td>9</td>
<td>JAK2_V617F (42%)</td>
<td>ASXL1_884X (43%)</td>
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<td>U2AF1_Q157R (45%)</td>
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<td>JAK2_V617F (39%)</td>
<td>CBL_C3965 (51%)</td>
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<td>SF3B1_K700E (26%)</td>
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<td>15</td>
<td>JAK2_V617F (98%)</td>
<td>DNMT3A_V687G (94%)</td>
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<td>16</td>
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<td>18</td>
<td>MPL_W515K (49%)</td>
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<td>JAK2_V617F (69%)</td>
<td>ASXL1_0758* (34%)</td>
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<td>JAK2_V617F (41%)</td>
<td>ASXL1_HHCRR3MA630G (21%)</td>
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<td>22</td>
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<td>ASXL1_AGGS640X (51%)</td>
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<td>23</td>
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<td>25</td>
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<td>ZBTB33_R537H (17%)</td>
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<td>26</td>
<td>CALR_52b_del (41%)</td>
<td>FCGBP_G44655 (25%)</td>
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<td>JAK2_V617F (46%)</td>
<td>MTA2_D288V (15%)</td>
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<td>28</td>
<td>CALR_KKRK374X (84%)</td>
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<td>30</td>
<td>JAK2_V617F (19%)</td>
<td>EZH2_F120X (53%)</td>
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<td>32</td>
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<td>ASXL1_S1044X (35%)</td>
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<tr>
<td>33</td>
<td>JAK2_V617F (97%)</td>
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</tr>
<tr>
<td>34</td>
<td>JAK2_V617F (44%)</td>
<td>ASXL1_L775* (17%)</td>
</tr>
</tbody>
</table>

**15/34** show a **decrease** in MAF in some or all somatic mutations

- **44%** Reduction in all MAFs
- **47%** Reduction in 1 or more MAFs (but not all)
- **9%** Stable
- **Increase in 1 or more MAFs (but not all)**
- **Increase in all MAFs**

**16/34** have **stable** MAF

**3/34** patient have **increased** MAFs

- No new mutations in up to 660 days of follow-up
- No progression to AML
- Elevated baseline blast counts (N=24) improved or resolved in 71% of patients

**These data suggest** **bomedemstat may be a disease-modifying therapy**

Source: Imago interim, unaudited and ongoing Phase 2 data of Investigational Product
Bomedemstat in MF: Total Symptom Score (TSS)

**TSS Changes at 24 weeks**
- 18/20 (90%) had a decrease in TSS at 24 weeks
- 6/20 (30%) had a decrease of ≥50% at 24 weeks

**Patients with TSS >20 at baseline**
- 15/16 (94%) had a decrease in TSS at 24 weeks
- 5/16 (31%) had a decrease of ≥50% at 24 weeks

Source: Imago interim, unaudited and ongoing Phase 2 data of Investigational Product
Bomedemstat in MF: Spleen Volume Reduction

OVERVIEW: Spleen Volume Change at 12/24 weeks

16/18 (89%) had a decrease in spleen volume at 24 weeks

1/18 (6%) had a decrease ≥35% at 24 weeks

Source: Imago interim, unaudited and ongoing Phase 2 data of Investigational Product
Opportunity for Combination Therapy in MF

Preclinical model shows additive activity reducing spleen size without additive thrombocytopenia
Safety and Tolerability Profile of Bomedemstat in MF

Uniquely positioned among all treatments for MPN

- No dose limiting toxicities up to 6 mg/kg
- No genotoxicity or mutagenicity
- No deaths related to study drug
- Out of 1,086 AEs reported, 72 were SAEs
- Ten SAEs attributed by investigators as possibly, probably or definitely related to bomedemstat
- Overall favorable safety and tolerability profile

Source: Imago interim and unaudited Phase 2 data
Pipeline & Upcoming Milestones
## Pipeline & Upcoming Milestones

<table>
<thead>
<tr>
<th>Essential Thrombocythemia (bomedemstat)</th>
<th>DISCOVERY</th>
<th>IND ENABLING</th>
<th>PHASE 1</th>
<th>PHASE 2</th>
<th>PHASE 3</th>
<th>NEXT ANTICIPATED MILESTONE(S)</th>
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<tbody>
<tr>
<td><strong>Enrolling</strong></td>
<td></td>
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<td>Data updates 2021 and 2022; Phase 3 FPI</td>
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<tr>
<td>Myelofibrosis (bomedemstat)</td>
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<td>Enrollment complete</td>
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<td>Myelofibrosis* (bomedemstat + ruxolitinib)</td>
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<td>Enrollment complete</td>
<td>FPI 2021; Data updates 2022</td>
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<td>Polycythemia Vera* (bomedemstat)</td>
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<td></td>
<td>Lead Optimization</td>
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<td>Enrolling</td>
<td>Nominate IND candidate</td>
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<tr>
<td>Hemoglobinopathies (NCE)</td>
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<td>Nominate IND candidate</td>
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<tr>
<td>Solid Tumors (NCE, combination)</td>
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</table>

*Investigator Sponsored Trial

**6/30/21 pro forma cash & equivs: $245 million**

Data presentations anticipated at major hematology meetings in 2021

Regulatory updates, including End-of-Phase 2 meetings, prior to Phase 3 studies

FPI: First patient dosed. NCE: New chemical entity.