BeiGene

R&D Investor Day

July 18, 2023

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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	***Julia Wang**, CFO*
Path to Global Oncology Leadership	***John V. Oyler**, Co-Founder, Chairman and CEO*
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

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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

RESEARCH

~1,100
world-class scientists

Broad preclinical programs, ~50% with first-in-class potential

$1.4B collaboration fees

CLINICAL DEVELOPMENT

3,000+
clinical development colleagues* in 48 regions

Successful track record of developing differentiated molecules

COMMERCIAL

3,500+
competitive commercial team with **500+** in NA/EU

CORNERSTONE MEDICINES

BRUKINSA and tislelizumab

Cornerstone commercial medicines with huge **global potential**

MANUFACTURING

~750
In-house people and capabilities with **cost advantage** and **agility**
Small molecules and biologics **(55,000L expanding to up to 200,000L)**

*Includes full-time service professionals

Leading Global Oncology Powerhouse

Largest dedicated oncology R&D team

Broadest 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

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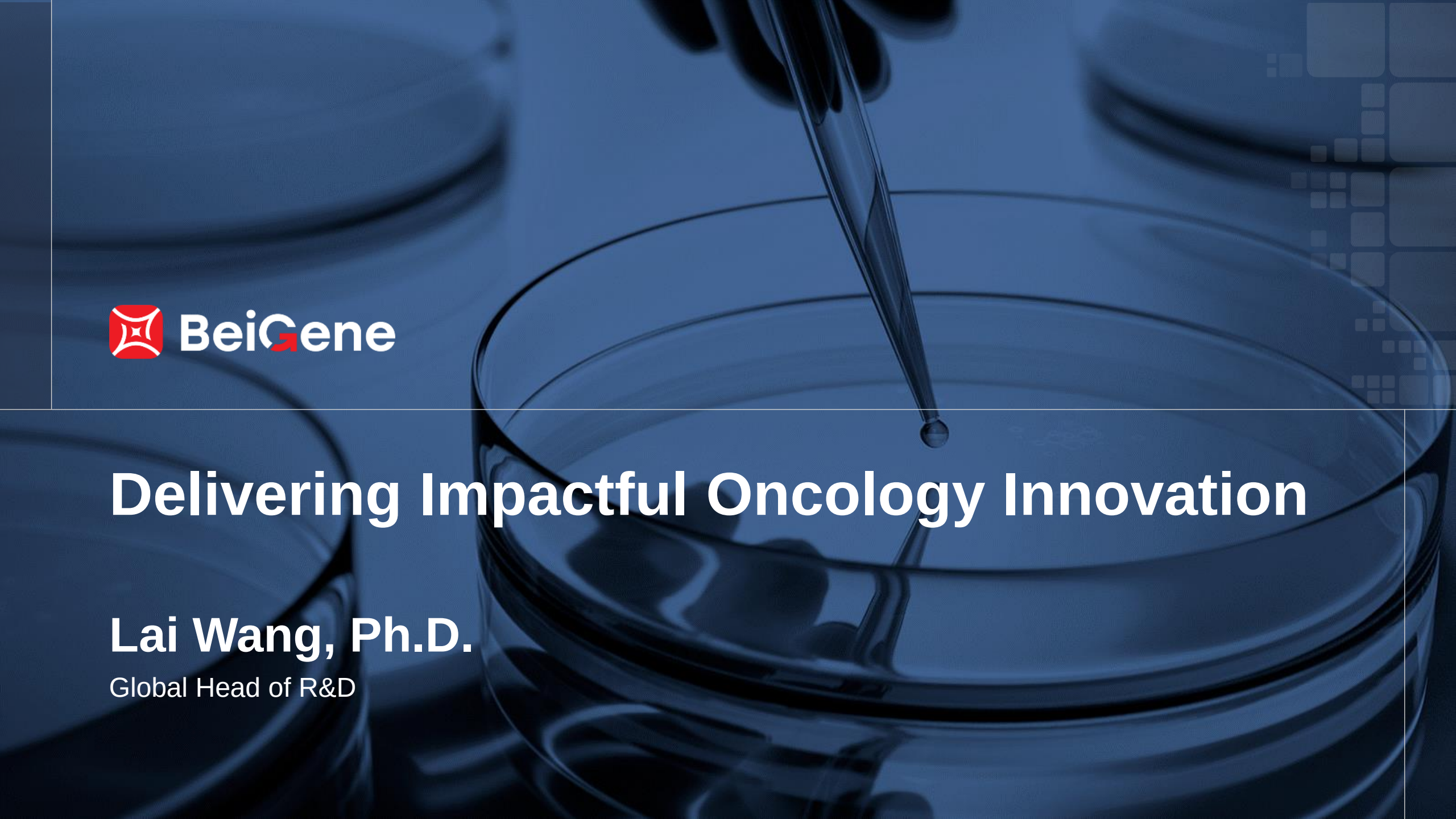


Delivering Impactful Oncology Innovation

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

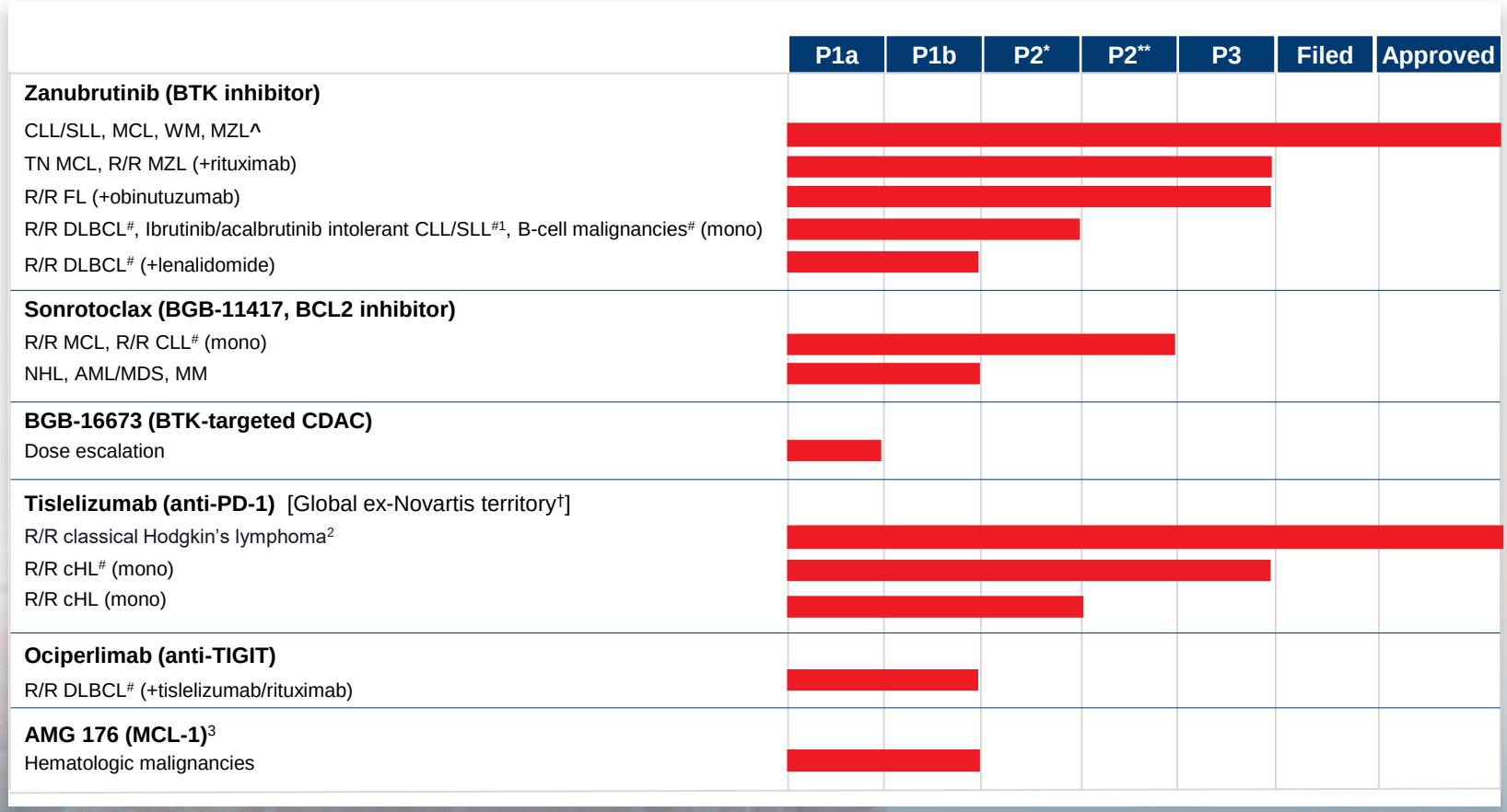
Emerging as **heme leader** with potential best-in-class/first-in-class assets addressing broad range of malignancies, including **BTKi, BCL2i, BTK degrader**

Going beyond **immuno-oncology** in solid tumor portfolio with **oncogenic signaling targeted therapies** and **TAA-driven therapies**

NME – New Molecular Entity PCC – Preclinical Candidate PoC – Proof of Concept TAA – Tumor Associated Antigen

Hematology Pipeline with 3 Important Programs

BRUKINSA, sonrotoclax (BCL2i) and BGB-11673 (BTK degrader) all with compelling data



*^ U.S.: CLL,R/R MCL[1], WM & R/R MZL[1]; China: R/R MCL[2], R/R CLL/SLL[2] & R/R WM[2]; EU[3]: 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, Lichtenstein 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.*

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Solid Tumor Pipeline is Growing

Expanding beyond I-O with targeted therapies

Late Stage

	P1	P2*	P2**	P3	Filed	Approved
Tislelizumab (anti-PD-1) [Global ex-Novartis territory†]						
China: 1L squamous and non-squamous NSCLC, 2/3 L NSCLC, 2/3 L HCC[1], R/R PD-L1+ UC[1], 2L ESCC, MSI-H or dMMR solid tumors[1], 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 ESCCandGC/GEJ, neo ESCC# (+chemo)						
1L HCC # (+lenvatinib), solid tumors# (+fruquintinib, +lenvatinib)						
Pamiparib (PARP 1/2 inhibitor)						
3L BRCA-mutated ovarian cancer						
2L PSOC maintenance (mono)#						
1L GC maintenance (mono)						
Solid tumors (+TMZ (chemo))						
Ociperlimab (anti-TIGIT)						
1L PD-L1+ stage IV NSCLC (+tislelizumab)						
2L PD-L1+ ESCC, 2L+CC (+tislelizumab), 1L HCC (+tislelizumab+BAT1706)						
1L LS-SCLC(+tislelizumab+cCRT), 1L NSCLC (+tislelizumab+chemo)						
Solid tumors (+tislelizumab)						
Sitravatinib[2] (multi-kinase inhibitor)						
2/3L NSCLC (+ tislelizumab)						
GC/GEJC#, 2/3L ESCC # (Mono, + tislelizumab)						
Solid tumors (Mono, + tislelizumab)						
Zanidatamab[3] (ZW25, HER2, bispecific antibody)						
1L HER2+ GEA (+ chemo ± tislelizumab)						
Biliary tract cancers (Mono)						
Tarlatamab[4,5] (AMG 757, DLL3 x CD3)						
SCLC, neuroendocrine prostate cancer[6]						

Early Stage

	P1a	P1b	P2*
LBL-007[5] (anti-LAG-3) Solid tumors (+ tislelizumab)			
BGB-A445 (anti-OX40) Solid tumors (+tislelizumab)			
Surzebiclimab (BGB-A425, anti-TIM-3) Solid tumors (+/- tislelizumab)			
Lifirafenib[6] (RAF Dimer) B-Raf- or K-RAS/N-RAS-mutated solid tumors (+mirdametinib)			
Brimarafenib[7] (BGB-3245, B-Raf inhibitor) Solid tumors			
BGB-10188 (PI3-Kδ 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			
Acapatamab[8] AMG160, PSMA x CD3) Prostate cancer, NSCLC			
AMG 199[3,8] (MUC-17 x CD3) Solid tumors			
Latikafusp[8] (AMG 256, IL-21m/PD-1) Solid tumors			
Xaluritamig[8] (AMG509, STEAP1 X CD3) Prostate cancer			

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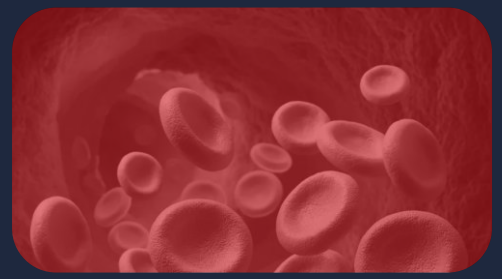
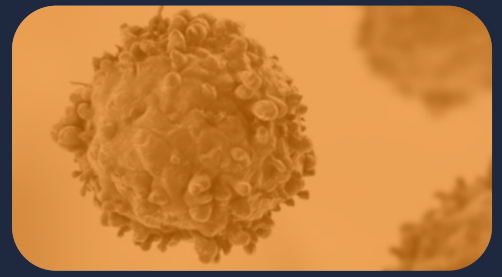
Deep Dive on R&D

Oncology leadership driven by an extensive portfolio and exceptional science



Leading in Hematology

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



Advancing Broad Solid Tumor Portfolio

Moving beyond IO with focus on lung and other key tumors with several potential blockbuster programs

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

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

Hematology

Solid Tumors

Research Innovation

BeiGene

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

BTK inhibitor

Brukinsa® zanubrutinib 80mg capsules

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

BCL2 inhibitor

Sonrotoclax

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

BTK CDAC

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**

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

Hypothesis: sustained inhibition

- Engineered to exhibit **high potency, bioavailability,** and **kinase selectivity** with aim of **reducing off-target toxicities** while maintaining continuous **high BTK inhibition**

4,800+ patients enrolled globally

- Safety and efficacy of **BRUKINSA** assessed in numerous indications **across the globe, in 35+ trials**

Two major Phase 3 head-to-head trials against ibrutinib

- **ORR** and **PFS** in **R/R CLL/SLL** patients shown to be superior to **ibrutinib**
- **WM** patients showed a consistent trend of **deeper** and **more durable** responses than ibrutinib

BTKi with approvals in most diseases

- Broadest label: **CLL/SLL, WM, MCL, MZL**
- sNDA in US and EU in **FL**

Expanding development program

- **Evaluating novel combinations:** with both external and internal programs **across a spectrum of hematological malignancies**

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.

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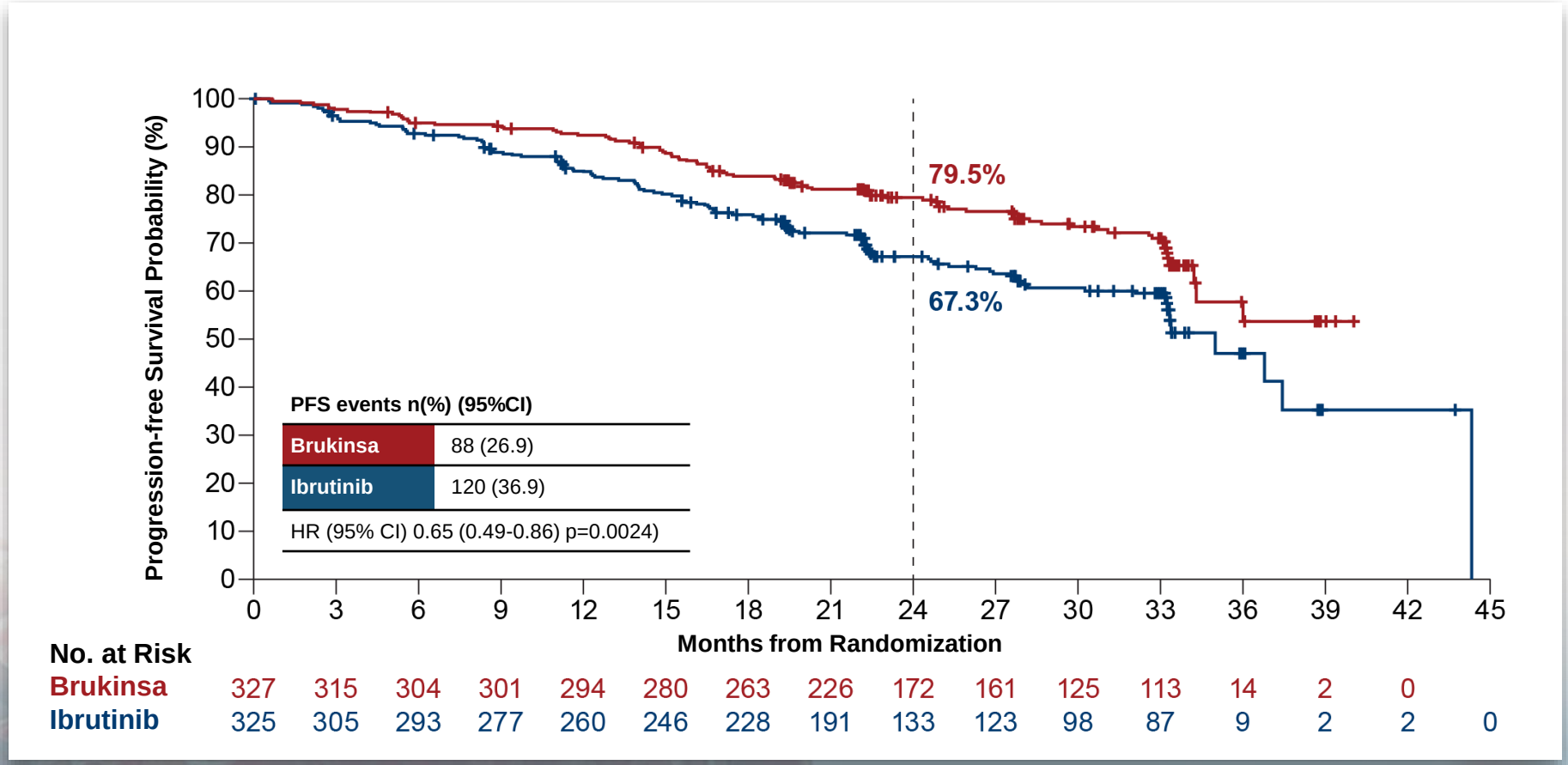
PFS significantly superior to ibrutinib in ALPINE – median follow-up of 29.6 months



Data cutoff: 8 Aug 2022

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

Brown J et al. NEJM 2022. DOI: 10.1056/NEJMoa2211582

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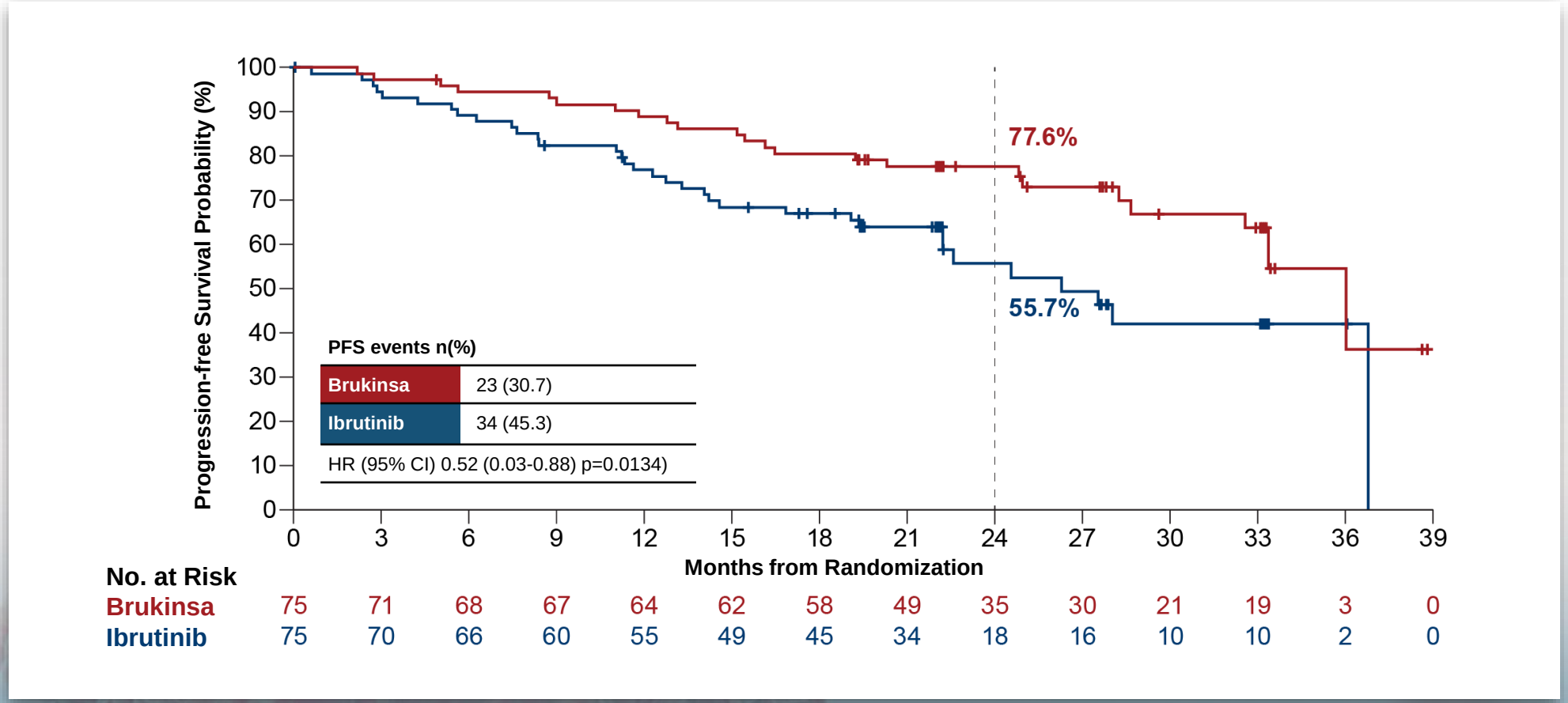
BRUKINSA

Improved PFS in pre-defined subset of patients with del(17p)/*TP53*^{mut}



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

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BRUKINSA

Consistent PFS superiority in all ALPINE sensitivity analyses

Analysis Type	HR (95% CI)
Accounting for treatment discontinuation without PD	**0.56** (0.38, 0.84)
Accounting for new therapies without PD	**0.63** (0.48-0.84)
Accounting for death due to COVID-19	**0.62** (0.45-0.84)
Accounting for drug interruption	**0.71** (0.53-0.95)

Data cutoff: 8 Aug 2022

Presented at the 34th French Society of Hematology Congress, March 30, 2023

BRUKINSA
Lower rate of cardiac events, treatment discontinuation and deaths

- **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*

Cardiac deaths. One death not listed due to myocardial infarction with ibrutinib discontinuation due to diarrhea 14 days prior to the fatal event.

	BRUKINSA (n=324)	ibrutinib (n=324)
Serious cardiac adverse events	**6 (1.9%)**	**25 (7.7%)**
Cardiac adverse events leading to treatment discontinuation	**1 (0.3)**	**14 (4.3)**
Atrial fibrillation	0	5 (1.5)
Cardiac arrest	0	2 (0.6)
Cardiac failure	0	2 (0.6)
Cardiac failure acute	0	1 (0.3)
Congestive cardiomyopathy	0	1 (0.3)
Myocardial infarction	0	1 (0.3)
Palpitations	0	1 (0.3)
Ventricular extrasystoles	1 (0.3)	0
Ventricular fibrillation	0	1 (0.3)

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

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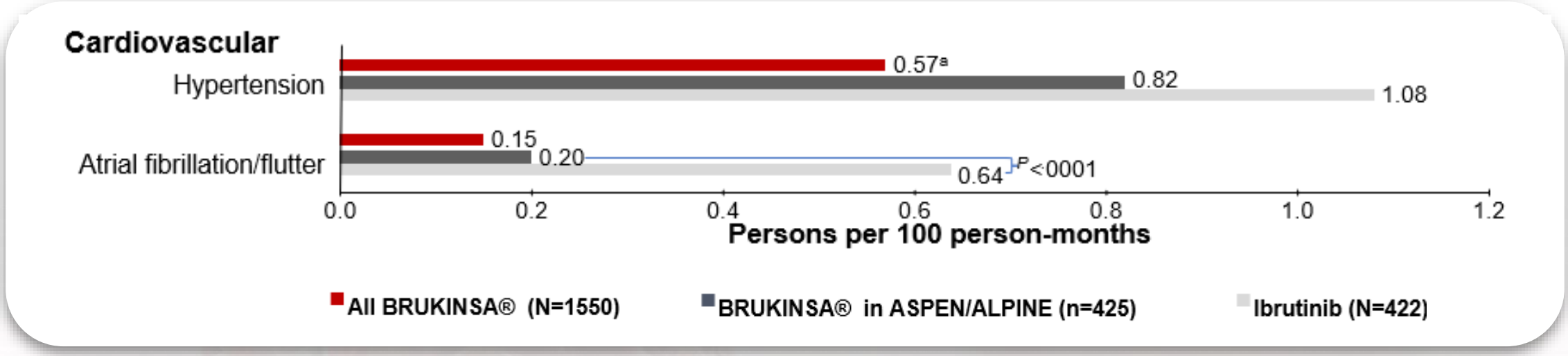
BRUKINSA

Lower incidence of cardiovascular events vs. ibrutinib reinforces better safety profile



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

Brown, et al. Presented at EHA 2023 Hybrid Congress; June 8-15, 2023; Frankfurt, Germany. Abstract P631.

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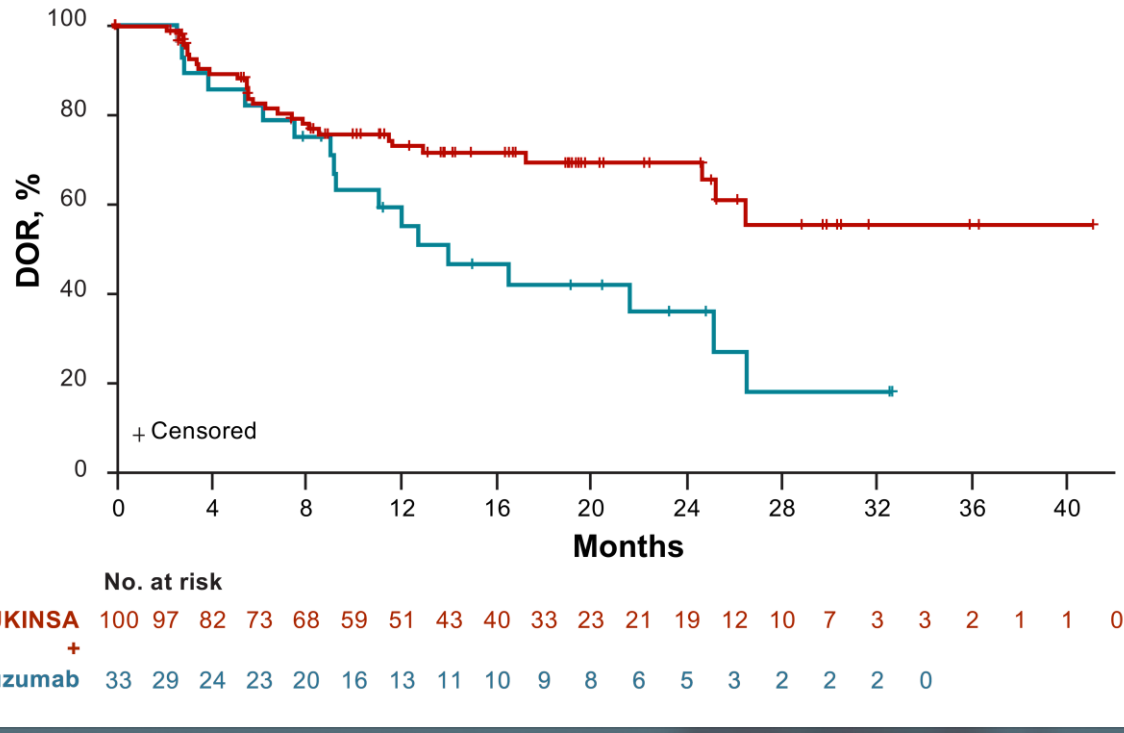
BRUKINSA

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

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

Duration of Response (DOR) by IRC

Median study follow up 20.2 months



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BRUKINSA
Superiority core to hematology

Superior to Chemo in TN CLL

- SEQUOIA: Superior PFS, favorable safety profile
- Category 1 NCCN guidelines for CLL

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

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

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

Sonrotoclax

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

Sonrotoclax
BGB-11417
(BCL2 inhibitor)

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

Studies with potential for registration are highlighted in red text

Sonrotoclax

FIH answers questions on optimizing dose and ramp-up schedule and potential coml

	Dose-Finding		Expansion
Monotherapy Sonrotoclax	**Part 1** *Independent disease-specific cohorts:* *R/R CLL/SLL, FL, DLBCL, MCL, MZL, WM*	→ RP2D (per cohort) →	**Part 2** *Independent disease-specific cohorts:* *R/R CLL/SLL, FL, DLBCL, MCL, MZL, WM*
Combination Sonrotoclax + BRUKINSA	**Part 3** *Independent disease-specific cohorts:* *R/R CLL/SLL, MCL; TN CLL/SLL*	→ RP2D (per cohort) →	**Part 4** *Independent disease-specific cohorts:* *R/R CLL/SLL, MCL; TN CLL/SLL*
Combinations Sonrotoclax + obinutuzumab Sonrotoclax + obinutuzumab + BRUKINSA	**Part 5** *TN CLL/SLL*	→ RP2D (per cohort) →	**Part 6** *TN CLL/SLL*

BGB-11417-101 Study

Sonrotoclax: BGB-11417-101

High objective and deep responses in TN and R/R CLL mono and combo

Efficacy (Overall Response Rate)

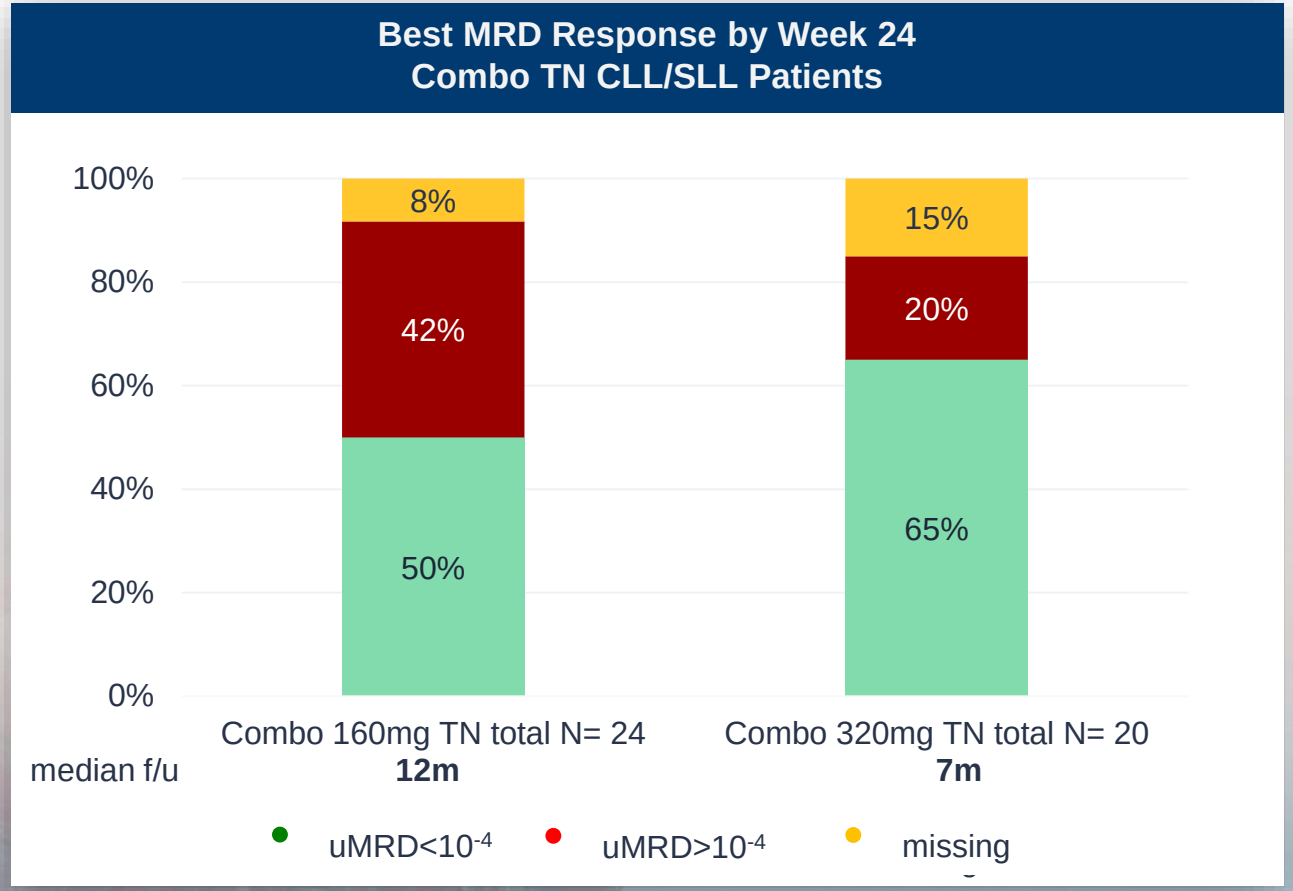
Response, n (%)	R/R sonrotoclax* (n=47)	R/R sonrotoclax + BRUKINSA (n=34)	TN sonrotoclax + BRUKINSA (n=94)
Number treated w/ sonrotoclax	**47**	**34**	**94**
Efficacy evaluable treated with sonrotoclax	**35**	**25**	**56**
ORR, n (%)	**23 (65.7)**	**24 (96)**	**56 (100)**
CR	8 (22.8)	**11 (44)**	15 (26.8)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

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



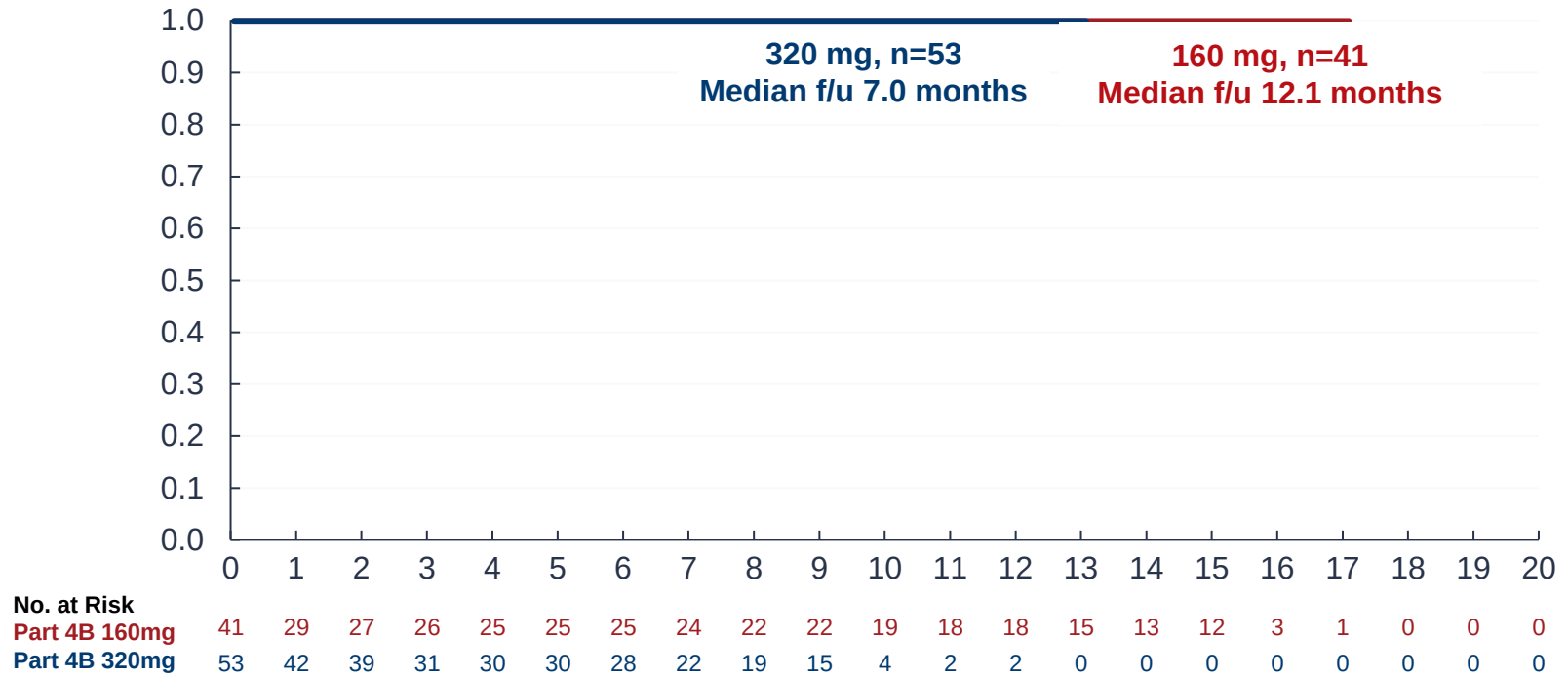
- **Higher uMRD rate, and more rapid uMRD with increasing dose**
- **Higher uMRD rate with longer treatment duration**
  - uMRD at ≥ 12m treatment: 69% (11/15) at 160mg; 1/1 at 320 mg

MRD Evaluable Set at Week 24 is defined as patients who reached Week 24 visit and had at least one post-baseline MRD sample by Week 24 on target dose, excluding those with baseline MRD levels <10^-4.

Sonrotoclax and BRUKINSA Combination

All 94 patients in TN CLL remain on study progression free

Kaplan Meier Plot of Progression Free Survival in TN CLL/SLL Patients Safety Analysis Set



Source: ADLS, ADTTE. Data cutoff: 21MAY2023. Data extraction: 23MAY2023.
/bgb_11417/bgb_11417_101/bb_20230521_tnell/dev/pgm/tlfs/f-eff-km.sas 25MAY2023 23:30 f-14-2-1-1-eff-km-pfs.rft

Data cut-off date: 21May2023 for TN CLL

Sonrotoclax

CLL monotherapy and combination demonstrates promising safety and tolerability

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

- **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**

TEAE: treatment-emergent adverse event. * Monotherapy data in R/R CLL/SLL was pooled analysis of 101 and 102

Sonrotoclax

Planned Phase 3 study in treatment naïve CLL with fixed duration treatment

Previously Untreated CLL/SLL

BRUKINSA + Sonrotoclax FD
12 cycles after 3 cycles Z mono

Venetoclax + Obinutuzumab FD

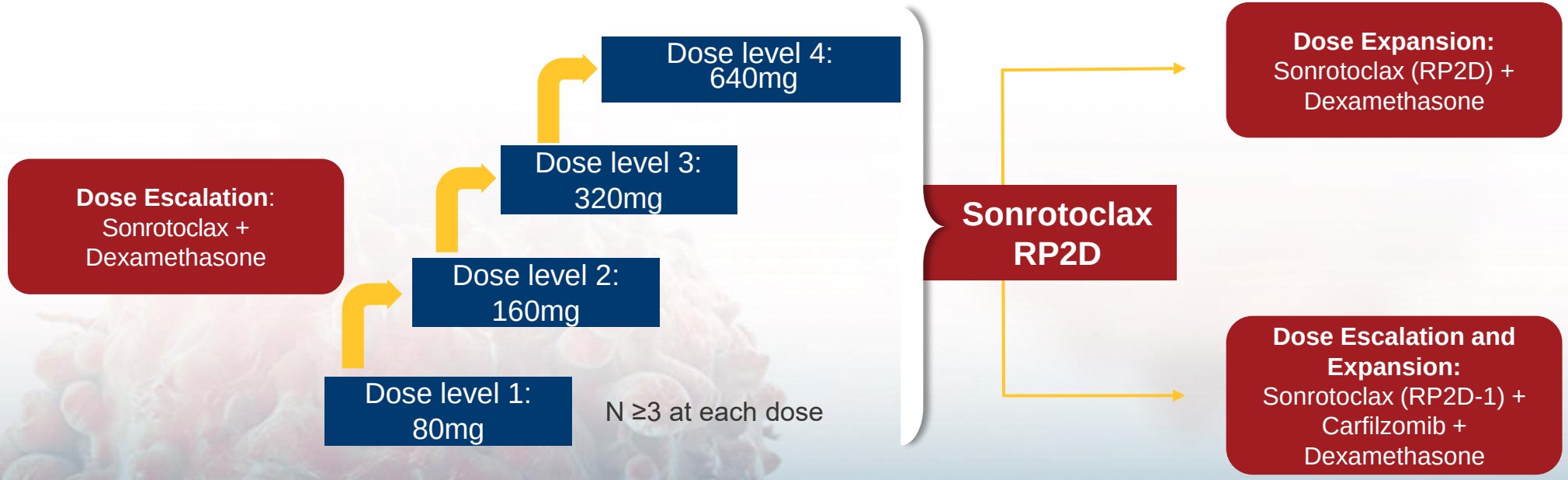
Stratified by:

- 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

Sonrotoclax

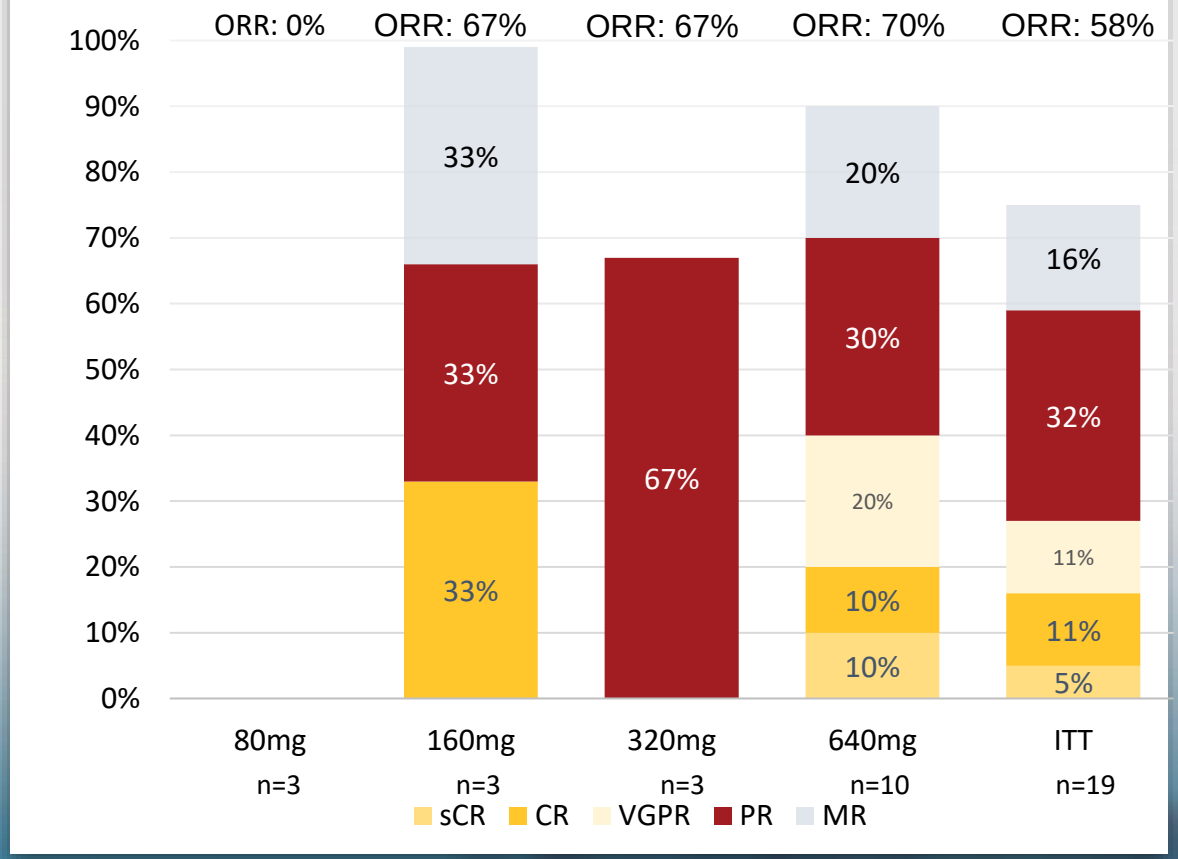
Phase 1 study R/R multiple myeloma with t(11,14) mutation



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%

ORR R/R Multiple Myeloma Harboring t(11,14)



**1 patient in the 640mg cohort is unevaluable at time of data cut-off*

Kumar Blood 2017; 130;2401. Kaufman Am J Hematol 2021; 96:418

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

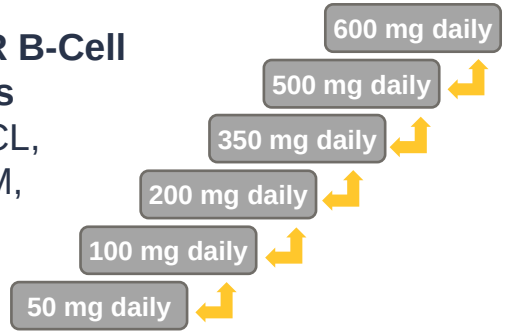
- **Become backbone** for patients progressing after BRUKINSA **as mono or combo with sonrotoclax**
- Degradation may **expand disease areas** where there is a clear rationale (e.g., LBCL)

BTK CDAC

BGB-16673-101 Phase 1a/b study in B-cell malignancies

Dose Escalation

Selected R/R B-Cell Malignancies
(MZL, FL, MCL, CLL/SLL, WM, DLBCL, RT)



Safety Expansion

Up to 20 patients enrolled at each dose level.

RP2D

Dose Expansion

Cohort 1:
Post BTKi R/R CLL/SLL

Cohort 2:
Post BTKi R/R MCL

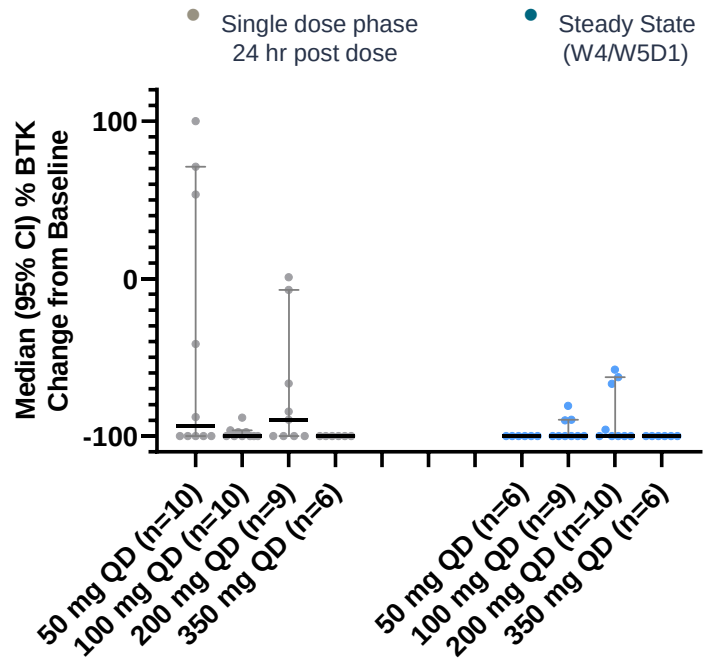
Objectives

- Characterizing safety / PK / biomarker properties, MTD, and RP2D in escalation and safety expansion
- Safety/ efficacy at the RP2D in dose expansion

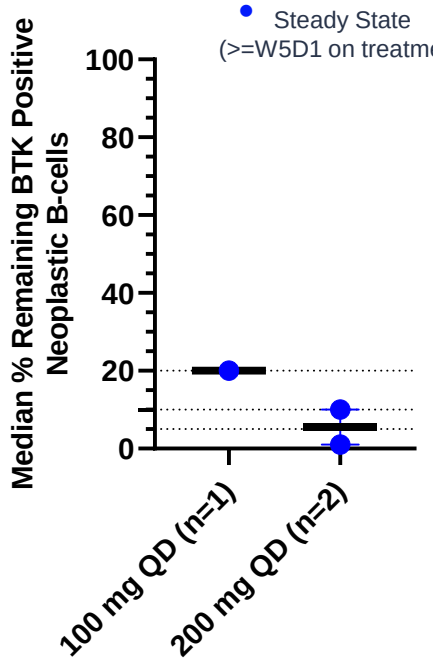
BTK CDAC

Strong BTK inhibition starting at lowest dose and dose-dependent inhibition in tissue

Percent BTK Reduction at Steady State and Single Dose Phase in Blood



Percent of Remaining BTK Positive Neoplastic B-cells in Lymph Node



- 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

BTK CDAC

BGB-16673-101 preliminary safety: no hypertension or atrial fibrillation observed to date

Safety Overview	N=27
Patients with any grade AE (%)	25 (92.6)
Grade ≥3 AEs	11 (40.7)
Serious AEs	10 (37.0)
AEs reported as DLTs *	1 (3.7)
AEs leading to death §	1 (3.7)
AEs leading to dose hold	6 (22.2)
AEs leading to dose reduction	2 (7.4)
AEs leading to discontinuation	0 (0)

mFU: 3.5mo

Adverse Events of Interest (Pooled, %)	N=27	
	Any Gr	G3+
Any Bleeding	12 (44.4)	2 (7.4)
Neutropenia	6 (22.2)	4 (14.8)
Diarrhea	6 (22.2)	0
Amylase/Lipase Increased	6 (22.2)	1 (3.7)
Any Infection	10 (37.0)	4 (14.8)
Anemia	1 (3.7)	1 (3.7)
Thrombocytopenia	1 (3.7)	1 (3.7)
Arthralgia	2 (7.4)	0
Atrial Fibrillation	0	0
Hypertension	0	0
Fatigue	2 (7.4)	0

Unofficial DCO 17 Jun 2023

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

BTK CDAC

BGB-16673-101 – good overall response rate (ORR) per dose level and histology

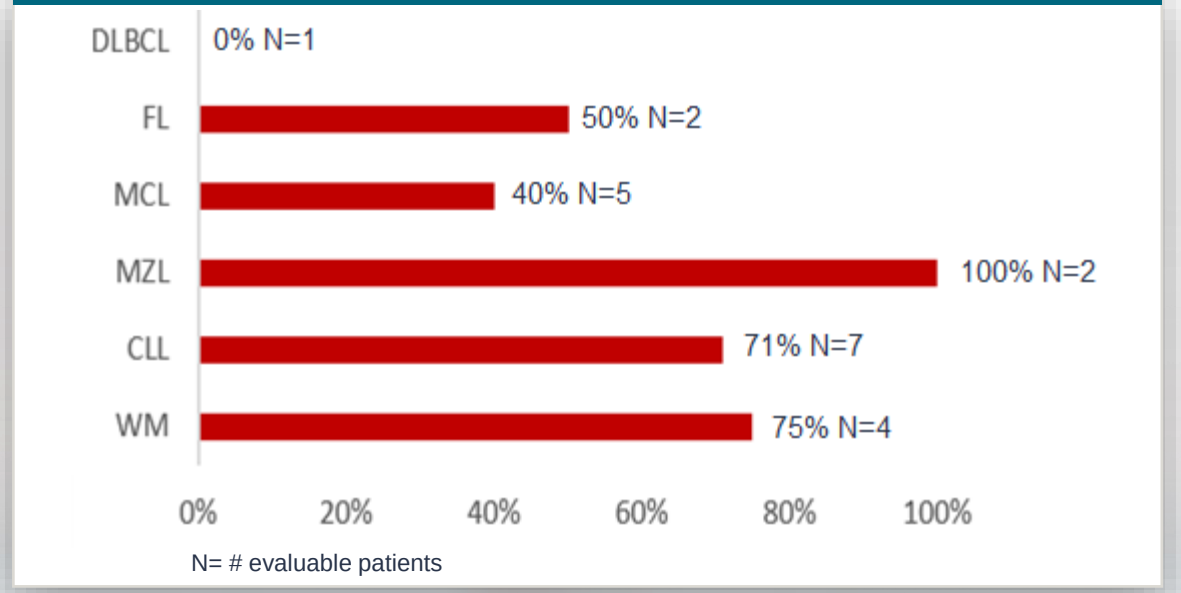
ORR by Dose Level

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

mFU: 3.5mo

Unofficial DCO 15 Jun 2023

ORR by Histology



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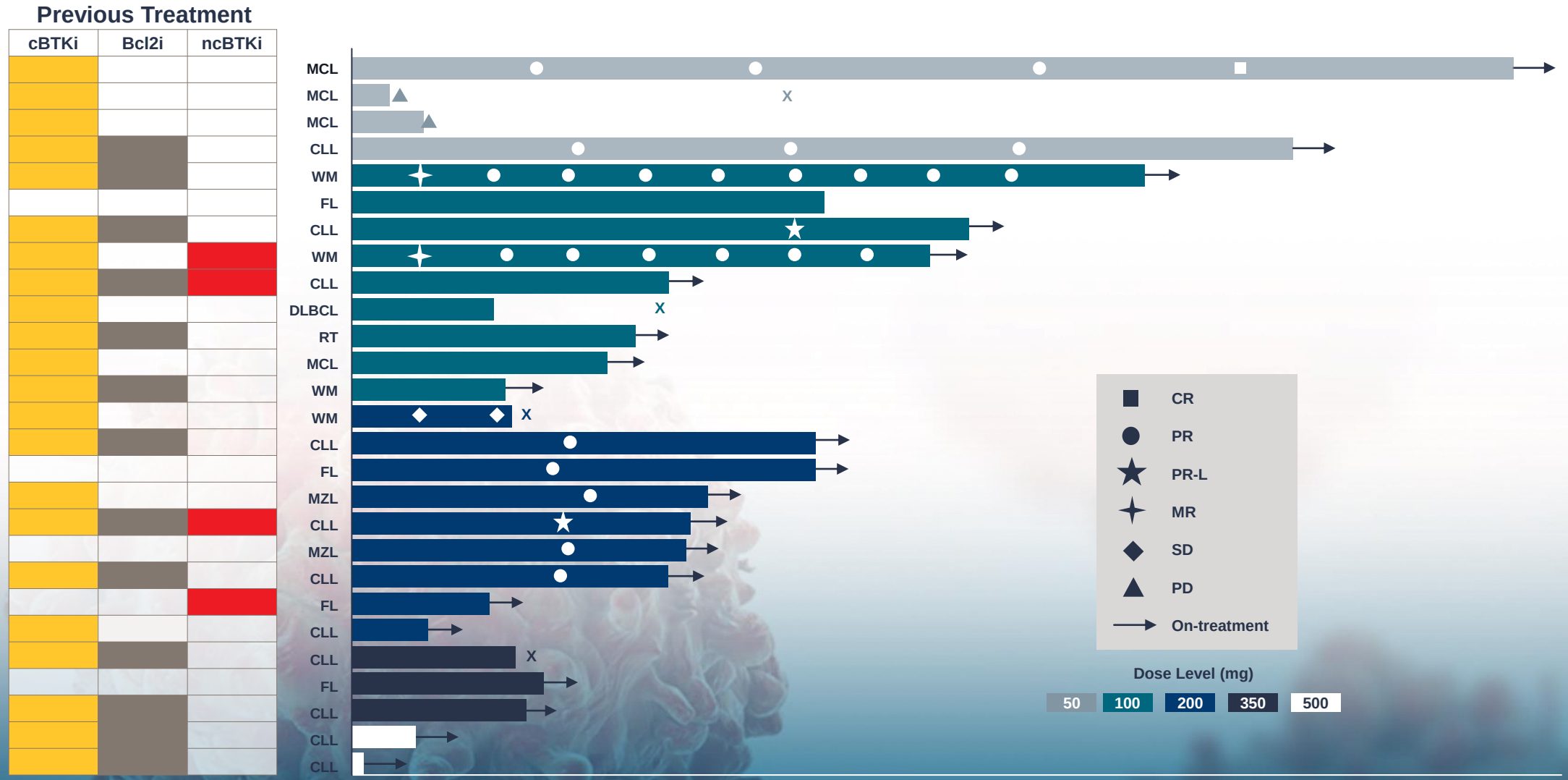
BTK CDAC

BGB-16673-101 efficacy by patient, with promising durability data



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

BeiGene

BTK CDAC Program Summary

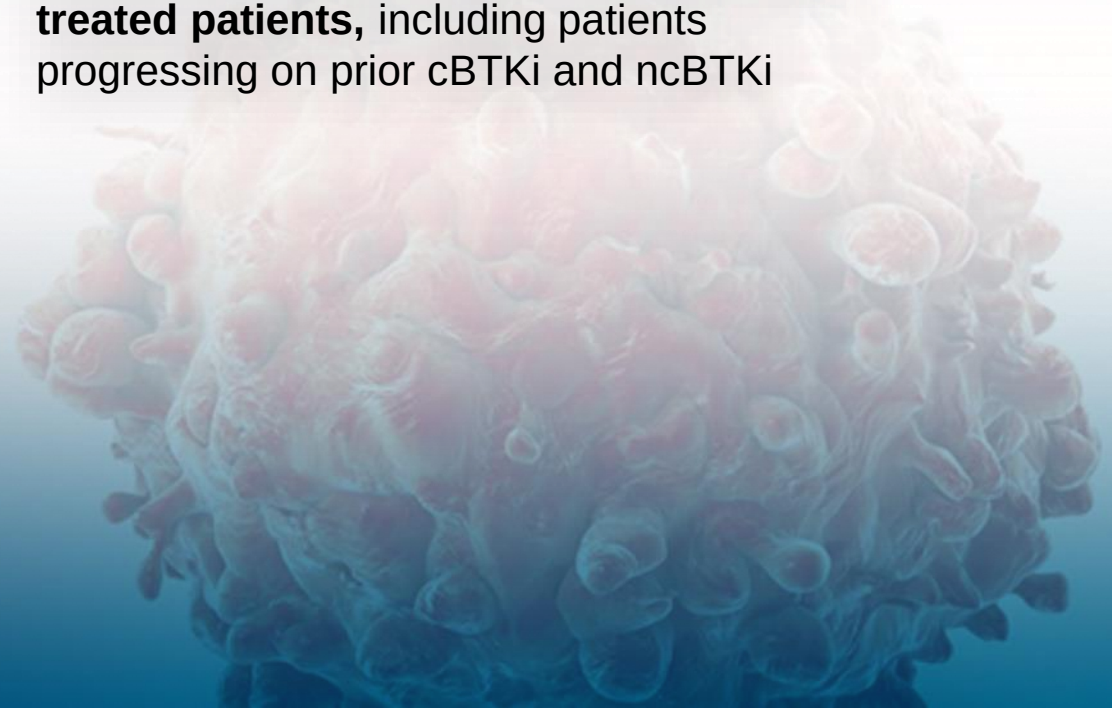
Well positioned to overcome resistance to covalent and noncovalent BTKi

Promising Early Efficacy and PD

- **Deep BTK degradation** in PD studies
- **Promising efficacy signal in heavily pre-treated patients,** including patients progressing on prior cBTKi and ncBTKi

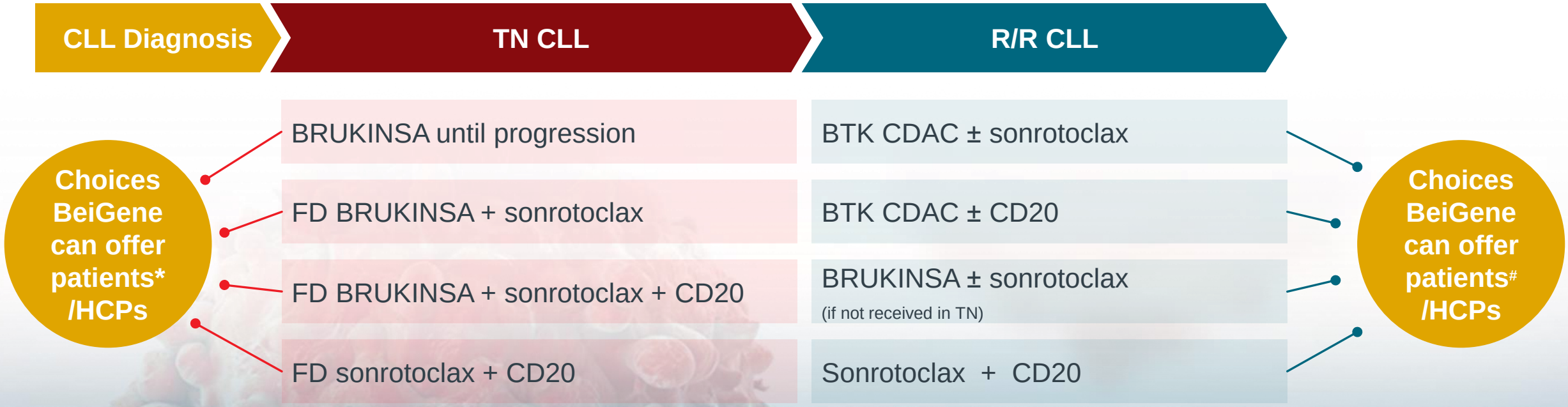
Good Safety Profile

- **Toxicity appears favorable** compared to other BTK degraders
- **No hypertension or atrial fibrillation** observed at this point



Covering the Entire CLL Patient Journey

Confirming our leadership in the treatment of this disease setting



*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

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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**

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

Hematology

Solid Tumors

Research Innovation

BeiGene

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

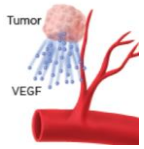
RATIONALE-302
2L ESCC: FDA on-site GMP inspection complete and BLA review progressing

Regulatory submissions underway to **expand to rest of world**

Reduced cost through optimization, internalization, and scale

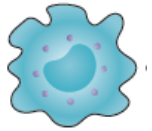
PD-1 Centered Pan Tumor Immuno-Oncology Pipeline

Extensive tumor microenvironment modulating approaches

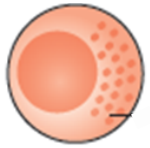


Endothelial Cell

Cytotoxic T-Cell

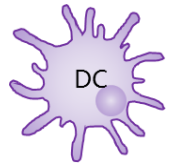


Myeloid Derived Suppressor Cell



NK Cell

Treg Cell



Dendritic Cell

PD-1 mAb Tislelizumab

Over 20 I-O and targeted molecules to pair with tislelizumab

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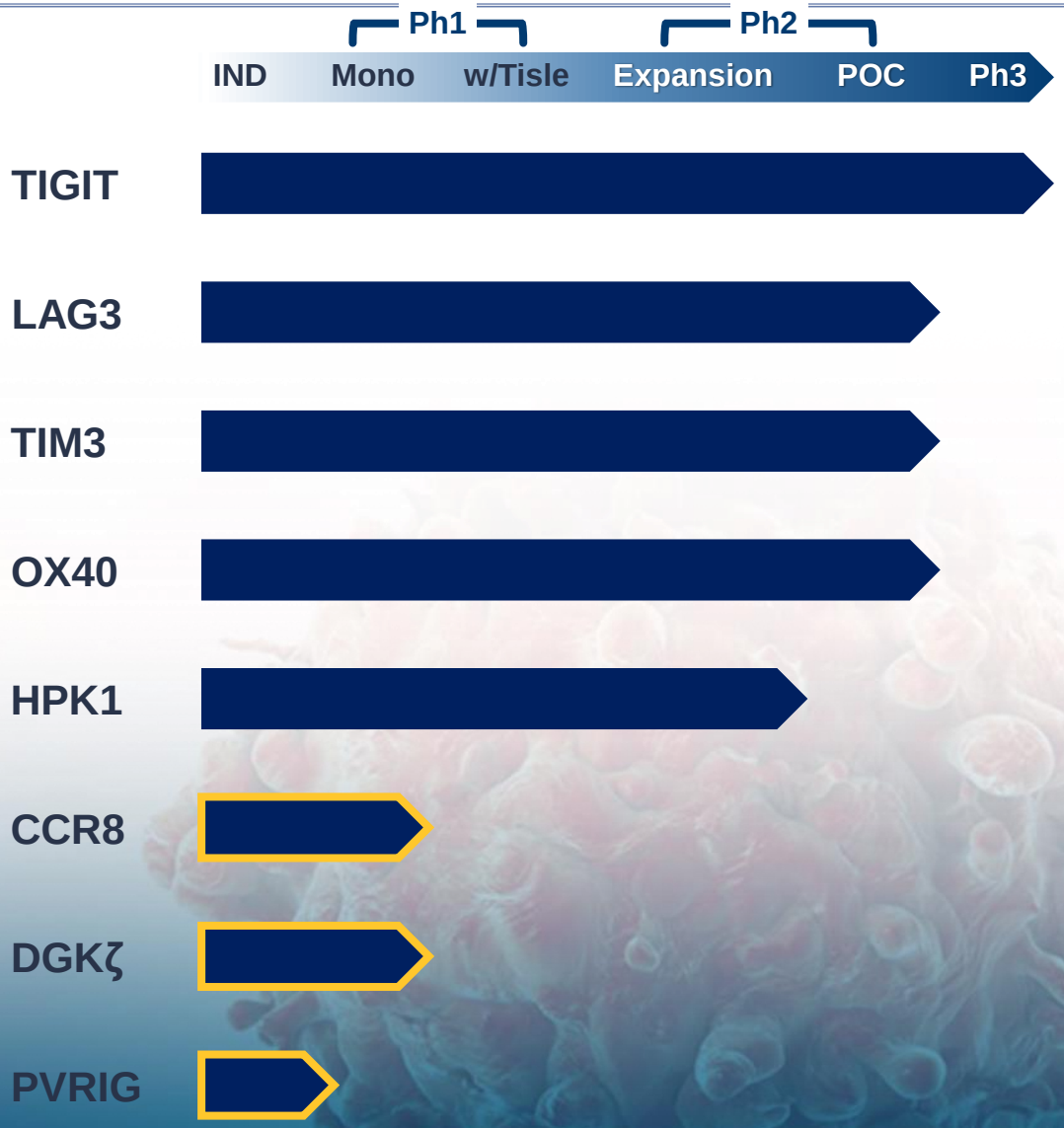
Next Wave of Immuno-Oncology Programs

Will synergize in combination with tislelizumab



Next Wave of IO Assets

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

- 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)

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

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

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

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

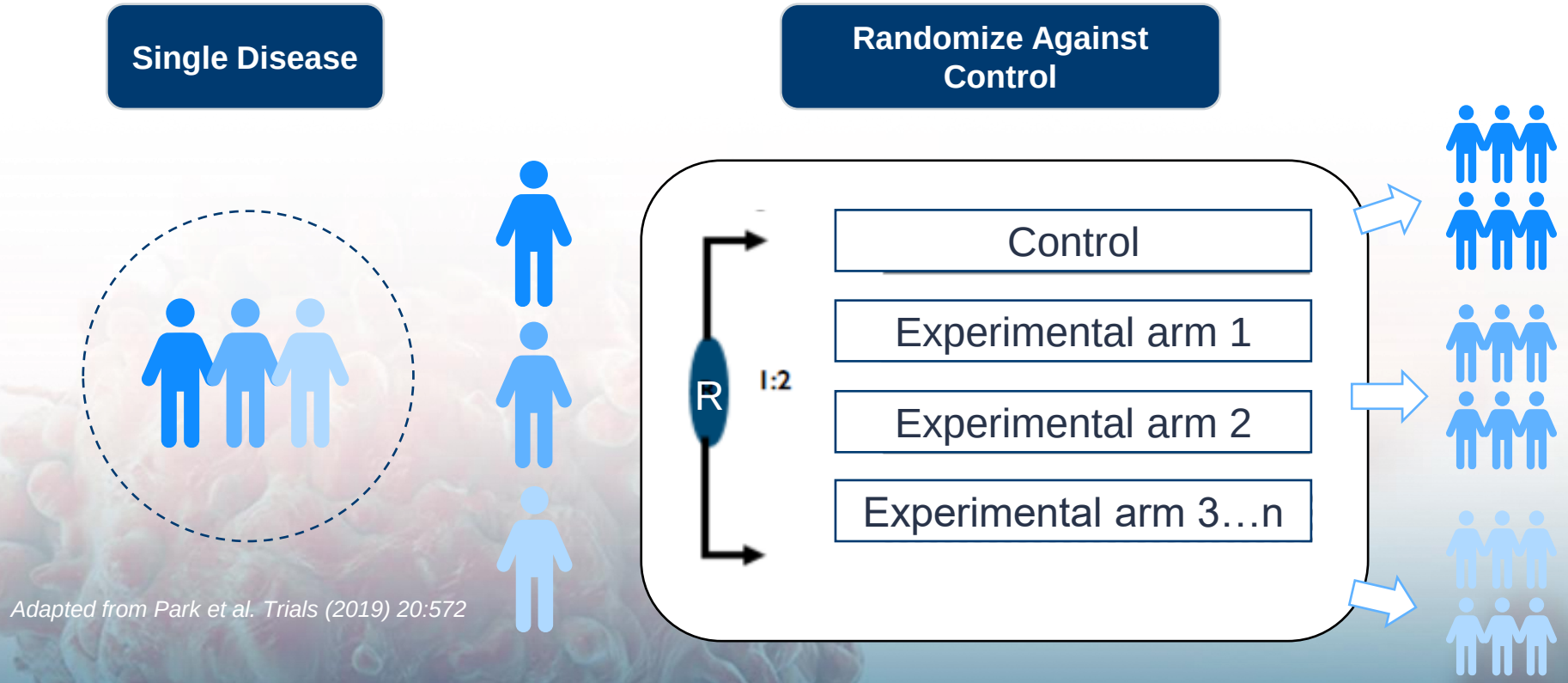
- FIC potential – activator of T and NK cells
- IND submitted with FIH in **3Q23**

- 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



Adapted from Park et al. Trials (2019) 20:572

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

Innovative Solid Tumor Portfolio

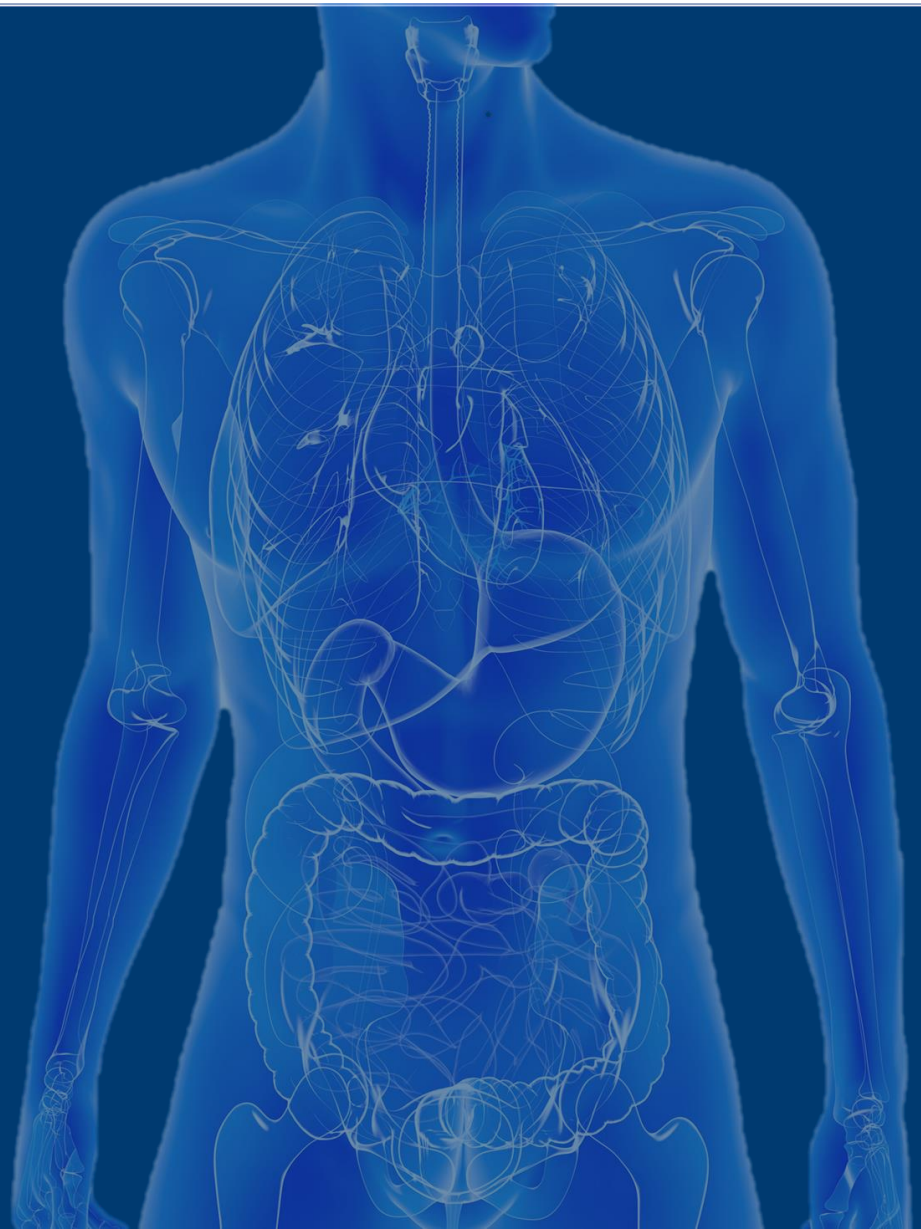
Accelerating programs in priority tumor types

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

BeiGene

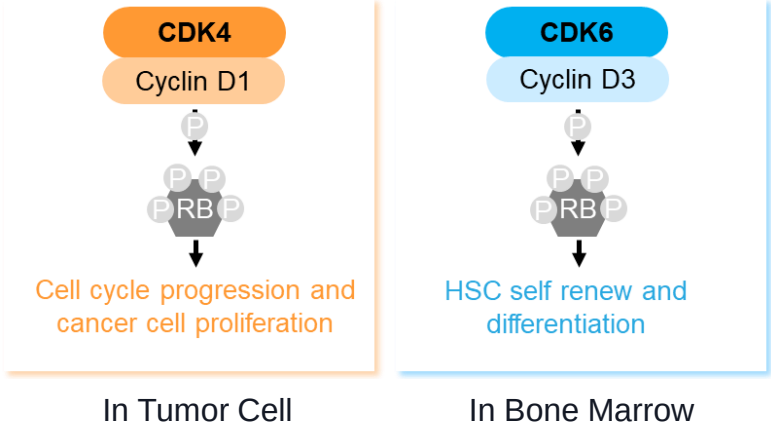
CDK4 Inhibitor

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

Drug	Palbociclib	Ribociclib	Abemaciclib
Dose Limiting Toxicities	Neutropenia	Neutropenia	Fatigue, Diarrhea
Potential Cause	CDK6 Inhibition	CDK6 Inhibition	Off-target to CDK9, GSK3β and CAMKIIs
CDK4/6 Selectivity	**1.3**	**7.7**	**16**



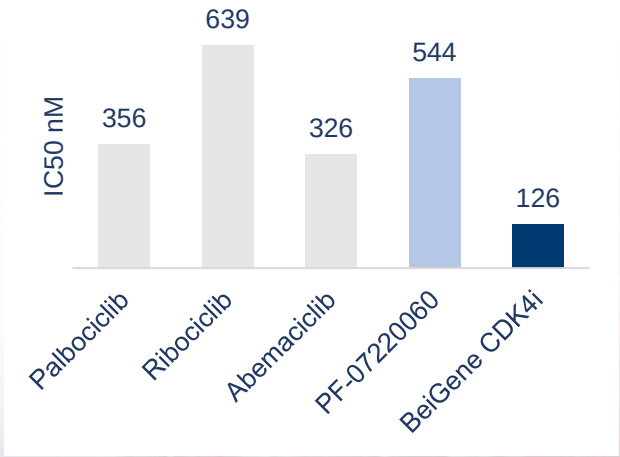
In Tumor Cell | In Bone Marrow

BeiGene

CDK4 Inhibitor

Highly potent and selective, with robust efficacy and improved tolerability*

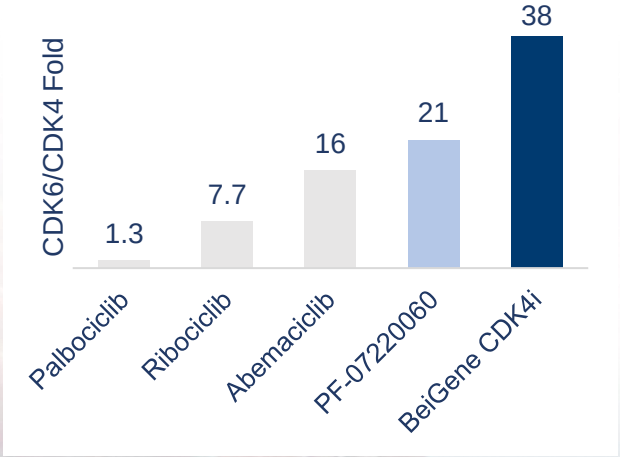
CDK4i Shows the Strongest CDK4 Potency



MCF-7 proliferation assay to assess CDK4 potency

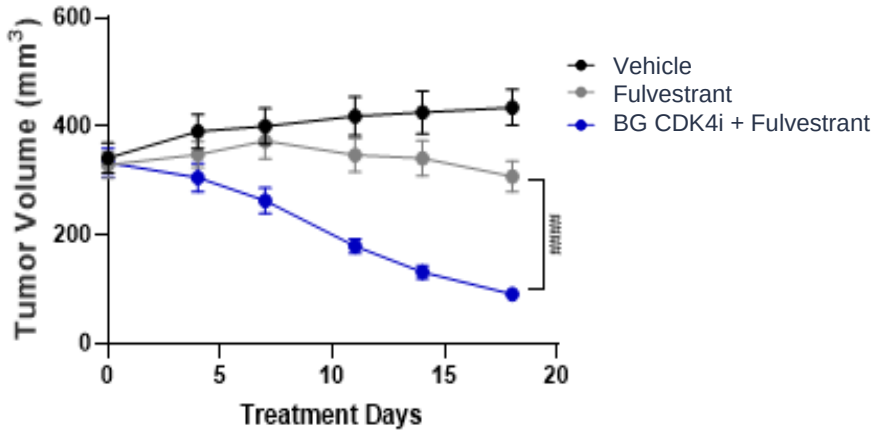
**CDK4 cellular IC50 measured through pRB in Jeko-1; CDK6 cellular IC50 measured through pRB in Pfeiffer with CDK4 KO*

CDK4i Has the Best CDK4/CDK6 Selectivity



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

CDK4i Shows Strong Efficacy in Combo with Fulvestrant



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

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

*In preclinical models

BeiGene

Leverage R&D Innovation to Generate Next Wave of Programs

Priority tumor types with blockbuster potential

Priority Tumor Types

Diversified Therapeutic Modalities

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

Future Cornerstone Programs

- *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**

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

Hematology

Solid Tumors

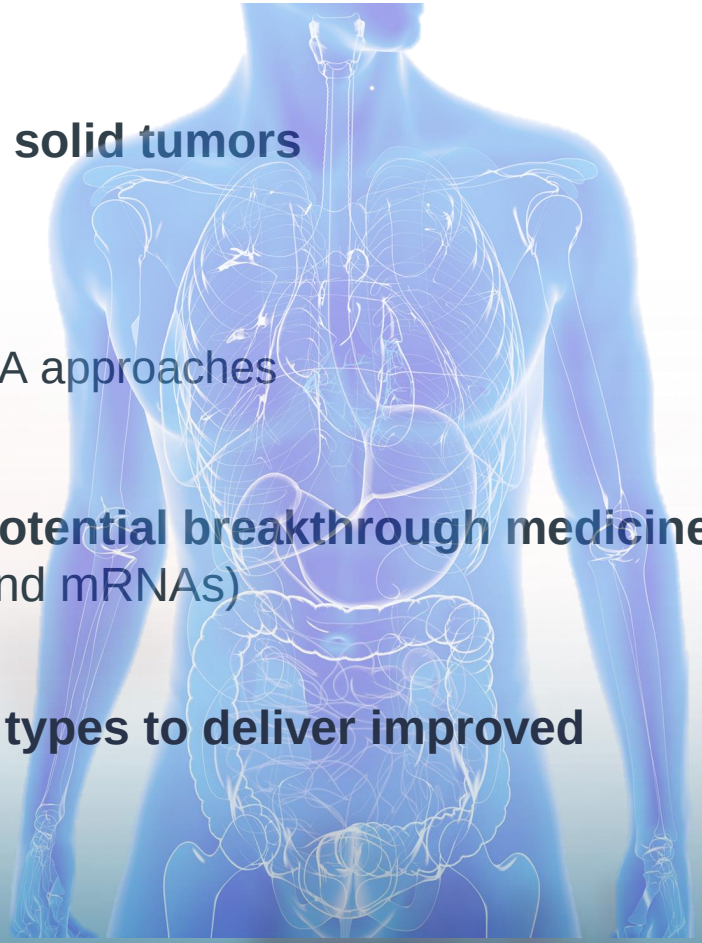
Research Innovation

BeiGene

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

2028 market size

Lung	$53B
Upper GI	$12B
Head and Neck	$5B
Breast	$42B
Colorectal	$8B

Hematology

2028 market size

B-Cell Malignancies	$45B
AML and MDS	$13B
Multiple Myeloma	$28B

Current disease areas

New tumor type expansion

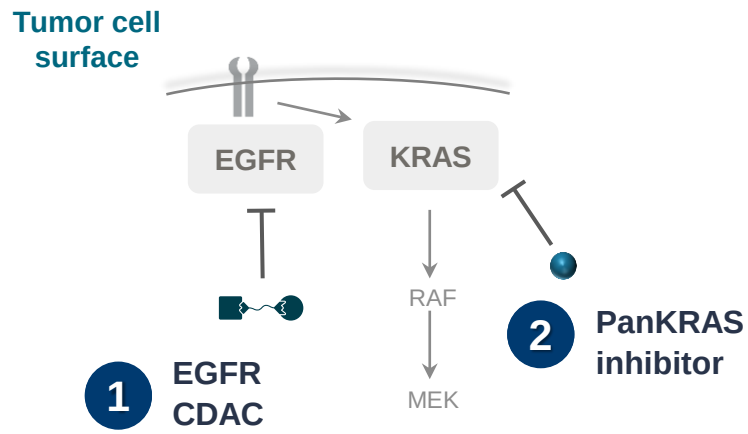
2028 WW Market Size estimates by EvaluatePharma
Upper GI includes GC, HCC, ESCC
B-cell Malignancies includes NHL (including DLBCL), CLL, and others (including MCL, MZL, WM, FL, SLL)

Lung Cancer Portfolio with FIC/BIC Potential

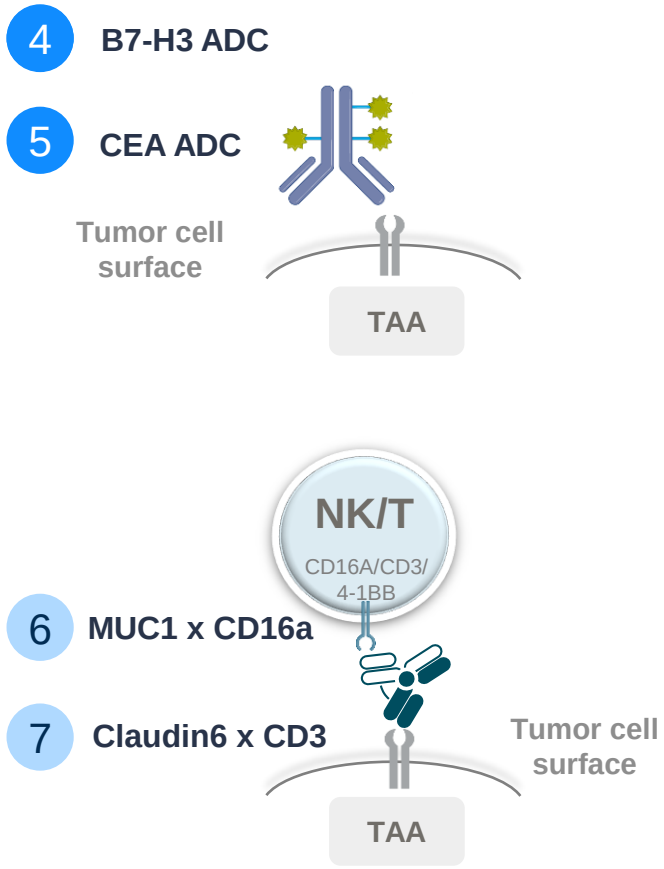
Over 30 scientifically driven targets with diverse modalities - highlighting 7

Small molecule | ADC | Bi/tri-specific

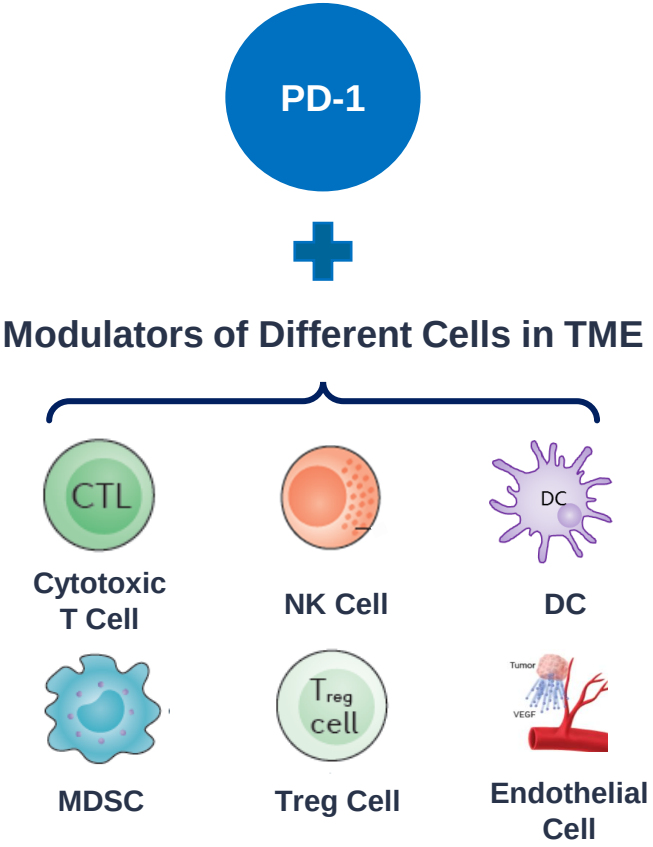
Oncogenic Signaling Targeting Therapies



Tumor Associated Antigens (TAAs)-Mediated Therapies



PD-1 Centered Pan Tumor Immuno-Oncology Therapies



BsAb: bispecific antibody; CDAC: chimeric degradation activating compound; ADC: antibody drug conjugate

BeiGene

1 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

MoA	Osimertinib-Sensitive Mutation	Osimertinib-Resistant Mutation	Destroy Scaffolding Function
3G TKI	✓	✗	✗
4G TKI	✓	✓	✗
CDAC	✓	✓	✓

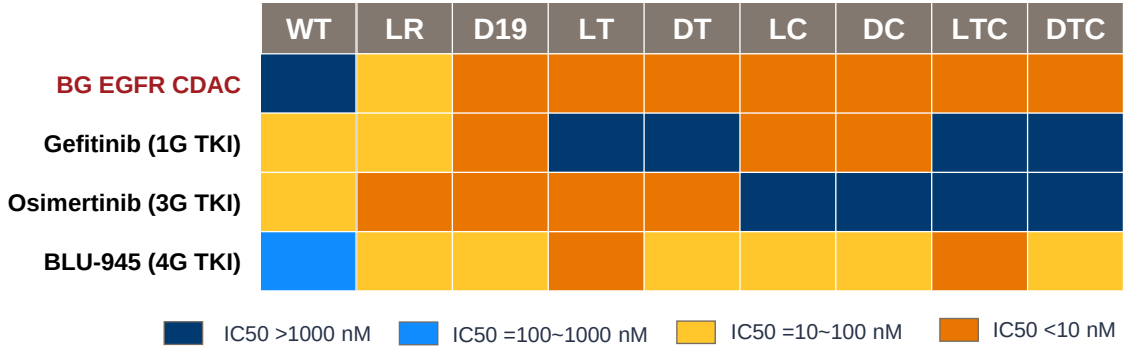
** 2020 Globocan; Wang P, et al. J ThoracDis. 2017, 9(7): 1973-1979; Wen S, et al. Oncologist. 2019, 24(11):e1070-e1081; J Clin Oncol . 2022 Feb 20;40(6):611-625.*

1 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

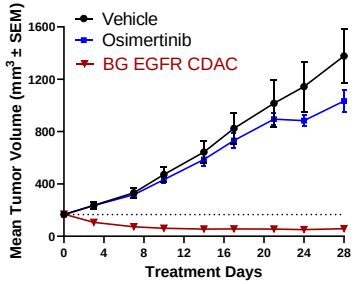
WT: wild-type; LR: L858R; D19: exon 19 deletion; DT: exon 19 deletion/T790M; LT: L858R/T790M; DC: exon 19 deletion/C797S; LC: L858R/C797S; DTC: exon 19 deletion/T790M/C797S; LTC: L858R /T790M/C797S

Broadest EGFRmut Coverage While Sparing WT

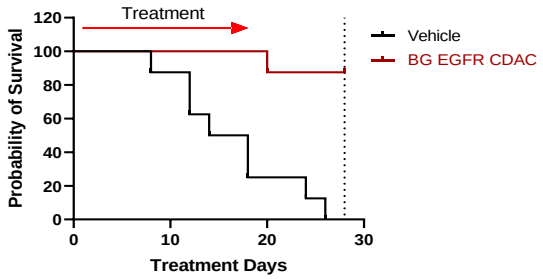


Robust Efficacy in Both Subcutaneous and Intracranial Xenograft Models

Osimertinib Resistant H1975-D19/C797S Model



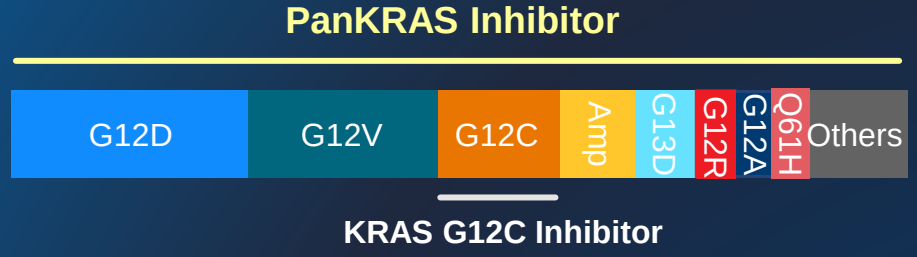
H1975 (L858R/T790M) Intracranial Model



2 PanKRAS Inhibitor

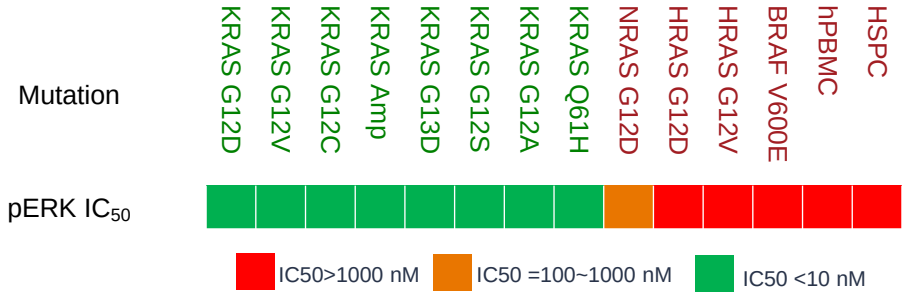
Addressing broad range of KRAS mutations in multiple tumor types

- 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**



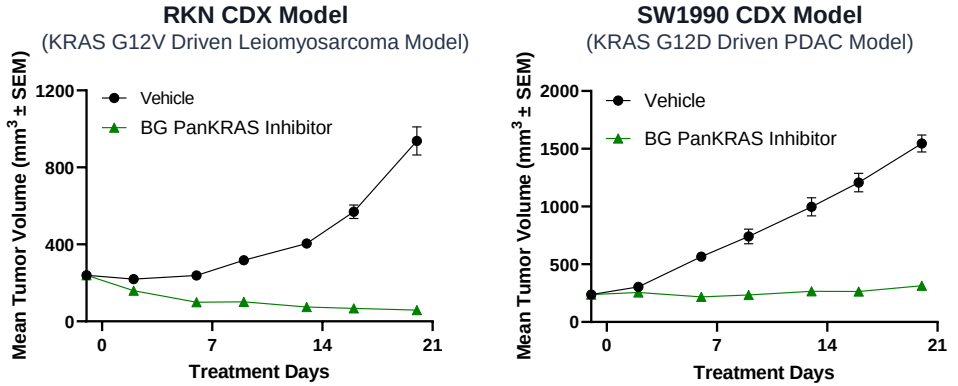
- **Adult mice with inducible KRAS KO appeared normal and healthy#,** suggesting low risk with inhibiting WT KRAS by panKRAS inhibitor
- **Highly potent across different KRAS mutations** with good selectivity against N/HRAS
- **Candidate selection in 2023 and to enter clinic in 2024**

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



hPBMC: Human peripheral blood mononuclear cells; HSPC: human hematopoietic stem/progenitor cell

Strong Anti-Tumor Efficacy in KRAS-Driven Xenograft Models



** Pharmacol Res. 2019 Jan;139:503-511.; Zhu, C.et al. Mol Cancer 21, 159 (2022); J Thorac Dis 2020;12(7):3776-3784*
 # Data on file

BeiGene

3 MTA-Cooperative PRMT5 Inhibitor

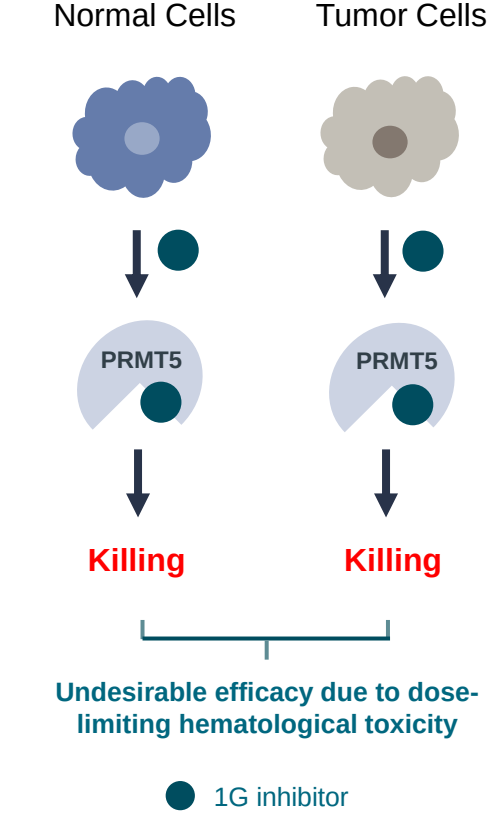
Next-generation PRMT5 inhibitor avoiding hematologic toxicity

- **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**

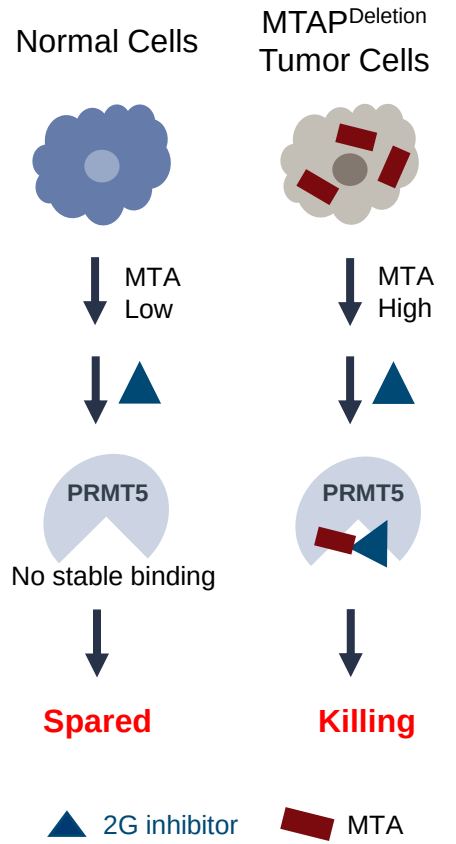
PRMT5: protein arginine methyltransferases 5; MTA: methylthioadenosine; MTAP: methylthioadenosine phosphorylase

**2020 Globocan; Konstantinos. M et al. Science. 2016, 351(6278): 1208-1213.*

1G PRMT5 Inhibitor Has Hematologic Toxicity



2G MTA-Cooperative PRMT5 Inhibitor Spares Normal Cells



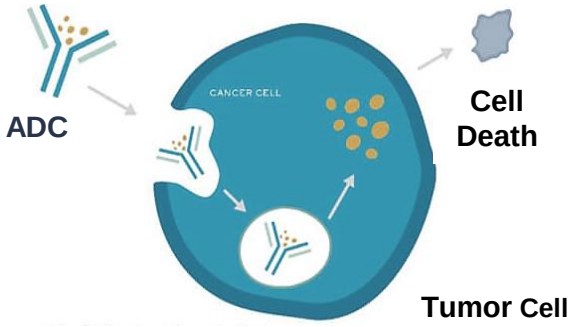
BeiGene

Tumor Associated Antigens as Tumor-Targeting Therapies

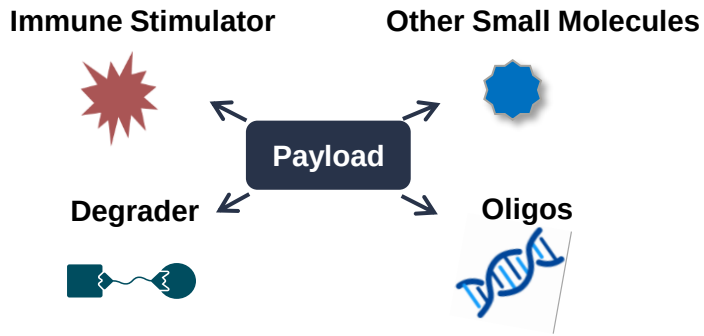
Broad applicability with multiple therapeutic modalities

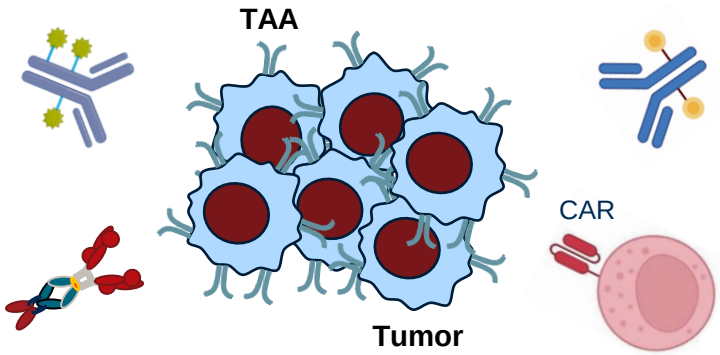
Research Innovation

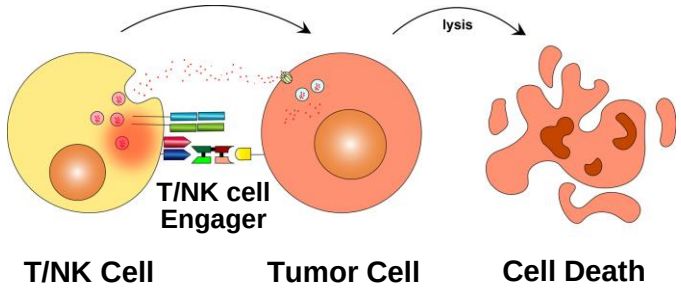
Toxin ADC
Current portfolio focus

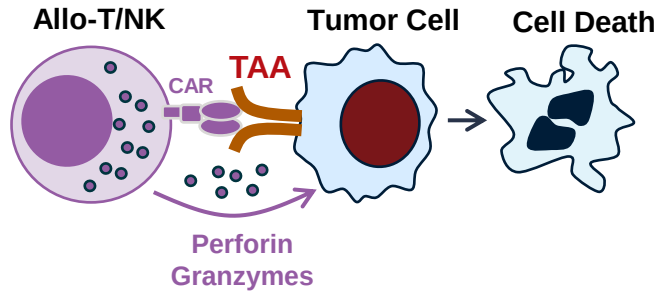


New concept ADC to unleash the power of drug conjugate









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

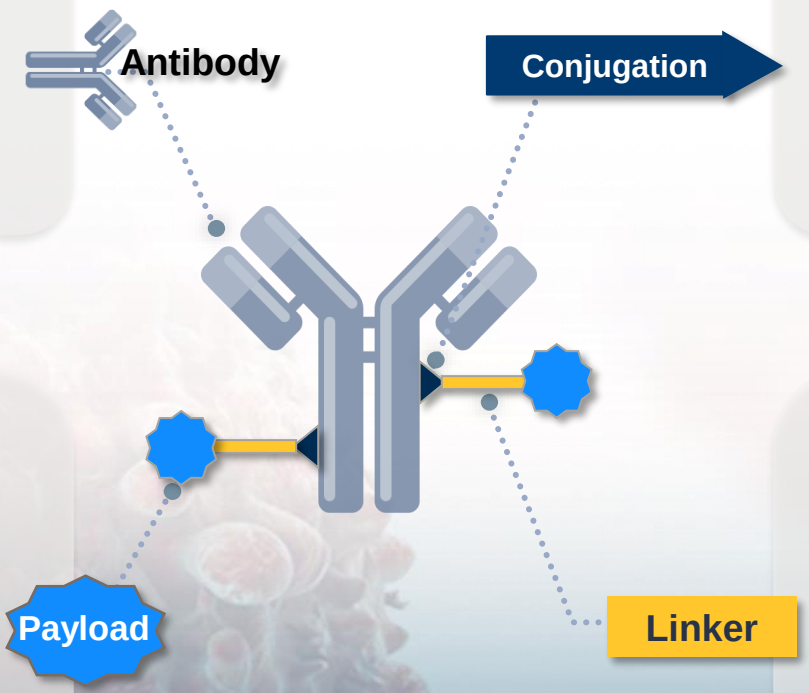
BeiGene

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



Homogeneous conjugation creates more uniform DAR/better stability

- Site specific
- Stable conjugator

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

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

4 B7-H3 ADC

BIC potential with stable DAR8 and strong bystander effect

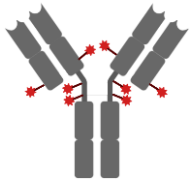
- **Highly expressed in multiple tumor types**, including lung, GI, gynecological cancers
  - B7-H3 moderate to high expression: 39% in lung adenocarcinoma, 84% in lung squamous cell carcinoma
- **Clinical validation** by lead competitor DS-7300 in small cell lung cancer and prostate cancer
- **Differentiated 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**

B7-H3 Expression	LUSC	LUAD	ESCC	CRC	HCC	OC	EC
B7-H3 Medium/High (H-score 101-300)	84%	39%	80%	23%	43%	25%	89%

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

BG B7-H3 ADC



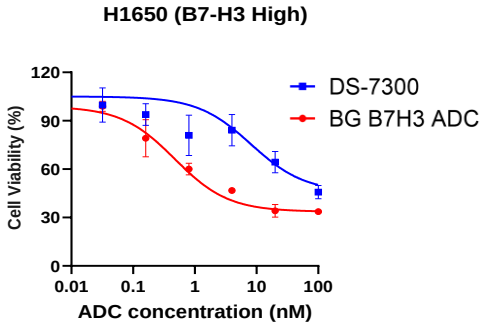
Attribute	DS-7300	BG B7H3 ADC	BeiGene Advantage
DAR	4	8	Higher DAR
Payload-Linker	DXd-GGFG	TopoI inhibitor-hydrophilic linker	Stronger bystander effect
Conjugation	Traditional Cysteine conjugation	Stable conjugator	Better stability

DS-7300 is B7-H3 ADC lead competitor from Daiichi Sankyo
TopoI, Topoisomerase I

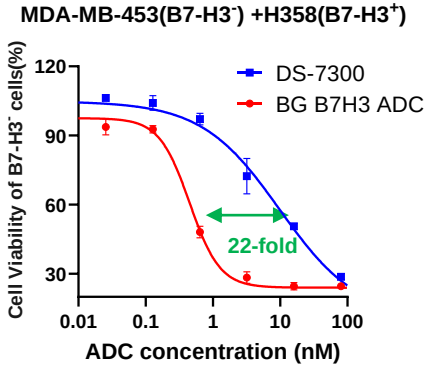
BeiGene

4 B7-H3 ADC
Active in DS-7300 insensitive and resistant models

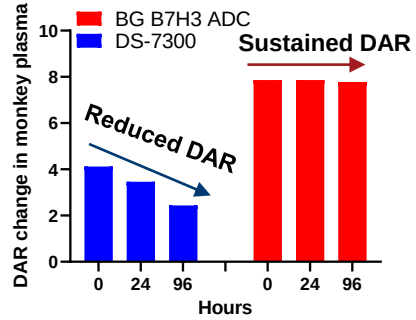
Superior On-Target Killing



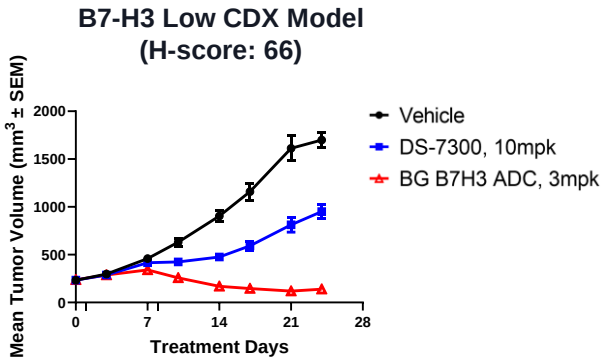
Better Bystander Killing

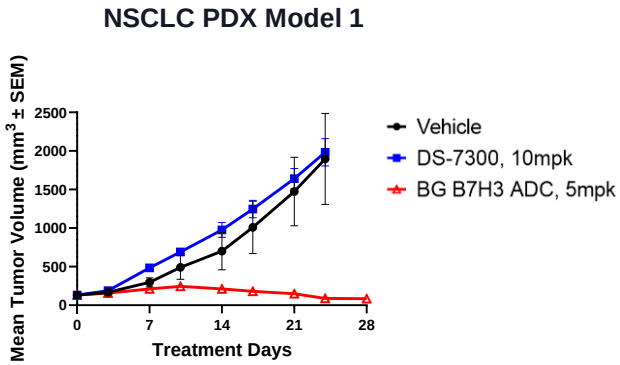


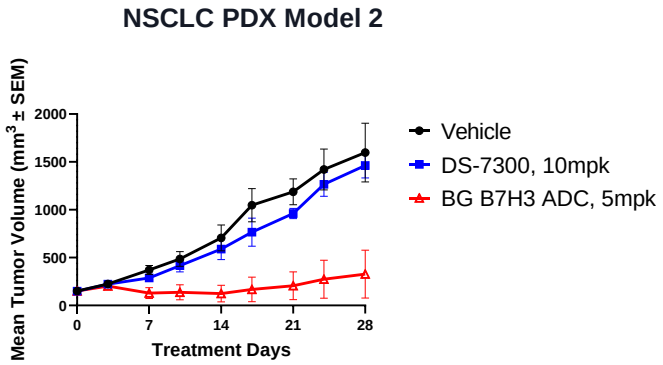
Higher DAR Stability in Monkey PK Study



Robust Tumor Shrinkage in Lead Competitor Insensitive/Resistant Models







Lead Competitor biosimilar used as benchmark
Data is from preclinical models

5 CEA ADC

BIC potential by expanding target population to CEA$^{Med/Low}$ lung and GI cancers

- **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^{High} lung cancer and 7% in CEA^{Med} lung cancer
  - Minimal efficacy in CRC and gastric cancer
- Differentiated ADC design to **expand into lung cancer pts with CEA$^{Med/Low}$ and GI cancer pts**
  - Different payload strategy: topoisomerase I (topoI) inhibitor
  - High DAR (8), stable conjugator and hydrophilic linker design
- **Candidate selected and to enter clinic in 2024**

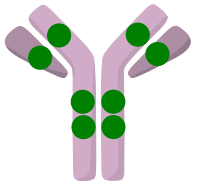
** SAR701 is in short for SAR408701*

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

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

BeiGene's CEA ADC with Differentiated ADC Design

BG CEA ADC



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

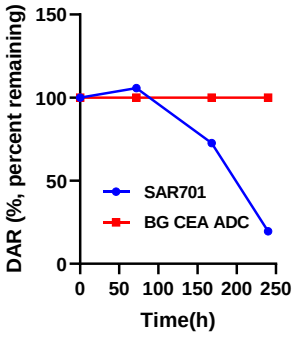
SAR701 is CEA ADC lead competitor from Sanofi

5 CEA ADC

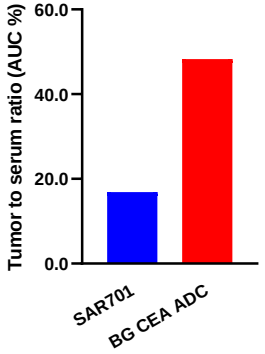
Better stability, tumor exposure, and bystander effects for better efficacy*

Better DAR Stability and Tumor Killing Than Lead Competitor

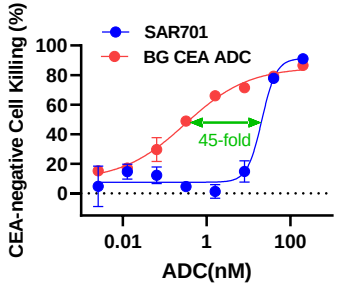
DAR Stability in Mouse PK Study



Tumor ADC Distribution



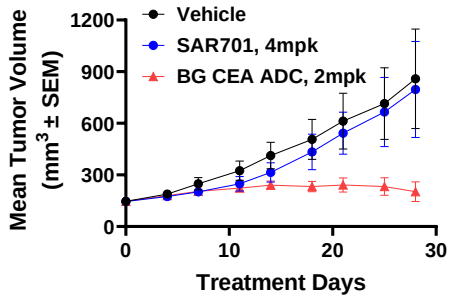
Bystander Killing



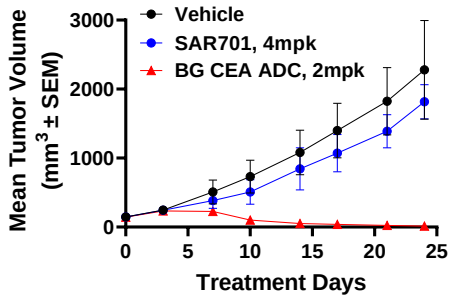
Lead Competitor biosimilar used as benchmark

Superior Efficacy in Lead Competitor Resistant Primary Tumor Models

Colon PDX Model (CEA^{Medium})



Gastric PDX Model (CEA^{Medium})



Lead Competitor biosimilar used as benchmark
Data is from preclinical models

BeiGene

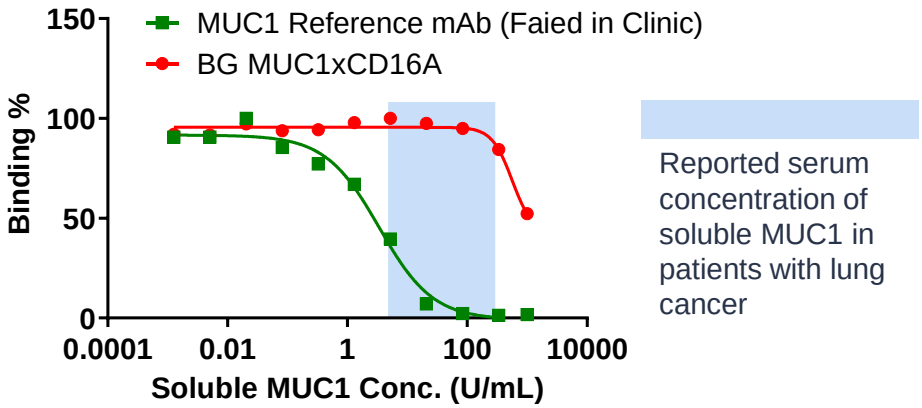
6 MUC1 x CD16A BsAb

Potential FIC MUC1 NK cell engager avoiding soluble MUC1 sink effect

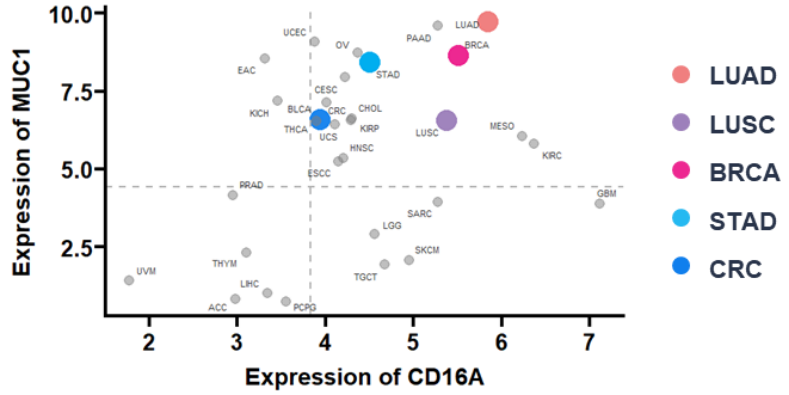
- **Highly expressed in lung, GI and breast cancers**, e.g., ~90% lung adenocarcinoma is MUC1 moderate/high*
- **Target MUC1 membrane proximal epitope** to avoid sink effect via minimal soluble MUC1 binding
- **Pursue NK engaging BsAb** since NK activating receptor CD16A is highly expressed in MUC1+ 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

Reduced Interference by Soluble MUC1



CD16A Highly Expressed in MUC1+ Tumors



Claudin6 x CD3 BsAb

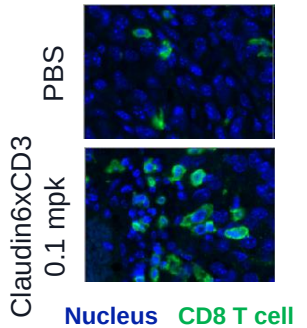
Highly tumor specific TAA/T-cell engager to treat lung & gynecologic cancers

- **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**

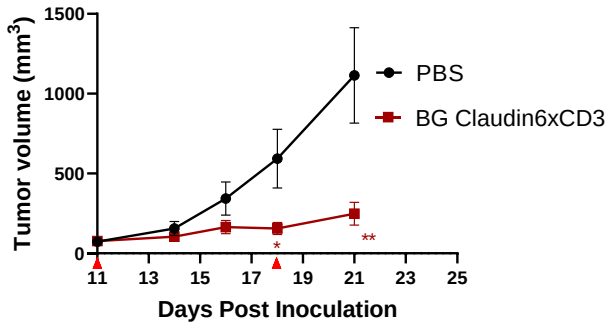
** Amgen, AACR Annual Meeting 2022*

Induces T-Cell Infiltration and Robust Efficacy in Immune Cold Tumor

T Cell Infiltration into Tumor

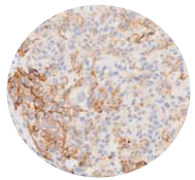


B16F10-hClaudin6 Syngenic Model

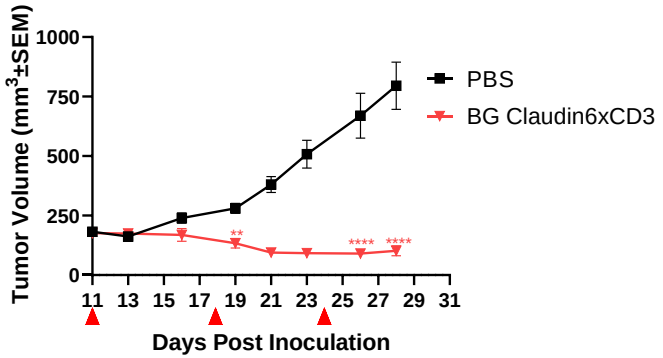


Elicits Strong Efficacy in Antigen-Heterogenous Model

Claudin6 Heterogeneous Expression in OV90 Tumor



OV90/hPBMC Transfer Model



BeiGene

Broad Targeted Therapy Lung Cancer Portfolio

Complementing immuno-oncology pipeline

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

PROGRAM	LUNG	UPPER GI	COLORECTAL	BREAST	HEAD and NECK	B CELL MALIGNANCY	AML/ MDS	PAN TUMOR
SM a	IND			IND				
SM b				Discovery				
Pan KRAS	Discovery	Discovery	Discovery					
SM c	Discovery	Discovery	Discovery					
PRMT5	Discovery	Discovery			Discovery			
CDK4				Discovery				
SM d				Discovery				
SM e				Discovery				
DGKz								IND
SM f								Discovery
SM g								Discovery
SM h								Discovery
BTK						Commercial		
BCL-2						Clinical	Clinical	
RAF	Clinical							
B-Raf	Clinical							
PI3K-δ						Clinical		Clinical
HPK-1								Clinical
SMAC	Clinical	Clinical	Clinical	Clinical	Clinical			
EGFR CDAC	IND							
CDAC								Discovery
BTK CDAC						Clinical		
CCR8								IND
PVRIG								IND
mAb a								Discovery
mAb b						Discovery		
PD1								Commercial
TIGIT								Clinical

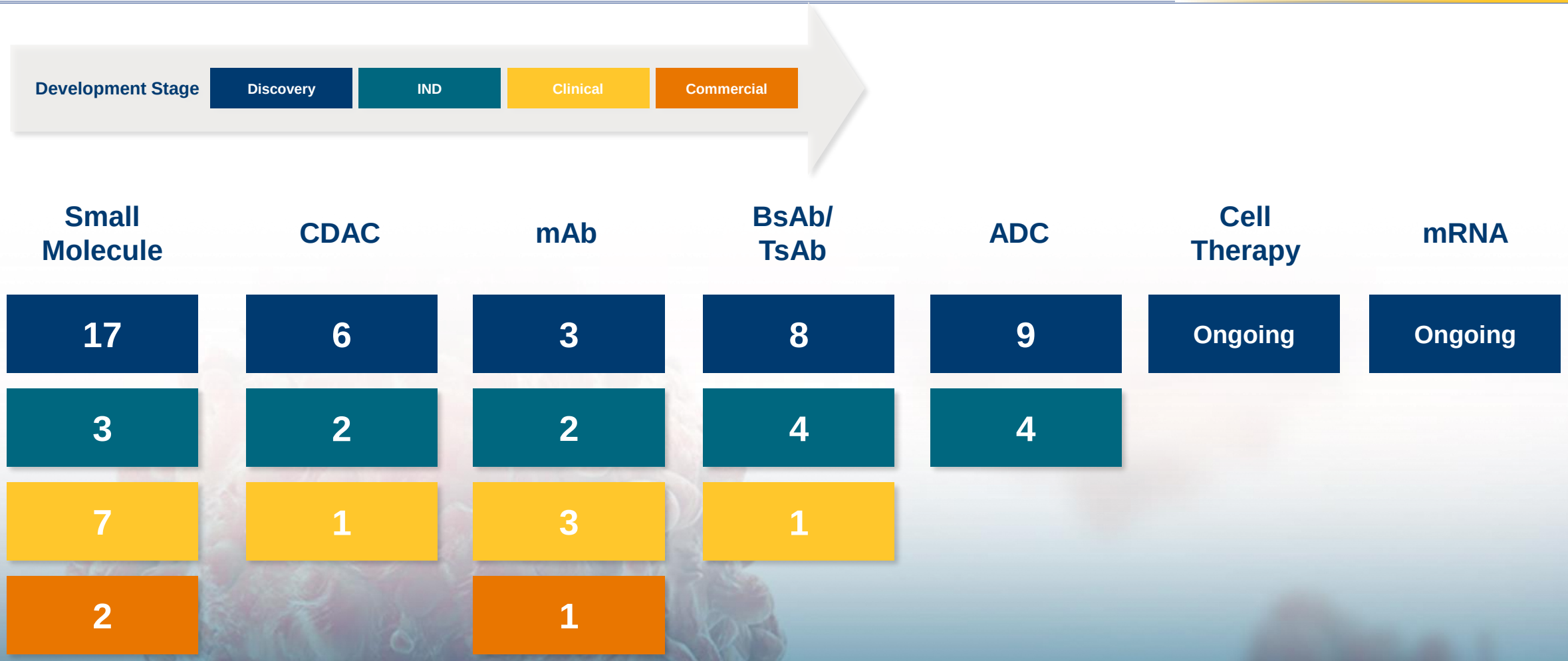
PROGRAM	LUNG	UPPER GI	COLORECTAL	BREAST	HEAD and NECK	B-CELL MALIGNANCY	AML/ MDS	PAN TUMOR
TIM-3								Clinical
OX40								Clinical
CT a								Discovery
CT b							Discovery	
B7H3 ADC	IND	IND	IND	IND	IND			
CEA ADC	IND	IND	IND					
ADC a	IND	IND		IND				
ADC b	Discovery	Discovery			Discovery			
ADC c		Discovery	Discovery	Discovery	Discovery			
ADC d	Discovery	Discovery	Discovery	Discovery	Discovery			
ADC e	Discovery	Discovery	Discovery	Discovery				
ADC f	Discovery	Discovery	Discovery	Discovery				
ADC g				Discovery				
ADC h						Discovery		
ADC i							Discovery	
ADC j	Discovery	Discovery	Discovery	Discovery				
BsAb a		IND						
MUC1 x CD16A	IND	IND	IND	IND				
BsAb b	Discovery							
Claudin6 x CD3	IND							
BsAb c		Discovery	Discovery		Discovery			
BsAb d							Discovery	
BsAb e						Discovery		
CEA x 4-1BB	Clinical	Clinical	Clinical					
TsAb a						Discovery		
TsAb b							Discovery	
TsAb c	IND							
Recombinant a								IND

Discovery | IND | Clinical | Commercial

SM: small molecule; mAb: monoclonal antibody; CT: cell therapy; BsAb: bispecific antibody; TsAb: trispecific antibody; recombinant: recombinant protein

Diversified Modalities and Broad Technology Platforms

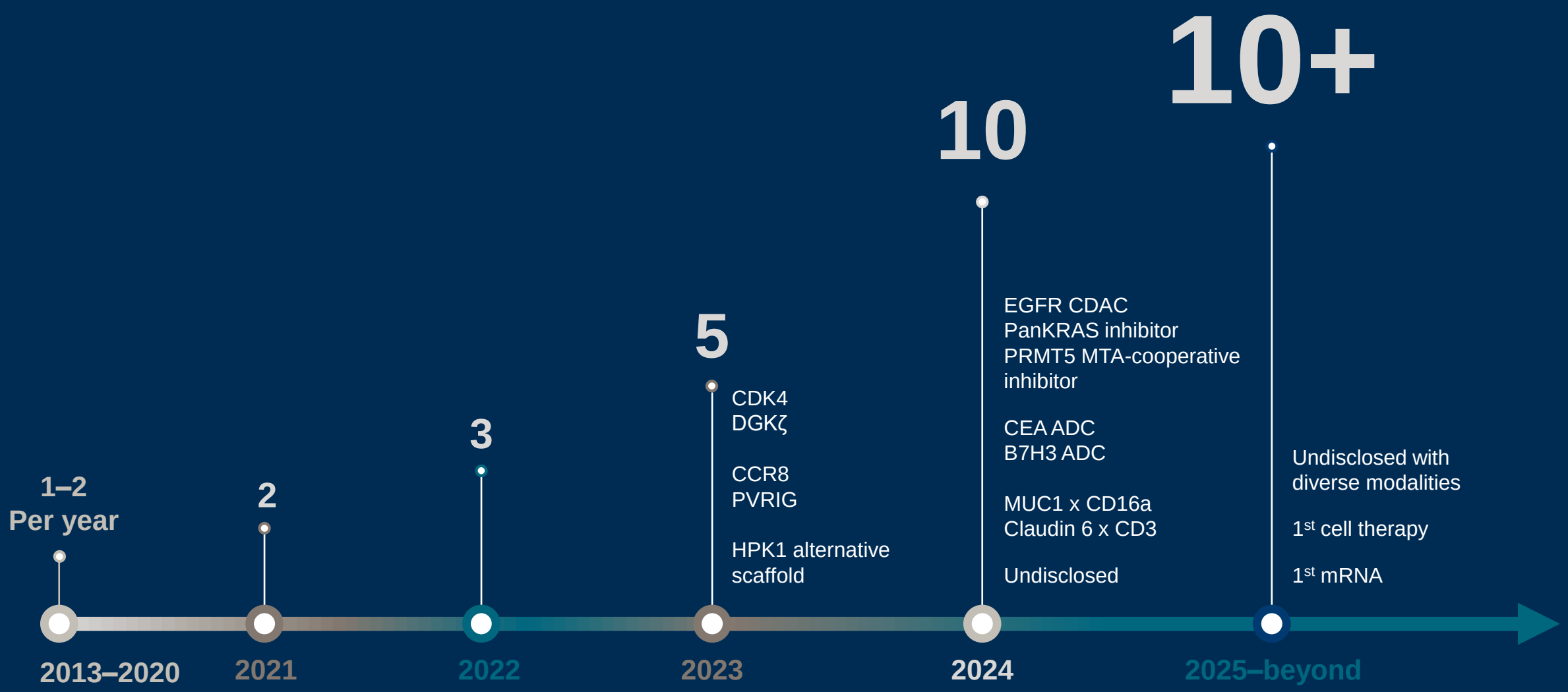
Accelerating innovations at scale



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



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Key Takeways from Research Innovation

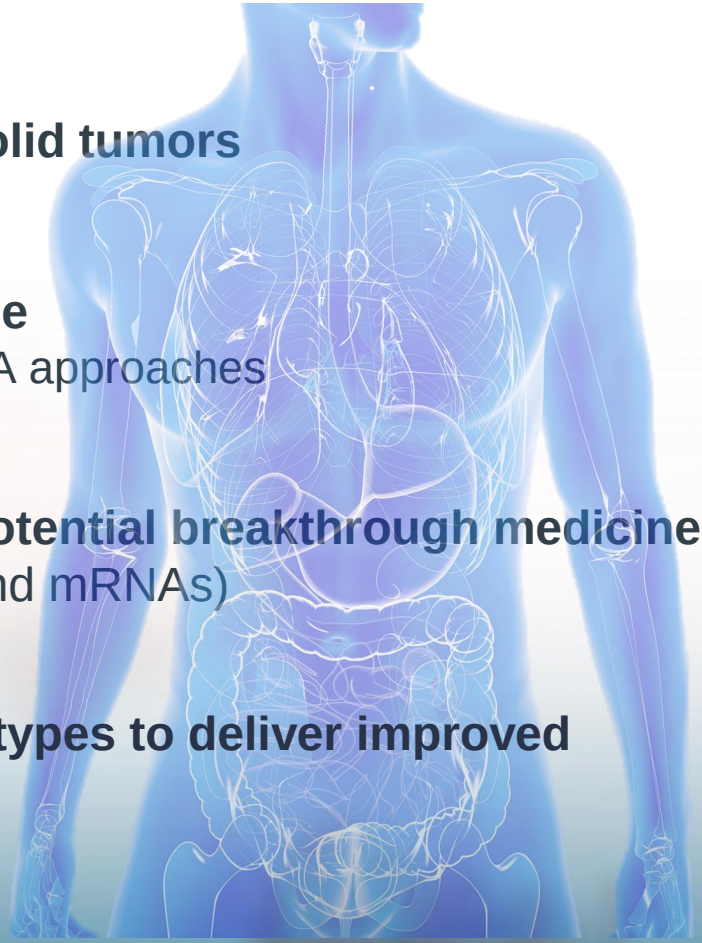
~1,100 innovative scientists delivering 10 new treatment changing molecules per year*

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

2. **A comprehensive tumor type approach with lung cancer as example**
   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

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

Broadest 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

Building Global Hematology Oncology Franchise Leadership

$22B market opportunity – Emerging leadership

BRUKINSA

$17B+ BTKi market by 2028, with $15B in heme-onc and $2B+ outside of oncology

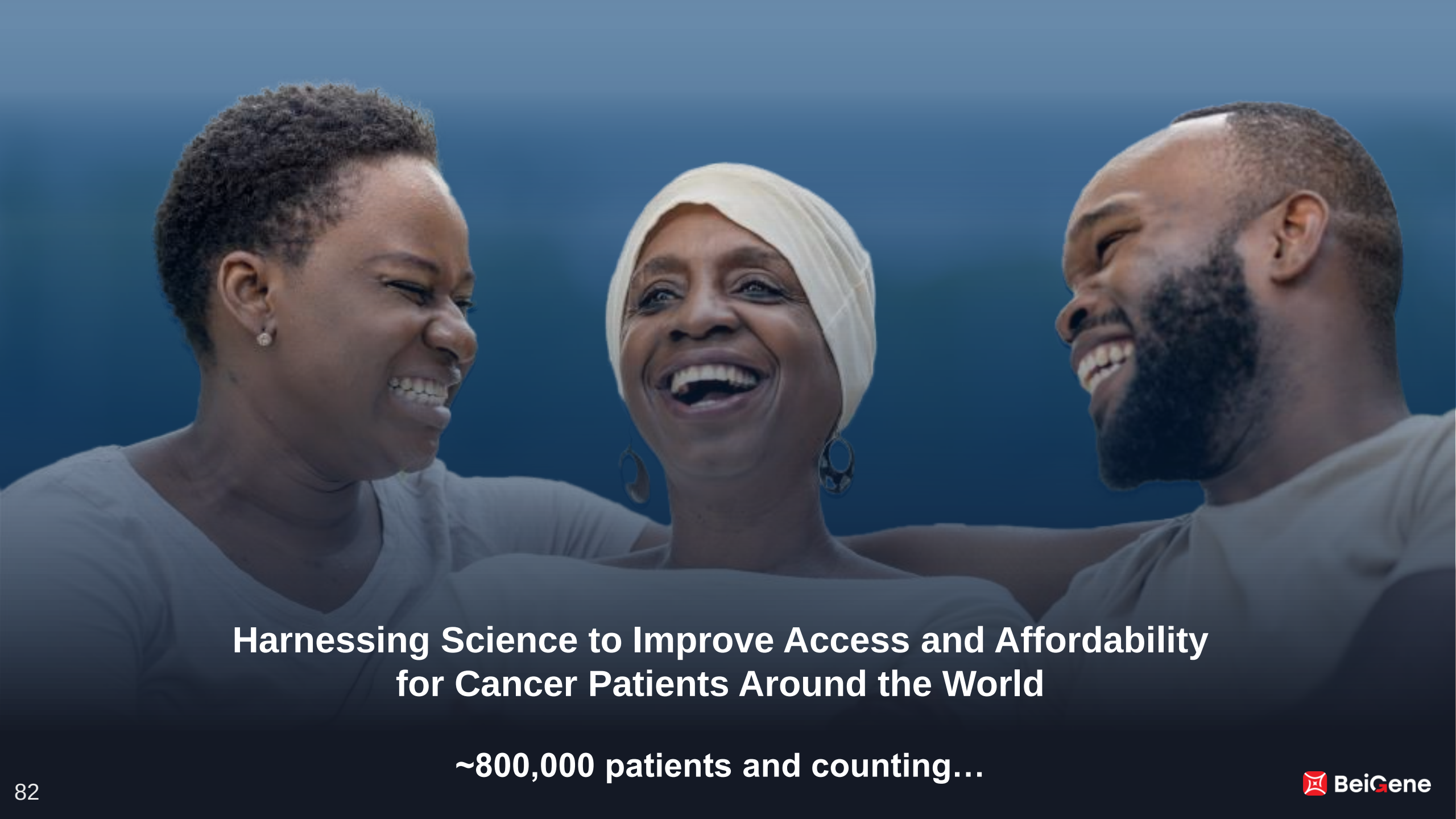
Sonrotoclax BCL2i

$4B+ BCL2i hematology market by 2028

BGB-16673 BTK CDAC

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

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*
		Approval in EU for 2L ESCC
		Approval in China for 1L HCC
		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

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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

Josh Neiman
Chief Commercial Officer, North America and Europe

Christiane Langer, M.D.
SVP, Global Medical Affairs (Ex-China)

BeiGene

Thank You!

Breakout Sessions and Panelists

1 Research

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Global Head of R&D

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VP, Head of Biologics

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CMO, Solid Tumors

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3 Commercial and Medical Affairs

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SVP, Global Medical Affairs
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4 Manufacturing and Supply Chain

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