R&D Meeting

Sumitomo Pharma
Innovation today, healthier tomorrows

February 17, 2026

Sumitomo Pharma Co., Ltd.

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Accordingly, due to various subsequent factors, forecasts, plans, goals, and other statements may not be realized as described, and actual financial results, success/failure or progress of development, and other projections may differ materially from those presented herein.

Information concerning pharmaceuticals and other products (including those under development) contained herein is not intended as advertising or as medical advice.

Today's Agenda

1. R&D Progress and Strategy

Managing Executive Officer
Research and Development Division
Senior Vice President, Head of Research and Development Division
Chief Development Officer, Sumitomo Pharma America, Inc.

Yumi Sato

2. Two Key Oncology Compounds in Development

- Selective Menin Inhibitor — Enzomenib
- PIM1 Kinase Inhibitor — Nuvisertib

Global Oncology Strategy Lead
Lead Senior Vice President, Sumitomo Pharma America, Inc.

Masashi Murata

3. Q&A Session

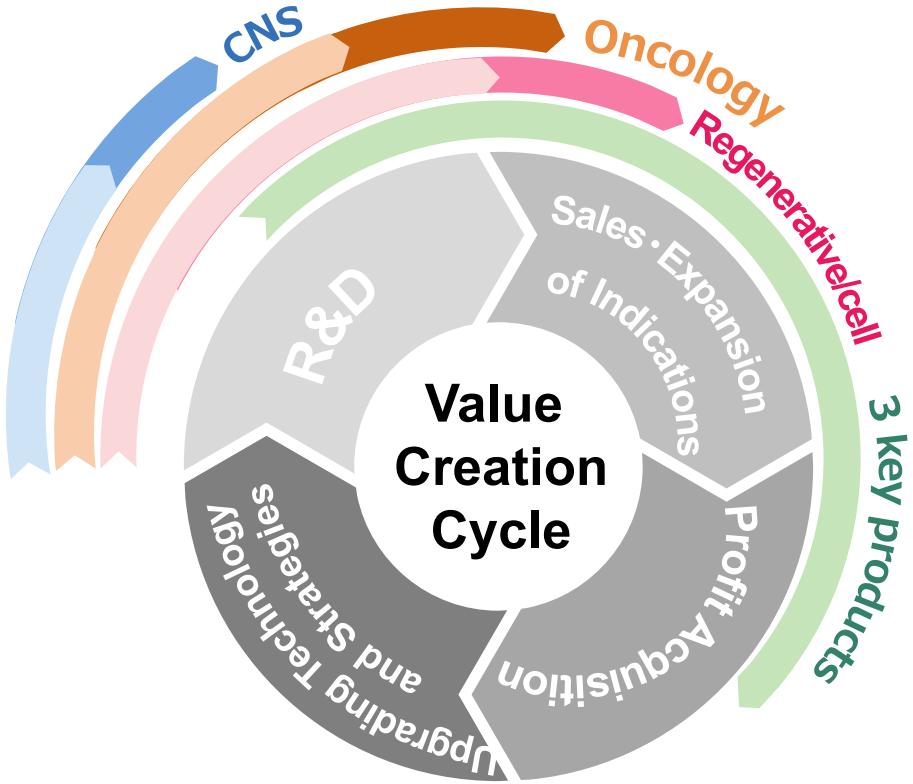
R&D Progress and Strategy

Managing Executive Officer
Research and Development Division
Senior Vice President, Head of Research and Development Division
Chief Development Officer, Sumitomo Pharma America, Inc.

Yumi Sato

Reboot 2027

✓ **Rebuild our foundation as an R&D-driven pharmaceutical company while continuing the fundamental structural reforms**
✓ **Reconstruct the Value Creation Cycle driven by our in-house innovation to pave the way toward recovery**



Three key products

Establish the Group's revenue base through sales expansion
Expand to 250 billion yen (FY2027)

Regenerative medicine/cell therapy

Start the iPS cell-based drug business with the approval and launch of iPS-PD
Expand the business in collaboration with RACTHERA

Oncology

Dedicate resources as a top priority and promote the fastest development (by leveraging partnerships)
enzomenib launch, nuvisertib NDA submission (FY2027)

CNS

Resume development using accumulated expertise and key technologies
Expected to become a revenue base after LOE of the three key products

- Stabilize the revenue base through sales expansion of the three key products
- Initiate the construction of the Value Creation Cycle through the commercialization of Regenerative medicine/cell therapy and Oncology businesses

Progress Toward the Milestones Set for FY2025

1. Progress in Oncology (details will be provided in the next section)

- ✓ Enzomenib (DSP-5336) : Initiated the confirmatory part of the monotherapy Ph2 study for relapsed/refractory acute leukemia with KMT2A rearrangements, aiming for approval in Japan and the U.S.
  Presented the latest data, including results from combination therapy with venetoclax/azacitidine (Ven/Aza), at ASH 2025
- ✓ Nuvisertib (TP-3654) : Advanced Ph1/2 study of monotherapy and combination therapy with momelotinib for relapsed/refractory myelofibrosis, aiming for approval in Japan and the U.S
  Presented the latest data at ASH
- ✓ SMP-3124：Advanced the Ph1/2 study

2. Progress in Regenerative Medicine/Cell Therapy

- ✓ Raguneprocel: Submitted the manufacturing and marketing authorization application in Japan in August 2025 under the Sakigake Designation, and scheduled for review at the Regenerative Medicine and Biologics Committee on Feb. 19, 2026
  Advanced the investigator-initiated clinical study using non-frozen cells and the company-sponsored clinical study using cryopreserved cells in the U.S.
- ✓ HLCR011 (retinal pigment epithelial tear, Japan) and DSP-3077 (retinitis pigmentosa, U.S.): Advanced company-sponsored clinical studies

3. Infectious Diseases

- ✓ Universal Influenza Vaccine (fH1/DSP-0546LP): Observed acceptable tolerability of the novel adjuvant and increases in LAH antibody titers in the interim analysis of the Ph1 study. Continued analyses of cross-reactivity and viral activity

Development Pipeline (as of January 30, 2026)

Area	Generic name/Product code	Mechanism of action, etc.	Planned indication(s)	Development stage
Psychiatry & Neurology	DSP-0038	Serotonin 5-HT_{2A} receptor antagonist and serotonin 5-HT_{1A} receptor agonist	Alzheimer's disease psychosis	Phase 1
	DSP-0187*	Selective orexin 2 receptor agonist	Narcolepsy	Phase 1
	DSP-3456	Metabotropic glutamate receptor 2/3 negative allosteric modulator (mGluR2/3 NAM)	Treatment resistant depression	Phase 1
	DSP-0378	Gamma-aminobutyric acid (GABA) $_A$ receptor positive allosteric modulator	Progressive Myoclonic Epilepsy Developmental Epileptic Encephalopathy	Phase 1
	DSP-2342	Serotonin 5-HT_{2A} and 5-HT_7 receptor antagonist	To be determined	Phase 1
	CT1-DAP001/DSP-1083 (Japan)	Allogeneic iPS [induced pluripotent stem] cell-derived dopaminergic neural progenitor cells	Parkinson's disease/Investigator-initiated study	MAA submitted in August 2025
	CT1-DAP001/DSP-1083 (U.S.)	Allogeneic iPS cell-derived dopaminergic neural progenitor cells	Parkinson's disease/Investigator-initiated study, Company- sponsored clinical study	Phase 1/2
	HLCR011(Japan)	Allogeneic iPS cell-derived retinal pigment epithelial cells	Retinal pigment epithelium tear	Phase 1/2
	DSP-3077(U.S.)	Allogeneic iPS cell-derived retinal sheet	Retinitis pigmentosa	Phase 1/2
Oncology	Enzomenib/DSP-5336	Selective menin inhibitor	Acute leukemia	Phase 2
	Nuvisertib/TP-3654	PIM1 kinase inhibitor	Myelofibrosis	Phase 1/2
	SMP-3124	CHK1 inhibitor	Solid tumors	Phase 1/2
	DSP-0390	EBP inhibitor	Glioblastoma	Phase 1
Others	KSP-1007	β-lactamase inhibitor	Complicated urinary tract and intraabdominal infections, Hospital-acquired bacterial pneumonia	Phase 1
	fH1/DSP-0546LP	Split, Adjuvanted vaccine	Influenza virus prophylaxis	Phase 1

*Development rights: Japan, China, and certain Asian countries

Sumitomo Pharma

Basic Strategy and Key Success Factors (KSFs)

Drive the Value Creation Cycle by discovering new value through in-house drug discovery research and creating and maximizing value through clinical development

Basic Strategy

1. Leverage our strengths in small- to medium-molecule drug discovery and regenerative medicine as core modalities, while maximizing and accelerating opportunities through strategic focus on Oncology and CNS as our priority disease areas
2. Prioritize early detection of patient signals, identify the Value Inflection Point, and execute agile exit strategies

Our KSFs

1. Define priority disease areas and build long-term expertise within these areas to strengthen organizational execution capability and increase the probability of success
2. Define core functions, enhance their capabilities, and gain flexibility to adapt to change in order to strengthen competitiveness
3. Reduce R&D risk through collaborative research and development, while leveraging partnerships to validate and strengthen our organizational execution capability

SMP's Priority Disease Areas and Breakthrough Drug Discovery Capabilities

Priority Disease Areas: Oncology and CNS

1. Capitalize on Market Size and Medical Needs

- Capitalize on the large market size and high unmet medical needs in each area by identifying new opportunities for **expanding opportunities for use of small molecules and regenerative cell therapies**

2. Fully Leverage Our Drug Discovery Strengths (see right figure)

- Leverage strong capabilities in the design and development of synthetic small- to medium-molecule compounds **to address highly challenging molecular targets**
- Target areas where conventional modalities have struggled by leveraging our technological strengths **through the enhanced functionality of small-molecule compounds and cutting-edge iPS cell–based modalities**
- Enhance the probability of clinical success by leveraging robust translational capabilities, including PDX models*, non-rodent nonclinical evaluation systems, and iPSC-derived disease models

3. Ensure Continuity of the R&D Pipeline

- Further expand and reinforce the oncology R&D pipeline **centered on ORGOVYX®, enzomenib, and nuvisertib**
- **Advance the regenerative cell product raguneprocel toward commercialization** and establish a sustainable CNS pipeline

FDA Priority Review Designation Track Record (Since 2012)

• **Nuvisertib**	Oncology Fast Track (2025)
• **Enzomenib**	Oncology Fast Track (2024)
• **KSP-1007**	Infectious Diseases Fast Track (2022)
• **DSP-7888**	Oncology Breakthrough Therapy (2020)
• **Ulotaront**	CNS Breakthrough Therapy (2019)

✓ Pursue the potential of small-molecule drug discovery amid declining capabilities across Japanese pharma, and maintain a top-tier position in Japan ,building on a consistent track record of FDA Priority Review designations (synthetic products: 4 small molecules, 1 synthetic peptide)

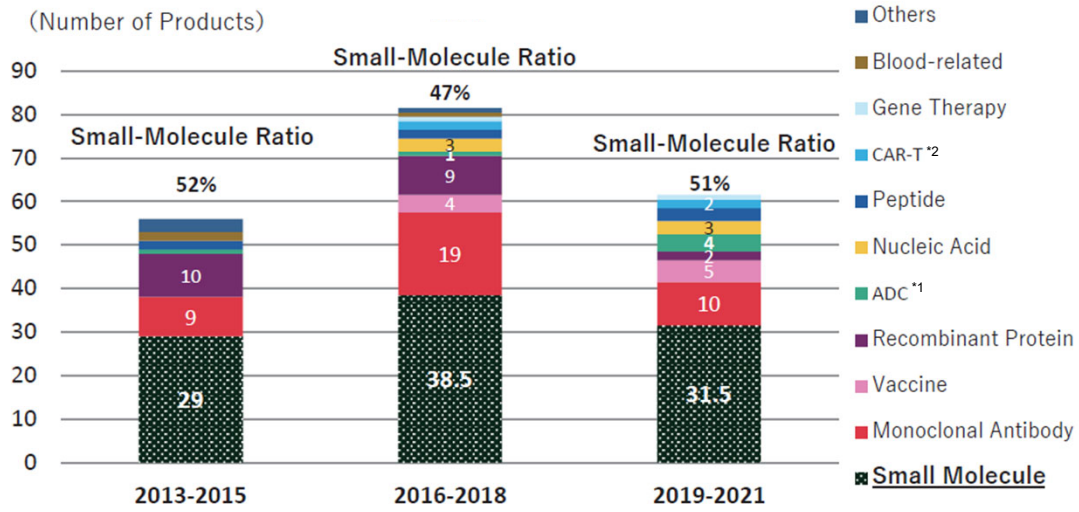
* PDX models: Patient-Derived Xenograft models, A preclinical model in which tumor tissue derived from a patient is transplanted into an immunodeficient mouse to evaluate drug efficacy

OPIR Views and Actions No.70 Nov. 2023; Nationality of Patent-Generating Institutions for NME Approvals in Japan, the U.S., and Europe — Comparison of Approved New Molecular Entity Pharmaceuticals Containing New Active Substances in Japan, the U.S., and Europe —

Source: Prepared by the Office of Pharmaceutical Industry Research (OPIR) based on publicly available information from PMDA, FDA, and EMA, and on Clarivate Analytics Cortellis Competitive Intelligence

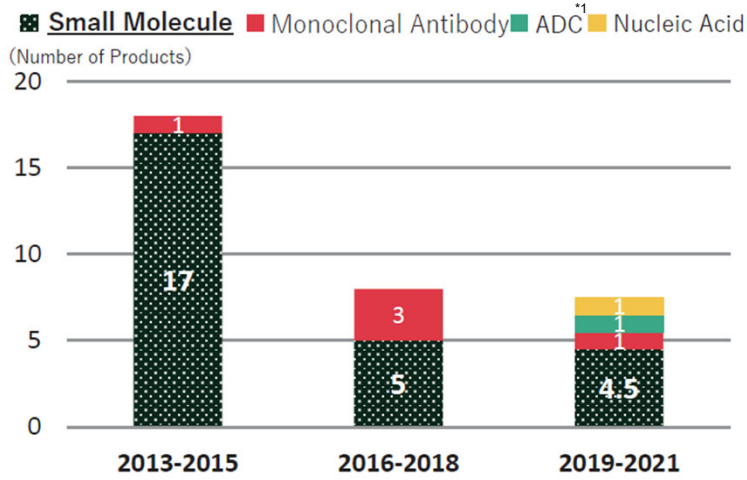
Competitiveness Comparison of Synthetic Small-Molecule Drug Discovery (U.S. vs Japan)

Annual Trend of Modalities in the U.S.



*1 ADC: Antibody-drug conjugate
*2 CAR-T: Chimeric Antigen Receptor T-cell

Annual Trend of Modalities in Japan



Note1: The numbers represent the count of products. When multiple institutions are listed as applicants, the count is evenly allocated by nationality.
Note2: Products that were approved in two or more regions (Japan, the U.S., and Europe) and received their first approval from any regulatory authority in or after 2013.

U. S.: Continually pursuing the potential of small-molecule drug discovery

➢ Despite the increasing variety of new modalities, the number of approvals for synthetic small-molecule drugs has been maintained

Japan: A significant decline in synthetic small-molecule drug discovery capability following a shift toward new modalities

➢ Although new modalities such as antibodies have been adopted, They have not filled the gap and approvals have declined overall

Expansion Strategy in Oncology

- ✓ Leverage our in-house products, pipeline assets, and technology platforms **to drive both pipeline enhancement and the creation of next-generation therapies**
- ✓ In parallel, explore new targets and technological foundations to **build R&D structure capable of sustainable growth**

Leverage Our In-House Products

Tier 01

Create next-generation therapies originating from ORGOVYX®

▶

Ensure continuity in the prostate cancer franchise

Leverage Our In-House Pipeline

Tier 02

Identify new indication opportunities for enzomenib and nuvisertib

▶

Expand the hematologic malignancy pipeline

Leverage Our In-House Technology Platform (Liposomal NM*)

Tier 03

Advance development of SMP-3124

- Verify technological platforms
- Validate targets of encapsulated compounds

▶

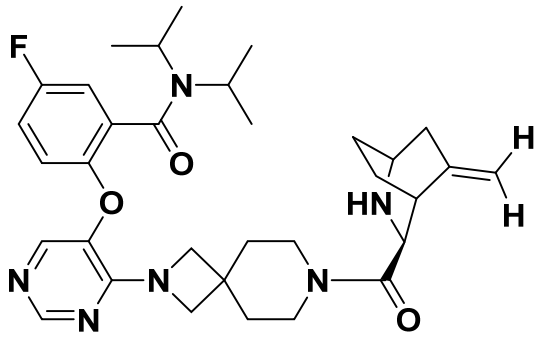
Drive expansion from both the “technology” and “target” perspectives

Sumitomo Pharma

* Liposomal NM: Liposomal Nanomedicine

Enzomenib（DSP-5336）

Leveraged co-creation with academia as a starting point, combining our precise compound-design and synthesis capabilities with forward-looking competitive profile design to generate well-differentiated assets



- A small-molecule compound **with inhibitory protein-protein binding activity for a highly challenging target**
- Compound design with a complex structure **that breaks from conventional norms for orally available small molecules**
- **A robust patent portfolio** amid intense competition

Discovery of drug targets driven by co-creation with academia

- ✓ Engaged Dr. Akihiko Yokoyama, an early pioneer in menin science, as Principal Investigator through the DSK Project*1

Compound design capabilities and MD simulation*2

- ✓ Discovered new target binding sites and designed compounds with efficient binding properties
- ✓ Organic synthesis capability enabled the practical production of industrially scalable compounds

Profile design anticipated intense competitive environments

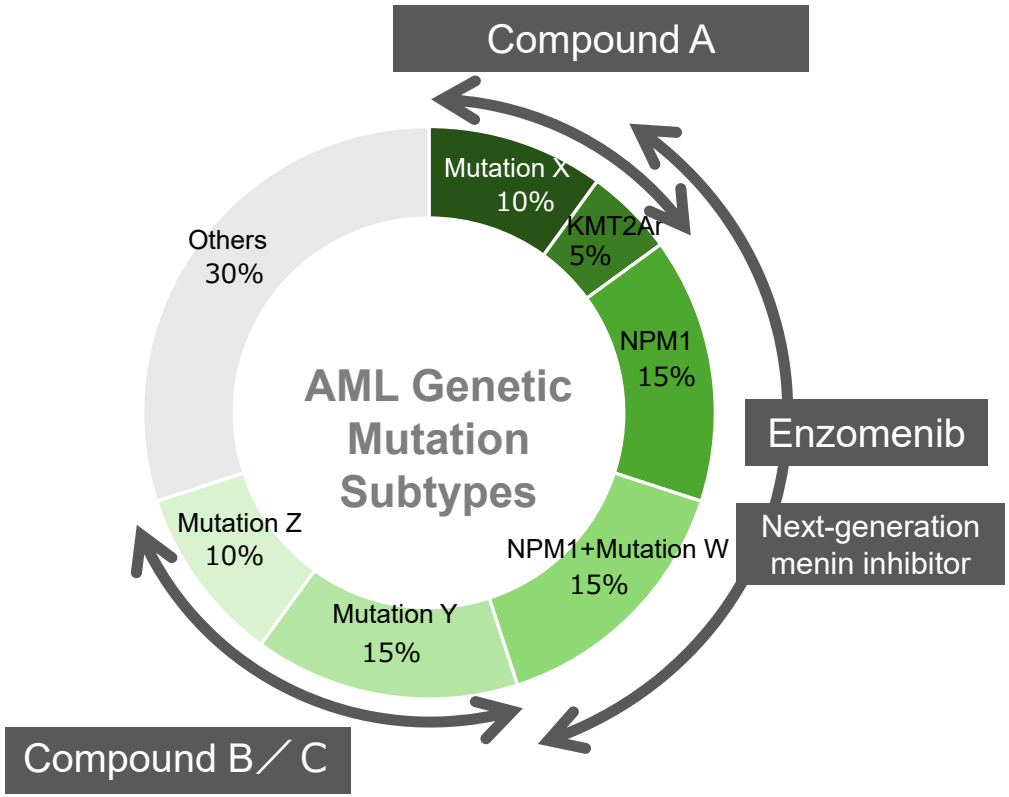
- ✓ Prioritized strong pharmacological activity while minimizing cardiotoxicity in light of competitive pressures
- ✓ Developed biomarkers to enable patient stratification and clinical efficacy prediction

*1 DSK Project: A cancer drug discovery project under an industry–academia collaboration between Kyoto University and Sumitomo Pharma, from fiscal year 2011 (start of the first term) to fiscal year 2020 (end of the second term)

*2 MD simulation: Molecular Dynamics simulation A computational method that reproduces the movements of compounds and proteins at the atomic level and evaluates binding modes and stability

Drug Discovery Coverage for Acute Myeloid Leukemia (AML)

Expect our pipeline to address approximately 70% of AML genetic mutation subtypes, building a comprehensive portfolio for AML treatment



Enzomenib

- KMT2A rearrangements (KMT2Ar) and NPM1 mutations (NPM1m)
- Monotherapy and combination therapy with VEN/AZA*
- Ph2 study ongoing

Next-generation menin inhibitor:

- Non-response/relapse (acquired resistance) to menin inhibitors
- Monotherapy and combination therapy
- Nonclinical studies in progress

Compound A / B / C:

- Treatment-resistant (Mutations X / Y / Z)
- Monotherapy and combination therapy
- Nonclinical studies in progress

*Ven: Venetoclax / Aza: Azacitidine

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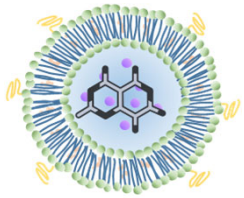
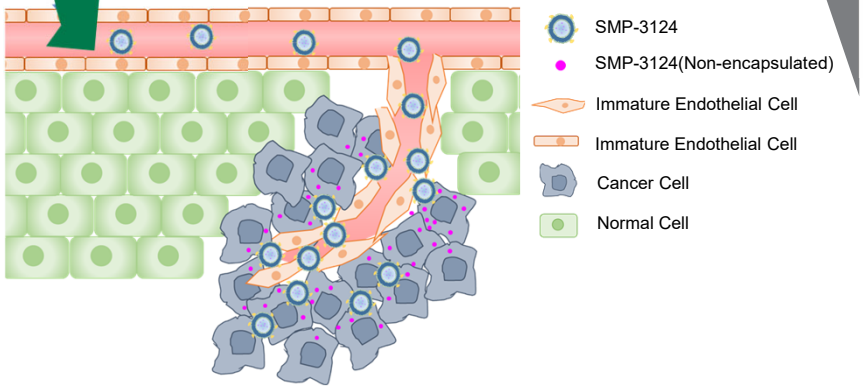
SMP-3124

- ✓ **Leveraged established precedents in product design to improve the probability of clinical success**
- ✓ **Validated Liposomal Nanomedicine technologies through the development of SMP-3124**



- Encapsulated a small-molecule CHK1 inhibitor, whose target relevance in tumors has been clinically validated, into a liposomal formulation
- Utilized liposome/nanoparticle platforms with substantial accumulated clinical evidence



Discovery of drug targets driven by co-creation with academia

- ✓ Through collaborative research with the Department of Obstetrics and Gynecology at Kyoto University (Dr. Mandai and Dr. Hamanishi), evaluated our kinase HTS library using clinical ovarian cancer specimens
- ✓ Identified CHK1 inhibition as an optimal therapeutic target for ovarian cancer

Profile design based on prior precedents

- ✓ Recognized that the primary development challenge of first-generation CHK1 inhibitors was treatment-related side-effects rather than insufficient efficacy

Liposomal Nanomedicine technology

- ✓ Achieved sustained in-vivo release and preferential tumor accumulation to maintain local drug concentrations, reduce side effects, and enhance efficacy
- ✓ Capability for the design and synthesis of encapsulated compounds (selective CHK1 inhibitors) suited to the physicochemical properties of liposomal formulations
- ✓ Expanded applicability across a broad range of therapeutic targets through these capabilities

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Drug Discovery Strategy in CNS

- ✓ **CNS area continues to show persistent unmet medical needs, as therapeutic development has long since stagnated**
- ✓ **Although CNS drug discovery is highly challenging and presents high entry barriers for competitors, steadily advancing our programs by leveraging our extensive experience, robust assets, and unique strengths**

Compounds with strong predicted clinical success

Tier 01

- DSP-0378（$GABA_A$ receptor PAM*）
- Candidate compound for improving motor symptoms in Parkinson's disease

- ✓ Ensure clear evidence supporting therapeutic effects
- ✓ Leverage unique mechanisms that differentiate from existing therapies
- ✓ Verify effectiveness concisely by using objective measures

Disease-modifying therapies for neurodegenerative diseases

Tier 02

- Multiple disease-modifying candidates centered on Parkinson's disease

- ✓ Identify actionable drug targets informed by advancing disease biology
- ✓ Leverage the advantages of small molecules in removing intracellular brain aggregates
- ✓ Detect early efficacy signals in patients using advanced biomarker technologies

Therapies for psychiatric symptoms associated with neurological diseases

Tier 03

- DSP-2342（$5\text{-}HT_{2A}$・$5\text{-}HT_7$ receptor antagonist）
- Multiple candidate compounds for improving multiple psychiatric symptoms

- ✓ Apply the experience and assets accumulated from the drug discovery of LATUDA® and ulotaront to neurodegenerative diseases
- ✓ Detect early efficacy signals by leveraging biomarkers in biologically homogeneous disorders

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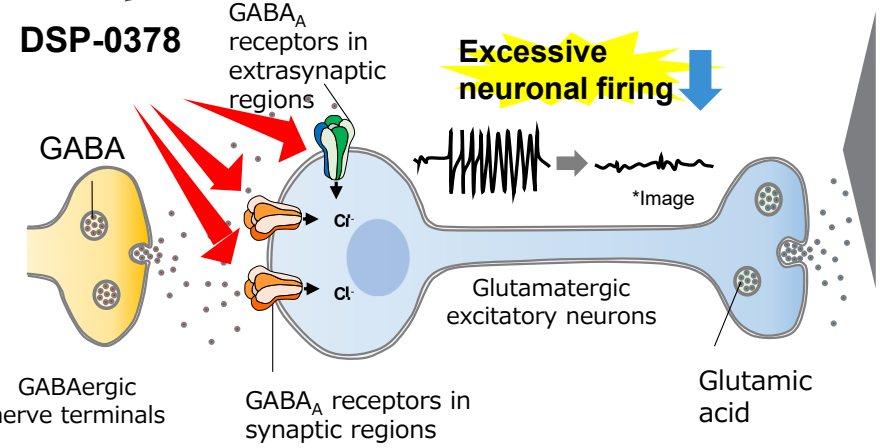
*PAM: Positive Allosteric Modulator

DSP-0378

- ✓ **Applied a proprietary symptom-based screening strategy to a compound library designed for antiepileptic drugs**
- ✓ **Identified a unique mechanism of action against a clinically validated therapeutic target**

- ✓ **Unique pharmacological effects**
- ✓ **Favorable brain penetration**
- ✓ **Desirable safety profile**



Experience from previous antiepileptic drug programs

- ✓ Leveraged compound libraries optimized for antiepileptic drugs discovery, built through development of EXCEGRAN® and DSP-0565
- ✓ Utilized these libraries to efficiently identify optimal candidate compounds

Identification of clinically validated targets through a proprietary pharmacological evaluation strategy

- ✓ Identified optimal candidate compounds for refractory epilepsy through phenotype screening*1 that reflects clinical manifestations and subsequently characterized their mechanism and site of action through multiple pharmacological evaluation systems
- ✓ Observed unique activity on the clinically validated target, the GABA$_A$ receptor, that is distinct from existing therapeutics

Design of translational biomarkers

- ✓ Identified a translational biomarker (EEG) using NHP*2 models that reliably translates pharmacological effects into clinical outcomes

*1 Phenotype screening: A method for narrowing down compounds by evaluating their effects on disease-relevant features observed in cells or animal models
*2 NHP: Non-human primate

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R&D Goals for the Medium- to Long-Term

FY2025 | FY2026 | FY2027 | FY2028–FY2030 | FY2031–FY2033

Sumitomo Pharma's R&D capabilities

- Capabilities of medicinal chemistry for challenging targets
- World-leading technology and experience in iPS cells
- R&D cycle based on data and expertise obtained from enzomenib and nuvisertib
- Drug discovery and translational research platform in CNS
- AI and digital technology to drive innovation

+

Development and CMC organization that can execute through to commercialization

World's first practical use of iPS cell-derived products

✓ Obtain domestic conditional and time-limited approval for iPS-PD program

NDA submission and launch of two oncology compounds

✓ Launch of enzomenib
✓ NDA submission of nuvisertib

Launch of CNS pipelines

✓ iPS-PD program in the U.S.
✓ DSP-0378
✓ Therapeutic agent for improving symptoms in Parkinson's disease

Expansion of the regenerative medicine/cell therapy business

✓ HLCR011, DSP-3077, etc.

Launch of next-generation pipelines

✓ Hematological malignancies, Rare neurological & degenerative diseases
✓ Launch of SMP-3124

Commercialization by partners
Ulotaront, DSP-0187, infectious disease products, etc.

Two Key Oncology Compounds in Development

Global Oncology Strategy Lead
Lead Senior Vice President, Sumitomo Pharma America, Inc.

Masashi Murata

Sumitomo Pharma

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- ✓ In parallel, explore new targets and technological foundations to **build R&D structure capable of sustainable growth**

Leverage Our In-House Products

Tier 01

Create next-generation therapies originating from ORGOVYX®

▶

Strengthen continuity in the prostate cancer franchise

Leverage Our In-House Pipeline

Tier 02

Advance indication acquisition and expansion for enzomenib and nuvisertib

▶

Expand the hematologic malignancy pipeline

Leverage Our In-House Technology Platform (Liposomal NM*)

Tier 03

Advance development of SMP-3124

- Verify technological platforms
- Validate targets of encapsulated compounds

▶

Drive expansion from both the “technology” and “target” perspectives

Sumitomo Pharma

* Liposomal NM: Liposomal Nanomedicine

Enzomenib

Mechanism of action	Selective menin inhibitor
Development stage	Phase 2
Planned indication	Acute leukemia（KMT2A rearrangements, NPM1 mutations）

Enzomenib

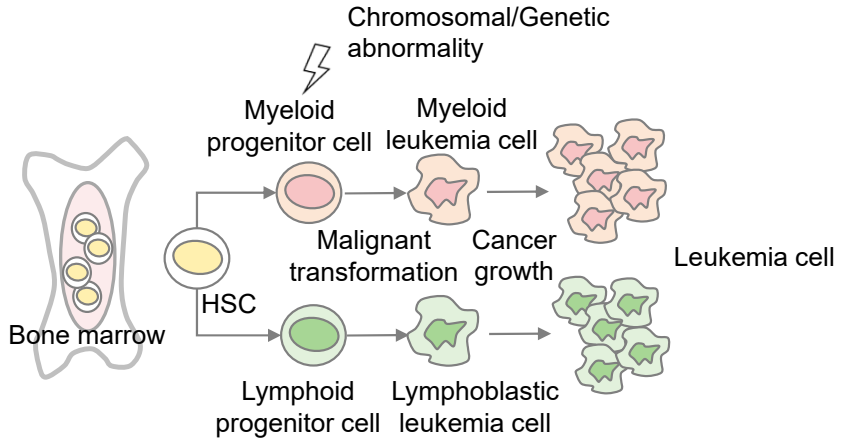
Disease Background of Acute Myeloid Leukemia (AML)

- ✓ Progresses rapidly, **requiring urgent diagnosis and treatment**
- ✓ **Relapse in approximately 50% of** patients resulting in a poor prognosis with a median survival of **5–6 months**

Pathophysiology / Clinical Symptoms

Immature leukemic cells proliferate**, leading to the rapid progression of systemic clinical symptoms**, as follows:

- Fever, fatigue, dizziness, headache, vomiting
- Anemia, bleeding, infections
- Neuropsychiatric symptoms
- Lymphadenopathy, hepatosplenomegaly



*Hematopoietic stem cell

Prognosis / Outcomes

- **Relapse in approximately 50% of patients** even after achieving complete remission with treatment
- Worse prognosis depending on the genetic mutations present in individual patients
- **Low 5-year overall survival rate in adults of approximately 30%** (high proportion of elderly patients, limited tolerance to intensive chemotherapy)
- **5-year overall survival rate of approximately 65% in pediatric and adolescent patients**

	U.S.	Japan
New Patients/year*	Appox.21,000 /year	Approx.8,000 /year

* Estimated based on the GlobalData Epidemiology Database (2025)

Enzomenib

Current Treatment Landscape and Unmet Medical Needs

- ✓ **Limited effective therapies for relapsed/refractory cases**, few treatment options for elderly or transplant-ineligible patients.
- ✓ Increasing availability of mutation-specific targeted therapies, **yet inadequate treatment available**

Induction Therapy

Can the patient tolerate intensive chemotherapy?

Yes（Fit patients）	No（Unfit patients）
• **7+3 regimen (intensive chemotherapy)** • Combination therapy with molecularly targeted therapy • Administration for several weeks	• **Ven/Aza[*1] combination therapy** • Combination therapy with molecularly targeted therapies • Administration for several weeks

Consolidation Therapy

- **Administer chemotherapy for several months to achieve complete eradication of leukemia cells**
- High-dose cytarabine (＋ molecularly targeted therapy)
- Allogeneic hematopoietic stem-cell transplantation in cases of high relapse risk

Maintenance Therapy

- Oral Aza or molecularly targeted therapy
- **Long-term administration (2–3 years) to reduce the risk of relapse**
- Treatment option for patients ineligible for intensive chemotherapy or transplantation

Relapse / Refractory Treatment

- Re-induction

*1 Ven: Venetoclax / Aza: Azacitidine

Genetic mutations	Proportion in newly diagnosed AML[*2]	Approved molecular targeted therapy[*2]
IDH1/2	18-35%	3
NPM1	25-35%	2
FLT3	25-31%	3
DNMT3A	14-23%	-
CTNNB1	22%	-
CEBPA	5-20%	-
TP53	10-15%	-
RUNX1	10-15%	-
SRSF2	12-13%	-
KMT2Ar	3-10%	1

*2 Internal survey (as of January 2026)

Enzomenib

Development Strategy for Enzomenib

- ✓ **Prioritize obtaining approval for enzomenib monotherapy in patients with relapsed/refractory AML**
- ✓ In parallel, **pursue indication expansion into frontline AML, including use in induction therapy and maintenance therapy**

Induction Therapy

Can the patient tolerate intensive chemotherapy?

Yes（Fit patients）

- **7+3 regimen (intensive chemotherapy)**
- Combination therapy with molecularly targeted therapy
- Administration for several weeks

No（Unfit patients）

- **Ven/Aza*combination therapy**
- Combination therapy with molecularly targeted therapies
- Administration for several weeks

Consolidation Therapy

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- Treatment options for patients ineligible for intensive chemotherapy or transplantation

Relapse / Refractory Treatment

- Re-induction

Aim to obtain approval for Ven/Aza combination therapy

Under consideration for development

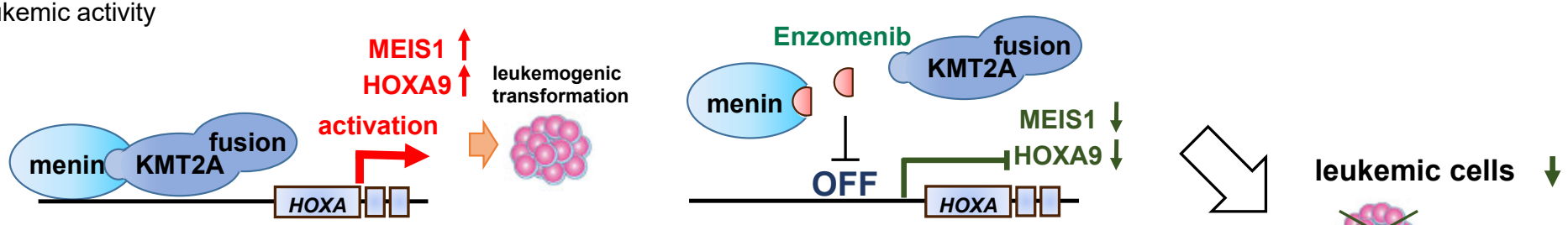
First planned indication (NDA submission planned in Japan and U.S.)

* Ven: Venetoclax / Aza: Azacitidine

Clear Target Specificity Based on Disease Mechanisms

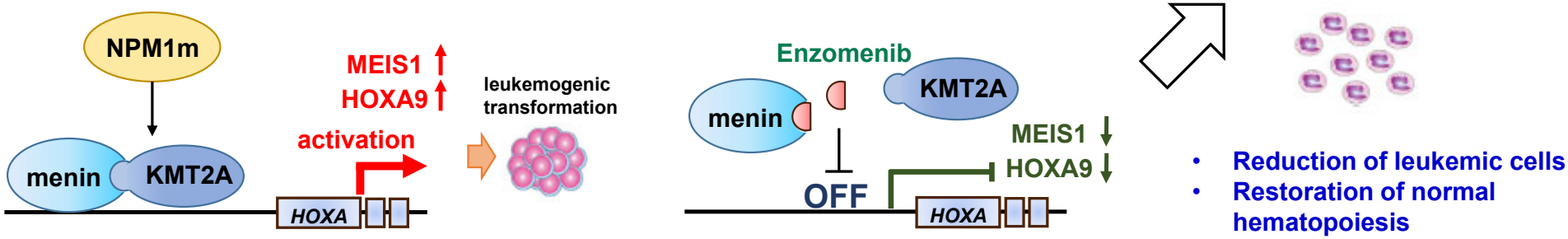
➢ KMT2A-rearranged leukemia

KMT2A fusion proteins drive leukemogenesis by interacting with menin and upregulating HOXA genes (e.g., HOXA9)
Enzomenib **inhibits the menin–KMT2A interaction and suppresses the aberrant transcriptional activity in leukemic cells**, thereby exerting anti-leukemic activity



➢ NPM1-mutated leukemia

In AML with NPM1 mutations, leukemogenesis is driven by upregulation of HOXA genes through the interaction between wild-type KMT2A and menin. Enzomenib **inhibits the wild-type KMT2A–menin interaction and suppresses the aberrant transcriptional activity in leukemic cells**, thereby exerting anti-leukemic activity

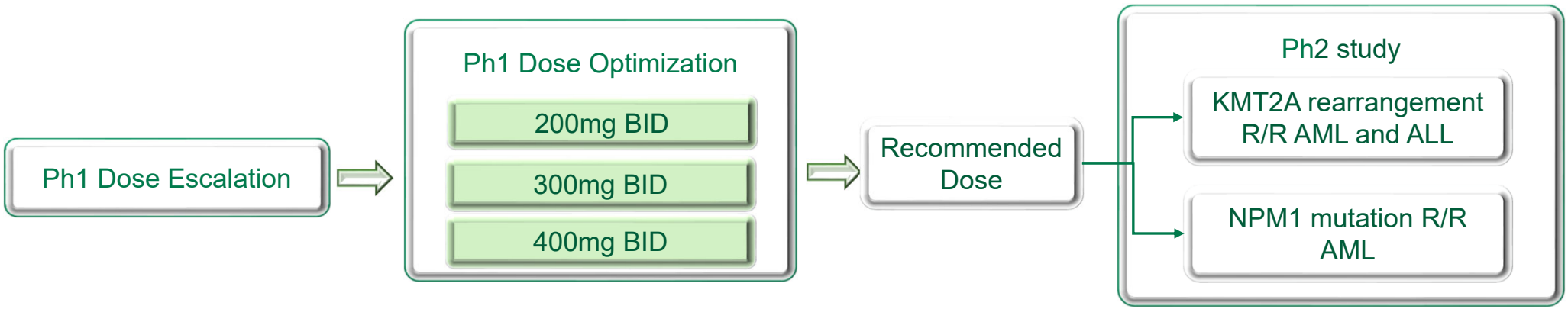


- **Reduction of leukemic cells**
- **Restoration of normal hematopoiesis**

Enzomenib

Oral presentation data at ASH 2025 Annual Meeting
Data cut-off: October 4, 2025

Monotherapy for Relapsed/Refractory Acute Leukemia



BID: Twice Daily
R/R: Relapsed/Refractory
AML: Acute Myeloid Leukemia
ALL: Acute Lymphoblastic Leukemia

CR: Complete Remission
CRh: Complete Remission with Partial Hematologic Recovery
CRi: Complete Remission with Incomplete Blood Count Recovery
MLFS: Morphologic Leukemia-Free State
CRc: CR, CRh or CRi
ORR: CR, CRh, Cri, or MLFS
OS: Overall Survival
EFS: Event-Free Survival

【Primary Endpoint】CR+CRh rate

【Secondary Endpoints】

CRc, ORR, Time to CR/CRh, Time to ORR, Duration of CR/CRh, Duration of ORR, Transfusion independence, OS, EFS

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Data cut-off: October 4, 2025

Monotherapy for Relapsed/Refractory Acute Leukemia

Safety: Monotherapy

Favorable tolerability of monotherapy

✓ No dose-limiting toxicities (DLTs), treatment-related deaths, or treatment discontinuations were observed, indicating favorable tolerability

Treatment-Emergent Adverse Events (TEAEs) with an incidence of ≥20% (n=116)

Preferred Term	Any grade	Gr ≥ 3
HEMATOLOGIC		
Febrile neutropenia	32 (27.6%)	31 (26.7%)
Platelet count decreased	26 (22.4%)	25 (21.6%)
Neutrophil count decreased	25 (21.6%)	24 (20.7%)
NON-HEMATOLOGIC		
Nausea	46 (39.7%)	4 (3.4%)
Vomiting	32 (27.6%)	2 (1.7%)
Diarrhea	30 (25.9%)	1 (0.9%)
Sepsis	29 (25.0%)	28 (24.1%)
Decreased appetite	28 (24.1%)	4 (3.4%)
Headache	28 (24.1%)	2 (1.7%)
Hypokalemia	27 (23.3%)	0

TEAEs related to enzomenib with an incidence of ≥20% (n=116)

Preferred Term	Any grade	Gr ≥ 3
HEMATOLOGIC		
Platelet count decreased	9 (7.8%)	8 (6.9%)
Neutrophil count decreased	8 (6.9%)	8 (6.9%)
Leukocytosis	7 (6.0%)	3 (2.6%)
NON-HEMATOLOGIC		
Nausea	19 (16.4%)	1 (0.9%)
Differentiation syndrome	15 (12.9%)	9 (7.8%)
Vomiting	13 (11.2%)	1 (0.9%)
Dysgeusia	7 (6.0%)	0
Diarrhea	6 (5.2%)	0

Note: QTc interval prolongation was reported in 9.5% of patients (Grade 3 in 2.6%)

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Data cut-off: October 4, 2025

Efficacy: Monotherapy

Monotherapy for Relapsed/Refractory Acute Leukemia

Evidence suggestive of efficacy with monotherapy

- ✓ KMT2A rearrangement AML/ALL (300 mg BID): CR+CRh rate: 40.0%; median OS: 11.8 months
- ✓ NPM1 mutation AML: CR+CRh rate: 37.5–50.0%

	KMT2A rearrangement
	300mg BID n=15
Overall Response Rate (CR/CRh/CRi/MLFS)	73.3%
Composite CR rate (CR/CRh/CRi)	60%
CR+CRh rate	40%
Median Time to CR/CRh	1.6 months
Duration of CR/CRh	12.5 months (n=11)
Median Overall Survival	11.8 months

	NPM1 mutation		
	200mg BID n=10	300mg BID n=7	400mg BID n=8
Overall Response Rate (CR/CRh/CRi/MLFS)	60%	57.1%	37.5%
Composite CR rate (CR/CRh/CRi)	50%	42.9%	37.5%
CR+CRh rate	50%	42.9%	37.5%
Median Time to CR/CRh	3.7 months		
Duration of CR/CRh	5.7 months (n=11)		
Median Overall Survival	8.5 months		

BID: Twice daily
CR: Complete Remission
CRh: Complete Remission with Partial Hematologic Recovery
CRi: Complete Remission with Incomplete Blood Count Recovery
MLFS: Morphologic Leukemia-Free State

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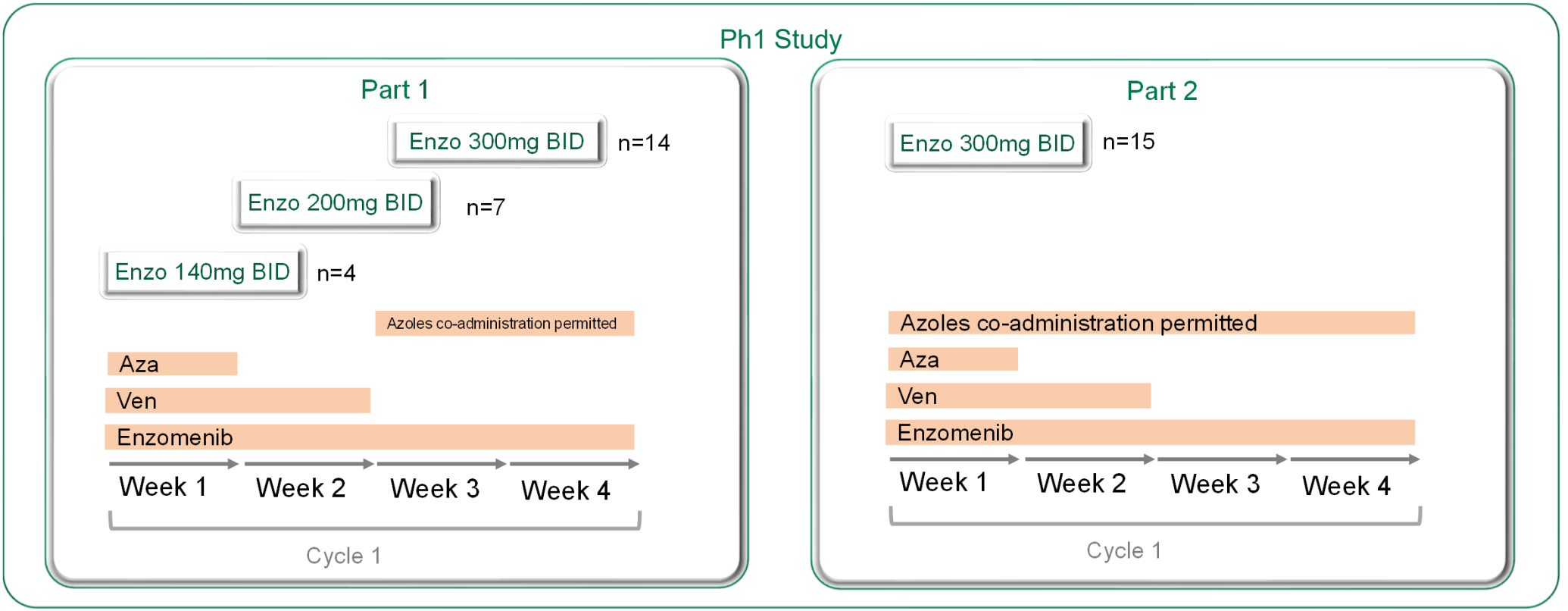
Combination Therapy with Ven/Aza for Relapsed/Refractory AML



【Primary Endpoints】 Safety, Tolerability, Pharmacokinetics, etc.
【Secondary Endpoints】 CRc, ORR, Time to CR/CRh, Time to ORR, Duration of CR/CRh, Duration of ORR, Transfusion independence, OS, EFS, etc.

Enzo: Enzomenib Ven: Venetoclax Aza: Azacitidine
BID: Twice daily
CR: Complete Remission
CRh: Complete Remission with Partial Hematologic Recovery
CRi: Complete Remission with Incomplete Blood Count Recovery
MLFS: Morphologic Leukemia-Free State
ORR: CR, CRh, Cri, or MLFS

Oral presentation data at ASH 2025 Annual Meeting
Data cut-off: October 4, 2025

Combination Therapy with Ven/Aza for Relapsed/Refractory Acute Myeloid Leukemia

Safety: Combination with Ven/Aza

Favorable tolerability of combination therapy

✓ No dose-limiting toxicities (DLTs), treatment-related deaths, or treatment discontinuations were observed, **indicating a favorable safety profile**

TEAEs with an incidence of ≥25% (n=40)

Preferred Term	Any grade	Gr ≥ 3
HEMATOLOGIC		
Platelet count decreased	21 (52.5%)	18 (45.0%)
WBC count decreased	15 (37.5%)	14 (35.0%)
Neutrophil count decreased	14 (35.0%)	14 (35.0%)
Anemia	10 (25.0%)	8 (20.0%)
Febrile neutropenia	10 (25.0%)	9 (22.5%)
NON-HEMATOLOGIC		
Nausea	19 (47.5%)	0
Constipation	16 (40.0%)	0
Diarrhea	15 (37.5%)	0
Hyperphosphatemia	11 (27.5%)	0
Vomiting	11 (27.5%)	1 (2.5%)
Arthralgia	10 (25.0%)	1 (2.5%)

TEAEs related to either Enzomenib, Ven or Aza with an incidence of ≥15%(n=40)

Preferred Term	Any grade	Gr ≥ 3
HEMATOLOGIC		
Platelet count decreased	18 (45.0%)	16 (40.0%)
WBC count decreased	14 (35.0%)	13 (32.5%)
Neutrophil count decreased	12 (30.0%)	12 (30.0%)
Anemia	9 (22.5%)	7 (17.5%)
Lymphopenia	6 (15.0%)	5 (12.5%)
NON-HEMATOLOGIC		
Nausea	10 (25.0%)	0
Diarrhea	8 (20.0%)	0
AST increased	6 (15.0%)	0
Constipation	6 (15.0%)	0

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Enzomenib

Oral presentation data at ASH 2025 Annual Meeting
Data cut-off: October 4, 2025

Efficacy: Combination with Ven/Aza

Combination Therapy with Ven/Aza for
Relapsed/ Refractory Acute Myeloid Leukemia

Evidence suggestive of combination therapy efficacy for relapsed/refractory AML

- ✓ In relapsed/refractory AML, the ORR was 77%, and the composite CR rate was 50%, indicating a potentially high level of clinical activity
- ✓ Consistent activity was observed across dose levels, supporting the promise of the combination regimen

Overall population	140mg BID + Ven/Aza 100mg n=4	200mg BID + Ven/Aza 100mg n=6	300mg BID + Ven/Aza 100mg n=8	300mg BID + Ven/Aza 50-100mg n=8	Total n=26
	Without azoles	Without azoles	Without azoles	With azoles	
Overall Response Rate (CR/CRh/CRi/MLFS)	100%	83%	62.5%	80%	77%
Composite CR rate (CR/CRh/CRi)	50%	50%	50%	50%	50%

BID: Twice daily
CR: Complete Remission
CRh: Complete Remission with Partial Hematologic Recovery
CRi: Complete Remission with Incomplete Blood Count Recovery
MLFS: Morphologic Leukemia-Free State

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Enzomenib

Potential Best-in-Class Profiles as a Selective Menin Inhibitor

- In the acute leukemia field, although targeted therapies corresponding to genetic mutations have been approved, treatment satisfaction remains insufficient
- **Enzomenib has shown encouraging tolerability and efficacy as monotherapy, as well as favorable safety and efficacy in combination with Ven/Aza**, suggesting a **potentially best-in-class profile**
- **The ongoing confirmatory Phase 2 study will be accelerated** to accelerated with the aim of obtaining approval in Japan and the U.S. for **relapsed/refractory acute leukemia with KMT2A rearrangement or NPM1 mutation** (target launch: FY2027)
- In addition, leveraging the safety and efficacy profiles observed with monotherapy and with Ven/Aza combination, we will pursue further development opportunities such as **expanding indications to newly diagnosed acute leukemia (induction therapy, maintenance therapy)** and **exploring disease areas beyond acute leukemia**.

Nuvisertib

Mechanism of action	PIM1 kinase inhibitor
Development phase	Phase 1/2
Planned indication	Myelofibrosis（MF）

PIM: Proto-oncogene proviral Integration site for Moloney murine leukemia virus

Nuvisertib

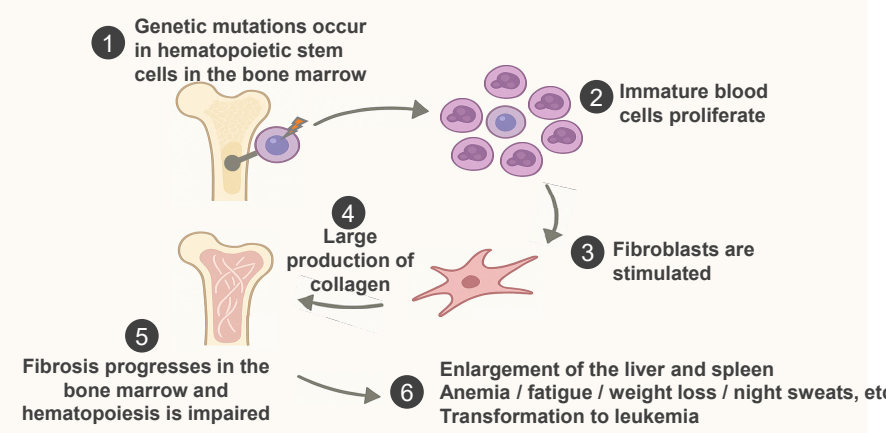
Disease Background of Myelofibrosis (MF)

- ✓ A hematologic cancer caused by genetic mutations in hematopoietic stem cells inducing bone marrow fibrosis and impairing normal hematopoiesis
- ✓ **Progressive symptoms,** including hepatosplenomegaly, general fatigue, bone pain, anemia, and infections, resulting in **significant reduction in quality of life**
- ✓ Regular blood transfusions required in many patients due to anemia, along **with frequent progression to relapsed/refractory stages**

Disease / Symptoms

Abnormal proliferation of hematopoietic stem cells causing fibrosis of the bone marrow, abolishing normal hematopoiesis and inducing extramedullary hematopoiesis in the liver and spleen.

- Hepatosplenomegaly
- Systemic symptoms such as night sweats, fatigue, bone pain
- Moderate to severe anemia, bleeding, susceptibility to infections
- Asymptomatic at diagnosis in approximately 20% of patients



Prognosis / Progression

- Regular blood transfusions required due to anemia
- Frequent progression to relapsed/refractory disease
- Potential transformation to leukemia
- Post-diagnosis survival: U.S. 4–6 years, Japan 3–6 years

	U.S.	Japan
New patients/year*	Approx. 2,200 /year	Approx. 600 /year

* Estimated based on the Global Data Epidemiology Database (2026)

Treatment Options and Unmet Medical Needs

Current standard treatment centered on **JAK inhibitor**
Limited treatment options in patients with low platelet counts, with disease progression leading to **transition to 2L or 3L therapy**
Bone marrow transplantation as the only curative option, with **a strong need for therapies with novel mechanisms of action**

1L (first-line therapy)

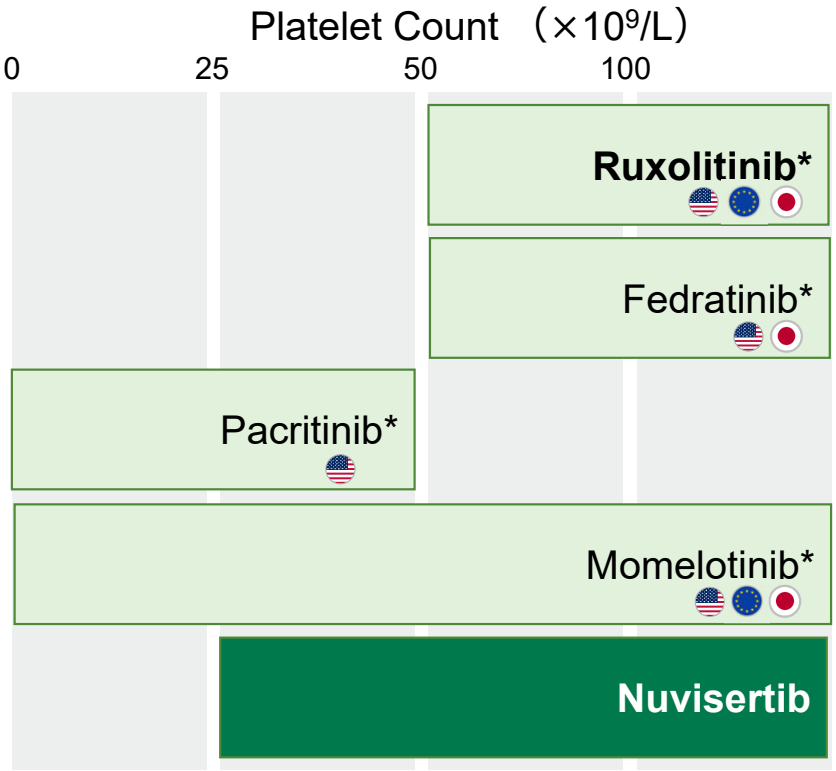
- **Ruxolitinib (a JAK inhibitor) is the most commonly used treatment**
- **Patients with low platelet counts have limited treatment options**
- The treatment duration: approximately 12 months
- Bone marrow transplantation (the only curative treatment) may be considered in patients with adequate age and organ function

2L (second-line therapy)

- **Up to 50% of 1L patients transition to 2L therapy** due to inadequate response or disease progression
- **A JAK inhibitor or investigational agent different from the 1L regimen**
- The treatment duration: 6–9 months
- Bone marrow transplantation is considered for patients with no response or insufficient response to drug therapy, or for those harboring high-risk mutations

3L (refractory/ advanced disease)

- **Up to 23% of 2L patients transition to 3L therapy**
- The treatment duration: up to 10 months
- When symptoms worsen and drug therapy is no longer effective, supportive care (transfusions, nutritional management, infection prevention) is used to maintain quality of life



*Approved JAK inhibitors. Momelotinib was approved in the US and EU in 2023, and in Japan in 2024

Key Characteristics of Nuvisertib

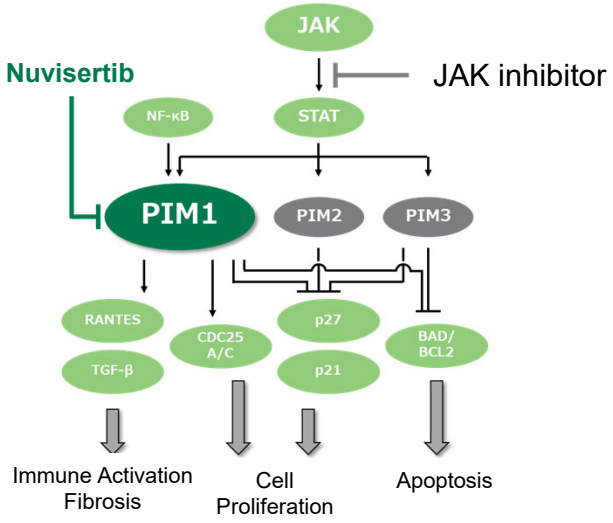
■ **Mechanism of Action and Drug Concept**

- Inhibits PIM1 kinase and acts on multiple pathways involved in the pathogenesis of MF, including not only **the JAK/STAT pathway but also NF-κB**
- Observe promising results such as spleen volume reduction, improvement in bone marrow fibrosis, and prolonged survival in non-clinical models

■ **Points of Differentiation**

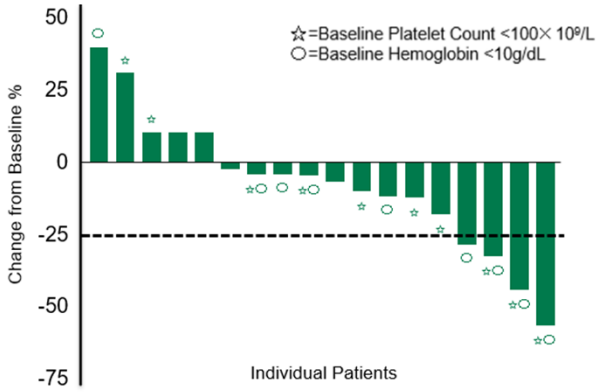
- Possesses a **mechanism distinct from JAK inhibitors**, offering strong potential as a combination therapy
- Exhibits high selectivity for PIM1, with **expectations of reducing the risk of hematologic toxicity**
- Observed **improvements in spleen volume and total symptom score (TSS)** in the Ph1/2 study, even with monotherapy

Drug concept: PIM1 inhibitor

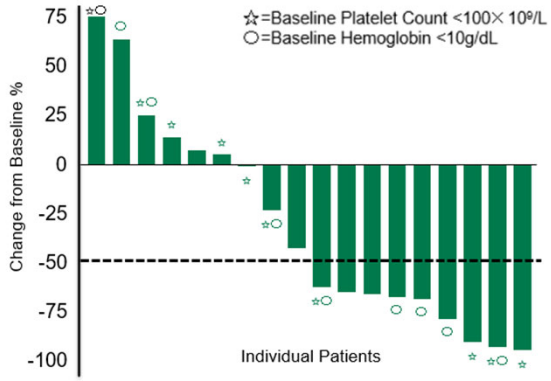


Spleen volume reduction and improvement in total symptom score (TSS) with monotherapy (ASH2025)

Best Changes in Spleen Volume at Any Time (n=20)
720mg BID SVR25: 20% (4/20)



Best Changes in TSS at Any Time (n=20)
720mg BID TSS50: 9/20 (45%)



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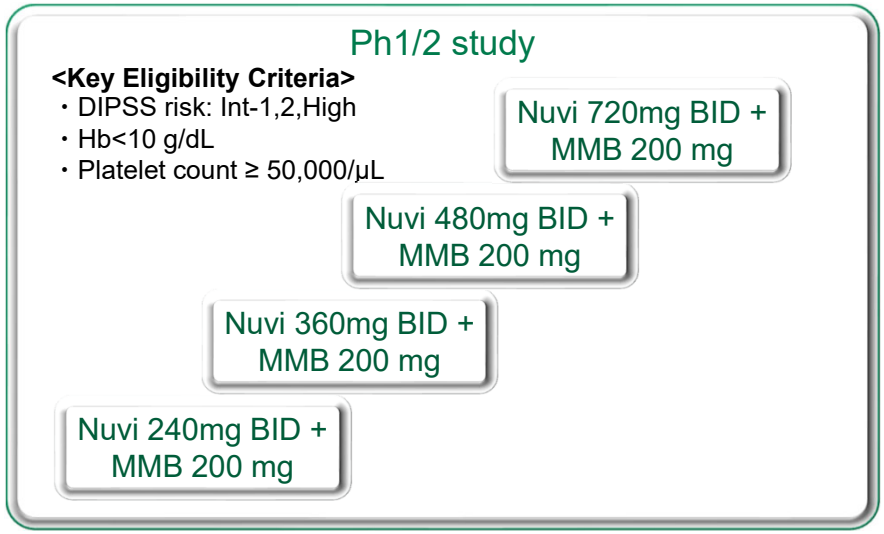
Combination Therapy with Momelotinib for Relapsed/Refractory Myelofibrosis



【Primary Endpoints】
Safety, Tolerability

【Secondary Endpoints】
Spleen volume reduction
Total symptom score (TSS) reduction
Overall survival
Bone marrow fibrosis change
Pharmacokinetics

Nuvi: Nuvisertib
MMB: Momelotinib
BID: Twice daily
DIPSS: Dynamic International Prognostic Scoring System - an international scoring system used to dynamically assess prognosis in myelofibrosis
Int-1: Intermediate-1 (intermediate-risk 1) Int-2: Intermediate-2 (intermediate-risk 2) High: High risk
Hb：Hemoglobin

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Safety: Combination with Momelotinib

Favorable tolerability of combination therapy

- ✓ Among the 18 patients included in the safety evaluation, **only one DLT was observed at 360 mg BID** (thrombocytopenia requiring transfusion)
- ✓ Overall, **the combination with momelotinib is expected to have generally favorable tolerability**
- ✓ The main adverse events were Grade 1–2 gastrointestinal symptoms (diarrhea, nausea, vomiting), which were manageable

Incidence of Dose-Limiting Toxicities (n=18)

Nuvisertib + Momelotinib 200mg QD	n=18	DLT
240mg BID	4	0
360mg BID	8	1 (Thrombocytopenia)
480mg BID	5	0
720mg BID	1	0

BID: Twice daily

TEAEs with an Incidence of ≥20% (n=18)

Preferred Term	Grade1	Grade2	Grade3
Diarrhea	10 (55.6%)	4 (22.2%)	0
Nausea	7 (38.9%)	2 (11.1%)	1 (5.6%)
Vomiting	3 (16.7%)	2 (11.1%)	1 (5.6%)
Fatigue	0	4 (22.2%)	0
Blood creatinine increased	2 (11.1%)	2 (11.1%)	0
Decreased appetite	1 (5.6%)	3 (16.7%)	0
Urinary tract infection	0	4 (22.2%)	0
Thrombocytopenia	0	2 (11.1%)	2 (11.1%)

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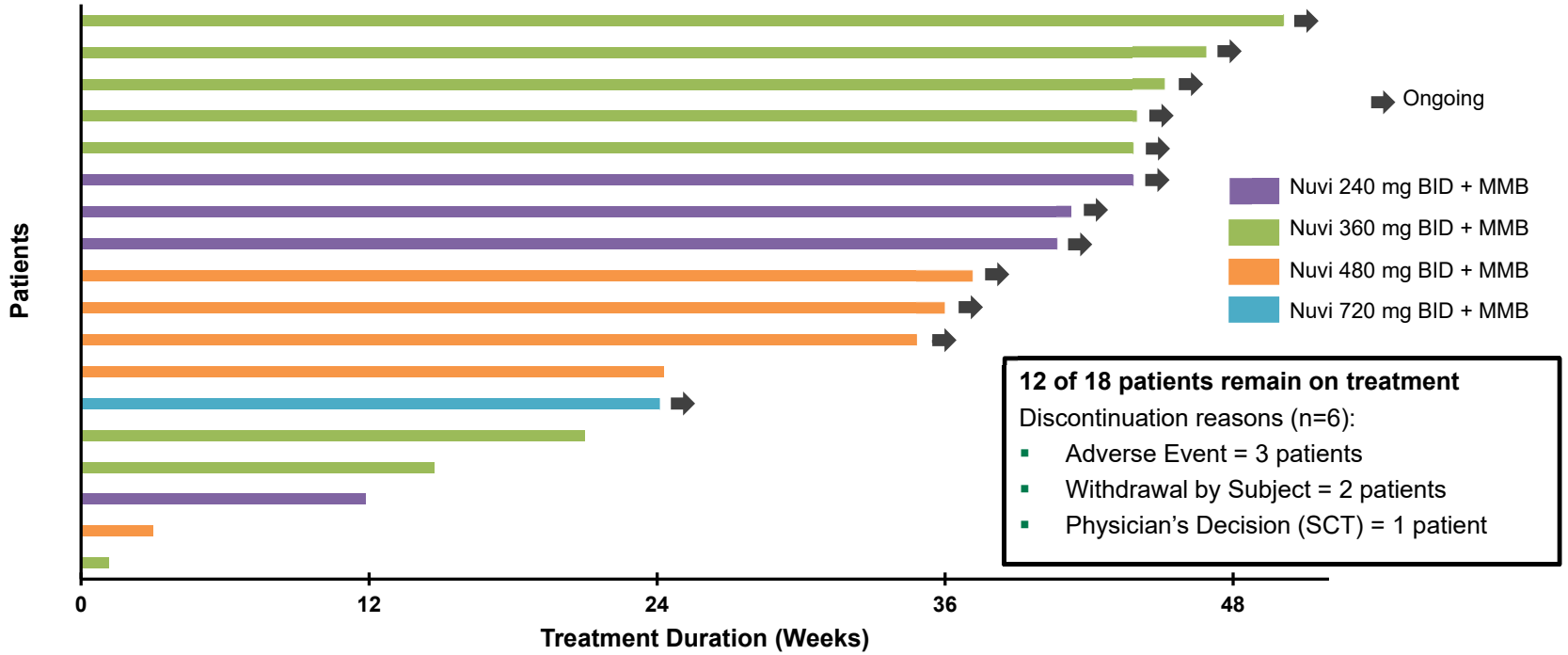
Safety: Combination with Momelotinib

Combination Therapy with Momelotinib for Relapsed/Refractory Myelofibrosis

Favorable long-term safety profile of combination therapy

- ✓ The treatment discontinuation rate through Week 24 was 28%, which is comparable to that observed in the momelotinib monotherapy Ph3 study
- ✓ No increase in discontinuations due to safety reasons was observed, indicating **a safety profile supportive of long-term combination therapy**



12 of 18 patients remain on treatment

Discontinuation reasons (n=6):

- Adverse Event = 3 patients
- Withdrawal by Subject = 2 patients
- Physician's Decision (SCT) = 1 patient

Nuvi: Nuvisertib
MMB: Momelotinib
BID: Twice daily

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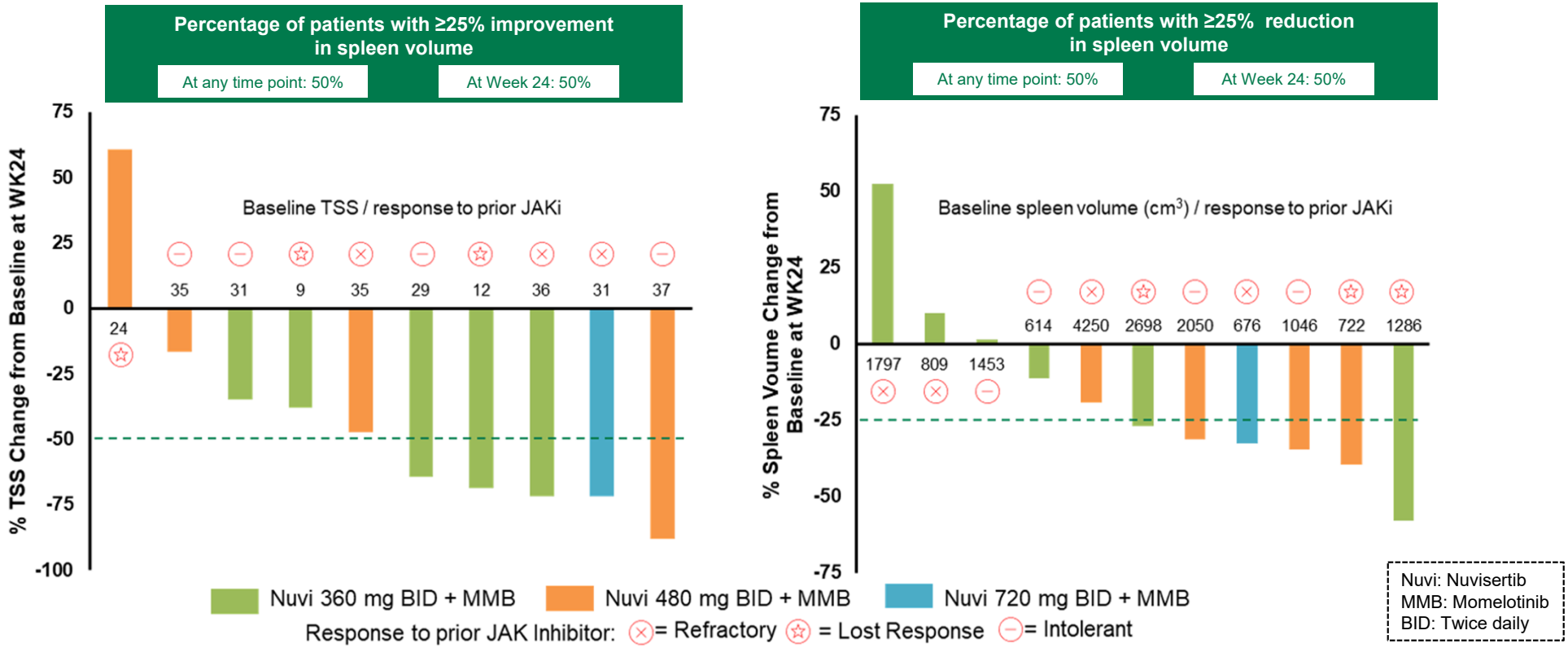
Efficacy: Combination with Momelotinib

Combination Therapy with Momelotinib for Relapsed/Refractory Myelofibrosis

Improvements in Total Symptom Score (TSS) and Spleen Volume

✓ **Improvements in both TSS and spleen volume** even in JAK-inhibitor non-responders and in high-risk patients with anemia



Nuvi: Nuvisertib
MMB: Momelotinib
BID: Twice daily

Note: Efficacy-Evaluable Population (n=12 patients who received ≥360 mg BID and either completed ≥24 weeks of treatment or discontinued due to treatment-emergent adverse events or disease progression)

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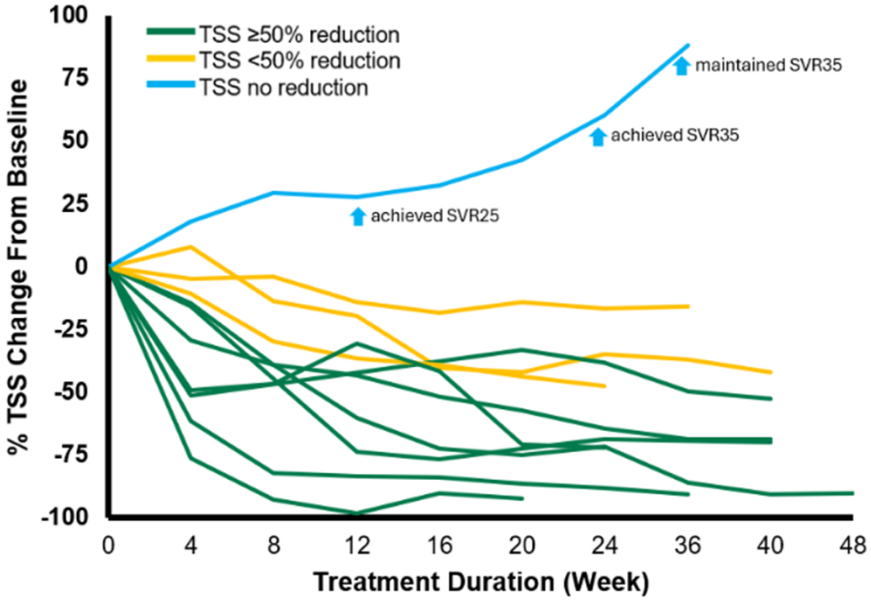
Efficacy: Combination with Momelotinib

Combination Therapy with Momelotinib for Relapsed/Refractory Myelofibrosis

Sustained Improvement in TSS from Early in Treatment

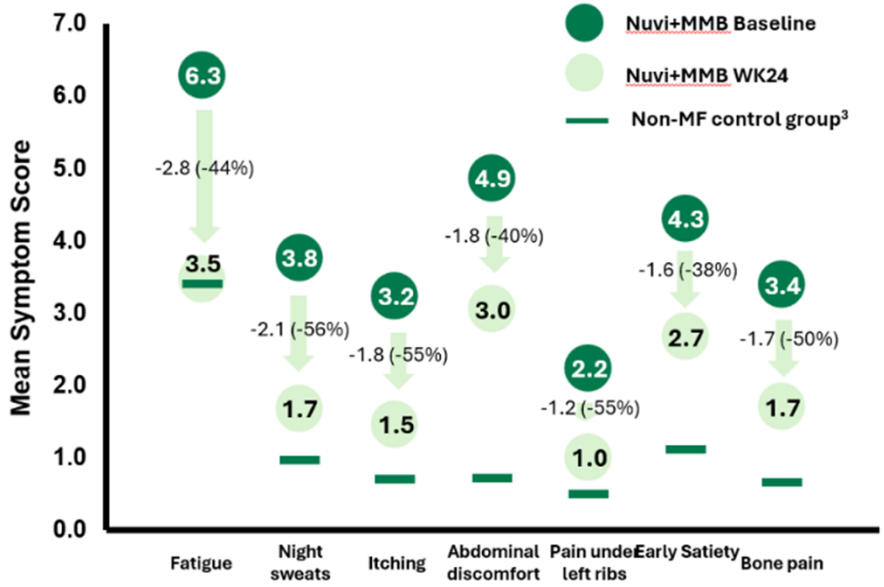
- ✓ Improvements in TSS observed as early as Week 4, with **effects appearing to sustain over the long term**
- ✓ Notable improvements in fatigue scores, with **some patients achieving levels approaching those of healthy individuals**

Early and sustained symptom reduction in TSS*1



*1 Efficacy-Evaluable Population: n=12 patients who received ≥360 mg BID and either completed ≥24 weeks of treatment or discontinued due to treatment-emergent adverse events or disease progression)

Absolute Changes in individual symptoms*2



*2 Evaluable population: n=10 patients with both baseline and WK24 symptoms data (evaluable dose= nuvisertive 360mg BID or higher + momelotinib 200mg QD)

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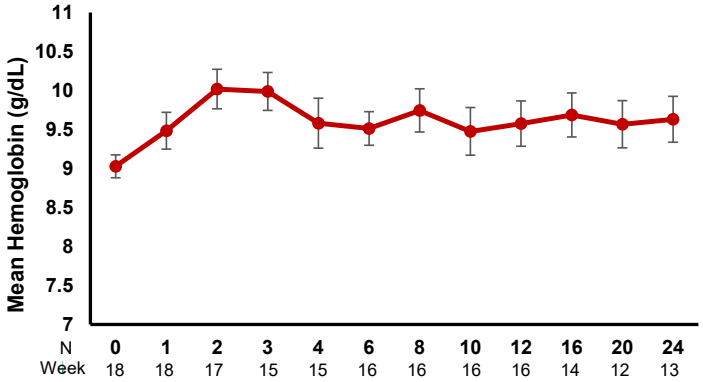
Anemia Improvement Observed in Combination Therapy with Momelotinib

Combination Therapy with Momelotinib
for Relapsed/Refractory Myelofibrosis

- ✓ **Maintain stable hemoglobin levels and platelet counts during combination therapy with nuvisertib and momelotinib**
- ✓ Improvements in anemia in 9 of 16 evaluable patients (56%)
  - ✓ Major Response: 3 patients (≥12 weeks without transfusion and ≥1.5 g/dL increase in hemoglobin)
  - ✓ Minor Response: 6 patients (Transfusion-dependent: ≥50% reduction in transfusion frequency; Transfusion-independent: ≥12 weeks without transfusion and ≥1.0 g/dL increase in hemoglobin)
- ✓ **Improvements in patients previously treated with JAK inhibitors and in those at high risk due to anemia, suggesting the potential of nuvisertib as a promising combination therapy option**

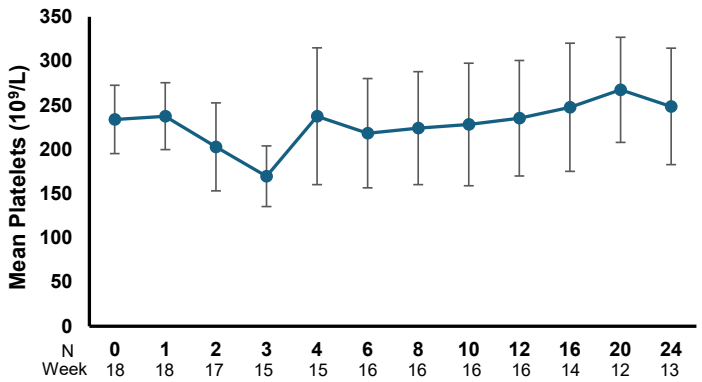
Hemoglobin Improvement

n=18; Mean ± SEM



Platelet Stability

n=18; Mean ± SEM



Nuvisertib

Potential as a First-in-Class PIM1 Kinase Inhibitor

- In MF, where JAK inhibitor is the standard of care, **treatment options with alternative mechanisms of action are extremely limited**
- Nuvisertib has
  - **demonstrated tolerability and efficacy both as monotherapy and in combination with momelotinib**
  - shown clinical activity even in patients previously treated with JAK inhibitors
- Based on these data, **initiation of a pivotal Phase 3 study is planned within FY2026 to support regulatory approval (target launch: FY2028)**
- Furthermore, with an eye toward expansion into disease areas beyond MF, **we aim to maximize its value as a compound with a novel mechanism of action**

Expansion Strategy in Oncology

- ✓ Leverage our in-house products, pipeline assets, and technology platforms **to drive both pipeline enhancement and the creation of next-generation therapies**
- ✓ In parallel, explore new targets and technological foundations to **build R&D structure capable of sustainable growth**

Leverage Our In-House Products

Tier 01

Create next-generation therapies originating from ORGOVYX®

▶

Strengthen continuity in the prostate cancer franchise

Leverage Our In-House Pipeline

Tier 02

Advance indication acquisition and expansion for enzomenib and nuvisertib

▶

Expand the hematologic malignancy pipeline

Leverage Our In-House Technology Platform (Liposomal NM*)

Tier 03

Advance development of SMP-3124

- Verify technological platforms
- Validate targets of encapsulated compounds

▶

Drive expansion from both the “technology” and “target” perspectives

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* Liposomal NM: Liposomal Nanomedicine


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Innovation today, healthier tomorrows