Today’s Participants

Xiaodong Wang, Ph.D.  
Chairman of Scientific Advisory Board & Co-Founder

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

Eric Hedrick, M.D.  
Chief Advisor

Howard Liang, Ph.D.  
CFO & Chief Strategy Officer

Jane Huang, M.D.  
Chief Medical Officer, Hematology

Yong (Ben) Ben, M.D.  
Chief Medical Officer, Immuno-Oncology

Lai Wang, Ph.D.  
SVP, Head of Global Research & APAC Clinical Development
Agenda

Howard Liang, Ph.D.
CFO and Chief Strategy Officer
Disclosures

- 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 pre-clinical and early-stage clinical programs and plans; recent clinical data for BeiGene’s product candidates and approvals of its products; the conduct of late-stage clinical trials and expected data readouts; additional planned commercial product launches; the advancement of and anticipated clinical development, regulatory milestones and commercialization of BeiGene’s products 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 products and drug candidates, if approved; BeiGene’s ability to obtain and maintain protection of intellectual property for its technology and drugs; BeiGene’s reliance on third parties to conduct drug development, manufacturing and other services; BeiGene’s limited operating history and BeiGene’s ability to obtain additional funding for operations and to complete the development and commercialization of its drug candidates; the impact of the COVID-19 pandemic on the Company’s clinical development, commercial and other operations, as well as those risks more fully discussed in the section entitled “Risk Factors” in BeiGene’s most recent quarterly report on Form 10-Q, as well as discussions of potential risks, uncertainties, and other important factors in BeiGene’s subsequent filings with the U.S. Securities and Exchange Commission. All information in this press release is as of the date of this press release, and BeiGene undertakes no duty to update such information unless required by law.

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- This presentation and the accompanying oral presentation contain data and information obtained from third-party studies and internal company analysis of such data and information. BeiGene has not independently verified the data and information obtained from these sources. Forward-looking information obtained from these sources is subject to the same qualifications noted above.
Agenda

• **Introduction** – Howard Liang

• **Opening Remarks** – Xiaodong Wang
  BeiGene’s research foundation

• **BeiGene’s Drug Discovery Engine** – Video Presentation
  People, capabilities, and facilities

• **Research and Internally Developed Assets** – Lai Wang
  TIGIT, Bcl-2, OX40, and HPK1

• **In-Licensed Programs** – Eric Hedrick
  Sotorasib (AMG 510), Sitravatinib, and Zanidatamab (ZW25)

• **Concluding Remarks** – John V. Oyler

• **Q&A**
Opening Remarks

Xiaodong Wang, Ph.D.
Chairman of Scientific Advisory Board, Co-Founder and Member of Board of Directors
Founded with the Goal of the Best Medicine for the Most People

The founding of a science-based company 10 years ago in China

- Why in China?

Our philosophy: BeiGene believes the path to the best medicine is following the science

- Believe in a core research principle to follow the science: evidence and logic, not hearsay or portfolio decoration
- Pursue programs with best-in-class differentiation
- Be unafraid to terminate subpar programs at any time

BeiGene’s research team is productive and has delivered

- Two drugs approved and a third one at the filing stage, all with clinically differentiated properties
- 11 internally developed molecules advanced into the clinic, all with pre-clinically differentiated properties
BeiGene’s Drug Discovery Engine Video Presentation
The Discovery Engine

Lai Wang, Ph.D.
SVP, Head of Global Research and APAC Clinical Development
Executive Summary

BeiGene has built an exceptional research organization with broad capabilities & scope

- Strong organization built over last decade and attracted outstanding talent
- Broad capabilities exist in this team to attack cancer through many modalities and targets

This team has shown proven internal research track record of success

- 11 molecules delivered to the clinic in the first 10 years
- Two of these approved and one at the filing stage
  - Outstanding clinical data demonstrated for each

We have created a robust early clinical pipeline

- Potentially differentiated compounds against TIGIT, Bcl-2, OX40
- Potentially first-in-class program in HPK1
- Compelling internal combination opportunities
- Planning to accelerate TIGIT program into Phase 3
The Discovery Engine

- Research Organization, Capabilities
- Proven Internal Research Track Record
- Robust Promising Pipeline
Integrated Research Capabilities Offer Opportunities to Address Wide Range of Biological Problems

• Comprehensive small molecule and biologics discovery engine
• Efficient portfolio management
• Striving for seamless transition to manufacturing and clinical development
• Moving beyond oncology to areas such as I/I
• Cutting-edge tools such as PROTAC, bispecific Ab, and ADC
• Pursuing 10+ potentially best-in-class and first-in-class projects with the plan to double that in one year
Expansion of BeiGene Research

**Beijing Research Center**
(ONLY 1\(^{st}\) AND 2\(^{nd}\) FLOOR)
- Team size <200
- 6-8 preclinical programs

**Beijing Research Center**
- Team size 350+
- ~12 preclinical programs

**Beijing Research Center**
**Shanghai Research Center**
- Team size 650+
- Capability for ~24 preclinical programs

- 2011–2018
- TODAY
- PLANNED IN ONE YEAR
The Discovery Engine

• Research Organization, Capabilities

• **Proven Internal Research Track Record**

• Robust Promising Pipeline
Proven Internal Research Track Record

**Two approved products, with a third at filing stage**

- **BRUKINSA (zanubrutinib) approved in US and China**
  - First China-discovered compound to be approved by the FDA and granted Breakthrough Therapy Designation
  - Highly selective, complete and sustained target inhibition in tumor tissue
  - Improved safety profile shown in Phase 3 head to head trial despite missing the primary efficacy endpoint
- **Tislelizumab approved in China for 2 indications, with 3 additional indications under review**
  - Differentiated MOA by completely removing Fc function, thus avoiding macrophage mediated T-cell elimination
  - High complete response rate in lead indication cHL
- **Pamiparib at filing stage in China**
  - Demonstrated brain penetration in preclinical models, potential for treating brain tumor and brain metastasis
  - Not a drug pump substrate, preventing a potential resistance mechanism that has been reported for other PARPi in clinic

**11 molecules discovered in-house and advanced into clinic in the last 10 years**

- Broad range of I/O programs including differentiated OX-40, TIGIT
- Compelling and challenging Bcl-2 program
The Discovery Engine

- Research Organization, Capabilities
- Proven Internal Research Track Record
- Robust Promising Pipeline
## Internal Capabilities and Collaborations Create Robust Pipeline

25+ assets, 8 with global rights

<table>
<thead>
<tr>
<th>COMPOUND</th>
<th>(TARGET) / PROGRAM</th>
<th>DOSE ESC.</th>
<th>DOSE EXPANSION</th>
<th>PIVOTAL</th>
<th>COMMERCIAL RIGHTS</th>
<th>PARTNER</th>
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<td>BGB-A1217</td>
<td>(TIGIT) + tislelizumab</td>
<td>Solid tumors</td>
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<td>PH1a</td>
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<td>(TIM-3) Mono, + tislelizumab</td>
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<td>BGB-11417</td>
<td>(Bcl-2) Mono, + zanubrutinib</td>
<td>B-cell malignancies</td>
<td>Phase 1 study startup ongoing</td>
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<td>BGB-15025</td>
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<td>IND Enabling studies ongoing</td>
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<td>BGB-10188</td>
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<td>B-cell + solid malignancies</td>
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<td>Ilirafenib</td>
<td>(RAF dimer)</td>
<td>B-RafK-RAS/N-RAS mut, solid tumors</td>
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<td>BA3017</td>
<td>(CTLA4) Mono, + tislelizumab</td>
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<td>AMG 510</td>
<td>(KRAS G12C)</td>
<td>Solid Tumors, NSCLC, CRC</td>
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<td>AMG 701</td>
<td>(BCMA)</td>
<td>MM</td>
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<td>AMG 176</td>
<td>(Mcl-1, SM (l.v.))</td>
<td>Hematologic malignancies</td>
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<td>Amgen</td>
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<td>AMG 397</td>
<td>(Mcl-1, SM (oral))</td>
<td>Hematologic malignancies</td>
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<td>Amgen</td>
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<tr>
<td>AMG 330</td>
<td>(CD33)</td>
<td>Myeloid malignancies</td>
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<td>AMG 673</td>
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<td>AML</td>
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<td>SCLC</td>
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<td>AMG 160</td>
<td>(PSMA)</td>
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<td>AMG 506</td>
<td>(FAP x 4-1BB, DARPin®)</td>
<td>Solid Tumors</td>
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<td>Amgen</td>
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<td>AMG 199</td>
<td>(MUC17)</td>
<td>GO/GEJC</td>
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<td>Amgen</td>
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<tr>
<td>Sitravatinib</td>
<td>(multi-kinase inhibitor) + tislelizumab</td>
<td>NSCLC, RCC, OC, MEL</td>
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<td>Amgen</td>
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<td>Zanidatamab</td>
<td>(HER2, bispecific antibody)</td>
<td>Breast cancer, GEA</td>
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<td>Mirati</td>
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<td>ZW49</td>
<td>(HER2, bispecific ADC)</td>
<td>Planned (in Ph1 ex-China by Zymeworks)</td>
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<td>Zymeworks</td>
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<td>BGB-3245</td>
<td>(B-RAF)</td>
<td>Solid tumors</td>
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<td>SpringWorks1</td>
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<td>SEA-CD70</td>
<td>(anti-CD70)</td>
<td>Planned (starting Ph1 ex-Asia by Seattle Genetics)</td>
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<td>Seattle Genetics</td>
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<td>DKN-01</td>
<td>(OKK1) + tislelizumab ± chemo</td>
<td>Trials in GC/GEJC planned</td>
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<td>Leap Therapeutics</td>
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</tr>
</tbody>
</table>

†Addition compounds from Amgen collaboration not yet disclosed

Cancer Immunotherapy

- **Sitravatinib (TAM)**
  - MDSC

- **Sitravatinib**
  - CAB-CTLA4
  - OX40
  - TIGIT
  - Treg

- **TIM-3**
  - OX40
  - Dendritic Cell

- **Lenalidomide**

- **Sitravatinib (VEGFR)**
  - PD-1/L1
  - OX-40
  - TIM-3
  - TIGIT
  - FAP-4-1BB

- **Cytotoxic T Cell**
  - CTL

- **Natural Killer Cell**
  - NK cells

Source: Modified from Daniel S. Chen, Immunity, 2013; CAB=Conditional Active Biologics
Tumor-Targeted Therapy

**KrasG12Ci (NSCLC)**
**DLL3-CD3 (SCLC)**
**HER2 BsAB/BsAB-ADC (HER2+ GC, BTC, CRC)**
**MUC17-CD3 (GC)**
**PARPi (GC)**
**KrasG12Ci (CRC, PC)**
**BRAFi (CRC)**
**PSMA-CD3 (Prostate)**
**PARPi (Prostate)**

**HER2 BsAB/BsAB-ADC (HER2+ GC, BTC, CRC)**
**MUC17-CD3 (GC)**
**PARPi (GC)**
**KrasG12Ci (CRC, PC)**
**BRAFi (CRC)**

**PSMA-CD3 (Prostate)**
**PARPi (Prostate)**

**BTKi (NHL)**
**Revlimid (NHL)**
**Blincyto (NHL)**
**CD19-CD3 (NHL)**

**KrasG12Ci (NSCLC)**
**DLL3-CD3 (SCLC)**

**EGFRvIII-CD3 (GBM)**
**PARPi (GBM)**

**PARPi (BC)**
**HER2 BsAB/BsAB-ADC (HER2+ BC)**

**Blincyto (ALL)**
**Vidaza (AML/MDS)**
**Mcl1i (AML)**
**CD33-CD3 (AML)**
**FLT3-CD3 (AML)**
**CD70 (AML)**
**BTKi (CLL)**

**BTKi (NHL)**
**Revlimid (NHL)**
**Blincyto (NHL)**
**CD19-CD3 (NHL)**

**PARPi (OC)**

**Revlimgid (MM)**
**Kyprolis (MM)**
**Mcl1i (MM)**
**BCMA-CD3 (MM)**

**Small Molecule Biologics**
Robust Promising Pipeline

• BGB-A1217 (TIGIT Antibody)
• BGB-11417 (Bcl-2 Inhibitor)
• BGB-A445 (Non Ligand-Competing OX40 Antibody)
• BGB-15025 (HPK1 Inhibitor)
Executive Summary: TIGIT Program

Encouraging POC data on tiragolumab/atezolizumab (Roche) at ASCO 2020

BGB-A1217 (TIGIT mAb) is one of the three most advanced TIGIT antibodies with full Fc function and RP2D for PD-(L)1 combination

• ~4x more potent than tiragolumab in preclinical studies
• Competent Fc required for efficacy based on preclinical data
• Combination with tislelizumab generally well-tolerated, no DLT, recommended Phase 2 dose (RP2D) identified

Potential to compete globally, possibly transformative

• Registrational program being planned

Source: 1. Internal Data
TIGIT Ab Activates T/NK Cells by Blocking TIGIT and Ligand Interaction

- TIGIT shares its ligand PVR (CD155) and PVR-L2 (CD112) with the activating receptor CD226 (DNAM-1)

- BGB-A1217 blocks the binding of PVR/PVR-L2 to TIGIT and reactivates T effector cells and NK cells by:
  - Suppressing TIGIT-mediated inhibitory signaling
  - Increasing ligand availability for CD226 co-stimulatory receptor

Sources: Iguchi-Manaka et al., PLoS One, 2016; Johnston RJ et al., Cancer Cell, 2014
Study Design (CITYSCAPE)

**1L Stage IV NSCLC**
- EGFR/ALK wild-type
- Tumor PD-L1 TPS ≥ 1% by 22C3 IHC by local or central assay
- N=135

Stratification Factors:
- PD-L1 TPS (1-49% vs ≥ 50%)
- Histology (Non-Squamous vs Squamous)
- Tobacco use (yes vs no)

Results:

<table>
<thead>
<tr>
<th>ITT (TPS≥1%) N=135</th>
<th>PD-L1 high (TPS≥50%) n=58</th>
<th>PD-L1 low (TPS≥1-49%) n=77</th>
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</thead>
<tbody>
<tr>
<td>Tiragolumab + Tecentriq</td>
<td>Tiragolumab + Tecentriq</td>
<td>Tiragolumab + Tecentriq</td>
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<tr>
<td>Placebo + Tecentriq</td>
<td>Placebo + Tecentriq</td>
<td>Placebo + Tecentriq</td>
</tr>
</tbody>
</table>

**ORR % (Follow-up 10.9 months)**
- 37 21
- 66 24
- 16 18

**mPFS, months (Follow-up 10.9 months)**
- 5.55 3.88
- NE 4.11
- 4.04 3.58

- **Co-Primary Endpoints: ORR and mPFS**
- **Key Secondary Endpoints: Safety, DOR, OS, Patient-reported outcomes (PROs)**
- **Exploratory Endpoints: Efficacy analysis by PD-L1 status**

Updated Investigator-Assessed PFS: ITT

Updated Investigator-Assessed PFS: PD-L1 TPS ≥ 50%

Source: Rodriguez-Abreu, D et al., ASCO 2020; NE, Not evaluable.
Fc Effector Function Appears Critical for Anti-Tumor Activity of TIGIT Ab

Multiple MOA may exist for competent Fc
1. Fc/FcγR co-engagement enhances T cell responsiveness by enhancing the quality of immune synapse
2. Fc/FcγR engagement on myeloid cell creates proinflammatory TME by activating myeloid cells

Sources: Internal data; Adopted and modified from Dahan R. et al, Cancer Cell 2015; Jeremy D. Waight et al., Cancer Cell, 2019
## TIGIT Competitive Landscape

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<tr>
<th>FORMAT</th>
<th>HYPOTHESIS</th>
<th>DRUG NAME</th>
<th>COMPANY</th>
<th>COMBO DOSE</th>
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<tbody>
<tr>
<td>WT IgG1</td>
<td>WT IgG1 required for maximal efficacy based on preclinical studies</td>
<td>Tiragolumab</td>
<td>Roche</td>
<td>600 mg Q3W</td>
<td>Ph1 initiated in May 2016 Ph2 in cervical cancer planned in Jun 2020 Ph3 in SCLC initiated in Feb 2020 Ph3 in NSCLC initiated in Mar 2020</td>
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<tr>
<td></td>
<td></td>
<td>Vibostolimab</td>
<td>Merck</td>
<td>Not disclosed</td>
<td>Ph1 initiated in Dec 2016 Ph1/2 in melanoma planned in Apr 2020 Ph2 in NSCLC initiated in Jan 2020</td>
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<tr>
<td></td>
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<td>Etigilimab</td>
<td>OncoMed/Mereo</td>
<td>NA</td>
<td>Ph1 initiated in May 2017</td>
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<td></td>
<td></td>
<td>BGB-A1217</td>
<td>BeiGene</td>
<td>Not disclosed</td>
<td>Ph1 initiated in Aug 2019 with combo escalation from beginning</td>
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<td>TSGN-TG</td>
<td>Seattle Genetics</td>
<td>NA</td>
<td>Ph1 initiated in Apr 2020</td>
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<td>EOS-884448</td>
<td>iTeos</td>
<td>NA</td>
<td>Ph1 initiated in Feb 2020</td>
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<tr>
<td>Mutant IgG1</td>
<td>Less effective</td>
<td>AB-154</td>
<td>Arcus/Gilead</td>
<td>NA</td>
<td>Ph1 initiated in Aug 2018 Ph2 in NSCLC initiated in Jan 2020</td>
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<td>BMS-986207</td>
<td>BMS</td>
<td>NA</td>
<td>Ph1 initiated in Nov 2016</td>
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<tr>
<td>WT IgG4</td>
<td>Less effective</td>
<td>ASP-8374</td>
<td>Astellas/Potenza</td>
<td>NA</td>
<td>Ph1 initiated in Sep 2017</td>
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<td>COM902</td>
<td>Compugen</td>
<td>NA</td>
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<td>IBI-939</td>
<td>Innovent</td>
<td>NA</td>
<td>Ph1 planned in May 2020</td>
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**Mutant IgG1**

- **WT IgG1**
  - **Tiragolumab**
    - **Roche**
    - **600 mg Q3W**
      - **Ph1 initiated in May 2016**
      - **Ph2 in cervical cancer planned in Jun 2020**
      - **Ph3 in SCLC initiated in Feb 2020**
      - **Ph3 in NSCLC initiated in Mar 2020**
  - **Vibostolimab**
    - **Merck**
    - **Not disclosed**
      - **Ph1 initiated in Dec 2016**
      - **Ph1/2 in melanoma planned in Apr 2020**
      - **Ph2 in NSCLC initiated in Jan 2020**
  - **Etigilimab**
    - **OncoMed/Mereo**
    - **NA**
      - **Ph1 initiated in May 2017**
  - **BGB-A1217**
    - **BeiGene**
    - **Not disclosed**
      - **Ph1 initiated in Aug 2019 with combo escalation from beginning**
  - **TSGN-TG**
    - **Seattle Genetics**
    - **NA**
      - **Ph1 initiated in Apr 2020**
  - **EOS-884448**
    - **iTeos**
    - **NA**
      - **Ph1 initiated in Feb 2020**

**Mutant IgG1**

- **AB-154**
  - **Arcus/Gilead**
  - **NA**
    - **Ph1 initiated in Aug 2018**
    - **Ph2 in NSCLC initiated in Jan 2020**
  - **BMS-986207**
    - **BMS**
    - **NA**
      - **Ph1 initiated in Nov 2016**

**WT IgG4**

- **ASP-8374**
  - **Astellas/Potenza**
  - **NA**
    - **Ph1 initiated in Sep 2017**
  - **COM902**
    - **Compugen**
    - **NA**
      - **Ph1 initiated in Mar 2020**

**Not Disclosed**

- **IBI-939**
  - **Innovent**
  - **NA**
    - **Ph1 planned in May 2020**
BGB-A1217 Program Moving Aggressively Towards Registration Trial

- Four-fold more potent than tiragolumab (Roche)\(^1\)
- BGB-A1217 Phase 1 combination with tislelizumab ongoing in advanced solid tumors
- Generally well-tolerated, no DLT, **combination recommended phase 2 dose has been determined**
- Full target occupancy was observed in PBMCs at lowest dose level
- Moving aggressively towards registration trial

![Graph showing IC50 values](image)

**Source:** 1. Internal Data; DLT: does limiting toxicities; PBMCs: peripheral blood mononuclear cells.
Robust Promising Pipeline

- BGB-A1217 (TIGIT Antibody)
- BGB-11417 (Bcl-2 Inhibitor)
- BGB-A445 (Non Ligand-Competing OX40 Antibody)
- BGB-15025 (HPK1 Inhibitor)
Executive Summary: Bcl-2 Program

BGB-11417 is a potential best-in-class Bcl-2 inhibitor

- Potent Bcl-2 inhibitor, with potential to overcome resistance to venetoclax
- Ability to be dosed high if needed, e.g. for solid tumor indications
- More selective than venetoclax for Bcl-2 relative to Bcl-xL
- NOAEL in animal GLP tox studies with exposure close to 30-fold higher than predicted human therapeutic exposure\(^1\)
  - Well-positioned to be combined with zanubrutinib, BeiGene’s potentially best-in-class BTK inhibitor

BGB-11417 FIH study ongoing

- Dose escalation initiated early this year, currently at 80 mg QD, which is predicted to be equivalent to 400 mg of venetoclax
- Combination trial with zanubrutinib planned to be initiated H2 2020

Source: 1. Internal Data
BGB-11417 Was More Potent and Selective than Venetoclax in Biochemical and Cellular Assays

<table>
<thead>
<tr>
<th></th>
<th>BGB-11417</th>
<th>Venetoclax</th>
<th>Potency Improvement (Fold, BGB-11417/Venetoclax)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bcl-2 WT (Biochemical IC50, nM)</td>
<td>0.035</td>
<td>1.3</td>
<td>37</td>
</tr>
<tr>
<td>Bcl-2 G101V (Biochemical IC50, nM)</td>
<td>0.28</td>
<td>34</td>
<td>121</td>
</tr>
<tr>
<td>Bcl-2 WT (Cell Proliferation IC50, nM)</td>
<td>0.42</td>
<td>3.4</td>
<td>8.1</td>
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<tr>
<td>Bcl-2 G101V (Cell Proliferation IC50, nM)</td>
<td>4.6</td>
<td>75</td>
<td>16.3</td>
</tr>
<tr>
<td>Fold selectivity (TF-FRET assay) Bcl-xL; Mcl-1; Bcl-w; Bcl-2A1</td>
<td>&gt;1000; &gt;1000; &gt;1000; &gt;1000</td>
<td>325; &gt;1000; &gt;1000; &gt;1000</td>
<td></td>
</tr>
</tbody>
</table>

Bcl-2 G101V mutation emerged as resistance to Venetoclax in clinic

Source: Internal data, Blombery et. al., Cancer Discovery AACR Journals Dec 2018
BGB-11417 Was More Efficacious than Venetoclax in Both Wild-Type and Bcl-2-G101V Xenograft Models

**BGB-11417 is more efficacious in RS4:11 model**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mean Tumor Volume (mm³ ± SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vehicle</td>
<td>0</td>
</tr>
<tr>
<td>5 mpk</td>
<td>1000</td>
</tr>
<tr>
<td>15 mpk</td>
<td>2000</td>
</tr>
</tbody>
</table>

**BGB-11417 is efficacious in RS4:11-G101V model**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mean Tumor Volume (mm³ ± SEM)</th>
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<tr>
<td>15 mpk</td>
<td>2000</td>
</tr>
</tbody>
</table>

Source: Internal Data
Bcl-2 Inhibitors Demonstrated Activity in Solid Tumors

Breast cancer

- Bcl-2 is overexpressed in approximately 80% of primary ER+ breast cancer\(^1,2\). Bcl-2 is often expressed at high levels in poorer-prognosis luminal B tumors, as well as good-prognosis luminal A tumors\(^3\).

- Combining venetoclax with endocrine therapy had a tolerable safety profile and elicited notable activity in ER and Bcl-2-positive metastatic breast cancer. For 24 patients treated at the RP2D, the confirmed radiologic response rate was 54% and the clinical benefit rate was 75%\(^4\).

- Venetoclax 800 mg/day was selected as the RP2D in combination with tamoxifen; no higher doses were explored due to the potential “pill burden”, while BGB-11417 may not have this issue.

- Dual targeting of CDK4/6 and Bcl-2 pathways augmented tumor response in ER+ breast cancer. The effect was associated with increased apoptosis\(^5\).

SCLC

- Dual Bcl-2 and Bcl-xL inhibitor, navitoclax (ABT-263) showed preliminary clinical benefit in SCLC\(^6\).

- Preclinical cell line screen and PDX experiments showed high Bcl-2 expression conferred sensitivity of SCLC to venetoclax\(^7\).

Robust Promising Pipeline

- BGB-A1217 (TIGIT Antibody)
- BGB-11417 (Bcl-2 Inhibitor)
- **BGB-A445 (Non Ligand-Competing OX40 Antibody)**
- BGB-15025 (HPK1 Inhibitor)
Executive Summary: OX40 Program

BGB-A445 (OX40 agonist antibody) is differentiated from all other OX40 Abs in the clinic

• Does not disrupt OX40-OX40L engagement
  ➢ Retains OX40L signaling on antigen presenting cells
  ➢ Achieves maximal OX40 activation by keeping natural ligand stimulation
• Widely efficacious as monotherapy in preclinical models, including PD-1 resistant models
• Has shown combo effect with PD-1 Ab, TLR9 agonist, PI3Kδ inhibitor, sitravatinib and chemo in preclinical models

Phase 1 clinical trial ongoing

• Monotherapy dose escalation ongoing
• Combination dose escalation trial with tislelizumab is planned to start in H2 2020
BGB-A445 is a Non-ligand Blocking OX40 Antibody, Differentiated from Other Clinical OX40 Antibodies

At lower concentration, a ligand blocking OX40 Ab promotes T cell proliferation and activation.

At higher concentration, a ligand blocking OX40 Ab blocks OX40-OX40L interaction, impairing the activation of APC.

At lower concentration, a non-ligand blocking OX40 Ab promotes T cell proliferation and activation.

At higher concentration, a non ligand-blocking OX40 Ab does not affect OX40-OX40L interaction, maintaining APC activation and promoting maximum T cell proliferation and activation.

APC = antigen presenting cell
BGB-445 Showed Dose-Response, While Competitor’s OX40 Ab Showed Hook Effect in MC38 OX40 Humanized Mice Model

Competitor OX40 Abs Showed Limited Efficacy in Clinic, Mainly at Low Dose Levels

<table>
<thead>
<tr>
<th>Name</th>
<th>MED04562</th>
<th>MOXR0916</th>
<th>PF-04518600</th>
<th>BMS-986178</th>
<th>ABBV-368</th>
<th>GSK3174998</th>
</tr>
</thead>
<tbody>
<tr>
<td>Company</td>
<td>AstraZeneca</td>
<td>Genentech</td>
<td>Pfizer</td>
<td>BMS</td>
<td>Abbvie</td>
<td>GSK</td>
</tr>
<tr>
<td>Dose Range</td>
<td>0.03-10 mg/kg (mono) 0.04-0.4 mg/kg (combo)</td>
<td>0.01-20 mg/kg</td>
<td>0.01–10 mg/kg (mono) 0.1-3 mg/kg (combo)</td>
<td>0.3-5 mg/kg</td>
<td>0.01 to 3.0mg/kg</td>
<td>0.003-10 mg/kg</td>
</tr>
<tr>
<td>Dose Level of Objective Responses</td>
<td>Mono (50 pts): 1 PR@0.03 mg/kg 1 PR@3 mg/kg</td>
<td>Mono expansion @ 5mpk (17 pts): 2 PR</td>
<td>Mono (49 pts): 1 PR@0.1 mg/kg 1 PR@0.3 mg/kg</td>
<td>Combo with Nivo (16 pts): 3 PR, 1 PR@5mg/kg; other two unknown</td>
<td>Mono (36 pts): 1 PR@0.01 mg/kg</td>
<td>Mono (45 pts): 1 PR@0.3 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Combo with Durva (26 pts): 2 <a href="mailto:PR@0.1mg">PR@0.1mg</a>/kg 1 PR@0.4 mg/kg</td>
<td>Combo with Atezo (51 pts): 1 PR@0.01 mg/kg 1 PR@0.2 mg/kg</td>
<td>Combo with α4-1BB (37 pts): 2 PR@0.3 mg/kg</td>
<td></td>
<td></td>
<td>Combo with Pembro (96 pts): 3 CR, 5 PR; 1 CR at 0.1 mg/kg, others unknown</td>
</tr>
</tbody>
</table>

# OX40 Competitive Landscape

<table>
<thead>
<tr>
<th>DRUG NAME</th>
<th>DESCRIPTION</th>
<th>COMPANY</th>
<th>DISEASE</th>
<th>STATUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>MOXR-0916</td>
<td>IgG1 OX40 agonist Block OX40L</td>
<td>Roche</td>
<td>Solid tumors</td>
<td>Ph1 initiated in Aug 2014 Development discontinued in 2017</td>
</tr>
<tr>
<td>MEDI-0562</td>
<td>IgG1 OX40 agonist Block OX40L</td>
<td>MedImmune/AZ</td>
<td>Solid tumors</td>
<td>Ph1 initiated in Mar 2016 Ph1b in HNSCC/melanoma initiated in Jul 2018 Ph2 in OC initiated in Jun 2018 Development discontinued in 2019</td>
</tr>
<tr>
<td>PF-04518600</td>
<td>IgG2 OX40 agonist Block OX40L</td>
<td>Pfizer</td>
<td>Solid tumors</td>
<td>Ph1 initiated in Apr 2015 Ph2 in RCC initiated in Sep 2017 Ph2 in TNBC initiated in Jul 2019 Development discontinued in 2019</td>
</tr>
<tr>
<td>GSK-3174998</td>
<td>IgG1 OX40 agonist Block OX40L</td>
<td>GSK</td>
<td>Solid tumors</td>
<td>Ph1 initiated in Sep 2015 Ph1 in MM initiated in Oct 2019</td>
</tr>
<tr>
<td>BMS-986178</td>
<td>IgG1 OX40 agonist Block OX40L</td>
<td>BMS</td>
<td>Solid tumors</td>
<td>Ph1 initiated in Jun 2016</td>
</tr>
<tr>
<td>INCAGN-1949</td>
<td>IgG1 OX40 agonist Block OX40L</td>
<td>Agenus/Icyte</td>
<td>Solid tumors</td>
<td>Ph1 initiated in Oct 2016</td>
</tr>
<tr>
<td>ABBV-368</td>
<td>IgG1 OX40 agonist Block OX40L</td>
<td>Abbvie</td>
<td>Solid tumors</td>
<td>Ph1 initiated in May 2017 Ph1b in HNSCC initiated in Jan 2020</td>
</tr>
<tr>
<td>IBI-101</td>
<td>IgG1 OX40 agonist Block OX40L</td>
<td>Innovent</td>
<td>Solid tumors</td>
<td>Ph1 initiated in Dec 2018</td>
</tr>
<tr>
<td>INBRX-106</td>
<td>OX40 agonist</td>
<td>Inhibrix/Elpiscience</td>
<td>Solid tumors</td>
<td>Ph1 initiated in Dec 2019</td>
</tr>
<tr>
<td>BGB-A445</td>
<td>IgG1 OX40 agonist</td>
<td>BeiGene</td>
<td>Solid tumors</td>
<td>Ph1 initiated in early 2020</td>
</tr>
</tbody>
</table>
OX40 Ab Was More Efficacious than PD-1 in Mouse Syngeneic Models

- OX40 Ab was active in 11/13 models while PD-1 Ab was only active in 7/13 models
- In most models where both agents were active, OX40 Ab showed stronger anti-tumor activity.

Source: Internal Data
OX40 Ab Has Shown Combination Activity with PD-1 Ab, PI3Kδ Inhibitor, Sitravatinib, TLR9 Agonist, and Chemo

**MC38 model**

- **Vehicle**
- **OX40 Ab**
- **PD1 Ab**
- **OX40 Ab + PD1 Ab**

**EMT6 model**

- **Vehicle**
- **OX40 Ab (10/25 TF)**
- **PI3Kδi (1/25 TF)**
- **PI3Kδi+OX40 Ab (19/25 TF)**

**CT26 model**

- **Vehicle**
- **OX40 Ab (10/20 TF)**
- **sitravatinib (1/20 TF)**
- **sitravatinib + OX40 Ab (16/20 TF)**

**A20 model**

- **Vehicle**
- **TLR9 agonist (2/12 TF)**
- **OX40 Ab (3/12 TF)**
- **TLR9 agonist + OX40 Ab (8/12 TF)**

**CT26 model**

- **Vehicle**
- **OX40 Ab**
- **PD1 Ab + OX40 Ab**
- **OX40 Ab+PD1 Ab+Oxaliplatin+5-FU**
- **OX40 Ab+PD1 Ab+Carboplatin**

Source: Internal Data
Ph1 Study Design and Current Status of BGB-A445 (OX40 Antibody)

Dose Escalation

- A445 20 mg
- A445 60 mg
- A445 150 mg
- A445 300 mg
- A445 600 mg
- A445 20 mg + tislelizumab
- A445 60 mg + tislelizumab
- A445 150 mg + tislelizumab
- A445 300 mg + tislelizumab
- A445 600 mg + tislelizumab

Dose Expansion

RP2D

BGB-A445 ± tislelizumab
2-4 cohorts of 20-40 pts/cohort

- Dose escalation schedule is BGB-A445 Q3W ± tislelizumab Q3W.

Abbreviations: RP2D, recommended Phase 2 dose.
Robust Promising Pipeline

- BGB-A1217 (TIGIT Antibody)
- BGB-11417 (Bcl-2 Inhibitor)
- BGB-A445 (Non Ligand-Competing OX40 Antibody)
- BGB-15025 (HPK1 Inhibitor)
Executive Summary: HPK1 Program

**BGB-15025 is a potentially first-in-class HPK1 inhibitor**

- HPK1 is a key negative feedback regulator of TCR signaling; inhibition of HPK1 enhances T cell activation
- Robust combination anti-tumor activity with PD-1 Ab in preclinical animal models\(^1\)
- Preliminary tox study suggests wide therapeutic window (~20-50 fold)

**IND submission expected Q4 2020**

Source: 1. Internal Data
HPK1 Negatively Regulates T-cell Receptor Signaling

Phosphorylation of the adaptor SLP-76 by HPK1 leads to degradation of SLP-76 which is crucial for T-cell activation.

Sources: Adopted and modified from Koretzky G. et al Nature Review Immunology 2006; Hernandez et S. et al, Cell Reports 25, 80–94, 2018
Strong Scientific Evidence Supports Critical Role for HPK1 in T-Cell Activation and Anti-Tumor Immunity

T-cells with reduced HPK1 catalytic activity show enhanced activation upon αCD3 treatment

Tumor rejection in GL261 model in HPK1 kinase dead mice

* M91A mutation reduces HPK1 kinase activity by ~50% in T cells.
* P<0.05; ** P<0.01, *** P<0.001, **** P<0.001

Sources: Saity Hernande et al, Cell Reports 25, 80–94, 2018
BGB-15025 Demonstrated Significant *in Vitro and in Vivo* PD Effect

**BGB-15025 increased IL2 production in PBMC**

**Splenic pSLP76 PD (6hr post treatment)**

*P<0.05; ** P<0.01, *** P<0.001 vs. Vehicle

Source: Internal Data
BGB-15025 showed significant combo efficacy with PD-1 antibody at as low as 1 mg/kg in CT26WT syngeneic model.

BGB-15025 demonstrated significant combo effects with PD-1 Ab at as low as 1 mg/kg in CT26WT syngeneic model.

Source: Internal Data
Productive Discovery Engine

Always science driven, proven record in target selection

Full internal capabilities, efficient portfolio management

Cutting edge technologies such as PROTAC, ADC, bsAb

Robust early pipeline (25+ in clinical stage and 10+ in preclinical), including:

- BGB-A1217 (TIGIT): One of three most advanced programs, Fc effector function competent, accelerating to registration trials
- BGB-11417 (Bcl-2): Potent Bcl-2 inhibitor, potentially overcomes venetoclax resistance
- BGB-A445 (OX40): The only endogenous ligand non-competing agent
- BGB-15025 (HPK1): Potentially first-in-class, prevents T-cell exhaustion
In-Licensed Programs

Eric Hedrick, M.D.
Chief Advisor
Executive Summary

External collaborations contribute significantly to our clinical pipeline:
- 25+ molecules across 9 collaborations
- Complementary with existing internal clinical and research programs
- Diversification of therapeutic modalities (e.g. Amgen BiTE platforms)
- Expansion of IO tislelizumab-based combination opportunities

Added focus on key disease indications:
- PD-1 sensitive Asia-prevalent tumor types (lung, liver, gastric) sitravatinib, zanidatamab (ZW-25), etc.
- NSCLC: Sotorasib (AMG 510)
- HER2-expressing cancers (breast, gastric) zanidatamab/ZW49

Anticipate several programs entering late-stage development within the next 6-18 months
### Internal Capabilities and Collaborations Create Robust Pipeline

25+ assets, 8 with global rights

<table>
<thead>
<tr>
<th>COMPOUND</th>
<th>(TARGET) / PROGRAM</th>
<th>DOSE ESC.</th>
<th>DOSE EXPANSION</th>
<th>PIVOTAL</th>
<th>COMMERCIAL RIGHTS</th>
<th>PARTNER</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>PH1a</td>
<td>PH1b</td>
<td>PH2*</td>
<td>PH2**</td>
<td>PH3</td>
</tr>
<tr>
<td>BGB-A1217</td>
<td>(TIGIT) + tislelizumab</td>
<td>Solid tumors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BGB-A445</td>
<td>(OX40) + tislelizumab</td>
<td>Solid tumors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BGB-A425</td>
<td>(TIM-3) Mono, + tislelizumab</td>
<td>Solid tumors</td>
<td></td>
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<tr>
<td>BGB-A333</td>
<td>(PD-L1) Mono, + tislelizumab</td>
<td>Solid tumors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BGB-11417</td>
<td>(Bcl-2) Mono, + zanubrutinib</td>
<td>Cell malignancies</td>
<td>Phase 1 study startup ongoing</td>
<td></td>
<td></td>
<td>Global</td>
</tr>
<tr>
<td>BGB-15025</td>
<td>(HPK1) Mono, + tislelizumab</td>
<td>IND Enabling studies ongoing</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>BGB-10188</td>
<td>(P3CD3) Mono, + tislelizumab, + zanubrutinib</td>
<td>Cell + solid malignancies</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>lifirafenib</td>
<td>(RAF dimer)</td>
<td>B-Raf/K-Ras/N-Ras mut. Solid tumors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BA3017</td>
<td>(CTLA4) Mono, + tislelizumab</td>
<td>Phase 1 study startup ongoing</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**AMG 510** (KRAS G12C) | Solid Tumors, NSCLC, CRC |       |       |       |       |       |         |
| **AMG 701** | (BCMA) | MM |       |       |       |       |         |
| **AMG 176** | (Mcl-1, SM (i.v.)) | Hematologic malignancies |       |       |       |       |         |
| **AMG 397** | (Mcl-1, SM (oral)) | Hematologic malignancies |       |       |       |       |         |
| **AMG 330** | (CD33) | Myeloid malignancies |       |       |       |       |         |
| **AMG 673** | (CD33) | AML |       |       |       |       |         |
| **AMG 427** | (FLT3) | AML |       |       |       |       |         |
| **AMG 562** | (CD19) | NHL |       |       |       |       |         |
| **AMG 596** | (EGFRvIII) | Glioblastoma |       |       |       |       |         |
| **AMG 757** | (DLL3) | SCLC |       |       |       |       |         |
| **AMG 160** | (PSMA) | Prostate cancer |       |       |       |       |         |
| **AMG 506** | (FAP x 4-1BB, DARPin®) | Solid Tumors |       |       |       |       |         |
| **AMG 199** | (MUC17) | GC/GEJC |       |       |       |       |         |

**Sitratavinib** (multi-kinase inhibitor) + tislelizumab Mono, + tislelizumab | NSCLC, RCC, OC, MEL | Asia ex-Japan, AU, NZ | Mirati |

**Zanidatamab** (HER2, bispecific antibody) | Breast cancer, GEA | Asia ex-Japan, AU, NZ | Zymeworks |

**ZW49** (HER2, bispecific ADC) | Planned (in Ph1 ex-China by Zymeworks) | Asia ex-Japan, AU, NZ | Zymeworks |

**BGB-3245** (B-RAF) | Solid tumors | Asia ex-Japan | SpringWorks |

**SEA-CD70** (anti-CD70) | Planned (starting Ph.1 ex-Asia by Seattle Genetics) | Asia ex-Japan, AU, NZ | Seattle Genetics |

**DKN-01** (DKK1) + tislelizumab + cheemo | Trials in GC/GEJC planned | Asia ex-Japan, AU, NZ | Leap Therapeutics |

†Addition compounds from Amgen collaboration not yet disclosed

• **Sotorasib* (AMG 510)**
• Sitravatinib
• Zanidatamab (ZW25) (HER-2 Bispecific Antibody)
Executive Summary – Sotorasib* (AMG 510) Program

Small molecule covalent KRAS$^{G12C}$ inhibitor

- Historically difficult drug target; Amgen’s key discovery was a surface groove on KRAS$^{G12C}$ exploited to optimize potency and advance into clinic
- Estimated incidence of KRAS$^{G12C}$ lung cancer in Chinese patients roughly equivalent to U.S.
- Encouraging clinical activity in Phase 1; Phase 2 fully enrolled in both NSCLC and CRC

BeiGene entered collaboration with Amgen in October 2019 and is responsible for China clinical development of sotorasib (both China-specific development and China operations within Amgen global trials)

Clinical Program Status

- China participation in clinical trials expected to start 4Q 2020
- Global, potentially registrational Phase 2 trial in KRAS$^{G12C}$ NSCLC (CodeBreaK 100) is ongoing$^1$
- Global Phase 3 trial in KRAS$^{G12C}$ NSCLC (sotorasib vs docetaxel) initiated in June 2020$^2$

* AMG 510 (proposed INN Sotorasib) Source: 1. NCT03600883 2. NCT04303780
Sotorasib (AMG 510) in NSCLC
Phase 1 data from ESMO 2019

At 960mg RP2D N=13, PR 7 (54%), SD 6 (46), ORR 54%; Data cutoff: July 17, 2019
Sotorasib (AMG 510) in CRC
Data from ASCO 2020

At 960mg RP2D N=12, PR 1 (8%), SD 10 (83), ORR 8%; Data cutoff: July 17, 2019
In-Licensed Programs

- Sotorasib* (AMG 510)
- **Sitravatinib**
- Zanidatamab (ZW25) (HER-2 Bispecific Antibody)
Executive Summary - Sitravatinib Program*

Sitravatinib: small molecule multi-kinase inhibitor

- In addition to being a potent inhibitor of VEGFR, sitravatinib is also a potent inhibitor of: Axl, Tyro3, and MerTK
- These kinases are involved in tumor-associated macrophage activities (polarization and efferocytosis) that appear to be critical in establishment of an immuno-tolerant state
- Proof-of-concept when combined with PD-1 in PD-1 R/R NSCLC\(^1\) and UC\(^2\)

BeiGene is responsible for China development activities, including China-specific trials and China participation in global trials

Clinical Program Status

- BeiGene initiated multi-indication Phase 1b sitravatinib + tislelizumab studies in Nov 2018 in both PD-1 sensitive and insensitive tumor types (e.g. platinum resistant ovarian cancer)\(^3\)
- Phase 3 registration trial in NSCLC patients with sitravatinib in combination with a PD-1 on-going

Sitravatinib/Nivolumab Combination Has Significant Clinical Activity in Patients with PD(L)-1 Refractory Tumors

Non-squamous NSCLC

**Confirmed ORR**
- **16%**

**PCB - Prior Clinical benefit (i.e., RECIST defined partial, complete response or stable disease for at least 12 weeks [-2-week window permitted for radiograph scheduling]) followed by radiographic progression of disease.**

**Longest Duration of Treatment Exceed 52 weeks**

**MRTX-500: Overall Survival, PCB Cohort: 1 or 2 Prior Lines of Treatment, N=73, Data as of January 30, 2020**

**OS Median (95% CI): 18.1 months (9.0, NE)**
- Events/Censored: 29/44
- Median Follow-up: 11.4 months

**Sitravatinib + Nivolumab**

- *censored

Source: 1 ESMO 2018; 2. ASCO 2020
Sitravatinib/Tislelizumab Combination Preliminary Antitumor Activity in Platinum Resistant Ovarian Cancer

<table>
<thead>
<tr>
<th>Best Response</th>
<th>Total (N=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confirmed PR, n</td>
<td>4</td>
</tr>
<tr>
<td>Unconfirmed PR, n</td>
<td>3</td>
</tr>
<tr>
<td>SD, n</td>
<td>8</td>
</tr>
<tr>
<td>PD, n</td>
<td>2</td>
</tr>
<tr>
<td>Confirmed ORR, % (95% CI)</td>
<td>23.5 (6.8–49.9)</td>
</tr>
<tr>
<td>Median DOR, weeks (95% CI)</td>
<td>NR (12.29, NR)</td>
</tr>
<tr>
<td>DCR, % (95% CI)</td>
<td>88.2 (63.6–98.5)</td>
</tr>
<tr>
<td>Median PFS, weeks (95% CI)</td>
<td>18 (12.29, NR)</td>
</tr>
<tr>
<td>3-month PFS rate, % (95% CI)</td>
<td>88.2 (60.6–96.9)</td>
</tr>
<tr>
<td>6-month PFS rate, % (95% CI)</td>
<td>35.3 (9.0–63.8)</td>
</tr>
</tbody>
</table>

- Of 17 efficacy-evaluable patients, 7 had PR (4 confirmed PR), 8 had SD, and 2 had PD

Number of patients with baseline and post baseline target lesion SPD and BOR: 17 (100.0%)
Median = -20.6% (range: -81.3%, 32.6%)

Source: ESMO IO 2019
• Sotorasib* (AMG 510)
• Sitravatinib
• Zanidatamab (ZW25) (HER-2 Bispecific Antibody)
Executive Summary – Zanidatamab (ZW25) Program

Zanidatamab: bispecific antibody targeting two distinct HER2 epitopes¹

- Zanidatamab biparatopic – targets extracellular domain 2: ECD2 (trastuzumab binding domain) and ECD4 (pertuzumab binding domain)
- Unique binding geometries of zanidatamab promoted increased tumor cell binding and enhanced HER2 internalization compared with trastuzumab
- Stronger anti-tumor activity compared to trastuzumab is observed in preclinical studies
- Zanidatamab, as single agent, has shown encouraging anti-tumor activity across multiple HER2-expressing tumor types (Part 1 and 2, ZWI-ZW25-101 Ph1 Study)²

BeiGene is responsible for China development activities, including China-specific trials and China participation in global trials

Clinical Program Status

- Registration-enabling trial initiated in 2L HER2+ biliary tract cancer
- Ph2 combination studies are ongoing to support pivotal trials
- One registration-enabling studies are planned:
  - 1st line HER2+ gastroesophageal cancer

Source: 1. Zymeworks March 2020 corporate presentation; 2. Internal Data
Collaborations Are a Central Part of Our Comprehensive Pipeline

Broad set of ongoing clinical collaborations that significantly augment our internal discovery, ongoing clinical development programs, and commercial products (tislelizumab and zanubrutinib)

Many programs planned to go to pivotal/late-stage in next 6-18 months
Concluding Remarks

John V. Oyler
Chairman, Co-Founder & CEO
Summary: Research and Early Development

Internal R&D platform generating robust, sustainable pipeline

- **Proven**: Two approved medicines (and another at the filing stage) with excellent clinical profiles
- **Cutting Edge**: Utilizing cutting edge technologies to address a wide range of biological problems
- **Impactful**: All programs potentially best-in-class or first-in-class assets such as HPK1
- **Broad and Growing**: 350+ team growing to 650+

Built unique, sustainable competitive advantages

- **Clinical acceleration & lower cost**: 1,350+ team for China-inclusive global trials
- **Combinability with internal platform assets**: PD-1, BTK, PARP and growing
- **Science & medicine-based commercial team**: ~1,300; 6 commercial products; excellent pipeline
- **Internal manufacturing & preclinical capabilities**: Includes biologics, formulation, preclinical

Past collaborations successful, leverage our competitive advantages, & expand our portfolio … and capacity exists for this to continue to be a major source of growth

- Deep, promising collaboration pipeline
- Each competitive advantage has been demonstrated in collaborations
- Amgen collaboration provides further validation

Well positioned to act quickly & capture future internal & external breakthroughs
Recent Accomplishments and Upcoming Milestones

Past 10 Months (From 4Q19 – YTD)

Recent Accomplishments and Upcoming Milestones

Disclosed Milestones Over Next 18 Months

Preclinical Assets Advanced into Clinic

Trials Enrolled

Phase 3 Data Readouts

NDA Filings

Approvals or Launches

Assets Added Through Collaborations

Organizational Progress

Biologics manufacturing in process validation & expanded

Amgen transitional activities progressing

Angus Grant as Chief Business Executive

Early Data Readouts

Potential Phase 3* Readouts and Potential Filings

Potential NDA Filings or Regulatory Discussion

Commercial Portfolio

* Phase 3 or registrational enabling trials
Future Vision

Once in a lifetime period of transformation in our industry, which creates opportunities for smaller players to become leaders

BeiGene is one of the best positioned companies for this opportunity

On this journey, BeiGene is striving to

- Become an oncology and scientific leader
- Expand beyond oncology into other areas of need
- Continue to build sustainable competitive advantages
- Become the best global clinical organization – addressing the biggest issue of the industry
- Transform the industry to bring better medicine to more patients more affordably

Thank You!
Q&A

Participants:

- Xiaodong Wang, Ph.D.
- Eric Hedrick, M.D.
- John V. Oyler
- Yong (Ben) Ben, M.D.
- Howard Liang, Ph.D.
- Jane Huang, M.D.
- Lai Wang, Ph.D.
Thank You