R&D Investor Day

July 18, 2023
Forward-Looking Statements

Certain statements contained in this presentation and in the accompanying oral presentation, other than statements of fact that are independently verifiable at the date hereof, may constitute forward-looking statements. Examples of such forward-looking statements include statements regarding BeiGene’s research, discovery, and preclinical and early-stage clinical programs and plans; recent clinical data for BeiGene’s product candidates and approvals of its medicines; the conduct of late-stage clinical trials and expected data readouts; additional planned commercial product launches; and the advancement of and anticipated clinical development, regulatory milestones and commercialization of BeiGene’s medicines and drug candidates. Actual results may differ materially from those indicated in the forward-looking statements as a result of various important factors, including BeiGene’s ability to demonstrate the efficacy and safety of its drug candidates; the clinical results for its drug candidates, which may not support further development or marketing approval; actions of regulatory agencies, which may affect the initiation, timing and progress of clinical trials and marketing approval; BeiGene’s ability to achieve commercial success for its marketed medicines and drug candidates, if approved; BeiGene’s ability to obtain and maintain protection of intellectual property for its technology and medicines; BeiGene’s reliance on third parties to conduct drug development, manufacturing and other services; BeiGene’s limited experience in obtaining regulatory approvals and commercializing pharmaceutical products and its ability to obtain additional funding for operations and to complete the development and commercialization of its drug candidates and achieve and maintain profitability; the impact of the COVID-19 pandemic on BeiGene’s clinical development, commercial, regulatory and other operations, as well as those risks more fully discussed in the section entitled “Risk Factors” in BeiGene’s most recent periodic report filed with the SEC, as well as discussions of potential risks, uncertainties, and other important factors in BeiGene’s subsequent filings with the SEC. All information in this presentation is as of the date of this presentation, and BeiGene undertakes no duty to update such information unless required by law.

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Today’s Presenters

John V. Oyler  
Co-Founder, Chairman and CEO

Lai Wang, Ph.D.  
Global Head of R&D

Julia Wang  
Chief Financial Officer

Mehrdad Mobasher, M.D., M.P.H.  
Chief Medical Officer, Hematology

Mark Lanasa, M.D.  
Chief Medical Officer, Solid Tumors
Today’s Agenda

Introduction and Corporate Overview

John V. Oyler, Co-Founder, Chairman and CEO
Julia Wang, CFO

Path to Global Oncology Leadership

John V. Oyler, Co-Founder, Chairman and CEO
Lai Wang, Global Head of R&D

Delivering Impactful Oncology Innovation

• Leading in Hematology
• Advancing Broad Solid Tumor Portfolio
• Building Differentiated Research

Lai Wang, Global Head of R&D
Mehrdad Mobasher, CMO, Hematology
Mark Lanasa, CMO, Solid Tumors
Lai Wang, Global Head of R&D

Closing Remarks

John V. Oyler, Co-Founder, Chairman and CEO

Q&A Session

Management Team
Fully Integrated Global Biotech

Corporate Snapshot

$1.3B
2022 FY total product revenue (doubled vs. prior year)

17
Approved products

65+
BRUKINSA approved markets including EU

$3.8B
2023 1Q cash balance

Global Clinical Development

~140
Trials initiated in 48 countries and regions
Speed and cost advantaged

Attracting Top Global Talent

~10,000
Global headcount

Global Scale Manufacturing

42-acre biologics site
Princeton Innovation Center, NJ
Expanding biologics capacity up to 200,000L
Path to Global Oncology Leadership

John V. Oyler
Co-Founder, Chairman and CEO
Harnessing Science to Improve Access and Affordability for Cancer Patients Around the World

~800,000 patients and counting...
**Sustainable Competitive Advantages**

*Innovation with speed and lower cost to better serve patients around the world*

<table>
<thead>
<tr>
<th>RESEARCH</th>
<th>CLINICAL DEVELOPMENT</th>
<th>COMMERCIAL</th>
<th>CORNERSTONE MEDICINES</th>
<th>MANUFACTURING</th>
</tr>
</thead>
<tbody>
<tr>
<td>~1,100 world-class scientists</td>
<td>3,000+ clinical development colleagues* in 48 regions</td>
<td>3,500+ competitive commercial team with 500+ in NA/EU</td>
<td>BRUKINSA and tislelizumab</td>
<td>~750 In-house people and capabilities with cost advantage and agility</td>
</tr>
<tr>
<td>Broad preclinical programs, ~50% with first-in-class potential</td>
<td>Successful track record of developing differentiated molecules</td>
<td></td>
<td>Cornerstone commercial medicines with huge global potential</td>
<td>Small molecules and biologics (55,000L expanding to up to 200,000L)</td>
</tr>
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</table>

$1.4B collaboration fees

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*Includes full-time service professionals*
Leading Global Oncology Powerhouse

- Largest dedicated oncology R&D team
- Brodest reach of internally-run global clinical trials
- Innovative oncology pipeline with 23 development programs and 60+ discovery programs
- Emerging global leadership in hematology & foundation in solid tumors

Among mid-cap companies, up to $50bn market cap as of July 11, 2023.
Delivering Impactful Oncology Innovation

Lai Wang, Ph.D.
Global Head of R&D
Executive Summary
Leading a world-class global oncology organization with entrepreneurial culture

~1,100 innovative research scientists delivering 10 differentiated NMEs/year including many compelling, highly impactful programs starting from 2024

Faster from PCC to clinical PoC by >6 months at meaningfully reduced cost through in-house manufacturing and CRO-free clinical development model

Emerging as heme leader with potential best-in-class/first-in-class assets addressing broad range of malignancies, including BTKi, BCL2i, BTK degrader

Going beyond immuno-oncology in solid tumor portfolio with oncogenic signaling targeted therapies and TAA-driven therapies
Hematology Pipeline with 3 Important Programs
BRUKINSA, sonrotoclax (BCL2i) and BGB-11673 (BTK degrader) all with compelling data

<table>
<thead>
<tr>
<th>Zanubrutinib (BTK inhibitor)</th>
<th>CLL/SLL, MCL, WM, MZL^</th>
<th>TN MCL, R/R MZL (+rituximab)</th>
<th>R/R FL (+obinutuzumab)</th>
<th>R/R DLBCL*, ibrutinib/acalabrutinib intolerant CLL/SLL*, B-cell malignancies^ (mono)</th>
<th>R/R DLBCL* (+lenalidomide)</th>
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<tbody>
<tr>
<td>Sonrotoclax (BGB-11417, BCL2 inhibitor)</td>
<td>R/R MCL, R/R CLL* (mono)</td>
<td>NHL, AML/MDS, MM</td>
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<tr>
<td>BGB-16673 (BTK-targeted CDAC)</td>
<td>Dose escalation</td>
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<tr>
<td>Tislelizumab (anti-PD-1) [Global ex-Novartis territory†]</td>
<td>R/R classical Hodgkin’s lymphoma2</td>
<td>R/R chL* (mono)</td>
<td>R/R chL (mono)</td>
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<td>Ociperlimab (anti-TIGIT)</td>
<td>R/R DLBCL* (+tislelizumab/rituximab)</td>
<td></td>
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<tr>
<td>AMG 176 (MCL-1)^3</td>
<td>Hematologic malignancies</td>
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<thead>
<tr>
<th>P1a</th>
<th>P1b</th>
<th>P2</th>
<th>P2^</th>
<th>P3</th>
<th>Filed</th>
<th>Approved</th>
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^ U.S.: CLL/R/R MCL3; WM & R/R MZL^; China: R/R MCL2; R/R CLL/SLL2 & R/R WM; EU: CLL, WM & MZL. 1. Approved under accelerated approval. Continued approval for this indication may be contingent upon verification and description of clinical benefit in a confirmatory trial. 2. Conditionally approved in China. Full approval for these indications is contingent upon results from ongoing randomized, controlled confirmatory clinical trials. 3. The approval is applicable to all 27 EU member states, plus Iceland, Liechtenstein and Norway. *Some indications will not require a non-pivotal Phase 2 clinical trial prior to beginning pivotal Phase 2 or 3 clinical trials; **Confirmatory clinical trials post-approval are required for accelerated approvals; † Novartis owns commercial rights in United States, Canada, Mexico, the European Union, United Kingdom, Norway, Switzerland, Iceland, Liechtenstein, Russia, and Japan. 1. BGB-3111-215 trial in previously treated B-cell lymphomas intolerant of prior BTKI treatment. 2. Conditionally approved. Full approval for these indications is contingent upon results from ongoing randomized, controlled confirmatory clinical trials. 3. In collaboration with Amgen; commercial rights are in China.
Solid Tumor Pipeline is Growing
Expanding beyond I-O with targeted therapies

### Late Stage

**Tislelizumab (anti-PD-1)** [Global ex-Novartis territory†]
- China: 1L squamous and non-squamous NSCLC, 2/3 L NSCLC, 2/3 L HCC,
  R/R PD-L1+ UC, 2L ESCC, MSI-H or dMMR solid tumors, 1L NPC, 1L G/GEJ, 1L GC
  1L ESCC (+chemo), 1L HCC (mono)
- Neo/adjuvant NSCLC, 1L UBC, 1L SCLC (+chemo), early ESCC (+CRT)
- MSI-H/dMMR CRC (+mono), 1L ESCC/GC/GEJ, neo ESCC (+chemo)
- 1L HCC (+lenvatinib), solid tumors (+fruquintinib, +lenvatinib)
- Neo/adjuvant NSCLC, 1L UBC, 1L SCLC (+chemo), early ESCC (+CRT)
- 1L LS-SCLC (+tislelizumab+cCRT), 1L NSCLC (+tislelizumab+chemo)
- Solid tumors (+ TMZ (chemo))
- Solid tumors (+/− tislelizumab)
- Biliary tract cancers (Mono)
- Prostate cancer
- SCLC, neuroendocrine prostate cancer

### Early Stage

- **LBL-007** (anti-LAG-3)
  - Solid tumors (+ tislelizumab)
  - Solid tumors (+/− tislelizumab)
- **GBG-A445** (anti-OX40)
  - Solid tumors (+ tislelizumab)
- **Surzebiclimab** (GBG-A425, anti-TIM-3)
  - Solid tumors (+/- tislelizumab)
- **Lifirafenib** (RAF Dimer)
  - B-Raf- or K-RAS/N-RAS-mutated solid tumors (+/mirvatatib)
- **Brimafenib** (GBG-3245, B-Raf inhibitor)
  - Solid tumors
- **BGB-10188** (P13-K5 inhibitor)
  - Solid tumors (mono; +tislelizumab; +zanubrutinib)
- **BGB-15025** (HPK1 inhibitor)
  - Advanced solid tumors (+/- tislelizumab)
- **BGB-24714** (SMAC^ mimetic)
  - Dose escalation
- **BGB-B167** (CEA x 4-1BB bispecific)
  - Dose escalation
- **BGB-B167** (CEA x 4-1BB bispecific)
  - Dose escalation
- **Acapatamab** AMG160, PSMA x CD3)
  - Prostate cancer, NSCLC
- **AMG 199** (MUC17 x CD3)
  - Solid tumors
- **Latikafusp** (AMG 256, IL-21m/PD-1)
  - Solid tumors
- **Xaluritamig** (AMG509, STEAP1 X CD3)
  - Prostate cancer

### Solid Tumor Pipeline is Growing
Expanding beyond I-O with targeted therapies

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Advancing Broad Solid Tumor Portfolio
Moving beyond IO with focus on lung and other key tumors with several potential blockbuster programs

Leading in Hematology
Three potential best-in-class medicines addressing a market beyond CLL with complementary assets make us a major heme player

Building Differentiated Research
Exceptional ~1,100 strong team with track record of success that expects to be one of the most prolific teams including many compelling, highly impactful programs, moving forward with 10 NMEs per year
Executive Summary
Accelerating development as emerging leader in numerous hematologic malignancies

1. **Cement** BRUKINSA as best-in-class BTKi in CLL, and preferred option based on superior data

2. **Solidify leadership** in CLL with sonrotoclax (BCL2i) and BTK-CDAC while **amplifying** our impact in other B-cell malignancies with progressive treatment strategies such as fixed duration and rational sequencing

3. **Expand our footprint** into other hematological malignances:
   - Sonrotoclax in AML/MDS and multiple myeloma
   - BTK-CDAC in Richter’s transformation and large B-cell lymphoma
Hematology Portfolio
Emerging as global leader in hematology with differentiated programs

Superior and durable safety and efficacy across indications, including head-to-head vs ibrutinib.
Broadest label
CLL/SLL, WM, MCL, MZL
FL sNDA
$15B BTKi class projected in 2028

BTK inhibitor

Sonrotoclaax

500+ patients, with compelling efficacy and safety data
Initiating a Phase 3 in TN CLL and fast to market Phase 2s in MCL/WM
Register by developing in AML/ MDS and Multiple Myeloma
Potential BIC with ability to use by all physicians
$4B BCL2i market projected in 2028

BCL2 inhibitor

BGB-16673

50+ patients enrolled, PoC achieved with encouraging data
Robust development plans; fast to market indications and combinations starting in 2024
Potential in Richter's and LBCL given potency and distinct MOA
Development in BTKi resistant patients first but expand to larger patient population

BTK inhibitor

BCL2 inhibitor

BTK CDAC

Hematology
Best-in-class BTKi with a broad set of indications around the world

<table>
<thead>
<tr>
<th>Hypothesis: sustained inhibition</th>
<th>4,800+ patients enrolled globally</th>
<th>Two major Phase 3 head-to-head trials against ibrutinib</th>
<th>BTKi with approvals in most diseases</th>
<th>Expanding development program</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Engineered to exhibit high potency, bioavailability, and kinase selectivity with aim of reducing off-target toxicities while maintaining continuous high BTK inhibition</td>
<td>• Safety and efficacy of BRUKINSA assessed in numerous indications across the globe, in 35+ trials</td>
<td>• ORR and PFS in R/R CLL/SLL patients shown to be superior to ibrutinib</td>
<td>• Broadest label: CLL/SLL, WM, MCL, MZL</td>
<td>• Evaluating novel combinations: with both external and internal programs across a spectrum of hematological malignancies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• WM patients showed a consistent trend of deeper and more durable responses than ibrutinib</td>
<td>• sNDA in US and EU in FL</td>
<td></td>
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</table>

Hematology
BRUKINSA
Broad global registrational development program

**BRUKINSA**
(BTK inhibitor)

- **CLL/SLL**
  - Longer Follow-Up
  - Approved for use in R/R CLL/SLL and 1L CLL based on readout from ALPINE (R/R CLL/SLL) and SEQUOIA (1L CLL/SLL)

- **WM**
  - Longer Follow-Up
  - Approved for use across many regions based on readout from Phase 3 ASPEN trial demonstrating clinically meaningful efficacy and safety advantages of BRUKINSA in patients with WM

- **MCL**
  - Confirmatory Trial
  - Approved for use in R/R MCL
    - Ongoing Phase 3 MANGROVE investigating BRUKINSA + rituximab in 1L MCL

- **MZL**
  - Confirmatory Trial
  - Approved for use in R/R MZL
    - Ongoing Phase 3 MAHOGANY investigating BRUKINSA + rituximab in 1L

- **FL**
  - HA Submissions
  - Submissions accepted in EU & U.S. for R/R FL
    - Ongoing Phase 3 MAHOGANY investigating BRUKINSA + obinutuzumab in R/R FL

The approved indications (highlighted in red text) and may be different in different countries and HCPs should always consult the SmPC/PI approved in their country.
BRUKINSA
PFS significantly superior to ibrutinib in ALPINE – median follow-up of 29.6 months

Data from ALPINE with longer follow up (May 2023) will be submitted to an upcoming congress in 2023
• Separation of PFS KM curve continues
• Improvement in PFS sustained

No. at Risk
Brukinsa 327 315 304 301 294 280 263 226 172 161 125 113 14 2 0
Ibrutinib 325 305 293 277 260 246 228 191 133 123 98 87 9 2 2 0

PFS events n(%) (95%CI)
Brukinsa 88 (26.9)
Ibrutinib 120 (36.9)

HR (95% CI) 0.65 (0.49-0.86) p=0.0024

Data cutoff: 8 Aug 2022
Brown J et al. NEJM 2022. DOI: 10.1056/NEJMoa2211582
BRUKINSA
Improved PFS in pre-defined subset of patients with del(17p)/TP53mut

Data cutoff: 8 Aug 2022

KM curve can be compared with acalabrutinib vs ibrutinib efficacy in similar population in ELEVATE-RR with HR of 1.00

Brown J et al. NEJM 2022. DOI: 10.1056/NEJMoa2211582
BRUKINSA
Consistent PFS superiority in all ALPINE sensitivity analyses

<table>
<thead>
<tr>
<th>Analysis Type</th>
<th>HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accounting for treatment discontinuation without PD</td>
<td>0.56 (0.38, 0.84)</td>
</tr>
<tr>
<td>Accounting for new therapies without PD</td>
<td>0.63 (0.48-0.84)</td>
</tr>
<tr>
<td>Accounting for death due to COVID-19</td>
<td>0.62 (0.45-0.84)</td>
</tr>
<tr>
<td>Accounting for drug interruption</td>
<td>0.71 (0.53-0.95)</td>
</tr>
</tbody>
</table>

Data cutoff: 8 Aug 2022

Presented at the 34th French Society of Hematology Congress, March 30, 2023
• Lower rate of serious cardiac adverse events reported with BRUKINSA

• Fatal cardiac events:
  – BRUKINSA, n=0 (0%)
  – Ibrutinib, n=6 (1.9%)
    • 3 deaths within 4 months of ibrutinib initiation
    • 3 deaths 2-3 years after ibrutinib initiation; one without cardiac history

Data from ALPINE with longer follow up will be submitted to ASH 2023

• Favorable cardiac safety profile sustained
• No cardiac death with BRUKINSA

<table>
<thead>
<tr>
<th>Serious cardiac adverse events</th>
<th>BRUKINSA (n=324)</th>
<th>Ibrutinib (n=324)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 (1.9%)</td>
<td>25 (7.7%)</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Cardiac adverse events leading to treatment discontinuation</th>
<th>BRUKINSA (n=324)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (0.3)</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Atrial fibrillation</th>
<th>0</th>
<th>5 (1.5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac arrest</td>
<td>0</td>
<td>2 (0.6)</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>0</td>
<td>2 (0.6)</td>
</tr>
<tr>
<td>Cardiac failure acute</td>
<td>0</td>
<td>1 (0.3)</td>
</tr>
<tr>
<td>Congestive cardiomyopathy</td>
<td>0</td>
<td>1 (0.3)</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>0</td>
<td>1 (0.3)</td>
</tr>
<tr>
<td>Palpitations</td>
<td>0</td>
<td>1 (0.3)</td>
</tr>
<tr>
<td>Ventricular extrasystoles</td>
<td>1 (0.3)</td>
<td>0</td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
<td>0</td>
<td>1 (0.3)</td>
</tr>
</tbody>
</table>

Data cutoff: 8 Aug 2022
Brown J et al. NEJM 2022. DOI: 10.1056/NEJMoa2211582

Cardiac deaths. One death not listed due to myocardial infarction with ibrutinib discontinuation due to diarrhea 14 days prior to the fatal event.
Exposure-Adjusted Incidence Rate* for hypertension is 0.48 persons per 100 person-months excluding ALPINE (n=1,226)

*EAIR analysis can provide incidence over fixed time period, allowing comparison across trials

### BRUKINSA

Impressive efficacy in all indications with FL now filed - PDUFA in 1Q 2024

#### Duration of Response (DOR) by IRC

Median study follow up 20.2 months

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>BRUKINSA + obinutuzumab (n=145)</th>
<th>obinutuzumab (n=72)</th>
<th>2-sided P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ORR by IRC % (95% CI)</td>
<td>69.0 (60.8-76.4)</td>
<td>45.8 (34.0-58.0)</td>
<td>0.0012</td>
</tr>
<tr>
<td>CR</td>
<td>39.3</td>
<td>19.4</td>
<td>0.0035</td>
</tr>
<tr>
<td>PR</td>
<td>29.7</td>
<td>26.4</td>
<td>–</td>
</tr>
<tr>
<td>DOR by IRC Median (95%CI) months</td>
<td>NE (25.3-NE)</td>
<td>14.0 (9.2-25.1)</td>
<td>–</td>
</tr>
<tr>
<td>18-month DOR rate (95% CI) %</td>
<td>69.3 (57.8-78.2)</td>
<td>41.9 (22.6-60.1)</td>
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</tr>
</tbody>
</table>

**Graph:**

- **DOR, %**
- **No. at risk**
- **Months**
- **BRUKINSA + Obinutuzumab**
- **Censored**

**Legend:**
- **BRUKINSA**
- **Obinutuzumab**

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Hematology
BRUKINSA
Superiority core to hematology

Superior to Chemo in TN CLL
- SEQUOIA: Superior PFS, favorable safety profile
- Category 1 NCCN guidelines for CLL

Pivotal Studies in MCL, MZL and FL
- Accelerated approval in MCL and MZL
- Positive Phase 2 data in FL and sNDA accepted
- Consistent efficacy and safety across tested B-cell malignancies with deep and durable responses

Two Randomized Phase 3 Studies Superior to Ibrutinib
- ALPINE: superior efficacy and safety profile in R/R CLL - longer follow up will be submitted to ASH 2023
- ASPEN: improved efficacy and favorable safety profile in WM

Central to Future Clinical Development
- Pipeline programs with complimentary mechanisms of action with potential to improve outcomes in B-cell malignancies

Next generation best-in-class BTKi
Sonrotoclax
Potential BIC BCL2 inhibitor with differentiated profile

**More potent and specific**
- Greater potency vs. venetoclax in preclinical models
- Higher selectivity could translate to improved tolerability

**Enables broader usage**
- Shorter half life vs. venetoclax and no drug accumulation could lead to better safety profile
- Easier ramp-up for increased use by all physicians

**Improved clinical profile**
- With 500+ patients treated, clinical activity: durable responses even at low dose levels
- Safe and tolerable in combination with BRUKINSA; fixed duration induces deeper responses

**Development plan**
- Initiating Phase 3 registrational study - potential to be fixed duration SOC globally
- Monotherapy potential in post-BTKi setting; early registrations in WM and MCL

**Extends our footprint in other heme malignancies**
- Expand into other hematological malignancies: by pivotal studies in AML/MDS in combination, and MM with t(11,14); compelling data in combo with dexamethasone

**Hematology**
Sonrotoclax
500+ patients in global program including myeloid malignancies and multiple myeloma

Studies with potential for registration are highlighted in red text

B-Cell Malignancies
- Phase 1/2: Monotherapy and in combination with BRUKINSA in patients with B-cell malignancies (BGB-11417-101 globally and BGB-11417-102 in China)

Multiple Myeloma
- Phase 1/2: Monotherapy and in combination with dexamethasone ± carfilzomib (BGB-11417-105) in t(11;14) MM

Myeloid Malignancies
- Phase 1/2: In combination with azacitidine (BGB-11417-103) in R/R and 1L AML and MDS

MCL
- Phase 2: Potentially pivotal. Monotherapy in patients with R/R MCL post-BTKi in the BGB-11417-201 trial

CLL/SLL
- Phase 2: Potentially pivotal. Monotherapy in patients with R/R CLL/SLL post-BTKi in the BGB-11417-202 trial in China

WM
- Phase 2: Potentially pivotal. Monotherapy in patients with R/R WM post-BTKi and post-chemoimmunotherapy in the BGB-11417-203 global trial

CLL/SLL
- Phase 3: Pivotal. Fixed duration combination with BRUKINSA in TN CLL/SLL patients in the BGB-11417-301 global pivotal trial

Hematology
**Sonrotoclax**

**FIH** answers questions on optimizing dose and ramp-up schedule and potential combos.

### Monotherapy

- **Sonrotoclax**

### Combination

- **Sonrotoclax + BRUKINSA**

### Combinations

- **Sonrotoclax + obinutuzumab**
- **Sonrotoclax + obinutuzumab + BRUKINSA**

#### Dose-Finding

- **Part 1**
  - Independent disease-specific cohorts:
    - R/R CLL/SLL, FL, DLBCL, MCL, MZL, WM

- **Part 3**
  - Independent disease-specific cohorts:
    - R/R CLL/SLL, MCL; TN CLL/SLL

- **Part 5**
  - TN CLL/SLL

#### Expansion

- **Part 2**
  - Independent disease-specific cohorts:
    - R/R CLL/SLL, FL, DLBCL, MCL, MZL, WM

- **Part 4**
  - Independent disease-specific cohorts:
    - R/R CLL/SLL, MCL; TN CLL/SLL

- **Part 6**
  - TN CLL/SLL

**BGB-11417-101 Study**
## Efficacy (Overall Response Rate)

<table>
<thead>
<tr>
<th>Response, n (%)</th>
<th>R/R sonrotoclax* (n=47)</th>
<th>R/R sonrotoclax + BRUKINSA (n=34)</th>
<th>TN sonrotoclax + BRUKINSA (n=94)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number treated w/ sonrotoclax</td>
<td>47</td>
<td>34</td>
<td>94</td>
</tr>
<tr>
<td>Efficacy evaluable treated with sonrotoclax</td>
<td>35</td>
<td>25</td>
<td>56</td>
</tr>
<tr>
<td>ORR, n (%)</td>
<td>23 (65.7)</td>
<td>24 (96)</td>
<td>56 (100)</td>
</tr>
<tr>
<td>CR</td>
<td>8 (22.8)</td>
<td>11 (44)</td>
<td>15 (26.8)</td>
</tr>
<tr>
<td>PR</td>
<td>15 (42.9)</td>
<td>12 (48)</td>
<td>41 (73.2)</td>
</tr>
<tr>
<td>PR-L</td>
<td>–</td>
<td>2 (8)</td>
<td>0</td>
</tr>
<tr>
<td>SD</td>
<td>5 (14.3)</td>
<td>1 (4)</td>
<td>0</td>
</tr>
<tr>
<td>PD</td>
<td>5 (14.3)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Median Follow-up (months)</td>
<td>8.44 (0.1-29.6)</td>
<td>16.99 (0.6-26.3)</td>
<td>8.54 (0.6-18.2)</td>
</tr>
</tbody>
</table>

Data cut-off date for BGB-11417: 24Apr 2023 for R/R CLL, 21May2023 for TN CLL

*Monotheray data in R/R CLL/SLL was pooled analysis of 101 and 102

TN CLL/SLL patients on combination with at least 3 post-baseline response assessments (n=37)

ORR=100% and CR=35%
Sonrotoclax and BRUKINSA Combination
High and increasing undetectable MRD in TN CLL/SLL with increased dose levels

- Higher uMRD rate, and more rapid uMRD with increasing dose
- Higher uMRD rate with longer treatment duration
  - uMRD at ≥ 12m treatment: 69% (11/15) at 160mg; 1/1 at 320 mg
Sonrotoclax and BRUKINSA Combination
All 94 patients in TN CLL remain on study progression free
**Sonrotoclax**

CLL monotherapy and combination demonstrates promising safety and tolerability

<table>
<thead>
<tr>
<th>TEAE, n, %</th>
<th>Sonrotoclax Monotherapy( n=47 )</th>
<th>Sonrotoclax + BRUKINSA RR( N=27 )</th>
<th>Sonrotoclax + BRUKINSA TN( N=79 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any AEs</td>
<td>46 (97.9)</td>
<td>26 (96.3)</td>
<td>69 (87.3)</td>
</tr>
<tr>
<td>Grade ≥3</td>
<td>28 (59.6)</td>
<td>10 (37)</td>
<td>28 (35.4)</td>
</tr>
<tr>
<td>Serious AEs</td>
<td>16 (34)</td>
<td>4 (14.8)</td>
<td>9 (11.4)</td>
</tr>
<tr>
<td>Leading to death</td>
<td>2 (4.3)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Treated with sonrotoclax</strong></td>
<td>47</td>
<td>27</td>
<td>79</td>
</tr>
<tr>
<td>Leading to dose interruption</td>
<td>20 (42.6)</td>
<td>7 (25.9)</td>
<td>15 (19)</td>
</tr>
<tr>
<td>of sonrotoclax</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leading to dose reduction</td>
<td>1 (2.1)</td>
<td>0</td>
<td>3 (3.8)</td>
</tr>
<tr>
<td>of sonrotoclax</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leading to discontinuation</td>
<td>2 (4.3)</td>
<td>0</td>
<td>1 (1.3)</td>
</tr>
<tr>
<td>of sonrotoclax</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- No DLTs were observed to date with the combination therapy at any dose level
- TLS: No lab or clinical TLS reported for combo
- No increased complicated neutropenia, infection and diarrhea with combination
Sonrotoclax
Planned Phase 3 study in treatment naïve CLL with fixed duration treatment

Previously Untreated CLL/SLL

Stratified by:
- Age (<65yr, ≥65yr)
- IGHV
- del(17p)/TP53 mutation
- Geographic region (Asia vs non-Asia)

Primary endpoint: PFS superiority by IRC
Secondary endpoints: CR/CRI, uMRD at end of treatment, OS, ORR, DOR, PFS by INV, PRO, safety

BRUKINSA + Sonrotoclax FD
12 cycles after 3 cycles Z mono

Venetoclax + Obinutuzumab FD
Sonrotoclax
Phase 1 study R/R multiple myeloma with t(11,14) mutation

Dose Escalation and Expansion:
Sonrotoclax (RP2D) + Carfilzomib + Dexamethasone

Dose level 1: 80mg
Dose level 2: 160mg
Dose level 3: 320mg
Dose level 4: 640mg

N ≥3 at each dose

Dose Expansion:
Sonrotoclax (RP2D) + Dexamethasone

Dose Escalation and Expansion:
Sonrotoclax (RP2D-1) + Carfilzomib + Dexamethasone
Sonrotoclax + Dexamethasone: Higher ORR and deep responses in R/R multiple myeloma with t(11,14)

- No DLT at any dose level; no deaths associated with study treatment
- Most common TEAEs were insomnia (42%), fatigue (32%), nausea (26%), arthralgia (21%), and COVID-19 (16%)
- Competitor data: venetoclax mono in t(11;14) MM: ORR 40%

*1 patient in the 640mg cohort is unevaluable at time of data cut-off
**BGB-16673 BTK CDAC**
Chimeric degradation activation compound - a novel approach to BTK pathway

### CDAC platform
- **Bivalent molecule** that co-opts a process leading to degradation of target protein

### BTK CDAC
- **Mutation agnostic** mechanism allows for optimal sequencing
- May provide additional **potency benefits**
- Lack of IMiD activity (vs competitors) allows for improved safety

### Robust clinical plan
- **Two Phase 1 studies** currently enrolling (>50 patients to date)
- Enrollment in potential **pivotal expansion cohorts** 2024
- Combination trial 2024

### More heme malignancies
- Become backbone for patients progressing after BRUKINSA as mono or combo with sonrotoclax
- Degradation may expand **disease areas** where there is a clear rationale (e.g., LBCL)
Dose Escalation

Selected R/R B-Cell Malignancies
(MZL, FL, MCL, CLL/SLL, WM, DLBCL, RT)
- 600 mg daily
- 500 mg daily
- 350 mg daily
- 200 mg daily
- 100 mg daily
- 50 mg daily

Safety Expansion

Up to 20 patients enrolled at each dose level.

Dose Expansion

Cohort 1:
Post BTKi R/R CLL/SLL

Cohort 2:
Post BTKi R/R MCL

Objectives

- Characterizing safety / PK / biomarker properties, MTD, and RP2D in escalation and safety expansion
- Safety/efficacy at the RP2D in dose expansion
BTK CDAC
Strong BTK inhibition starting at lowest dose and dose-dependent inhibition in tissue

- Blood PD: steady state data week 4 or 5, show complete BTK degradation already observed in initial dose level
- Tissue PD: in lymph node, 20% of remaining BTK + tumor cells at 100 mg and as low as 1% at 200 mg dose
## Safety Overview

<table>
<thead>
<tr>
<th>Event</th>
<th>N=27</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with any grade AE (%)</td>
<td>25 (92.6)</td>
</tr>
<tr>
<td>Grade ≥3 AEs</td>
<td>11 (40.7)</td>
</tr>
<tr>
<td>Serious AEs</td>
<td>10 (37.0)</td>
</tr>
<tr>
<td>AEs reported as DLTs *</td>
<td>1 (3.7)</td>
</tr>
<tr>
<td>AEs leading to death §</td>
<td>1 (3.7)</td>
</tr>
<tr>
<td>AEs leading to dose hold</td>
<td>6 (22.2)</td>
</tr>
<tr>
<td>AEs leading to dose reduction</td>
<td>2 (7.4)</td>
</tr>
<tr>
<td>AEs leading to discontinuation</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

mFU: 3.5mo

## Adverse Events of Interest (Pooled, %)

<table>
<thead>
<tr>
<th>Event</th>
<th>Any Gr</th>
<th>G3+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any Bleeding</td>
<td>12 (44.4)</td>
<td>2 (7.4)</td>
</tr>
<tr>
<td>Neutropenia</td>
<td>6 (22.2)</td>
<td>4 (14.8)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>6 (22.2)</td>
<td>0</td>
</tr>
<tr>
<td>Amylase/Lipase Increased</td>
<td>6 (22.2)</td>
<td>1 (3.7)</td>
</tr>
<tr>
<td>Any Infection</td>
<td>10 (37.0)</td>
<td>4 (14.8)</td>
</tr>
<tr>
<td>Anemia</td>
<td>1 (3.7)</td>
<td>1 (3.7)</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>1 (3.7)</td>
<td>1 (3.7)</td>
</tr>
<tr>
<td>Arthralgia</td>
<td>2 (7.4)</td>
<td>0</td>
</tr>
<tr>
<td>Atrial Fibrillation</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Fatigue</td>
<td>2 (7.4)</td>
<td>0</td>
</tr>
</tbody>
</table>

* Rash maculopapular of face and leg
§ Sepsis and Septic Shock in context of possible disease progression

BTK CDAC
BGB-16673-101 preliminary safety: no hypertension or atrial fibrillation observed to date
### BTK CDAC
BGB-16673-101 – good overall response rate (ORR) per dose level and histology

#### ORR by Dose Level

<table>
<thead>
<tr>
<th>Dose Level</th>
<th># of Ongoing Pts / Total</th>
<th>ORR of Evaluable Pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>50mg</td>
<td>2/4</td>
<td>50% (2/4)</td>
</tr>
<tr>
<td>100mg</td>
<td>7/9</td>
<td>55% (5/9)</td>
</tr>
<tr>
<td>200mg</td>
<td>8/9</td>
<td>86% (6/7)</td>
</tr>
<tr>
<td>350mg</td>
<td>2/3</td>
<td>0% (0/1)</td>
</tr>
<tr>
<td>500mg</td>
<td>2/2</td>
<td>NE</td>
</tr>
<tr>
<td>TOTAL</td>
<td>78% (21/27)</td>
<td>62% (13/21)</td>
</tr>
</tbody>
</table>

#### ORR by Histology

<table>
<thead>
<tr>
<th>Histology</th>
<th>ORR Rate</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>DLBCL</td>
<td>0%</td>
<td>1</td>
</tr>
<tr>
<td>FL</td>
<td>50%</td>
<td>2</td>
</tr>
<tr>
<td>MCL</td>
<td>40%</td>
<td>5</td>
</tr>
<tr>
<td>MZL</td>
<td>100%</td>
<td>2</td>
</tr>
<tr>
<td>CLL</td>
<td>71%</td>
<td>7</td>
</tr>
<tr>
<td>WM</td>
<td>75%</td>
<td>4</td>
</tr>
</tbody>
</table>

N= # evaluable patients

mFU: 3.5mo
Unofficial DCO 15 Jun 2023
BTK CDAC
BGB-16673-101 efficacy by patient, with promising durability data

Previous Treatment
<table>
<thead>
<tr>
<th>cBTKi</th>
<th>Bcl2i</th>
<th>ncBTKi</th>
</tr>
</thead>
</table>

- MCL
- CLL
- WM
- FL
- MZL
- RT

Treatment Duration (Weeks)

- CR
- PR
- PR-L
- MR
- SD
- PD
- On-treatment

Dose Level (mg)
- 50
- 100
- 200
- 350
- 500

X patient discontinued study. DCO 15JU
BGB-16673-101 Efficacy By Patient N2023
Median number of prior treatments = 4
cBTKi - covalent BTKi ncBTKi - non-covalent BTKi

Hematology
<table>
<thead>
<tr>
<th>Promising Early Efficacy and PD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep BTK degradation in PD studies</td>
</tr>
<tr>
<td>Promising efficacy signal in heavily pre-treated patients, including patients progressing on prior cBTKi and ncBTKi</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Good Safety Profile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toxicity appears favorable compared to other BTK degraders</td>
</tr>
<tr>
<td>No hypertension or atrial fibrillation observed at this point</td>
</tr>
</tbody>
</table>
### CLL Diagnosis

#### TN CLL
- BRUKINSA until progression
- FD BRUKINSA + sonrotoclacl
- FD BRUKINSA + sonrotoclacl + CD20
- FD sonrotoclacl + CD20

#### R/R CLL
- BTK CDAC ± sonrotoclacl
- BTK CDAC ± CD20
- BRUKINSA ± sonrotoclacl (if not received in TN)
- Sonrotoclacl + CD20

### Choices BeiGene can offer

#### Patients*/HCPs

*The choice of therapy is driven by patient preference, PS, risk stratification, MRD assessment etc.

# The choice of therapy is driven by prior therapies and response, PS, patient preference and risk stratification.
Key Takeaways
Accelerating development as emerging leader across numerous hematologic malignancies

1. BRUKINSA as best-in-class BTKi for CLL based on ALPINE update adding confidence to its durable superior efficacy and safety vs. ibrutinib

2. Bring forward sonrotoclax and BTK-CDAC as best-in-class medicines

3. Develop evidence to support impactful and desirable treatment strategies including fixed duration and rational sequencing

4. Expand our footprint with sonrotoclax in AML/MDS, MM and BTK-CDAC Richter’s transformation and LBCL
Executive Summary
Improving outcomes for patients across broad range of solid tumors

1. Establish tislelizumab as a global standard of care PD-1 in multiple tumor types

2. Build best-in-class regimens leveraging tislelizumab combinations with next-wave IO, including new targets CCR8, DGKζ, and PVRIG

3. Expand into additional tumor types with novel agents that have blockbuster potential such as CDK4 selective inhibitor in breast cancer
Tislelizumab: (PD-1 mAb)
With broad reach and potential

**Patient Impact**

- More than **750,000** patients treated commercially
- Over 12,000 global patients in sponsored clinical trials
- Developing **subcutaneous injection** formulation (FIH 2023)

**Data**

- **RATIONALE-305**
  - 1L GC: Met primary endpoint (OS)
- **RATIONALE-312**
  - 1L ES-SCLC: Met primary endpoint (OS)

**Global Expansion and Scale**

- **RATIONALE-302**
  - 2L ESCC: FDA on-site GMP inspection complete and BLA review progressing
- Regulatory submissions underway to **expand to rest of world**
- Reduced cost through optimization, internalization, and scale
PD-1 Centered Pan Tumor Immuno-Oncology Pipeline
Extensive tumor microenvironment modulating approaches

Over 20 I-O and targeted molecules to pair with tislelizumab
Next Wave of Immuno-Oncology Programs
Will synergize in combination with tislelizumab

**Solid Tumors**

**Next Wave of IO Assets**

### TIGIT
- Phase 3 NSCLC PDL-1+ to complete enrollment end of 2023
- Five Phase 2 studies enrolled and nearing primary read-out (1,000+ patients)

### LAG3
- Phase 2 in 1L NSCLC, neoadj NSCLC, 1L HNSCC, 1L ESCC, and 1L CRC MTx
- Mono, tisle combo, and tisle/TIM3 triplet dose escalation complete (40 patients)

### TIM3
- Phase 2 in 1L HNSCC; Phase 1b in 2L+ NSCLC and HNSCC
- Mono, tisle combo, and tisle/TIM3 triplet dose escalation complete (113 patients)

### OX40
- Non-ligand blocking OX40 agonist (240 patients); Phase 2 dose established
- Phase 2 in 1L NSCLC, 2L+ NSCLC, UBC, RCC, and melanoma

### HPK1
- Phase 2 dose established; Dose expansions enrolling in 1L NSCLC and 2L+ ESCC
- Mono and tisle combo dose escalation (108 patients)

### CCR8
- BIC potential – unique binding epitope, which may facilitate more potent ADCC effect
- IND submitted with FIH in 3Q23

### DGKζ
- FIC potential – activator of T and NK cells
- IND submitted with FIH in 3Q23

### PVRIG
- BIC potential - strong binding affinity, ligand blockade potency. FIH in 4Q23
- Fc-competent which increased anti-tumor activity in pre-clinical models
Umbrella Trial Multiplier Effect
Efficient testing of efficacy of multiple interventions vs. standard of care in single study

Ability to efficiently and with lower cost test multiple combinations in one study

Single Disease

Randomize Against Control

Control
Experimental arm 1
Experimental arm 2
Experimental arm 3…n

Umbrella studies active in advanced and resectable NSCLC; HNSCC study in start-up

Adapted from Park et al. Trials (2019) 20:572
Innovative Solid Tumor Portfolio
Accelerating programs in priority tumor types

**NSCLC**
- EGFR-CDAC
- panKRAS
- MTA-Cooperative PRMT5
- B7H3-ADC
- CEA-ADC
- MUC1xCD16
- Claudin6xCD3

**Upper GI**
- CEA-ADC
- B7H3-ADC
- CEAx4-1BB*

**Colorectal**
- panKRAS
- CEAx4-1BB*
- CEA-ADC

**Head and Neck**
- SMAC Mimetic*
- B7H3-ADC

**Breast**
- CDK4
- B7H4-ADC**
- BCL2i*

* In the clinic
** Exclusive global option from Duality
CDK4 Inhibitor
Next-generation CDK4 inhibitor aiming for better efficacy and less toxicity

- CDK4/6 inhibitor class had huge commercial success in HR+/HER2- breast cancer (estimated peak sales over $18B worldwide)
  - 3 CDK4/6 inhibitors have been approved by FDA, but all with on-target toxicity
- **Selective CDK4 inhibitor is differentiated**
  - Improve efficacy and safety profile
  - Potential new indications, including lung, prostate, ovarian and endometrial cancer
  - Only one CDK4 inhibitor (PF-07220060) in Phase 1
- On track to enter clinic in 2023

### CDK4 Selective Inhibition for Better Efficacy and Less Toxicity

<table>
<thead>
<tr>
<th>Drug</th>
<th>Palbociclib</th>
<th>Ribociclib</th>
<th>Abemaciclib</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose Limiting Toxicities</td>
<td>Neutropenia</td>
<td>Neutropenia</td>
<td>Fatigue, Diarrhea</td>
</tr>
<tr>
<td>Potential Cause</td>
<td>CDK6 Inhibition</td>
<td>CDK6 Inhibition</td>
<td>Off-target to CDK9, GSK3β and CAMKIIIs</td>
</tr>
<tr>
<td>CDK4/6 Selectivity</td>
<td>1.3</td>
<td>7.7</td>
<td>16</td>
</tr>
</tbody>
</table>

- **CDK4**
  - Cyclin D1
  - Cell cycle progression and cancer cell proliferation
  - In Tumor Cell

- **CDK6**
  - Cyclin D3
  - HSC self renew and differentiation
  - In Bone Marrow

Solid Tumors
CDK4 Inhibitor
Highly potent and selective, with robust efficacy and improved tolerability*

CDK4i Shows the Strongest CDK4 Potency

CDK4i Has the Best CDK4/CDK6 Selectivity

CDK4i Shows Strong Efficacy in Combo with Fulvestrant

Well tolerated in GLP TOX study without neutropenia and GI toxicity issues

*In preclinical models

MCF-7 proliferation assay to assess CDK4 potency
*CDK4 cellular IC50 measured through pRB in Jeko-1; CDK6 cellular IC50 measured through pRB in Pfeiffer with CDK4 KO

Cellular CDK6 IC50 divided by cellular CDK4 IC50 to assess CDK6 selectivity*

MCF-7: HR+, HER2- breast cancer model

Vehicle
Fulvestrant
BG CDK4i + Fulvestrant
Leverage R&D Innovation to Generate Next Wave of Programs
Priority tumor types with blockbuster potential

Priority Tumor Types

Future Cornerstone Programs

Diversified Therapeutic Modalities

- Small Molecule
- CDAC
- mAb
- BsAb/TsAb
- ADC
- Cell Therapy
- mRNA

- B7-H3 ADC
- CDK4
- EGFR CDAC
- PanKRAS
- PRMT5
Key Takeaways
Improving treatment for patients across broad range of solid tumors

1. Establish tislelizumab as a global standard of care PD-1 in multiple tumor types

2. Build best-in-class regimens leveraging tislelizumab combinations with next-wave IO, including new targets CCR8, DGKζ, and PVRIG

3. Expand into additional tumor types with novel agents that have blockbuster potential such as CDK4 selective inhibitor in breast cancer
Research Innovation

Lai Wang, Ph.D.
Global Head of R&D
Executive Summary
~1,100 innovative scientists delivering 10 new treatment changing molecules per year*

1. Develop diverse and compelling programs across hematology and solid tumors

2. Detail our tumor type approach with lung cancer portfolio
   3 exciting small molecules, 2 ADCs and 2 bi-specifics with differentiated TAA approaches

3. Lead the industry in breadth of novel modality designs to deliver potential breakthrough medicines
   (small molecules, CDACs, mAbs, bi/tri-specifics, ADCs, cell therapies and mRNAs)

4. Combine differentiated targets with novel modalities across tumor types to deliver improved patient outcomes (as with our lung cancer portfolio)

*starting from 2024
# Broad Oncology Coverage in Current Tumor Types
Expanding into new tumor types to deliver broader patient impact

## Solid Tumors

<table>
<thead>
<tr>
<th>Tumor Type</th>
<th>2028 Market Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung</td>
<td>$53B</td>
</tr>
<tr>
<td>Upper GI</td>
<td>$12B</td>
</tr>
<tr>
<td>Head and Neck</td>
<td>$5B</td>
</tr>
<tr>
<td>Breast</td>
<td>$42B</td>
</tr>
<tr>
<td>Colorectal</td>
<td>$8B</td>
</tr>
</tbody>
</table>

## Hematology

<table>
<thead>
<tr>
<th>Malignancy Type</th>
<th>2028 Market Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>B-Cell Malignancies</td>
<td>$45B</td>
</tr>
<tr>
<td>AML and MDS</td>
<td>$13B</td>
</tr>
<tr>
<td>Multiple Myeloma</td>
<td>$28B</td>
</tr>
</tbody>
</table>

---

2028 WW Market Size estimates by EvaluatePharma
Upper GI includes GC, HCC, ESCC
B-cell Malignancies includes NHL (including DLBCL), CLL, and others (including MCL, MZL, WM, FL, SLL)
Lung Cancer Portfolio with FIC/BIC Potential
Over 30 scientifically driven targets with diverse modalities - highlighting 7
EGFR CDAC
Truly differentiated MOA to completely abolish EGFR signaling

• Address large EGFRmut patient population
  • ~50% lung adenocarcinoma in Asian and 15% in Caucasian*

• Potentially best-in-class strategy - degradation
  • Induce more sustained signaling inhibition by eliminating the EGFR protein in the cells
  • Target broad EGFR mutations
  • Destroy EGFR scaffold function to minimize compensatory signaling via heterodimerization with other receptor tyrosine kinases

• Candidate selected and to enter clinic in 2024

Differentiated MoA of EGFR CDAC

<table>
<thead>
<tr>
<th>MoA</th>
<th>Osimertinib-Sensitive Mutation</th>
<th>Osimertinib-Resistant Mutation</th>
<th>Destroy Scaffolding Function</th>
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<tr>
<td>3G TKI</td>
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<td>✗</td>
<td>✗</td>
</tr>
<tr>
<td>4G TKI</td>
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<tr>
<td>CDAC</td>
<td>✓</td>
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</table>

**EGFR CDAC**
Targeting broad range of EGFR mutations while sparing WT

- Highly potent across EGFR mutations sparing WT EGFR
- Highly selective in proteome panel
- Desirable oral bioavailability supporting daily dosing in clinic
- Robust efficacy in both osimertinib-sensitive and resistant models
- Good brain penetration in preclinical models

---

**Broasted EGFRmut Coverage While Sparing WT**

<table>
<thead>
<tr>
<th></th>
<th>WT</th>
<th>LR</th>
<th>D19</th>
<th>LT</th>
<th>DT</th>
<th>LC</th>
<th>DC</th>
<th>LTC</th>
<th>DTC</th>
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<td>Gefitinib (1G TKI)</td>
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<td>BLU-945 (4G TKI)</td>
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</tbody>
</table>

**Robust Efficacy in Both Subcutaneous and Intracranial Xenograft Models**

**Osimertinib Resistant H1975-D19/C797S Model**

**H1975 (L858R/T790M) Intracranial Model**

WT: wild-type; LR: L858R; D19: exon 19 deletion; DT: exon 19 deletion/T790M; LT: L858R/T790M; DC: exon 19 deletion/C797S; LTC: L858R/T790M/C797S; DTC: L858R/T790M/C797S;
PanKRAS Inhibitor
Addressing broad range of KRAS mutations in multiple tumor types

- KRAS mutations found in ~19% of all tumor types*
  - 9% in lung adenocarcinoma in Asia and 33% in Caucasian
  - 43% in CRC & 87% in pancreatic ductal adenocarcinoma

- Addressing broad KRAS mutations

  PanKRAS Inhibitor

  - Adult mice with inducible KRAS KO appeared normal and healthy#, suggesting low risk with inhibiting WT KRAS by panKRAS inhibitor
  - Highly potent across different KRAS mutations with good selectivity against N/HRAS
  - Candidate selection in 2023 and to enter clinic in 2024

# Data on file

Robust Activity in KRAS Dependent Cell Lines, Yet Spares KRAS Independent Cells

Strong Anti-Tumor Efficacy in KRAS-Driven Xenograft Models

hPBMC: Human peripheral blood mononuclear cells; HSPC: human hematopoietic stem/progenitor cell
MTA-Cooperative PRMT5 Inhibitor
Next-generation PRMT5 inhibitor avoiding hematologic toxicity

- 2nd generation, MTA-cooperative PRMT5 inhibitor selectively kills MTAP-deleted tumor cells avoiding normal hematological cells
- MTAP-deletion is found in 15% of all tumor types*
  - 8% in lung adenocarcinoma and 19% in lung squamous cell carcinoma
  - 10% in gastric adenocarcinoma and 28% in esophageal adenocarcinoma
- Promising pharmacological properties
  - Good brain penetration
  - Desirable half-life supports daily dosing
- Candidate selection in 2023 and to enter clinic in 2024

1G PRMT5 Inhibitor Has Hematologic Toxicity

- Normal Cells
- Tumor Cells
- PRMT5
- Killing
- Undesirable efficacy due to dose-limiting hematological toxicity
- 1G inhibitor

2G MTA-Cooperative PRMT5 Inhibitor Spares Normal Cells

- Normal Cells
- MTAP Deletion
- Tumor Cells
- MTA Low
- MTA High
- PRMT5
- No stable binding
- Spared
- Killing
- 2G inhibitor
- MTA

PRMT5: protein arginine methyltransferases 5; MTA: methylthioadenosine; MTAP: methylthioadenosine phosphorylase

Tumor Associated Antigens as Tumor-Targeting Therapies
Broad applicability with multiple therapeutic modalities

Toxin ADC
Current portfolio focus

New concept ADC to unleash the power of drug conjugate

Immune cell engaging BsAb/TsAb
Current portfolio focus

Allogenic cell therapy as emerging direction to develop breakthrough therapeutics

ADC: antibody drug conjugate; BsAb: bispecific antibodies; TsAb: trispecific antibodies; CAR: chimeric antigen receptor
Next Generation ADC Platform
Novel approaches to payload, linker, and conjugation for BIC ADC

**Powerful discovery engine generating fit-for-purpose Abs with high quality and efficiency**
- Monoclonal Ab
- Multi-specific Ab

**Diversified toxin toolbox to fit different TAAs and indications**
- Proprietary Topoisomerase I inhibitor optimized for strong bystander effect
- Hydrophilic linkers to enable higher DAR with MMAE payload
- Proprietary PBD payload with pro-drug design to improve safety margin

**Homogeneous conjugation creates more uniform DAR/better stability**
- Site specific
- Stable conjugator

**Hydrophilic linker platform with various cleavage mechanisms**
- Fine-tuned hydrophilicity to reduce aggregation
- Tandem release linker to minimize systemic payload release
- Neutrophil protease insensitive linker to reduce neutropenia toxicity with MMAE

**Payload Conjugation Linker Antibody**
B7-H3 ADC
BIC potential with stable DAR8 and strong bystander effect

- Highly expressed in multiple tumor types, including lung, GI, gynecological cancers
  - B7-H3 moderate to high expression: 39% in lung adenocarcinoma, 84% in lung squamous cell carcinoma
- Clinical validation by lead competitor DS-7300 in small cell lung cancer and prostate cancer
- Differentiated drug design with BIC potential
  - High DAR (DAR8) enhance payload delivery
  - Proprietary drug-linker with strong bystander effect to address tumor heterogeneity
  - Stable conjugator to improve stability and tumor presence
- Candidate selected and to enter clinic in 2024

<table>
<thead>
<tr>
<th>B7-H3 Expression</th>
<th>LUSC</th>
<th>LUAD</th>
<th>ESCC</th>
<th>CRC</th>
<th>HCC</th>
<th>OC</th>
<th>EC</th>
</tr>
</thead>
<tbody>
<tr>
<td>B7-H3 Medium/High (H-score 101-300)</td>
<td>84%</td>
<td>39%</td>
<td>80%</td>
<td>23%</td>
<td>43%</td>
<td>25%</td>
<td>89%</td>
</tr>
</tbody>
</table>

Michiko Yamato et al., Mol Cancer Ther, 2022
LUSC: Lung squamous cell carcinoma; LUAD: Lung adenocarcinoma; ESCC: Esophageal squamous cell carcinoma; CRC: Colorectal cancer; HCC: Hepatocellular carcinoma; OC: Ovarian cancer; EC: Endometrial carcinomas

BeiGene’s B7-H3 ADC: Differentiated Molecular Design

<table>
<thead>
<tr>
<th>Attribute</th>
<th>DS-7300</th>
<th>BG B7H3 ADC</th>
<th>BeiGene Advantage</th>
</tr>
</thead>
<tbody>
<tr>
<td>DAR</td>
<td>4</td>
<td>8</td>
<td>Higher DAR</td>
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<tr>
<td>Payload-Linker</td>
<td>DXd-GGFG</td>
<td>Topo inhibitor-hydrophilic linker</td>
<td>Stronger bystander effect</td>
</tr>
<tr>
<td>Conjugation</td>
<td>Traditional Cysteine conjugation</td>
<td>Stable conjugator</td>
<td>Better stability</td>
</tr>
</tbody>
</table>

DS-7300 is B7-H3 ADC lead competitor from Daiichi Sankyo
Topol, Topoisomerase I
**B7-H3 ADC**
Active in DS-7300 insensitive and resistant models

### Superior On-Target Killing

![Graph showing cell viability for B7-H3 ADC vs. DS-7300 in H1650 (B7-H3 High) cells.](image)

### Better Bystander Killing

![Graph showing cell viability for B7-H3 ADC vs. DS-7300 in MDA-MB-453(B7-H3-) + H358(B7-H3+) cells.](image)

### Higher DAR Stability in Monkey PK Study

![Graph showing sustained DAR and reduced DAR with DS-7300 and BG B7H3 ADC.](image)

### Robust Tumor Shrinkage in Lead Competitor Insensitive/Resistant Models

- **B7-H3 Low CDX Model (H-score: 66)**
  - Vehicle
  - DS-7300, 10mpk
  - BG B7H3 ADC, 3mpk

- **NSCLC PDX Model 1**
  - Vehicle
  - DS-7300, 10mpk
  - BG B7H3 ADC, 5mpk

- **NSCLC PDX Model 2**
  - Vehicle
  - DS-7300, 10mpk
  - BG B7H3 ADC, 5mpk

---

*Lead Competitor biosimilar used as benchmark*
*Data is from preclinical models*
Cancer Type | High CEA Expression | Medium to Low CEA Expression | Anti-Tubulin Sensitivity
--- | --- | --- | ---
Lung adenocarcinoma | 7% | 31% | Yes
Gastric | 26% | 22% | Yes
Colorectal | 51% | 36% | No

BeiGene’s CEA ADC with Differentiated ADC Design

**Attribute** | **SAR701** | **BG CEA ADC** | **BeiGene Advantage**
--- | --- | --- | ---
**Payload** | DM4 | Proprietary TopoI inhibitor | • Stronger bystander effect
• Payload MoA is better fit for target indications
**DAR** | 4 | 8 | • Higher DAR
**Linker** | SPDB disulfide | Hydrophilic | • Better ADC stability
**Conjugation** | Lysine | Cystine (w/ stable conjugator) | • Better ADC homogeneity and stability

* SAR701 is in short for SAR408701

**CEACAM5 (CEA) is a well-established TAA highly expressed in multiple cancer types**

**Lead competitor SAR701* achieved clinical PoC in lung cancer, room for further patient impact**

  - Only 20% ORR in CEA\textsuperscript{High} lung cancer and 7% in CEA\textsuperscript{Med} lung cancer
  - Minimal efficacy in CRC and gastric cancer

**Differentiated ADC design to expand into lung cancer pts with CEA\textsuperscript{Med/Low} and GI cancer pts**

  - Different payload strategy: topoisomerase I (topol) inhibitor
  - High DAR (8), stable conjugator and hydrophilic linker design

**Candidate selected and to enter clinic in 2024**

* Stéphanie Decary et al., Clin Cancer Res, 2020 Dec 15;26(24): 6589-6599

SAR701 is CEA ADC lead competitor from Sanofi
CEA ADC
Better stability, tumor exposure, and bystander effects for better efficacy*

Better DAR Stability and Tumor Killing Than Lead Competitor

DAR Stability in Mouse PK Study

Tumor ADC Distribution

Bystander Killing

Colon PDX Model (CEA\textsuperscript{Medium})

Gastric PDX Model (CEA\textsuperscript{Medium})

Superior Efficacy in Lead Competitor Resistant Primary Tumor Models

Notes:
- Lead Competitor biosimilar used as benchmark
- Data is from preclinical models

*Research Innovation
Reduced Interference by Soluble MUC1

CD16A Highly Expressed in MUC1+ Tumors

- Highly expressed in lung, GI and breast cancers, e.g., ~90% lung adenocarcinoma is MUC1 moderate/high*
- Target MUC1 membrane proximal epitope to avoid sink effect via minimal soluble MUC1 binding
- Pursue NK engaging BsAb since NK activating receptor CD16A is highly expressed in MUC1+ tumors
- Differentiated design of MUC1 x CD16A BsAb to enhance NK cell engagement and tumor cell killing
  - High binding affinity for CD16A
  - WT Fc to engage FcR binding without increasing NK cell fratricide
  - Spatially close between MUC1 and CD16A arms
- Candidate selected and to enter clinic in 2024

*Data on file
LUAD: lung adenocarcinoma; LUSC: lung squamous cell carcinoma; BRCA: breast invasive carcinoma; STAD: stomach adenocarcinoma; CRC: colorectal cancer
Claudin6 x CD3 BsAb
Highly tumor specific TAA/T-cell engager to treat lung & gynecologic cancers

- **Very clean TAA, highly tumor-specific**
  - Overexpressed in ~30% non-squamous lung cancer and additional cancer types including ovarian cancer*

- **Claudin6 specificity is challenging to achieve**
  - Claudin9 differing by 3 amino acids from Claudin6 has broad expression in normal tissues

- **Key highlights of BeiGene’s Claudin6 x CD3 BsAb**
  - Highly selective against Claudin9
  - Adopted Fab x ScFv format to shorten spatial distance between Claudin6 and CD3 arms for better tumor cell killing
  - Efficacious in immune cold tumor model
  - Designed to overcome antigen heterogeneity through low antigen dependency and bystander effect

- **Candidate selected and to enter clinic in 2024**

* Amgen, AACR Annual Meeting 2022
We broadly cover lung cancer patient segments

- EGFR mutation, KRAS mutation, and MTAP deletion account for over half of the lung cancers
- MUC1, B7-H3, CEA and Claudin6 represent distinct but overlapping populations in lung, providing multiple approaches to target lung cancer

**Fully integrated CMC and manufacturing capabilities** across multiple modalities empower a fast path to the clinic

Established early-stage clinical trial network and internalized clinical development capability enable **quick clinical proof-of-concept**

(have engaged with over 700 global clinical sites for lung cancer trials)

**Diverse and innovative opportunities** in internal combinations with other targeted therapies or IO agents
Oncology Portfolio Heatmap
Deeply invested in key tumor types with multiple modalities

**Programs and Targets**
- **Lung**
  - TIM-3
  - Ox40
  - CT a
  - CT b
  - B7-H3
  - CEA
  - ADC a
  - ADC b
  - ADC c
  - ADC d
  - ADC e
  - ADC f
  - ADC g
  - ADC h
  - ADC i
  - ADC j
  - BsAb a
  - MUC1 x CD16A
  - Claudin6 x CD3
  - MUC1
  - BsAb c
  - BsAb d
  - BsAb e
  - CEA x 4-1BB
  - TsAb a
  - TsAb b
  - TsAb c
  - Recombinant a
- **Upper GI**
- **Colorectal**
- **BREAST**
- **Head and Neck**
- **B-cell malignancy**
- **AML/MDS**
- **PAN tumor**

**Sm**: small molecule; **mAb**: monoclonal antibody; **CT**: cell therapy; **BsAb**: bispecific antibody; **TsAb**: trispecific antibody; **Recombinant**: recombinant protein.
### Diversified Modalities and Broad Technology Platforms
Accelerating innovations at scale

<table>
<thead>
<tr>
<th>Development Stage</th>
<th>Small Molecule</th>
<th>CDAC</th>
<th>mAb</th>
<th>BsAb/TsAb</th>
<th>ADC</th>
<th>Cell Therapy</th>
<th>mRNA</th>
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<td>Discovery</td>
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</table>

mAb: monoclonal antibody; BsAb/TsAb: bispecific/trispecific antibody; CDAC: chimeric degradation activating compound; ADC: antibody drug conjugate
Accelerating Next Wave of Innovation
15+ molecules planned to enter the clinic in next 18 months

- EGFR CDAC
- PanKRAS inhibitor
- PRMT5 MTA-cooperative inhibitor
- CEA ADC
- B7H3 ADC
- MUC1 x CD16a
- Claudin 6 x CD3
- Undisclosed

Undisclosed with diverse modalities
1st cell therapy
1st mRNA

10+

10

5

CDK4
DGKζ
CCR8
PVRIG
HPK1 alternative scaffold

1–2 Per year
2013–2020

2
2021

3
2022

5
2023

10
2024

2025–beyond
Develop diverse and compelling programs across hematology & solid tumors

A comprehensive tumor type approach with lung cancer as example
3 exciting small molecules, 2 ADCs and 2 bi-specifics with differentiated TAA approaches

Lead the industry in breadth of novel modality designs to deliver potential breakthrough medicines
(small molecules, CDACs, mAbs, bi/tri-specifics, ADCs, cell therapies and mRNAs)

Combine differentiated targets with novel modalities across tumor types to deliver improved patient outcomes (as with our lung cancer portfolio)

*starting from 2024
R&D Key Takeaways
Scientific innovation with quality and speed to better serve patients around the world

1. **Leading in hematology:** Developing potentially BIC sonrotoclax (BCL2i) and FIC BTK CDAC in addition to BRUKINSA and expanding to additional heme malignancies

2. **Advancing broad solid tumor portfolio:** Expanding beyond I-O into oncogenic signaling target therapies and TAA therapies; targeting additional important tumor types with novel agents

3. **Research innovation:** broad portfolio with scientifically driven molecules based on diversified modalities across tumor types to improve patient outcomes
Closing Remarks

John V. Oyler
Co-Founder, Chairman and CEO
Leading Global Oncology Powerhouse

Largest dedicated oncology R&D team

Brodest reach of internally-run global clinical trials

Innovative oncology pipeline with 23 development programs and 60+ discovery programs

Emerging global leadership in hematology & foundation in solid tumors

Among mid-cap companies, up to $50bn market cap as of July 11, 2023
$17B+ BTKi market by 2028, with $15B in heme-onc and $2B+ outside of oncology

$4B+ BCL2i hematology market by 2028

• Hundreds of thousands of patients with BTKi resistance
• Potential BTK market expansion through DLBCL and other indications

$22B market opportunity – Emerging leadership

Source: Evaluate Pharma as of July 11, 2023, projection for 2028
Harnessing Science to Improve Access and Affordability for Cancer Patients Around the World

~800,000 patients and counting...
Striving to Reach Billions Worldwide
## Key Catalysts in 2H 2023

### Data Readouts
- **BRUKINSA (BTK inhibitor)**: ALPINE PFS long-term follow-up data
- **Sonrotoclax (BCL2 inhibitor)**: Phase 1/2 data
- **BGB-16673 (BTK degrader)**: Phase 1 data

### Regulatory Actions
- **Tislelizumab (PD-1 antibody)**: Approval in U.S. for 2L ESCC*
- **Sonrotoclax (BCL2 inhibitor)**: Approval in EU for 2L ESCC
- **Tislelizumab (PD-1 antibody)**: Approval in China for 1L HCC
- **Sonrotoclax (BCL2 inhibitor)**: 1L ESCC and GC filings in NVS territory

### Pipeline Progress
- **Sonrotoclax (BCL2 inhibitor)**: Initiate global Phase 3 trial in CLL in combination with BRUKINSA
- **Ociperlimab (TIGIT inhibitor)**: Complete enrollment in AdvanTIG-302 trial in NSCLC
- **CCR8, DGKζ, PVRIG, CDK4i**: Initiate first-in-human trials

*Original PDUFA date deferred*
Q&A Session & Panelists

John V. Oyler
Co-Founder, Chairman and CEO

Mehrdad Mobasher, M.D., M.P.H.
Chief Medical Officer, Hematology

Lai Wang, Ph.D.
Global Head of R&D

Mark Lanasa, M.D.
Chief Medical Officer, Solid Tumors

Julia Wang
Chief Financial Officer

Josh Neiman
Chief Commercial Officer, North America and Europe

Christiane Langer, M.D.
SVP, Global Medical Affairs (Ex-China)
Thank You!
Breakout Sessions and Panelists

1. Research
   - Lai Wang, Ph.D.
     Global Head of R&D
   - Chichi Huang
     VP, Head of Biologics

2. Clinical Development
   - Mark Lanasa, M.D.
     CMO, Solid Tumors
   - Mehrdad Mobasher
     M.D., M.P.H.
     CMO, Hematology

3. Commercial and Medical Affairs
   - Josh Neiman
     Chief Commercial Officer
     North America and Europe
   - Christiane Langer, M.D.
     SVP, Global Medical Affairs
     (Ex-China)

4. Manufacturing and Supply Chain
   - Kyu-Sung Lee
     SVP, Global Head of Technical Operations and Manufacturing
   - Kyoung Lim
     VP, Supply Chain