BeOne’s Innovative Pipeline Progress

Investor R&D Day

BeOne

JUNE 26, 2025

Forward-looking Statements

Certain statements contained in this presentation and in the accompanying oral presentation, other than statements of fact that are independently verifiable at the date hereof, constitute forward looking statements. Examples of such forward-looking statements include statements regarding the projected size of the oncology market and related sectors; BeOne's research, discovery, pre-clinical and early-stage clinical programs and plans including proof of concept timing; the advancement of and anticipated clinical development and the conduct of late-stage clinical trials; expected data readouts and approvals; projected regulatory milestones and commercialization of BeOne's medicines; the ability of BeOne's assets to meaningfully outperform current medicines and address all lines of therapy; the potential for BeOne to have a significant market share in hematologic diseases; the ability for BeOne to become the most impactful global oncology company; and BeOne's future growth and financial performance. Actual results may differ materially from those indicated in the forward-looking statements as a result of various important factors, including BeOne'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; BeOne's ability to achieve commercial success for its marketed medicines and drug candidates, if approved; BeOne's ability to obtain and maintain protection of intellectual property for its medicines and technology; BeOne's reliance on third parties to conduct drug development, manufacturing, commercialization and other services; BeOne's limited experience in obtaining regulatory approvals and commercializing pharmaceutical products; BeOne's ability to obtain additional funding for operations and to complete the development of its drug candidates and maintain profitability, as well as those risks more fully discussed in the section entitled "Risk Factors" in BeOne's most recent periodic report filed with the U.S. Securities and Exchange Commission ("SEC"), as well as discussions of potential risks, uncertainties, and other important factors in BeOne's subsequent filings with the SEC. Except where otherwise noted, all information in this presentation is as of the date of this presentation, and BeOne undertakes no duty to update such information unless required by law.

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Some of the clinical data in this presentation relating to BeOne's investigational drug candidates is from pre-clinical studies or early phase, single-arm clinical trials. When such data or data from later stage trials are presented in relation to other investigational or marketed drug products, the presentation and discussion are not based on head-to-head trials between BeOne's investigational drug candidates and other products unless specified in the trial protocol. BeOne is still conducting pre-clinical studies and clinical trials and, as additional patients are enrolled and evaluated, data on BeOne's investigational drug candidates may change.

Definitive conclusions cannot be drawn from cross-trial comparisons or anticipated data as they may be confounded by various factors and should be interpreted with caution.

Agenda

●	Welcome, safe harbor, and agenda	**Liza Heapes**, Senior Director, Investor Relations
01	Global oncology leadership	**John V. Oyler**, Co-Founder, Chairman and CEO
02	R&D strategy and overview	**Lai Wang, PhD**, Global Head of R&D
03	Hematology portfolio	**Lai Wang** **Remus Vezan, MD, PhD,** VP, Hematology Clinical Development **Jacob D. Soumerai**, **MD,** Assistant Professor of Medicine, Harvard Medical School Hematologist / Oncologist, Mass General Hospital Cancer Center **Amit Agarwal, MD, PhD,** VP, Hematology Clinical Development
		◆ BREAK ◆
04	Solid tumor portfolio	**Mark Lanasa, MD, PhD**, SVP, Chief Medical Officer, Solid Tumors **Dr. Shom Goel, MBBS, PhD**, Group Leader and Medical Oncologist, Peter MacCallum Cancer Centre
05	Summary	**Lai Wang**
06	Q&A	**BeOne management team and key opinion leaders** Moderator: **Aaron Rosenberg**, CFO
07	Closing	**John V. Oyler**

Global oncology leadership

John V. Oyler
Co-Founder, Chairman and CEO

BeOne is a *global oncology* company that was *built differently* to deliver innovative medicines faster, more equitably and affordably to patients around the world

Purpose built with sustainable competitive advantages

Global access

6 continents,
40 offices,
11,000+
colleagues

Leading science

1,200+
proven
research
team

3,700+
global
development
and medical
affairs team

Superior returns

In-house manufacturing
including $800M U.S. flagship facility in New Jersey

We have treated **>1.7 million patients** with our medicines globally

Our commitment to business sustainability

BeOne is driven by purpose:
Transform cancer care and transcend borders to enable more affordable access to more patients here, and all around the world

What you will hear today – industry leadership in R&D

01

Our unique R&D model is delivering

- Proven quality, speed, cost, and technological advantages
- Diversified modalities, proprietary combinations, and scientific leadership

02

Hematology - serial innovation to drive sustained leadership

- Wholly-owned combinations of foundational assets BRUKINSA, sonrotoclax, and BTK CDAC to comprehensively address unmet needs in CLL
- Broadening beyond CLL, lymphomas e.g., B cell malignancies, MM

03

Solid tumors – breadth and depth across areas of focus

- Burgeoning franchises in breast, lung, and GI cancers each anchored by potentially best-in-class assets and combinations

Updates and new programs

Hematology

BRUKINSA Sonrotoclax BTK CDAC TCEs

Breast/Gyn

CDK4i B7-H4 ADC CDK2i/CDAC KAT6A/B

Lung

PRMT5i MAT2Ai EGFR CDAC/TsAb

GI

PanKRASi/CDAC RAS(ON) FGFR2b ADC

I&I

IRAK4 CDAC

CDAC – chimeric degradation activating compound, TCE - T-cell engager, ADC – antibody drug conjugate, TsAb – trispecific antibody

R&D strategy and overview

Lai Wang, PhD
Global Head of R&D

BeOne R&D stands at an inflection point

Proven capabilities converging to create a unique R&D model

Prolific research organization

Time, cost, and quality advantaged clinical development infrastructure

Global manufacturing and process development

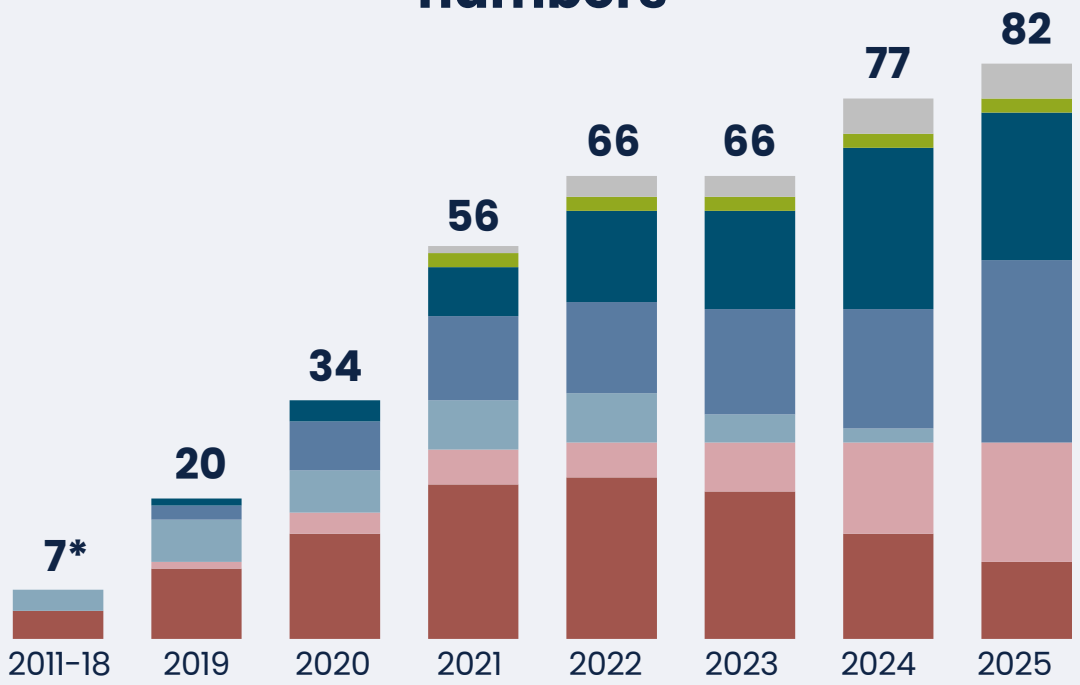
Superior R&D returns

Global commercial access

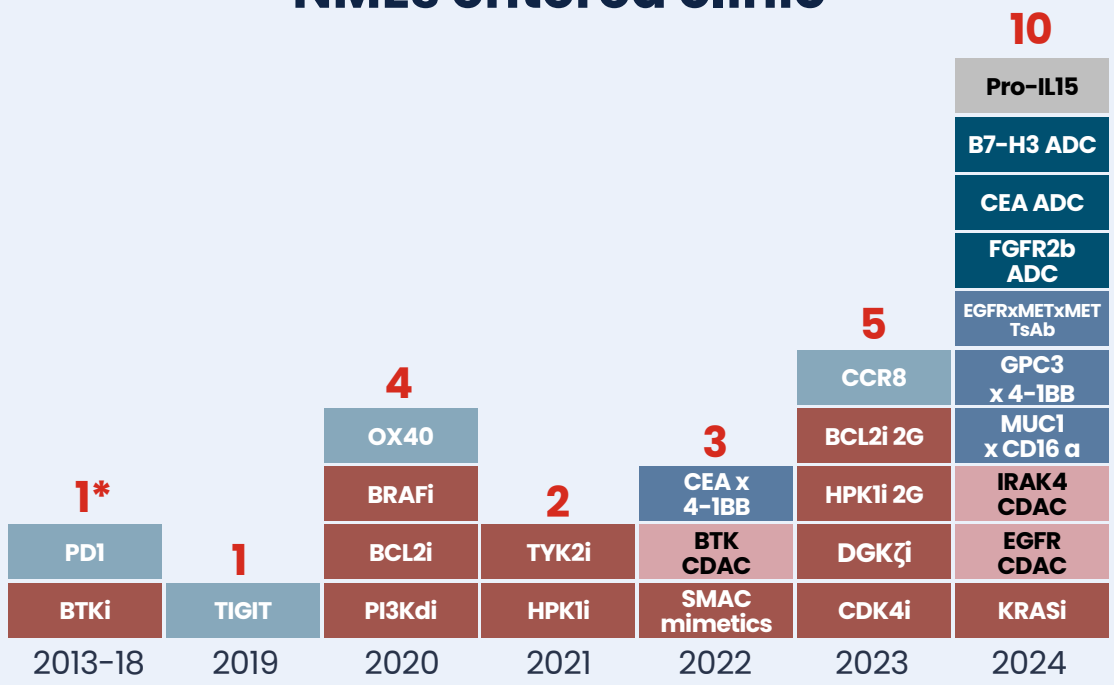
Evolution of BeOne's scaled research organization

New era of prolific, quality scientific output based on diversified modalities

Preclinical program numbers



Internally developed NMEs entered clinic



Traditional SM | CDAC | mAb | Bs/Ts Ab | ADC | Cell Therapy | Others

*Average numbers per year; SM, Small Molecule; CDAC, Chimeric degradation activation compound; ADC, Antibody drug conjugate; Bs/Ts Ab: Bispecific/Trispecific Antibody

We focus on delivering high-quality innovation

Committed to designing superior molecules with exceptional profiles

- Successful innovation with the same approach as BRUKINSA and now in parallel across many programs
- Perform the critical experiment and only advance high-quality molecules to clinic
- Halt programs that don't meet our high standard
  - Over the past 3.5 years, 60+ pre-clinical programs terminated

Sonrotoclax

More potent and selective design aiming to improve efficacy and safety, respectively

Shorter half-life for ease of TLS* monitoring

FGFR2b ADC

First-in-class, retain ligand binding to avoid on-target ocular toxicity induced by mAb

IRAK4 CDAC

First-in-class, fast ternary complex formation to drive complete degradation

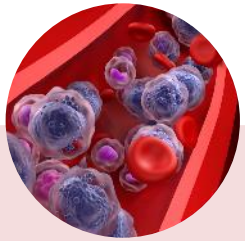
No QT issue

TLS, Tumor lysis syndrome

Establish scientific leadership in protein degradation - CDAC

Over 20 programs including degrader antibody conjugate (DAC)

Hematology



- BTK CDAC
- CDAC 6
- CDAC 7
- CDAC 8
- DAC 1
- DAC 2

Breast / Gynecologic

- CDK2 CDAC
- CDAC 9
- CDAC 10
- CDAC 11
- CDAC 12
- DAC 3

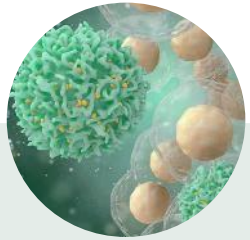
Lung

- EGFR CDAC
- KRAS CDAC
- CDAC 9
- CDAC 13
- CDAC 14
- CDAC 15

Gastrointestinal

- KRAS CDAC
- CDAC 9
- CDAC 13

Other Tumors



- CDAC 16
- CDAC 17
- DAC 3

Non-Oncology

- IRAK4 CDAC
- CDAC 18
- CDAC 19
- CDAC 20

Discovery | IND | Clinical

GI, Gastrointestinal; CDAC, Chimeric degradation activation compound; DAC, Degrader antibody conjugate; Cut-off date: MAY2025

Address various challenges in drug discovery via our industry-leading CDAC platform

Spare tissue-specific toxicity

Target X

Drug undruggable

Multiple targets

Destroy scaffold function

BTK CDAC, IRAK4 CDAC

Solve low potency issue

KRAS CDAC

Gain selectivity

EGFR CDAC, CDK2 CDAC

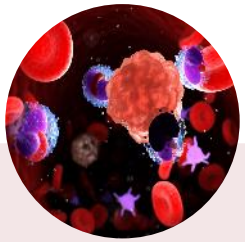
Reduce on-target resistance

BTK CDAC, EGFR CDAC

Emerging player in ADC field

Robust dual-TAA and novel payload ADC pipeline to drive next waves of innovation

Hematology



Novel payload ADC

- Novel DAC 1
- Novel DAC 2
- Novel ADC 2

mADC

- mADC 6

Breast / Gynecologic

Bs/TsADC

- B7-H4xHER3

Novel payload ADC

- Novel ADC 3
- Novel DAC 3

mADC

- B7-H4 ◆

Lung

Novel payload ADC

- RAS (ON)i ADC
- Novel ADC 3
- Novel ADC 4

mADC

- B7-H3 ◆
- CEA ◆
- ADAM9
- mADC 7
- mADC 8

Bs/TsADC

- EGFRxMETxMET
- Bs/TsADC 3
- Bs/TsADC 4
- Bs/TsADC 5
- Bs/TsADC 6

Gastrointestinal

Novel payload ADC

- RAS (ON)i ADC
- Novel ADC 4

mADC

- B7-H3 ◆
- FGFR2b ◆
- CEA ◆

Bs/tsADC

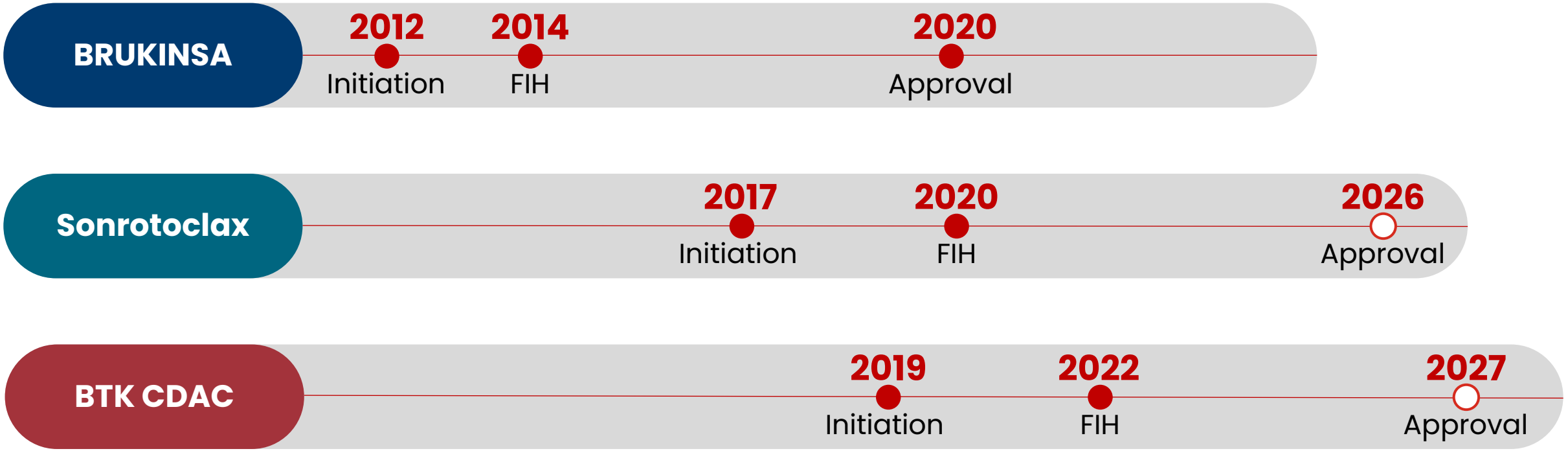
- EGFRxMETxMET
- Bs/TsADC 3
- Bs/TsADC 4
- Bs/TsADC 7
- Bs/TsADC 8
- Bs/TsADC 9
- Bs/TsADC 10

◆ **Clinical stage**

ADC, Antibody drug conjugate; DAC, degrader antibody conjugate; mADC, monospecific ADC; BsADC, bispecific ADC; TsADC, trispecific ADC; TAA, tumor associated antigen
Cut-off date: MAY2025

Technology platforms enable us to build disease franchise

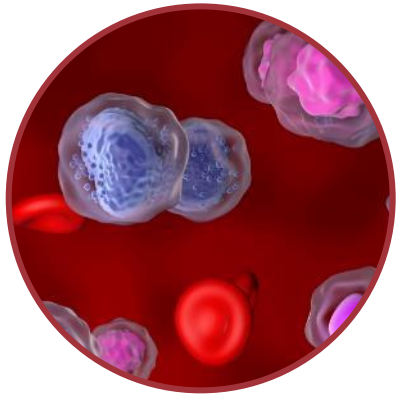
Our CLL franchise has been a great success



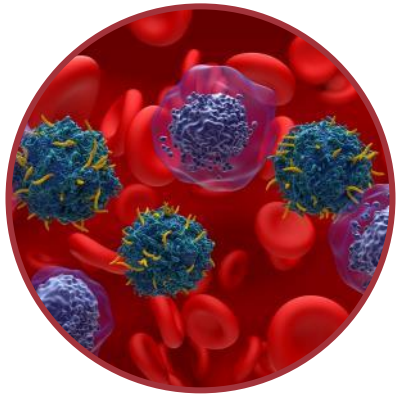
Approval indicates first (anticipated) CLL approval in a major market (U.S., EU, JP, CN)
FIH, First in human

Now, we can scale success across our disease areas of focus much faster

Build a deep pipeline by aiming to deliver 8-10 highly differentiated NMEs into the clinic in each of the following disease areas in the next 3-6 years



B-Cell Malignancies



AML/MDS

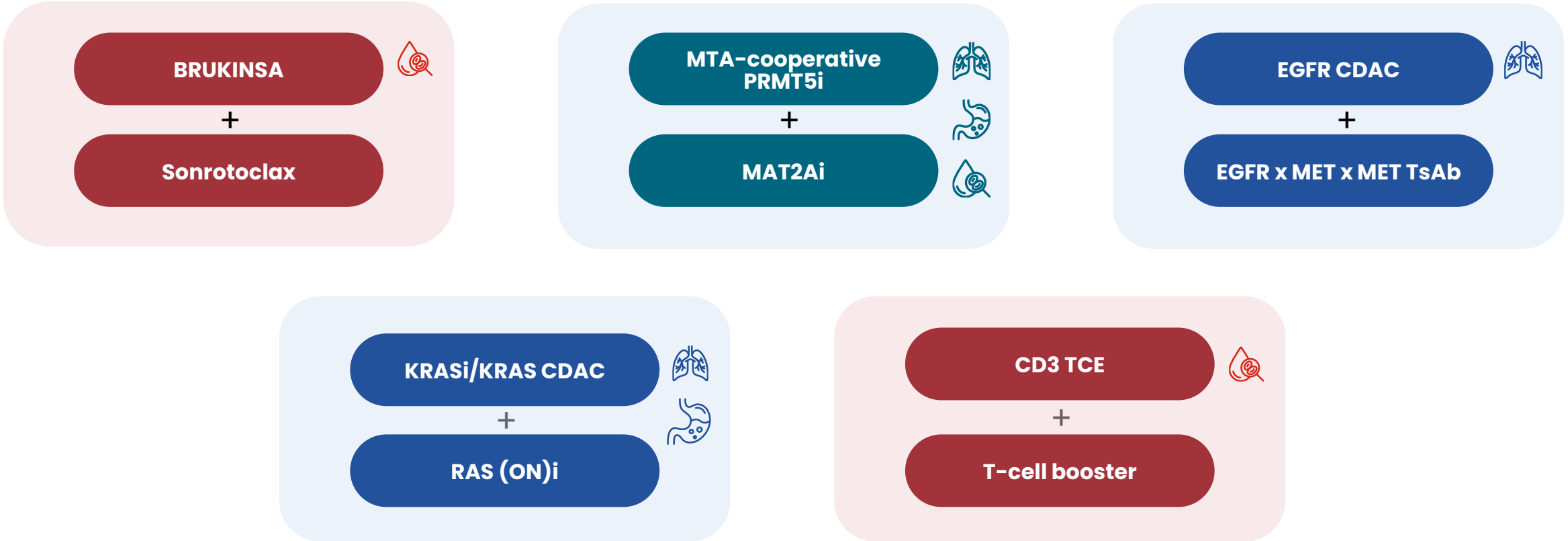
Breast/ Gynecological

Lung

Gastrointestinal

Immunology and Inflammation (I&I)

Deep pipeline enables unique and proprietary combinations



TCE, T-cell engager; TsAb, trispecific antibody; Target(i): target inhibitor

Our philosophy, scale, and sense of urgency has led to an industry-leading pipeline

Approved | Ph 3 | Ph 1/2

Heme | Lung | Breast / Gyn | GI | Other Cancers | I&I

Molecule	Indication	Phase
BRUKINSA® Zanubrutinib (BTKi)	TN CLL/SLL	Approved
	R/R CLL/SLL	Approved
	R/R WM and TN WM	Approved
	R/R FL	Approved
	R/R MCL	Approved
	R/R MZL	Approved
	TN MCL	Phase 3
	B-Cell Malignancies	Phase 2
	pMN	Phase 3
TEVIMBRA® Tislelizumab (PD1 mAb)	1L ES-SCLC	Approved
	IL NonSq NSCLC	Approved
	1L Sq NSCLC	Approved
	2/3L NSCLC	Approved
	Neo/Adj NSCLC	Approved
	IL ESCC	Approved
	2L ESCC	Approved
	1L GC/GEJC	Approved
	1L HCC	Approved
	2/3L HCC	Approved
	R/R cHL	Approved
	1L NPC	Approved
	Late Line MSI-H or dMMR	Approved
	2L UBC	Approved
	1L UBC	Phase 3
	SubQ Formulation	Phase 1

Molecule	Indication	Phase
Sonrotoclax (BCL2i)	TN CLL/SLL	Phase 3
	R/R CLL/SLL	Phase 3
	R/R MCL	Phase 3
	R/R WM	Phase 2
	R/R MM with t(11;14)	Phase 1
	AML/MDS	Phase 1
	B-Cell Malignancies	Phase 1
BGB-16673 (BTK CDAC)	R/R CLL/SLL	Phase 3
	R/R MCL	Phase 2
	B-Cell Malignancies	Phase 1
	Chronic spontaneous urticaria	Phase 1
IMDELLTRA® Tarlatamab (DLL3 x CD3 BITE®)[1]	1L ES-SCLC	Phase 3
	2L ES-SCLC	Phase 3
	LS-SCLC	Phase 3
	3L+ ES-SCLC	Phase 2
	2L+ ES-SCLC SubQ Formulation	Phase 1
ZIIHERA® Zanidatamab (HER2 BsAb)[2]	2L+ HER2+ BTC	Approved
	1L HER2+ GEA	Phase 3
BLINCYTO® Blinatumomab (CD3 x CD19 BITE®)[1]	R/R B-ALL SC	Phase 2
PARTRUVIX® Pamiparib (PARPi)	3L+ PSOC & PROC/PSOC	Approved
BGB-43395 (CDK4i)	BC and Solid Tumors	Phase 1
BG-68501 (CK2i)[3]	BC and Solid Tumors	Phase 1
BG-C9074 (B7-H4 ADC)[4]	BC and Solid Tumors	Phase 1
BGB-B455 (CLDN6 x CD3 BsAb)	Gyn and Solid Tumors	Phase 1

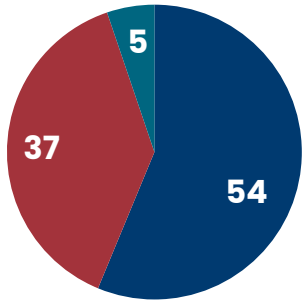
Molecule	Indication	Phase
BGB-21447 (BCL2i 2G)	HR+/HER2- BC	Phase 1
	B-cell Malignancies	Phase 1
BG-60366 (EGFR CDAC)	Lung Cancers	Phase 1
BG-T187 (EGFRxMETxMET TsAb)	Lung Cancers	Phase 1
BGB-58067 (MTA Coop. PRMT5i)	Lung Cancers	Phase 1
	GI Cancers	Phase 1
BG-89894 (MAT2Ai)[5]	Lung Cancers	Phase 1
	GI Cancers	Phase 1
BGB-53038 (PanKRASi)	Lung Cancers	Phase 1
	GI Cancers	Phase 1
BG-C477 (CEA ADC)	Lung Cancers	Phase 1
	GI Cancers	Phase 1
BGB-B3227 (MUC1 x CD16A BsAb)	Lung Cancers	Phase 1
	GI Cancers	Phase 1
BGB-2033 (GPC3 x 4-1BB BsAb)	GI Cancers	Phase 1
BG-C137 (FGFR2b ADC)	GI Cancers	Phase 1
BGB-A3055 (CCR8 mAb)	Solid Tumors	Phase 1
BGB-26808 (HPK1i)	Solid Tumors	Phase 1
BGB-C354 (B7-H3 ADC)	Solid Tumors	Phase 1
BGB-R046 (IL-15 Prodrug)	Solid Tumors	Phase 1
BGB-30813 (DGKζi)	Solid Tumors	Phase 1
Xaluritamig (STEAP1 x CD3 XmAb®)[1]	mCRPC	Phase 1
BGB-45035 (IRAK4 CDAC)	Immunology & Inflammation	Phase 1

1) Amgen collaboration, 2) Zymeworks/Jazz collaboration, 3) Ensem collaboration, 4) DualityBio collaboration, 5) CSPC collaboration

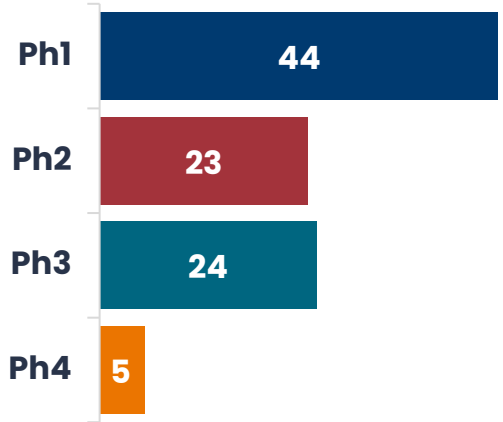
Proliferation of trials across phases, modalities, and disease areas

Assets with active clinical development	Ongoing clinical trials	Ongoing or planned submissions	Total approvals
30	96	154 *(83 countries)*	225 *(75 countries)*

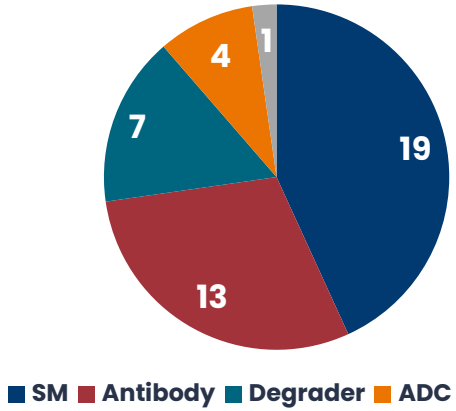
of trials by TA



of trials by phase

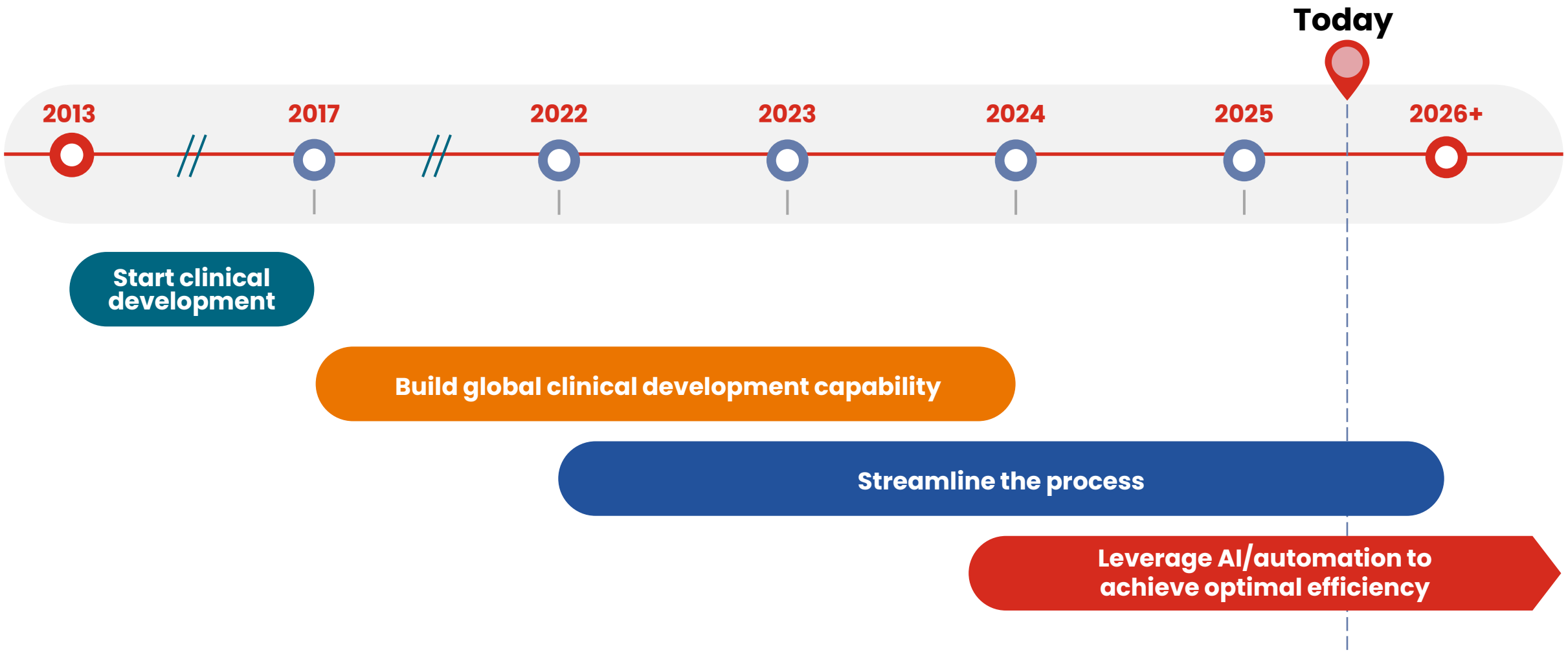


of Phase 1 trials by modality

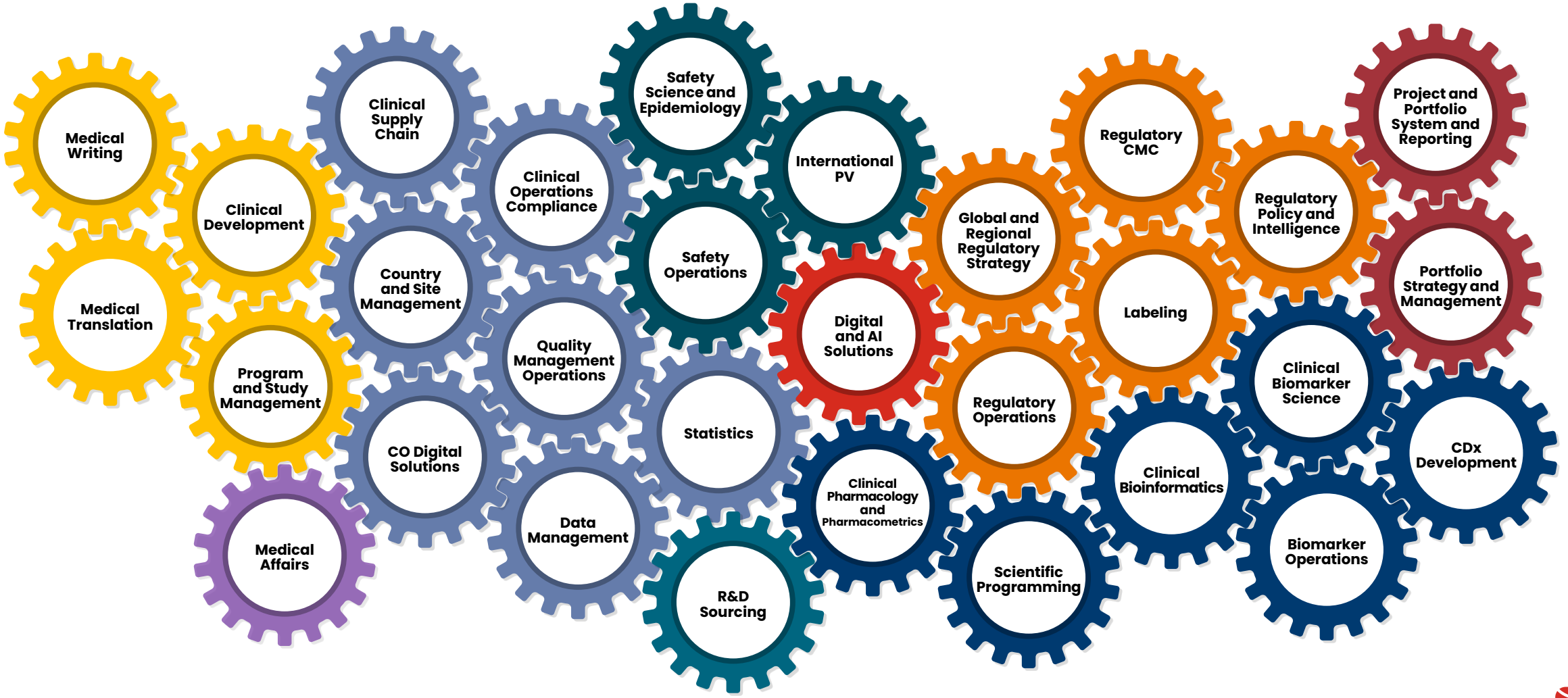


Ongoing trials as of 28MAY2025 includes trials with Start-up, Enrolling, Maintenance, Close-out status
Planned submissions through 2H26

It took substantial effort to build our global clinical development organization, but it is now firing on all cylinders

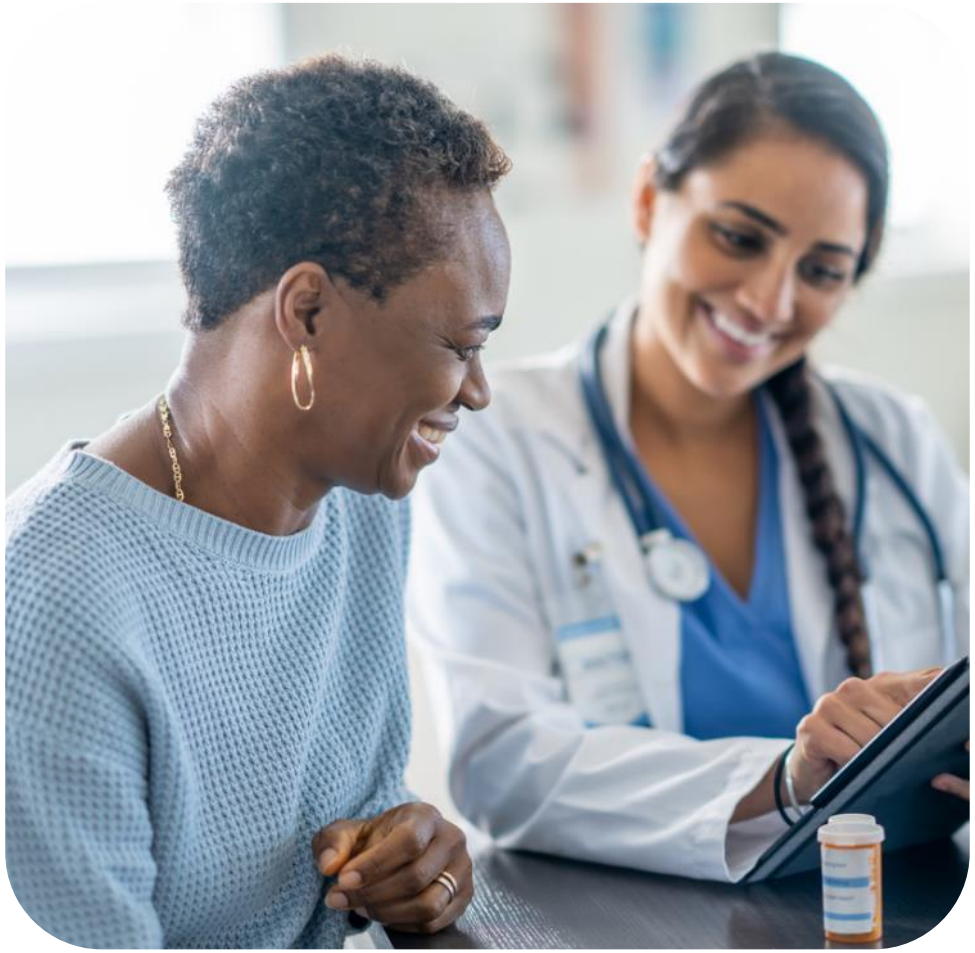


Our 3,700+ global clinical development team is complex and spans many functions



CDx: Companion diagnostic; PV: Pharmacovigilance

Leveraging internal global clinical capabilities to drive speed, cost, quality, and site access



Tremendous speed all around the world, including in hard to access regions

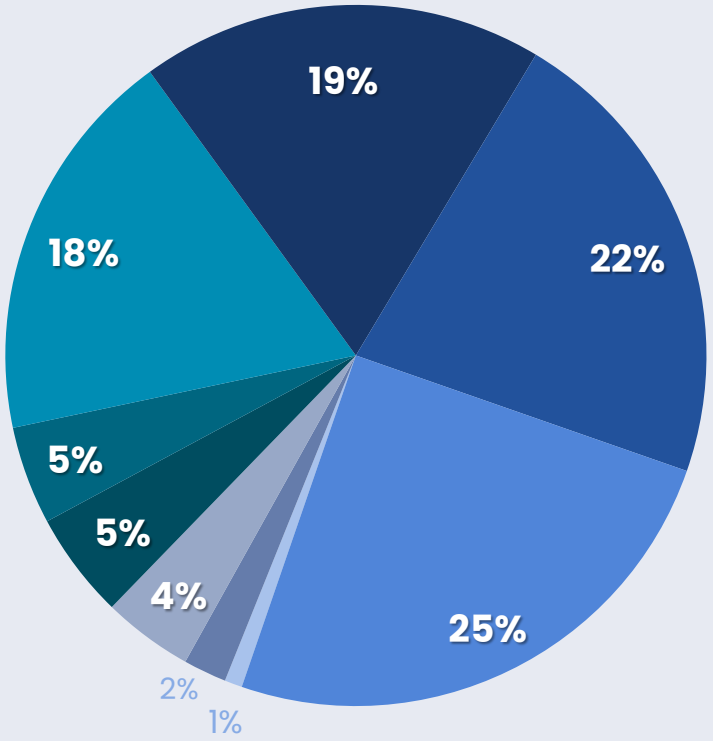
Over **100 inspections** by 15 different health authorities across 10+ countries with **zero critical findings**

Lower cost trials by eliminating CRO with internal operating model

Established strong **direct site relationships** to enable operational **efficiency**

BeOne's patient enrollment spans diverse regions, reflecting our commitment to global representation

Global patient enrollment in 2025



- Southeast Asia
- Middle East
- Eastern Europe
- Japan and Korea
- LATAM
- Australia and New Zealand
- Western Europe
- North America
- China

Broad and diverse global enrollment across distinct regions

Growing representation in emerging markets

Data as of 27MAY2025

Streamline drug development process toward perfection, delivering with rigor and speed

Start of GLP tox study to first subject enrolled (FSE) in Phase 1a

9.9 months
(12 programs)

3.6 weeks in average from draft tox report to protocol finalization

1.2 weeks from protocol finalization to EC submission in AUS

2.6 weeks from last piece of data to IND submission in U.S.

Time per dose escalation cohort

7.1 weeks
(13 programs/88 cohorts)

Real-time data cleaning

2-4 days from last patient completing safety evaluation to next dose level decision (SMC meeting)

Allow early patient screening **~2 weeks** before SMC meetings

Initiate dose expansion after recommended dose identified

4.4 weeks
(3 programs)

Initiate CDP discussion **~9m prior** to the predicted completion of dose escalation

Completion of critical documents earlier (protocol amendment, IND/EC submission) for site readiness prior to identification of recommended dose for expansion

Data represents FSE, dose escalation cohort, or Ph1 dose expansions initiated since 01JAN2024

CDK4i and BTK CDAC examples illustrate how we interrogate every development step to maximize efficiency

CDK4i (BGB-43395)

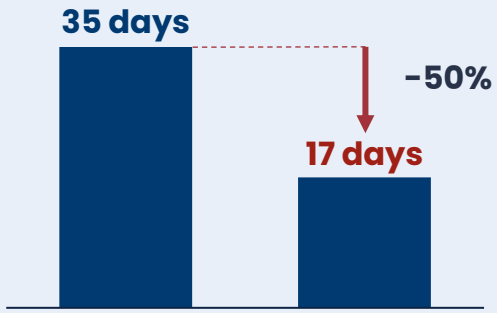
- Enrolled **300+ patients within ~18 months**
- Opened 67 sites in 11 countries
- **Evaluated 22 dose** levels, including 10 mono and 12 combo cohorts
- **Initiated 1L letrozole combo in CDK4/6i** naïve patients **13 days** after finishing DLT evaluation of this combo in 2L+ by strategically **opening sites** in regions with **limited CDK4/6i availability** (Brazil, Malaysia, Moldova, and Thailand)

BTK CDAC (BGB-16673)

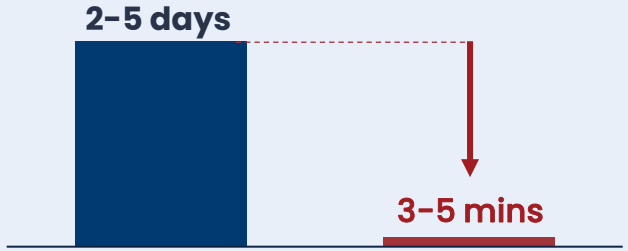
- Enrolled **600+ patients** in the past 3 years
- Opened ~100 sites in 22 countries
- Effectively leveraging backfill and safety expansion cohorts to generate dose response data
- **Efficient path to RP2D/RP3D** with manageable risk
  - **4 weeks** from **recommended dose for expansion** to initiating potentially pivotal Phase 2
  - **2 months** from submission of FDA RP2D briefing book **to starting pivotal Phase 3**

Our internal model enables use of AI/automation to further improve efficiency

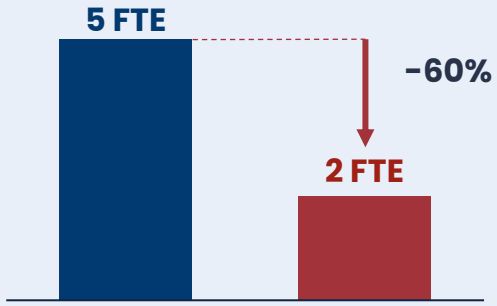
50% time reduction from data-cutoff to database lock



>99% faster in table/figure preparation for safety monitoring committee meeting



3 FTE savings per study in data analysis



90% efficiency increase in SAE review and summary[1]

No AI/automation | Leveraging AI/automation

Note: All data comparisons are with BeOne-sponsored studies
[1]Applied in 5200 serious adverse event (SAE) cases of 54 studies

BeOne R&D strategy to transform patients' lives

1. Develop a **deep and impactful portfolio** in focused disease areas
2. Execute **fast-to-POC** for value maximization

BeOne
R&D
Overall Strategy

3. Initiate **combination therapies early** to win
4. Advance **only transformative medicines to late-stage** development

ENABLED BY:

1,200+ researchers covering **diverse modalities**, advancing science with **urgency and agility**

Maximizing clinical **trial efficiency** with **CRO-free model**, powered by **automation** and **AI technologies**

Hematology portfolio

Lai Wang, PhD
Global Head of R&D

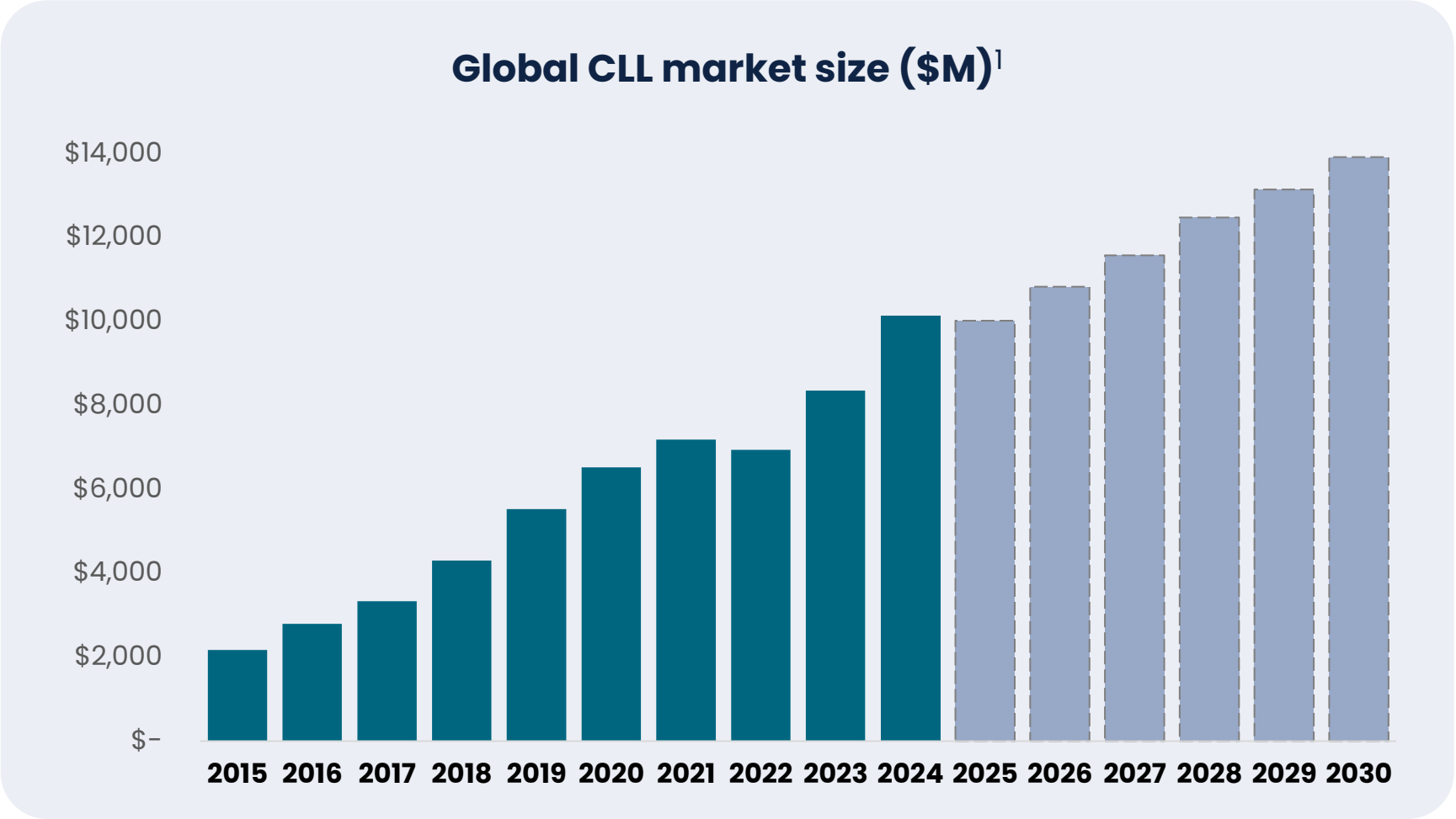
Remus Vezan, MD, PhD
Vice President, Hematology Clinical Development

Amit Agarwal, MD, PhD
Vice President, Hematology Clinical Development

Novel therapies have transformed the CLL treatment paradigm

Key FDA approvals in CLL

- **2013:** Gazyva
- **2014:** Imbruvica
- **2016:** Venclexta
- **2019:** Calquence
- **2023:** **BRUKINSA**
- **2023:** Jaypirca



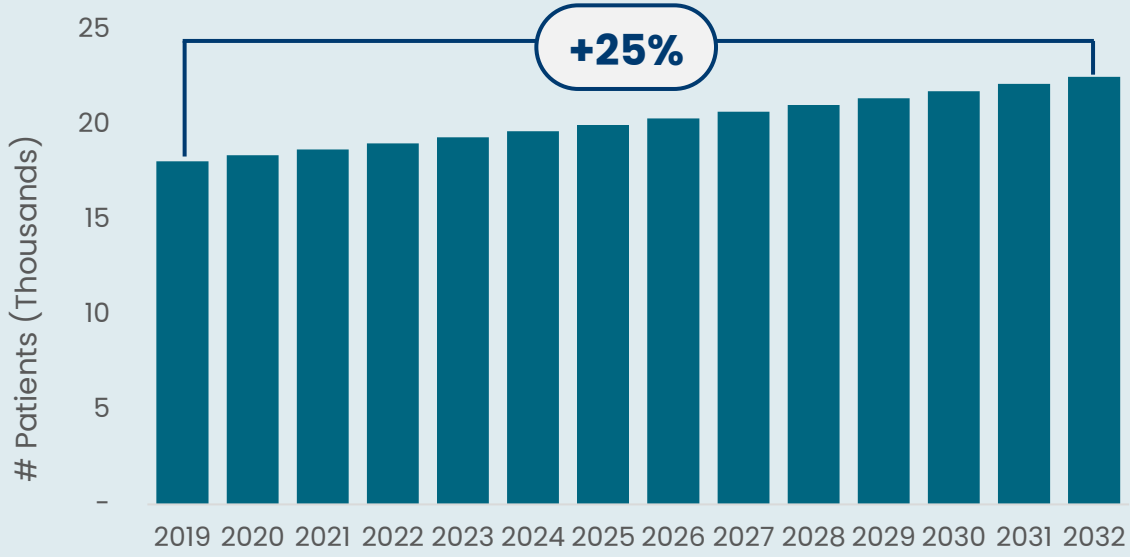
[1] EvaluatePharma, accessed 27MAY2025

Yet there is still substantial unmet need in the treatment of CLL

Fixed duration

- Patients and physicians are interested in the prospect of time-limited therapy
- However, current first-generation venetoclax-based regimens fail to address this unmet need due to efficacy, safety and convenience challenges
- We believe that patients should not have to accept an unfavorable treatment outcome in exchange for time off treatment

R/R CLL epidemiology[1]



Novel options are needed for the increasing number of patients who will relapse after 1L therapy

[1] DelveInsight, U.S. + EU5 + JP

BeOne is the only company with potentially best-in-class assets across three foundational CLL MOAs

Key CLL mechanisms

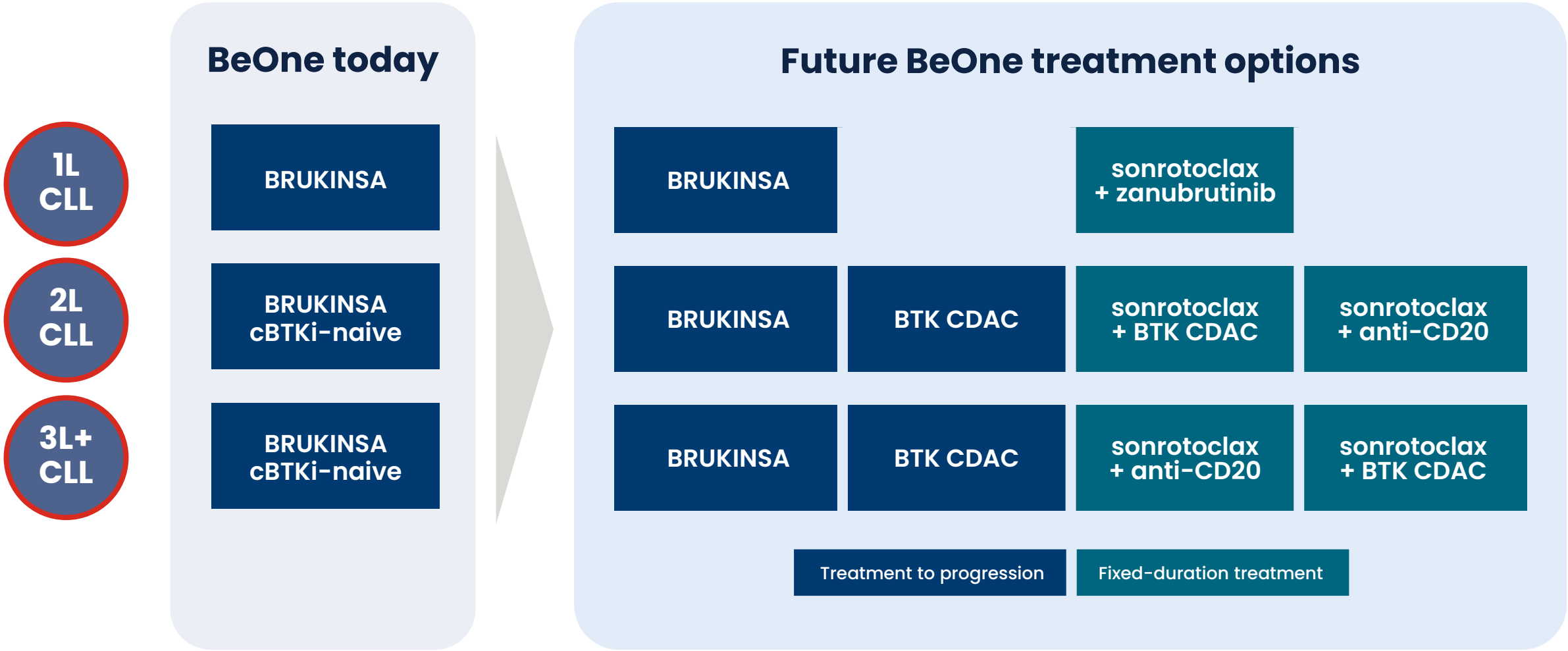
	BTKi	BCL2i	BTK degrader
BeOne	✓	✓	✓
abbvie	◐	◐	●
AstraZeneca	●		
Lilly	●		

✓ Wholly-owned best-in-class/potentially best-in-class medicine

● Wholly-owned medicine

◐ Partnered medicine

BeOne is well-positioned to address CLL unmet need with our wholly-owned portfolio



BRUKINSA

BRUKINSA continues to drive value for patients globally

Brukinsa® zanubrutinib

1. **U.S. revenue leader**
2. The **only** BTKi to show **PFS superiority** over ibrutinib in head-to-head Phase 3 CLL trial
3. Launched in Japan and **now approved in 75 markets**
4. **11 new or expanded reimbursements,** including in Japan, EU, and Brazil
5. **200K+ patients** treated globally

Recent publications
Support best-in-class profile and unmet needs

"A network meta-analysis of BTKis found **zanubrutinib to be the most efficacious treatment** for patients with high-risk R/R CLL.**"**[1]

— blood advances

"These analyses further support the benefits of zanubrutinib as a first-line **treatment for patients with CLL regardless of del(17p) or IGHV status.**"[2]

— ICML

"Based on a hypothetical scenario of a clinical practice of 100 patients **treated with zanubrutinib instead of ibrutinib**, the model estimated that approximately **13 patients would avoid disease progression** or death over 24 months.**"**[3]

"Overall, this study's results suggest that **zanubrutinib is a clinically effective and cost-saving alternative** to ibrutinib for the treatment of patients with R/R CLL.**"**[3]

— JMCP

"Zanubrutinib demonstrated a more favorable safety profile than ibrutinib, with fewer severe adverse events.**"**[4]

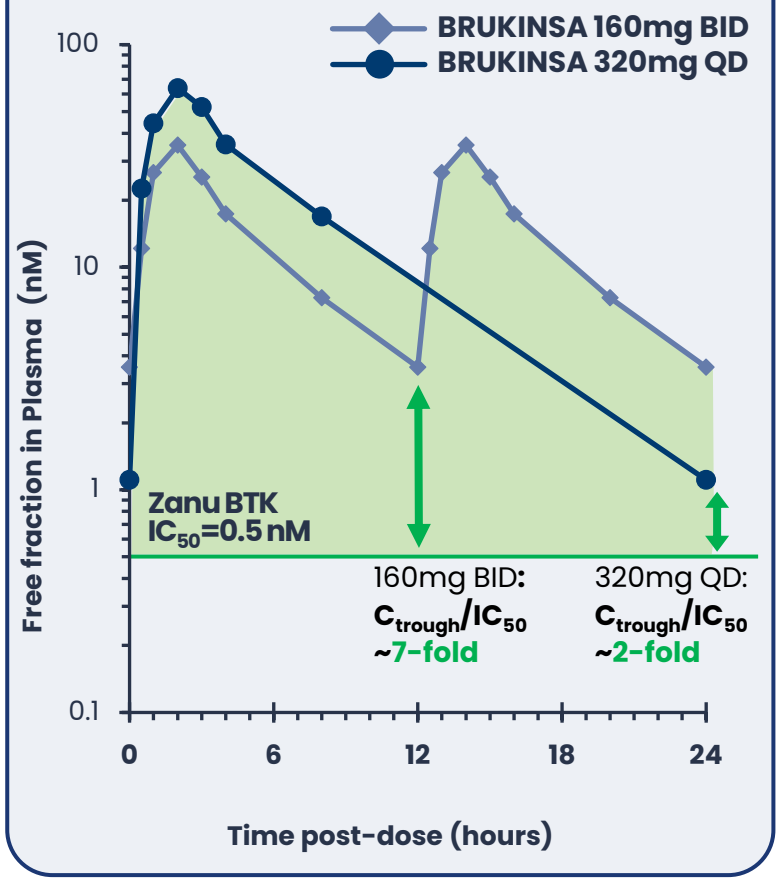
— Hematological ONCOLOGY

[1] Shadman M, Brown JR, et al. Comparative efficacy of Bruton tyrosine kinase inhibitors in the treatment of relapsed/refractory chronic lymphocytic leukemia: A network meta-analysis. Blood Adv. 2025: 2024014523
[2] Munir T et al., Efficacy of continuous zanubrutinib vs fixed-duration venetoclax in combination with obinutuzumab in treatment-naïve chronic lymphocytic leukemia: a matching-adjusted indirect comparison. ICML, 2025
[3] Chanan-Khan A, Hanna K, Xue M, et al. Number needed to treat and associated cost analysis of zanubrutinib vs ibrutinib in chronic lymphocytic leukemia. J Manag Care Spec Pharm. 2025; 31(5): 482-490
[4] Fan F, Liu X, Su Z, et al. Comparative safety of ibrutinib versus zanubrutinib in patients with Chronic Lymphocytic Leukemia: A Prospective Cohort Study. Hematol Oncol. 2025; 43(2): e70041

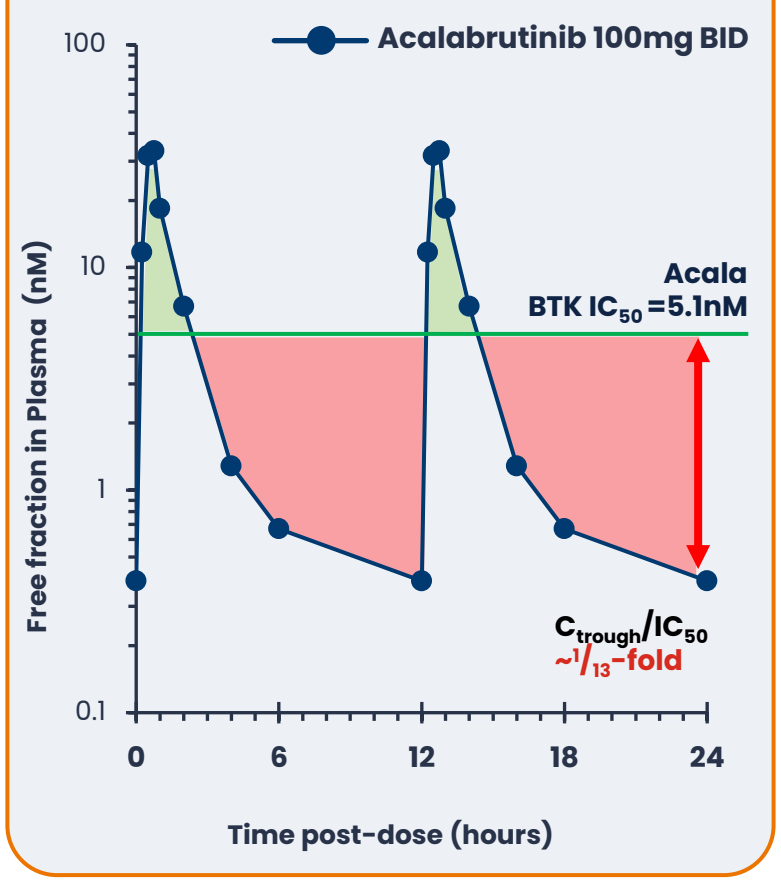
BRUKINSA designed from inception to be best-in-class

Hypothesis: complete and sustained BTK inhibition would result in best-in-class profile

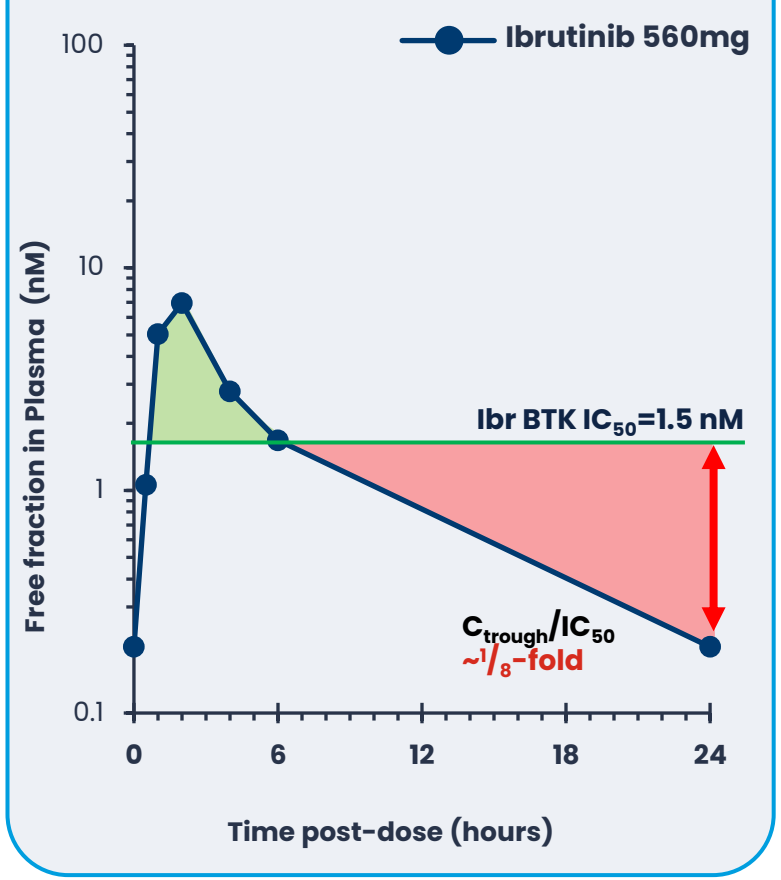
BRUKINSA PK vs. IC_{50}[1]



Acalabrutinib PK vs. IC_{50}[2]



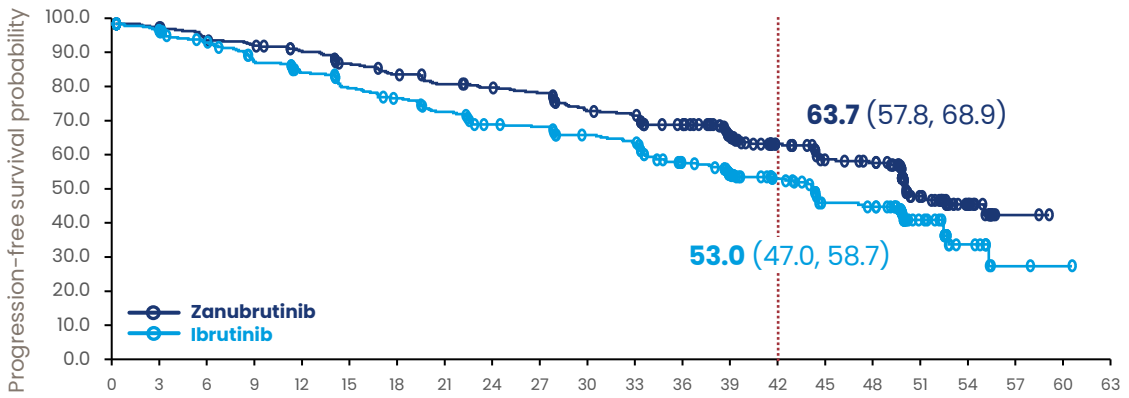
Ibrutinib PK vs. IC_{50}[3]



[1] Health Canada Product Monograph
[2] Byrd et al., NEJM, 2015; Zhou et al., Pharmacometrics Syst. Pharmacol. (2019) 8, 489–499
[3] Advani, et al., JCO 2013.; NDA Clinical Pharmacology Review {NDA 205552, ibrutinib}
The clinical significance of complete inhibition has not been established. In the absence of head-to-head data, definitive conclusions regarding comparative safety and efficacy cannot be drawn

BRUKINSA is the only BTKi to demonstrate PFS superiority over ibrutinib in head-to-head Phase 3 R/R CLL trial

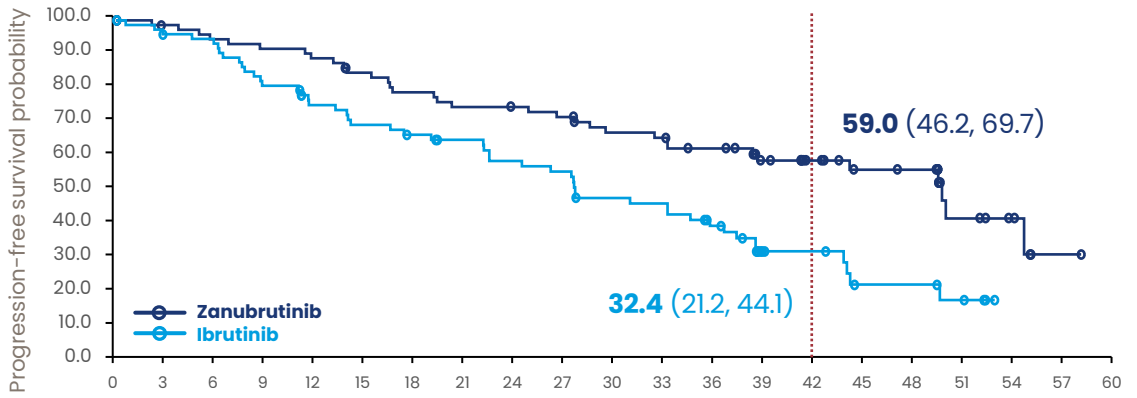
PFS superiority in all-comer population[1]



Months from randomization	0	3	6	9	12	15	18	21	24	27	30	33	36	39	42	45	48	51	54	57	60	63
No. subjects at risk **Zanubrutinib**	327	313	301	295	286	268	257	247	241	236	214	208	189	151	128	108	103	43	19	2	0	0
Ibrutinib	325	304	292	271	256	238	227	213	197	194	182	173	147	116	101	76	73	30	10	2	1	0

	Zanubrutinib	Ibrutinib
# of events (%)	134 (41.0%)	160 (49.2%)
HR (95% CI):	0.66 (0.52, 0.84)	
nominal *p*-value	0.0005	

PFS in del(17p)/TP53 subset consistent with ITT patient population[1]



Months from randomization	0	3	6	9	12	15	18	21	24	27	30	33	36	39	42	45	48	51	54	57	60
No. subjects at risk **Zanubrutinib**	75	71	68	66	64	59	55	52	51	49	44	43	39	31	25	20	19	8	5	1	0
Ibrutinib	75	70	68	59	52	48	45	42	38	36	30	29	23	13	11	6	6	4	0	0	0

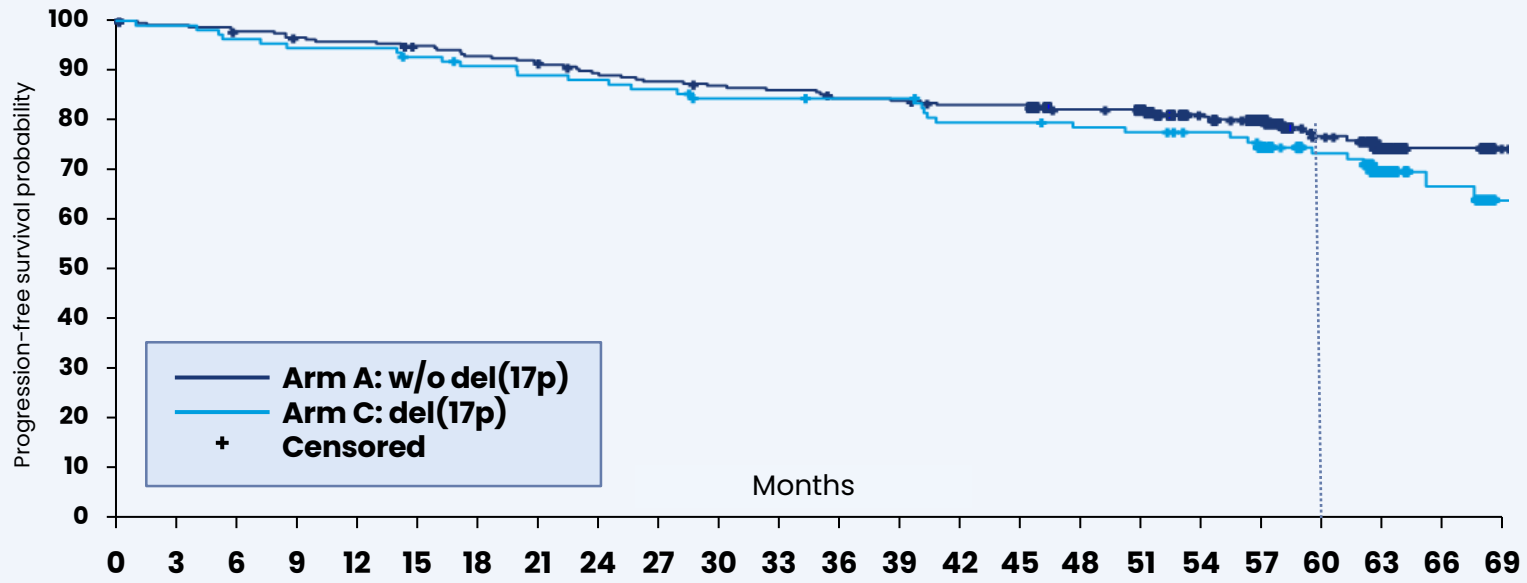
	Zanubrutinib	Ibrutinib
# of events (%)	36 (48.0%)	51 (68.0%)
HR (95% CI):	0.48 (0.31, 0.75)	
nominal *p*-value	0.0019	

[1] Brown et al., Blood, 2024; COVID adjusted

BRUKINSA demonstrated impressive PFS in SEQUOIA study in treatment naïve CLL/SLL patients regardless of risk status

- Presence of del(17p) is associated with ~2-fold greater risk of death due to CLL[3]
- SEQUOIA Arm C is largest prospective cohort of uniformly treated patients with del(17p) TN CLL/SLL
- **60-month PFS** with zanubrutinib was **72.2%** in TN CLL/SLL patients **with del(17p)**, similar to **75.8%** observed in patients **without del(17p)**

PFS of del(17p) in Arm C[1] vs. PFS of w/o del(17p) in Arm A[2]

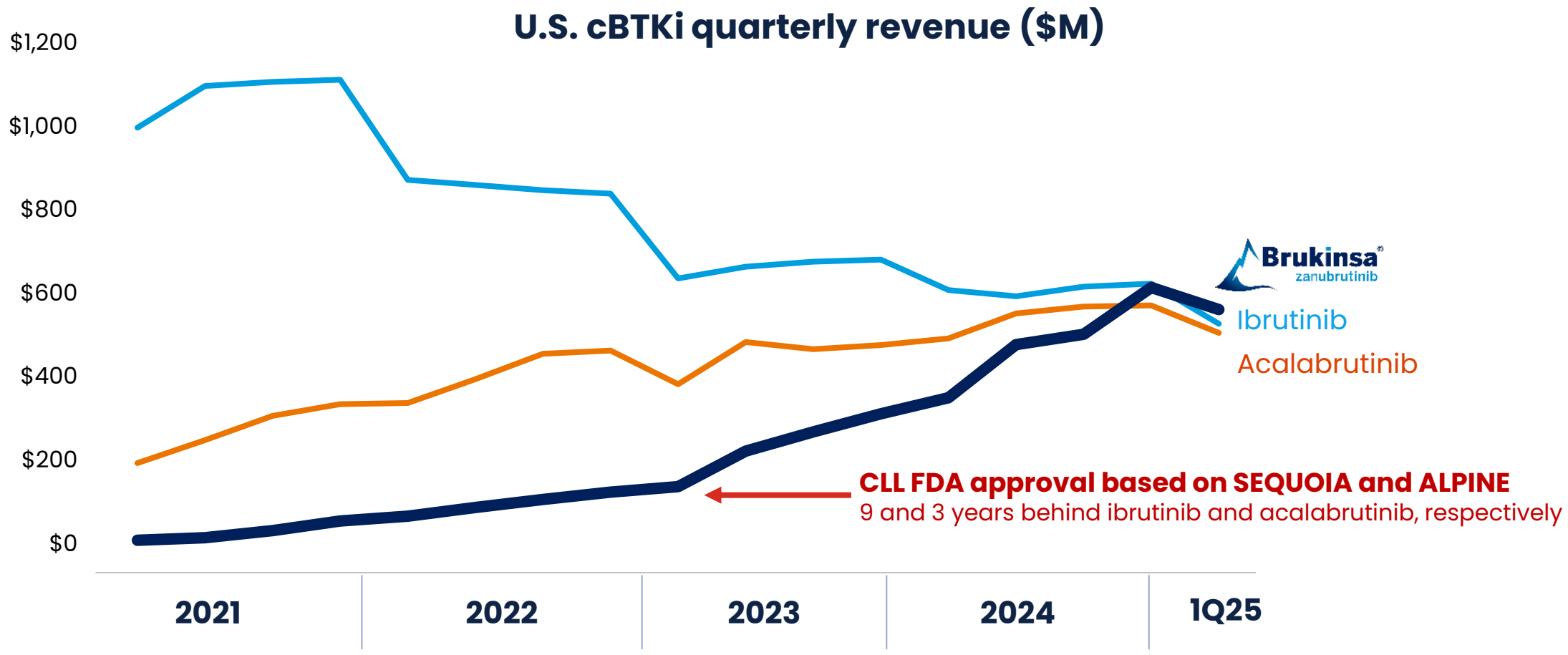


Number of subjects at risk:

Arm A	241	238	234	230	228	224	219	214	208	205	201	200	195	192	190	183	178	164	153	89	81	19	19	2
Arm C	110	109	106	104	104	101	98	96	94	93	89	89	88	86	82	81	80	78	76	66	62	24	22	1

[1] Tam et al., ASCO, 2025
[2] Shadman et al., JCO, 2024
[3] Wang et al. Blood Cancer J. 2021
CLL, chronic lymphocytic leukemia; OS, overall survival; PFS, progression-free survival; SLL, small lymphocytic lymphoma; TN, treatment-naïve

Designing the right molecule and conducting the right experiment ultimately leads to market leadership despite later entry



Sonrotoclax, combinations, and fixed duration

Ideal fixed-duration treatment regimen must meet the following criteria

Deep response

- Measured by MRD at the time of discontinuation – need to be comfortable when stopping therapy

Sustained PFS

- Comparable to BTKi continuous therapy

Safety

- Adding only minimal AEs over BTKi monotherapy continuous
- No TLS, and no death/OS detriment

Convenience

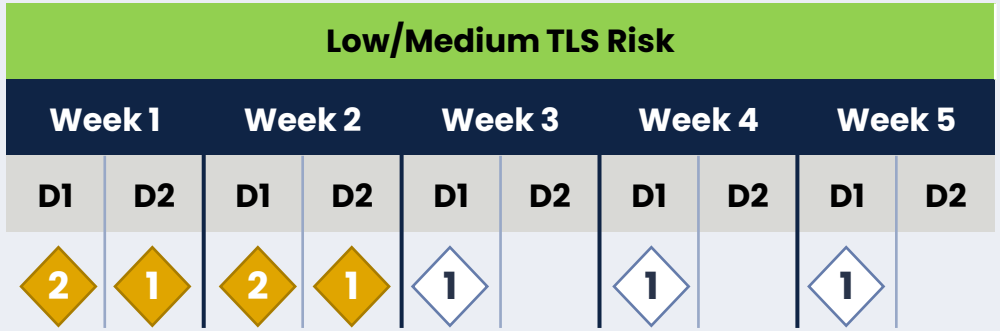
- No required hospitalizations
- Fewer clinic visits and less monitoring during ramp-up
- Usable in the community oncology centers

Current venetoclax-based fixed-duration treatments for CLL are suboptimal

HCPs often cite issues with venetoclax – there is clear need for a next-generation BCL2i

Venetoclax ramp-up and monitoring[1]

5-7 clinic visits



8 clinic visits including two overnight

- Hospitalization required
- Hospitalization considered
- 4 Tests pre-dose, 4, 8, 12h
- 2 Tests pre-dose, 6-8h (ven), 4-6h (sonro)
- 1 1 test pre-dose

"Ven's ramp-up is a little annoying, but it is only required because it is an effective Tx"

- U.S. Community HCP[2]

"Monitoring [venetoclax] can be very annoying. You want a treatment that simplifies staff responsibilities"

– U.S. Community HCP[2]

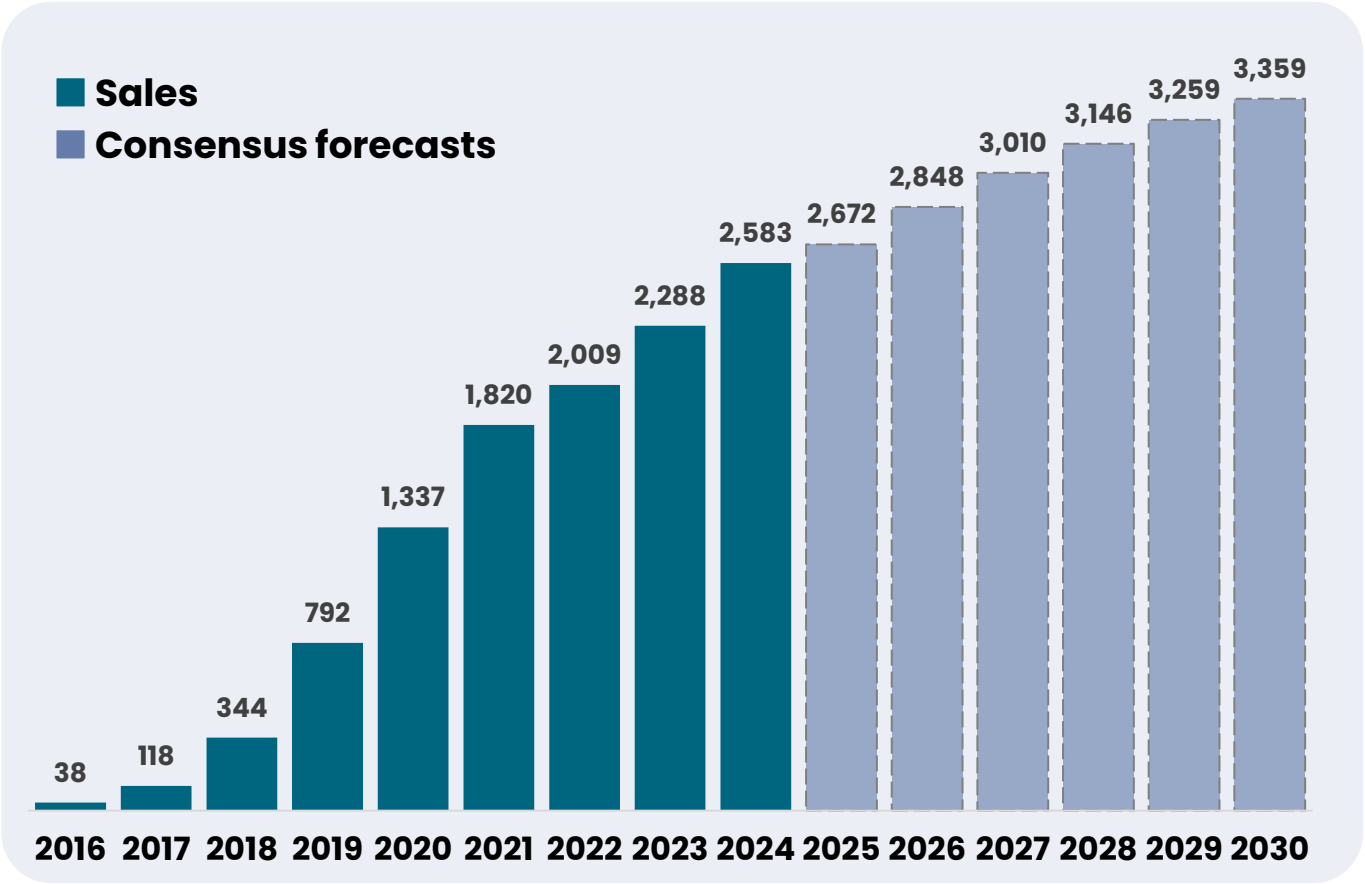
"Longer post-administration monitoring means greater likelihood of hospitalization"

- U.S. Community HCP[2]

[1] Venetoclax U.S. package insert
[2] Primary Market Research, NOV2023

Despite its substantial use limitations, venetoclax is a blockbuster product

Global venetoclax revenue ($M)[1]



- Venetoclax-based regimens represent ~25% of new patient starts in CLL, yet the vast majority of use lies in the academic setting
- We believe there is substantial opportunity for a next-gen BCL2i to unlock the full potential of this class

[1] EvaluatePharma, accessed 27MAY2025

Sonrotoclax is designed to be best-in-class on efficacy, with favorable safety and convenient handling

	Sonrotoclax	Venetoclax	Implication in clinic
Potency (IC_{50})	**0.014 nM**	**0.20 nM**	**14-fold more potent**, deeper target inhibition to eliminate the most difficult to treat tumor cells
Selectivity (vs. BCLxL)	**2000X**	**325X**	**6-fold improved selectivity** for potentially better tolerability
$T_{1/2}$ in clinic	**~5 h**	**26 h**	**Aiming for only one clinic visit for sonro ramp-up for most patients;** no accumulation leading to ease of TLS monitoring during ramp-up

The clinical significance of pre-clinical data has not been established. There are no head-to-head data comparing sonrotoclax and venetoclax

Sonrotoclax

Speaker introduction: Dr. Jacob Soumerai, key opinion leader in CLL and lymphomas

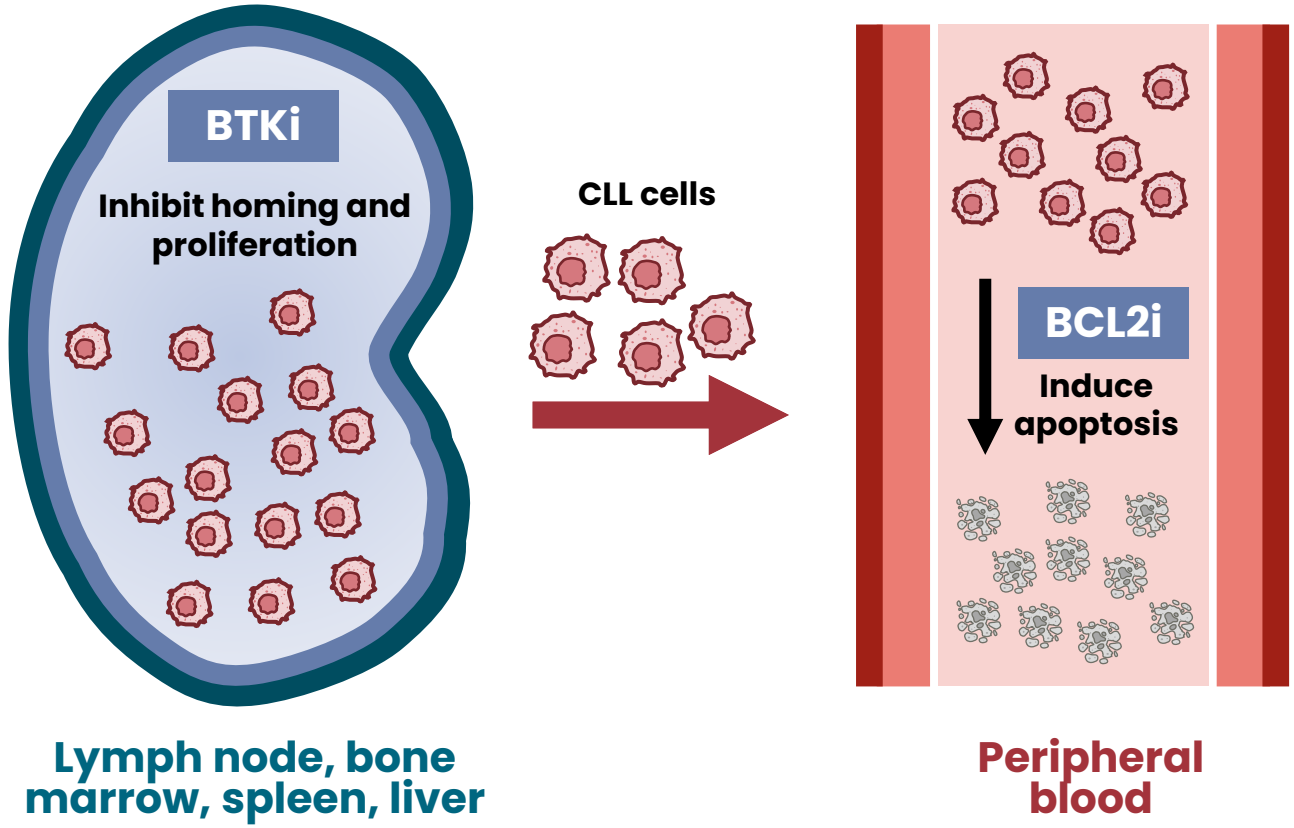
Jacob D. Soumerai, MD

Assistant Professor of Medicine, Harvard Medical School
Hematologist/Oncologist, MGH Cancer Center

- Hematologist/oncologist and clinical investigator in lymphoma and CLL at the Massachusetts General Hospital Cancer Center
- Dr. Soumerai's clinical trial and translational research efforts focus on the development of innovative therapeutic combinations in CLL and B-cell lymphomas
- A major theme running through his research relate to identifying novel biomarkers with potential to guide personalized therapy approaches
- Recipient of multiple awards, like NIH K08 Award, Lymphoma Research Foundation Career Development Award, Kraft Translational Research Award and others
- 50+ publications as first author, with recent publications in high-impact journals including Blood, JCO, Lancet Oncology, Lancet Haematology

MOA of the synergistic effect of BTK and BCL2 inhibitors[1,2,3]

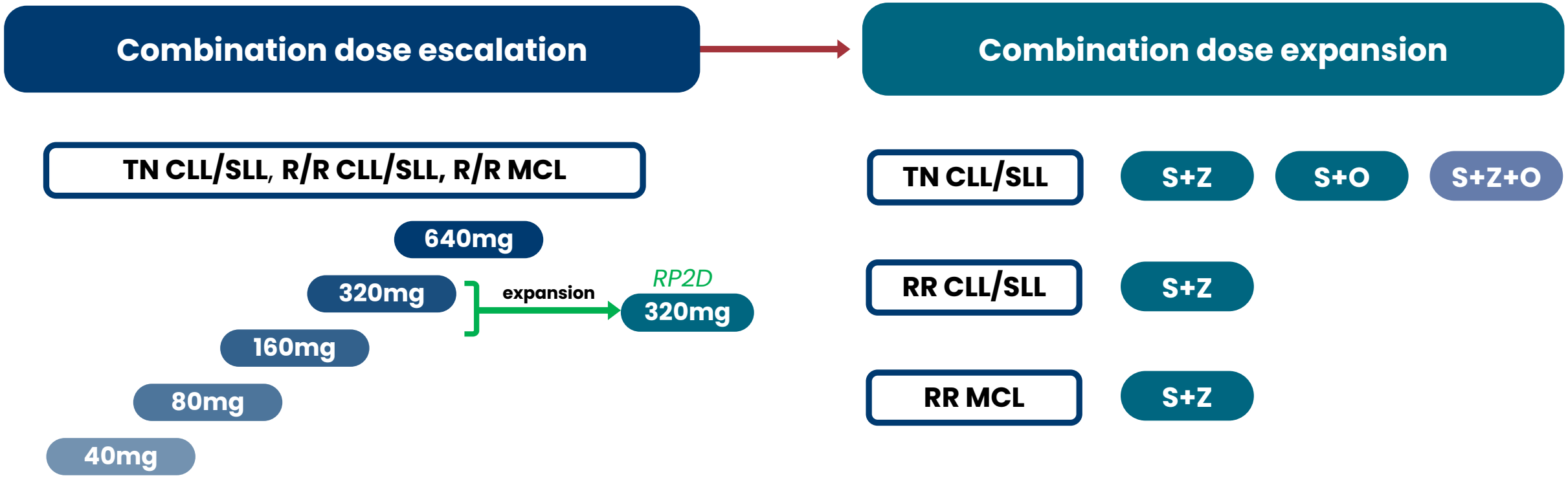
- Besides anti-proliferation, BTKi also prevents CLL cells' homing in lymphoid tissues/organs and promotes cell migration to peripheral blood
- Then, BCL2i can effectively kill CLL cells in peripheral blood
- Distinct and complementary MoA of BTKi and BCL2i brings synergistic effects



[1] Zygmunciak P, et al. Int J Mol Sci. (2025)
[2] Zhang J, et al. Biomark Res. (2022)
[3] Cervantes-Gomez F, et al. Clin Cancer Res. (2016)

BGB-11417-101: Phase 1/2, open-label, sonrotoclax dose-escalation/expansion study in B-cell malignancies

- BGB-11417-101 study is a Ph 1/2 study that evaluated sonrotoclax monotherapy or in combinations with zanubrutinib ± sonrotoclax ± obinutuzumab in multiple B-cell indications, at various dose levels



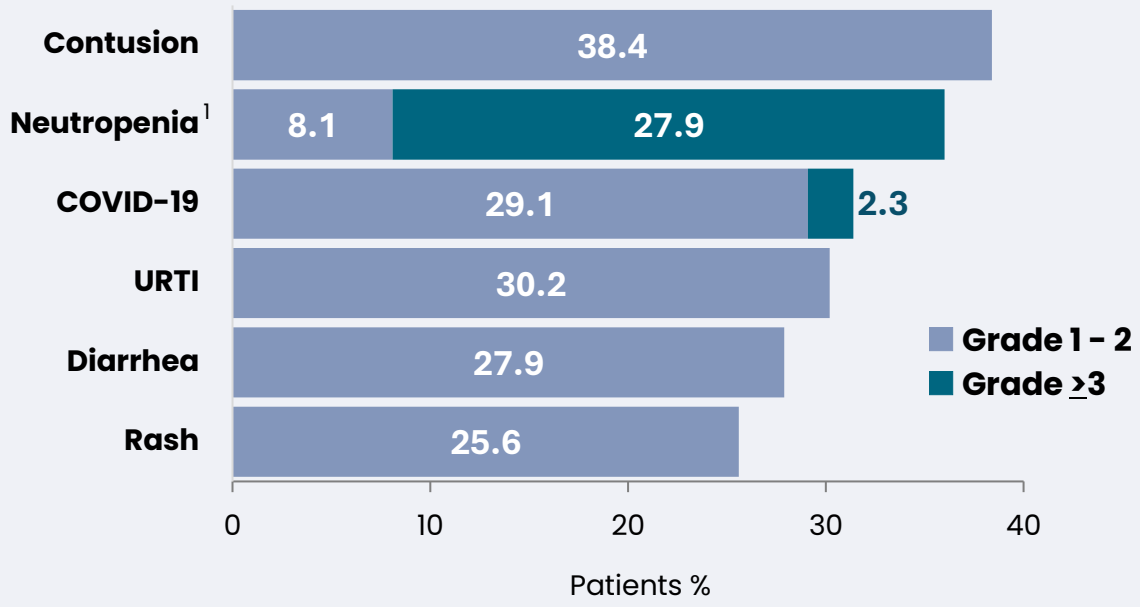
CLL/SLL, chronic lymphocytic leukemia/small lymphocytic lymphoma; MCL, mantle cell lymphoma; MTD, maximum tolerated dose; R/R, relapsed/refractory; RP2D, recommended phase 2 dose; TN, treatment naive

TEAEs observed with sonrotoclax + zanubrutinib in TN CLL/SLL were mostly low grade and transient

TN CLL/SLL

- No TLS (clinical or laboratory)
- Neutropenia was transient and did not lead to higher rates of grade ≥3 infections
- No AE of febrile neutropenia reported
- No new safety signals observed with longer follow-up
- No TEAE leading to death

sonrotoclax 320mg + zanubrutinib (n=86)
Median follow-up (range): 25.53 mo (3.1-36.0)



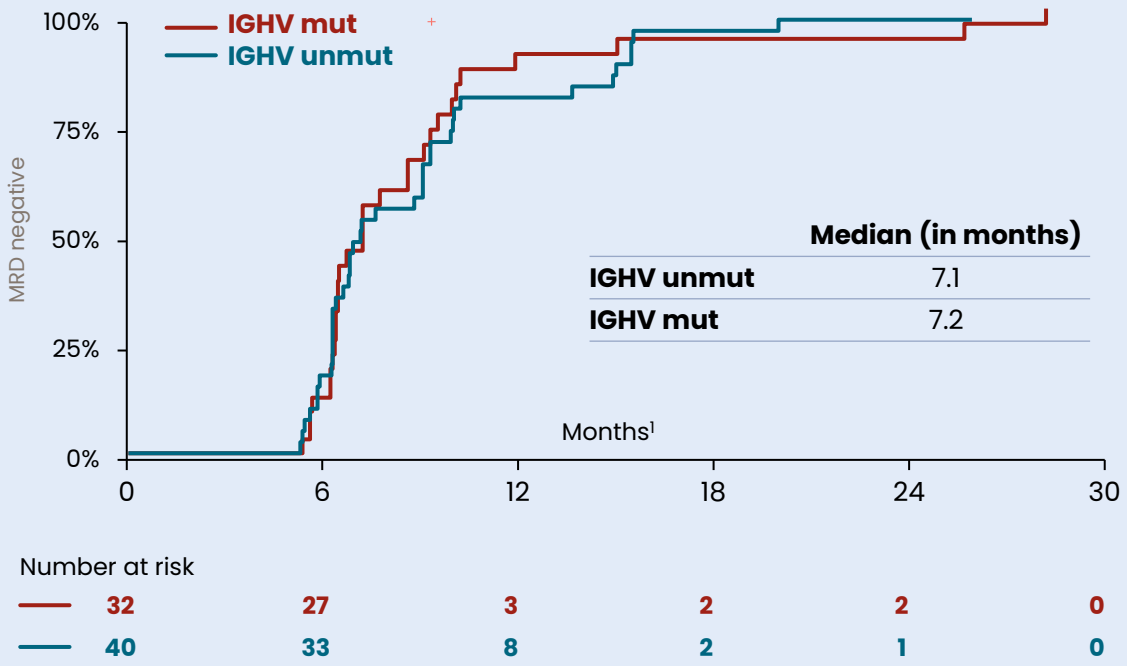
TEAEs in ≥25% of all patients

[1] Includes the combined preferred terms *neutrophil count decreased* and *neutropenia*
DCO: 01MAR2025

Sonrotoclax + zanubrutinib achieved fast and deep response in TN CLL/SLL regardless of risk status

TN CLL/SLL

Time to blood uMRD sonro(320mg)+zanu

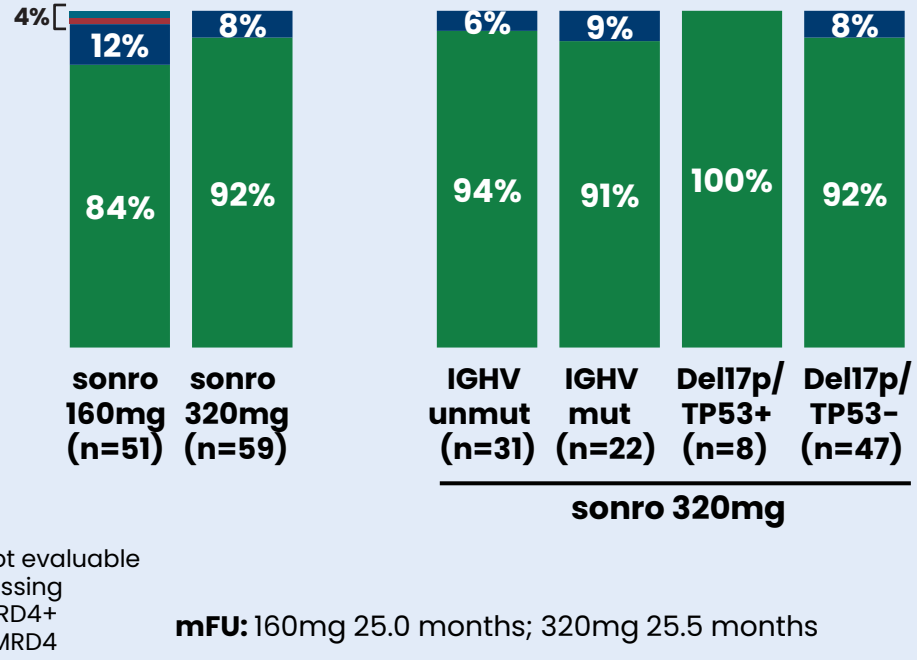


	Median (in months)
IGHV unmut	7.1
IGHV mut	7.2

Number at risk

IGHV mut	32	27	3	2	2	0
IGHV unmut	40	33	8	2	1	0

uMRD by week 48[2,3]



mFU: 160mg 25.0 months; 320mg 25.5 months

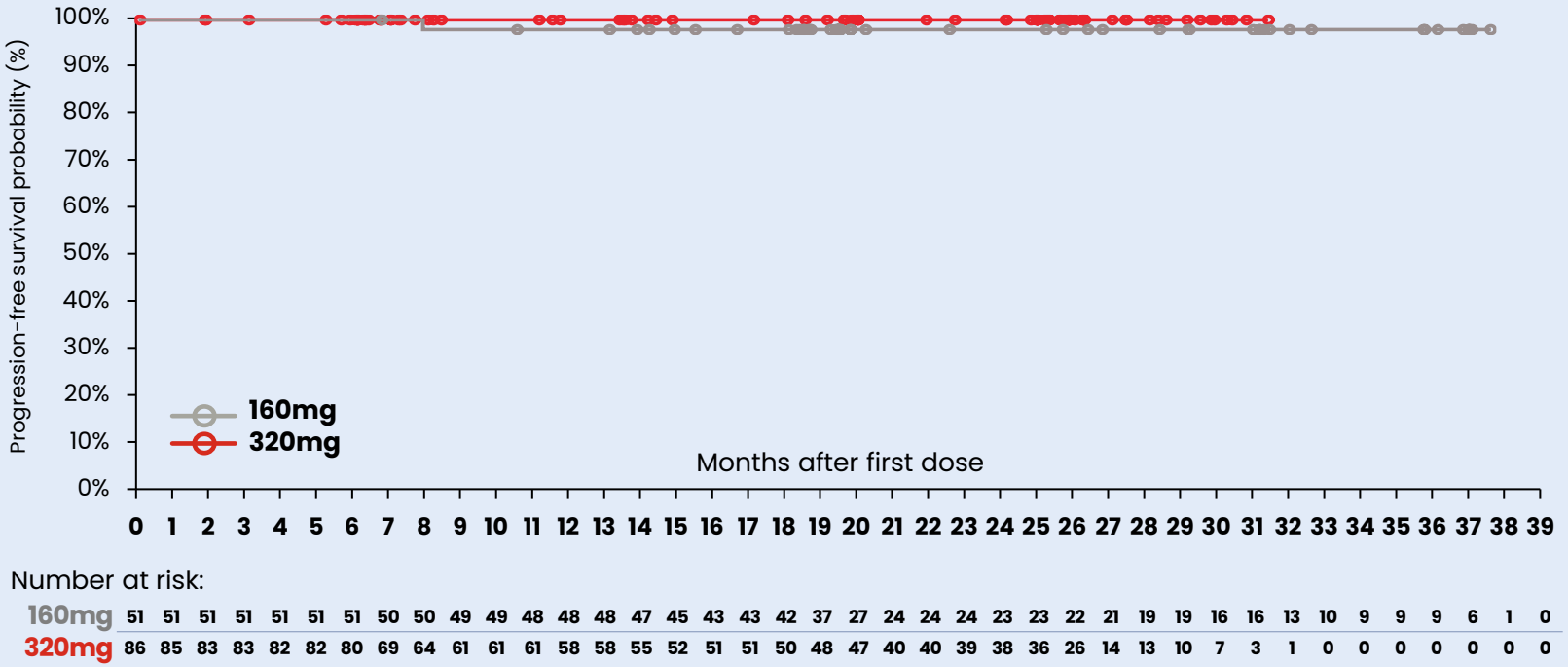
[1] From day 1 of zanubrutinib monotherapy treatment
[2] As measured by ERIC flow cytometry panel uMRD4 is defined as less than 1 CLL cell per 10,000 leukocytes ($<10^{-4}$); MRD is best reported within a 2-week window following the week 48 assessment
[3] Number of weeks at target dose, following zanubrutinib monotherapy and sonrotoclax ramp-up to target dose, different from [1]
DCO: 01MAR2025

Sonrotoclax + zanubrutinib high uMRD rate translates into impressive PFS in TN CLL/SLL

TN CLL/SLL

- No PFS events at 320mg dose and only one progression at 160mg dose
- Patients (n=35) who reached week 96 and elected to stop therapy[1] all in remission with a time off median of three months (range 1-12 months)

No PFS events observed at 320mg dose



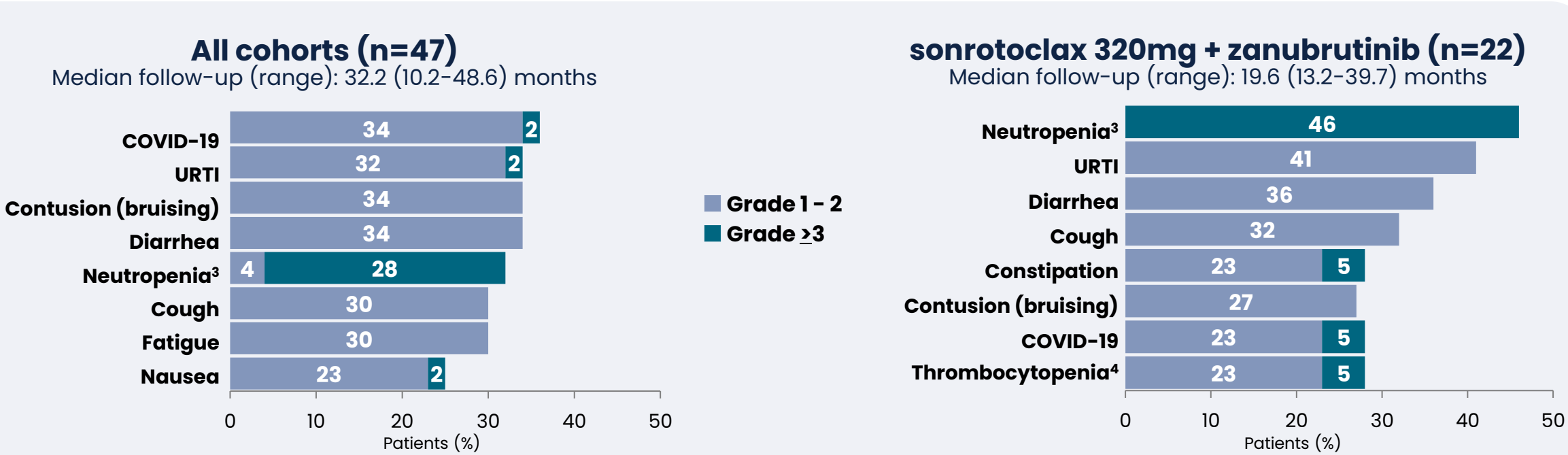
[1] Patients had the option to electively discontinue therapy after 96 weeks of combination

DCO: 01MAR2025

TEAEs observed with sonrotoclax + zanubrutinib in R/R CLL/SLL were mostly low grade and transient[1]

R/R CLL/SLL

- No clinical or laboratory TLS and no febrile neutropenia observed
- No dose reductions occurred due to diarrhea
- No TEAE leading to death



TEAEs in ≥25% of all patients and those treated at sonrotoclax RP2D of 320mg[2]

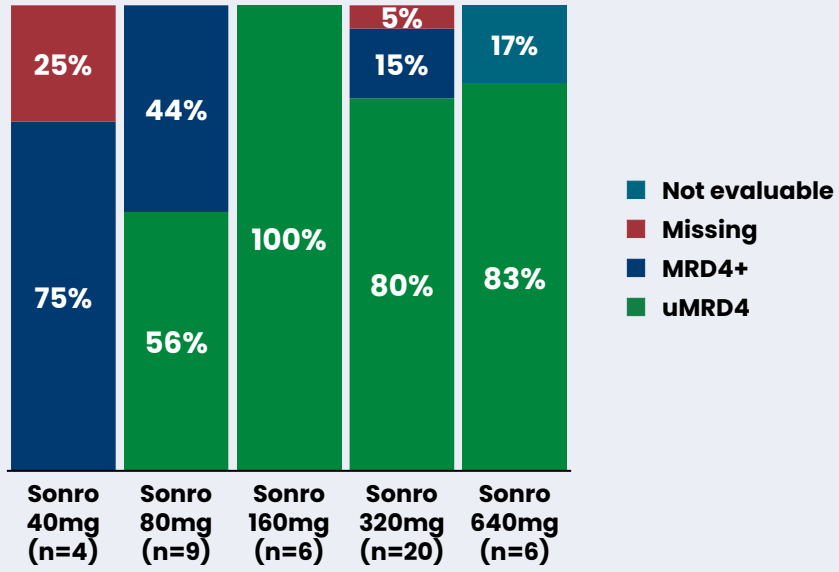
[1] Cheah, EHA, 2025
[2] Grade is listed as worst grade experienced by patient on any drug
[3] Neutropenia combines preferred terms neutrophil count decreased and neutropenia
[4] Thrombocytopenia combines preferred terms platelet count decreased and thrombocytopenia
RP2D, recommended phase 2 dose; TEAE, treatment-emergent adverse event; TLS, tumor lysis syndrome; URTI, upper respiratory tract infection

Sonrotoclax + zanubrutinib demonstrated high uMRD rate with impressive PFS in R/R CLL[1]

R/R CLL/SLL

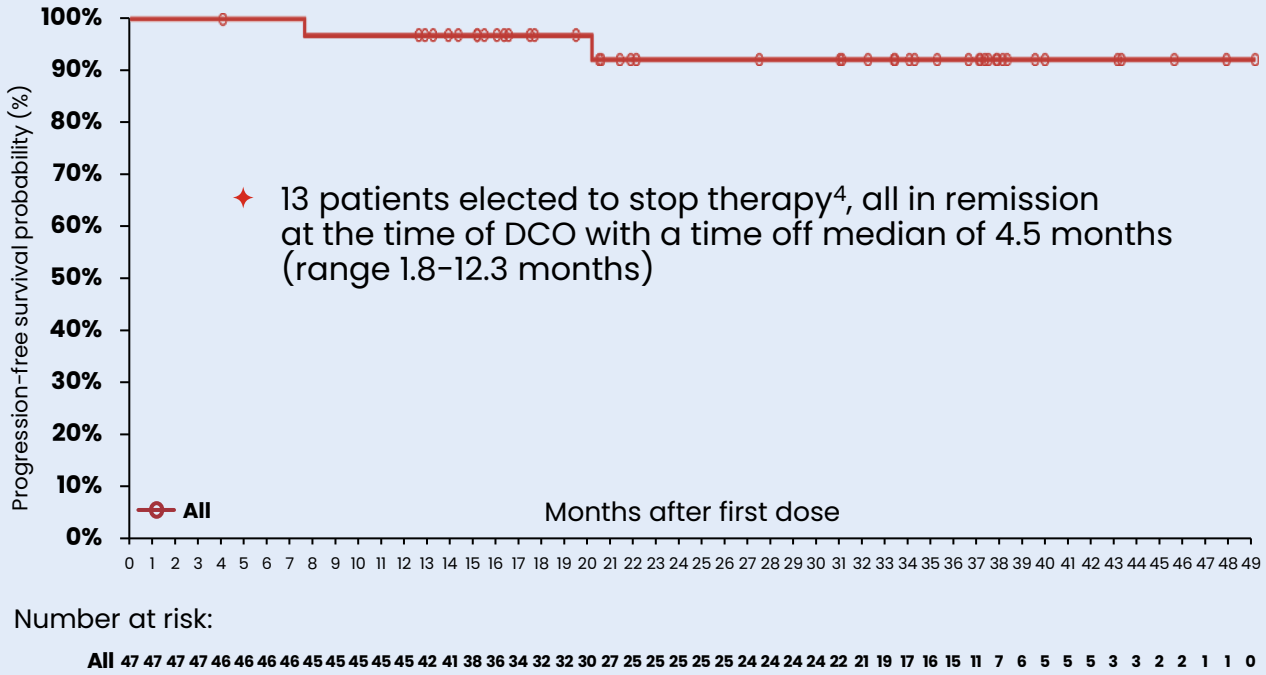
- SZ combo demonstrated high uMRD rate, with 80% uMRD at the RP2D (320mg)
- To date, only two PFS events occurred on the study across all dose levels, one at 40mg dose and one at 320mg dose

uMRD by week 48[2,3]



mFU:
32.2 months (combined all dose levels) 320mg – 19.6 months

Only two PFS events across dose levels



- 13 patients elected to stop therapy[4], all in remission at the time of DCO with a time off median of 4.5 months (range 1.8-12.3 months)

Number at risk:

All 47 47 47 47 46 46 46 46 46 45 45 45 45 45 42 41 38 36 34 32 32 30 27 25 25 25 25 25 24 24 24 24 22 21 19 17 16 15 11 7 6 5 5 5 3 3 2 2 1 1 0

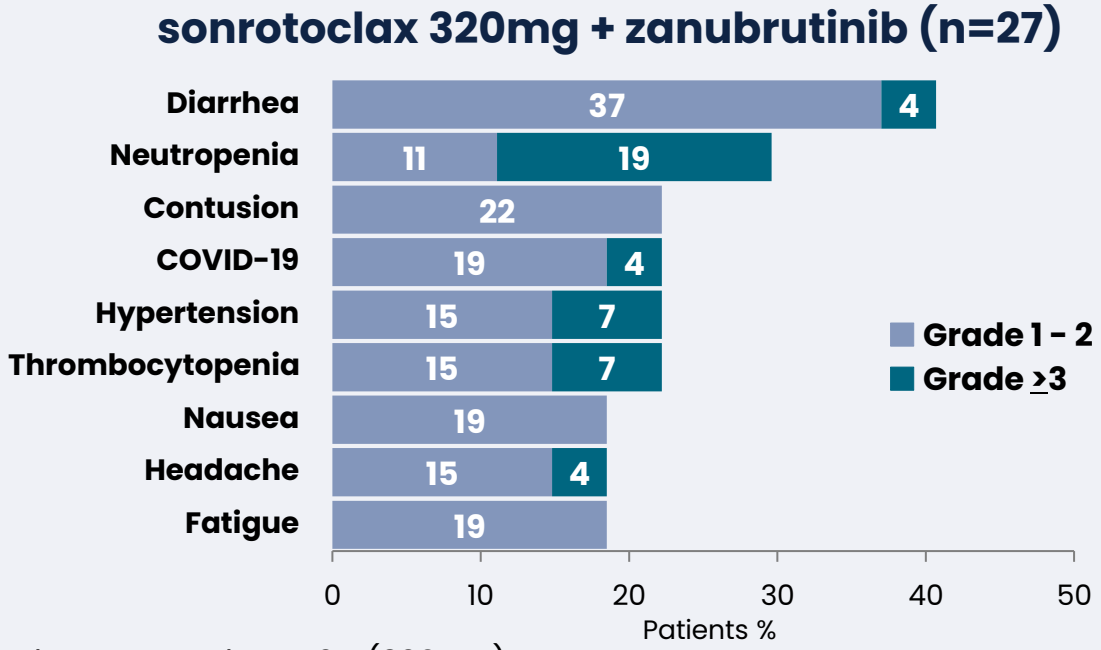
[1] Cheah, EHA, 2025
[2] As measured by ERIC flow cytometry panel uMRD4 is defined as less than 1 CLL cell per 10,000 leukocytes ($<10^{-4}$); MRD is best reported within a 2-week window following the week 48 assessment
[3] Number of weeks at target dose, following zanubrutinib monotherapy and sonrotoclax ramp-up to target dose
[4] Patients had the option to electively discontinue therapy after 96 weeks of combination

Sonrotoclax + zanubrutinib in R/R MCL was generally well tolerated and showed a safety profile similar to CLL[1]

R/R MCL

- Safety profile was generally similar across all doses tested, with no clinical or laboratory TLS observed and no cases of atrial fibrillation
- MTD was not reached with sonrotoclax up to 640mg; 320mg was chosen as RP2D combination with zanubrutinib in MCL

Patients, n (%)	Sonro 320mg + zanu (n=27)	All (N=51)
Any TEAEs	26 (96.3)	48 (94.1)
Grade ≥3	14 (51.9)	28 (54.9)
Serious TEAEs	7 (25.9)	15 (29.4)
Leading to death	1 (3.7)	3 (5.9)[2]
Leading to zanubrutinib discontinuation	4 (14.8)	8 (15.7)[3]
Treated with sonrotoclax, n (%)	24 (88.9)	46 (90.2)
Leading to death	0	1 (2.0)[4]
Leading to sonro dose reduction	0	0



TEAEs in ≥15% of patients and in those at the sonrotoclax RP2D (320mg)

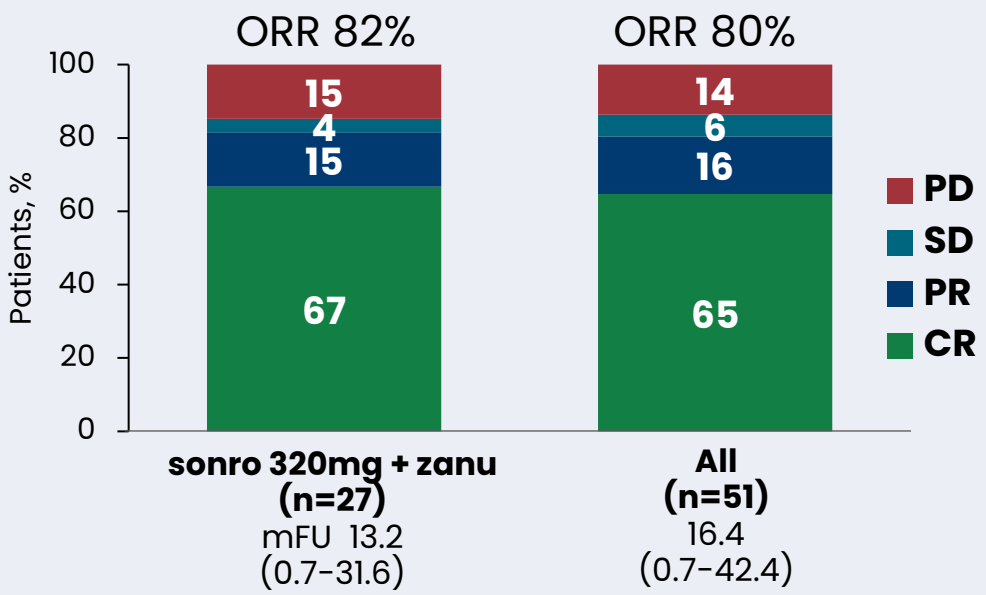
[1] Cordoba, EHA, 2025
[2] Pleural effusion (80mg; due to PD), pneumonia (160mg), abdominal sepsis (320mg)
[3] Lymph node pain (160mg, due to PD), diarrhea (320mg), MDS (160mg), abdominal sepsis (320mg), pneumonia (160mg), diarrhea (80mg), cryptococcal meningoencephalitis (320mg), abdominal pain (320mg)
[4] Pneumonia (160mg)
RP2D, recommended phase 2 dose; ; TEAE, treatment-emergent adverse event

Sonrotoclax + zanubrutinib demonstrated deep and durable responses in R/R MCL[1]

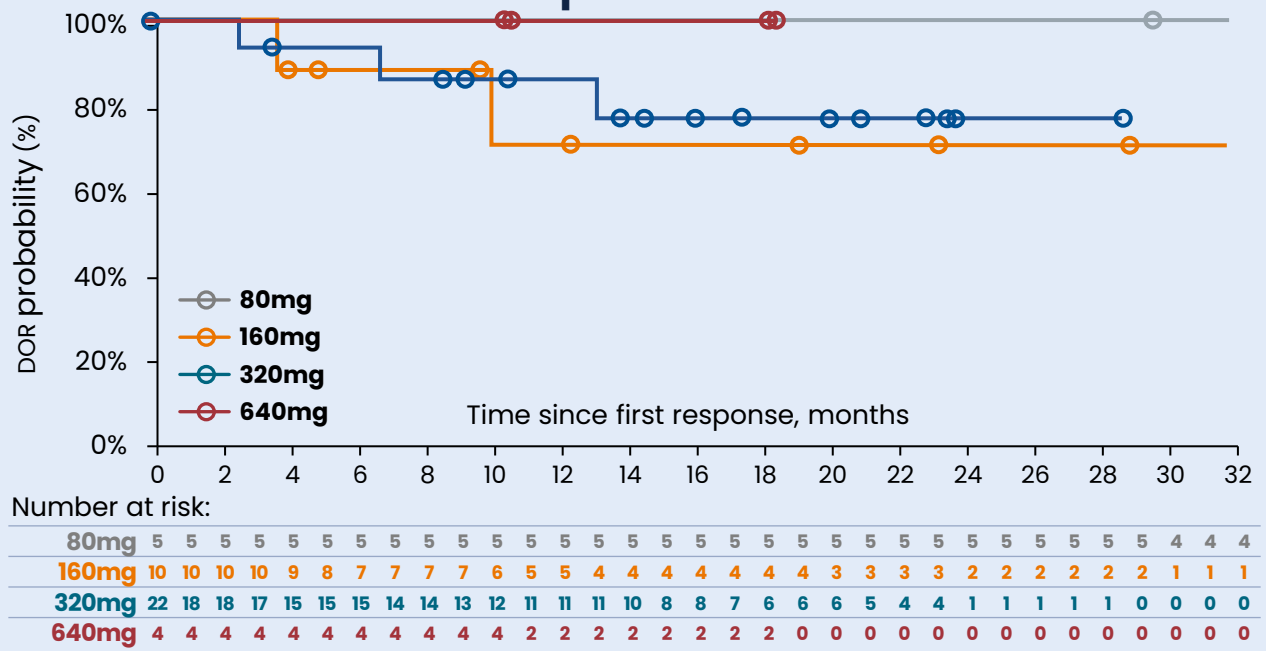
R/R MCL

- ORR[2] was 82% with CR rate of 67% in 320mg dose group
- Median DOR[3] in all patients was not reached; DOR rate at 24 months was 84.0% with median follow-up of 17.7 months
- 16 of 18 patients who achieved CR remain in CR at a median follow-up of 13 months

Best overall response rate



Median DOR[c] in all patients was not reached

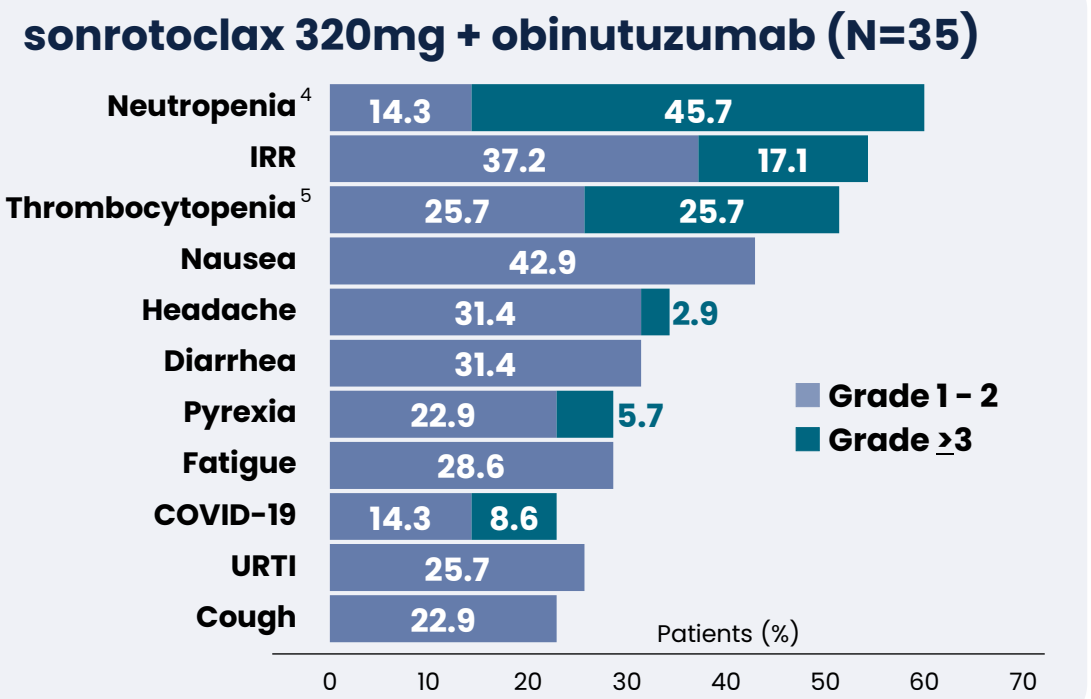


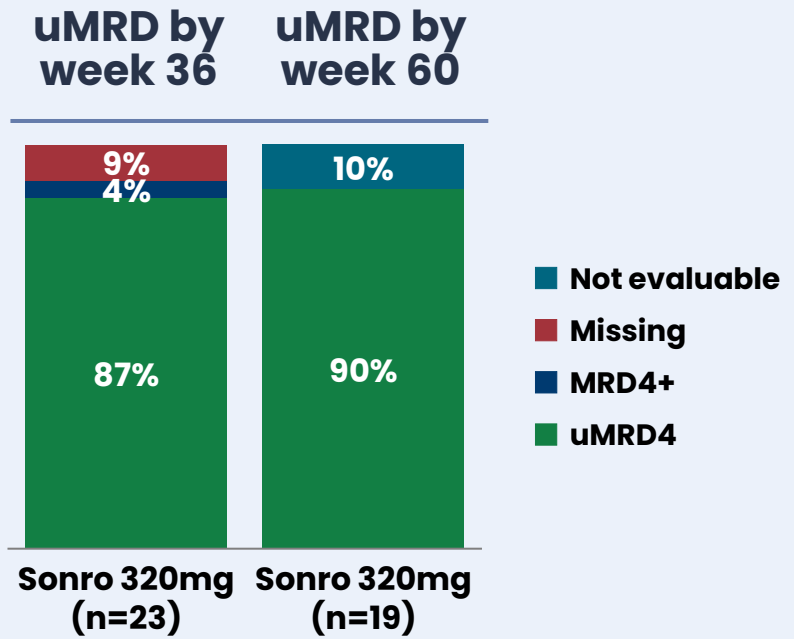
[1] Cordoba, EHA, 2025
[2] Responses were assessed per Lugano 2014 criteria and percentage of responding patients who had at least one post-baseline tumor assessment after dosing with sonrotoclax unless treatment was discontinued due to clinical progression or death prior to tumor assessment
[3] For all patients as treated (n=51)
BOR, best overall response; BTK, Bruton tyrosine kinase; CR, complete response; ORR, overall response rate; PD, progressive disease; PR, partial response; SD, stable disease

Sonrotoclax + obinutuzumab[1] shows promising efficacy in TN CLL/SLL

TN CLL/SLL

- Favorable safety profile with no case of TLS during sonrotoclax ramp up
- Quick and deep uMRD rate, 87% and 90% of evaluable patients achieved uMRD by week 36 and 60, respectively[2,3]
- Only two PFS events up to the DCO

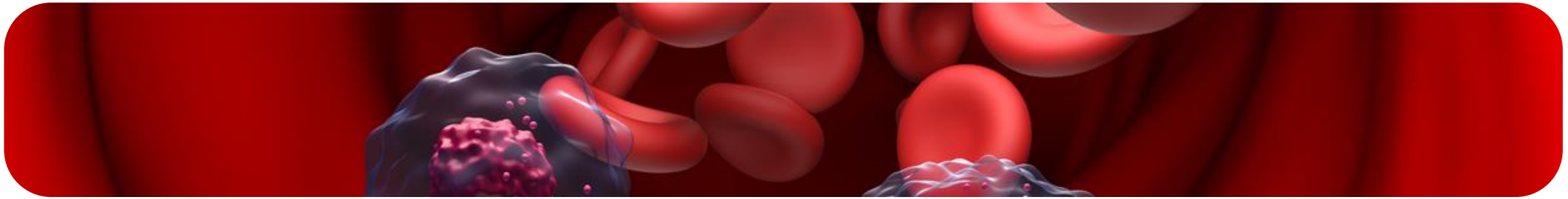




Median follow up – 13.5 months

[1] Combination treatment consist of 6 cycles of obinutuzumab (C1-C6;) and 14 cycles (c2-c15) or 26 cycles of sonrotoclax (C2-C27)for MRD4+ at C16. 1 cycle=4 weeks
[2] As measured by ERIC flow cytometry panel uMRD4 is defined as less than 1 CLL cell per 10,000 leukocytes (<10-4); MRD is best reported within a 2-week window following the week 48 assessment
[3] Number of weeks on therapy from first obinutuzumab dose
[4] Neutropenia combines preferred terms neutrophil count decreased and neutropenia
[5] Thrombocytopenia combines preferred terms platelet count decreased and thrombocytopenia
DCO: 01MAR2025
IRR, infusion related reaction; URTI, upper respiratory tract infection

All oral sonrotoclax + zanubrutinib has potential to be best in disease combination, including fixed-duration therapy



Sonrotoclax + zanubrutinib combination:

- Demonstrates **favorable safety profile** in patients with CLL/SLL and MCL, including **a more convenient ramp up with no cases of TLS in this combination**
- Demonstrates **impressive efficacy** in TN and R/R CLL/SLL and R/R MCL with responses continuing to deepen over time and **high uMRD rates observed in CLL regardless of risk factors**
- **Has the potential to become the new standard in CLL and MCL**

ZS has best-in-class potential vs. VO, IV, and AV on efficacy, safety, and convenience

		Precedent fixed-duration				
	ZS[1]	VO[2]	VO[3]	IV[4]	IV[5]	AV[7]
Population	all-comers	unfit	fit	unfit	all-comers	fit
uMRD	91%	76%	87%	55%	77%	34%
36-mo PFS	100% 24 mo. PFS	82%	88%	77%	90%[6]	77%
Grade ≥3 TEAEs	45%	80%	80%	75%	NR	54%
TEAE leading to death	0%	9%	4%	6.6%	NR	5.5%

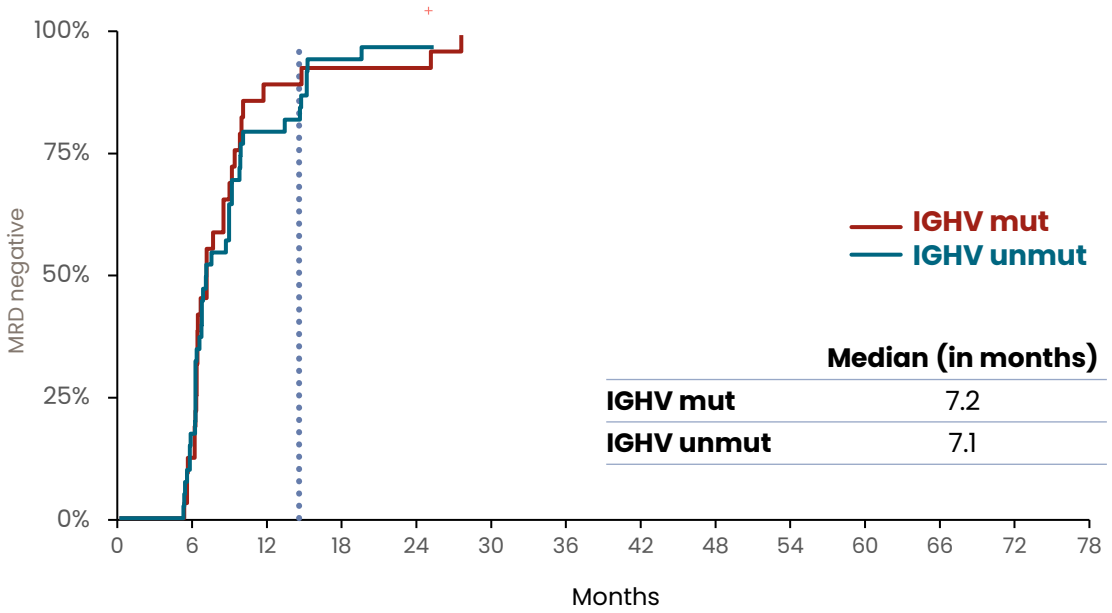
Not currently approved in U.S.

We are optimizing ramp-up scheduling for sonrotoclax and are optimistic that for vast majority of patients (>90%), **only one clinic visit** is required for sonrotoclax ramp-up after zanubrutinib lead-in

[1] CELESTIAL 101 - Soumerai et al., ASH, 2024; 320mg cohort
[2] CLL14 – Al-Sawaf, The Lancet, 2020
[3] CLL13 - Eichorst et al., NEJM, 2023
[4] GLOW - Niemann et al., Lancet, 2023, estimated PFS value for all patients
[5] CAPTIVATE fixed duration - Tam et al., Blood, 2022
[6] CAPTIVATE – Allan, CCR, 2023, estimated PFS value for all patients
[7] AMPLIFY - Brown et al., NEJM 2025
In the absence of head-to-head data, definitive conclusions regarding comparative safety and efficacy cannot be drawn; estimated PFS values; NR = not reported

ZS combination had much faster kinetics with time to blood uMRD than IV combo regardless of IGHV status

Time to blood uMRD ZS (CELESTIAL 101)[1]

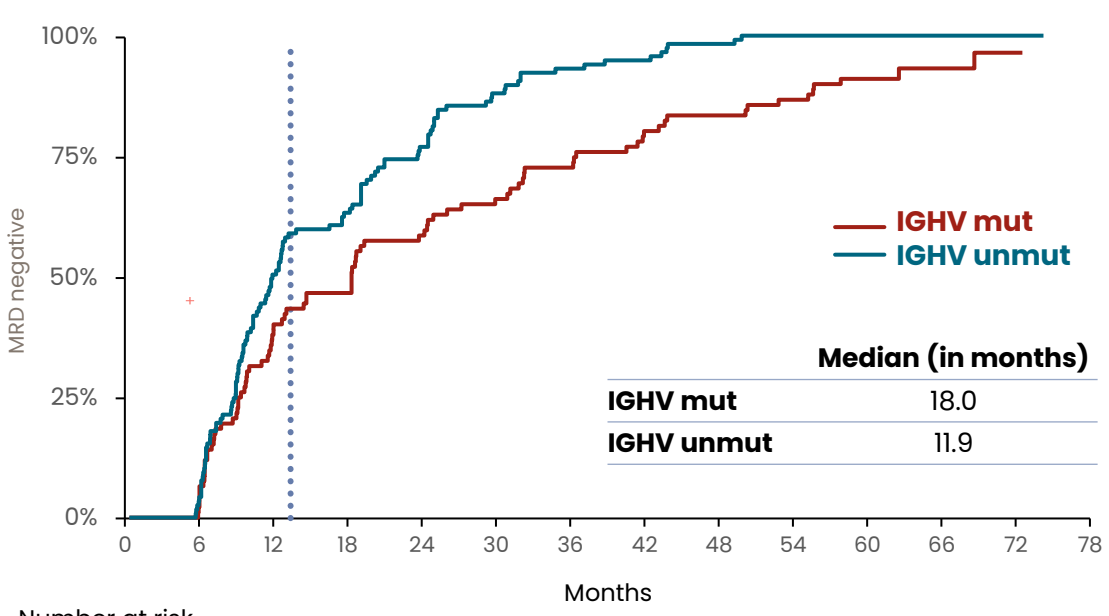


	Median (in months)
IGHV mut	7.2
IGHV unmut	7.1

Number at risk

IGHV mut	32	27	3	2	2	0
IGHV unmut	40	33	8	2	1	0

Time to blood uMRD IV (FLAIR study)[2]



	Median (in months)
IGHV mut	18.0
IGHV unmut	11.9

Number at risk

IGHV mut	97	86	60	48	40	36	27	23	20	17	13	11	0	0
IGHV unmut	123	111	61	47	29	18	14	11	8	6	6	6	6	0

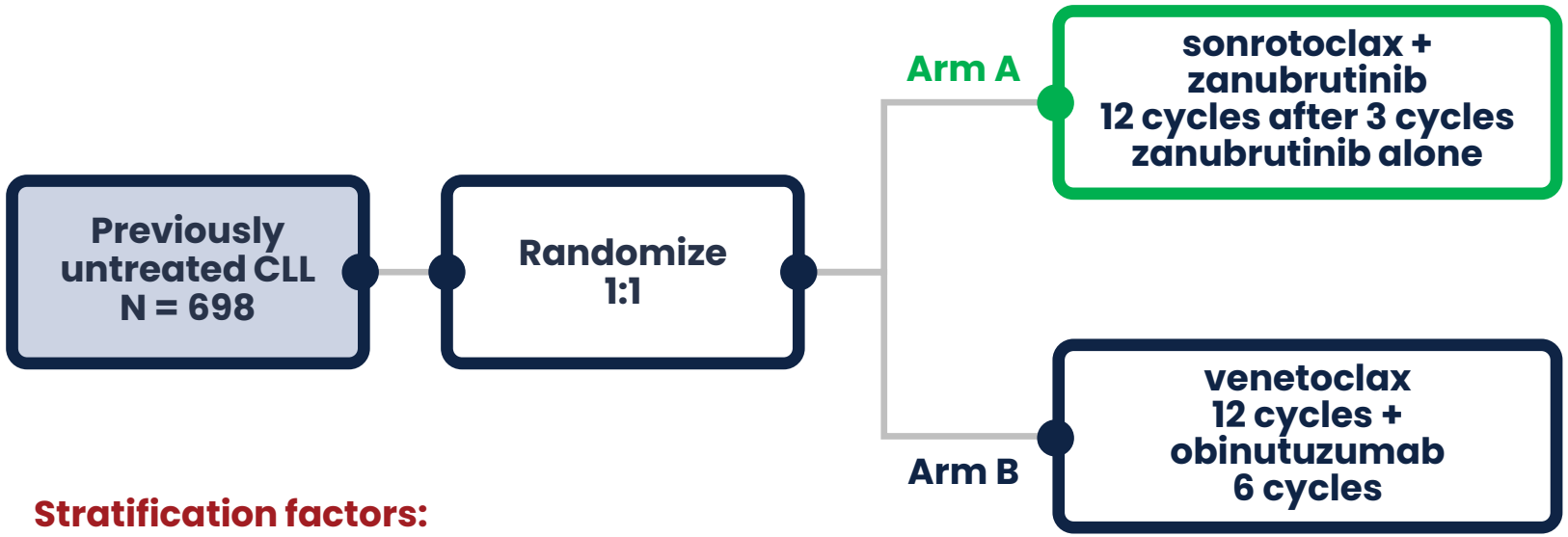
[1] Internal data, DCO: 01MAR2025
[2] Munir, EHA 2025

CELESTIAL-TNCLL (301)[1]: fixed duration sonro+zanu vs. ven+obin

Only global Phase 3 trial designed to demonstrate PFS superiority over current FD SOC

TN CLL

Completed enrollment in February 2025



Primary endpoint

- PFS

Key secondary endpoints

- CRR
- OS

Stratification factors:

- Age (<65 years vs. ≥ 65 years)
- IGHV (mutated vs. unmutated)
- del(17p)/TP53

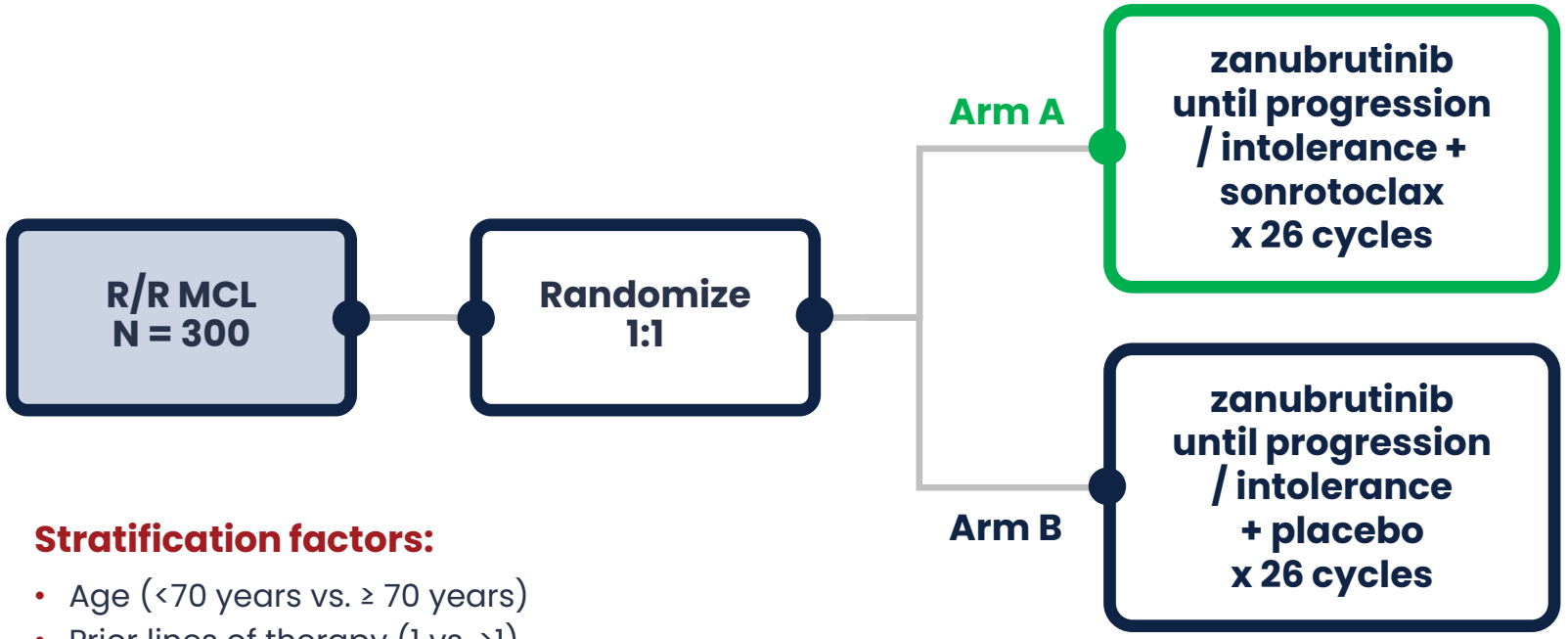
[1] NCT06073821

CELESTIAL-RRMCL (302)[1]: sonrotoclax + zanubrutinib vs. zanu

Phase 3 global study design

R/R MCL

Enrollment ongoing



Stratification factors:

- Age (<70 years vs. ≥ 70 years)
- Prior lines of therapy (1 vs. >1)
- Geographic regions

Primary endpoint

- PFS

Key secondary endpoints

- OS

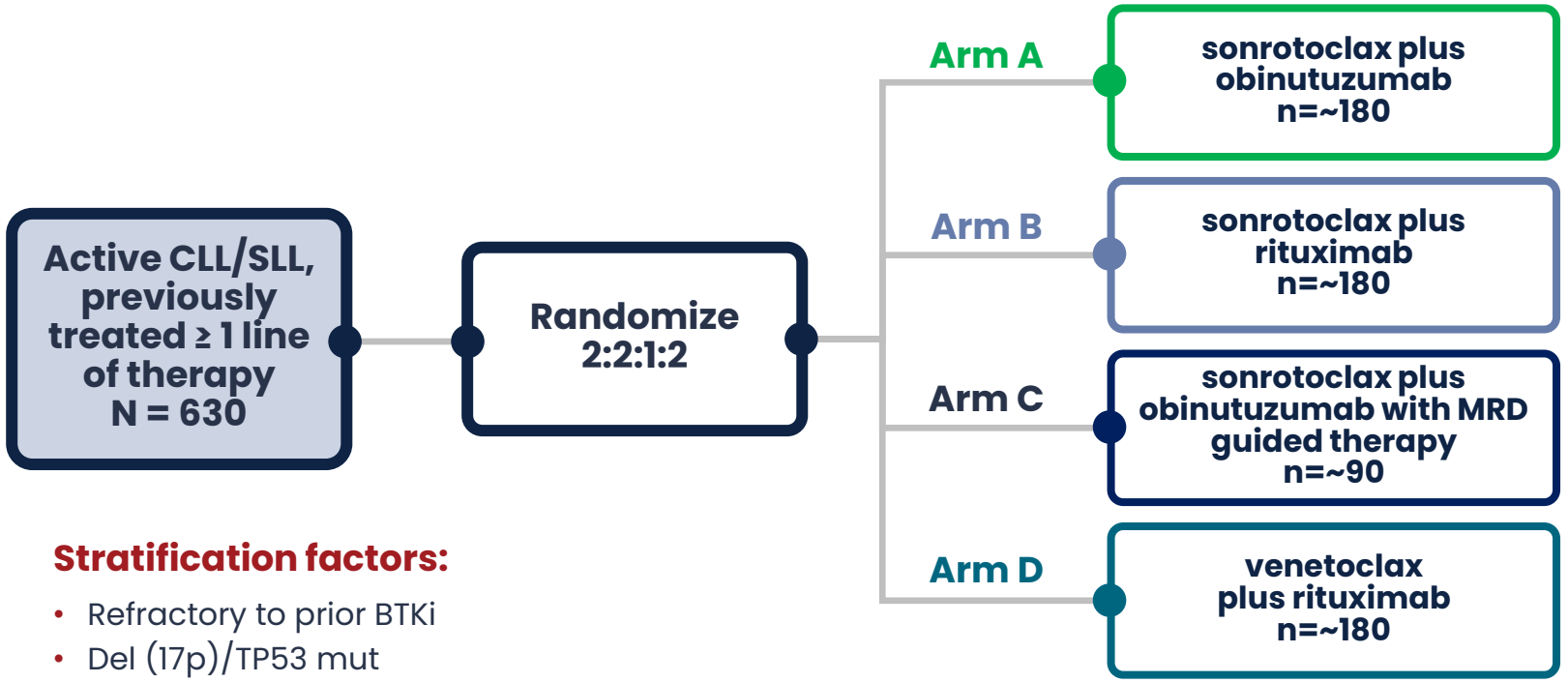
[1] NCT06742996

CELESTIAL-RRCLL (303)[1]: sonro+anti-CD20 vs. ven+anti-CD20

A head-to-head trial to demonstrate superiority of sonrotoclax over venetoclax

R/R CLL

First site initiated



Stratification factors:

- Refractory to prior BTKi
- Del (17p)/TP53 mut
- Prior BCL2i exposure

Primary endpoint

- PFS of SO vs. VR

Key secondary endpoints

- PFS of SR vs. VR
- uMRD
- CRR
- OS

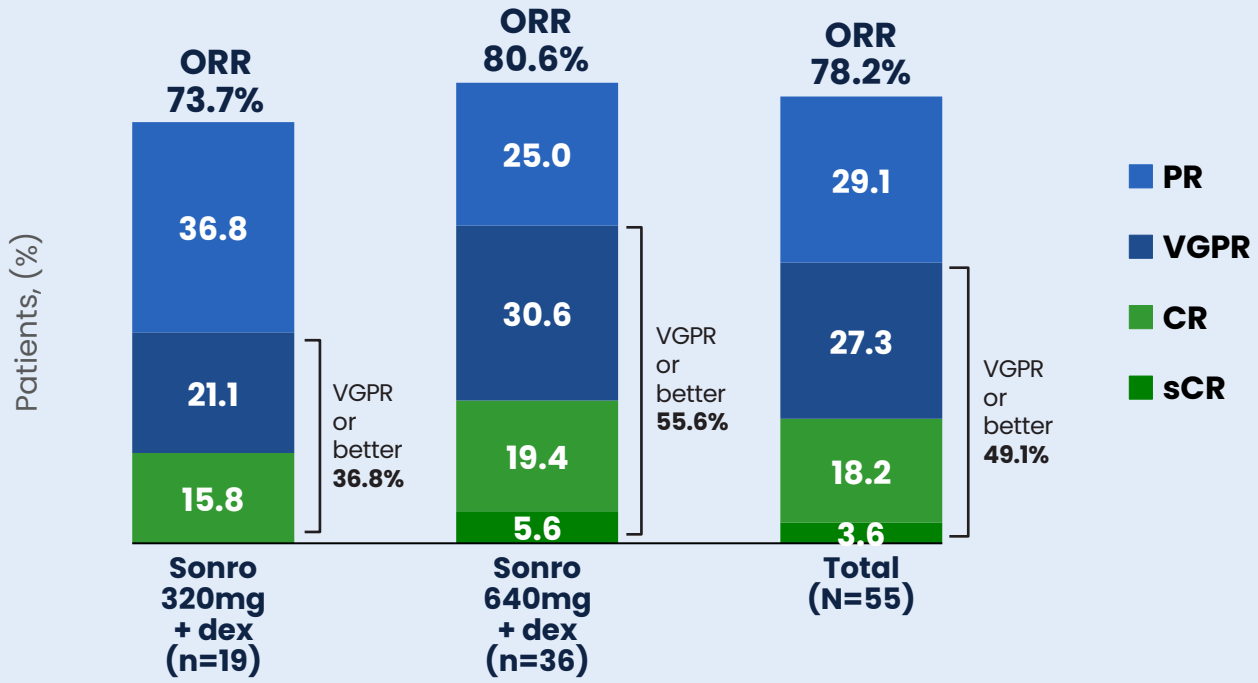
[1] NCT06943872

We are moving aggressively towards registration in multiple myeloma patients with t(11;14)

MM

- t(11;14) represents ~20% of the multiple myeloma population
- Sonrotoclax ORR compares favorably to previous study with venetoclax (ORR-62%[1])
- Sonrotoclax+dara+dex dose escalation ongoing, without any new safety signals
- Pivotal phase 3 study in 2L+ MM for sonrotoclax triplet with the use of an intermediate endpoint for accelerated approval is being planned

Sonrotoclax+dex shows promising efficacy



[1] Mateos et al. Results from the randomized, open-label phase 3 CANOVA study of venetoclax-dexamethasone versus pomalidomide-dexamethasone in patients with t(11;14)-positive relapsed/refractory multiple myeloma. IMS Annual Meeting Sep 2023

CR, complete response; dex, dexamethasone; ORR, overall response rate; PR, partial response; VGPR, very good partial response; sCR, stringent complete response

Sonrotoclax has broad development program with registrational plans in multiple indications

Indication	Regimen	Study details	Early clinical development	Registrational trial
TN CLL/SLL	+zanubrutinib	108: ramp-up optimization	Ongoing	
		204: COC study	Ongoing	
		CELESTIAL 301: vs. venetoclax+obinutuzumab	Ongoing	
R/R CLL/SLL	Monotherapy	CELESTIAL 202: ph2 for AA (CN)	Filed	
	+anti-CD20	CELESTIAL 303: vs. venetoclax+rituximab	Start-up	
R/R MCL	Monotherapy	CELESTIAL 201: ph2 for AA	Filed[1]	
	+zanubrutinib	CELESTIAL 302: vs. zanubrutinib	Ongoing	
R/R WM	Monotherapy	CELESTIAL 203: ph2 for AA	Ongoing	
R/R MM	+anti-CD38, dex	105: dose escalation/expansion	Ongoing	Under planning
TN, R/R AML	+azacitidine	103: dose escalation/expansion	Ongoing	

[1] Submitted in China, global filings anticipated in 2H25

AA, accelerated approval potential; COC, contribution of components; dex, dexamethasone

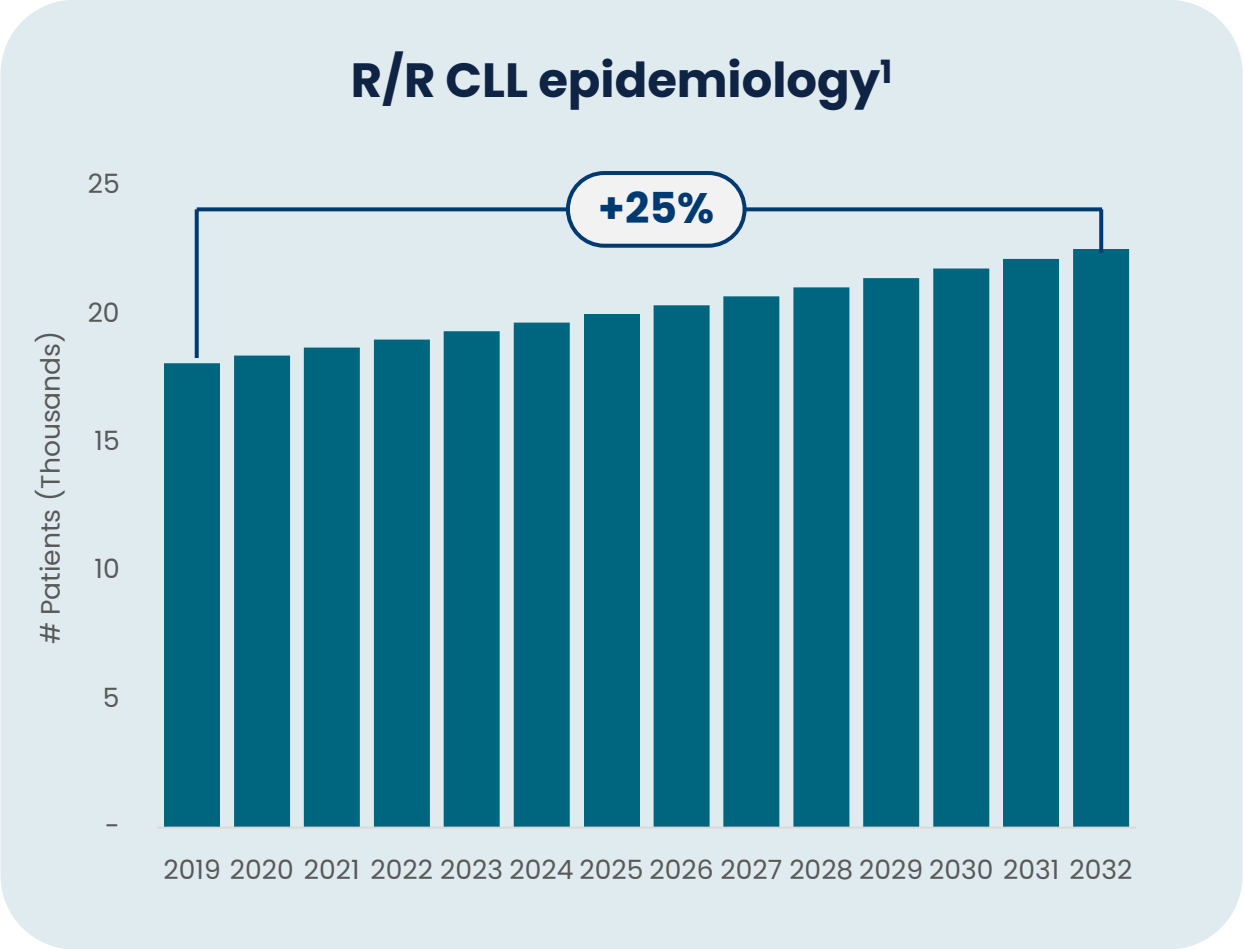
Sonrotoclax initial market filings started based on two Phase 2 single-arm monotherapy studies in R/R CLL and R/R MCL

Indication	Countries	Target filing
R/R CLL	China only	Filed in April 2025 in China
R/R MCL	Global	Filed in May 2025 in China; Global filing 2H25

- CDE granted Priority Review for both filings
  - CLL filing was completed in 2.5 months from DCO
- Global filing in R/R MCL in 2H25

BTK CDAC
(BGB-16673)

BGB-16673 designed to address unmet need in CLL and other B-cell malignancies



- Unmet needs in CLL are evolving and while there have been improvements in outcomes in treatment-naïve CLL patients, there is an **enduring unmet need especially in the relapsed setting**
- Novel options are needed for the increasing number of patients who will relapse after 1L therapy
- BGB-16673 is potential **first-in-class, orally available BTK degrader**

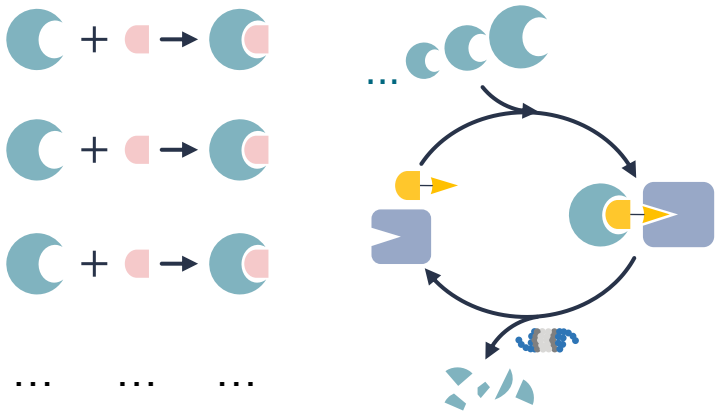
[1] DelveInsight, U.S. + EU5 + JP

CDAC emerging as enhanced approach to target BTK pathway

Degraders offer significant advantages over small molecule inhibitors

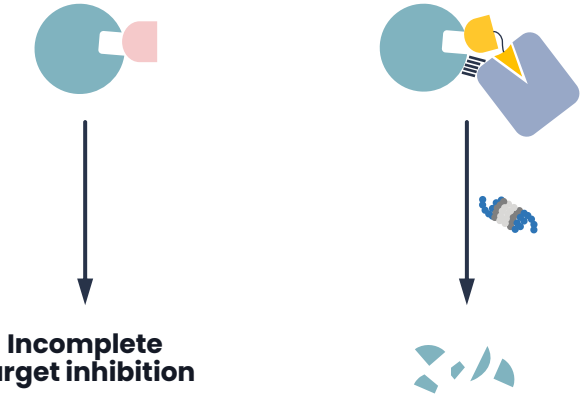
Catalytic activity[1]

Inhibitor	CDAC
Blocks one target protein	Degrades multiple target proteins



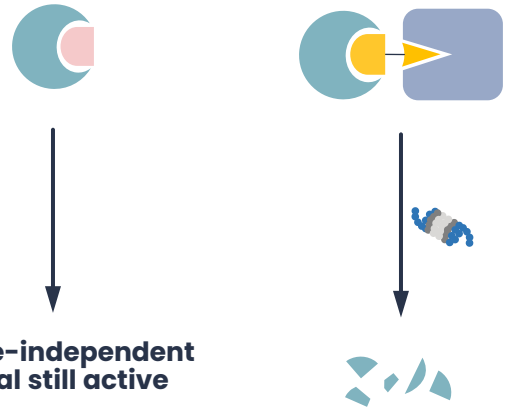
Higher barrier to resistance[2]

Inhibitor	CDAC
Mutations that weaken binding abolish activity	Retains activity despite mutations that impair binding



Disrupts scaffold function[3]

Inhibitor	CDAC
Active only against kinase-mediated signaling	Nullifies all associated signaling



Wild-type BTK | Mutant BTK | E3 ligase | Proteasome | BTK CDAC | Traditional BTK inhibitor

[1] Yoon H. et al J Clin Invest. 2024;134(1):e175265
[2] Feng X et al; Poster presented at EHA 2023; #P1239
[3] Yuan et al J Biol Chem. 2022 Nov; 298(11)

CaDAnCe 101: Phase 1/2, BGB-16673 monotherapy dose-escalation/expansion study in R/R B-cell malignancies

Phase 1

Part 1: dose escalation and expansion

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

Part 1f: expansion

Selected B-cell malignancies BTK naïve
(CLL/SLL, MCL, WM, RT, MZL)

Phase 1 (except 1f)	Pts (n)
R/R CLL	66
R/R WM	36
R/R FL/MZL	46
Other B-cell malignancies	46

Phase 2

Cohort 1:
Post BTKi/BCL2i R/R CLL/SLL

Cohort 2-7:
B-cell malignancies

Potential pivotal cohort with enrollment ongoing and >100 pts enrolled to date

BTK, Bruton tyrosine kinase; cBTKi, covalent Bruton tyrosine kinase inhibitor; CLL/SLL, chronic lymphocytic leukemia/small lymphocytic lymphoma; DLBCL, diffuse large B-cell lymphoma; FL, follicular lymphoma; MCL, mantle cell lymphoma; MZL, marginal zone lymphoma; R/R, relapsed/refractory; RT, Richter transformation; WM, Waldenström macroglobulinemia

R/R CLL patients from CaDAnCe-101 phase 1 study are heavily pretreated with high-risk features[1]

R/R CLL

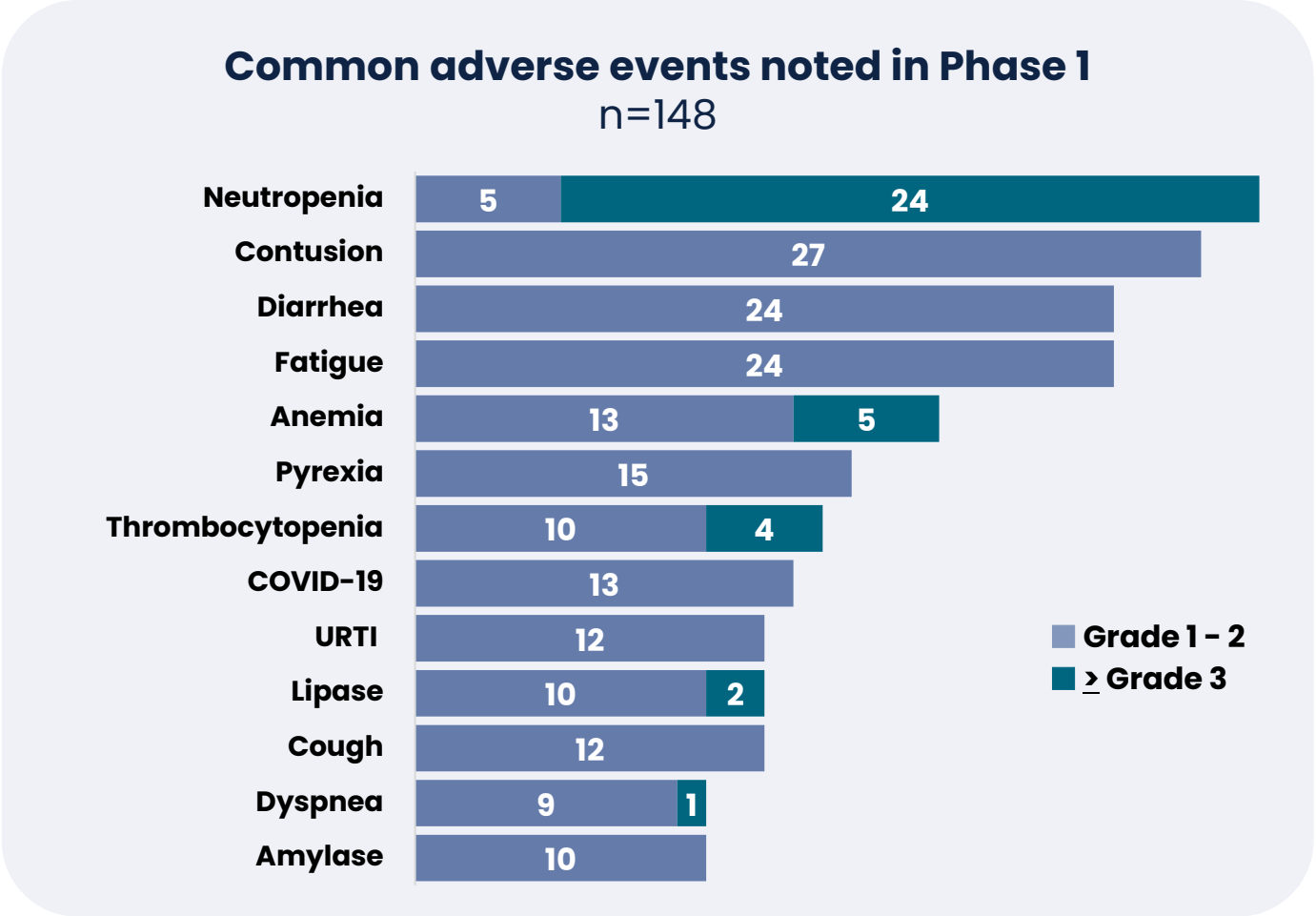
Patient characteristics	Total (N=66)
Age, median (range), years	70 (47-91)
CLL/SLL risk characteristics at study entry, n/N with known status (%)	
Binet stage C	29/62 (46.8)
Unmutated IGHV	38/49 (77.6)
del(17p) and/or *TP53* mutation[2]	43/66 (65.2)
Complex karyotype (≥3 abnormalities)	22/44 (50.0)

Patient characteristics	Total (N=66)
Mutation status, n/N (%)	
BTK mutation present	24/63 (38.1)
PLCG2 mutation present	10/63 (15.9)
No. of prior lines of therapy, median (range)	4 (2-10)
Prior therapy, n (%)	
Chemotherapy	47 (71.2)
cBTK inhibitor	62 (93.9)
ncBTK inhibitor	14 (21.2)
BCL2 inhibitor	54 (81.8)
cBTK + BCL2 inhibitors	42 (63.6)
cBTK + ncBTK + BCL2 inhibitors	12 (18.2)
Discontinued prior BTK inhibitor due to PD, n/N (%)[3]	55/62 (88.7)

[1] Scarfo, EHA, 2025
[2] Analyzed centrally by next-generation sequencing with limit of detection of 0.1%
[3] The remaining seven patients discontinued prior BTK inhibitor due to toxicity (n=4), treatment completion (n=2), and other (n=1)
DCO: 03MAR2025
BCL2, B-cell lymphoma 2; BTK, Bruton tyrosine kinase; cBTK, covalent Bruton tyrosine kinase; CLL, chronic lymphocytic leukemia; ncBTK, noncovalent Bruton tyrosine kinase; PD, progressive disease; SLL, small lymphocytic lymphoma

In large group of late line R/R B-cell malignancy patients, BGB-16673 has shown tolerable safety profile

- The AE profile of BGB-16673 is consistent with the MOA and related to on target BTK degradation
- Treatment related AE leading to discontinuation occurring in <5% of these very late line patients
- Off target AEs associated with BTK inhibitors including arrythmia, hypertension have not been noted higher than the baseline rate in this population



DCO: 03MAR2025

In heavily pretreated R/R CLL patients, significant responses have been observed[1]

R/R CLL

Response rates	200mg (n=16)	Total (N=66)
Best overall response, n (%)		
CR/CRi	1 (6.3)	**3 (4.5)**
PR[2]	12 (75.0)	44 (66.7)
PR-L	2 (12.5)	9 (13.6)
SD	0	5 (7.6)
PD	1 (6.3)	2 (3.0)
Discontinued prior to first assessment	0	3 (4.5)
Overall response rate, n (%)[3]	**15 (93.8)**	**56 (84.8)**
Disease control rate, n (%)[4]	15 (93.8)	61 (92.4)
Time to first response, median (range), months[5]	2.9 (2.6-8.3)	2.8 (2.0-19.4)
Time to best response, median (range), months	3.4 (2.6-13.8)	3.4 (2.0-19.4)
Duration of exposure, median (range), months	16.2 (2.9-24.6)	12.9 (0.2-29.6)

Responses are seen in patients with high-risk features

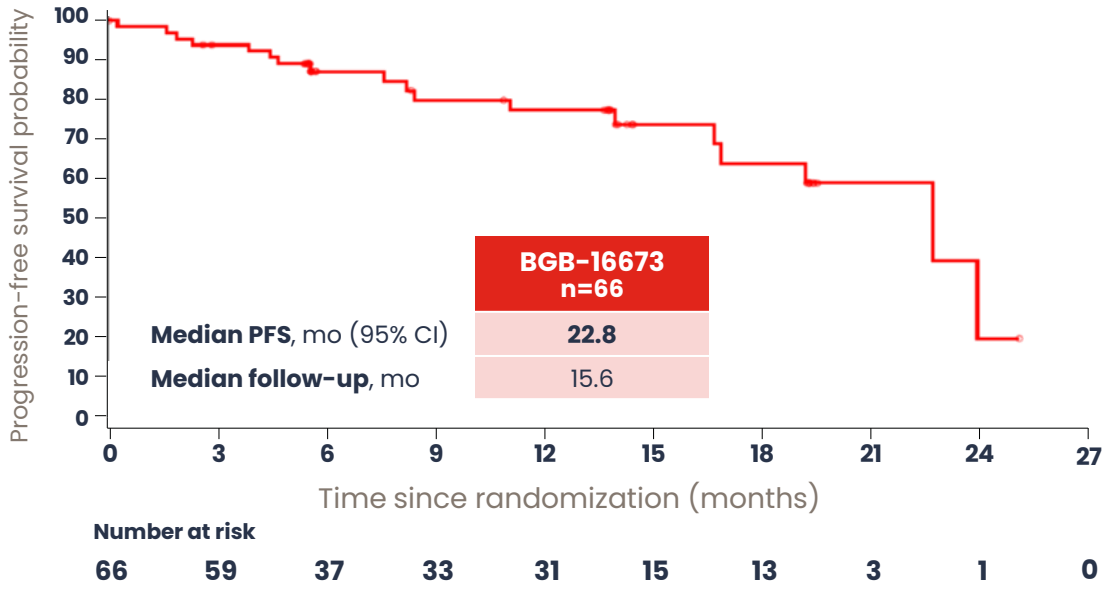
cBTKi+BCL2i exposure (n=42)	**90.5%**
cBTKi+ncBTKi+BCL2i exposure (n=12)	**75%**
BTK mutation (n=24)	**75%**
PLCγ2 mutation (n=10)	**90%**

[1] Scarfo, EHA, 2025
[2] Of 44 patients with PR, 12 achieved all nodes normalized
[3] Includes best overall response of PR-L or better
[4] Includes best overall response of SD or better
[5] In patients with a best overall response of PR-L or better
CR, complete response; CRi, complete response with incomplete marrow recovery; PD, progressive disease; PR, partial response; PR-L, partial response with lymphocytosis; SD, stable disease

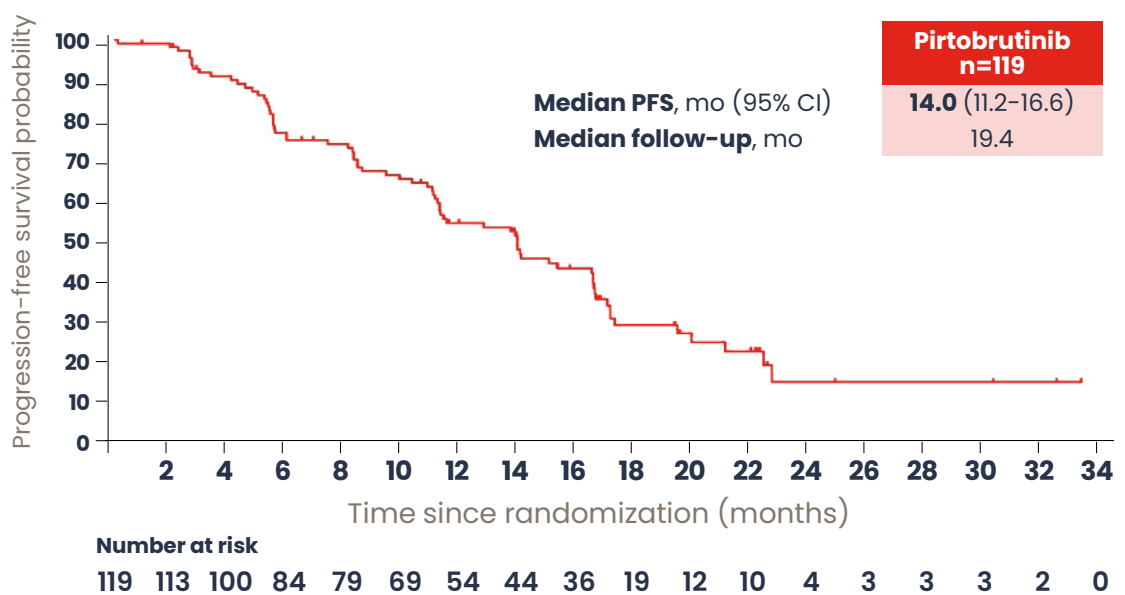
Emerging data for R/R CLL provides confidence to conduct H2H superiority trial vs. pirtobrutinib

R/R CLL

BGB-16673: PFS by investigator[1]



Pirtobrutinib: PFS by IRC[2]



	CaDAnCe-101 (BTK CDAC)	**BRUIN321** (pirtobrutinib)
Median prior lines of therapies	4	3
BTKi+BCL2i exposed	82%	50%
Prior BTKi discontinuation due to PD	89%	71%

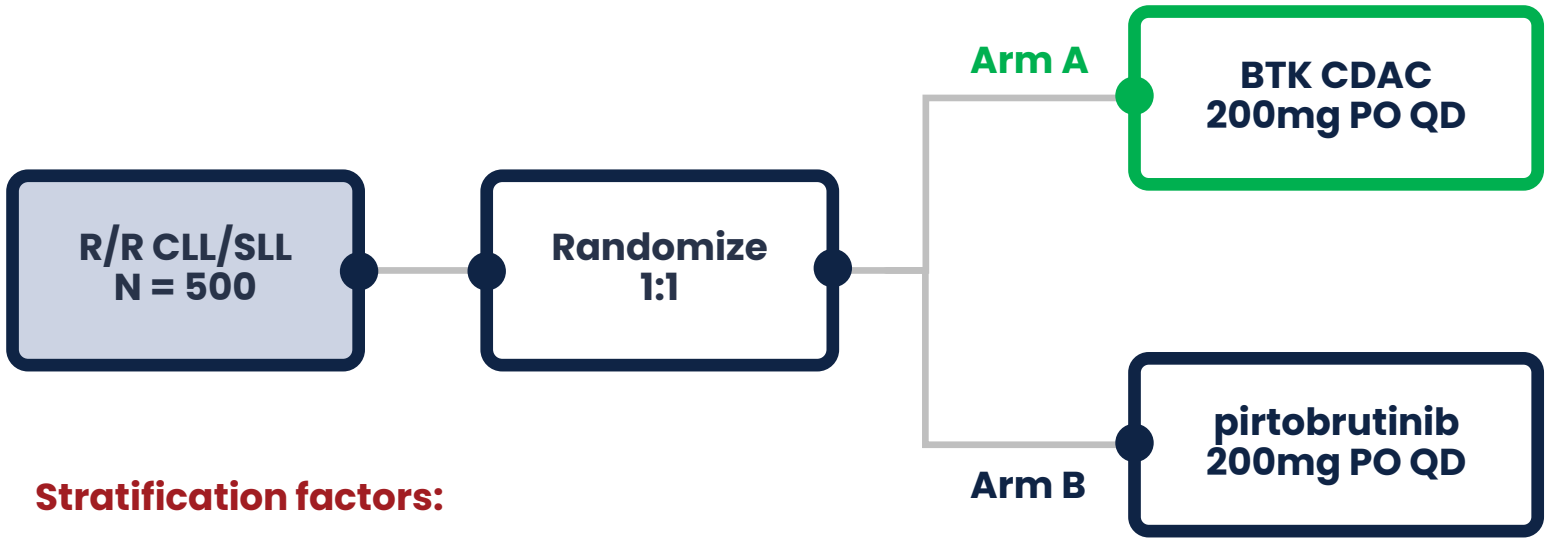
[1] Scarfo L. et all EHA 2025
[2] Sharnan J. et al ASH 2024

CaDAnCe 304[1]: BTK CDAC vs. pirtobrutinib

Global phase 3 in R/R CLL/SLL post-cBTKi

R/R CLL

First patient expected in 2H 2025
CLL dose for Phase 3 agreed with FDA



Stratification factors:

- Prior BCL2i
- del(17p)/TP53
- Refractory to last cBTKi

Primary endpoint

- PFS

Key secondary endpoints

- OS

[1] NCT06973187

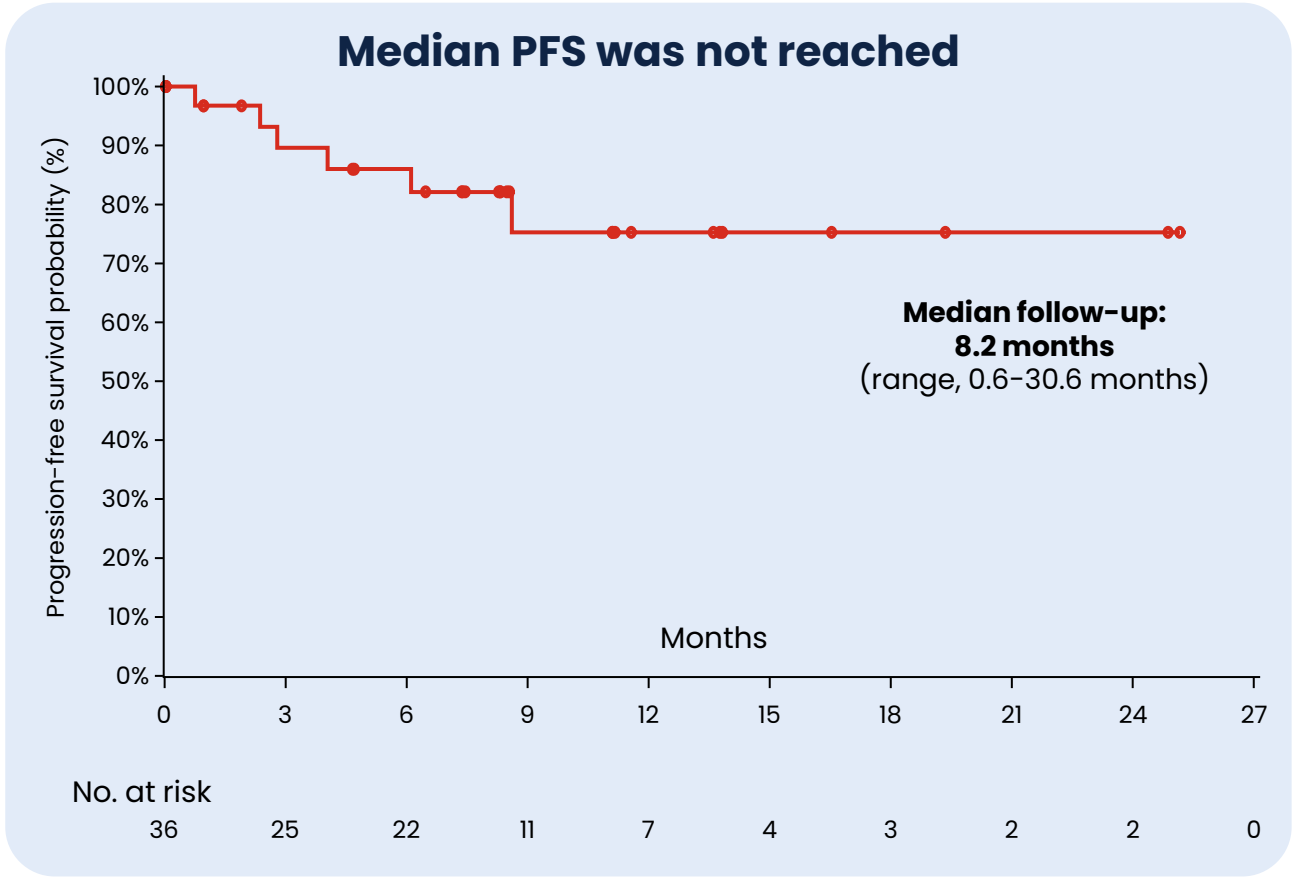
CaDAnCe 101: deep and durable responses seen in R/R WM[1]

Responses across all risk groups including with prior cBTKI and ncBTKI

R/R WM

Response rates	Total[2] (N=32)
Best overall response, n (%)	
VGPR	10 (31.3)
PR	14 (43.8)
MR	3 (9.4)
SD	3 (9.4)
PD	1 (3.1)
Discontinued prior to first assessment	1 (3.1)
ORR, n (%)[3]	27 (84.4)
Major response rate, n (%)[4]	24 (75.0)
Disease control rate (DCR), n (%)[5]	30 (93.8)

- Responses were observed starting at the lowest dose (100mg; 10/10) and in patients with prior cBTK inhibitor (27/32) or ncBTK inhibitor (4/4)



[1] Frustaci, EHA, 2025
[2] Efficacy-evaluable population
[3] Includes best overall response of MR or better
[4] Includes best overall response of PR or VGPR
[5] Includes best overall response of SD or better
WM = Waldenström macroglobulinemia, cBTK, covalent BTK; MR, minor response; ncBTK, noncovalent BTK; PD, progressive disease; PR, partial response; SD, stable disease; VGPR, very good partial response

CaDAnCe 101: efficacy data in R/R FL and MZL[1]

In heavily pretreated patients, BGB-16673 has shown favorable risk-benefit profile

R/R FL, MZL

- BGB-16673 had **durable antitumor activity** with short time to response in heavily pretreated patients with NHL, including those with BTK inhibitor–resistant disease
- **ORR was 41.7% in patients with FL and 50% in those with MZL**
- Four patients achieved CR (FL, n=1; MZL, n=3)

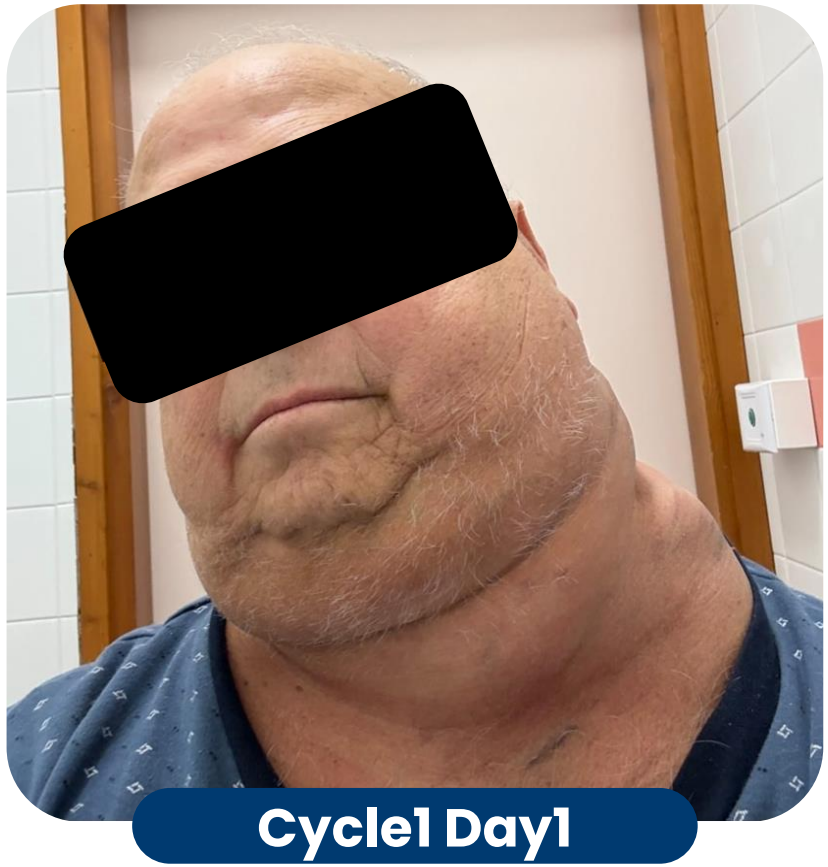
Responses by histology for R/R FL and MZL patients from CaDAnCe-101 study

Response rates	FL (n=12)	MZL (n=20)
Best overall response, n (%)		
CR	1 (8.3)	3 (15.0)
PR	4 (33.3)	7 (35.0)
SD	3 (25.0)	5 (25.0)
PD	3 (25.0)	3 (15.0)
ORR, n (%)[2]	5 (41.7)	10 (50.0)
Disease control rate, n (%)[3]	8 (66.7)	15 (75.0)
Time to first response, median (range), months[4]	2.6 (2.3-3.3)	2.9 (2.6-9.9)
Duration of response, median (95% CI), months[4]	9.5 (5.7-NE)	10.8 (2.8-NE)

[1] Zinzaini, ICML, 2025
[2] Includes best overall responses of PR or CR
[3] Includes best overall responses of SD or better
[4] In patients with best overall response better than SD
CR, complete response; NE, not estimable; ORR, overall response rate; PD, progressive disease; PR, partial response; SD, stable disease FL, follicular lymphoma; MZL marginal zone lymphoma; TEAE, treatment-emergent adverse event

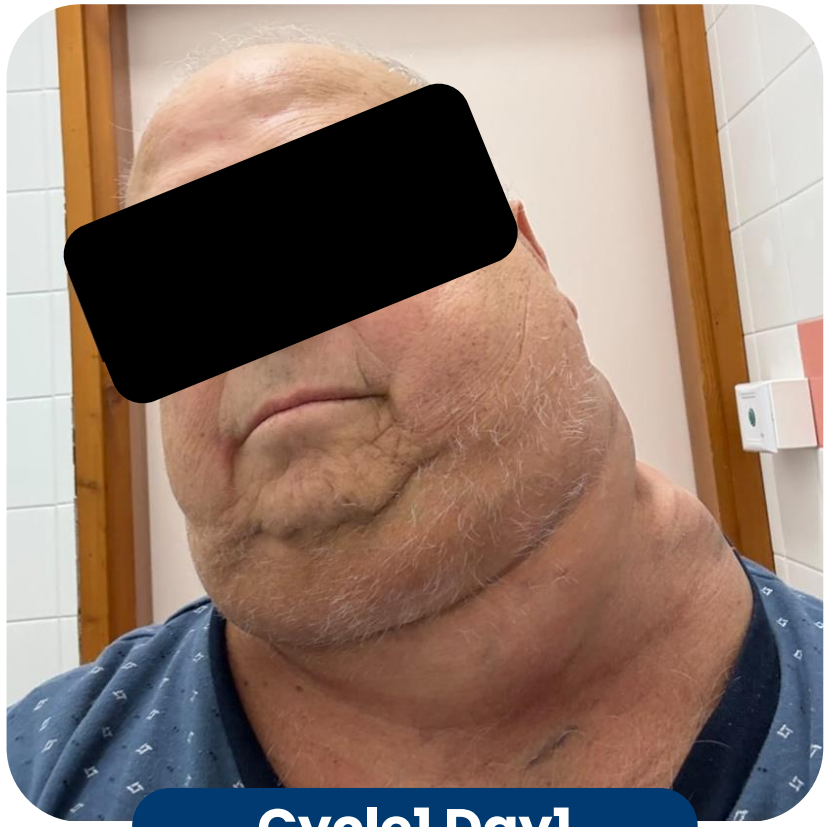
Responses have been observed in patients with Richters Transformation indicating the potential of the mechanism

- 69-year-old male
- Diagnosed with CLL in 2009 and treated with multiple treatments
- Diagnosed with Richters transformation in 2024
- Progressed after initial therapy of R-CHOP for RT and enrolled in CaDAnCe-101 trial



Responses have been observed in patients with Richters Transformation indicating the potential of the mechanism

- 69-year-old male
- Diagnosed with CLL in 2009 and treated with multiple treatments
- Diagnosed with Richters transformation in 2024
- Progressed after initial therapy of R-CHOP for RT and enrolled in CaDAnCe-101 trial



After only 3 weeks of treatment!

BGB-16673 has a broad development program with registrational plans in multiple indications

Indication	Regimen	Study details	Early clinical development	Registrational trial
TN CLL/SLL	Monotherapy	CaDAnCe 101: dose escalation/expansion	Ongoing	
	+zanubrutinib	CaDAnCe 104: dose escalation/expansion	Ongoing	
	+sonrotoclax		Ongoing	
R/R CLL/SLL	Monotherapy	CaDAnCe 101: phase 2 part for AA	Ongoing	
		CaDAnCe 302, 303[1]: vs. investigator's choice	Ongoing	
		CaDAnCe 304: vs. pirtobrutinib	Start-up	
	+sonrotoclax	CaDAnCe 104: dose escalation/expansion	Ongoing	Under planning
	+anti-CD20 bispecifics		Ongoing	
B-cell malignancies including WM and NHL	Monotherapy	CaDAnCe 101: dose escalation/expansion	Ongoing	WM under planning
	+sonrotoclax	CaDAnCe 104: dose escalation/expansion	Ongoing	
	+zanubrutinib		Ongoing	
	+anti-CD20 bispecifics		Ongoing	

✦ **We plan to file CaDAnCe 101 in CLL globally in 2026 for accelerated approval**

[1] 303 is China-only study
AA, accelerated approval potential

Recap and looking ahead

BeOne has comprehensive registrational program to address all CLL segments for treatment naïve and relapsed settings

Indication	Treatment	Study details	Phase 2	Phase 3	Approval
TN CLL/SLL	Continuous use	Zanubrutinib monotherapy vs. BR	Approved		
	Fixed duration	Zanubrutinib + sonrotoclax vs. VO	Ongoing		
R/R CLL/SLL	Continuous use	Zanubrutinib vs. ibrutinib	Approved		
		Sonrotoclax monotherapy (AA[1])	Filed		
		BGB-16673 monotherapy (AA[2])	Ongoing		
		BGB-16673 monotherapy vs. investigator's choice	Ongoing		
		BGB-16673 monotherapy vs. pirtobrutinib	Start-up		
	Fixed duration	Sonrotoclax + anti-CD20	Start-up		
		Sonrotoclax + BGB-16673	Under planning		

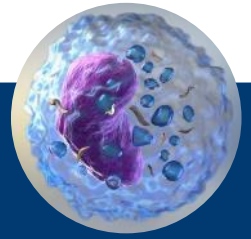
BTKi | BTK CDAC | BCL2i

[1] China only; global filing for MCL anticipated in 2H25
[2] Global filing anticipated in 2026
BR, bendamustine + rituximab; VO, venetoclax + obinutuzumab

B-cell malignancy pipeline outlook – CLL and beyond

Focus on immune cell-based modalities to develop practice-changing therapies

B-cell malignancy



- BTKi
- BCL2i
- BTK CDAC
- CD19 x CD20 x CD3 ◆
- BAFFR x CD22 x CD3 ◆
- CD20 x CD28 ◆
- CD20 x 4-1BB ●
- CD19 iγδT ◆

Oncogenic signal cluster

Cornerstone assets established the franchise leadership

- BTKi
- BCL2i
- BTK CDAC
- BCL2i 2G

Deliver greater patient impact by fully harnessing the killing function of immune cells

T-cell engager cluster

- CD19 x CD20 x CD3 ◆
- BAFFR x CD22 x CD3 ◆
- CD79b x TAA 1 x CD3 ●
- CD20 x CD28 ◆
- CD20 x 4-1BB ●

Cell therapy cluster

- CD19 iγδT ◆
- TAA iαβT ●

Macrophage engager

- Dual TAA x engager ●

Small molecule | Protein degrader | Bi/Tri-specific | Cell Therapy

● Discovery ◆ Investigational New Drug (IND)

Fully exploiting the killing potential of T-cells by CD3 T-cell engager and T-cell booster combinations[1]

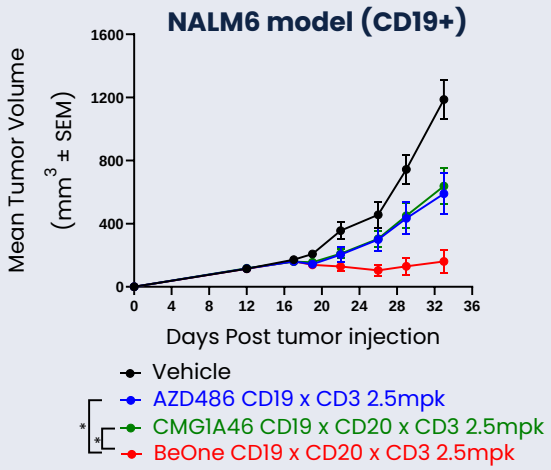
CD19 x CD20 x CD3 TsAb

- Broader coverage and mitigation of antigen loss issue
  - CD19 and CD20 dual targeting covers ~95% B-NHL cells
- Robust efficacy in all CD19 and CD20 positive cells
- On track to enter clinic in 2026

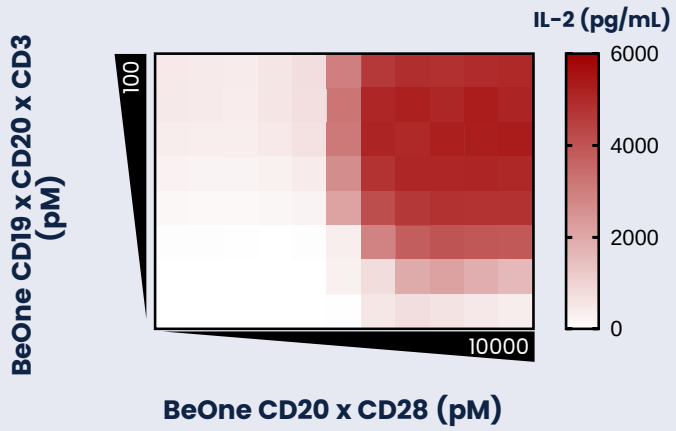
CD20 x CD28 BsAb

- CD20 selected due to higher tumor cell density than other TAAs
- Strong CD28 affinity enables superior T-cell activity boosting
- On track to enter clinic in early 2027

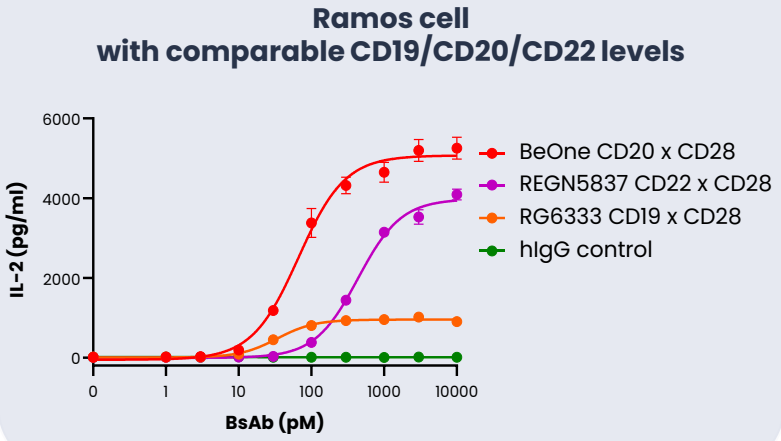
Better activity than leading competitors



Stronger T-cell activation with combination of CD19 x CD20 x CD3 and CD20 x CD28



Superior T-cell activity enhancement versus leading competitors

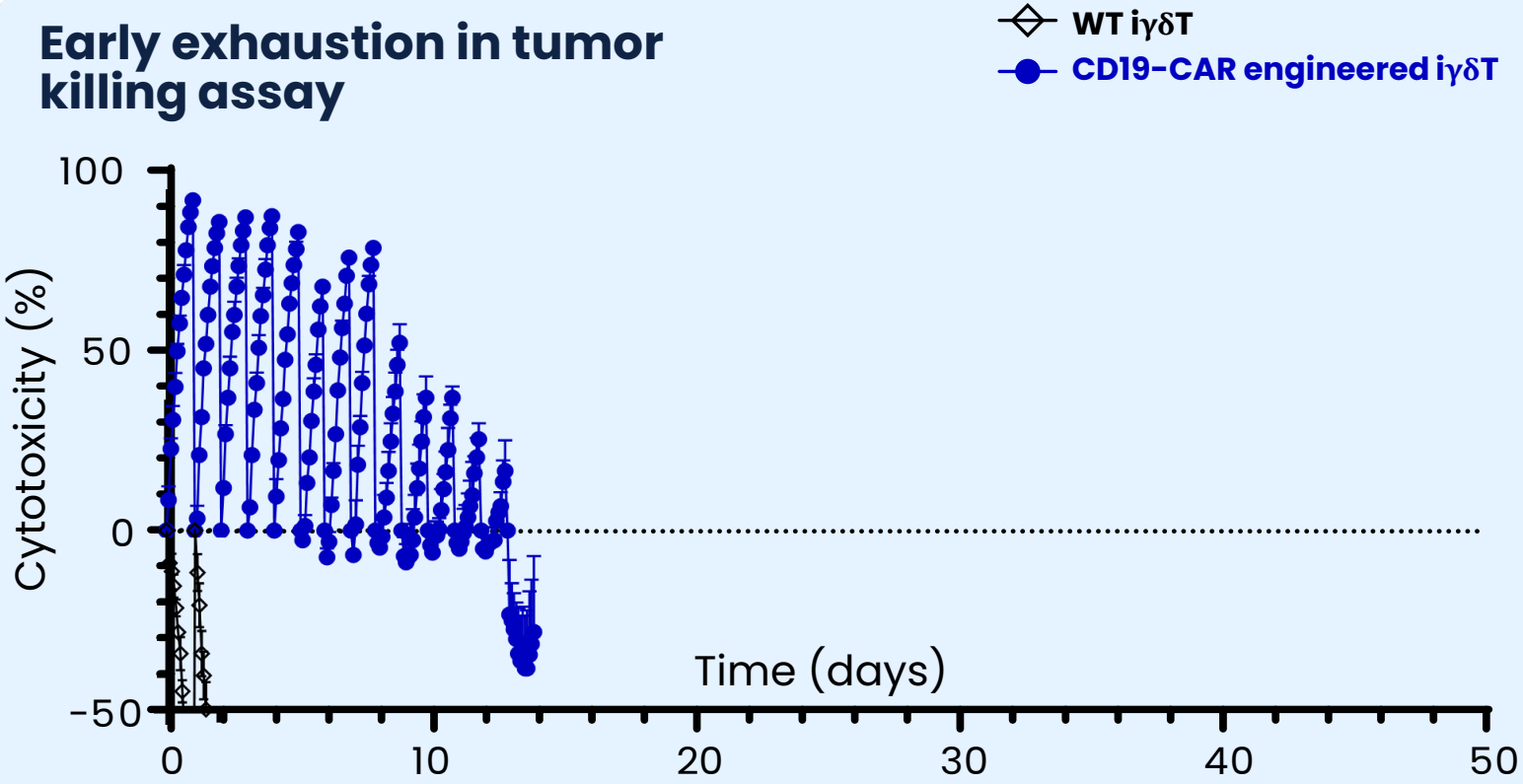


[1] Biosimilar molecules of AZD486, CMG1A46, REGN5837 and RG6333 were used in studies; In the right panel, 5pM epcoritamab was used to induce the first T-cell activation signal
TsAb, Tri-specific antibody; BsAb, bispecific antibody

Our first cell product, CD19-CAR iγδT, potentially provides a solution to persistence issues associated with allo-CAR-T

- Despite impressive clinical outcomes, autologous cell therapies have limited adoption
- Our proprietary iPSC derived allogeneic γδT cell therapy platform:
  - Highly optimized for γδT differentiation (purity>99%) and expansion (>10M fold)
  - Integrates **unique signal converter** that synergizes with the additional proprietary genetic edits (>10) to enhance efficacy and persistence
- On track to enter clinic in 2026

Early exhaustion in tumor killing assay



Fresh tumor cells were added daily to assay without changing the iPSC-derived γδT cells to determine durability of tumor cell killing w/o IL2 and IL15

Our first cell product, CD19-CAR iγδT, potentially provides a solution to persistence issues associated with allo-CAR-T

- Despite impressive clinical outcomes, autologous cell therapies have limited adoption
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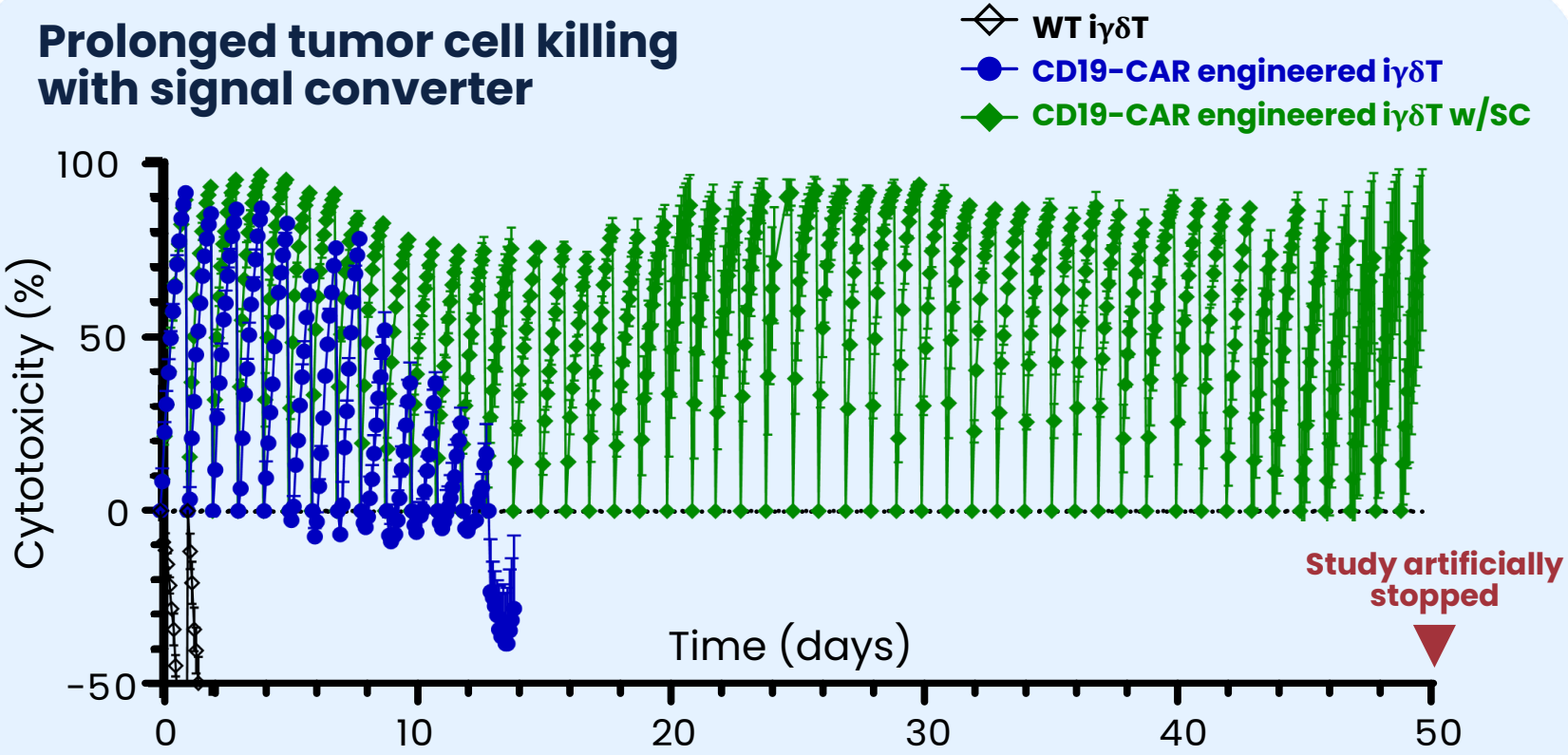
Prolonged tumor cell killing with signal converter

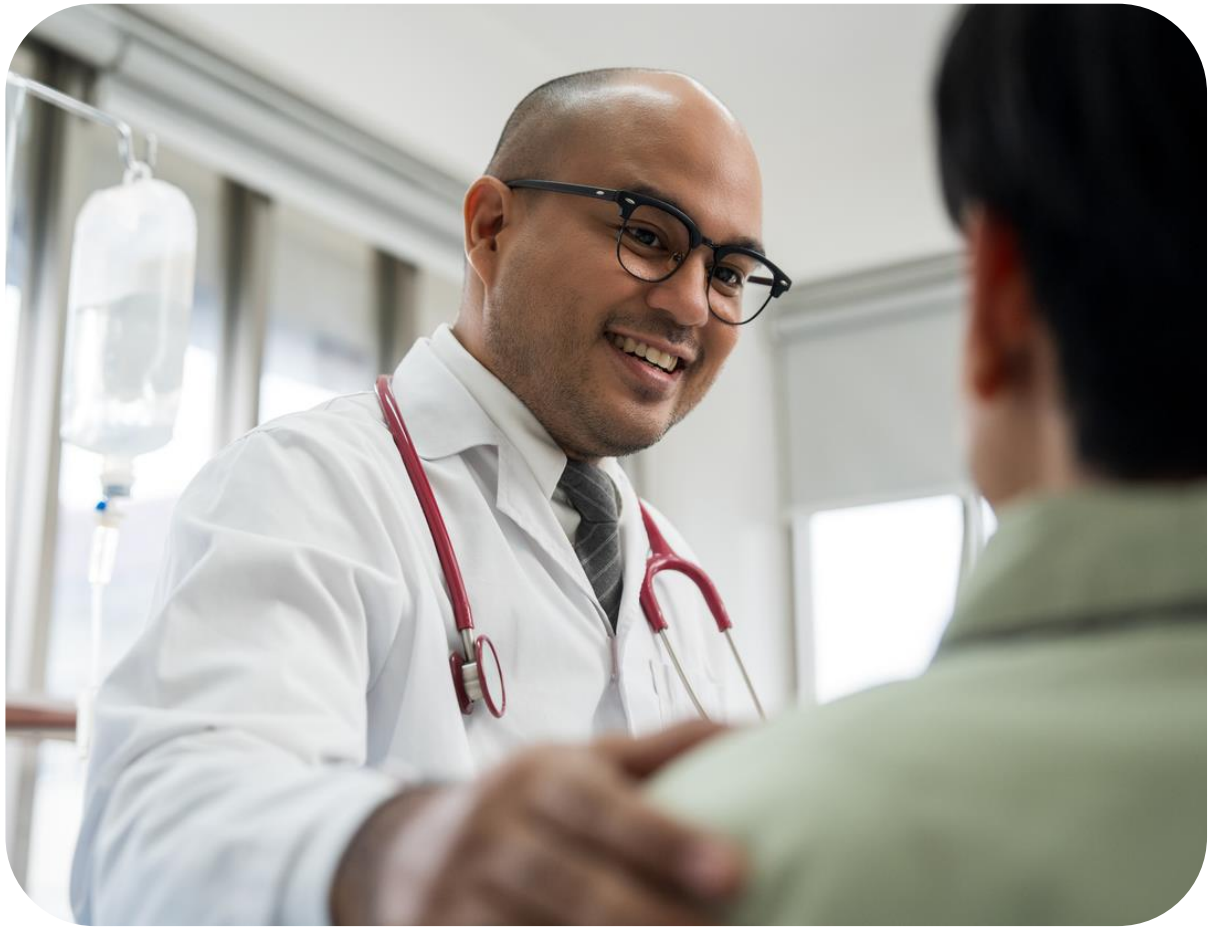


Fresh tumor cells were added daily to assay without changing the iPSC-derived γδT cells to determine durability of tumor cell killing w/o IL2 and IL15

BRUKINSA is just the start

We are building a franchise

- **Provide options for all CLL segments.** A systematic development plan will help develop BeOne combinations across the disease spectrum
- **Accelerate development** with unparalleled speed from POC to multiple Phase 3 trial initiations planned for sonrotoclax and BTK CDAC
- **Compelling continued innovation for B-cell malignancies beyond CLL** with novel trispecific/bispecific TCEs and iPSC derived cell therapy platforms that have the potential to transform the field



10-minute break

Solid tumor portfolio

Mark Lanasa
Senior Vice President, Chief Medical Officer, Solid Tumors

Our pipeline includes diverse modalities and mechanisms across disease franchises

Breast/Gynecologic

- CDK4i
- CDK2i[2]
- BCL2i 2G
- KAT6A/Bi[1]
- B7-H4 ADC[2]
- Claudin6 x CD3 BsAb
- CDK2 CDAC[1]

Lung

- PanKRASi
- MTA Cooperative PRMT5i
- MAT2Ai[2]
- CEA ADC
- B7-H3 ADC
- EGFR CDAC
- EGFR x MET x MET TsAb
- EGFR x MET x MET ADC[1]

Gastrointestinal

- PanKRASi
- MTA Cooperative PRMT5i
- MAT2Ai
- FGFR2b ADC
- CEA ADC
- GPC3 x 4-1BB BsAb
- MUC1 x CD16A BsAb

Pan-tumor

- HPK1i
- DGKζi
- CCR8 mAb
- IL-15 prodrug

Small molecule | Protein degrader | Bi/Tri-specific | mAb | ADC | Cytokine therapy

[1] Not yet in the clinic

[2] BeOne has global rights for CDK2i (Ensem partnership), B7-H4 ADC (DualityBio partnership), MAT2Ai (CSPC Zhongqi Pharmaceutical Technology)

Target(i), target inhibitor; CDAC, chimeric degradation activating compound; ADC, antibody drug conjugate; BsADC, bispecific ADC; TsADC, trispecific ADC; BsAb, bispecific antibody; TsAb, trispecific antibody

Breast and gynecologic cancers

Establishing an innovative breast and gynecological franchise

Breast / Gynecological

- CDK4i
- CDK2i
- BCL2i 2G
- KAT6A/Bi
- B7-H4 ADC
- Claudin6 x CD3 BsAb
- CDK2 CDAC[2]

Cell cycle cluster

Replace SOC CDK4/6 inhibition with selective CDK inhibitors

- CDK4i
- CDK2i
- CDK2 CDAC ◆

ER cluster

Novel MOAs establish new paradigms

- KAT6A/Bi ◆
- BCL2i 2G
- Novel CDAC 1 ●

ADC cluster

- B7-H4 ADC
- B7-H4 x HER3 BsADC 1 ◆
- Novel payload ADC ●
- Novel payload DAC ●

Immune cell engagers

- Claudin6 x CD3 BsAb
- MUC1 x CD16A BsAb

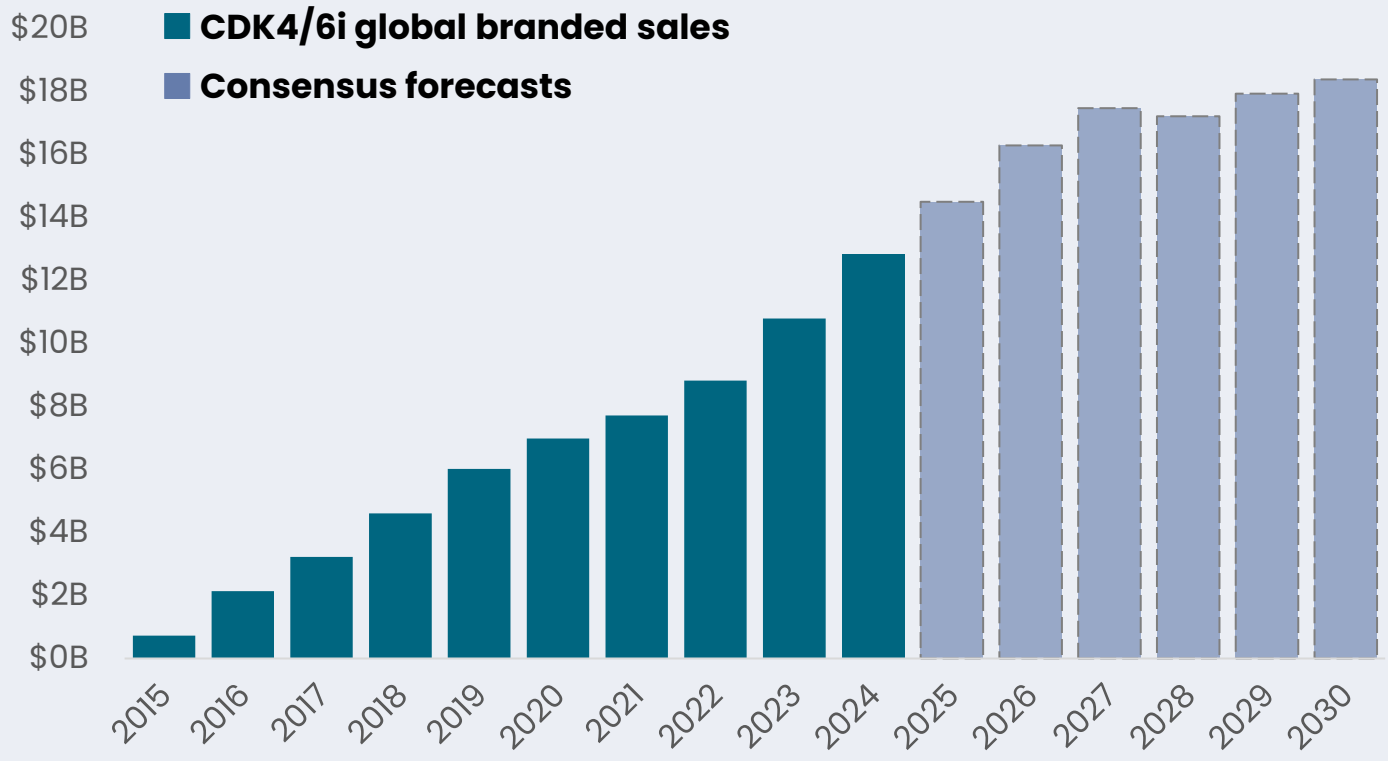
Small molecule | Protein degrader | Bi/Tri-specific | ADC

● Discovery ◆ Investigational New Drug (IND)

Target(i), target inhibitor; CDAC, chimeric degradation activating compound; ADC, antibody drug conjugate; BsADC, bispecific ADC; BsAb, bispecific antibody

CDK4/6 inhibitors represent one of largest drug classes in solid tumors at ~$13B and growing

Global CDK4/6i market is large, and still growing[1]



CDK4/6i are blockbusters yet **leave room for improvement**

Issues with CDK4/6i and what is unmet need

- **Heme tox that drives dosing interruptions** and holidays, and therefore suboptimal target coverage
- **Safety concerns** due to off-target inhibition

Hypothesis: CDK4 selective inhibitor can ameliorate these issues and drive superior therapeutic benefit for patients

[1] EvaluatePharma, accessed 27MAY2025

BGB-43395 (CDK4 inhibitor)

Thoughtfully designed to have superior potency and selectivity vs. atirmociclib

Selective CDK4 inhibitor sparing CDK6 and off-target toxicities

- Closed time gap with atirmociclib (Pfizer) in 1L HR+ breast cancer
- Emerging best-in-class profile with low hematologic toxicity at active PD dose levels

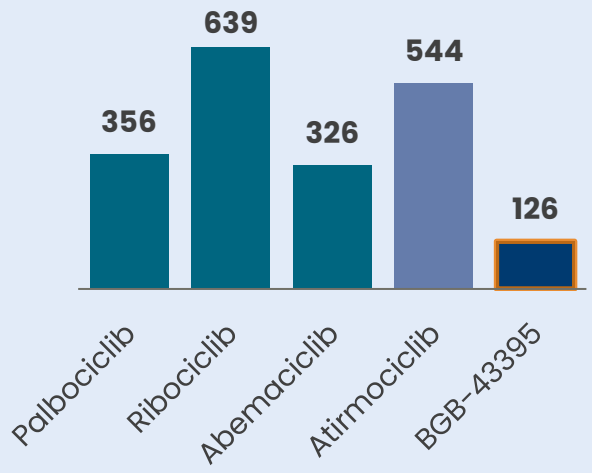
Clinical progress

- 300+ patients enrolled across dose escalation and expansion cohorts in HR+/HER2- MBC as of June 3, 2025
- Favorable tolerability, PD and clinical responses in extensively pretreated patients

Planning underway for Phase 3 studies in 1L and 2L HR+ breast cancer with 2L start as early as 4Q25

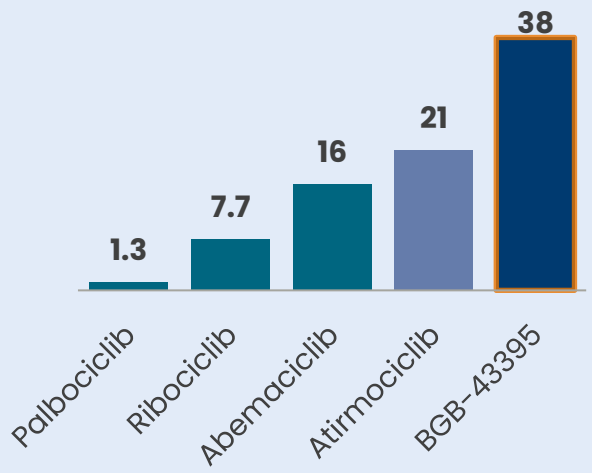
BeOne CDK4i presents the highest CDK4 potency and CDK6 selectivity

CDK4i potency



MCF-7[1] proliferation assay to assess CDK4 potency

CDK4/CDK6 selectivity



Cellular CDK6 IC50 divided by cellular CDK4 IC50 to assess CDK6 selectivity: 1) CDK4 cellular IC50 measured by p-RB in Jeko-1; 2) CDK6 cellular IC50 measured by p-RB in Pfeiffer CDK4 KO cell

[1] MCF-7, breast cancer cell line, is a commonly used model of early-stage breast cancer that is estrogen receptor positive and noninvasive
CDK4/6, cyclin-dependent kinase 4/6; HER2,human epidermal growth factor receptor 2; HR, hormone receptor; MBC, metastatic breast cancer

BGB-43395 (CDK4 inhibitor)

Speaker introduction: Dr. Shom Goel, key opinion leader in breast cancer

Dr. Shom Goel, MBBS, PhD

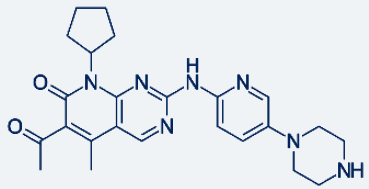
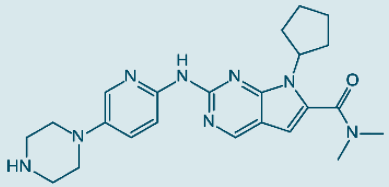
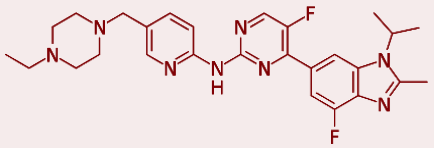
Group Leader and Medical Oncologist, Peter MacCallum Cancer Centre

- Associate Professor and Clinician-Scientist at the University of Melbourne and Peter MacCallum Cancer Centre
- Global PI / Translational PI for four randomized clinical trials in breast cancer
- Chair of the American Society of Clinical Oncology Education Committee (2023)
- Recent Snow Fellowship awardee and Era of Hope Scholar Award (U.S. Department of Defense)
- 60+ publications, with recent publications in high-impact journals including Nature, Cancer Cell, and Nature Cancer

BGB-43395 (CDK4 inhibitor)

Three CDK4/6 inhibitors approved, all with toxicity issues and loss of efficacy over time

- CDK4/6 inhibitors block the CDK4/6 complex and prevent the phosphorylation of the RB protein
- This stops the tumor cycle from progressing past the G1 phase, inhibiting further tumor cell growth and causing tumor regression
- More potent inhibition of CDK4 prevents emergence of resistance

	Palbociclib[1]	Ribociclib[1]	Abemaciclib[1]
			
Heme toxicity	Neutropenia	Neutropenia	Neutropenia
GI toxicity	Low	Low	Diarrhea
Suspected cause of key toxicity	Inhibition of CDK6	Inhibition of CDK6	Off targeting of CDK9, GSK3β and CAMKIIs

[1] Palbociclib (Ibrance®, Pfizer), abemaciclib (Verzenio®, Eli Lilly), and ribociclib (Kisqali®, Novartis) are approved CDK4/6 inhibitors indicated for the treatment of HR+/HER2– advanced or metastatic breast cancer

BGB-43395 (CDK4 inhibitor): study design and enrollment

Global dose escalation

- **Monotherapy** — n=60
- **Fulvestrant combo** — Doses to 800mg BID, n=64
- **Letrozole combo** — Doses to 800mg BID, n=54
- **Elacestrant combo ER⁺/HER2⁻ BC**[1]

Dose optimization (target n=120)

- **Fulvestrant combo** — 240mg, 400mg, 600mg BID, n=62
- **Letrozole combo** — 240mg, 400mg, 600mg BID, n=44

Cohort expansion (target n=80)

- **Fulvestrant combo** — 2L HR+ BC
- **Letrozole combo** — CDK4/6i Naïve BC

Region	Enrolled patients (as of 09JUN2025)
France	79
United States	59
Brazil	39
Australia	38
China	22
Korea	19

Region	Enrolled patients (as of 09JUN2025)
Japan	12
Moldova	9
Malaysia	4
New Zealand	2
Thailand	1
Total study enrollment	**284**

Completed | In progress | Not commenced

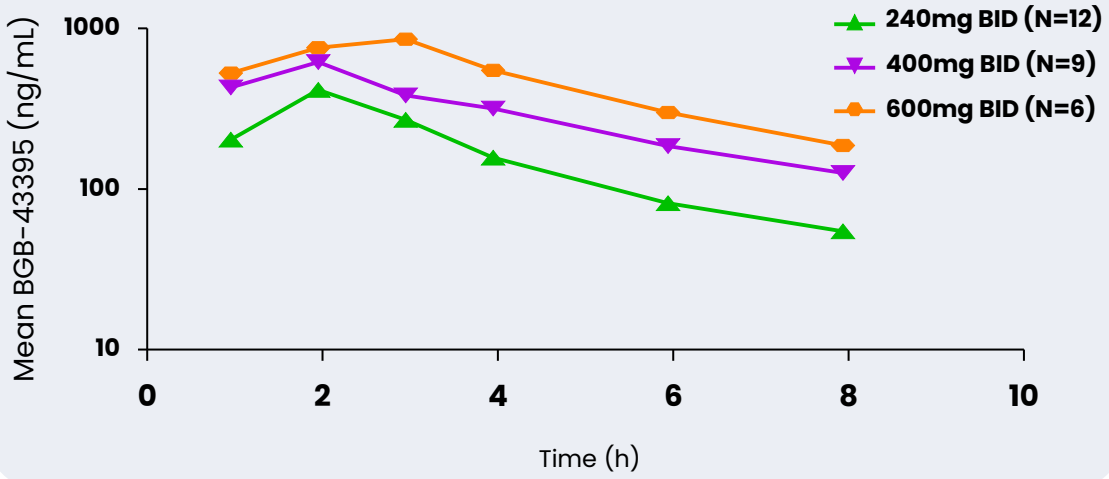
[1] This data will enable subsequent development of this combination

BGB-43395 (CDK4 inhibitor): pharmacokinetic data

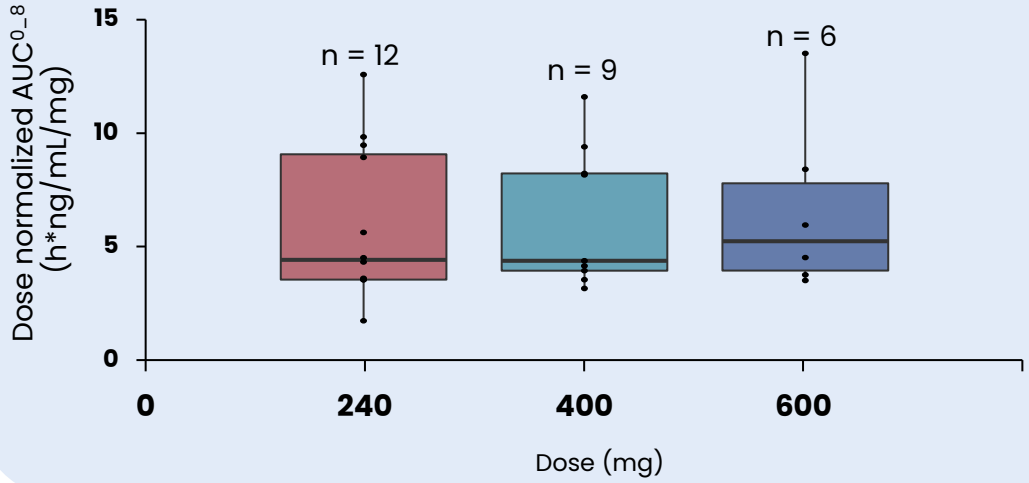
Linear PK with no major impact of race or co-meds (fulvestrant/letrozole)

- BGB-43395 exhibited rapid absorption and linear PK within available dose range
- Mean elimination half-life is 13 hours
- BGB-43395 exposures are not impacted by co-administration with either fulvestrant or letrozole
- No major differences in exposures observed between non-Asian and Asian populations

Preliminary mean BGB-43395 plasma concentration-time profiles[1]



Dose normalized AUC across doses PK is dose proportional



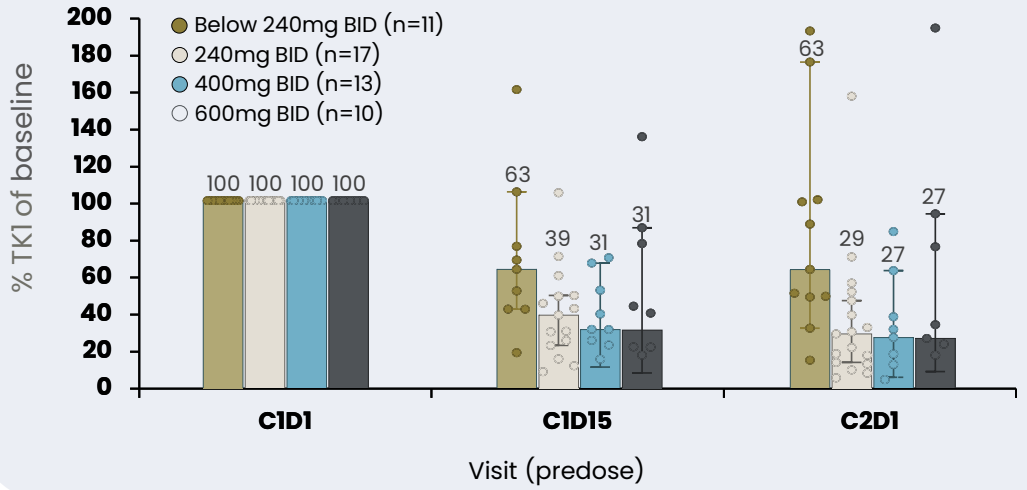
[1] Caveat: Non-compartmental analysis (NCA) is performed using nominal time points
DCO: 10MAR2025

BGB-43395 (CDK4 inhibitor): pharmacodynamic data

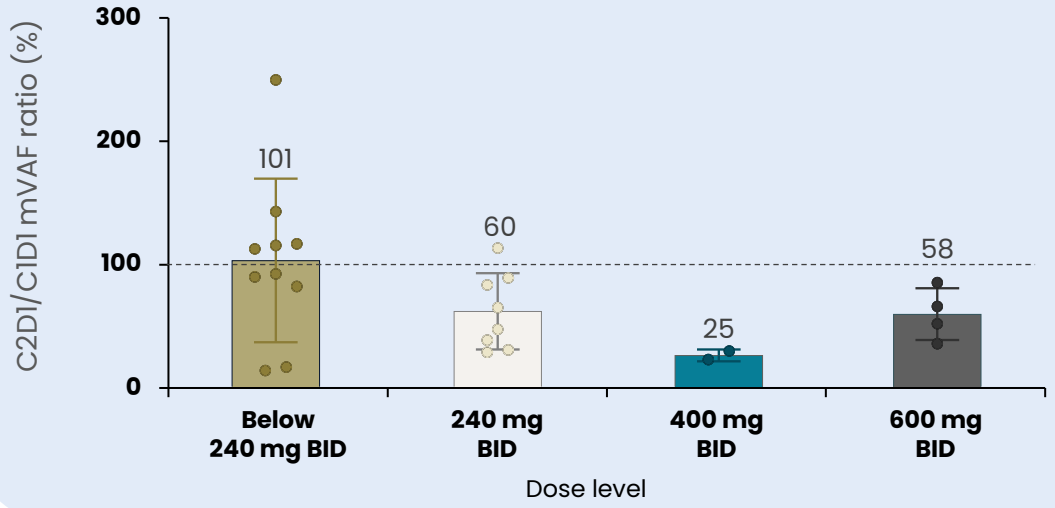
Both TK1 and ctDNA data suggest 240mg BID doses and above are biologically active

- Dose-dependent and durable PD biomarker modulation in blood (TK1 activity) was observed across dose levels
- Clinically meaningful PD effects were achieved at doses 240mg BID and above in fulvestrant combo cohort
- Early (C2D1) ctDNA decrease was observed starting from 240mg BID in fulvestrant combo

Plasma TK1[1] activity change post BGB-43395 +fulvestrant treatment (Dose-escalation, BC-only)



ctDNA mVAF[2] change post BGB-43395 +fulvestrant treatment (Dose-escalation, BC-only)



[1] TK1, Thymidine kinase, reduction to 20% defined as goal, since several published datasets indicate an 80% TK1 reduction level from baseline, when CDK4/6 inhibitors used at full dose plus ET in the metastatic setting. Yap T et al ASCO Annual meeting 2023 (atirmociclib), Malorni L et al EJC 2023 (ribociclib)

[2] mVAF, Mean variant allele fraction, represents the average proportion of mutant DNA sequences within a sample, reflecting the overall tumor burden

Plot: median with 95% CI

DCO: 12MAY2025

BGB-43395 (CDK4 inhibitor): safety data

Well-tolerated in combination with fulvestrant, with improved hematological tolerability

- Well-tolerated safety profile across range of doses under consideration for future study
- Diarrhea is low grade and readily manageable with no associated treatment discontinuations due to diarrhea
- Potential best-in-class hematologic safety profile

n (%)	CDK4i + fulvestrant (240 – 600mg BID)			
	Dose escalation (N=37)		Dose optimization (n=61)[1]	
Patient with any TEAE	36 (97.3%)		54 (88.5%)	
Related TEAE	35 (94.6%)		51 (83.6%)	
≥ G3 TEAE	19 (51.4%)		14 (23%)	
≥ G3 Related TEAE	10 (20.7%)		12 (19.7%)	
TRAE leading to discontinuation	1 (2.7%)		1 (1.6%)	
Key AEs	**All Grade TEAE**	**Gr3+ TEAE**	**All Grade TEAE**	**Gr3+ TEAE**
Diarrhea	83.8%	5.4%	65.6%	8.2%
Anemia	13.5%	0%	11.5%	1.6%
Neutrophil count decreased	21.6%	16.2%	29.5%	8.2%
Platelet count decreased	5.4%	2.7%	6.6%	1.6%

[1] Patients included those who were randomized by DCO: 26MAY2025

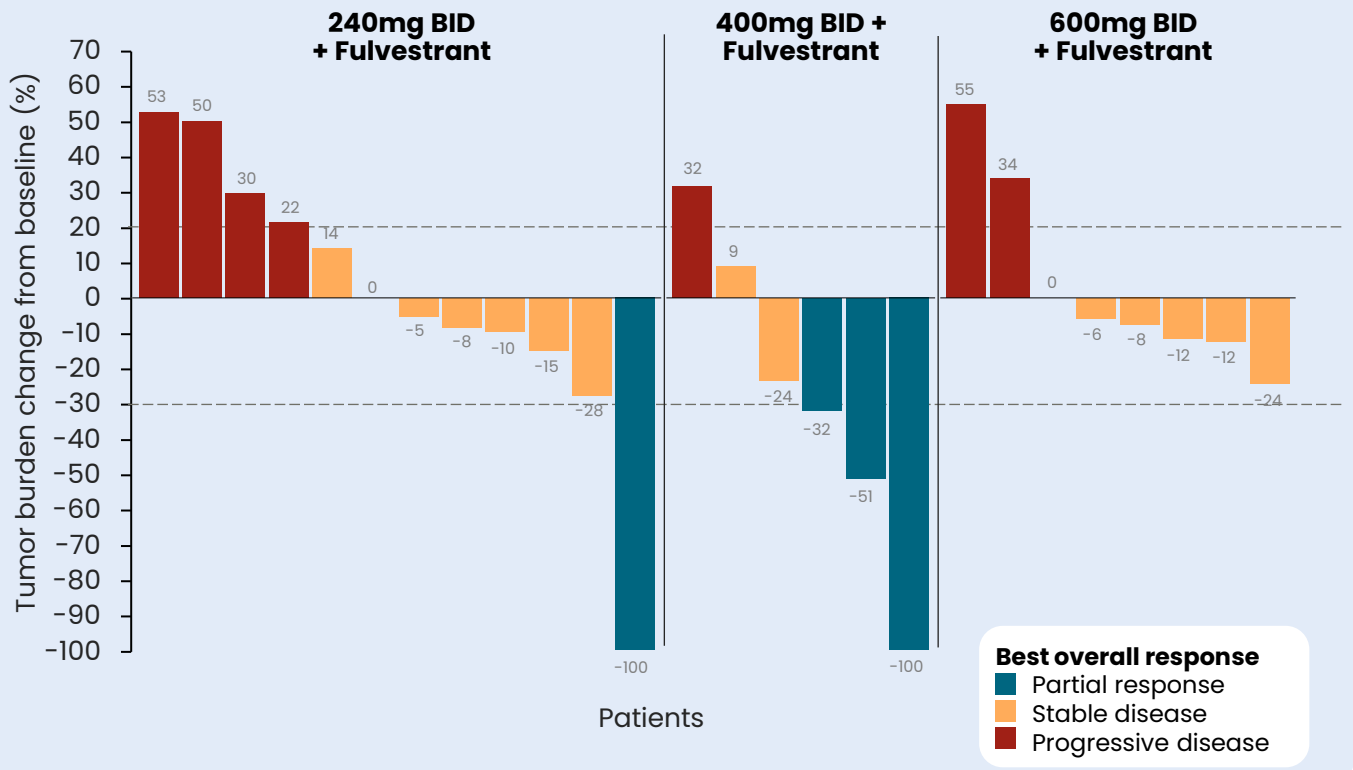
BGB-43395 (CDK4 inhibitor): combination with fulvestrant

Preliminary anti-tumor activity in extensively pretreated dose escalation patients

BGB-43395 + Fulvestrant Dose escalation	N=29; Breast Cancer	N=37; Total[1]
Number of prior lines of therapy in metastatic setting • Median (range) • >= 3 Lines	**4 (0-11)** **90%**	**4 (0-11)** **73%**
Prior chemotherapy (including ADC)	**100%**	**100%**
Prior endocrine therapy	**100%**	**84%**
Prior CDK4/6 inhibitor(s)	**93%**	**76%**
Median follow-up (months)	**3.0**	**2.7**
Objective response rate[2]	**11% (2 of 19)**	**15% (4 of 27)**

Breast cancer patients with 'bone only' metastatic disease are eligible for dose escalation but are not response evaluable (19 of 29 response evaluable)

Investigator-assessed best change from baseline in target lesion sum of diameters[1]



[1] Breast, endometrial, and ovarian cancer.

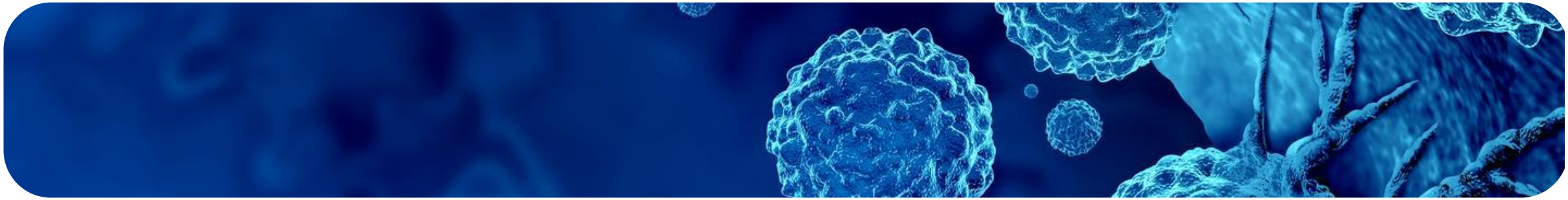
[2] Unconfirmed ORR among the response-evaluable patients

Waterfall plot includes data from 27 response evaluable patients in BGB-43395 + fulvestrant dose escalation

DCO: 26MAY2025

BGB-43395 (CDK4 inhibitor)

BeOne's CDK4i pre-clinical differentiation translates to meaningful clinical results



Key findings

- Strong pharmacodynamic effect across range of well-tolerated doses
- Potential best-in-class hematologic safety profile; diarrhea is low grade and readily manageable
- Emerging evidence of clinical efficacy in extensively pretreated, post-CDK4/6 patients at doses with pharmacodynamic activity
- Clear path to registration-enabling studies in next 6–12 months

Next steps

- Read-out of dose-optimization randomized expansions informs both dose selection and final proof-of-concept for registrational intent studies
- Elacestrant combination enables broader development with different ET backbones, such as oral SERDs in future

Target(i), target inhibitor; ET, endocrine therapy; SERD, selective estrogen receptor degrader

BG-C9074[1](B7-H4 ADC)

Potential first-in-class ADC for patients with B7-H4 expressing tumors

Target expressed in breast and gynecologic tumors with limited expression in normal tissues

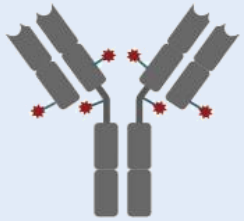
- Demonstrated dose-dependent in vivo efficacy good internalization, and bystander killing

Clinical Progress

- 95 patients enrolled in mono dose escalation and safety expansion as of June 2, 2025
- Responses noted across multiple tumor types and at various dose levels with manageable toxicity
- Tumor-specific expansion cohorts to begin enrollment 3Q25

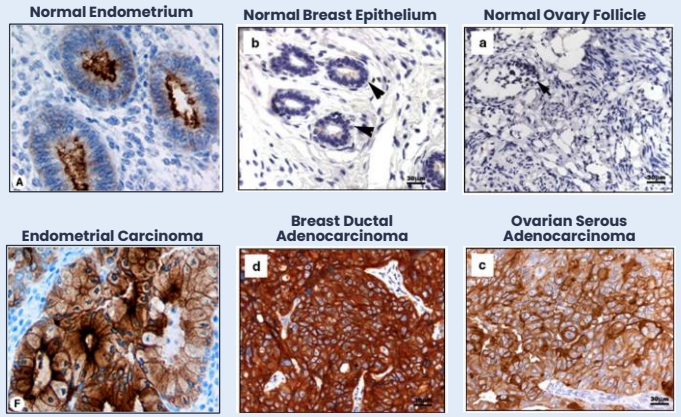
First data disclosure at ASCO 2025 with clinical updates today

Robust and clinically validated drug linker design differentiates our molecule



- Non-P-gp substrate[2] TOP1i payload
- Strong bystander effect
- DAR6 to balance efficacy and toxicity

High B7-H4 expression in tumor tissue of breast, endometrial, ovarian cancer[3]



[1] BG-9074 in licensed from Duality named DB-1312

[2] P-glycoprotein (P-gp) is a protein that can cause multidrug resistance (MDR) in cancer cells by preventing the uptake of many drugs (substrates), including anticancer drugs. Not being a P-gp substrate reduces drug resistance

[3] Clin Cancer Res. 2023 Mar 14;29(6):1086-1101

BG-C9074 (B7-H4 ADC): early clinical data

Well-tolerated monotherapy across clinically relevant dose levels

n (%)	Safety profile at selected monotherapy dose levels				
	4mg/kg (n=23)	5mg/kg (n=7)	6mg/kg (n=26)	6.5mg/kg (n=12)	**Total[1]** (n=68)
Patients with any TEAE	**22 (95.7%)**	**7 (100%)**	**25 (96.2%)**	**12 (100%)**	**66 (97.1%)**
Related	21 (91.3%)	7 (100%)	24 (92.3%)	11 (91.7%)	**63 (92.6%)**
Related >= Grade 3	2 (8.7%)	0 (0%)	10 (38.5%)	5 (41.7%)	**17 (25%)**
Related serious	1 (4.3%)	1 (14.3%)	3 (11.5%)	2 (16.7%)	**7 (10.3%)**
Related leading to dose modification	1 (4.3%)	0 (0%)	9 (34.6%)	3 (25%)	**13 (19.1%)**
Selected AE ≥Grade 3					
Neutropenia	1 (4.3)	0 (0.0)	7 (26.9)	5 (41.6)	**13 (19.1)**
Thrombocytopenia	0 (0.0)	0 (0.0)	4 (15.3)	2 (16.7)	**6 (8.8)**
Leukopenia	0 (0.0)	0 (0.0)	2 (7.6)	1 (8.3)	**3 (4.4)**
Nausea	0 (0.0)	0 (0.0)	2 (7.7)	0 (0.0)	**2 (2.9)**

- Most common TEAEs: nausea (low grade), neutropenia, and fatigue
- Most common Gr≥3 TEAE: neutropenia and thrombocytopenia
- Six DLTs:
  - Fatigue (n=1)
  - Neutropenia (n=1)
  - Febrile neutropenia (n=2)
  - Platelet count decreased (n=2)
- No adverse events leading to drug discontinuation or death

[1] Include response evaluable patients in breast and gynecological cancers
DCO: 02JUN 2025
TEAE, treatment emergent adverse events; DLT, dose limiting toxicities

BG-C9074[1] (B7-H4 ADC): early clinical data

Compelling early antitumor activity observed in Breast and Gynecological Cancers

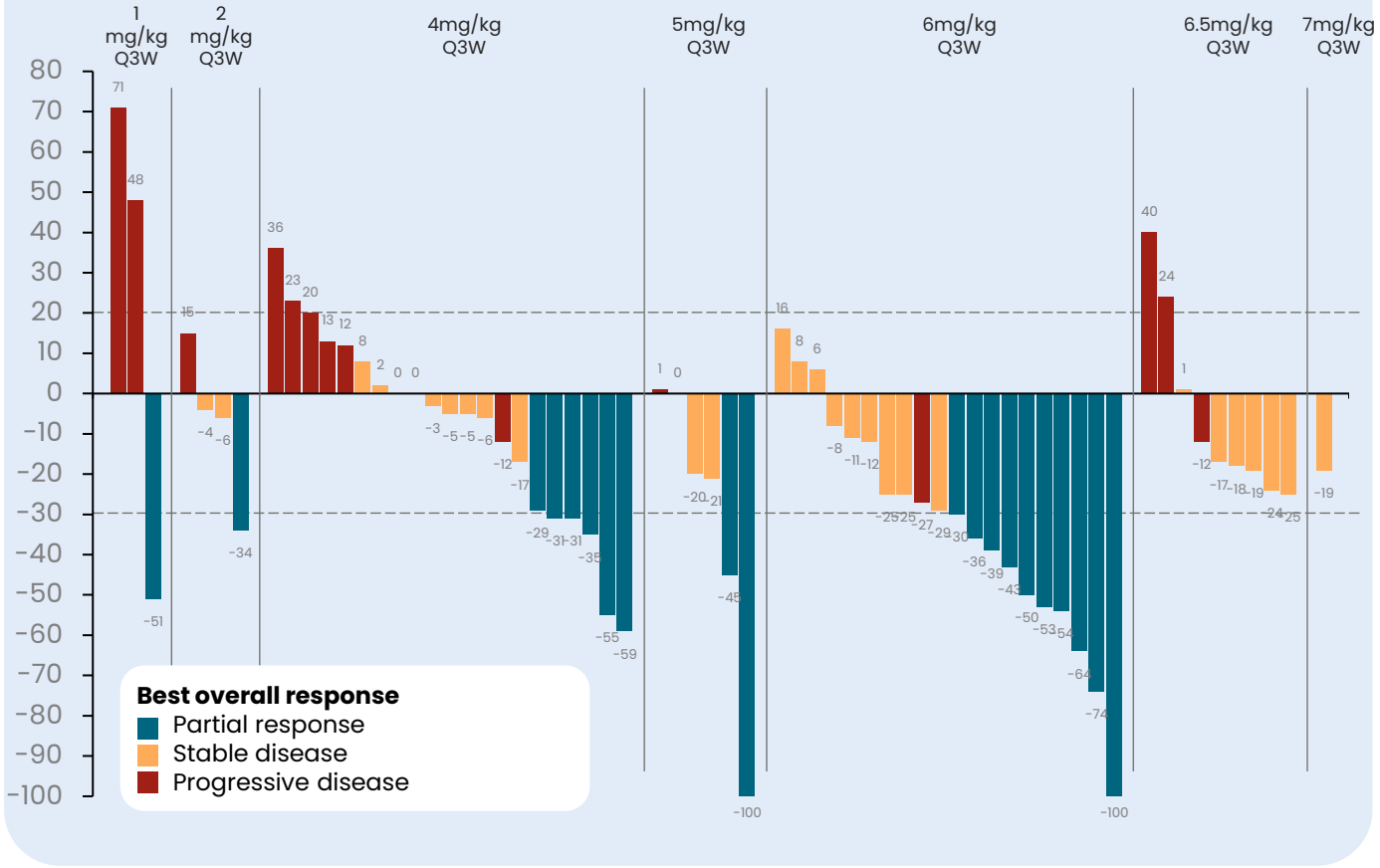
95 patients treated at 9 dose levels

- 6 enrolled tumor types; median 4 prior lines of therapy

Emerging dose-response relationship in breast, ovarian, and endometrial cancers[2]:

- Confirmed ORR 24%, unconfirmed ORR 29% among 68 efficacy evaluable patients
- 14 of 20 responses ongoing as of June 2, 2025
- Activity at 6mg/kg Q3W: confirmed ORR 43%, unconfirmed ORR 48%

Investigator-assessed best change from baseline in target lesion sum of diameters (BC and GYN)



[1] BG-9074 in licensed from Duality named DB-1312
[2] 83 patients enrolled with breast and gynecological cancers
DCO: 02JUN2025

BG-C9074 (B7-H4 ADC): biomarker data

Greater response rates in patients with higher levels of B7-H4 expression

Efficacy observed across a range of tumor types:

- Selecting patients with higher level B7-H4 expression enriches ORR
- Favorable safety profile at doses with strong early response rates

Tumor specific expansion cohorts in breast and gynecological malignancies to open in 3Q25

- Planning underway for registrational intent studies

Stronger efficacy in B7-H4 biomarker enriched patients

	BG-C9074	
	Breast and Gynecological	
N	68	33
Dose level	≥1mg/kg	≥1mg/kg
B7-H4 expression cutoff	None	High
uORR[1]	29.4%	42.4%

[1] Unconfirmed;
DCO: 02JUN2025

BG-68501 (CDK2 inhibitor)

Potent and selective - manageable safety and early signs of monotherapy activity in BC

Differentiated CDK2i vs. competitors with better potency, selectivity, CDK family kinome selectivity

Potential synergy with CDK4i in solid tumors, including CDK4/6i-resistant breast cancer

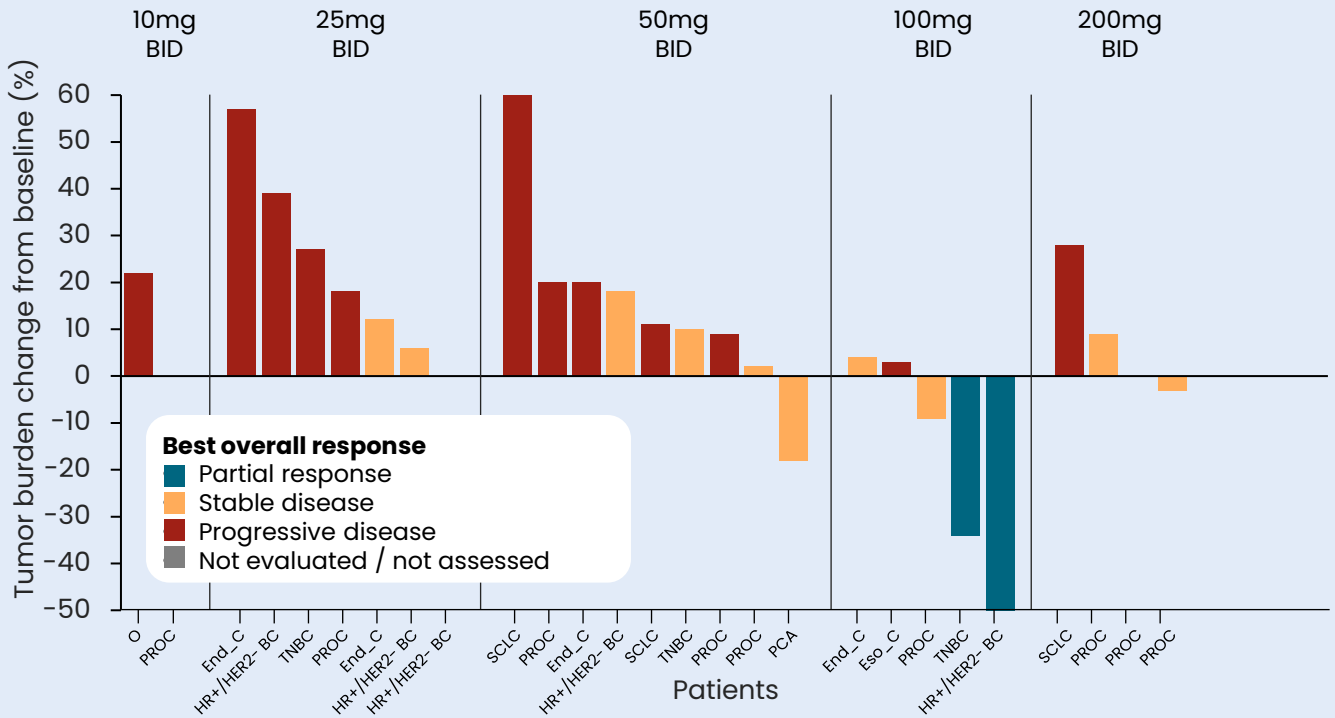
First data disclosure at ASCO 2025[1]

49 patients with pre-treated solid tumors received ascending doses of BG-68501

Preliminary safety: No DLTs; Most common AEs include Nausea and vomiting (61%), and fatigue (25%). GI toxicity improved with scheduled anti-emetics

Preliminary efficacy: 2 uPR (6%) (both BC), 15 (39%) SD in 33 patients (mFU of 2.9 months)

Investigator-assessed best change from baseline in target lesion sum of diameters



Response is unconfirmed. This plot shows post-baseline tumor assessments excluding patients without target lesions up to the initiation of new anti-cancer therapy or the first PD in the efficacy-evaluable population.

[1] Joshi et al ASCO Annual Meeting 2025, Abstract 3115

BC, breast cancer; BID, twice per day; End_C, endometrial cancer; Eso_C, esophageal cancer; HER2, human epidermal growth factor receptor 2; HR, hormone receptor; O, other: fallopian tube high grade serous; PCA, prostate cancer; PROC, platinum refractory or resistant ovarian cancer; SCLC, small cell lung cancer; TNBC, triple-negative breast cancer

Our breast cancer franchise is further extended by new and differentiated molecules

- **CDK2 CDAC**
  - potential for superior potency and improved toxicity profile compared to small molecule CDK2 inhibitors

- **KAT6A/B inhibitor**
  - encouraging preclinical activity for both monotherapy and in combination with fulvestrant when compared to a leading competitor, PF-07248144 (Pfizer)[1]

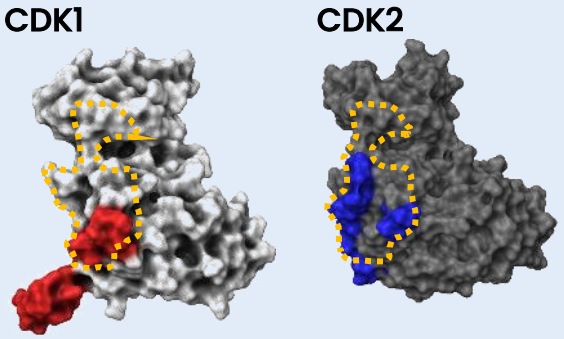
[1] Mukohara T. et. Al Nature Medicine 2024

BG-75098 (CDK2 CDAC)

Potential first-in-class CDK2 degrader with improved potency, selectivity, and PK

- Catalytic MoA to elicit superior target inhibition over small molecule inhibitors
- Much improved CDK1 selectivity compared to small molecule inhibitors due to additional selectivity provided by tertiary complex formation with E3 ligase
- Long predicted $T_{1/2}$ to support QD dosing and better target coverage with flat PK at steady state
- On track to enter clinic by YE 2025

CDAC gains selectivity via unique interaction between CDK2 and E3



Predicted E3 and CDK1/2 interaction region
CDK1 specific region
CDK2 specific region

Selectivity comes from using rigid linkers that directs E3 binding to CDK2 specific regions, enabling stable interactions with CDK2 but not CDK1

BG-75098 presents exceptional potency and selectivity profile

Degradation assay[1]	CDK2 DC50 (nM)	CDK1 DC50 (nM)
BeOne CDK2 CDAC	**1-13**	**› 10,000**

Anti-proliferation assay[2]	CDK2 cellular IC50 (nM)	CDK1/2 selectivity (fold)
Pfizer CDK2i	380	6
Incyte CDK2i	156	5
BeOne CDK2 CDAC	**14**	**› 714**

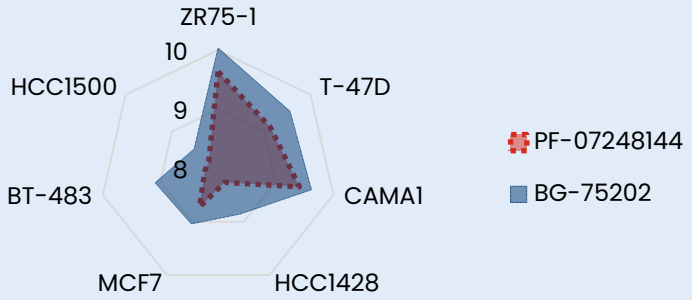
[1] Study was carried out in a variety of CDK2-dependent and independent cell lines
[2] Anti-proliferation assay in CDK2-dependent OVCAR3 and CDK1-dependent TOV21G cell lines

BG-75202 (KAT6A/B inhibitor)

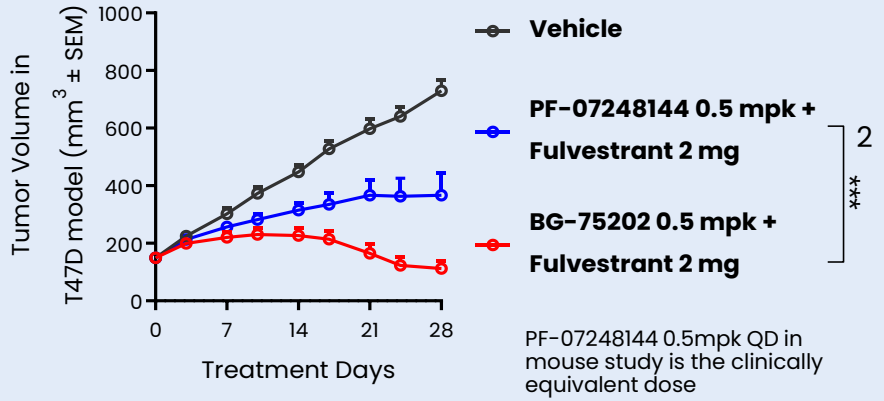
Potential best-in-class with opportunity to achieve better efficacy and safety

- Improved efficacy in both monotherapy and combination setting in preclinical models, comparing to PF-07248144[1]
- Over 10-fold higher selectivity against KAT7 comparing to PF-07248144, potentially lowering hematology tox
- Prioritize development in HR+HER2-BC in combination with endocrine therapy, with and without BG-43395
- On track to enter clinic by YE 2025

Greater potency in HR+ HER2- breast cancer cell lines (pIC50)



Stronger efficacy in combo with fulvestrant



PF-07248144 0.5mpk QD in mouse study is the clinically equivalent dose

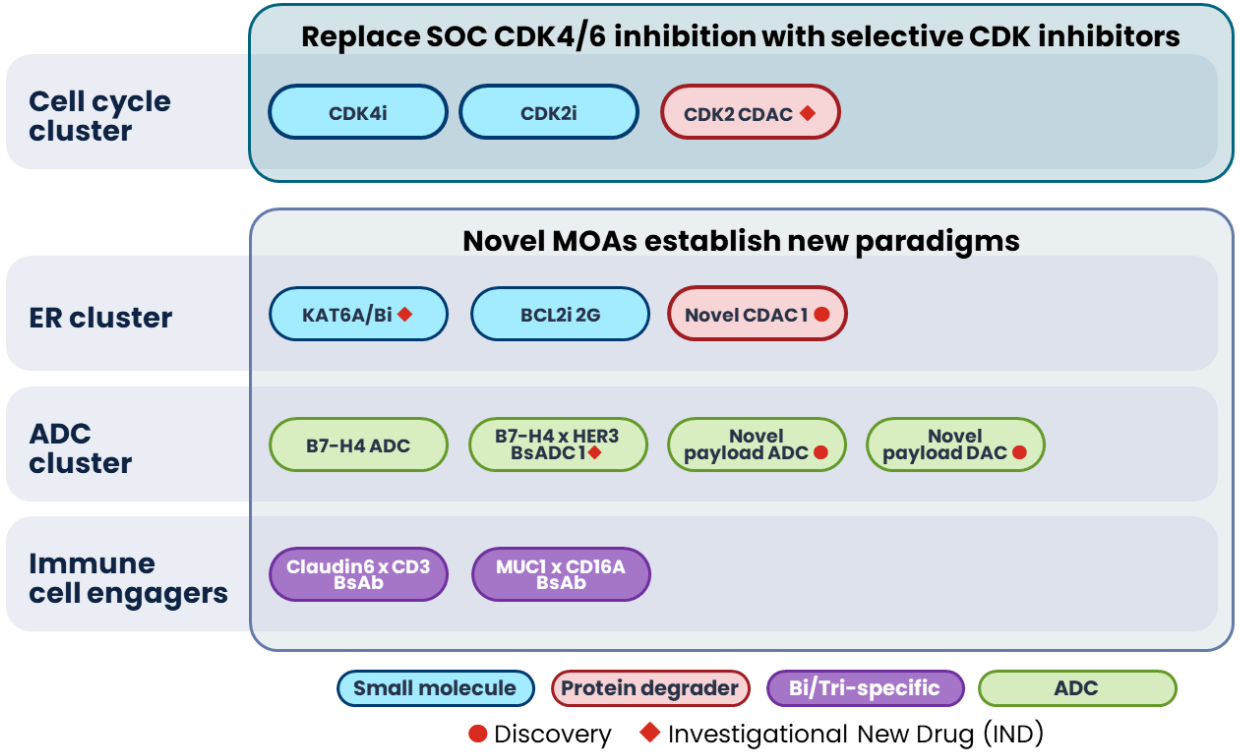
[1] PF-07248144 is KAT6A/B inhibitor from Pfizer

[2] *** P<0.001

Tremendous progress in first 18 months since establishing breast/gynecologic cancer franchise

Highly promising pipeline including several modalities (small molecule, CDAC and ADCs) with rapidly evolving dose-defining and POC data generation with potential for first pivotal study by YE (CDK4i)

- **CDK4i**: emerging data supports differentiated and potentially BIC profile; progressing towards Phase 3
- **B7-H4 ADC**: very promising efficacy and manageable toxicity; subsequent studies in OC, EC and BC in planning
- **BCL2i**: Phase 1b study ongoing to evaluate differentiated HR+ BC development opportunity
- **CDK2i:** manageable toxicity and early evidence of efficacy in advanced BC as monotherapy
- **CDK2 CDAC and KAT6A/Bi:** potentially differentiated based on improved selectivity and potency



Target(i), target inhibitor; CDAC, chimeric degradation activating compound; ADC, antibody drug conjugate; BsADC, bispecific ADC; BsAb, bispecific antibody

Lung cancer franchise

Building an industry-leading lung cancer franchise

Broad and deep pipeline with proprietary, innovative and intentionally designed molecules

Lung

- PanKRASi
- MTA Cooperative PRMT5i
- MAT2Ai
- B7-H3 ADC
- CEA ADC
- EGFR x MET x MET TsADC
- EGFR CDAC
- EGFR x MET x MET TsAb

MTAP-del cluster

- MTA Cooperative PRMT5i
- MAT2Ai

EGFRm cluster

- EGFR CDAC
- EGFRxMETxMET TsAb
- EGFRxMETxMET TsADC ◆

KRASm cluster

- panKRASi
- RAS (On)i ◆
- KRAS CDAC ●
- RAS (ON) G12Di ●
- RAS (ON) ADC ●

ADC cluster

- B7-H3 ADC
- CEA ADC
- ADAM9 ADC ◆
- mADC 1-2 ●
- EGFRxMETxMET TsADC ◆
- Novel Bs/TsADC 1-4 ●
- Novel payload ADC 1-2 ●

Small molecule | Protein degrader | Bi/Tri-specific | ADC

● Discovery ◆ Investigational New Drug (IND)

Target(i), target inhibitor; CDAC, chimeric degradation activating compound; ADC, antibody drug conjugate; BsADC, bispecific ADC; TsADC, trispecific ADC; BsAb, bispecific antibody; TsAb, trispecific antibody

BGB-58067 (PRMT5i) and BG-89894 (MAT2Ai)

Synergistic combination between MTA-cooperative PRMT5 inhibitor and MAT2A inhibitor

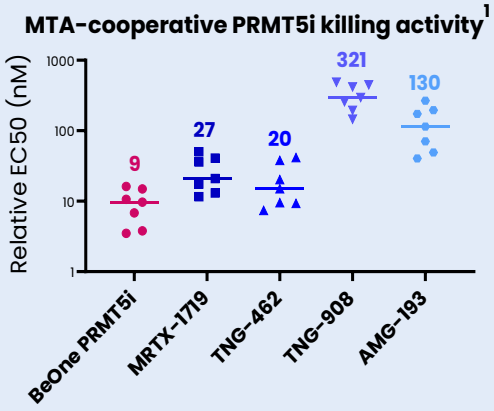
Only company with both clinical stage PRMT5i and MAT2Ai

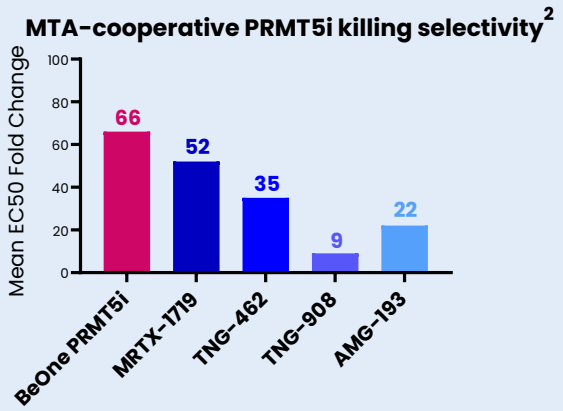
- Potential best-in-class PRMT5i with superior potency, selectivity, and brain penetration

Clinical progress

- Both programs in monotherapy dose escalation
- PRMT5i shows better than expected, dose dependent PK profile
- Favorable safety and tolerability profile with no DLT observed
- Early responses observed at the second dose level

BeOne PRMT5i shows higher potency and selectivity

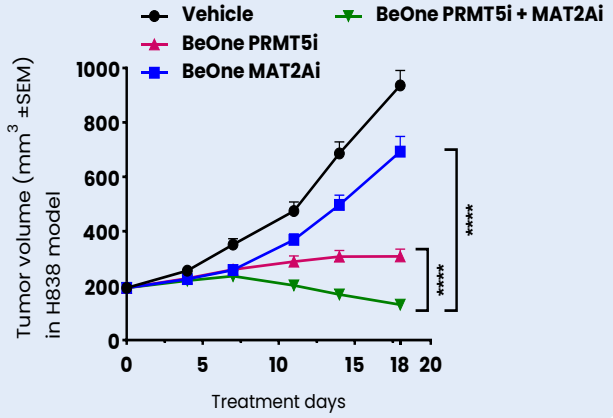




BeOne PRMT5i and MAT2Ai are highly brain penetrative

Compound	BGB-58067	BG-89894
Kpuu, CSF[3]	**55%**	**102%**

BeOne PRMT5i + MAT2Ai induce robust efficacy in animal model



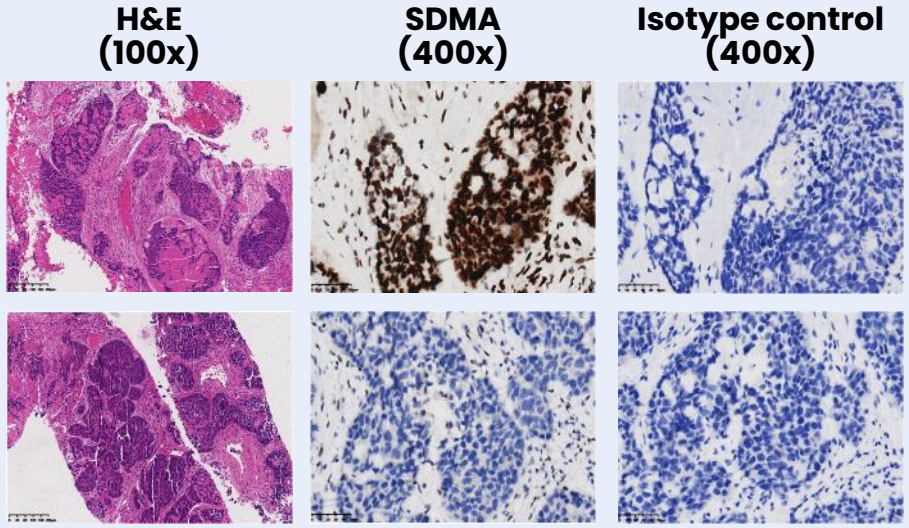
[1] Different dots in the "Tumor Cells" panel indicate different tumor cell lines. Del, deletion;
[2] Mean EC50 fold change of cell killing in 7 $MTAP^{DEL}$ and 2 $MTAP^{WT}$ cell lines; **** $p<0.0001$
[3] Kpuu,CSF represents a measurement of how well a drug moves from the bloodstream into the CSF in cynomolgus monkeys, used for assessment of blood-brain penetration

BGB-58067 (PRMT5i): pharmacodynamic data

Target engagement is confirmed by rapid SDMA[1] reduction in tumor and plasma

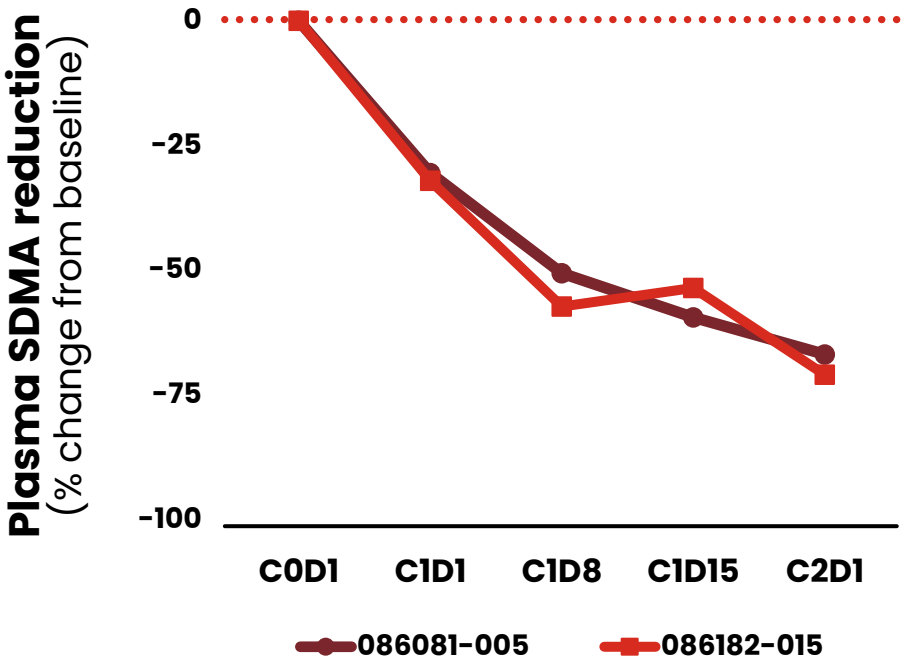
Target engagement with MTAP-del vs. normal cells in the tumor tissue of MTAP-del ESCC patient

(dose level 1; paired lung biopsy)



	Tumor Cell SDMA H-score	Stromal cell SDMA+ percentage
Baseline	300	100%
C2D1	0	90%

Rapid plasma SDMA reduction observed at dose level 1 (n=2)



[1] SDMA, symmetrical dimethylization, reduced levels particularly in MTAP-deleted cells can be associated with increased protein stability, potentially promoting tumor growth and metastasis

BGB-58067 (PRMT5i): clinical safety and efficacy data

Favorable safety profile with early efficacy signals

Safety

- **Favorable safety profile observed in 27 enrolled patients across four dose levels**
  - Most common TRAEs were nausea (18.5%) and anemia (11.1%)
  - No significant hematological toxicity
  - No DLTs, ≥ Gr3 treatment-related AEs, serious treatment-related AEs, or TRAEs leading to discontinuation/modification

Efficacy

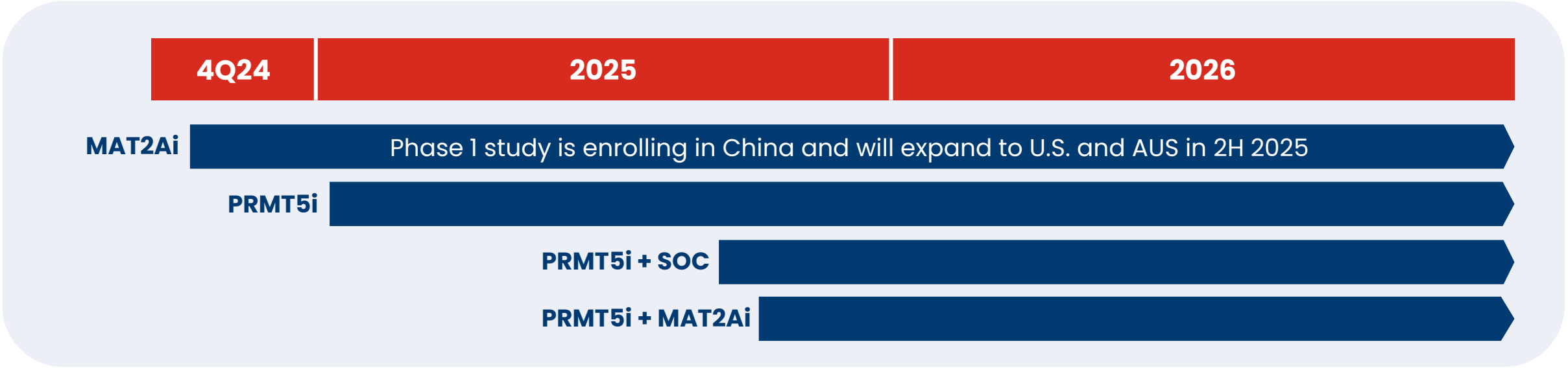
- Target efficacious range achieved at second dose level due to better than anticipated clinical PK
- **Three objective responses[1] in histologically distinct tumor types** at second dose level

[1] Responses - 1 confirmed, 2 unconfirmed, all ongoing
DCO: 29MAY2025

BGB-58067 (PRMT5i) and BG-89894 (MAT2Ai)

BeOne is only company with both PRMT5i and MAT2Ai in the clinic

Clinical stage	BeOne	Amgen	BMS	IDEAYA	AstraZeneca
PRMT5i	✓	✓	✓	X	✓
MAT2Ai	✓	X	X	✓	X



The bars represent phase 1 study timelines; the start of the bar represents the first patient in for the cohort(s)

BG-60366 (EGFR CDAC)

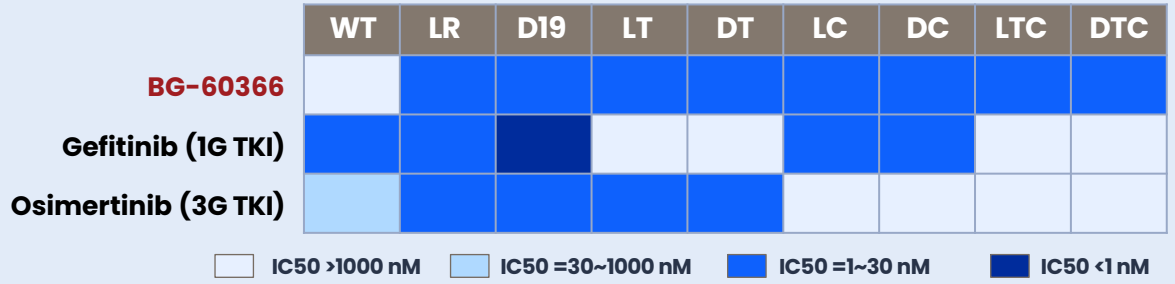
Truly differentiated MoA to completely, selectively abolish EGFR signaling

- Highly potent against all typical EGFR mutations and major resistant forms
- Sparing wild-type EGFR and good proteomic selectivity
- Deep target degradation and strong efficacy in animal models

Clinical progress

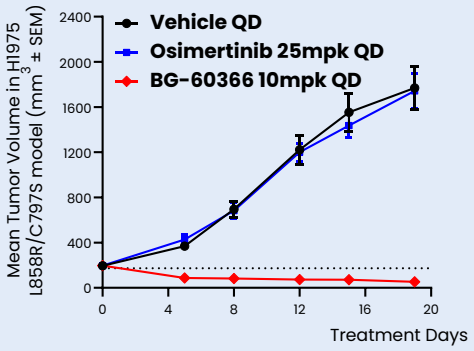
- In mono dose escalation
- Encouraging PK profile with good oral absorption (T_{max}=~4 hours), high exposure and long elimination ($T_{1/2}$ ~4.8 days)
- BG-60366+3G TKI combo planned to start in 2H25

Broadest EGFR mutation coverage w/o hitting WT EGFR

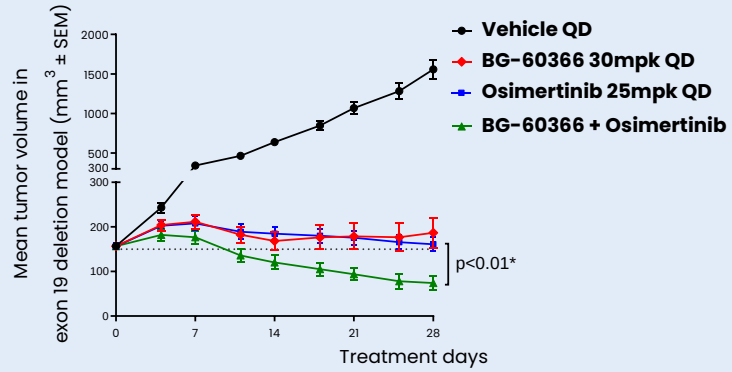


Robust efficacy in both osimertinib-sensitive and resistant models

Address osimertinib resistance



Deeper response when combined with osimertinib



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

BG-T187 (EGFRxMETXMET TsAb)

Best-in-class potential EGFRxMETxMET dual targeting TsAb with unique antibody design

- Superior and optimal MET inhibition with biparatopic MET antibody construct
- Pre-clinical data suggest stronger efficacy than amivantamab in MET-driven models
- Potential lower EGFR on-target skin toxicity due to weaker killing activity of primary keratinocyte[1] than amivantamab

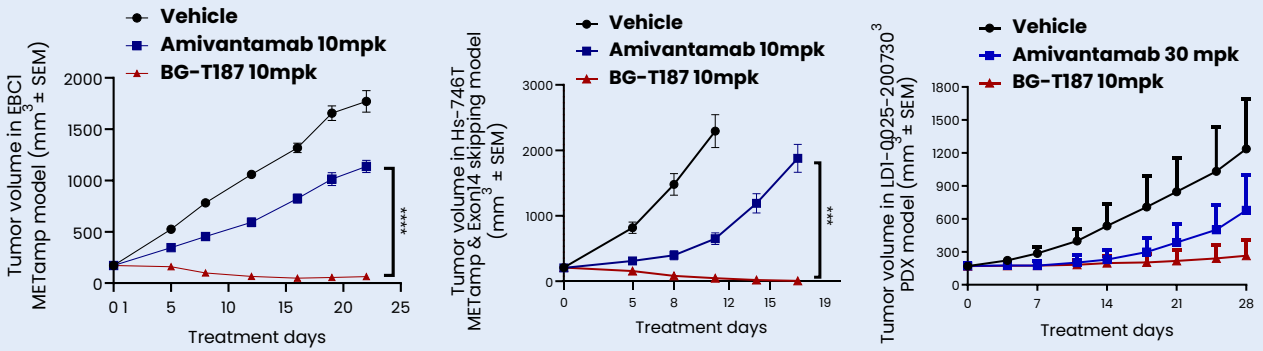
Clinical progress

- In mono dose escalation
- Subcutaneous formulation planned to enter clinic in 3Q25

Best-in-class cellular profile among EGFR x METx MET dual targeting Ab[2]

EGFR x MET dual targeting Ab	BG-T187	Amivantamab
Strong EGFR signaling inhibition	✓	✓
Strong MET signaling inhibition	✓	X
Weak primary keratinocyte killing	✓	X

Superior efficacy in MET-driven and TKI resistant xenograft models

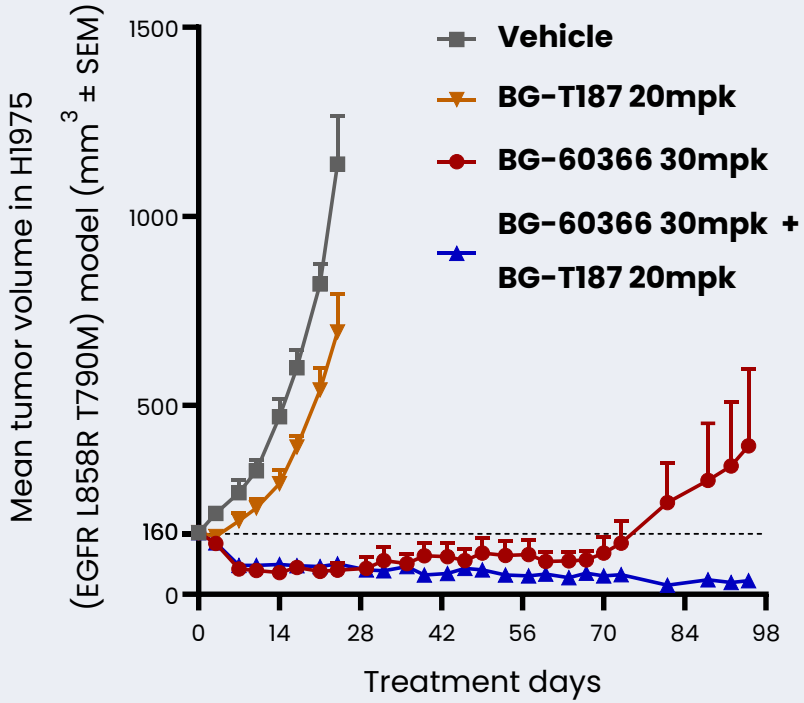


[1] HEKn, primary human epidermal keratinocytes isolated from neonatal foreskin, in the cellular study;
[2] Biosimilars of competitors' EGFR x MET antibodies were used;
[3] Osimertinib-resistant model, MET IHC 3+; EGFR L858R; ; ***, p<0.001, ****, p<0.000
The clinical significance of pre-clinical data has not been established
TsAb, trispecific antibody

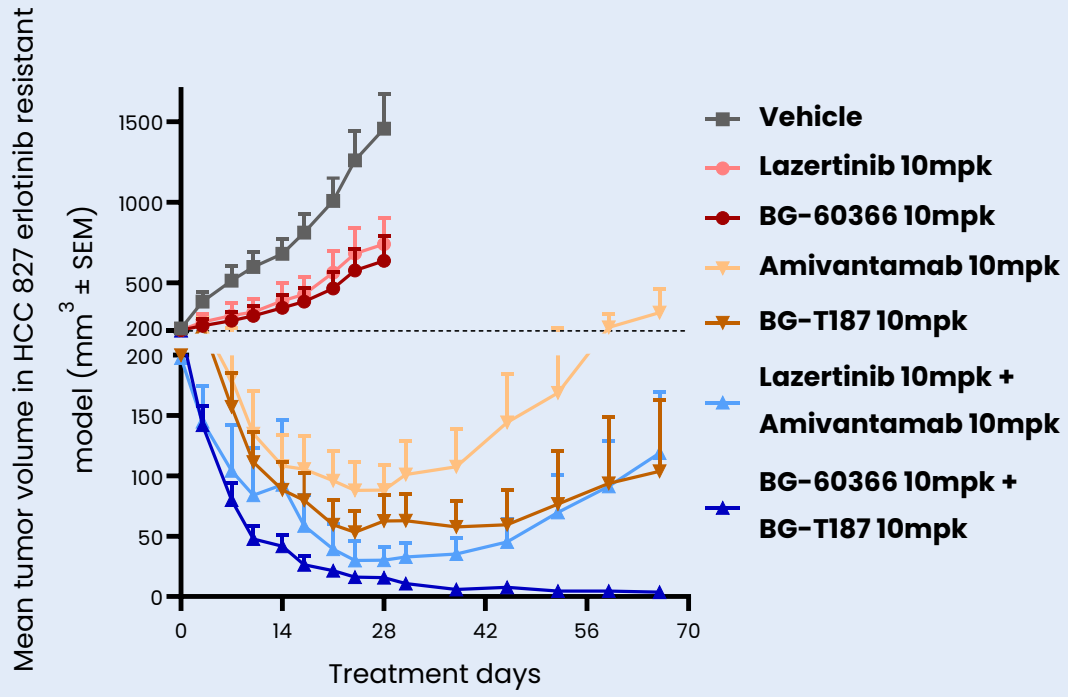
BG-60366 (EGFR CDAC) and BG-T187 (EGFRxMETxMET TsAb)

Combination generates durable response in both EGFR TKI-sensitive and resistant models

EGFR TKI-responsive model



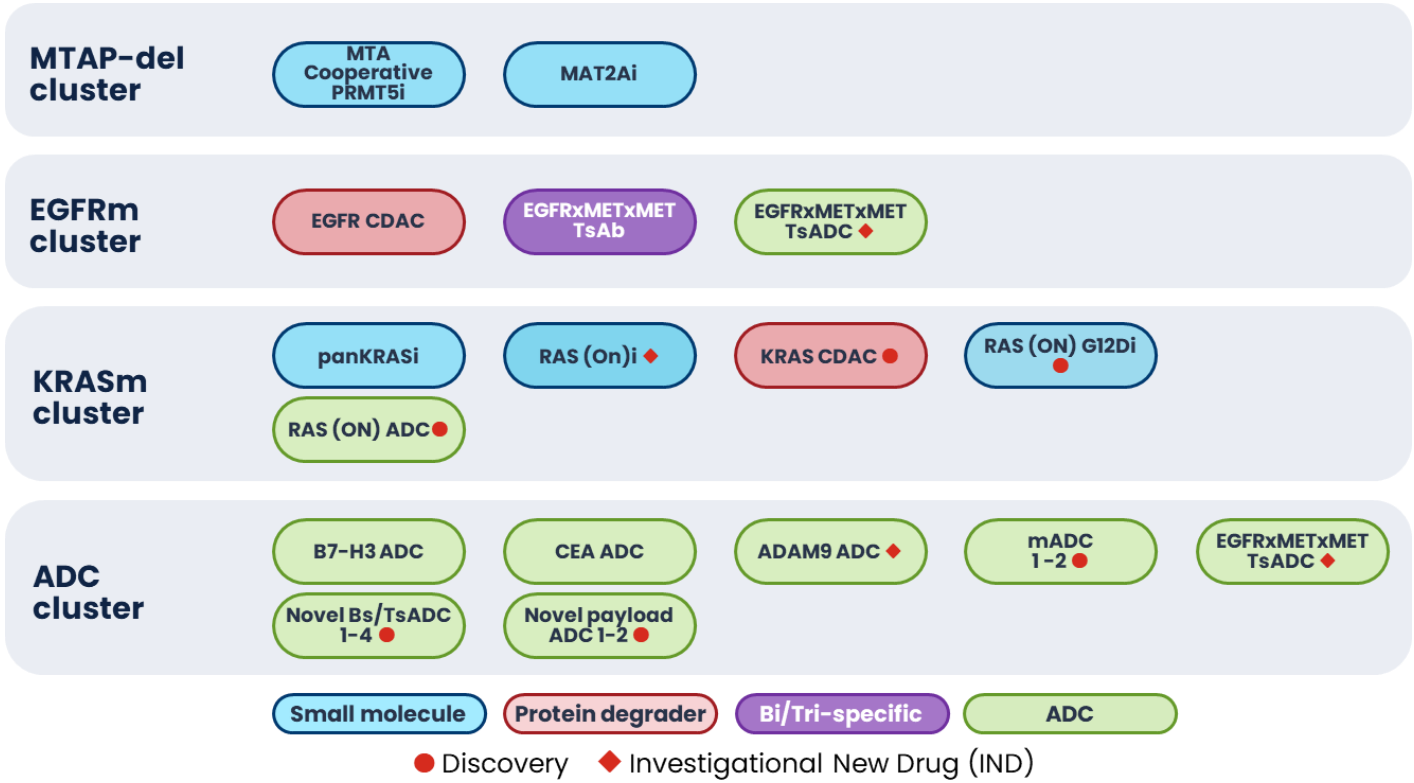
EGFR TKI-resistant model



TKI, tyrosine kinase inhibitor

Broad and deep lung cancer portfolio with diverse and differentiated assets

- **MTAP-del cluster**: PRMT5i and MAT2Ai are foundational molecules for MTAP-del NSCLC and other MTAP-del tumors. Early data support differentiated and best-in-class profiles
- **EGFR cluster**: differentiated and FIC molecules provide opportunity for substantial patient impact via in-portfolio combination and in combination with 3rd generation TKI
- **Novel ADCs**: multi-specific ADCs have potential to both broaden eligible population and improve therapeutic window. Novel payloads will further advance tumor-selective targeting



Target(i), target inhibitor; CDAC, chimeric degradation activating compound; ADC, antibody drug conjugate; BsADC, bispecific ADC; TsADC, trispecific ADC; BsAb, bispecific antibody; TsAb, trispecific antibody

Gastrointestinal cancer franchise

Designing and delivering innovation in GI oncology

Gastrointestinal

- KRASi
- MTA Cooperative PRMT5i
- MAT2Ai
- FGFR2b ADC
- CEA ADC
- GPC3 x 4-1BB BsAb
- MUC1 x CD16A BsAb

KRAS cluster	PanKRASi	RAS (On)i ◆	KRAS CDAC ●	RAS (ON) G12Di ●	RAS (ON) ADC ●
MTAP-del cluster	MTA Cooperative PRMT5i	MAT2Ai			
EGFR cluster	EGFRxMETxMET TsAb	EGFRxMETxMET ADC ◆			
ADC cluster	CEA ADC	FGFR2b ADC	B7-H3 ADC	Novel Bs/Ts ADC 1-6 ●	Novel Payload ADC ●
Immune cell engager	GPC3 x 4-1BB BsAb	MUC1 x CD16A BsAb			

Small molecule | Protein degrader | Bi/Tri-specific | ADC

● Discovery ◆ Investigational New Drug (IND)

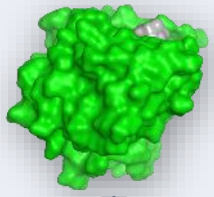
Target(i), target inhibitor; CDAC, chimeric degradation activating compound; ADC, antibody drug conjugate; BsADC, bispecific ADC; TsADC, trispecific ADC; BsAb, bispecific antibody; TsAb, trispecific antibody

Targeting KRAS biology to maximize clinical benefit

Combinations to generate greater therapeutic benefits

RAS-GDP complex

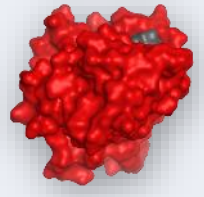
OFF



1G KRASi

2G KRAS CDAC — Improve potency

RAS-GTP complex



ON

1G RAS(ON)i — RAS(ON) G12Di

2G RAS(ON) ADC — Mitigate on-target toxicity

Clinical

IND

Discovery

New molecules for KRAS pathway poised to deliver greater patient impact

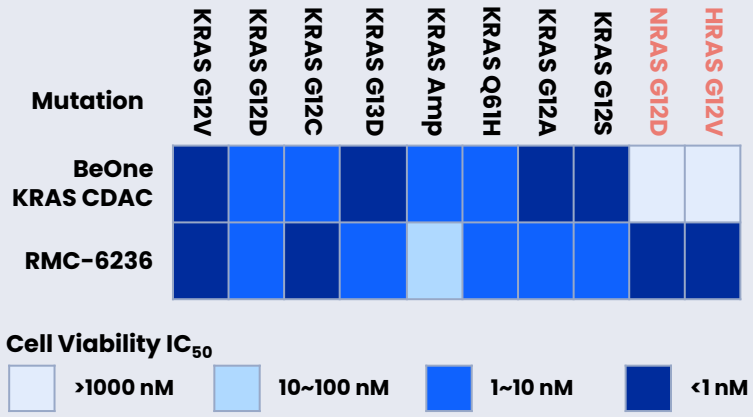
KRAS CDAC

- Highly potent and selective, broad spectrum for KRAS mutations
- Good pharmacological property, supporting daily dosing
- Near complete target degradation at efficacious dose
- Expected to enter clinic 2H26

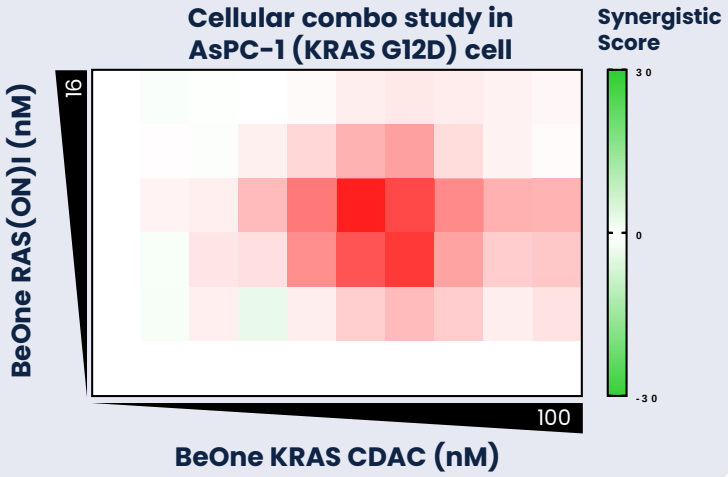
Brain-penetrant RAS(ON)i

- High brain penetration, 39% Kpuu[1] in mice
- Significantly lower anticipated efficacious dose enabled by better potency and PK properties
- Expected to enter clinic by 2H26

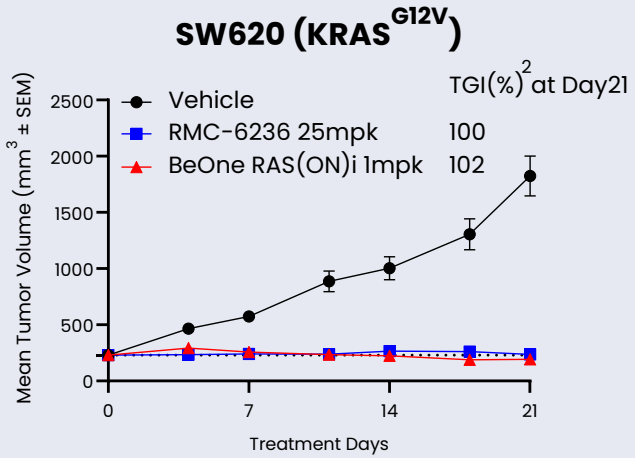
KRAS CDAC exhibits superior potency and selectivity over RAS(ON)i



Marked increase of potency upon KRAS CDAC and RAS (ON)i combination



BeOne RAS (ON)i produces robust efficacy at a much lower dose level



[1] Kpuu represents a measurement of how well a drug moves from the bloodstream into the CSF in cynomolgus monkeys, used for assessment of blood-brain penetration
[2] TGI, tumor growth inhibition

BG-C137 (FGFR2b ADC)

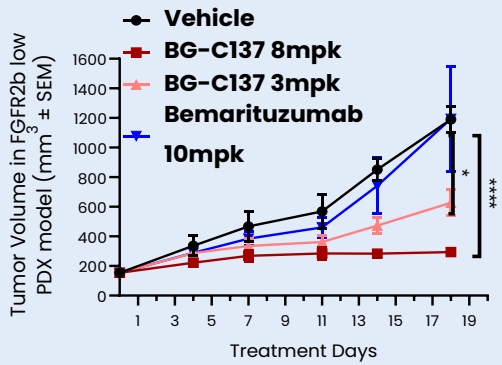
Potential first-in-class with best-in-modality design

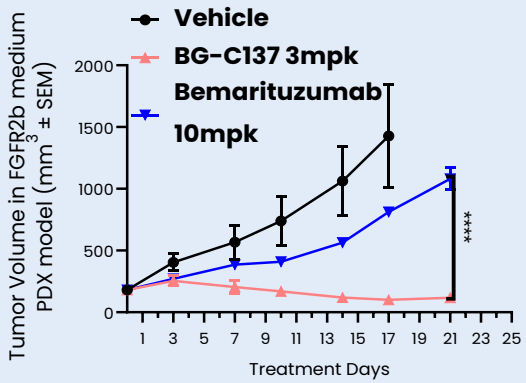
- Superior efficacy over bemarituzumab[1] in animal models with heterogeneous FGFR2b expression
- Spares corneal toxicity via unique epitope with weaker ligand blockade

Clinical progress

- In 4th dose level cohort of monotherapy dose escalation
- Objective responses observed with no corneal or ocular AEs reported to date

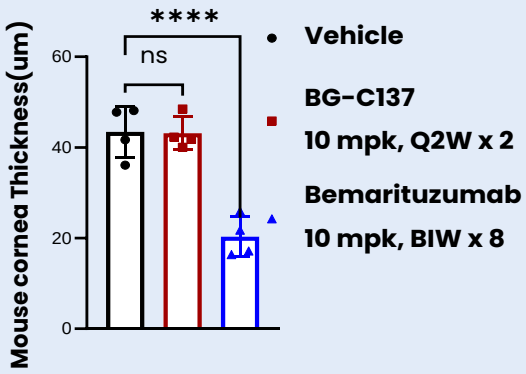
BG-C137 produces stronger efficacy in xenograft models[2]





BG-C137 has lower FGFR2b ligand blocking activity and does NOT induce corneal tox in animal studies[3]

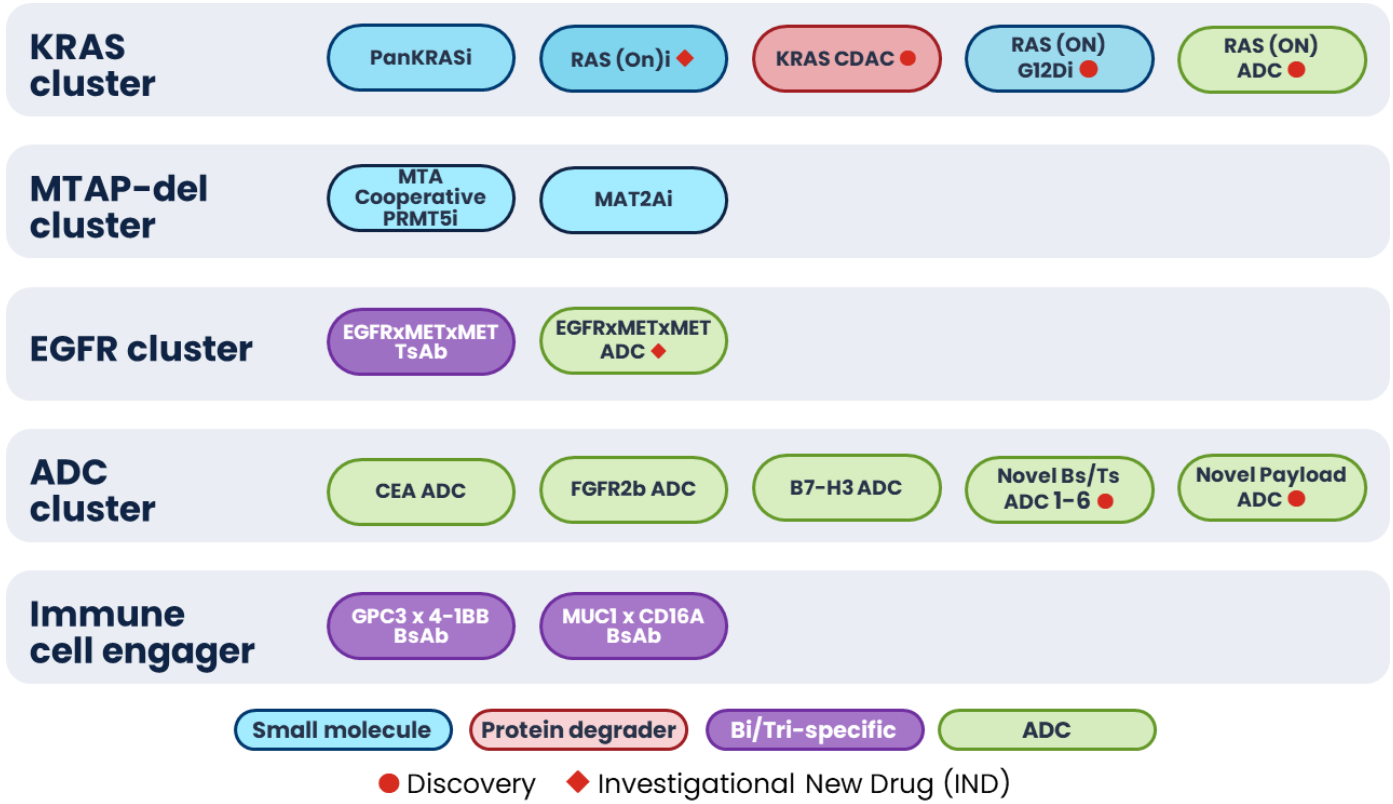
Molecule	Block FGF7-FGFR2b	Block FGF10-FGFR2b
BG-C137	Weak	NO
Bemarituzumab	Strong	Strong



[1] Bemarituzumab is FGFR2b mAb from Amgen, whose comboination with chemotherapy has shown Phase 2 efficacy in gastric cancer but 26% rate of treatment discontinuation due to on-target corneal toxicity
[2] BG-C137 was dosed once, bemarituzumab was dosed BIW
[3] No ocular adverse findings in monkey GLP TOX study

Exciting GI cancer portfolio with deep focus on targeting RAS pathway

- **KRAS**: deep commitment to developing differentiated RAS pathway targeting therapies via comprehensive multi-modality approach
- **MTAP-del**: exciting combination opportunity with PRMT5i and MAT2Ai for MTAP-deleted pancreatic cancer
- **ADCs**: multiple first-in-class (FIC) and/or best-in-class opportunities (BIC). FGFR2b ADC emerging profile indicates superior safety profile with FIC potential in gastric cancer



Target(i), target inhibitor; CDAC, chimeric degradation activating compound; ADC, antibody drug conjugate; BsADC, bispecific ADC; TsADC, trispecific ADC; BsAb, bispecific antibody; TsAb, trispecific antibody

From innovation to impact: executing a strategic and rapidly progressing solid tumor portfolio to help more patients

- **Completely reshaped** clinical-stage solid tumor portfolio in only two years enabling in-portfolio combinations across three disease areas
- **Compelling continued innovation** with two to three additional clinical-stage molecules in each disease area every year
- **Accelerate development** with unparalleled speed to POC with multiple Phase 3 trial initiations planned in 2026 for CDK4i and B7-H4 ADC, and possibly PRMT5i and FGFR2b ADC

Summary

Lai Wang
Global Head of R&D

BGB-45035 (IRAK4 CDAC)

First-in-class with deep and sustained IRAK4 degradation

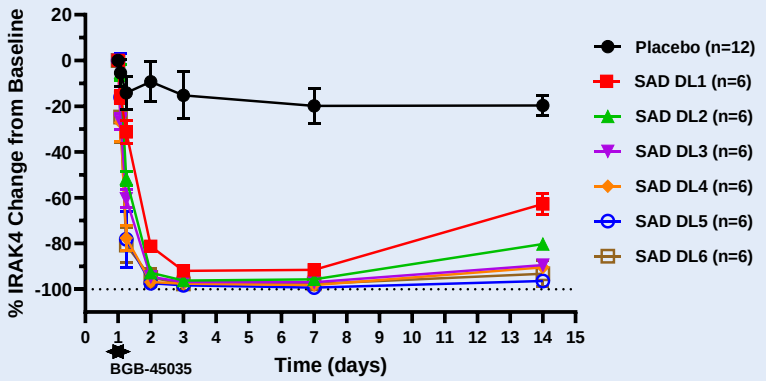
- Designed with best-in-class properties:
  - Faster and deeper IRAK4 degradation with stronger cytokine inhibition compared to KT474 in preclinical models
  - No QT prolongation

Clinical progress

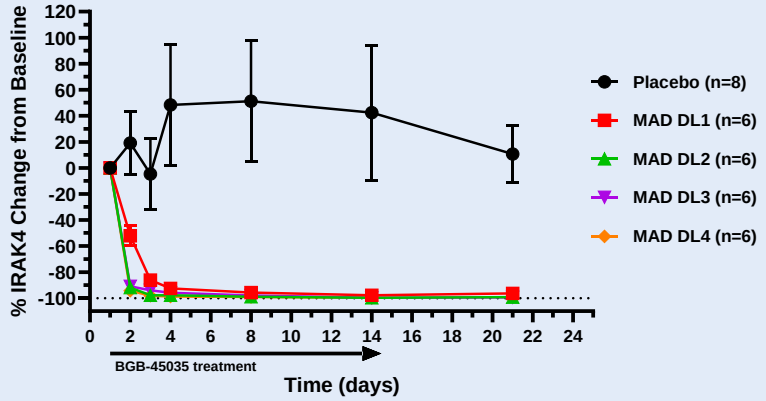
- SAD and MAD completed
- Phase 1b in prurigo nodularis (PN)/atopic dermatitis initiated (AD)
- Long half-life in human (60-96 hours)
- Complete and sustained IRAK4 degradation in peripheral blood up to 7 days after a single dose of BGB-45035
- Phase 2 to start in 2H25; POC for tissue IRAK4 degradation expected before YE 2025

IRAK4 degradation in blood

SAD



MAD



MAD, multiple ascending dose; SAD, single ascending dose

We have demonstrated the potential for BeOne's innovative oncology pipeline to impact patient lives

Industry leadership in R&D

01 Our **sheer research output** and number of **clinical catalysts** are **unprecedented**; we are at **an inflection point** that our capabilities are coming together to create a unique R&D model; we are pioneering **first-in-class** and **best-in-class** therapies

02 **BRUKINSA is just the start.** We have thoughtfully considered all permutations of how the CLL field will evolve and have designed for the best solution regardless with **sonrotoclax and BTK-CDAC**

03 We are approaching our priority solid tumor histologies, **breast/gynecological cancers, lung cancers and GI cancers**, with the **same mindset of serial innovation** as we did in hematology

Key late-stage catalysts in 2025 and 2026

Asset	Catalyst	H1 2025	H2 2025	2026
BRUKINSA	MANGROVE TN MCL Ph3 PFS interim analysis		●	
Sonrotoclax	CELESTIAL-TNCLL (301) Ph3 enrollment completion (+BRUKINSA)	✓		
	CELESTIAL-RRMCL (302) Ph3 initiation (+BRUKINSA)	✓		
	CELESTIAL-RRCLL (303) Ph3 initiation (+anti-CD20)	●		
	R/R MCL Ph2 data readout and AA submission if data support[1]	✓	●	
	R/R CLL Ph2 data readout and CN AA submission	✓		
BTK CDAC (BTK CDAC)	CaDAnCe-302 R/R CLL vs. Investigator's Choice (IR/BR/VR) Ph3 initiation	✓		
	CaDAnCe-304 R/R CLL H2H vs. pirtobrutinib Ph3 initiation		●	
	CaDAnCe-101 R/R CLL Ph2 data readout - potentially pivotal			●
TEVIMBRA	1L ESCC U.S. approval	✓		
	1L ESCC and 2L ESCC JP approval	✓		
	1L SCLC EU approval	✓		
	1L NPC EU approval		●	
	Neo/adj NSCLC EU approval		●	
	1L GC subcutaneous formulation Ph3 initiation		●	
	1L GC JP approval			●
Zanidatamab[2] + TEVIMBRA	HERIZON-301 1L HER2+ GEA Ph3 readout		●	
IMDELLTRA® (Tarlatamab)[3]	2L SCLC Ph3 readout	✓		
	3L SCLC Ph2 readout	✓		

✓ **achieved** ● ● **planned**

[1] CN submission in H1 2025 complete, global submission in H2 2025 planned, [2] Zymeworks/Jazz collaboration, [3] Amgen collaboration

Key early-stage catalysts in 2025 and 2026

Asset	Catalyst	H1 2025	H2 2025	2026
CDK4i	POC Data	✓		
	2L HR+/HER2- mBC Ph3 initiation		●	
B7-H4 ADC[1]	POC Data	✓		
	Ph3 initiation			●
PanKRASi	POC Data		●	
EGFR CDAC	POC Data		●	
CDK2i[2]	POC Data		●	
B7-H3 ADC	POC Data		●	
CEA ADC	POC Data		●	
FGFR2b ADC	POC Data		●	
IRAK4 CDAC	POC Data*		●	
PRMT5i	POC Data		●	
PRMT5i + MAT2Ai[3] **combination**	POC Data			●
EGFRxMETxMET TsAb	POC Data			●

✓ achieved ● ● planned

[1] DualityBio collaboration, [2] Ensem collaboration, [3] CSPC collaboration
* Tissue PD
Note: Catalyst external data presentation subject to conference calendar

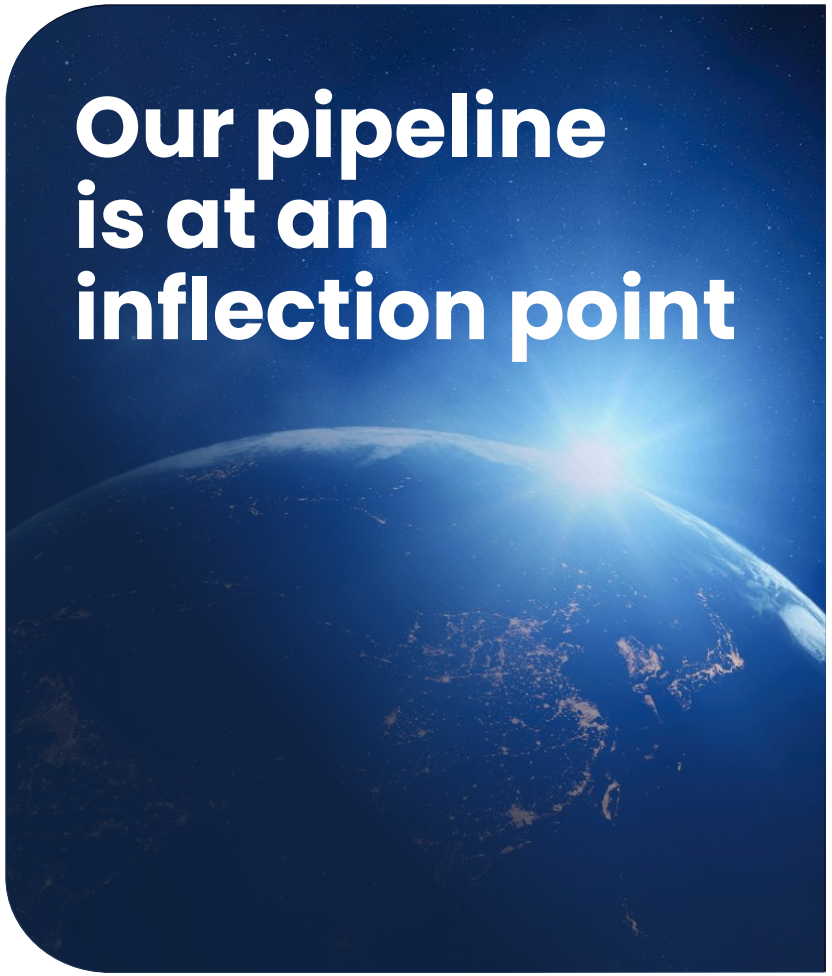
Q&A

BeOne management team and guest speakers

Closing

John V. Oyler
Co-Founder, Chairman and CEO

What you should take away from today



20 **Near-term milestones in the next 18 months, including:**

Registrational data readouts / potential approvals:

- Sonrotoclax (R/R CLL and R/R MCL Phase 2 AA*)
- BTK CDAC (R/R CLL Phase 2 readout - potentially registrational)

10+ POC data readouts across pipeline

Pivotal trial initiations:

- BTK CDAC vs. pirtobrutinib
- CDK4i and B7-H4 ADC

*AA – accelerated approval

Thank You

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BeOneMedicines.com

BeOne

Appendix

BeOne clinical pipeline
(snapshot of our entire pipeline)

Updated: 22 June 2025

Phase 1

Sonrotoclax — BCL2i
- 101 B-cell malignancies
- 102 B-cell malignancies
- 103 AML/MDS
- 105 R/R MM t(11;14)
- 108 Dose ramp-up

BGB-43395 — CDK4i
- 101/102 BC & Solid tumors

BGB-53038 — PanKRASi
- 101 Solid tumors

BG-C9074[1] — B7-H4 ADC
- 101 Solid tumors

BG-60366 — EGFR CDAC
- 101 Solid tumors

BG-58067 — MTA Coop. PRMT5i
- 101 Solid tumors

BG-89894[2] — MAT2Ai
- 101 Solid tumors

BGB-45035 — IRAK4 CDAC
- 101 Immunology & Inflammation

BG-68501[3] — CDK2i
- 101 BC & Solid tumors

BG-C354 — B7-H3 ADC
- 101 Solid tumors

BG-C477 — CEA ADC
- 101 Solid tumors

BG-C137 — FGFR2b ADC
- 101 Solid tumors

BG-T187 — EGFR x MET x MET TsAb
- 101 Solid tumors

BGB-16673 — BTK CDAC
- 101 B-cell malignancies
- 102 B-cell malignancies
- 104 B-cell malignancies
- 107 Chronic spontaneous urticaria**

BGB-21447 — BCL2i 2G
- 101 B-cell malignancies
- 102 Metastatic breast cancer

Xaluritamig[4] — STEAP1 x CD3 XmAb®
- 20180146 mCRPC

BGB-R046 — IL-15 prodrug
- 101 Solid tumors

BGB-B2033 — GPC3 x 4-1BB BsAb
- 101 Solid tumors

BGB-B3227 — MUC1 x CD16A BsAb
- 101 Solid tumors

BGB-26808 — HPK1i
- 101 Solid tumors

BGB-30813 — DGKζi
- 101 Solid tumors

BGB-A3055 — CCR8 mAb
- 101 Solid tumors

Tislelizumab — PD1 mAb
- 103 SubQ formulation

BGB-B455 — CLDN6 x CD3 BsAb
- 101 Solid tumors

Tarlatamab[4] — DLL3 x CD3 BiTE®
- 20240124 2L+ SCLC**
- 20230298 2L+ SCLC**

Phase 2

Zanubrutinib — BTKi
- 215 B-cell malignancies

BGB-16673 — BTK CDAC
- 101 R/R CLL
- 102 R/R CLL

Sonrotoclax — BCL2i
- 203 R/R WM
- 204 TN CLL/SLL

Blinatumomab[4] — CD3 x CD19 BiTE®
- 20190359 R/R BP-ALL

Tarlatamab[4] — DLL3 x CD3 BiTE®
- 20230273 3L SCLC
- 20240092 2L+ SCLC**

Phase 3

Zanubrutinib — BTKi
- 306 TN MCL
- 308 R/R MZL, R/R FL
- 309 pMN

BGB-16673 — BTK CDAC
- 302 R/R CLL
- 303 R/R CLL/SLL
- 304 R/R CLL/SLL**

Sonrotoclax — BCL2i
- 301 TN CLL
- 302 R/R MCL
- 303 R/R CLL/SLL**

Tislelizumab — PD1 mAb
- 310 1L UBC
- 314 R/R cHL

Pamiparib — PARPi
- 302 2L MTx gBRCAm PSOC

Zanidatamab[5] — HER2 BsAb
- 301 1L HER2+ GEA

Tarlatamab[4] — DLL3 x CD3 BiTE®
- 20210004 2L SCLC
- 20200041 1L ES-SCLC
- 20230016 LS-SCLC

Accepted Submissions
Major markets

Zanubrutinib — BTKi
- 114 Tablet formulation (EU)

Sonrotoclax — BCL2i
- 201 R/R MCL (CN)
- 202 R/R CLL (CN)

Tislelizumab — PD1 mAb
- 315 Neo/adj NSCLC (EU)
- 309 1L NPC (EU)
- Alternate dosing Q6W (US, CN)

Zanidatamab[5] — HER2 BsAb
- 203 HER2+ 2L BTC (CN)

Heme · Breast / Gyn · Other Cancers · Lung · GI · I&I

Accepted submissions shown for major markets US, CN, EU, JP
1) DualityBio collaboration, 2) CSPC collaboration, 3) Ensem collaboration, 4) Amgen collaboration, 5) Zymeworks/Jazz collaboration.
Please refer to our most recent 10-K filing for a full list of our commercial products, including in-licensed products, as well as commercial rights and collaboration details
** Trial is listed on clinicaltrials.gov, but may not have subjects enrolled

Acronyms (1 of 2)

1L	1st-line
2G	2nd generation
2L	2nd-line
3L	3rd-line
AA	Accelerated approval
ADC	Antibody drug conjugate
AI	Artificial intelligence
AML	Acute myeloid leukemia
APAC	Asia Pacific
ASCO	American Society of Clinical Oncology
ASH	American Society of Hematology
AV	Acalabrutinib + venetoclax
AVO	Acalabrutinib + venetoclax + obinutuzumab
B-Cell	B lymphocytes
B7-H3	Tumor associated antigen
B7-H4	Tumor associated antigen
BCL2	B-cell lymphoma 2
BD	Business development
BIC	Best-in-class
BID	Twice daily
BiTE	Bi-specific T-cell engager
BP-ALL	B-precursor acute lymphoblastic leukemia
BR	Bendamustine + rituximab
BRCA	"Breast Cancer" gene
BsAb	Bispecific antibody
BTK	Bruton's tyrosine kinase
CCR8	C-C motif chemokine receptor 8
CD19	Cluster of differentiation 19
CD3	Cluster of differentiation 3
CDAC	Chimeric Degradation Activation Compound
CDK2	Cyclin-dependent kinase 2
CDK4	Cyclin-dependent kinase 4
CEA	Carcinoembryonic antigen
cHL	Classical hodgkins lymphoma
CI	Confidence Interval
CIT	Chemoimmunotherapy
CLDN6	Claudin6
CLL	Chronic lymphocytic leukemia
CMR	CMR/Clarivate
CN	China
COVID-19	Coronavirus Disease 2019
CRC	Colorectal cancer
CRO	Contract research organization
CSPC (Collaboration)	CSPC Zhongqi Pharmaceutical Technology
del(11q)	Deletion of part of chromosome 11
del(17p)	Deletion of part of chromosome 17
DGKζ	Diacylglycerol kinase zeta
DLBCL	Diffuse large B-cell lymphoma
DLL3	Delta-like canonical notch ligand 3
DNA-PK	DNA-dependent protein kinase
EGFR	Epidermal growth factor receptor
EGFRmut / EGFRm	EGFR mutation
EOT	End of treatment
EMEA	Europe, the Middle East and Africa
ES-SCLC	Extensive-stage small-cell lung cancer
ESCC	Esophagea squamous cell carcinoma
EU	European Union
FCR	Fludarabine, cyclophosphamide, rituximab
FDA	U.S. Food and Drug Administration
FGFR2b	Fibroblast growth factor receptor 2 isoform IIIb
FIC	First-in-class
FIH	First-in-human
FL	Follicular lymphoma
FMI	Foundation Medicine, Inc
FTE	Full-time employee
FULV	Fulvestrant
FY	Full year
GAAP	Generally accepted accounting principles
gBRCAm	Germline BRCA mutation
GC	Gastric cancer
GEA	Gastroesophageal adenocarcinoma
GI	Gastrointestinal
GLP	Good laboratory practice
GPC3	Glypican-3
GYN	Gynecological
H1	1st half
H2	2nd half
H2H	Head-to-had
HEME	Hematology
HER2	Human epidermal growth factor receptor 2
HNSCC	Head & neck squamous cell carcinoma
hPBMC	Human peripheral blood mononuclear Cells
HPK1	Hematopoietic progenitor kinase 1
HR	Hazard ratio
HSPC	Human hematopoietic stem/progenitor cell
i	Inhibitor
I&I	Immunology & Inflammation
IC50	Half maximal inhibitory concentration
IL-15	Interleukin-15
IO	Immuno-oncology
IRA	Inflation Reduction Act
IRAK4	Interleukin-1 receptor-associated kinase 4
IRC	Independent review committee
ITT	Intent-to-Treat
JCO	Journal of Clinical Oncology
JP	Japan
KRAS	Kirsten rat sarcoma viral oncogene homolog

Acronyms (2 of 2)

LATAM	Latin America
LC	Lung cancer
LoE	Loss of exclusivity
LS-SCLC	Limited-stage small-cell lung cancer
mAb	Monoclonal antibody
MAD	Multiple ascending dose
MAT2A	Methionine adenosyltransferase 2A
mBC	Metastatic breast cancer
MCL	Mantle cell lymphoma
mCRPC	Metastatic castration resistant prostate cancer
MDS	Myelodysplastic syndromes
MET	Mesenchymal epithelial transition factor
Mg	Milligrams
MM	Multiple myeloma
MoA	Mechanism of action
mRNA	Messenger RNA
MSS-CRC	Microsatellite stable colorectal cancer
MTA Cooperative PRMT5	Methylthioadenosine cooperative protein arginine methyltransferase 5
MTx	Maintenance therapy
MUC1	Mucin 1
MZL	Marginal zone lymphoma
NDA	New drug application
NEJM	New England Journal of Medicine
Neo/adj	Neoadjuvant/adjuvant
NME	New molecular entity
NPC	Nasopharyngeal carcinoma
NPS	New patient share
NSCLC	Non small-cell lung cancer
ONC	Oncology
OS	Overall survival
PanKRAS	Molecule that targets all KRAS mutations
PARP	Poly(ADP-ribose) polymerases
PBMC	Peripheral blood mononuclear cells
PD	Progressive disease
PD1	Programmed cell death protein 1
PFS	Progression-free survival
Ph1	Phase 1
Ph2	Phase 2
Ph3	Phase 3
Pirto	Pirtobrutinib
pMN	Primary membranous nephropathy
POC	Proof of concept
PSOC	Platinum-sensitive ovarian cancer
Q1	First quarter
Q2	Second quarter
Q3	Third quarter
Q4	Fourth quarter
Q6W	Every 6-week dosing
QD	Once daily dosing
R&D	Research and development
ROI	Return on Investment
ROW	Rest of world
R/R	Relapsed / refractory
SAD	Single ascending dose
SAE	Serious adverse event
SCLC	Small-cell lung cancer
SLL	Small lymphocytic lymphoma
SMAC	Second mitochondrial activator of caspase
SoC	Standard of care
ST	Solid tumor
STEAP1	Six-transmembrane epithelial antigen of prostate 1
SubQ	Subcutaneous formulation
t(11;14)	Chromosomal translocation 11-14
TA	Therapeutic area
TCE	T-cell engager
TCR	T-cell receptor
TLR	Toll like receptor
TLS	Tumor lysis syndrome
TOX	Toxicity
TN	Treatment naïve
TP53	Tumor suppressor gene
TsAb	Trispecific antibody
UBC	Urinary / bladder cancer
uIGHV	Unmutated immunoglobulin heavy chain variable region
uMRD	Undetectable minimal residual disease
U.S.	United States of America
VEN	Venetoclax
VI	Venetoclax + ibrutinib
VO	Venetoclax + obinutuzumab
WDs	Working days
WM	Waldenström's macroglobulinemia
XmAb®	XmAb® is a registered trademark of Xencor, Inc.
Z	Zanubrutinib
ZS	Zanubrutinib + sonrotoclax