BeyondSpring PHARMACEUTICALS

Transforming Cancer Care with Immune-modulating Therapies

NASDAQ: BYSI

January 2026

Non-Confidential

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Investment Highlights

Plinabulin: Unique Immune Modulator	First-in-class small molecule: a unique, reversible tubulin depolymerizing agent with MoA of dendritic cell maturation and prevention chemotherapy induced neutropenia
Durable Anti-cancer Efficacy and Safety	>700 cancer patients treated with good tolerability; Demonstrated overall survival benefits and reduction of severe neutropenia in 2L/3L NSCLC EGFR Wide Type vs. docetaxel
Efficacy Potential in ICI Failed Patients	Promising efficacy data in ICI (immune checkpoint inhibitors) combos in patients with various cancers after ICI-failure
SEED: Novel TPD Platform & Pipeline	SEED: Robust pipeline with lead oncology asset, an oral RBM39 degrader entered phase 1 study; Investments and R&D Collaborations from Eli Lilly & Eisai
Intellectual Property	Strong Global Patent Protection for Plinabulin and SEED Platform and Pipeline

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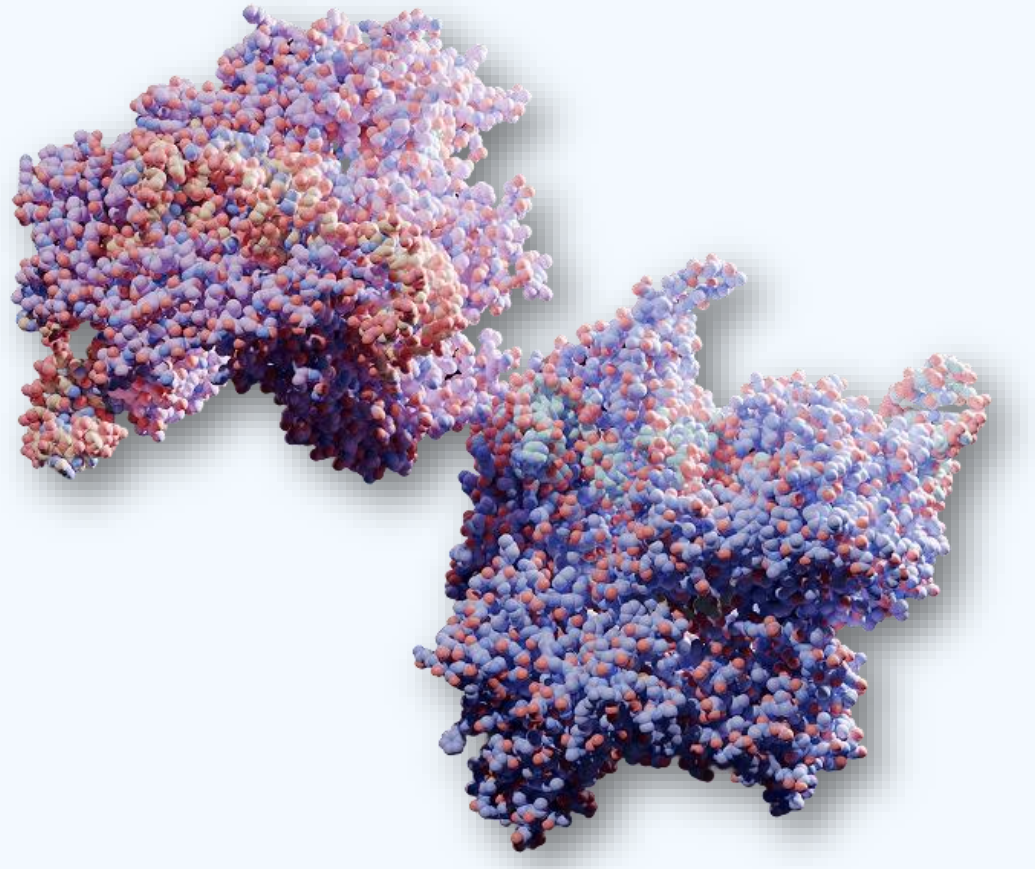


Plinabulin: Unique Dual Mechanism with Immune and Safety Benefits

- **Reversible tubulin binder** – distinct from taxanes, vincas, or colchicine; does not disrupt microtubule dynamics
- **Immune modulation** – induces dendritic cell maturation and primes T cells by activating GEF-H1
- **Neutropenia mitigation** – reduces chemotherapy-induced neutropenia by stimulating GMP progenitor cells via GEF-H1
- **Positive benefit/risk Ratio**: improve anti-cancer efficacy and tolerability in combination

Plinabulin, a Differentiated Late-Stage Oncology Asset with Broad Potential

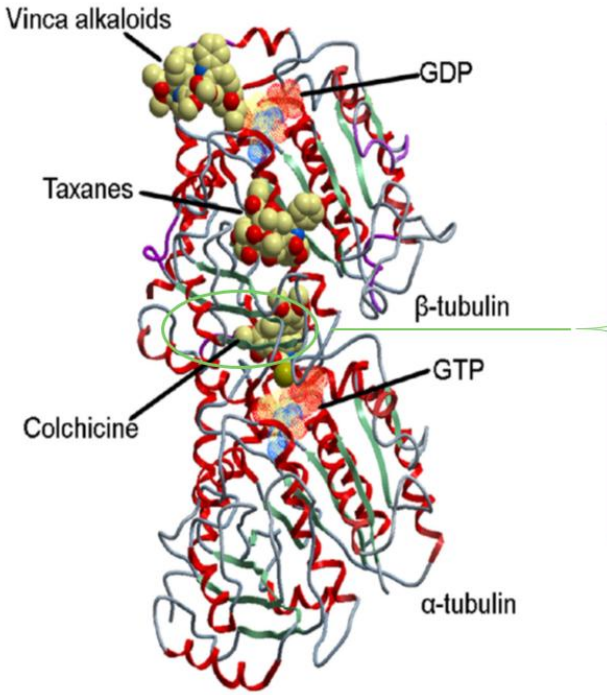
- **Robust clinical foundation**: >700 cancer patients treated with good tolerability.
- **Unique MoA**: Brain-penetrating, reversible tubulin binder driving dendritic cell maturation and T cell activation (Chem 2019, Cell Reports 2019, Med 2025); reduces chemotherapy-induced neutropenia (JAMA Oncology 2020). Synergizes with chemo, ADC, radiation, and checkpoint inhibitors.
- **Extended patent protection:** Composition of Matter (NCE) Patent Protection to 2036; Likely patent extension to 2041 based on Hatch-Waxman Act.
- **Validated benefit**: Dublin-3 Phase 3 trial (n=559) demonstrated significant OS, PFS, ORR improvement with durable long-term survival benefits and significant reduction in grade 4 neutropenia in "Plinabulin + Docetaxel" vs Docetaxel alone in 2L/3L NSCLC EGFR WT (LANCET RM 2024).
- **Regulatory momentum**: with productive discussions with US FDA and EMA, we are in late-stage clinical development in non-squamous NSCLC patients progressed on PD-1/L1 inhibitors, which is MoA-targeted, homogeneous and high-need group.

Plinabulin Monohydrate is a Brain-Penetrant, Reversible Tubulin Depolymerizing Agent with Differential Binding from Other Tubulin Targeting Agents

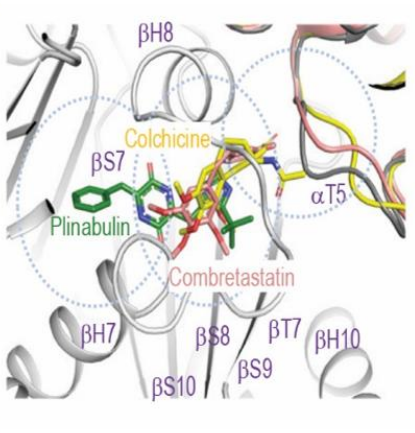
Plinabulin Monohydrate is a unique tubulin binder and does not change tubulin dynamics

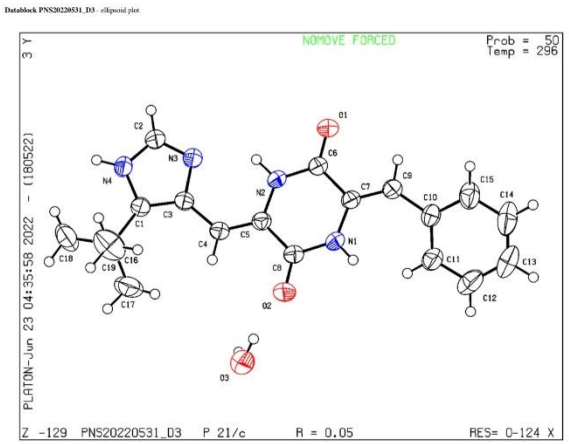
Plinabulin Monohydrate is a New Chemical Entity (NCE) with Composition-of-matter patent protection to 2036, with potential extension to 2041.

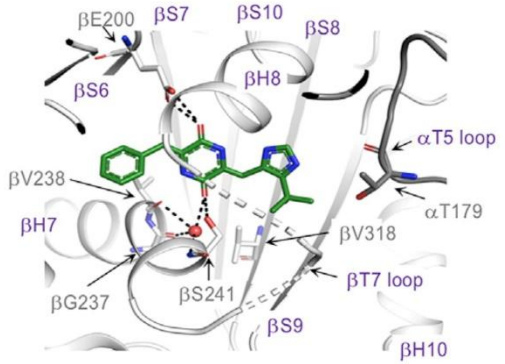
Tubulin Binding Sites



Plinabulin Binds to β-Tubulin, Near the Colchicine Site[1]





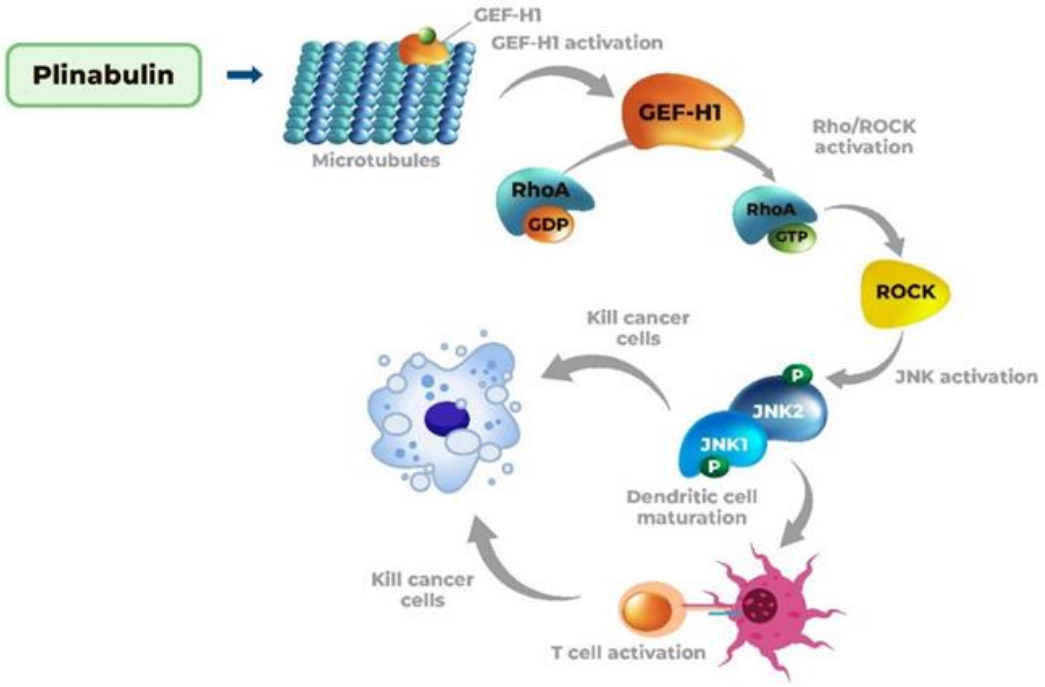


1. La Sala et al., 2019 Chem 5(11): 2969-2986

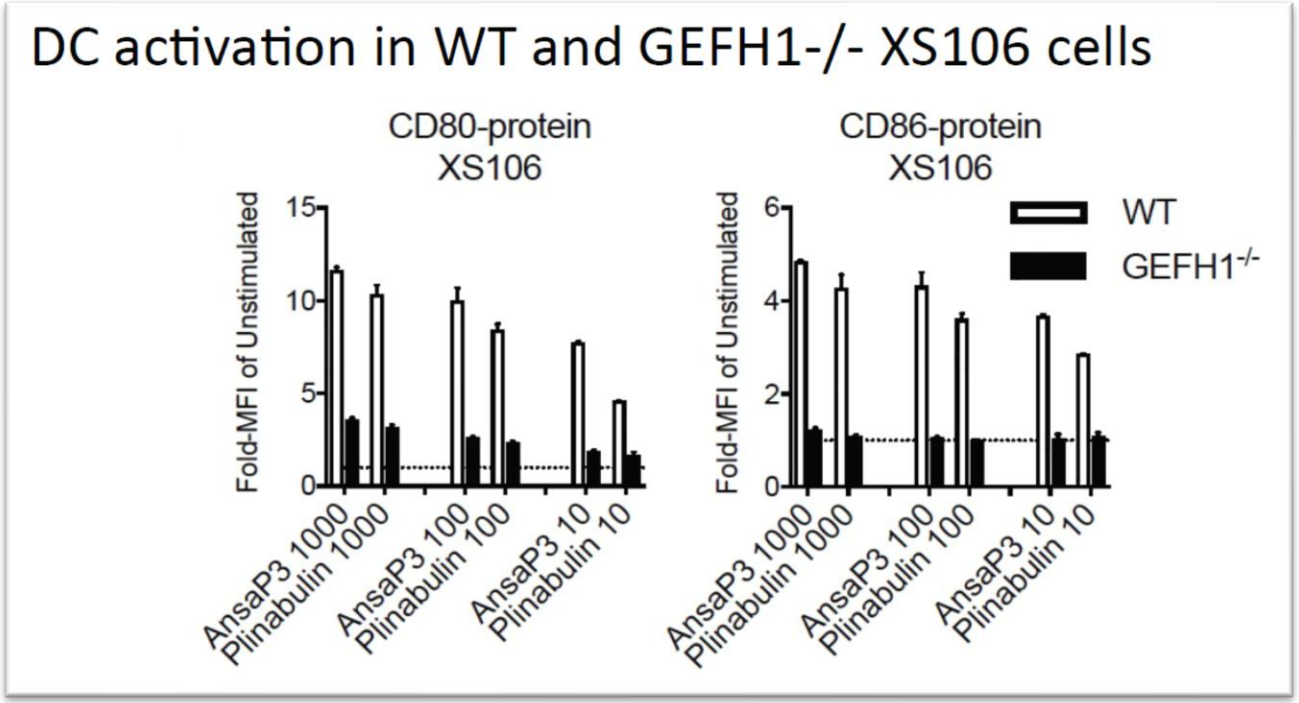
Plinabulin Modulates Innate and Adaptive Immunity via GEF-H1 Activation and Dendritic Cell (DC) Maturation

By depolymerizing microtubules, plinabulin releases and activates immune-regulatory protein GEF-H1
✓ DC Maturation is Key to Prime T cells for Adaptive Immunity with Durable Response[1].

Plinabulin Novel Target GEF-H1 activates RhoA/ROCK pathway, leading to DC Maturation[2]



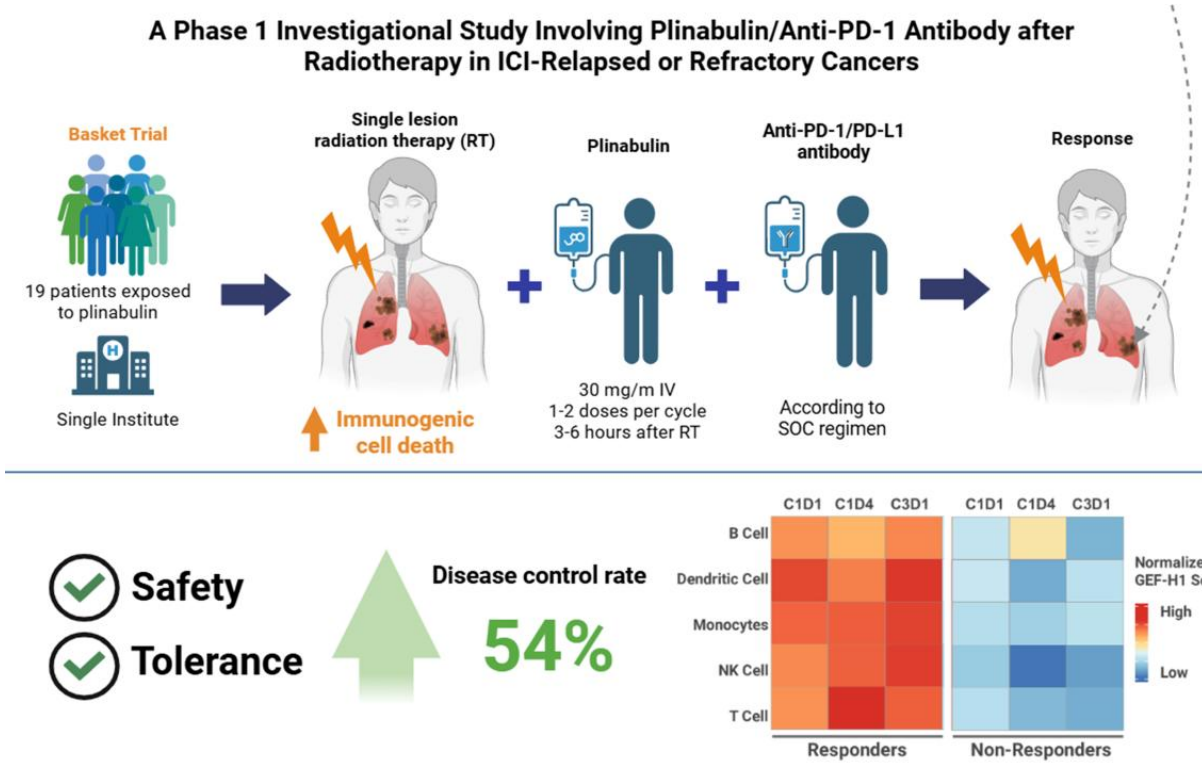
In WT DC cells, plinabulin can induce DC maturation, but not in GEF-H1 deleted DC cells[3]
CD80 and CD86 up-regulation are biomarkers for DC maturation



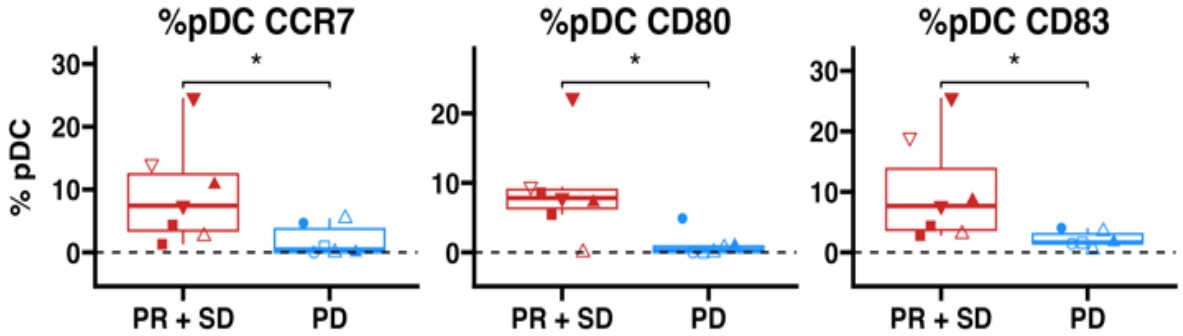
1.Steinman Annual Review of Immunology 30: 1-22 (2012); 2. Kashyap et al., Cell Reports 28(13): 3367-3380 (2019); 3. AnsaP3, a Maytansinoid cytotoxic positive control compound, is too toxic for human study

Plinabulin-Responding Patients Show Early Immune Activation Evidenced by Rapid DC Maturation in the Peripheral Blood

CCR7, CD80 and CD83 are rapidly upregulated at cycle 1 Day 4 in responding (PR + SD) patients[1]



Dendritic Cell Maturation & Migration



▽ Fibrolamellar HCC ● Melanoma □ MSI-H CRC △ RCC — PD

■ Hodgkin lymphoma ○ Merkel Cell Carcinoma ▽ NSCLC ▲ SCCHN — PR + SD

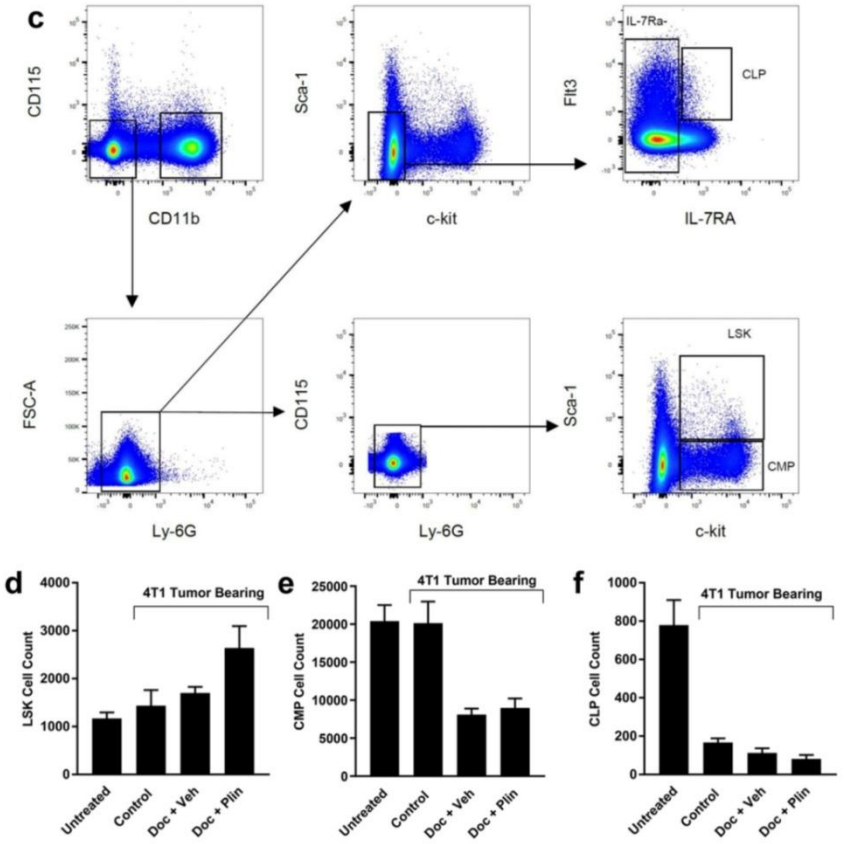
1. Lin et al., Med 6(10):100752 (2025)

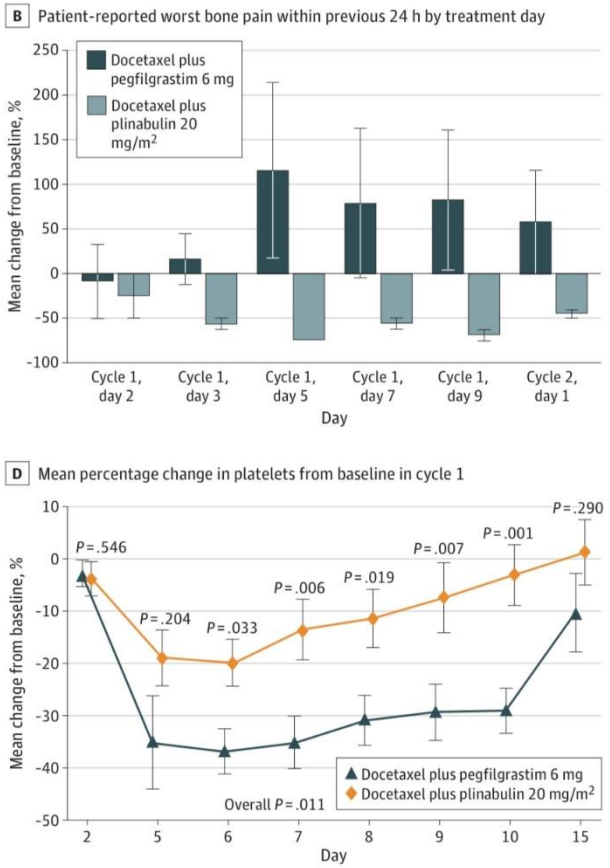
Plinabulin Unique MoA in Reducing Chemotherapy-induced Neutropenia

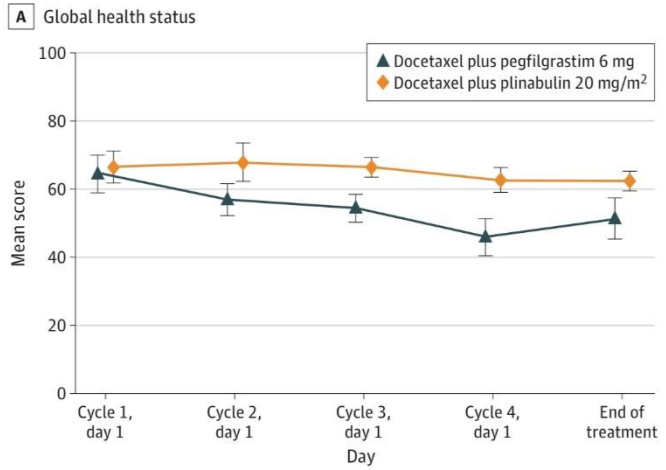
Demonstrated in 6 clinical studies for docetaxel and other chemo with early onset action

Plinabulin: non-G-CSF based treatment for CIN[1]
- Generates HSPC, including LSK cells
- GEF-H1 is shown to regulate HSPC proliferation[2]

Different from G-CSF, Plinabulin cause limited bone pain and limited platelet decrease, sustained QoL[3]



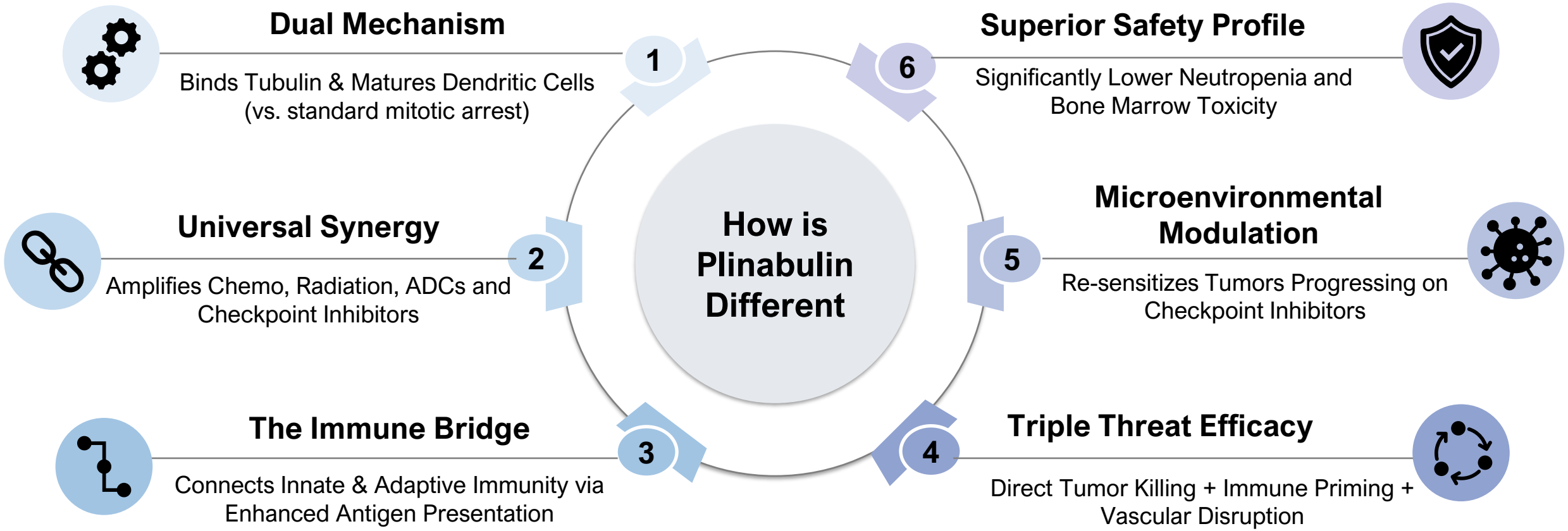




1. Tonra et al. Plinabulin ameliorates neutropenia induced by multiple chemotherapies through a mechanism distinct from G-CSF therapies. Cancer Chemother Pharmacol 85(2): 461-8 (2020).
2. Derek et al. Arhgef2 (GEF-H1) regulates mitotic spindle orientation in hematopoietic stem cells and is essential for productive hematopoiesis. Blood Advances 5(16): 3120-33 (2021)
3. Blaney et al. Plinabulin ameliorates neutropenia induced by multiple chemotherapies through a mechanism distinct from G-CSF therapies. JAMA Oncology 6(11): e204429 (2020)

Plinabulin Differentiated Profile

Mechanism Supported | Clinically Validated | Favorable Safety | Scalable Opportunity



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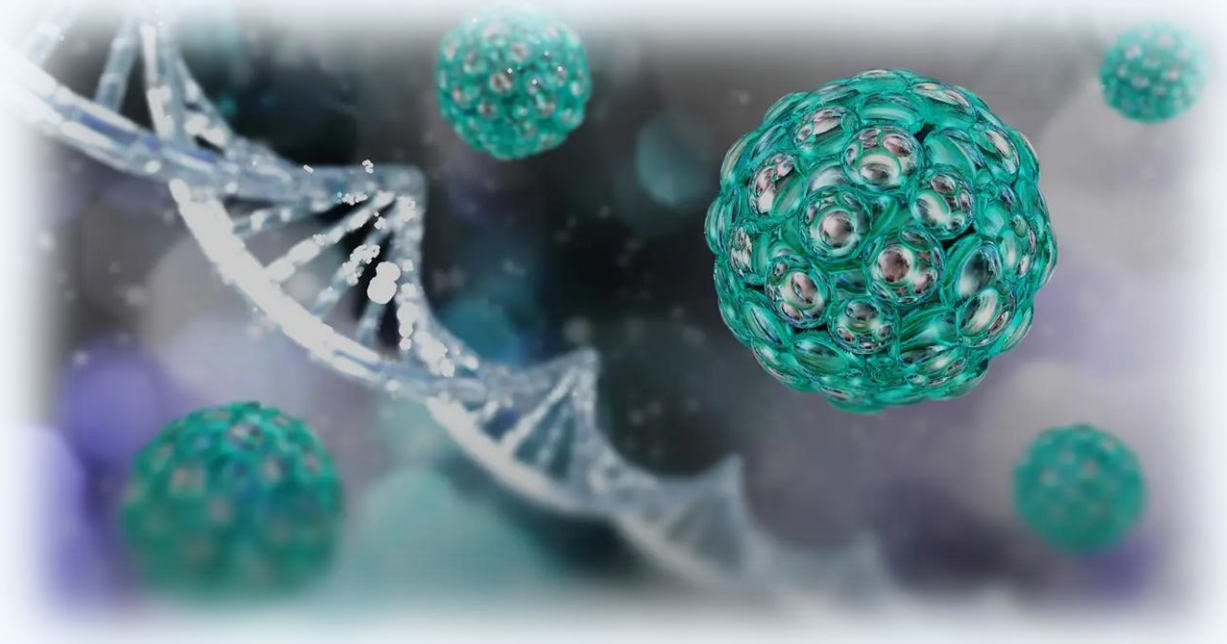
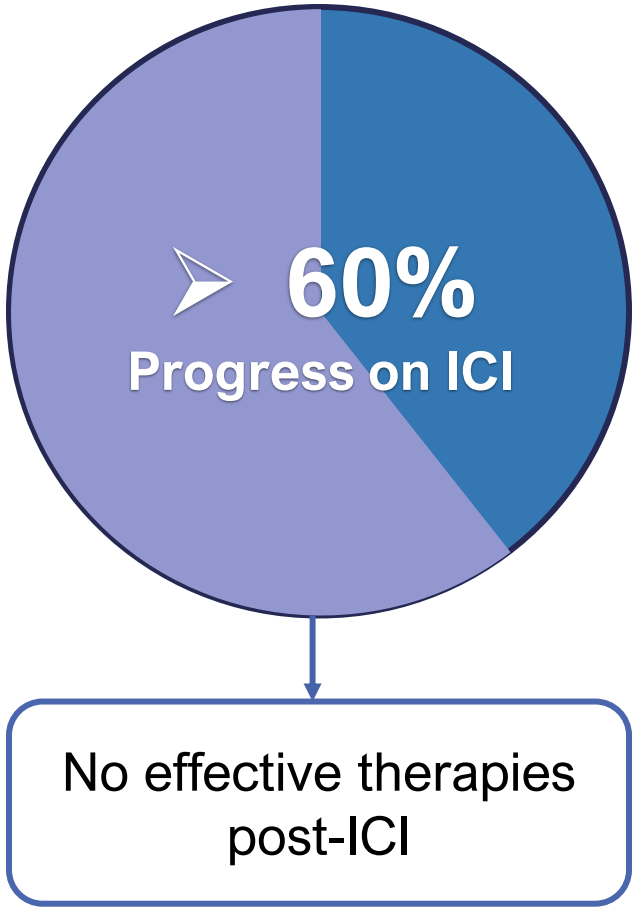


Post-ICI NSCLC: A Large, Growing and Unmet Clinical Challenge

- Limited treatment options after PD-1/L1 inhibitor progression
- Chemotherapy remains the only option, with poor outcome and high toxicity
- Urgent need for differentiated therapy with immune re-sensitization

Post-ICI Landscape: Massive Unmet Medical Need, No Approved Therapies



Immune Checkpoint Inhibitors (ICIs) Have Transformed Cancer Care

Around US$60B/year Success Story with a Critical Gap

- Approved in 20+ cancer types
- Nearly $60B in global annual sales
- Have redefined first-line treatment in NSCLC and other solid tumors

Current Options are Limited and Toxic

- >60% patients develop "acquired resistance" due to T cell exhaustion and/or antigen presenting cell (APC) pathway alterations.[1] After progression, ICIs are no longer used owing to limited efficacy
- Plinabulin's unique DC-maturation MoA could help mitigate acquired resistance to ICIs, as *dendritic cells are the most potent antigen-presenting cells and can prime T cell function - strengthening the cancer-immunity cycle.*
- Current options include chemotherapy, which is associated with severe neutropenia

Urgent Opportunity

- No newly approved therapies specifically address ICI resistance/progression
- Significant clinical and commercial opportunity

1. Memon et al. Cancer Cell 42, 209–224 (2024).

Docetaxel Remains a Global Standard of Care (SOC) in Post-ICI NSCLC, EGFR WT, Despite Limited Benefit and Substantial Toxicity

Current Standard of Care

Docetaxel Overview

- Approved >25 years ago
- Remains the NCCN-recommended standard of care for 2L/3L NSCLC with no targetable alterations
- Used after progression on anti-PD-(L)1 antibody ± chemotherapy
- Used in real world practice across U.S., EU, Japan, and China

Limitations

- Median OS: ~9 months
- 40% experience severe neutropenia

Industry-wide Phase 3 Trials Summary

Industry-wide Phase 3 Trials Summary

- **7 global trials** including ADCs and anti-PD-(L)1 combos **did not improve OS vs. docetaxel** [1]
- **#8 failed global trial** PRAGMATICA-LUNG (SWOG S2302) — ASCO 2025 [2]
  - N=838, randomized 1:1, Ramucirumab + pembrolizumab (mOS 10.1 Mo) vs. SOC (mOS 9.3 Mo), HR 0.99, p=0.46
- **#9 failed global trial** COSTAR (GSK) – 07/2025
  - N=758, TIM-3 + PD-1 + docetaxel vs. PD-1 + docetaxel vs. docetaxel. Triple combo & combo did not improve OS vs. docetaxel
- **#10 failed global trial** LATIFY (AZ) – 12/2025
  - N=594, Durvalumab + Ceralasertib (ATR Inhibitor) did not improve OS vs. docetaxel.
- **#11 Prgm ended ABBIL1TY** (Genmab) – 12/2025
  - N=702, Acasunlimab (PD-L1x4-1BB) + Pembro vs Doc

1. Malinou J et al., ASCO 2024; 2. Dragnev KH etal. ASCO 2025

"Plinabulin and Docetaxel" Demonstrated Significant OS benefit vs. Docetaxel in a Global Phase 3 Study with Stage IIIb/IV NSCLC EGFR WT Patients (Dublin-3, n=559)

Docetaxel + Plinabulin vs. Docetaxel + Placebo in Patients with EGFR Wild-Type NSCLC

Study Plan

- Global, randomized, single-blinded (to patients)
- Stratified by region (Asia/non-Asia), prior line (2L or 3L), ECOG (0-1/2), Prior PD-1/PD-L1 (yes/no)

Primary endpoint

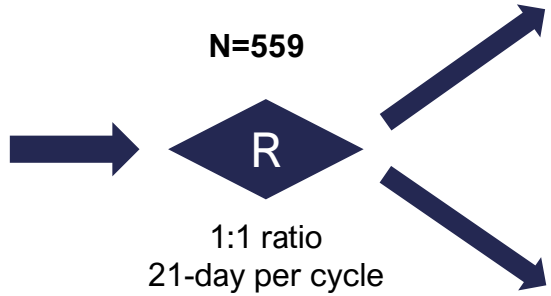
Overall survival (OS)

Secondary endpoints

- ORR, PFS
- Percent of patients without severe neutropenia (Day 8, cycle 1)
- Month 24 and 36 OS rate
- DoR
- Q-TWiST; QoL
- Proportion of patients who received docetaxel >8 cycles, >10 cycles and >12 cycles

Inclusion Criteria:

- Non-squamous or squamous NSCLC
- Stage IIIb/IV
- ECOG ≤ 2
- Progression during or after treatment with one or two treatment regimens containing a platinum
- Must have at least one measurable lung lesion
- Prior checkpoint inhibitor therapy allowed



1:1 ratio
21-day per cycle
Plinabulin IV Infusion: 1 or 2 dose per cycle

DP:
Docetaxel
(75 mg/m2, day 1)
+ **Plinabulin**
(30 mg/m2, day 1, 8)

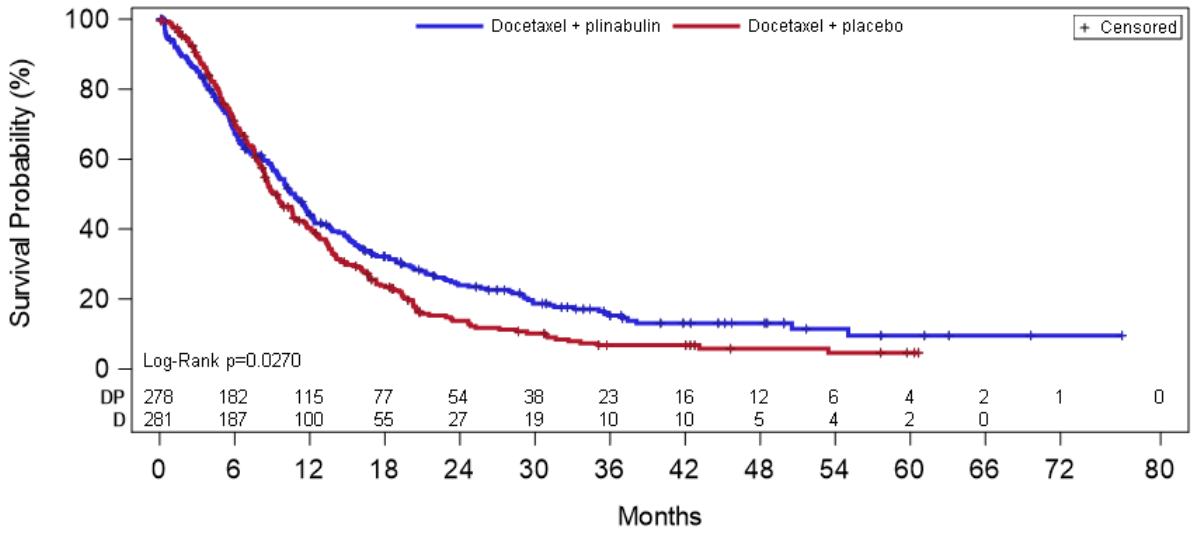
D:
Docetaxel
(75 mg/m2, day 1)
+ Placebo (day 1, 8)

Publication: Lancet Respir Med 12(10): 775-786 (2024)

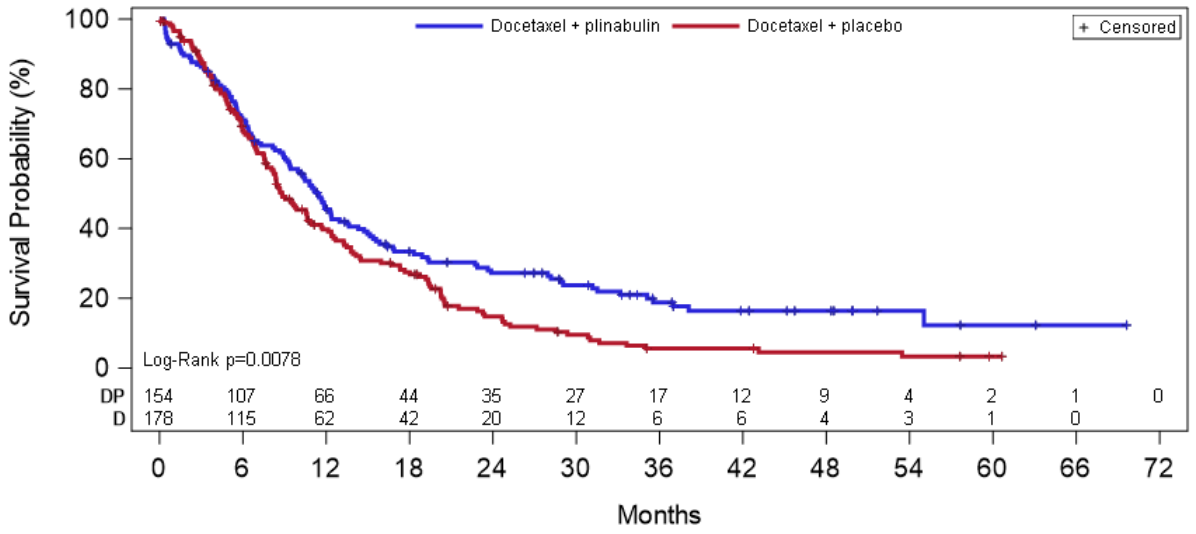
Dublin-3: Consistent OS Benefit in 24-month Follow-up after Database Lock

Non-squamous OS HR=0.72

ITT (Intention-to-Treat)



Non-squamous



ITT	N	Median OS (95% CI)	HR	Log rank P value
Docetaxel	281	9.3 (8.35, 10.65)		
Plinabulin + Docetaxel	278	10.8 (9.37, 11.97)	**0.81 (0.68, 0.98)**	**p = 0.0270**

Non-squamous	N	Median OS (95% CI)	HR	Log rank P value
Docetaxel	178	8.81 (7.73, 10.65)		
Plinabulin + Docetaxel	154	11.37 (9.37, 12.95)	**0.72 (0.57, 0.92)**	**P = 0.0078**

Publication: Lancet Respir Med 12(10): 775-786 (2024)

Plinabulin + Docetaxel (DP) is Safer Than Docetaxel (D) Adjusted by Exposure

The combination has Significant lower Grade 3 or 4 exposure-adjusted event rates vs. Docetaxel

	Grade 3 or 4 Event Rates Adjusted by Exposure		Grade 4 Only Event Rates Adjusted by Exposure	
	DP (n=274)	D (n=278)	DP (n=274)	D (n=278)
Descriptive Statistics				
Number of patients with events, n (%)	203 (74·1)	212 (76·3)	61 (22·3)	121 (43·5)
Total number of years of dose regimen	83·43	71·65	83·43	71·65
Total number of events	814	783	141	221
Observed event rate per year	9·76	10·93	1·69	3·08
Estimated event rate per year (95% CI)	**9·76 (9·11, 10·45)**	**10·93 (10·19, 11·72)**	**1·69 (1·43, 1·99)**	**3·08 (2·70, 3·52)**
Treatment Difference				
RR vs Docetaxel + Placebo (95% CI)	**0·89 (0·81, 0·98)**		**0·55 (0·44, 0·68)**	
P-value vs· Docetaxel + Placebo	**0·0235**		**<0·0001**	

Plinabulin and Docetaxel arm had more cycles of treatment vs. Docetaxel alone.

Publication: Lancet Respir Med 12(10): 775-786 (2024)

Dublin-3: Plinabulin + Docetaxel with Well-tolerated Safety Profile

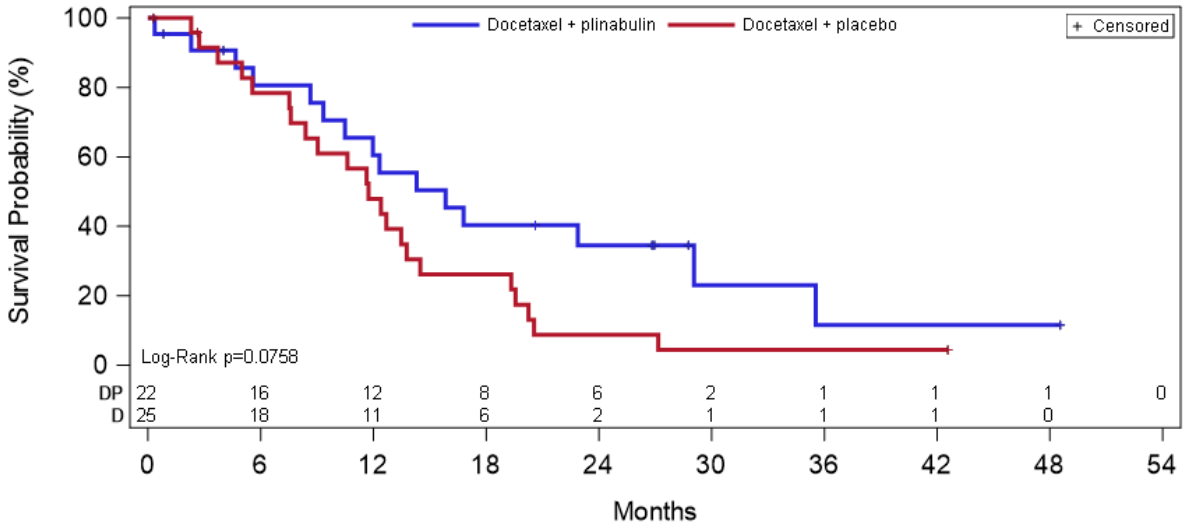
	Docetaxel + Placebo N=278 n (%)			Docetaxel + Plinabulin N=274 n (%)		
TEAE	All grades	Grade 3	Grade 4	All grades	Grade 3	Grade 4
Any	276 (99.3)	85 (30.6)	119 (42.8)	273 (99.6)	141 (51.5)	52 (19.0)
Hematological						
Anemia	121 (43.5)	13 (4.7)	0	137 (50.0)	15 (5.5)	0
WBC decreased	189 (68.0)	102 (36.7)	33 (11.9)	160 (58.4)	47 (17.2)	32 (11.7)
Neutrophil count decreased	196 (70.5)	46 (16.5)	107 (38.5)	142 (51.8)	48 (17.5)	39 (14.2)
Platelet count decreased	48 (17.3)	2 (0.7)	1 (0.4)	77 (28.1)	12 (4.4)	6 (2.2)
Other TEAEs						
Diarrhea	62 (22.3)	3 (1.1)	0	118 (43.1)	23 (8.4)	1 (0.4)
Constipation	80 (28.8)	1 (0.4)	0	95 (34.7)	1 (0.4)	0
Nausea	67 (24.1)	0	0	100 (36.5)	3 (1.1)	0
Vomiting	39 (14.0)	1 (0.4)	0	82 (29.9)	6 (2.2)	0
Abdominal pain	23 (8.3)	1 (0.4)	0	42 (15.3)	0	0
Abdominal distension	13 (4.7)	0	0	29 (10.6)	2 (0.7)	0
Lung infection	42 (15.1)	23 (8.3)	1 (0.4)	31 (11.3)	15 (5.5)	2 (0.7)
Blood pressure increased	16 (5.8)	8 (2.9)	0	93 (33.9)	50 (18.2)	0
Hepatic enzyme increased	45 (16.2)	1 (0.4)	0	47 (17.2)	2 (0.7)	0
Weight decreased	24 (8.6)	0	0	32 (11.7)	1 (0.4)	0
Cough	77 (27.7)	2 (0.7)	0	64 (23.4)	1 (0.4)	0
Dyspnea	47 (16.9)	6 (2.2)	6 (2.2)	38 (13.9)	5 (1.8)	1 (0.4)
Hemoptysis	27 (9.7)	1 (0.4)	0	31 (11.3)	4 (1.5)	1 (0.4)

Publication: Lancet Respir Med 12(10): 775-786 (2024)

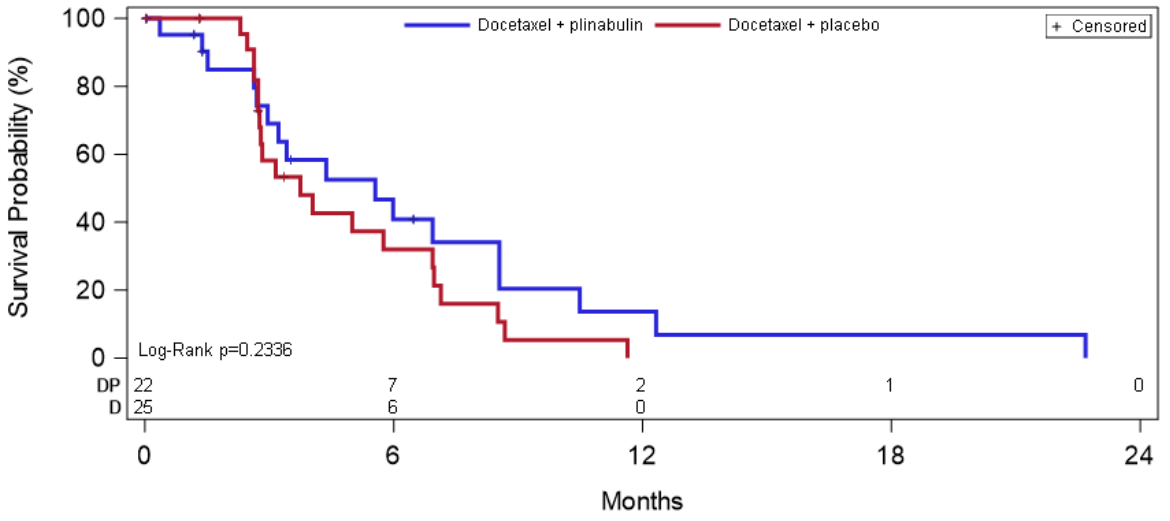
Dublin-3: Post-hoc analysis in Plinabulin Mechanism-Based Population in 2L/3L Non-squamous EGFR WT NSCLC and Progressed on PD-1/L1 Inhibitors

Clinically Meaningful Improvement of OS, PFS, and ORR in Plinabulin + Docetaxel (DP) vs. Docetaxel (D) – NACLC 2025

mOS extension of 4.1 months
(DP 15.8 vs. D 11.7 months, HR 0.55)



mPFS extension of 1.8 months
(DP 5.6 vs. D 3.8 months, HR 0.67)



ORR Improvement (DP 18.2% vs. D 8.0%)

> 60% western patients	Docetaxel + Placebo (N=25) n (%)	Docetaxel +Plinabulin 30 mg/m² (N=22) n (%)
ORR(CR+PR), n/N (%)	**2/25(8.00%)**	**4/22(18.18%)**
95% CI	(0.00,18.63)	(2.06,34.30)

Post-hoc data of Docetaxel is similar to Docetaxel data from TROPION-LUNG01[1] in similar patients

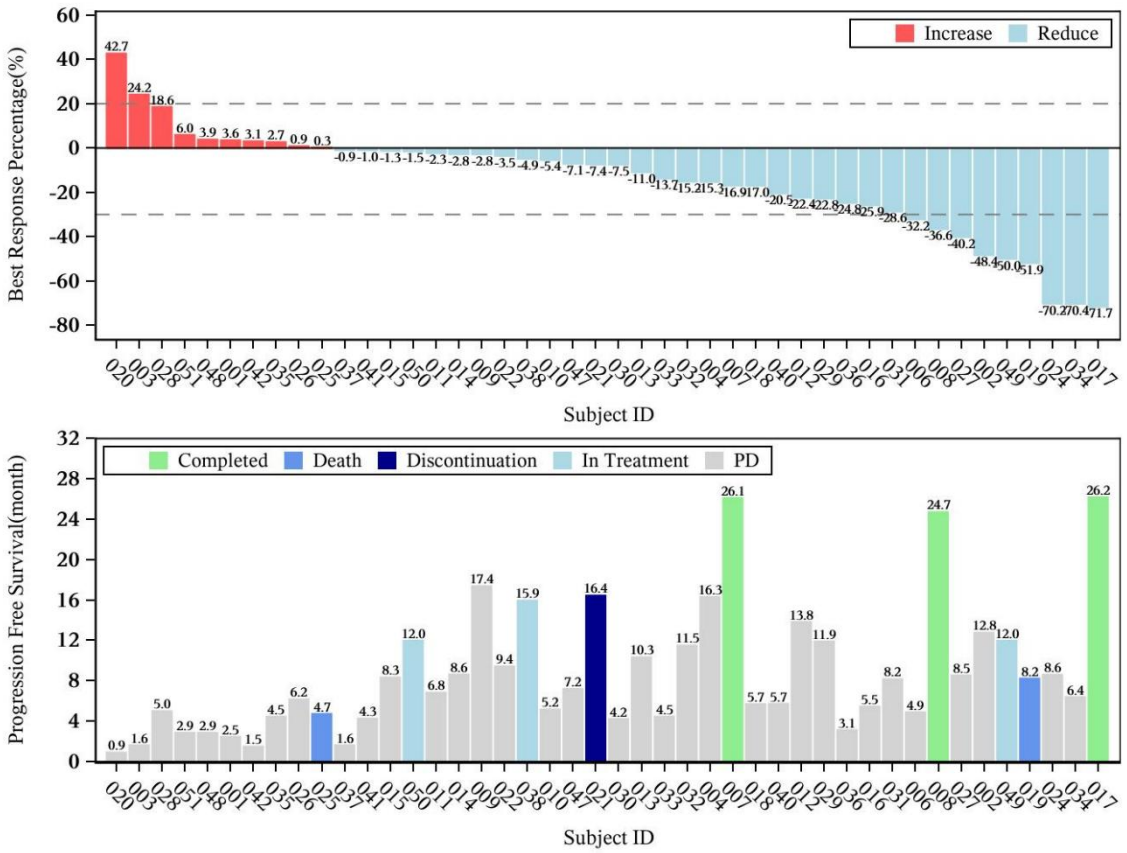
✓ mOS: 11.8 months; mPFS: 3.7 months; ORR: 12.8%

1. Ahn et al. Thoracic Oncology 43(3): 260-272 (2024)

Study 303 (Plinabulin + Docetaxel + Keytruda) in 2L/3L Metastatic NSCLC, Immediately Progressed on PD-1/L1 Inhibitors

Primary Endpoint (n=47) – SITC 2025 Presentation	
Confirmed ORR（RECIST 1.1）	**18.2%**
Secondary Endpoint	
Median PFS（RECIST 1.1）	**7.0 months**
Median OS	**16 months+, Not reached**
Median DoR（RECIST 1.1）	7.2 months
Disease Control Rate (DCR)	**85.1%**
12 months OS%	79%
24 months OS%	**66%**

Best Change (%) in Target Lesions and PFS



Median follow-up at data cutoff (30 September 2025) was 14.3 months. Median age was 67 (44-83); 80.9% male and 19.1% female. 72.3% were current or former smokers. Histology included 63.8% with non-squamous cell carcinoma, 36.2% with squamous cell carcinoma.

Planned Dublin-4 Study As a Confirmatory Phase 3 Study

Mechanism-Enriched Patients for High Chance of Clinical Success

Dublin-4: "Plinabulin + Docetaxel" vs. "Docetaxel" in 2L/3L non-squamous NSCLC, EGFR WT, progressed on PD-1/L1 with PFS ≥ 3 months

✓ Defined, homogeneous group with clear unmet need and Plinabulin MoA alignment

Scientific Rationale

✓ **Tumor Vasculature Targeting:** Plinabulin acts on tumor vasculature → Dublin-3 OS HR 0.72 in non-squamous NSCLC patients. Avastin, a vasculature targeting agent, only approved in non-squamous NSCLC patients.

✓ **Immune Re-sensitization:** Plinabulin has the potential to overcome ICI "acquired resistance" due to T-cell exhaustion / APC pathway mutation[1] by dendritic-cell (DC) maturation MoA, as DC is the most potent APC → restores T-cell priming → strengthens the Cancer-Immunity Cycle

Supporting Clinical Data in NSCLC patients progressed on PD-1/L1 Inhibitors

✓ *Dublin-3 (DP):* OS HR 0.55, mOS: 15.8 vs 11.7 months (Plinabulin + docetaxel DP vs docetaxel D), ORR 18.2% vs. 8.0%

✓ *303 Study (DP+PD-1): 24*-month OS rate 66% (mOS 16 month+), mPFS 7.0 month, ORR 18.2%

1. Memon et al. Cancer Cell 42, 209–224 (2024).

Plinabulin + Docetaxel: Addressing Unmet Needs in 2L/3L NSCLC, EGFR WT, After Prior PD-1/L1 Failure, Positioned as Potentially New Standard of Care

Severity of Unmet Need	Current SOC - Docetaxel	Potential Solution - Plinabulin + Docetaxel
●●●	Lack of durable response post-immunotherapy	Strong immune re-priming, durable OS benefit
●●●	Immune exhaustion, “cold” tumors	Converts tumors to “hot”
●●	Limited efficacy with Docetaxel	More cycles and more dose of docetaxel; Improved OS, PFS, and ORR
●●	High chemo-induced neutropenia	Reduces Grade 4 neutropenia
●●	EGFR wild-type population underserved	Target EGFR wild type patients

Plinabulin Clinical Studies in Multiple Cancers

>700 cancer patients treated with Plinabulin with good tolerability

	Indication/Target	Program	Preclinical	Phase 1	Phase 2	Phase 3	Registration	Trial Name / Collaborator
Late stage	**NSCLC (2nd/3rd line)**	Plinabulin + Docetaxel						Study 103 (DUBLIN-3) - OS, PFS, ORR benefit [1]
Late stage	**CIN Prevention**	Plinabulin alone or + Pegfilgrastim						Studies 105 & 106 [2, 3] (PROTECTIVE-1 & PROTECTIVE-2)
	NSCLC (2L/3L) progressed on PD-1/L1 Inhibitor	Plinabulin + Pembrolizumab + Docetaxel						Study 303
	ES-SCLC (1L)	Plinabulin + Pembrolizumab + Etoposide / Platinum						Study 302
	Eight types of cancers Failed PD-1/L1 Inhibitor	Plinabulin + PD-1/PD-L1 + Radiation						THE UNIVERSITY OF TEXAS MD Anderson Cancer Center [4]

- Mechanisms not restricted to lung cancer; other solid tumors may benefit, with early signals in liver, head and neck, prostate, breast, and ES-SCLC.
- Strong rationale for IO and ADC combination strategies.

1. Han et al., Lancet Resp Med 12(10): 775-786 (2024), 2. Blaney et al. JAMA Oncol 6(11): e204429 (2020);
3. Blaney et al. JAMA Network Open 5(1): e2145446 (2022); 4. Lin et al., Med 6(10):100752 (2025)

First-in-Class Agent Plinabulin: Potentially Transforming Oncology Treatment with Novel Mechanisms and Clinically Meaningful Benefits in NSCLC and Beyond

SIMPLE

Easy to use

Day 1 and 8 use in a cycle, intravenous infusion of 30-60 minutes

SAFE

Safety Benefit

Reduce AEs including chemotherapy-induced neutropenia

DURABLE

Clinical Benefit

Overall survival and durable response

SEED THERAPEUTICS

De Novo Molecular Glues for Protein Targets Beyond the Reach of Traditional Therapies

SEED: A Clinically Advancing Molecular Glue Company with Validated Pharma Partnerships and Near-term Data

Targeted protein degradation (TPD) focused on developing novel “molecular glues”

TPD Potential	• Addressing 80% of disease-causing proteins considered “undruggable” by traditional methods
Technology Platform	• Target-centric: Differentiation in using novel E3 ligases among 640 E3s for protein of interest, featured as one of leading TPD companies by two Nature review articles in 2024 • R&D collaborations with Lilly and Eisai with potential value exceeding $2.3 billion plus royalties
Robust Pipeline	• 6 Key Programs (3 internal; 3 partnered) across oncology, neurodegeneration, and immunology • ST-01156, an RBM39 degrader (oncology): preliminary Phase 1 clinical data in 2H 2026 • Oral Tau degrader (neurodegeneration): current cell activity; in vivo PK expected in 2H 2025
Finance	• ~$60M in equity, collaboration upfronts, and milestones since inception • $30M Series A-3 closed in September 2025
World-class team	• World-class founding team: Co-founders are scientific leaders in TPD E3 ligase structures and ubiquitin biology, including Nobel prize Winner Dr. Avram Hershko.

Experienced Team with Successful Track Record

Founders

- Nobel Prize winner
- Pioneers in TPD

Management Team

- 40 INDs
- 12 NDAs

Board Members

- Experienced science, business and legal experts
- Board members from Lilly and Eisai

Pioneers of E3 Biology as Scientific Foundation for SEED's TPD 2.0 Strategy

Pioneering structural biology solving both major E3 classes – HECT and CRL (RING-based) – enabling rational E3 selection

Avram Hershko, MD, PhD

Nobel Laureate Co-discovered ubiquitin–mediated protein degradation, establishing the scientific basis for modern TPD

Ning Zheng, PhD

Solved the first CRL (Cullin-RING Ligase) structure, revealing the architecture of the largest human E3 ligase family

Michele Pagano, MD

International leader in CRL substrate biology and cell-cycle regulation, defining key ubiquitin pathways in cancer

Lan Huang, PhD

Chairman and CEO; Solved the first HECT E3 ligase structure, defining the major ubiquitin transfer mechanism central to TPD 2.0

The Founder's academic discoveries and independent scientific perspectives provide the foundational scientific framework for SEED's TPD 2.0 strategy. SEED's discovery, development, and operations are executed by the company's full-time management and R&D teams.

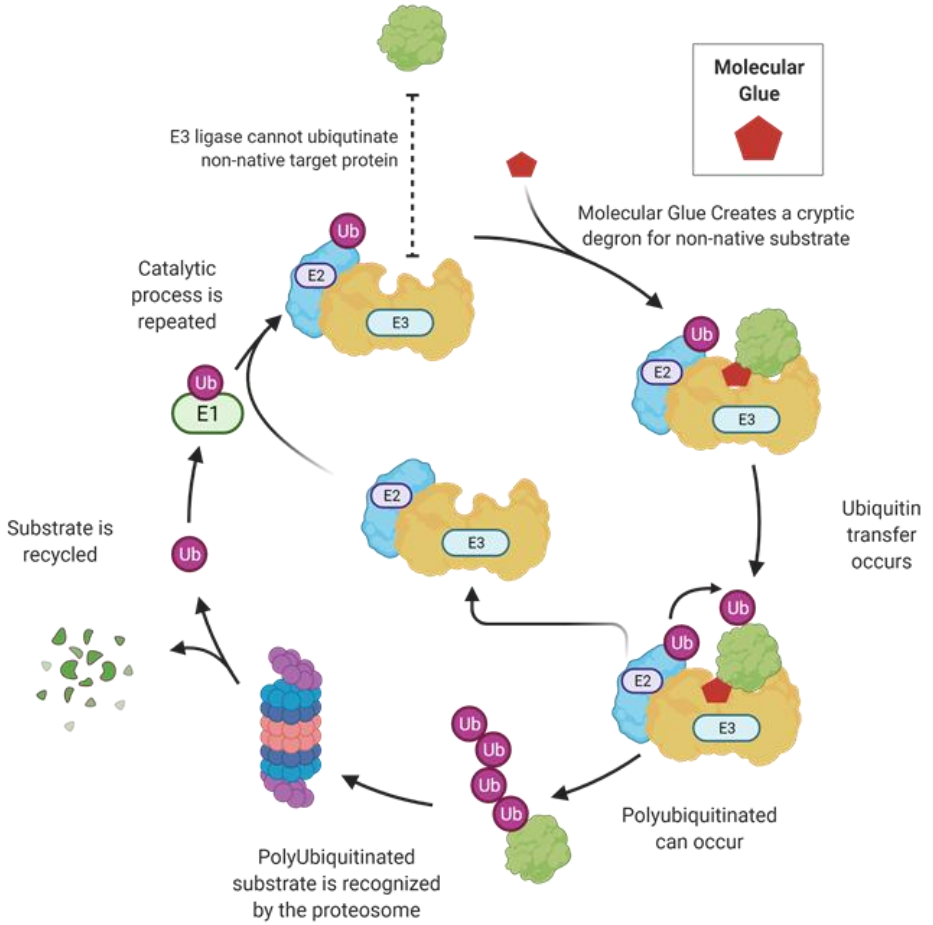
RITE3™ : A Rational, Multi-Dimensional Platform for Precise E3 Selection and Molecular Glue Engineering

Target-Centric Discovery
- Starts with disease-causing protein (POI)
- Leverages weak basal interactions of selected E3 and POI, resulting in Higher HTS hit rates

Expansive E3 Ligase Coverage and Unique Knowledge
- >600 E3 ligases (only 2 structure classes: HECT & RING, both solved by SEED co-founders)
- Enables degradation of undruggable proteins

Integrated Validation
- Structural biology + proteomics + cell assays
- Rapid validation & optimization of molecular glues

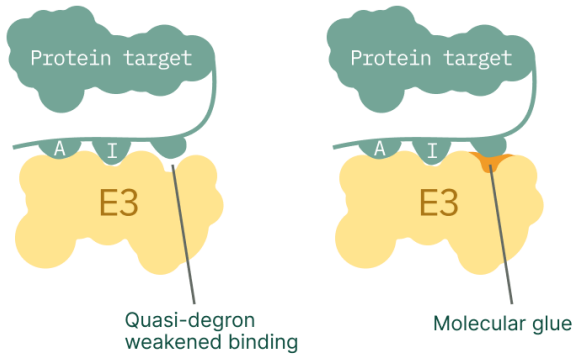
Broad & Validated Impact
- Oncology, neurodegeneration, immunology, virology
- Novel E3 ligases proven in internal & partnered pipelines



✓ **Expands the scope of degradable proteins**
✓ **Accelerates translation into therapies**

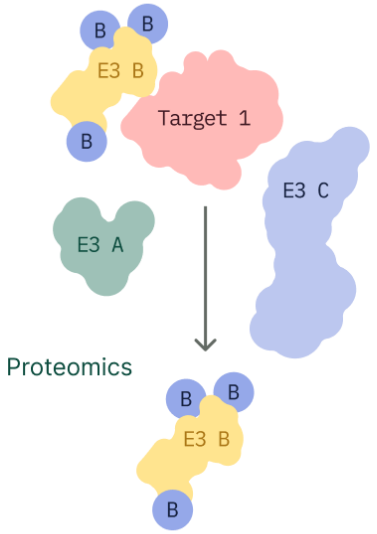
Integrated Structural, Cellular, and Biophysical Capabilities to Engineer Potent Molecular Glues

Quasi-Interface Analyses



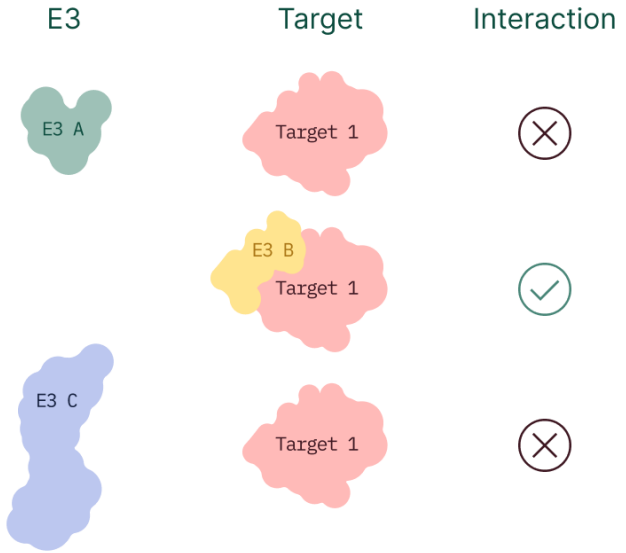
Computational and structural biology

LumID™: identify the right E3 near POI in live cell



Cell biology: real-time monitoring of target-E3 interactions

Basal Affinity Validation



Biophysics: distinguishing productive vs. non-specific interactions

Novel E3 Selection for Disease Protein of Interest

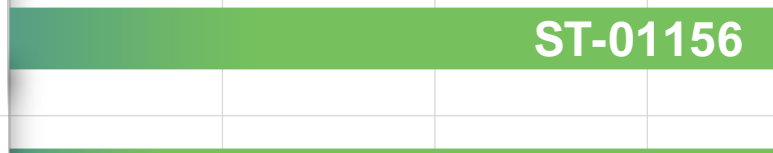
↓ High-throughput screening (E3/POI) + Optimization

Molecular Glue

*__Two Nature Review Papers:__ Garber, *Nature Biotechnology* 42(4):546-550 (2024); Mullard, *Nature Reviews Drug Discovery* 23 (11):799-802 (2024)

Advancing Six High-Value TPD Programs with Clinical Readouts Beginning in 2026

Indication	Target Protein	Milestones
Oncology	RBM39	✓ IND cleared with US and China FDA in 2H 2025 ✓ First patient dosed in Jan. 2026
	Undisclosed	
Neurodegeneration	Tau	2H 2025: In Vivo PK
	Undisclosed	
	Undisclosed	
Immunology	Undisclosed	



Internal Program Partnered Program

Lilly

- Research collaboration with Lilly on TPD with multiple targets
- $10 million upfront, and a $10 million equity investment in series A-2
- Up to $780 million in potential milestones and tiered royalties on sales

Eisai Global

- Series A-3 financing: first close of $24 million from investors led by Eisai in August 2024
- SEED-Eisai Collaboration: Milestone payments of up to $1.5 billion plus tiered royalties upon Eisai's exercise of their exclusive rights under the strategic research collaboration

RBM39: A clinically validated splicing dependency across multiple cancers

BIOLOGY

Why it matters

- Master regulator of oncogenic RNA splicing programs essential for tumor survival
- Splicing machinery is non-enzymatic and historically undruggable

MECHANISM

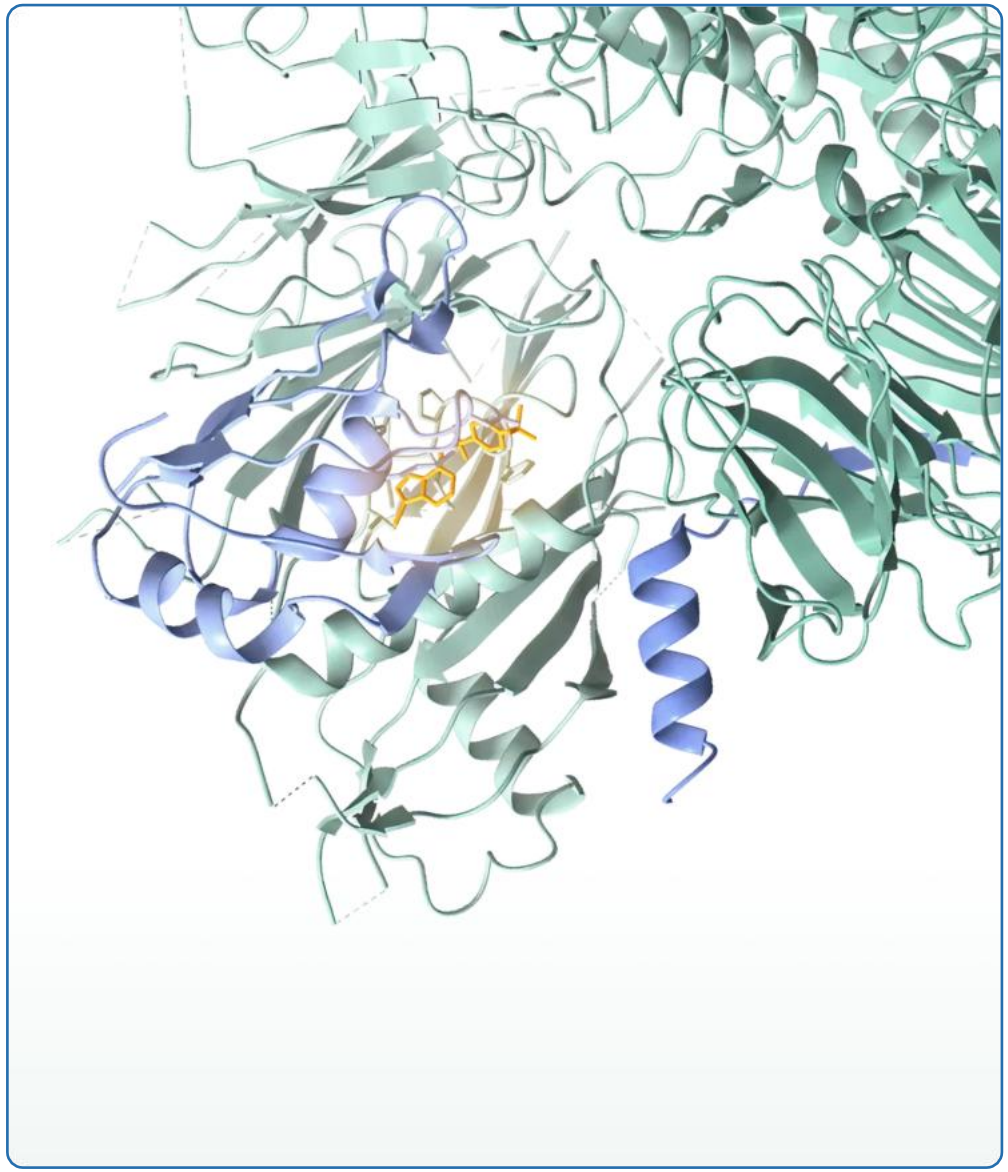
Why it works

- Molecular glue-mediated degradation via DCAF15 selectively eliminates RBM39
- Induces lethal mis-splicing in tumors while sparing normal cells via redundancy

VALIDATION

Why it’s de-risked

- Genetic + pharmacologic degradation drives strong anti-tumor effects
- Broad dependency across Ewing sarcoma and multiple solid tumors



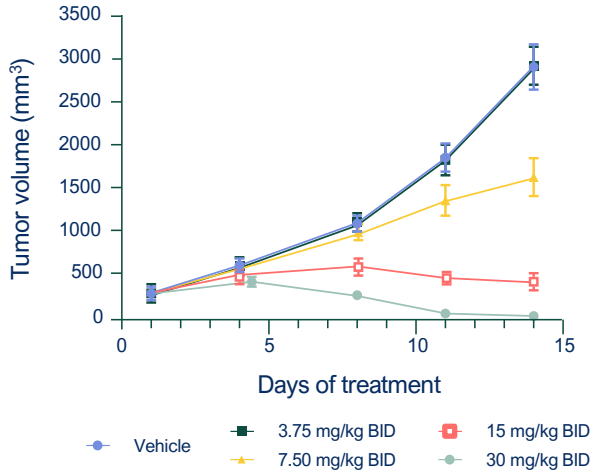
Ewing Sarcoma Is A Fusion-driven Disease with No New Drugs in the Past 30 Years

The cleanest biological proof point for RBM39 degradation.

- ST-01156 IND candidate eliminates EWS-FLI1 which causes 90% of ES cases
- Total tumor regression in vivo with precise target engagement
- FDA Orphan + Pediatric Rare Disease designation (2025)

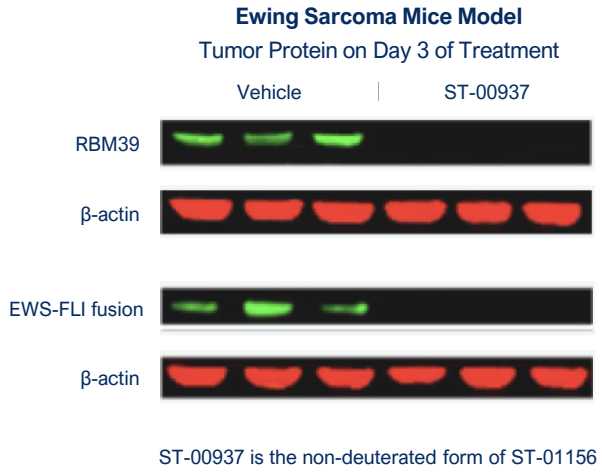
Complete tumor regression in Ewing Sarcoma

Rare pediatric and orphan cancer designation by US FDA



Precise target engagement

Total elimination of RBM39 and EWS-FLI fusion which causes 90% of Ewing Sarcoma

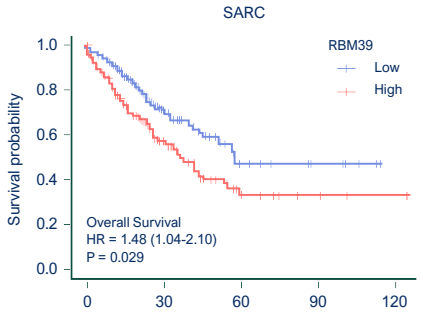


ST-00937 is the non-deuterated form of ST-01156

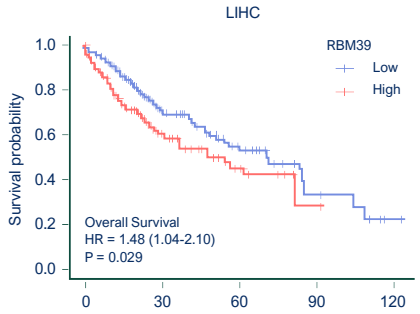
RBM39 Drives Progression in Rare and Large Tumor Indications

Neuroblastoma

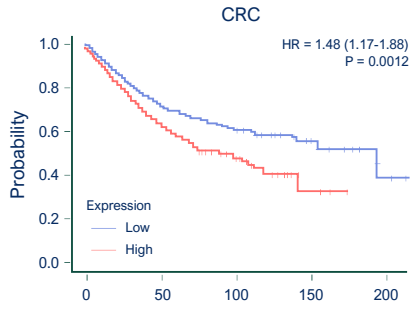
Sarcoma



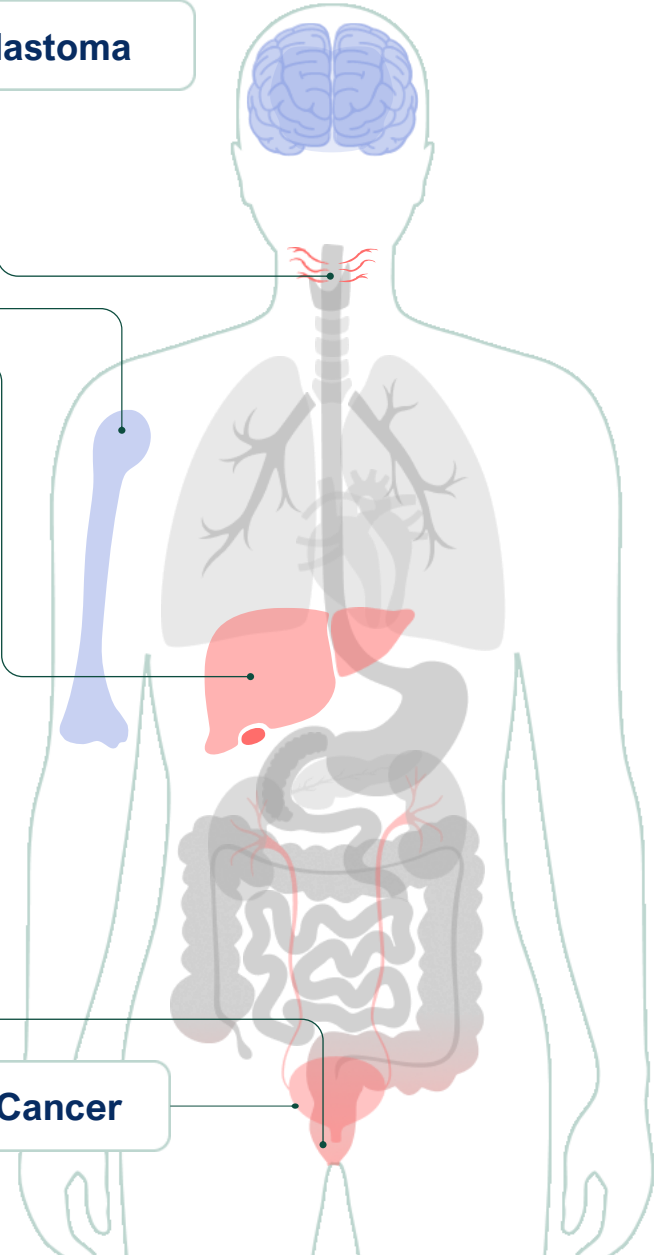
Hepatocellular Carcinoma



Colorectal Cancer



RBM39 overexpression leads to poor patient survival



Prostate Cancer

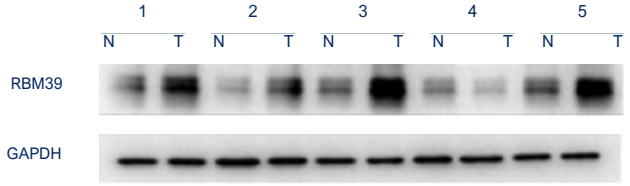
Pediatric tumors High prevalence tumors

Initial total addressable market = 1,067,280 patients

Strong Anti-tumor Activity in RBM39-dependent Liver & Colon Cancer Models

Colon Cancer

RBM39 high expression in colon cancer (T), not in normal tissue (N)

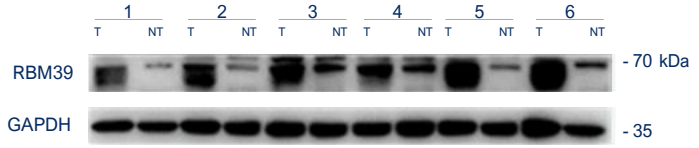


N = No Tumor
T = Tumor

Wang et al., J of Cancer, 2025

Liver Cancer

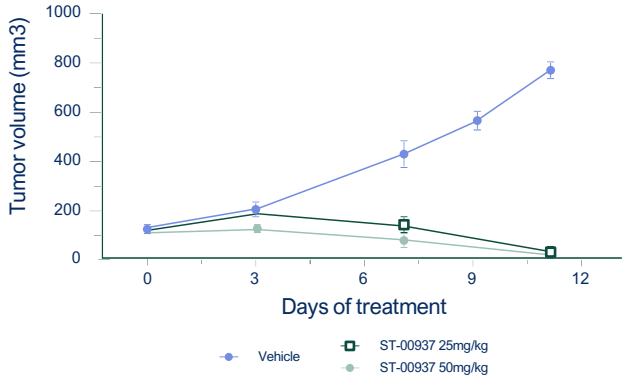
RBM39 high expression in liver cancer (T), not in normal tissue (NT)



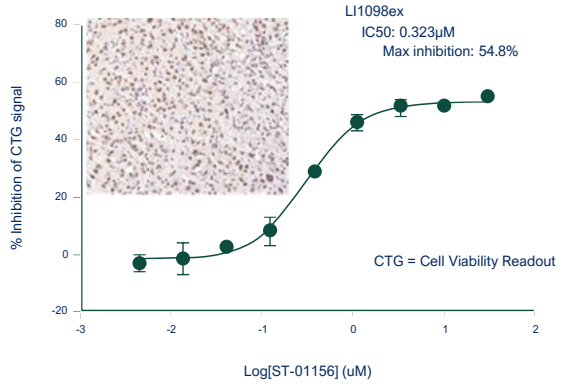
N = No Tumor
T = Tumor

Xia et al., Cell Death Discovery, 2023

Complete tumor regression in colon cancer model



ST-01156 targets RBM39-expressing patient-derived hepatocellular carcinoma cells



Clear Value Path for ST-01156: Target Engagement, Dose Expansion, and Early Efficacy Readouts – First Patient Dosed in January 2026

Focus on MoA-based Indications, Accelerating Clinical Development

- Rational clinical indications selection based on RBM39 Target Engagement
- Orphan and large cancer indications to be enriched for “MoA targeted” indications in the first clinical trials
- Preclinical in vivo data showed total tumor regression in a number of tumor models as monotherapy with favorable safety margin at >10× therapeutic exposure
- Experienced innovative oncology drug development team with investigators from US leading institutions, including Dana Farber, MSK, and MD Anderson Cancer Centers.

Breakthrough Investments Highlight the Value of Molecular Glues and Degraders

Discovery stage TPD assets
Upfront payments of $35 - $60M and $500M - $5B in potential milestones.

Pre-IND / IND stage TPD assets
$100- $300M in upfront payments and up to $2B potential milestones.

Clinical stage TPD assets (Phases I & II)
$150 - $650M in upfront payments, $350M investment and $2.1B in potential milestones.

BioSpace

Nurix Closes $120 Million Financing to Support Protein Degradation Program

BioSpace

Novartis Sticks With Monte Rosa in Second Molecular Glue Deal Worth up to $5.7B

Pharmaceutical Technology

Gilead eyes Kymera's 'adhesive' cancer drug in $750m deal

SEED Therapeutics — Why We Will Create Significant Value in 2026–2028

Clinically advancing first-in-class molecular glue degrader

- ST-01156 IND cleared in the U.S. and China
- First-in-human safety and target engagement data expected in 2026

Science built on the founders' pioneering E3 structural discoveries

- Solved structural biology of both major E3 classes: HECT and CRL (RING-based) ligases
- These insights power SEED's rational E3 selection and neo-substrate design via RITE3™

Mechanism-driven clinical strategy enabling rapid proof-of-concept

- Prioritized indications: Ewing sarcoma, Liver cancer, KRAS-mutant solid tumors, including colon cancer
- Strong PK/PD, regression models, and biomarker strategy support early efficacy readouts

Pharma-validated platform with significant non-dilutive value

- Lilly and Eisai collaborations with >$2.3B milestone potential
- Novel program portfolio across oncology and neurodegeneration

BeyondSpring PHARMACEUTICALS

Thank You

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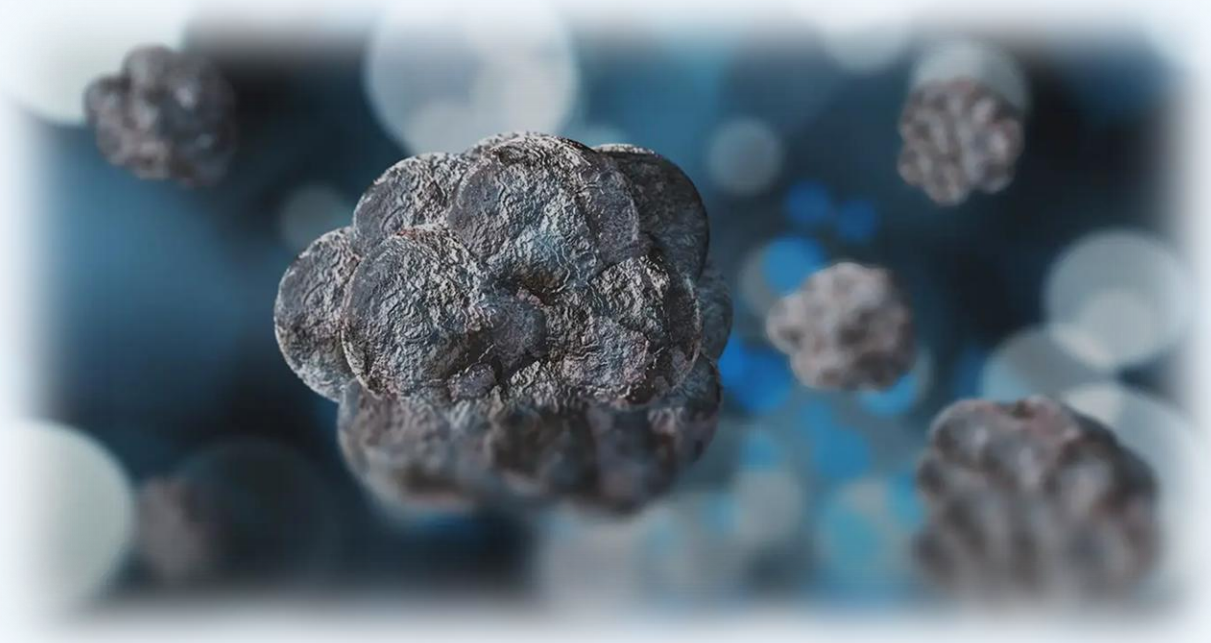
BeyondSpring Inc.



Appendix

Seasoned Management Team with Deep Global Biopharma Expertise

Lan Huang, PhD
Co-Founder, Chairman & Chief Executive Officer

Biotech entrepreneur with 20+ years across U.S. and China; founder of multiple companies, MSKCC-trained scientist with *Science*/*Nature* publications, inventor with oncology and dermatology patents.

June Lu, PhD
Chief Scientific Officer

Drug development leader with 25+ years' experience across oncology and autoimmunity, Endocyte/Novartis-trained, leading cross-functional R&D, target discovery, and clinical translation, with diverse publications/presentations and patents.

John Mao, PhD
SVP, Development

Senior biotech development executive with 30+ years advancing global programs, leading INDs, NDAs, and regulatory approvals across multiple therapeutic areas.

Helen Li, MD
VP, Clinical Science

Global clinical development leader with 20+ years, advancing NDAs and pharmacovigilance; instrumental in Plinabulin NSCLC development; former Sanofi and Roche contributor to multiple FDA/EMA approvals.

Qi Liu, MD, PhD
Medical Consultant

Global oncology leader with 20+ years' experience, expert in Phase I to III trials and novel modalities; former CMO at Elucida and Zai Lab, AstraZeneca executive, and trusted biotech advisor.

Hao Zhang
Senior Director, CMC

CMC and supply chain leader with nearly a decade at BeyondSpring, directing global manufacturing and regulatory operations for Plinabulin across IND, Phase III, and NDA stages.

Joy Jia
VP, Finance

Finance professional with a decade at BeyondSpring, overseeing SEC reporting, SOX compliance, audits, and capital markets activities, with strong experience in global accounting and cross-border financial operations.

Proven Board with Global Biopharma and Financial Expertise

Brendon Delaney

Director

Seasoned commercial leader with 25+ years' experience, leading global launches at Constellation, Immunomedics, Celgene, Novartis, and Genentech.

Jen Majeti, PhD, MBA

Director

Biotech investor and executive with 20+ years' experience, leading global collaborations at Erasca, Roche, BioDuro, and Amgen, with strong US - China lead.

Lan Huang, PhD

Co-Founder, Chairman and Chief Executive Officer

Biotech entrepreneur with 20+ years across U.S. and China; founder of multiple companies, MSKCC-trained scientist with *Science*/*Nature* publications, inventor with oncology and dermatology patents.

Mathew Kirkby, MA

Director

Board member with over 20 years of global banking leadership, with senior roles at HSBC, CIMB, RBS, and ABN AMRO across Asia and Europe.

Patrick Fabbio

Director

Board member and seasoned CFO with 25+ years of financial leadership across public and private life science companies, including Progenics, Catalent, WindMIL, and Sanofi.

Sihai Xu, MBA

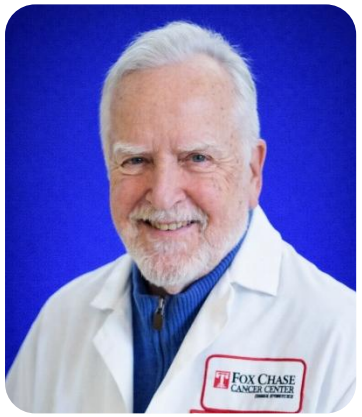
Director

Finance executive with nearly 30 years' experience in China, CFO and board member of BOJI Health, advising numerous pre-IPO, IPO, and restructuring transactions across China and Hong Kong.

Scientific Advisors with Global Oncology Leadership

Adam Grippin, MD, PhD
MD Anderson Cancer Center

Physician-scientist advancing RNA-based cancer immunotherapies, Nature-published innovator, contributor to first-in-human mRNA vaccines, and national co-chair for planned Phase III clinical trials.



John Ruckdeschel, MD
Fox Chase Cancer Center

Senior oncology leader and lung cancer expert; Professor at Fox Chase, former CEO of multiple NCI centers, author of 200+ publications, with extensive national leadership across ECOG, NCI, and IASLC.

Stephen Lin, MD
MD Anderson Cancer Center

Professor and physician-scientist specializing in thoracic malignancies, leading clinical trials and translational research to enhance radiotherapy and immunotherapy combinations through biomarker-driven strategies in lung and esophageal cancers.

Trevor Feinstein, MD
Piedmont Cancer Institute

Award-winning hematologist-oncologist with 50+ publications and global lung cancer expertise, leading research at Piedmont Cancer and chairing the Lung Disease Group for the OneOncology network.