OUR VISION: COMBINING TO CURE WITH BEST-IN-CLASS CANCER THERAPIES
Forward-looking Statements/Safe Harbor

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Arcus Has a Broad Portfolio of Molecules, Enabling Differentiated Combination Therapies

**Differentiated Small Molecules**
- **etrumadenant (AB928):** First-in-class dual $A_{2a} \& A_{2b}$ antagonist; generated early evidence of clinical activity in colorectal, prostate and lung cancers
- **quemlicluster (AB680):** First-in-class small-molecule CD73 inhibitor; generated early evidence of clinical activity in pancreatic cancer
- **AB521:** highly potent and selective HIF-2a-inhibitor; first study in healthy volunteers enrolling

**Enabling Antibodies**
- **domvanalimab (AB154):** Anti-TIGIT monoclonal antibody (mAb; Fc silent) – ongoing randomized Phase 2 study ARC-7 in 1L NSCLC; initiated ARC-10, our registrational trial to support both dom + zim and zim monotherapy approvals
- **AB308:** Anti-TIGIT mAb (Fc enabled); Phase 1/1b expansion enrolling
- **zimberelimab (AB122):** anti-PD-1 mAb; recently approved in China for classical Hodgkin Lymphoma (cHL)*

**Next Generation Programs**
- **AB598:** anti-CD39 mAb
- **AxI:** tyrosine kinase small molecule inhibitor

*Gloria Biosciences secured China approval; it holds all rights to zim in China and conducts its activities independent from Arcus.
In Nov. 2021, Gilead Exercised Options to Three Programs and Parties Added a Research Collaboration

• Exercised three of their options, including:
  – domvanalimab and AB308, our anti-TIGIT antibodies
  – etrumadenant, an A2a/2bR antagonist
  – quemliclustat, a small molecule CD73 inhibitor
• Arcus to receive option payments totaling $725mm and share expenses related to each optioned program
  – Transaction is subject to applicable antitrust clearance under the Hart-Scott Rodino Antitrust Improvements Act and other customary closing conditions
• If molecules from these 3 programs are approved, Arcus and Gilead will co-commercialize and equally share profits in the U.S. Gilead holds exclusive rights outside the U.S., subject to any rights of Arcus’s existing collaboration partners, and will pay Arcus tiered royalties that range from mid-teens to low-twenties ex-US
• Added a research collaboration to focus on two jointly selected novel targets

What This Means for Arcus and the Collaboration

✓ $725 million payment and cost sharing puts Arcus in a strong position to invest in its programs
✓ Accelerates and expands our collaboration activities
✓ Brings operational expertise with global reach
✓ Enables earlier alignment on clinical studies and priorities to move fast
✓ Accelerates the exploration of new cross-portfolio combinations, with first-in-class potential
Our Partnerships Greatly Expand & Accelerate Opportunities Inherent in Arcus’s Portfolio

<table>
<thead>
<tr>
<th>10-year “all-in” collaboration</th>
<th>5-year collaboration for Japan and other territories in Asia (ex-China)</th>
<th>Clinical collaboration for domvanalimab plus durvalumab</th>
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<tbody>
<tr>
<td>• Nearly $1.4b in investments from Gilead</td>
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<tr>
<td>- Nov. 18, 2021, Gilead exercised its option to three programs for which Arcus will receive a $725m payment following closing; closing expected by YE:2021</td>
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<tr>
<td>• $175mm upfront payment + $200mm equity investment at closing of 2020 collaboration agreement</td>
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<td>• Gilead obtained rights to zim + an option to current and future molecules</td>
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<td>• In 1Q21, increased their equity stake to 19.7%, with $220mm investment in Arcus</td>
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<td></td>
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<tr>
<td>• All expenses shared between parties</td>
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<tr>
<td>• Arcus retains U.S. co-commercial rights</td>
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<tr>
<td>• Upfront payment of $35mm and potential for additional option payments</td>
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<tr>
<td>• Up to $275mm in development, regulatory and commercial milestones per program</td>
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<tr>
<td>• Tiered royalties from high-single digit to mid-teens on net sales</td>
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<tr>
<td>• Facilitates global development &amp; commercialization of Arcus molecules</td>
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<td>• Companies collaborating on PACIFIC-8, an AstraZeneca-sponsored trial</td>
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<tr>
<td>• Further validates Arcus’s position at forefront of anti-TIGIT field</td>
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<td>• Leverages AstraZeneca’s leadership in the curative-intent Stage 3 NSCLC setting</td>
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<td>• Retained economics on respective molecules</td>
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<td>• Trial to be initiated in 4Q21</td>
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~$743M in cash as of 9/30/21 (excludes the Gilead opt-in payments announced in November)
### Six Ongoing Randomized Studies Targeting Four of the Most Prevalent Cancers

<table>
<thead>
<tr>
<th>Indication</th>
<th>Study</th>
<th>Line &amp; Regimen</th>
<th>Phase 1</th>
<th>Phase 1b</th>
<th>Phase 2</th>
<th>Phase 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NSCLC</strong></td>
<td>ARC-4</td>
<td>EGFRm 2L+ etruma + carbo/pem ± zim</td>
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<td></td>
<td>ARC-7</td>
<td>WT 1L, PD-L1 ≥50%, metastatic dom + zim ± etruma vs zim</td>
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<td></td>
<td>ARC-10</td>
<td>WT 1L, PD-L1 ≥50%, advanced or metastatic dom + zim vs zim vs chemo</td>
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<td></td>
<td>PACIFIC-8</td>
<td>Stage 3 NSCLC durva ± dom</td>
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<td>Anticipated in 4Q21</td>
</tr>
<tr>
<td><strong>CRPC</strong></td>
<td>ARC-6</td>
<td>2L+: etruma + zim + doce</td>
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<tr>
<td></td>
<td></td>
<td>2L+: etruma + zim + quemli</td>
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<tr>
<td><strong>CRC</strong></td>
<td>ARC-9</td>
<td>2L: etruma + zim + FOLFOX vs FOLFOX</td>
<td></td>
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<td></td>
<td></td>
<td>3L: etruma + zim + FOLFOX vs rego</td>
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<td></td>
<td></td>
<td>&gt;3L: etruma combinations</td>
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<tr>
<td><strong>PDAC</strong></td>
<td>ARC-8</td>
<td>1L quemli + zim + gem/nab-pac</td>
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<tr>
<td></td>
<td></td>
<td>2L quemli + zim + gem/nab-pac</td>
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</tbody>
</table>

Carbo/pem: carboplatin/pemetrexed; doce: docetaxel; dom: domvanalimab; durva: durvalumab; etruma: etrumadenant; gem/nab-pac: gemcitabine/nab-paclitaxel; quemli: quemiclustat; rego: regorafenib; zim: zimberelimab; CRC: colorectal cancer; CRPC: castrate-resistant prostate cancer; NSCLC: non-small cell lung cancer; PDAC: pancreatic ductal adenocarcinoma; WT: wild-type
ANTI-TIGIT PROGRAM
Anti-TIGIT Antibody Portfolio Positions Arcus as a Pioneer in the TIGIT Field

Domvanalimab’s advancement into registrational studies coupled with AB308’s rapid advancement into expansion cohorts reinforces Arcus as a leader in the development of anti-TIGIT therapies

Domvanalimab (Fc-silent)

• Blocks the TIGIT receptor on T-cells to prevent binding of CD155; does NOT deplete TIGIT-bearing immune cells
• No evidence of ADAs (which can impact clinical efficacy) to date
• 100% TIGIT occupancy on blood lymphocytes achieved
• Increased proliferation (Ki-67) of blood CD8 T cells, of a magnitude similar to what has been described for anti-PD-1 mAbs
• Ongoing randomized Phase 2 trial (“ARC-7”) and Phase 3 trial evaluating Dom + Zim vs. Zim vs. chemotherapy (“ARC-10”) with additional Phase 3’s in planning with Gilead

AB308 (Fc-enabled)

• Also blocks the CD155 interaction with TIGIT, critical for T cell activation
• Potential to deplete TIGIT-bearing cancer cells (e.g., myeloma, NHL)
• Recommended dose for expansion (RDE) selected in the Phase 1/1b ARC-12 study evaluating AB308 plus zim in advanced malignancies.

No Depletion of T-Cells or Peripheral T-Regs

Potential for Activity in Heme Malignancies
Randomized Phase 2 Study to Evaluate dom + zim vs. zim vs. etruma + dom + zim in 1L NSCLC (PD-L1 ≥ 50%)

Designed to evaluate the doublet of TIGIT + PD-1, as well as the triplet combination of TIGIT + PD-1 + A2a/2b receptor antagonist

1L NSCLC
PDL1 ≥ 50%
excluding EGFR/ALK mutations

Arm 1 (N=50)
zimberelimab monotherapy
(crossover to triplet allowed)

Arm 2 (N=50)
zim + dom ("Doublet")

Arm 3 (N=50)
zim + dom + etruma ("Triplet")

PFS & ORR co-primary endpoints

NCT#: NCT04262856
Summary of Efficacy Observations from IA2:

• Both domvanalimab-containing arms demonstrated differentiated clinical activity compared to that of zimberelimab alone.
  ─ Zimberelimab alone continued to demonstrate activity similar to that of other marketed anti-PD-1 antibodies in the setting.

• As expected with immunotherapy treatments, continued deepening of response and greater tumor shrinkage were observed in patients with longer follow-up in both domvanalimab-containing arms.
  ─ Since the previous interim analysis, the doublet continued to show encouraging activity relative to the monotherapy, and the triplet continued to numerically outperform the doublet.

• As of the data cut-off date for this interim analysis, data for progression-free survival (PFS) was immature but indicated that fewer events of progression or death had occurred in the domvanalimab-containing arms compared to zimberelimab alone.

Summary of Safety Observations from IA2:

• No unexpected safety signals were observed; the current safety profile for each molecule in the study appeared to be consistent with known and published immune checkpoint inhibitors in this setting.

• Early safety data from this interim analysis showed a lower incidence of infusion reactions relative to published numbers from other anti-TIGIT plus anti-PD-(L)1 clinical studies.
Phase 1/1b Trial to Evaluate Safety & Tolerability of AB308 + zim in Advanced Malignancies

**Status:**
- RDE for AB308 + zim combination has been established
- Multiple expansion cohorts allow for rapid signal seeking to inform future Phase 3 studies
- All five expansion cohorts are open for enrollment

**Dose-Escalation**

**AB308 + zim combination**
(Varying Q3/Q4 dose-schedules)

**Patients with advanced or metastatic hematological or solid tumors**

**Five Expansion Cohorts**
- Heme (R/R DLBCL)
- Melanoma (2L+)
- Gastric, GEJ, Esophageal (2L+)
- NSCLC (1L, PD-L1 high)
- Cervical (2L+)

zim: zimberelimab; Heme: hematology; R/R DLBCL: relapsed, refractory diffused large b-cell lymphoma; GEJ: esophagogastric junction; NSCLC: non-small cell lung cancer
2021 AND 2022 DATA READOUTS
## Multiple Study Milestones in 2021 and 2022

<table>
<thead>
<tr>
<th>COMBINATION / ARMS</th>
<th>SETTING</th>
<th>MILESTONE</th>
<th>ANTICIPATED TIMING</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ARC-4</strong></td>
<td>etruma + zim + carbo/pem vs. zim + carbo/pem</td>
<td>Randomized Phase 1/2 Trial in TKI R/R EGFR+ 2L+ NSCLC</td>
<td>- Randomized data</td>
</tr>
<tr>
<td><strong>ARC-6</strong></td>
<td>etruma + zim + SOC vs. SOC</td>
<td>Randomized Phase 2 Trial in 2L/3L mCRPC</td>
<td>- Preliminary data from an initial cohort - Randomized data</td>
</tr>
<tr>
<td><strong>ARC-7</strong></td>
<td>zim + dom vs. zim vs. zim + dom + etruma</td>
<td>Randomized Phase 2 Trial in 1L mNSCLC (PD-L1 ≥ 50%)</td>
<td>- First interim analysis - Expected data presentation</td>
</tr>
<tr>
<td><strong>ARC-8</strong></td>
<td>quemli + zim + gem/nab-pac</td>
<td>Phase 1/1b Trial in 1L mPDAC</td>
<td>- Initial dose-escalation data - Expected data presentation</td>
</tr>
</tbody>
</table>

Phase 3 Trial to Evaluate $\text{dom} + \text{zim}$ vs. $\text{zim}$ mono vs. $\text{chemo}$ in 1L NSCLC (PD-L1 ≥ 50%)

**Designed to enable potential approval of BOTH** $\text{zim}$ mono and $\text{zim} + \text{dom}$ combination
The Adenosine Axis Plays a Well-Established and Critical Role in Suppression of the Immune Response

1. ATP is released from tumor cells in response to cellular stresses

2. Enzymatic action of CD39 and CD73 convert extracellular ATP into adenosine.

3. Adenosine elicits its immunosuppressive effects by binding the A2a and A2b receptors on the surface of immune cells.

Tumor cell

CD39

ATP

AMP

CD73

Enzymatic action of CD39 and CD73 convert extracellular ATP into adenosine.

AMP

Adenosine

etrumadenant blocks adenosine from binding to A2aR and A2bR, blocking immunosuppression

CD73

quemliclustat blocks the function of CD73, preventing the conversion of AMP to adenosine

Tumor cell

A2a Receptor

A2b Receptor

T Cells

NK Cells

Myeloid Cells

NK Cells ↓ cytotoxicity

Myeloid Cells ↓ DC / T-cell activation

T Cells ↓ effector function ↓ cytotoxicity

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HIF-2α Inhibitor Program – AB521

- Entered clinic in 4Q21 via healthy volunteer Phase 1 study
- Genetic alterations in VHL are often causatively associated with increased HIF signaling and clear cell RCC development
- Clinical POC obtained in ccRCC and VHL disease with HIF-2α inhibitor belzutifan
- Opportunities to differentiate our program by combining with other portfolio molecules
  - e.g., upregulation of CD73 (adenosine) by hypoxia is believed to be mediated by HIF-1α; thus, strong rationale to combine AB521 with etrumadenant or quelmiclustat in RCC or several other tumor types characterized by a hypoxia gene signature

### Assay

<table>
<thead>
<tr>
<th>Assay</th>
<th>AB521</th>
<th>Belzutifan 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIF-2α 786-O Luc. IC₅₀ (nM)</td>
<td>10.0 ± 3.7</td>
<td>15.9 ± 7.4</td>
</tr>
<tr>
<td>HIF-2α SPA IC₅₀ (nM)</td>
<td>19.1 (n=1)</td>
<td>31.2 ± 9.7</td>
</tr>
<tr>
<td>VEGF Secretion IC₅₀ (nM)</td>
<td>30.5 ± 3.7</td>
<td>66.7 ± 37</td>
</tr>
</tbody>
</table>

1Data from Arcus test of molecule described as MK-6482 in Wehn et al., J Med Chem (2018) 61:9691