UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 6-K/A

Report of Foreign Private Issuer
Pursuant to Rule 13a-16 or 15d-16
under the Securities Exchange Act of 1934

For the month of **November 2026**

Commission File Number: 001-42484

ASCENTAGE PHARMA GROUP INTERNATIONAL
(Translation of Registrant's name into English)

68 Xinqing Road
Suzhou Industrial Park
Suzhou, Jiangsu
China
(Address of principal executive offices)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.

Form 20-F ☒ Form 40-F ☐

On November 17, 2026, Ascentage Pharma Group International ("Company") furnished an updated corporate investor deck to correct the information on slide 42 in meetings with investors at 2026 Jefferies Global Healthcare Conference in London. The corporate investor deck updates and supersedes the corporate investor deck previously furnished by the Company on November 16, 2026. A copy of the updated corporate investor deck is furnished as Exhibit 99.1 to this Report.

INDEX TO EXHIBITS

Exhibit Number	Exhibit Title
99.1	Corporate investor deck

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

ASCENTAGE PHARMA GROUP INTERNATIONAL

Date: November 17, 2026

/s/ Dajun Yang

Name: Dajun Yang
Title: Chief Executive Officer



Ascentage

Company Overview

6855 HKEX | AAPG Nasdaq

November 2026

Cautionary Note Regarding Forward-Looking Statements

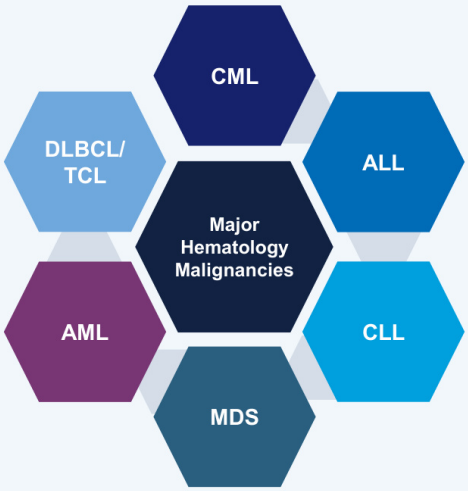
This presentation has been prepared by Ascentage Pharma Group International (the "Company") and includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 and Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. All statements, other than statements of historical facts, contained in this presentation may be forward-looking statements, including statements that express the Company's opinions, expectations, beliefs, plans, objectives, assumptions or projections regarding future events or future results of operations or financial condition. These forward-looking statements are subject to a number of risks and uncertainties that may cause actual results, levels of activity, performance or achievements to be materially different from the information expressed or implied by these forward-looking statements. For factors that could cause actual results to differ materially from the forward-looking statements in this presentation, please see the sections titled "Risk factors" and "Special note regarding forward-looking statements and industry data" in the Company's Form 20-F for the year ended December 31, 2025, filed with the SEC on April 29, 2026, and other filings with the SEC that the Company made or makes from time to time, and with respect to non-U.S. investors only, the sections headed "Forward-looking Statements" and "Risk Factors" in the prospectus of the Company for its Hong Kong initial public offering dated October 16, 2019 and other filings with the SEC and/or The Stock Exchange of Hong Kong Limited that the Company made or makes from time to time. The forward-looking statements contained in this presentation do not constitute profit forecast by the Company's management.

As a result of these factors, you should not rely on these forward-looking statements as predictions of future events. The forward-looking statements contained in this presentation are based on the Company's current expectations and beliefs concerning future developments and their potential effects and speak only as of the date of such statements. The Company does not undertake any obligation to update or revise any forward-looking statements, whether as a result of new information, future events or otherwise.

Addressing unmet medical needs.

Patients first. Innovation-driven. Science-based.

Building a leading, integrated global biopharmaceutical company.



Ascentage at a Glance

Two Novel Commercial Products[1]



Olverembatinib
BCR-ABL Inhibitor



Lisaftoclax
Bcl-2 Selective Inhibitor

Dual listed on Nasdaq and HKEX

6855 HKEX
HKEX IPO completed in October 2019

AAPG Nasdaq
Nasdaq IPO completed in January 2025

2026 6/30 cash balance: $279.4m

Global Patents and Publications

537
Issued Patents Worldwide

395
Issued Patents outside of China

100+
Publications

Robust Pipeline

7
Novel Active Clin. Compounds

10+
Indications

30+
INDs cleared

17
ODD by FDA And EMA

4
FTD by FDA

40+
Clinical Trials

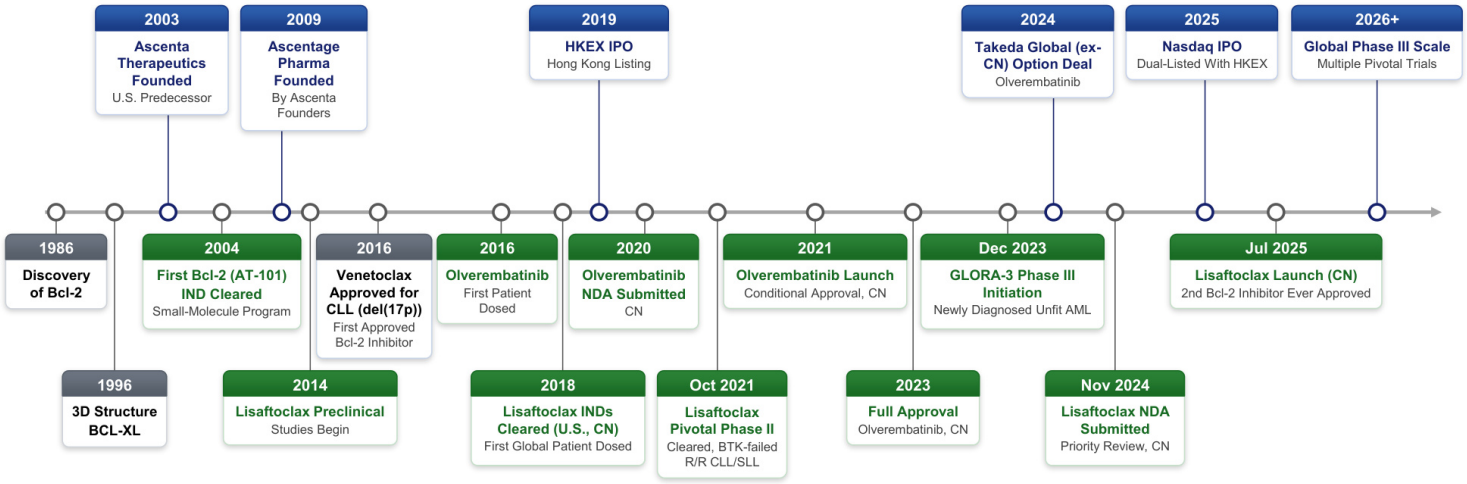


Corporate Overview

- **Global Team:** 880+ employees worldwide
- **Trials:** Nine ongoing global registrational trials, including 4 FDA-cleared Ph-III trials
- **Incorporated in the Cayman Islands;** U.S. HQ: Rockville, MD and China HQ: Suzhou with offices in Australia

1. Approvals granted by China's NMPA

Ascentage Evolved From Its Scientific Roots Into a Global, Commercial-Stage Hematology-Oncology Company



Sources: Company data; Muchmore SW et al., Nature 1996 (BCL-XL structure); Petros AM et al., PNAS 2001 (Bcl-2 structure); FDA approval of venetoclax (April 2016); NMPA approvals of Olverembatinib (November 2021, November 2023) and Lisaftoclax (July 2025); Takeda option agreement (June 2024); clinicaltrials.gov NCT03537482, NCT05147467, NCT06389292.
Events denoted in gray reflect external events.

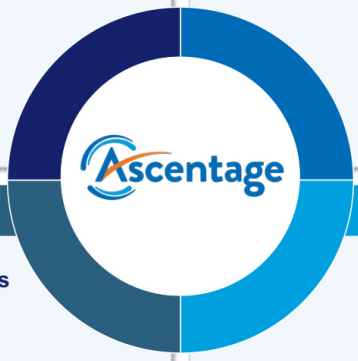
Ascentage: A Global, Commercial-Stage Hematology Oncology Platform With Multiple Paths to Value Creation

Differentiated Commercial Portfolio

- **Olverembatinib:** BIC potential 3G BCR::ABL1 TKI with activity across T315I-mutant and resistant CML
- **Lisaftoclax:** BIC potential, next-gen, Bcl-2 inhibitor with differentiated PK / DDI profile — four global Ph. III programs

Global Late-Stage Execution

- Novel, differentiated, product candidates in global development across **CML, HR-MDS and other hematologic malignancies**
- **Four FDA-cleared** Phase III programs
- **Nine ongoing** global registrational trials



Dedicated Commercial Capabilities

- **Two marketed products — $41.6M product sales** in 1H:26
- **Established U.S. commercial leadership** and planning dedicated sales force
- **300+ China commercial team,** targeting ~400 sales reps achieving **~80%** prescriber coverage

Innovative Emerging Pipeline

- **Proprietary BTK degrader, APG-3288**, targeting blockbuster oncology & autoimmune markets
- Additional programs across MDM2-p53, EED and other oncology targets
- Proprietary assets create combination synergy and lifecycle optionality

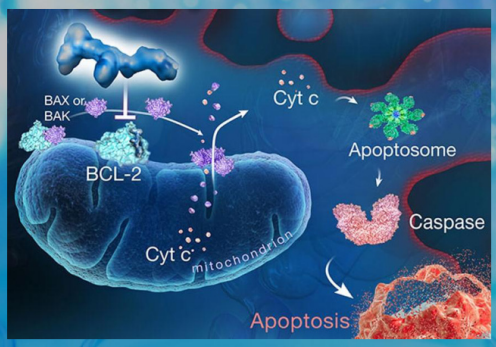
Commercial-Stage Portfolio, Multiple Global Registrational Programs, and Deep Pipeline

Asset	Indication	Clinical Program	Phase 1	Phase 2	Phase 3
Lisaftoclax[1] **Selective Bcl-2 Inhibitor**	**1L HR-MDS** + azacitidine (AZA)	**GLORA-4** *Global registrational trial*			
	CLL/SLL with suboptimal BTKi response + BTK inhibitor	**GLORA** *Global registrational trial*			
	1L CLL/SLL + acalabrutinib	**GLORA-2** *Multinational registrational trial*			
	1L elderly / unfit AML + AZA	**GLORA-3** *Multinational registrational trial*			
Olverembatinib[2] **3G BCR::ABL1 TKI**	**Resistant CML-CP**	**POLARIS-2** *Global registrational trial*			
	1L Ph+ ALL + chemo	**POLARIS-1** *Global registrational trial*			
	SDH-deficient GIST	**POLARIS-3** *Pivotal Ph. 2 registrational trial*			
APG-3288	**R/R B-cell malignancies**	**BTK Degrader** *Dose escalation ongoing*			
APG-5918	**T-cell lymphoma / prostate cancer**	**EED** *Phase 1 ongoing in U.S. & China*			
APG-2449	**ALK+ / ROS1+ NSCLC**	**FAK / ALK / ROS1** *Phase 3 ready*			
Alrizomadlin (APG-115)	**STS / AML / MDS**	**MDM2-p53** *MPN plan under evaluation*			
Pelcitoclax (APG-1252)	**AML / other indications**	**Bcl-2 / Bcl-xL** *Phase 1 site activating*			

1. Approved for CLL/SLL after BTKi therapy; marketed in China since Jul 2025.
2. Approved for T315I+ CML and TKI-resistant CML-CP; marketed in China since 2021.

Lisaftoclax

Bcl-2 Selective Inhibitor

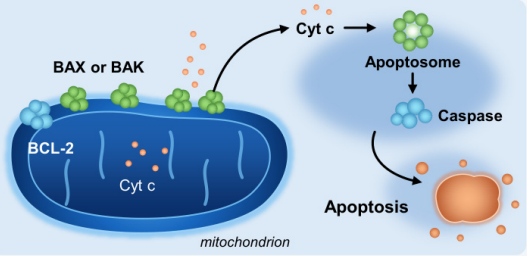


Bcl-2: A Critical Cancer Target With Decades of Drug-Development History

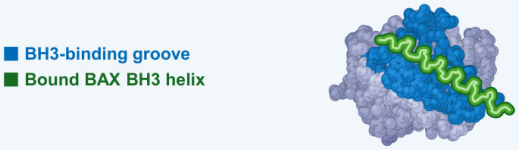
Ascentage's pioneering development efforts culminated in the first new selective Bcl-2 inhibitor approval in nine years

Structure & Function

Bcl-2 blocks the mitochondrial apoptosis pathway



Bcl-2/BAX BH3 complex



Regulates mitochondrial permeability and programmed cell death

Bcl-2 Overview



40+ years of Bcl-2 research

Critical Cancer Target
Bcl-2 blocks BAX/BAK pores to trap cytochrome c and inhibit Bcl-2, restoring apoptosis

Historically Hard to Drug
Complex mitochondrial biology and challenging protein–protein interactions

Lisaftoclax Approval: Key Milestone in Bcl-2 Innovation
Successfully developed and brought to market nine years after Venetoclax

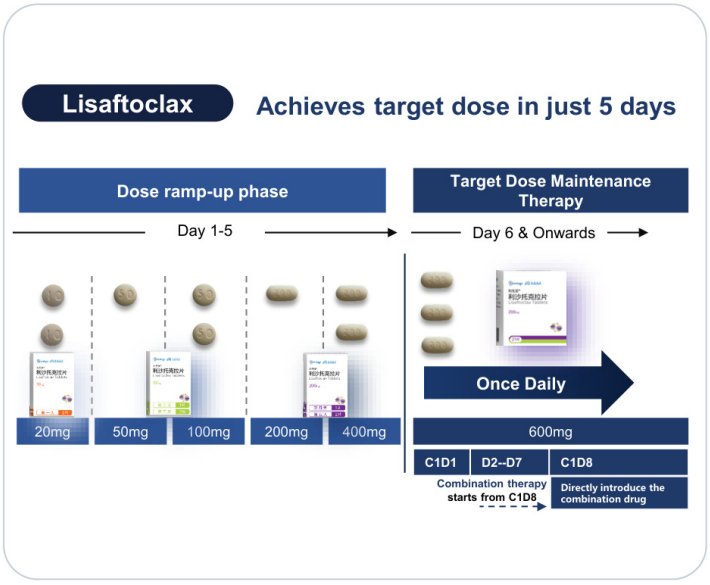
Notes: Structure: Bcl-2/BAX BH3 complex (PDB 2XA0, X-ray; Ku B, et al. Cell Res 2011); first Bcl-2 structure by NMR (Petros AM, et al. PNAS 2001; PDB 1G5M).
Sources: Tsujimoto Y, et al. Science 1984;226:1097–1099 (BCL2 identified via t(14;18)); Vogler M, et al. Signal Transduct Target Ther 2025;10:91; Wang Z, Tang M, Konopleva M. Oncogenesis 2026; U.S. FDA venetoclax approval (April 2016)

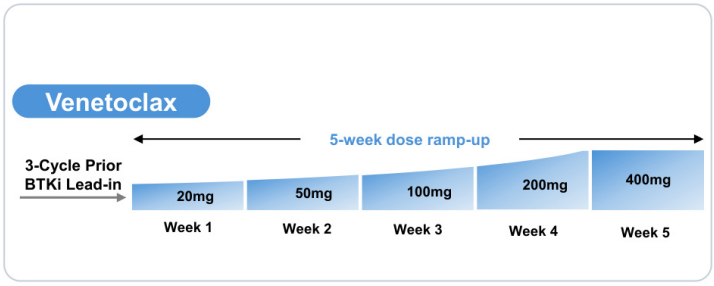
Lisaftoclax: Next-Gen Bcl-2 Selective Inhibitor Actively Advancing in Multiple Phase III Registrational Trials

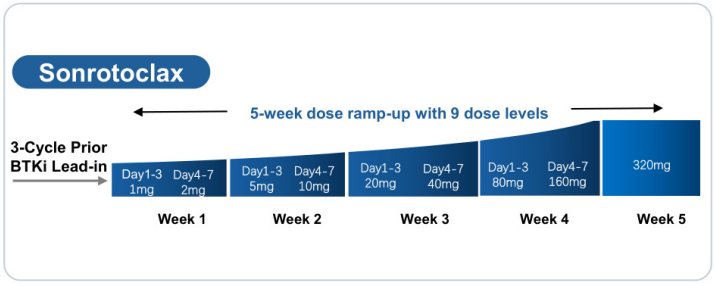
Program	Indication	Phase 1	Phase 2	Phase 3	Marketed
Pivotal Ph-II	CLL/SLL[1]	Single-agent	Coverage by NRDL[2]	Marketed in China since July 2025	
GLORA-4	First-Line HR-MDS	+ azacitidine (AZA)	FDA, EMA, PMDA, & CDE cleared	Global Phase III Registrational Trial	
GLORA	CLL/SLL sub-optimal BTKi response (Add-on)	+ BTK Inhibitor	FDA, EMA, and CDE cleared	Global Phase III Registrational Trial	
GLORA-2	First-Line CLL/SLL	+ acalabrutinib	EMA and CDE cleared	Multinational Phase III Registrational Trial	
GLORA-3	First-Line Elderly and Unfit AML	+ AZA	EMA and CDE cleared	Multinational Phase III Registrational Trial	

1. In July 2025, Lisaftoclax was approved by NMPA in China for the treatment of adult patients with CLL/SLL who have previously received at least one systemic therapy including BTK inhibitors. 2. Per company announcement on November 14, 2026.

Lisaftoclax Rapid 5-Day Dose Ramp-Up and No Prior Lead-In for Combination With BTKi





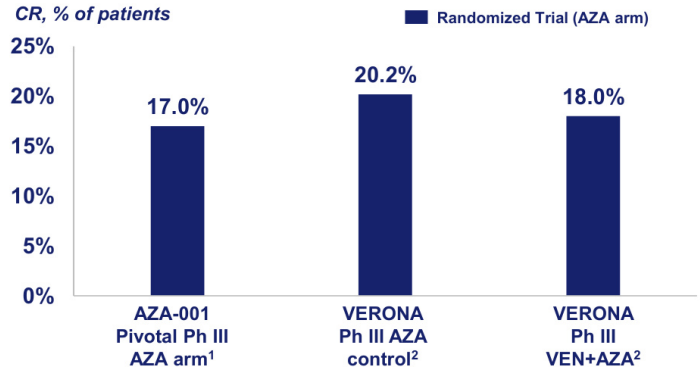


Sources: Lisaftoclax Tablets Prescribing Information. Cheson BD, et al. Oncologist. 2017 Nov;22(11):1283-1291. Sonrotoclax Tablets Prescribing Information; Venetoclax Tablets Prescribing Information.

HR-MDS Patients Have Seen No New Approved Frontline Therapy in Over 20 Years

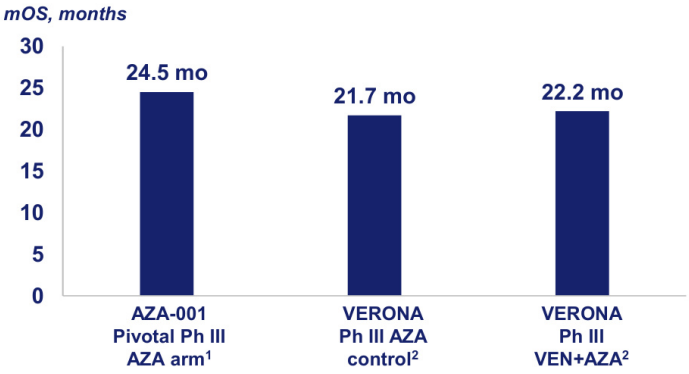
AZA remains standard of care, with VERONA (addition of Venetoclax), failing to improve CR or OS

Complete Response



- CR remains limited at ~20% in randomized AZA arms and <10% in U.S. real-world practice

Overall Survival



- OS remains at ~22–25 months across Ph. III trials

Despite AZA and ven-based treatment approaches, HR-MDS patients continue to face inadequate CR and OS outcomes

Sources: 1. Fenaux P, et al. Lancet Oncol 2009 (AZA-001, azacitidine arm n=179: CR 17%, mOS 24.5 mo); 2. Garcia-Manero G, et al. Clin Lymphoma Myeloma Leuk 2025 / AbbVie release 16 June 2025 (VERONA placebo + azacitidine arm n=253: CR 20.2%, mOS 21.7 mo); Notes: Separate studies in different populations with differing response criteria; not head-to-head comparisons.

GLORA-4: Designed to Overcome VERONA's Toxicity in HR-MDS

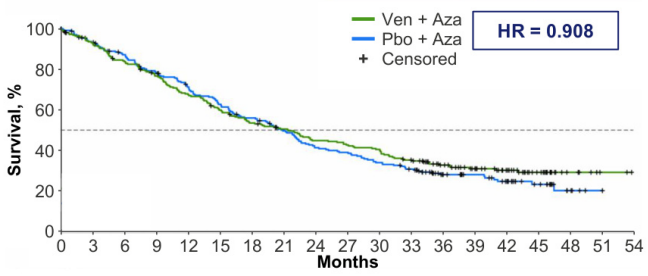
VERONA had ~2× higher AZA dose reductions with Ven + AZA limited time on therapy, with no significant OS benefit

Key Insights

1. **VERONA failed due to toxicity**
   ~2× higher AZA dose reductions with Ven + AZA
2. **Toxicity is critical in HR-MDS**
   Added myelosuppression limits time on therapy
3. **Lisaftoclax: Designed for tolerability**
   Differentiated pharmacology designed to avoid VERONA's toxicity
4. **GLORA-4: Dual primary endpoints**
   CR and OS, vs. OS alone in VERONA

Phase 3 VERONA Study — Primary Analysis

Median OS, ITT population (22.2 vs. 21.7 mos)[1]



	Ven + AZA	Pbo + AZA
AZA dose reductions	48.6%	27.2%

1. OS measured from first dose of study drug to death; censored at date last known alive. OS: ITT population (N=509); AZA dose reductions: safety population (N=501).
Source: Garcia-Manero G, et al. Primary analysis of the Phase 3 VERONA study (NCT04401748), SOHO 2025 poster MDS-1497; data cutoff April 10, 2025.

GLORA-4 Is Designed to Establish Lisaftoclax in Frontline HR-MDS

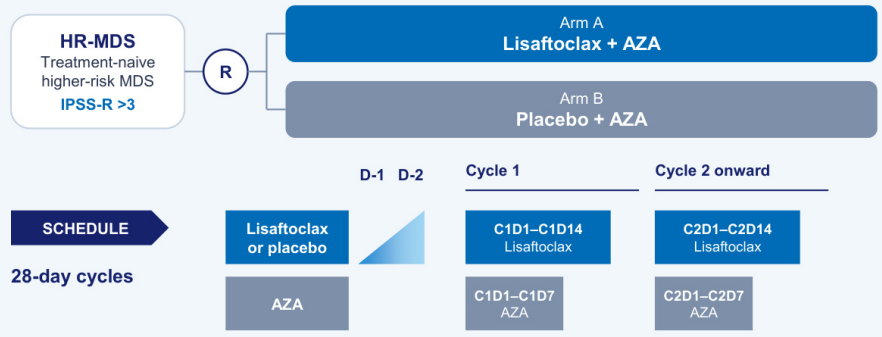
Global Randomized Phase III Evaluating Lisaftoclax + AZA In Frontline HR-MDS

Key Takeaways

- **GLORA-4 is currently the only registrational trial in frontline HR-MDS**
- Conducting under the **authorization from FDA, EMA, PMDA, and CDE**
- **Dual primary endpoints:**
  CR rate (per IWG 2023 response criteria),
  OS

Trial Design

Double-blind, randomized, placebo-controlled; same AZA backbone in both arms



Global Co-Leading PIs

Guillermo Garcia-Manero, MD	Xiaojun Huang, MD
Chair, Department of Leukemia, The University of Texas MD Anderson Cancer Center (MDACC); Leader of the MDS/AML Moon Shot Program, MDACC	*Chair, Department of Hematology, Peking University People's Hospital; Director, Institute of Hematology at Peking University*

Source: Company data.

AML Patients Need More Effective Options Across Frontline and Relapsed Disease

Bcl-2 inhibition is established in frontline AML, but patients progressing after venetoclax have limited effective options

Established Frontline SOC	**Ven + AZA remains global frontline standard** for older / intensive-chemotherapy-ineligible AML patients
Significant Post-Venetoclax Need	**Limited effective options exist for post-Ven patients,** substantial need to treat Ven-resistant patients
Resistance Biology Supports Combo Development Strategy	**MCL-1 / BCL-xL upregulation can drive resistance**; acquired Bcl-2 mutations are uncommon
Encouraging Early Clinical Activity to Date	**Lisaftoclax + AZA achieved ~32% ORR (7/22)[1]** in Ven-exposed R/R AML/MPAL

Lisaftoclax is a potential backbone targeted therapy addressing AML broadly, including Venetoclax-resistant

1. Early Phase Ib/II combination data: Lisaftoclax + azacitidine in venetoclax-exposed R/R AML/MPAL (n=22). Sources: Fathi AT, et al. N Engl J Med 2026;395:845-58 (PARADIGM, NCT04801797); Kadia T, et al. ASH 2025 (APG2575AU101).

Lisaftoclax Has Multiple Paths to Expand Across the Evolving AML Treatment Paradigm

GLORA-3 anchors the frontline opportunity, with potential in additional settings

1 Frontline Older / Unfit AML

- **GLORA-3 evaluating** Lisaftoclax + AZA vs. pbo + AZA
- Bcl-2 backbone has **potential for broader frontline AML use**

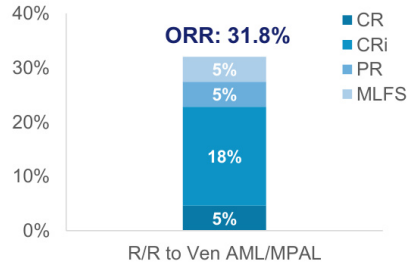
Independent Randomized Ph. 2 trial in frontline AML (PARADIGM):
Ven + AZA vs. induction chemotherapy
n=172; NEJM September 2026

14.5 vs. 6.2 mo Median EFS	**HR 0.57** Event-free survival (P=0.002)
60% vs. 40% Proceeded to transplant	**28% vs. 41%** Grade ≥3 infection

2 Post-Venetoclax / Resistance

- **Demonstrated treatment response after Venetoclax failure** in heavily pretreated population

ORR in Venetoclax Exposed AML/MPAL: Lisaftoclax + AZA (200/400/600/800 mg)
n=22 of 103 treated; ASH 2025



3 Potential Lisaftoclax Options for Broad AML Treatment

- **Lisaftoclax used widely instead of Ven**
- **Combo with Olverembatinib:** down-regulates **MCL-1**, a key Bcl-2 resistance pathway
- **Lisaftoclax backbone in combo with other novel, targeted AML therapies**

Sources: Fathi AT, et al. N Engl J Med 2026;395:845-58 (PARADIGM, NCT04801797); Kadia T, et al. ASH 2025 (APG2575AU101). Notes: PARADIGM evaluated venetoclax, not lisaftoclax; results are presented as BCL-2 class context and are not head-to-head comparisons. PARADIGM excluded patients with CBF fusions, FLT3 mutations, or NPM1 mutations (unless aged ≥60).

Single Agent Deep/Durable Response in BTKi failed R/R CLL/SLL

- **Phase II registrational study in patients with poor baseline characteristics:**
  - **100% R/R to both BTKi's and CD20 mAb-based R/R CLL**
  - **43% CK**
  - **27% high CK**

Efficacy Assessment	
ORR	**62.5%** (45/72)
MRD in PB assessment	
MRD	**21.8%** (12/55)
MRD in BM assessment	
MRD	**54.5%** (6/11)
mPFS, mo. (95% CI)	**23.89** (13.01-NR)

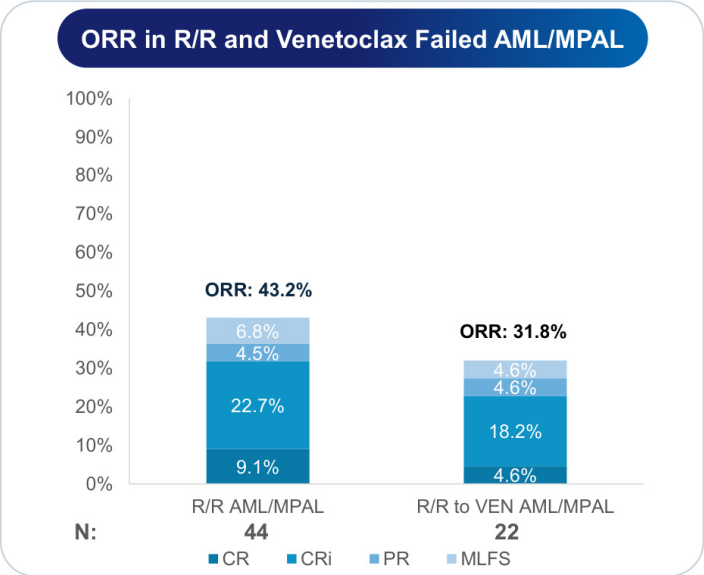
Grade≥ 3 TRAEs (>5% incidence), n = 77	
	Lisaftoclax 600 mg
Any grade ≥ 3 TRAE, n (%)	41 (53.2)
Preferred term, n (%)	
Neutrophil count decreased	21 (27.3)
Platelet count decreased	13 (16.9)
Anemia	7 (9.1)
Leukocyte count decreased	6 (7.8)

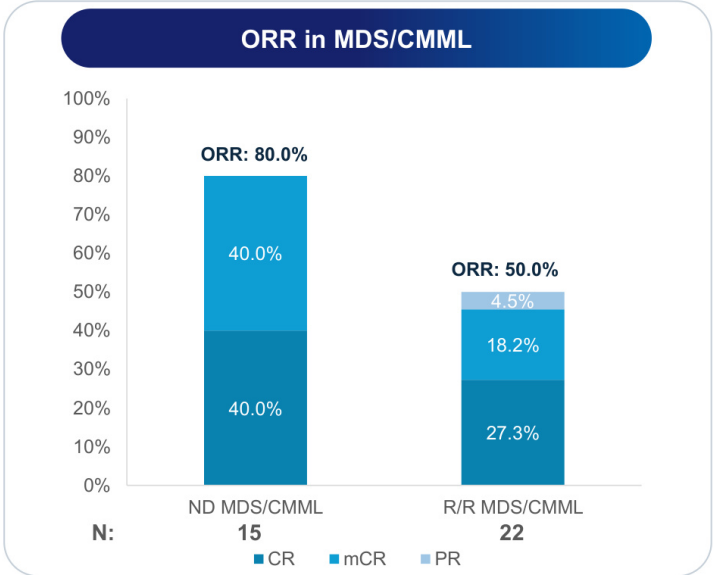
ASH 2025 Abstract. Zhou K, et al. Lisaftoclax monotherapy for BTKi-failed R/R CLL/SLL (APG2575CC201)[P]. 67th ASH Annu Meet, 2025. NCT05147467.
Company data cutoff as of July 25, 2025

Strong Clinical Activity in AML and MDS, Including Venetoclax Failed AML

Potential to overcome Venetoclax resistance in AML and HR-MDS

103 patients were treated with Lisaftoclax (200/400/600/800 mg) combined with AZA





ASH 2025 Abstract. Kadia T, et al. Lisaftoclax + azacitidine for myeloid malignancies (APG2575AU101)[P]. 67th ASH Annu Meet, 2025. Poster No.: 1641. NCT04964518.
Company data cutoff as of July 01, 2025 and enrolled 103 patients.
MLFS, Morphologic leukemia-free state

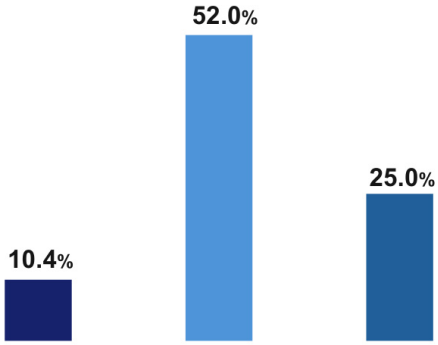
Lower Rates of SAE and Infection, No AE-Related Deaths

Lisaftoclax
CC201
R/R CLL/SLL

Venetoclax
M14-728
R/R del(17p) CLL/SLL

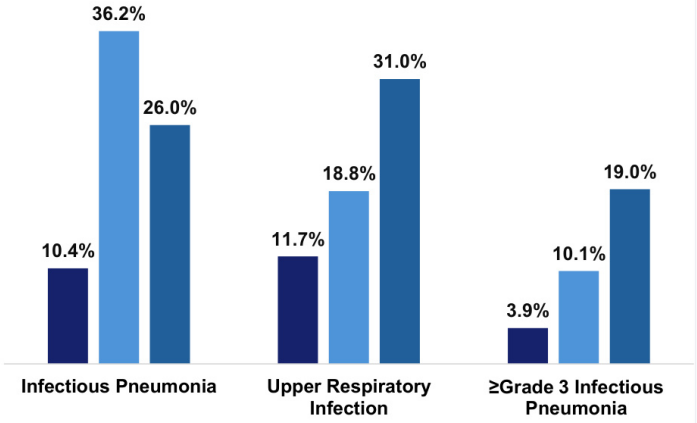
Sonrotoclax
BGB-11417-202
R/R MCL CLL/SLL

SAE Incidence in registration studies



- 2 deaths attributed to TRAE in Sonrotoclax study due to infectious pneumonia
- No deaths reported in Lisaftoclax study

Infection rate comparison



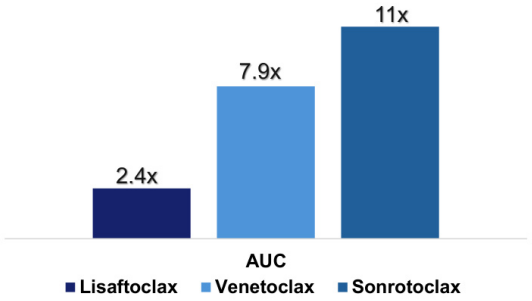
From Prescribing Information for Lisaftoclax, Sonrotoclax and Venetoclax

Lisaftoclax Has Better Drug Combinability

CLL/SLL patients are often elderly and immunocompromised, with frequent fungal infections

Commonly used antifungals are strong CYP3A4 inhibitors and does not affect Lisaftoclax PK

PK variability in combination with strong CYP3A4 inhibitors



- Strong CYP3A inhibitors contraindicated with Venetoclax and Sonrotoclax
- Lisaftoclax shows minimal fluctuation in plasma concentration when combined with strong CYP3A4 inhibitors

Lisaftoclax is safe in combination with P-gp or BCRP substrates and inhibitors

	Lisaftoclax	Venetoclax	Sonrotoclax
P-gp or BCRP substrates	**NO**	**YES**	**YES**
Combination	**No dose adjustment required**	**Dose reduction by at least 50%**	**Dose adjustment required**

- Some BTKi's are P-gp inhibitors, potentially causing DDI issues
- Unlike Venetoclax and Sonrotoclax, Lisaftoclax does not have any DDI issues with BTK inhibitors

From Prescribing Information for Lisaftoclax, Sonrotoclax and Venetoclax

Lisaftoclax: Next-Generation Selective Bcl-2 Inhibitor

Purpose-designed for potent Bcl-2 inhibition, shorter exposure, and treatment convenience

Lisaftoclax Compared to Venetoclax

1. **Efficacious in BTK-resistant WM PDX model** in which Venetoclax shows no effect
2. **Daily ramp-up** vs. weekly ramp-up
3. **Extremely low lab and clinical TLS**
4. **Less neutropenia and thrombocytopenia**
5. **Short T½ and exposure**: potentially lower risk with better safety profile
6. **The first single agent Bcl-2 inhibitor in BTK-failed CLL/SLL**

Product, Patient, Provider Attributes

When selectively targeting Bcl-2



Target dose
Daily ramp-up over days

PK properties
Short ~5-hour T½

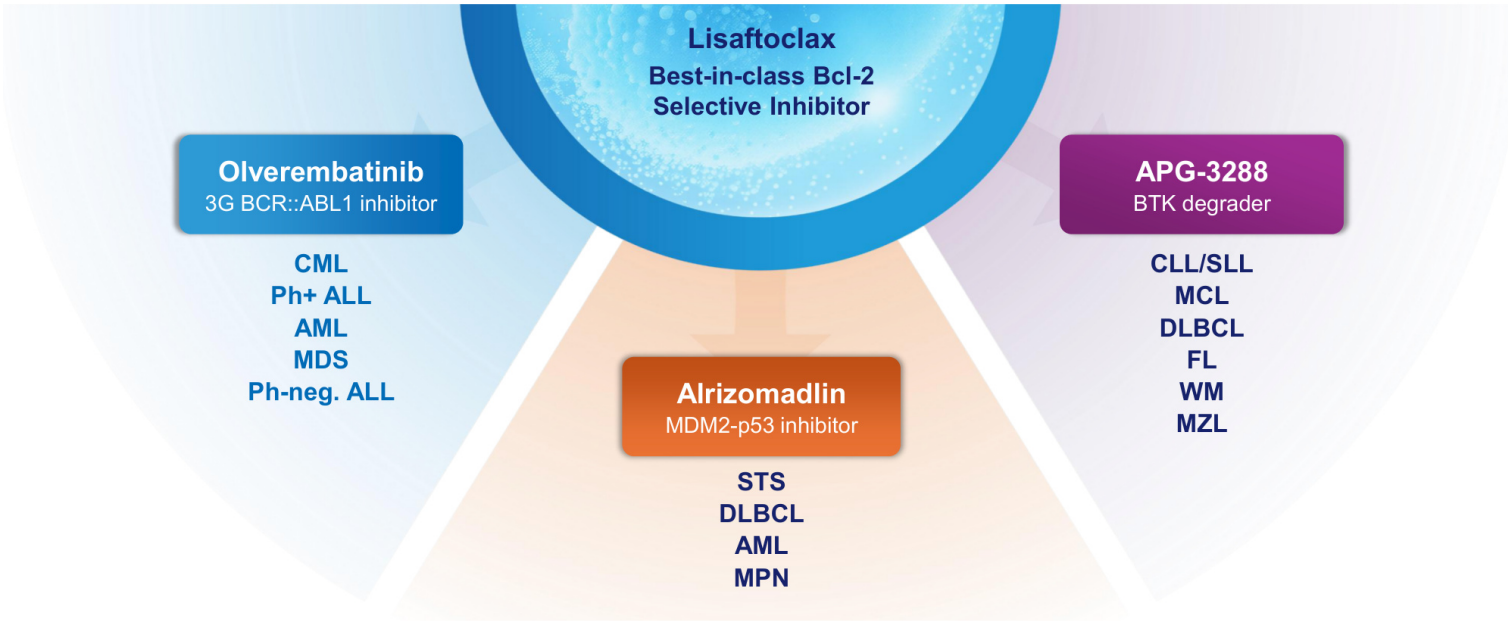
Combination use
Less concern of DDIs

"Less is More" Drug Profile Minimizes Exposure, Increases Potency, and Lowers Treatment Burden

Sources: Lisaftoclax and venetoclax prescribing information; Company data.

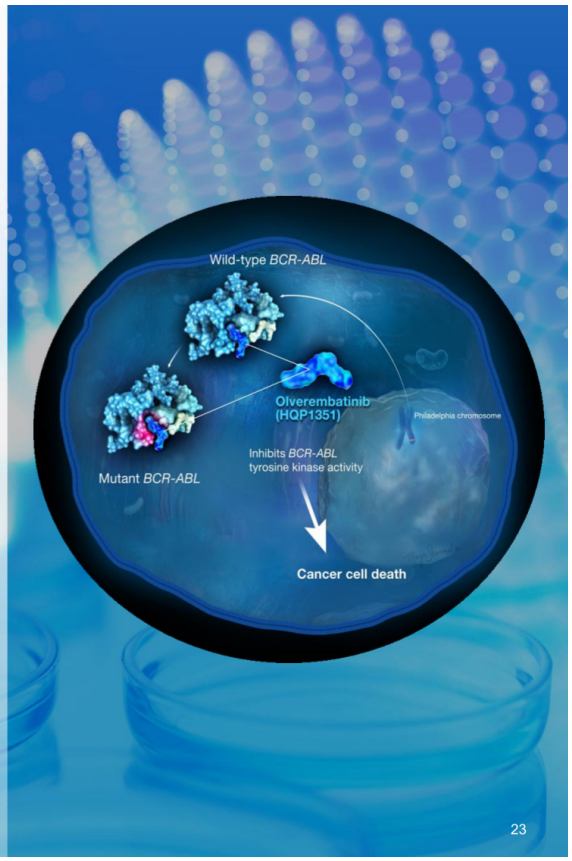
Lisaftoclax: Cornerstone Asset Poised for Proprietary Combo Strategy Across Multiple Hematologic Malignancies

Portfolio assets across BCR::ABL1, MDM2-p53 and BTK provide multiple potential combination partners



Olverembatinib (HQP1351)

3rd Generation BCR-ABL TKI



Key Shortcomings Remain with Current TKIs for CML

Despite therapeutic advances, efficacy, resistance and tolerability challenges persist across treatment classes

Current CML Treatment Landscape

1G TKI Imatinib	Transformed CML into a manageable chronic disease
2G TKIs Dasatinib, Nilotinib, Bosutinib	Improved MMR vs. Imatinib, but ineffective against T315I
3G Active-Site TKI Ponatinib	T315I activity, but carries boxed warning for AOEs
Allosteric Inhibitor Asciminib	Differentiated mechanism targeting the BCR::ABL1 myristoyl pocket

Persistent Unmet Needs

Limited Later-Line Efficacy & Durability
Only **25.5%** 24-week MMR with Asciminib (ASCEMBL)

Mutation-Driven Resistance
T315I and compound mutations limit treatment options

Tolerability / Sequencing
CV safety risks constrain use of potent later-line therapies

Sources: Réa D, et al. Blood 2021 (ASCEMBL; MMR at week 24 after ≥2 prior TKIs, asciminib arm); SCEMBLIX and ICLUSIG U.S. prescribing information.

Olverembatinib: 3G BCR::ABL1 Inhibitor With Full Mutation Coverage

Designed to address resistance with broad mutational-coverage and long-term tolerability issues of existing TKIs

Key Limitations of Existing CML TKIs	Olverembatinib: Key Attributes
✗ 2G TKIs do not address the T315I mutation	✓ Active across broad BCR::ABL1 mutation spectrum, including T315I and compound mutations
✗ Ponatinib black box warning constrains long-term use	✓ Tolerability supports durable dosing — including decreasing hematologic TRAEs over time
✗ Asciminib needs 5x dose for T315I mutants	✓ Active against the myristoyl-pocket and other mutations that confer asciminib resistance
✗ Options narrow after Ponatinib and Asciminib failures	✓ Demonstrated activity in Ponatinib and Asciminib failures (U.S. Phase 1b); POLARIS-2 global registrational trial enrolling

Real-World Activity In Patients Where Incumbent TKIs Are Ineffective: T315I, Post-Ponatinib And Post-Asciminib CML

Sources: SCEMBLIX, ICLUSIG and 2G TKI U.S. prescribing information; Jabbour E, et al. JAMA Oncol. 2025;11(1):28–35 (U.S. Phase 1b, N=62); Jiang Q, et al. ASH 2025, Poster 3788 (4-year follow-up); clinicaltrials.gov NCT06423911 (POLARIS-2); company data (BaF3 IC50 assay). Notes: Limitations reflect approved-agent labeling and published experience and are not head-to-head comparisons with Olverembatinib.

Olverembatinib in Global Phase III Registrational Trials

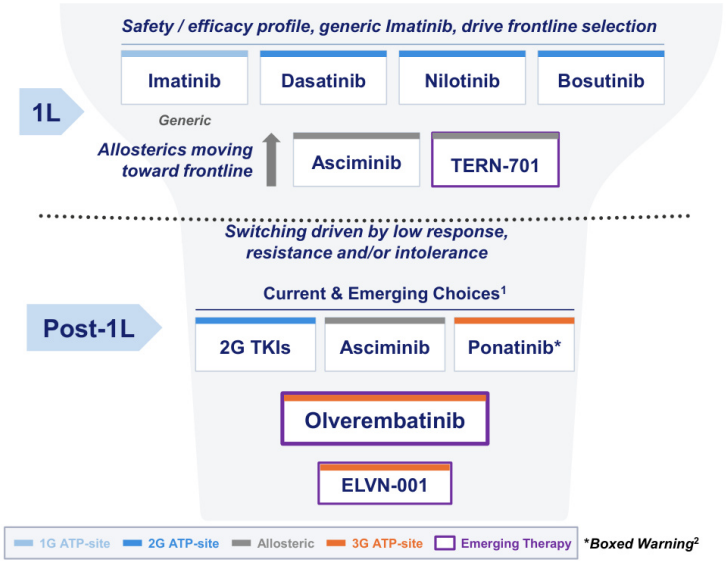
Clinical Program	Indications	Phase 1	Phase 2	Phase 3	Marketed
Pivotal Ph-II	CML-CP with/without *T315I* Mutation; CML-AP with *T315I* Mutation[1, 2]	Single-agent	Coverage by NRDL	Marketed in China since 2021	
POLARIS-2	CML	Single-agent	FDA, EMA, and PMDA cleared	Global Phase III Registrational Trial	
POLARIS-1	First-line Ph+ ALL	+ Chemo	FDA, EMA, PMDA, and CDE (with BTD) cleared	Global Phase III Registrational Trial	
POLARIS-3	SDH-deficient GIST	Single-agent	CDE cleared	Multinational Phase III Registrational Trial	

1. Approved in November 2021 in China for the treatment of adult patients with TKI-resistant CML-CP and CML-AP harboring the T315I mutation, has been included into the China 2022 NRDL effective March 1, 2023.
2. Approved in November 2023 in China for the treatment of adult patients with CML-CP resistant and or intolerant first and second generation TKIs, has been included into the China 2024 NRDL effective January 1, 2025.

CML Therapies Are Moving Earlier, Raising the Bar for Post-1L Differentiation

As a differentiated 3G TKI with mature clinical evidence, Olverembatinib is poised to emerge as a leader in post-1L CML

Prevailing CML Treatment Paradigm



Olverembatinib Evidence in Post-1L CML

1	**Heavily Pretreated Population**	**52%** ≥4 prior TKIs	**50%** Prior Ponatinib	**27%** Prior Asciminib
2	**Activity Across Resistant Disease**	**42.4%** MMR overall	**44.4%** MMR in T315I	**42.9%** MMR post-Ponatinib resistant
3	**Prospective Evidence Supporting Switching**	**54.3%** MMR at 6 mo, Olverembatinib **P<0.001**	**10.0%** MMR at 6 mo, existing TKI	**N=105** Prospective switching study
4	**Mounting Evidence of Durability**	**2021** Marketed in China since	**4-6 Year** Clinical follow-up	**Clinical +** China commercial experience

Olverembatinib: Jabbour E, et al. JAMA Oncol. 2025;11(1):28–35 (U.S. Phase 1b, N=62; prior therapy, MMR overall, T315I and post-ponatinib resistance); switching study EHA 2026 (EHA-4595 / PS1728, Wen B, N=105), prospective, multicenter, controlled trial vs. continued prior TKI in inadequate responders. SCEMBLIX and ICLUSIG U.S. prescribing information; Company data.
1. Selection depends on prior TKI exposure, mutation profile and durability. 2. Boxed Warning summarized from ICLUSIG (ponatinib) U.S. Prescribing Information; see full label for complete safety information.

Olverembatinib Positioned to Address a Meaningful 2L+ CML Opportunity

Large Treated Population and Continued Later-Line Treatment Support a Significant Commercial Opportunity

CML Treatment Landscape

		Prior / Initial TKI	Subsequent Sequencing	Status	Annual Opportunity	Patients on Treatment[1]
CORE OPPORTUNITY	Post-1L	**Prior ATP-binding TKI(s)** Resistance or intolerance	**Ponatinib / Asciminib** Other sequencing options	**POLARIS-2: resistant CML-CP and T315I (enrolling)**	**~13K[1]**	**~38K**
	1L	**ATP-binding TKI(s)** Imatinib, dasatinib, nilotinib, bosutinib	**Allosteric TKI(s)** Asciminib / other sequencing	***Potential Future Opportunity***	**~18K[2]**	**~55K**

Key Differentiation

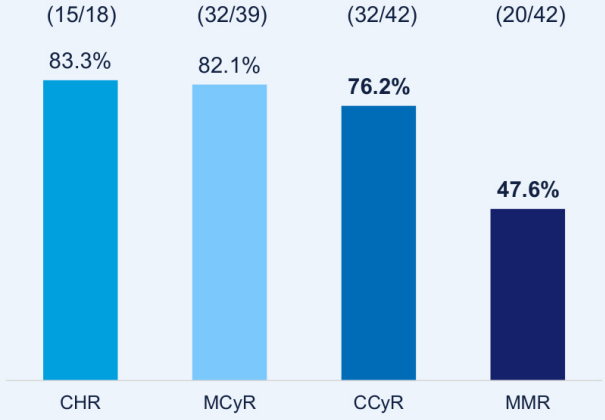
- Activity across **T315I, multi-TKI resistance, post-ponatinib, post-asciminib** and heavily pretreated patients
- **Proven durability demonstrated by post-approval real-world evidence**
- Global registrational strategy supported by **mature China experience**

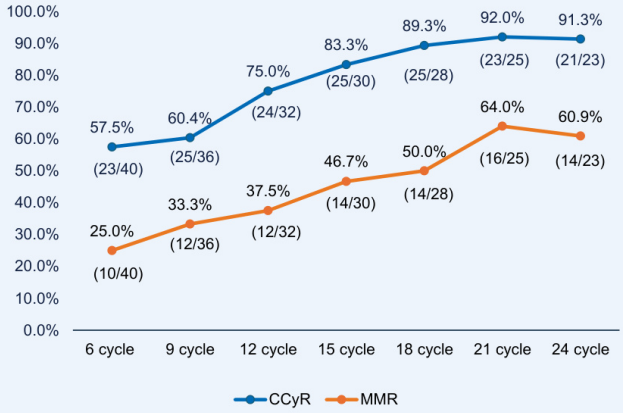
Resistance and Continued TKI Switching Support an Estimated $4–5B Global Post-1L CML Opportunity[3]

Sources: Terns Pharma Investor Presentation (March 2026), based on SEER 2024 estimates. Novartis ASCO Investor Event (June 2024). Merck Investor Presentation (March 2026), based on CancerMPact®, Oracle Life Sciences.
1. G7 (US, EU5, Japan) CML patients by line of therapy; reflects post-1L annual treatment starts. 2. CML incidence in G7 in 2025. 3. Company estimate based on historical, global branded CML TKI sales (2019–2023; Bloomberg). Assumes ~50% post-1L allocation and reflects potential growth in CML prevalence, premium branded pricing, and treatment switching.

Olverembatinib Shows Potential as a Second-Line Treatment in CML-CP

76% CCyR and 48% MMR in efficacy-evaluable patients, increasing response over time





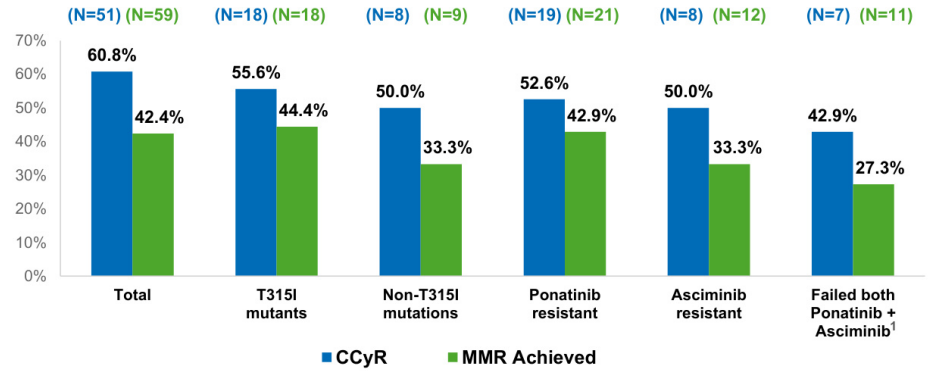
2026 ASCO. Li W, Zhang Y, Zhu H, et al. Updated efficacy and safety of Olverembatinib (HQP1351) as second-line therapy in patients with chronic phase-chronic myeloid leukemia (CP-CML).
Data cutoff: January 14, 2026.
CCyR, complete cytogenetic response; CHR, complete hematologic response; MCyR, major cytogenetic response; MMR, major molecular response.

Phase 1b Study in Heavily Pretreated CML-CP Patients (U.S.)

Principal Investigator: Elias Jabbour, MD | The University of Texas MD Anderson Cancer Center

- Heavily pretreated CML-CP patients: 52% ≥4 prior TKIs, 50% Ponatinib-pretreated, 27% Asciminib-pretreated
- 29% patients with T315I mutations

Efficacy Data



Sustained Treatment Duration

48 weeks	median treatment duration for CML-CP
3.2 years	longest treatment for CML-CP

Manageable Safety Profile

Most frequent Grade ≥3 TEAEs

Thrombocytopenia	**18%**
Increased blood creatine phosphokinase	**13%**

Other Grade ≥3 TEAEs were ≤5%

Consistent And Durable 2L+ Efficacy In Ponatinib- Or Asciminib-pretreated Patients, Including Those Who Failed Both

Sources: JAMA Oncology. Jabbour E, Oehler VG, Koller PB, et al. Olverembatinib After Failure of Tyrosine Kinase Inhibitors, Including Ponatinib or Asciminib: A Phase 1b Randomized Clinical Trial. JAMA Oncol. 2025;11(1):28–35
1. Elias Jabbour et al. ESH-iCMLf 26th Annual John Goldman Conference on Chronic Myeloid Leukemia 2024.

Olverembatinib Has Proven, Broad-Spectrum Coverage

- **Broad activity across T315I and compound BCR::ABL1 mutations**
- **Retains activity against myristoyl-pocket mutations associated with asciminib resistance; clinical activity after ponatinib and asciminib failure**

Mutated region	BaF3 mutant	Anti-proliferation assay, IC50 (nM)					
		Imatinib	Nilotinib	Dasatinib	Asciminib	Ponatinib	Olverembatinib
Wild-type	–	565±656	31±4	10±3	31±4	11	6±3
SH2-contact region	M351T	1298±542	37±4	8±4	47±34	13±1	9±1
Substrate-binding region	F359V	>10000	1710±635	598±624	6066±355	466±73	50±16
P-loop	E255K	8222±484	648±395	14±1	10	49±4	22±13
	Y253H	8936±1774	497±122	11±2	28±13	37±4	7±1
	E255V	7565±3268	587±151	29±15	24±4	56±1	27±11
	M244V	2963±83	236±152	40±1	5223±4899	75±42	41±8
Gatekeeper	T315I	>10000	3425±650	2525±322	148±14	33±11	24±10
Hinge region	F317L	526±56	89±8	11±1	6±3	7±1	8±3
	F311I	3547±223	226±122	13±0	107±1	30±8	23±13
SH3-contact region	V299L	1987±1237	103±6	118±2	562±552	10±4	8±4
T315I + other compound mutation	T315I/E255V	>10000	6467±4431	3571±1385	93±86	244±125	26±11
	T315I/F359V	>10000	4586±1397	3392±211	6631±1201	101±22	20±10
	T315I/G250E	>10000	8511±5599	5001±2939	7451±3057	130±16	33±2
	T315I/E255K	>10000	>10000	4706±803	8944±748	339±12	40±5
	T315I/E453K	8466±1628	>10000	4724±155	2931±74	130±5	61±27
	T315I/M351T	7603±1498	>10000	7683±3645	>10000	127±5	67±44
	T315I/F311I	7144±2459	>10000	4789±1739	7061±1423	438±88	78±46
	T315I/H396R	8953±5314	>10000	9286±3386	>10000	211±134	79±54
	T315I/Y253H	>10000	>10000	7080±3233	6981±2481	889±100	114±1
	T315I/F317L	>10000	>10000	>10000	860±96	688±412	117±23
	T315M	>10000	>10000	>10000	996±405	1987±1414	217±131
Other compound mutation	G250E/V299L	6486±2622	641±368	570±599	2601±2903	12±3	14±2
	F317L/F359V	7195±1729	926±24	50±12	5214±810	24±12	25±13
	Y253H/E255V	>10000	7026±2183	231±92	5014±2920	772±220	122
	T253H/F359V	>10000	>10000	110±1	>10000	432±23	311±35

IC50 in BaF3 cells expressing BCR::ABL1 mutants (mean ± SD): ■ ≤100 nM (sensitive) ■ 100–1,000 nM (moderate) ■ >1,000 nM (highly resistant)

In BaF3 Cells Stably Expressing BCR-ABL Mutants, Olverembatinib Is Active Against Those Mutations That Confer Asciminib Resistance[1]

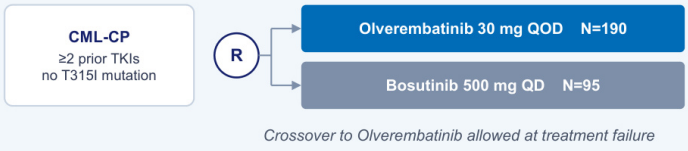
Sources: IC50 heatmap: company data (BaF3 anti-proliferation assay; mean ± SD). Post-ponatinib and post-asciminib activity: Jabbour E, et al. JAMA Oncol 2025.
1. Includes M244V, F359V/I/C, A337T, G463D or P465S mutations.

POLARIS-2 Design: Olverembatinib in CML-CP, Including T315I Mutations

Randomized comparison against bosutinib after ≥2 prior TKIs, with a single-arm T315I cohort; primary endpoint of MMR at 24 weeks in both parts

PART A Randomized vs bosutinib

Post-≥2 TKI, non-T315I CML-CP (N=285)

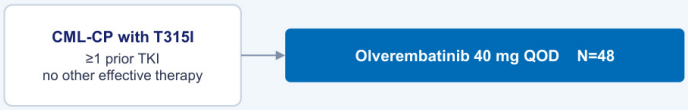


Crossover to Olverembatinib allowed at treatment failure

- Previously treated with ≥2 TKIs; T315I mutation excluded
- Randomized 2:1 to Olverembatinib 30 mg QOD or bosutinib 500 mg QD
- Crossover to Olverembatinib permitted at treatment failure

PART B Single-arm T315I cohort

T315I CML-CP after ≥1 prior TKI (N=48)



- Confirmed T315I mutation after ≥1 prior TKI
- Single-arm Olverembatinib 40 mg QOD
- Enrolls patients with no other effective therapy available

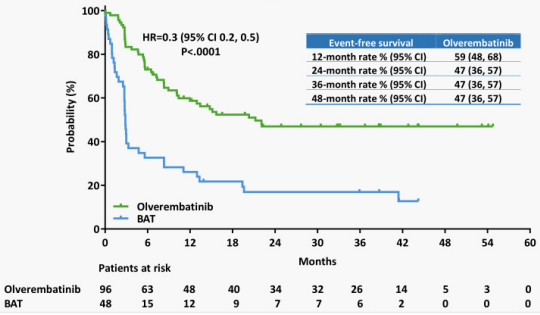
Primary Endpoint: MMR at 24 weeks
Secondary Endpoint: MMR at 96 weeks

Cleared by FDA, EMA, CDE and PMDA; global Phase III registrational trial (NCT06423911)

Sources: Company data (POLARIS-2 trial design).

Registrational Phase II Study Demonstrates Durable Benefit With 4-Year Follow-Up

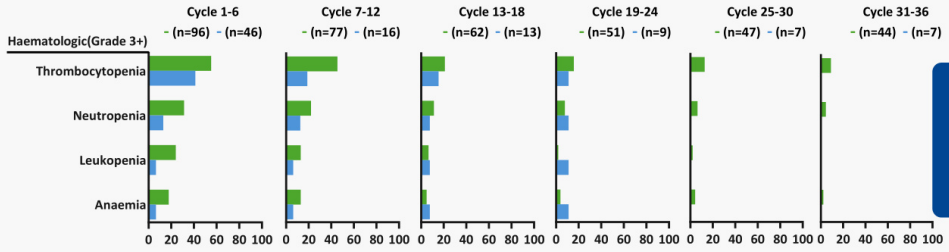
- **Significantly longer EFS and better tolerability than BAT arm**
- **Majority of patients included in study had all three 1G & 2G TKIs**



Event-free survival	Olverembatinib
12-month rate % (95% CI)	59 (48, 68)
24-month rate % (95% CI)	47 (36, 57)
36-month rate % (95% CI)	47 (36, 57)
48-month rate % (95% CI)	47 (36, 57)

EFS: All Patients with CML-CP

mEFS Olverembatinib treated: **21.2** months
mEFS BAT: 2.9 months



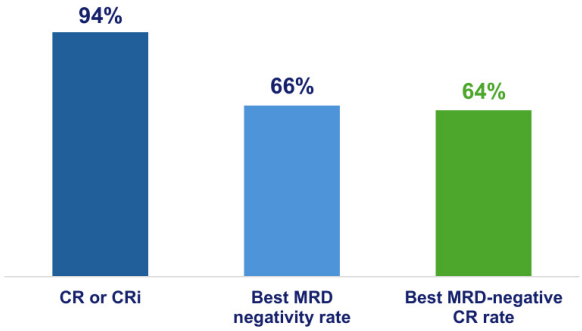
Hematologic ≥ 3 grade TRAEs decrease over cycles

Sources: ASH 2025 Abstract. Jiang Q, et al. Olverembatinib for TKI-resistant CML-CP: 4-yr follow-up (Phase 2)[P]. 67th ASH Annu Meet, 2025. Poster No.: 3788. NCT04126681
Company data as of Jan 13, 2025.

POLARIS-1 Part 1 Demonstrates Deep Responses in Frontline Ph+ ALL

Primary Endpoint: MRD Negativity CR Rate[1] by End of Three Cycles of Induction Therapy

53 evaluable patients



- 10 patients had IKZF1^{plus} genotype and 9 achieved MRD-negativity
- MRD-negative CR rate: 64.2%
- Surpasses Ponatinib's 34.4% MRD-negative CR rate from PhALLCON Trial

Olverembatinib in combination with low-intensity chemotherapy was well tolerated

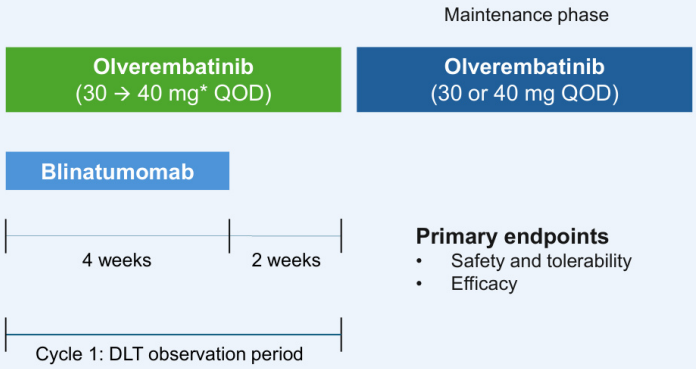
Summary of TEAEs	Overall (n = 55)
Any TEAE, n (%)	54 (98.2)
Grade ≥ 3 TEAE	48 (87.3)
Any SAE	34 (61.8)
Any TEAE leading to discontinuation	7 (12.7)
Any TEAE leading to death	2 (3.6)
Any TRAE, n (%)	43 (78.2)
Grade ≥ 3 TRAE	22 (40.0)
Any SAR	9 (16.4)

ASH 2025 Abstract. Chen S, et al. Olverembatinib + low-intensity chemo for ND Ph+ ALL (POLARIS-1)[P]. 67th ASH Annu Meet, 2025. Poster No.: 1574. NCT06051409.
Company data as of July 18, 2025
1. BCR-ABL/ABL1 ≤ 0.01% by qPCR

Olverembatinib + Blinatumomab: Clinical Strategy in CML-LBP and Ph+ BCP-ALL

Study Design and Regimen

- Phase 1b
- 3+3 dose escalation
- Up to five 6-week cycles



Primary endpoints
- Safety and tolerability
- Efficacy

*If 0/3 patients develop DLT, the dose escalates from 30 to 40 mg QOD.

Efficacy To Date Summary

91%
CR/CRi
10/11 evaluable patients

67%
MRD-negative (BCR::ABL1, MR4.0)
8/12 evaluable patients

80%
MRD-negative (flow cytometry, 10^{-4})
8/10 evaluable patients

Olverembatinib: Highly Competitive Among Emerging Therapies for CML / Ph+ ALL

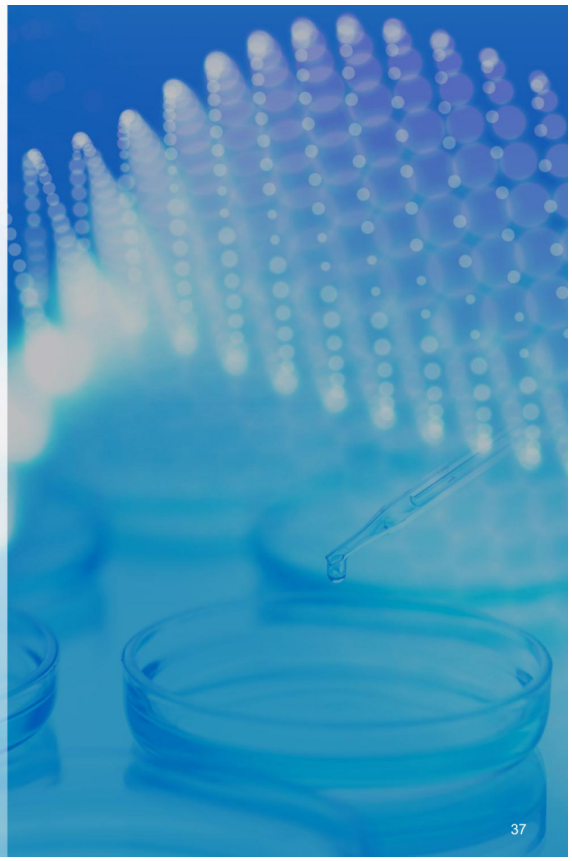
Peer Benchmarking Analysis

	Olverembatinib	ELVN-001	TERN-701
Development Maturity			
Stage of Development	Marketed*	Phase 1b	Phase 1/2
Long-term follow-up / durability evidence	Up to 6 years	✗	✗
Real-world evidence	✓	✗	✗
Resistant CML Activity *(% MMR achieved)*			
T315I-mutant CML	44.4%	None	None
Prospective randomized evidence	54.3% vs 10.0% (P<0.001)	None	None
Post-ponatinib	42.9%	Not reported	Not reported
Heavily pretreated / Post-Asciminib	33.3%	28–29%[1]	Not reported
Post-Ponatinib + Asciminib	27.3%	Not reported	Not reported
Breadth of Efficacy			
Transplant achievement	✓	✗	✗
BP-CML / Ph+ ALL	✓	✗	✗

Olverembatinib Is The Potential Leading Post-1L CML Option

Sources: Olverembatinib: JAMA Oncol. 2025;11(1):28–35 (U.S. Phase 1b, N=62); switch study EHA 2026 (EHA-4595 / PS1728, Wen B, N=105), a prospective, multicenter, controlled trial.
1. ELVN-001: newly MMR achieved by Week 24 in post-asciminib patients with 3–4 prior TKIs (28%, n=22) and 5+ prior TKIs (29%, n=15); 67% total / 60% achieved in those with 1–2 prior TKIs (n=6). Enliven Therapeutics, data cutoff 10 March 2026. *Approved for T315I+ CML and TKI-resistant CML-CP; marketed in China since 2021.

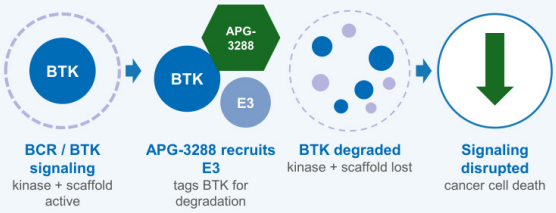
Clinical Pipeline



APG-3288: Differentiated BTK Degrader Designed to Address Resistance Across B-Cell Malignancies

Lead clinical candidate from proprietary protein degrader platform; global Phase 1 dose escalation underway

Targeted BTK Degradation



Degrades BTK to address both kinase and scaffolding functions

Designed for Broad Activity Across BTK Resistance Settings

Broad Mutant Coverage
Designed to overcome resistance to covalent and noncovalent BTKi's; binds wild-type BTK and treatment-emergent mutants with high affinity and selectivity

Distinct Degrader Mechanism
Targets both BTK kinase and scaffolding functions through selective protein degradation

Potent, Selective Degradation
Robust, sustained BTK degradation at low-nM concentrations across multiple B-cell lymphoma cell lines, with limited off-target kinase activity

Optimized Drug-Like Properties
Oral exposure, clean ADME profile, and potential CNS penetration

Proprietary Protein Degrader Platform

DC Nomination to FDA IND in ~7 Months
From APG-3288 project initiation to IND submission

Multiple Modalities
PROTACs, molecular glues and antibody-conjugated degraders

End-to-End Capabilities
Target validation through clinical translation

Global Phase 1 Underway

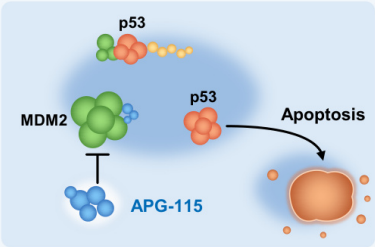
Development Status
- Global, multicenter, open-label Phase 1 dose escalation in R/R B-cell malignancies
- IND open in the U.S. and China

Preclinical Profile Supports Potential Differentiation Vs. Other Clinical-stage BTK Degraders

Source: Company data.

Alrizomadlin (APG-115): Orally Bioavailable, Highly Selective, Small Molecule Inhibitor Targeting MDM2-p53

Mechanism of Action



Reactivates wild-type p53 by inhibiting MDM2-mediated suppression

Clinical scope spans multiple rare oncology settings:

- ACC
- MPNST
- AML/MDS
- Pediatric solid tumors

APG-115 Overview

6 FDA Orphan Drug Designations

2 FDA Rare Pediatric Disease Designations

- **China's Starlight Program** provides additional development support for pediatric anticancer drug R&D
- **Global clinical footprint** across China, the U.S. and Australia
- **Flexible development strategy** across monotherapy and combination studies
- **Phase I/II data in salivary gland cancers**, including adenoid cystic carcinoma, demonstrated promising monotherapy activity with good tolerability[1]

Strategic Validation

- **Ipsen / Kartos acquisition sets class benchmark:** US$450M upfront + up to US$1.3B in milestones
- **Ipsen gained navtemadlin, a Phase III** ready asset as add-on to ruxolitinib in myelofibrosis
- **Large-cap acquisition validates** strategic appetite for MDM2–p53 assets
- APG-115 offers **broader development optionality** in hematologic malignancies and solid tumors

Class Validation Underscores APG-115's Strategic Value & Broad Development Optionality

Sources: Company data.
1. Pearson AT, et al. Phase I/II study of alrizomadlin (APG-115) in p53 wild-type salivary gland cancers. Abstract, 2024.

Alrizomadlin Alone or in Combination with Lisaftoclax Demonstrated Encouraging Objective Response Rates in Pediatric Patients with RMS or STSs (Cont'd)

Efficacy of Alrizomadlin + Lisaftoclax in patients with RMS and other tumors



- **12** evaluable patients achieved at least stable disease (DCR = 70.6%)
- **4** patients achieved PR (ORR = 23.5%)
- **2 of 7** patients with RMS achieved PR
- **1** patient with ES achieved CR with no uptake

2026 ASCO. Zhang YZ, Zhang Y, Lu SY, et al. Alrizomadlin (APG-115) alone or in combination with Lisaftoclax (APG-2575) for pediatric relapsed/metastatic rhabdomyosarcoma (RMS) and other soft-tissue sarcomas (STSs) Data cutoff: April 16, 2026.

Olverembatinib + Lisaftoclax is a Potentially Viable "Oral Dual-Targeted" Regimen for Pediatric Patients with R/R Ph+ ALL

Upcoming Poster (ASH 2026)*

Efficacy

	EOM*	C2D15	C2D27	C2D28
ORR (CR + CRi)	**55.6%** (5/9) 3 CR, 2 CRi	**88.9%** (8/9) all CR	**88.9%** (8/9) all CR	-
CCyR (by FISH)	**40.0%** (4/10)	**88.9%** (8/9)	-	-
MRD negativity (by cytometry)	**25.0%** (3/12)	**66.7%** (8/12)	-	**66.7%** (8/12)
MMR or better (via quantitative PCR)	-	-	-	**93.3%**
CMR	-	**21.4%** (3/14)	-	**20.0%** (3/15)

**EOM = end of olverembatinib monotherapy*

Efficacy, PK, and Safety

Patients

- 17 pediatric Ph+ ALL patients (median age 13): 14/16 R/R, 6/16 intolerant
- ABL1 KD mutations in 6/15 (40%), including T315I (n=4)

Efficacy

- Responses across dose levels and ABL1 variants

PK

- Exposure predictable and comparable to adults
- No significant accumulation or drug–drug interactions
- T½: Olverembatinib ~13–16 h; Lisaftoclax ~4–6 h
- Both agents detected in CSF, indicating CNS penetration

Safety

- No treatment-related deaths

Source: EHA 2026 Abstract. Zhang J, Wang J, Liu T, et al. Safety and preliminary efficacy of olverembatinib (HQP1351) combined with lisaftoclax (APG-2575) in pediatric patients with relapsed/refractory (R/R) Ph+ ALL. *Updated results to be presented at ASH 2026 (Poster #1547).

Multiple Upcoming Catalysts

Upcoming Clinical Milestones

Lisaftoclax
Bcl-2 inhibitor

- **GLORA-4** 1L HR-MDS
  Topline readout anticipated **2H 27**
- **GLORA-3** 1L Unfit AML
  Enrollment completion anticipated **YE 26 / 1Q 27**
- **GLORA-2** BTKi add-on 1L CLL/SLL
  Enrollment completed ✓
- **GLORA** BTKi-treated CLL/SLL
  Enrollment completion anticipated **YE 26 / 1H 27**

Olverembatinib
BCR::ABL1 inhibitor

- **POLARIS-2** ≥2 TKIs CML-CP
  Enrollment completion anticipated **1H 27**
- **POLARIS-1** 1L Ph+ ALL
  Enrollment completion anticipated **YE 26 / 1Q 27**

APG-3288

- **APG-3288** Global Phase 1
  Dose escalation / expansion data anticipated **1H 27**

APG-5918

- **APG-5918** R/R TCL Phase 1 data
  ASH oral presentation (#513): **Dec 13, 2026**
  Pivotal trial discussions ongoing

Experienced Leadership Team and Board of Directors

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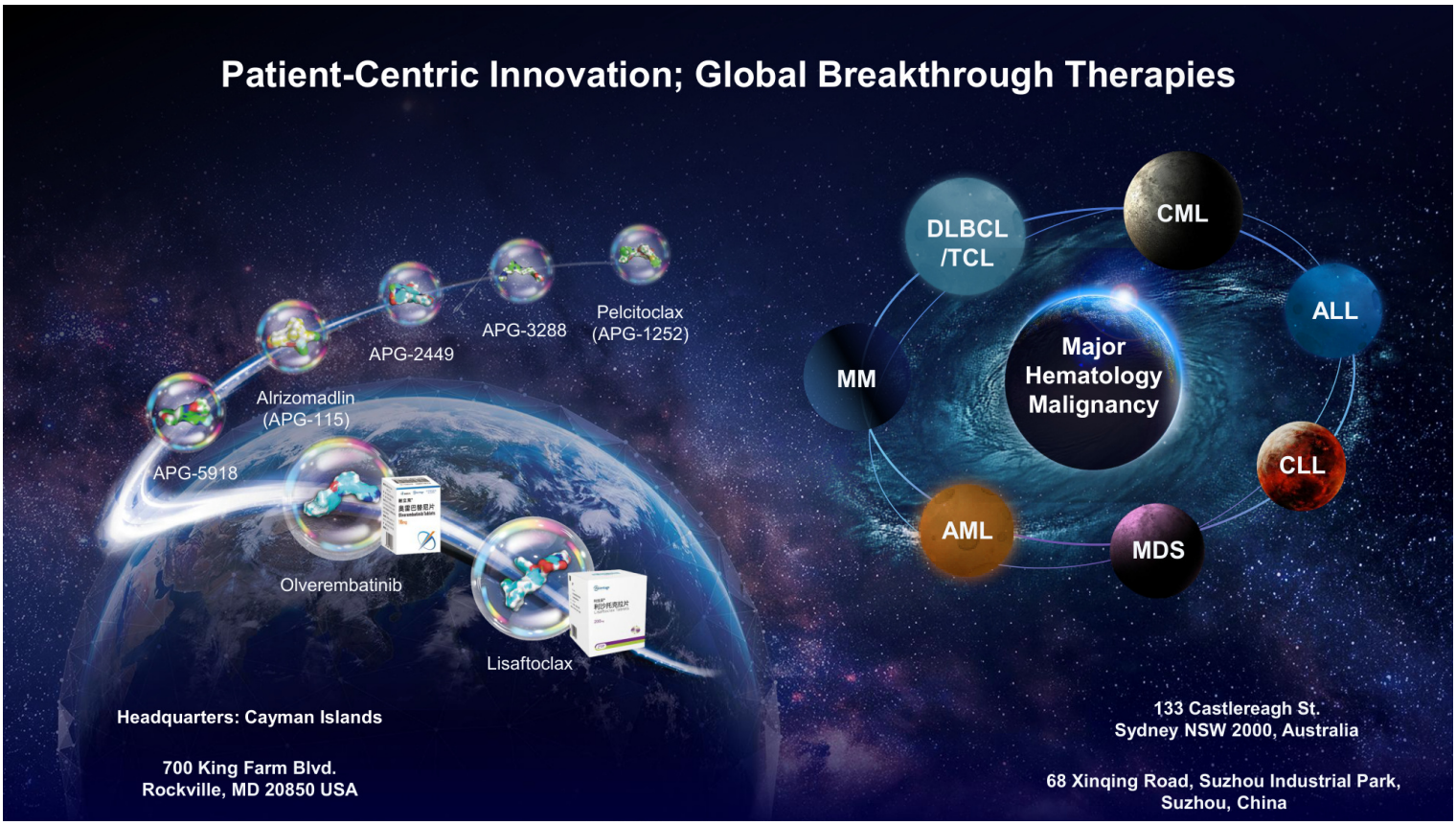
Patient-Centric Innovation; Global Breakthrough Therapies
APG-2449
APG-3288
Pelcitoclax
(APG-1252)
Alrizomadlin
(APG-115)
APG-5918
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