



BeyondSpring

PHARMACEUTICALS

**Transforming Cancer Care with
Immune-modulating Therapies**

NASDAQ: BYSI

March 2026

Non-Confidential



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By attending this presentation, you acknowledge that you will be solely responsible for your own assessment of the market and our market position and that you will conduct your own analysis and be solely responsible for forming your own view of the potential future performance of our business.

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 inhibitor) combos in patients with various cancer types after immune checkpoint inhibitors (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 Protections for Plinabulin and SEED Platform & Pipeline



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 recommended alone owing to limited efficacy
- Current limited options include chemotherapy, which is associated with severe neutropenia

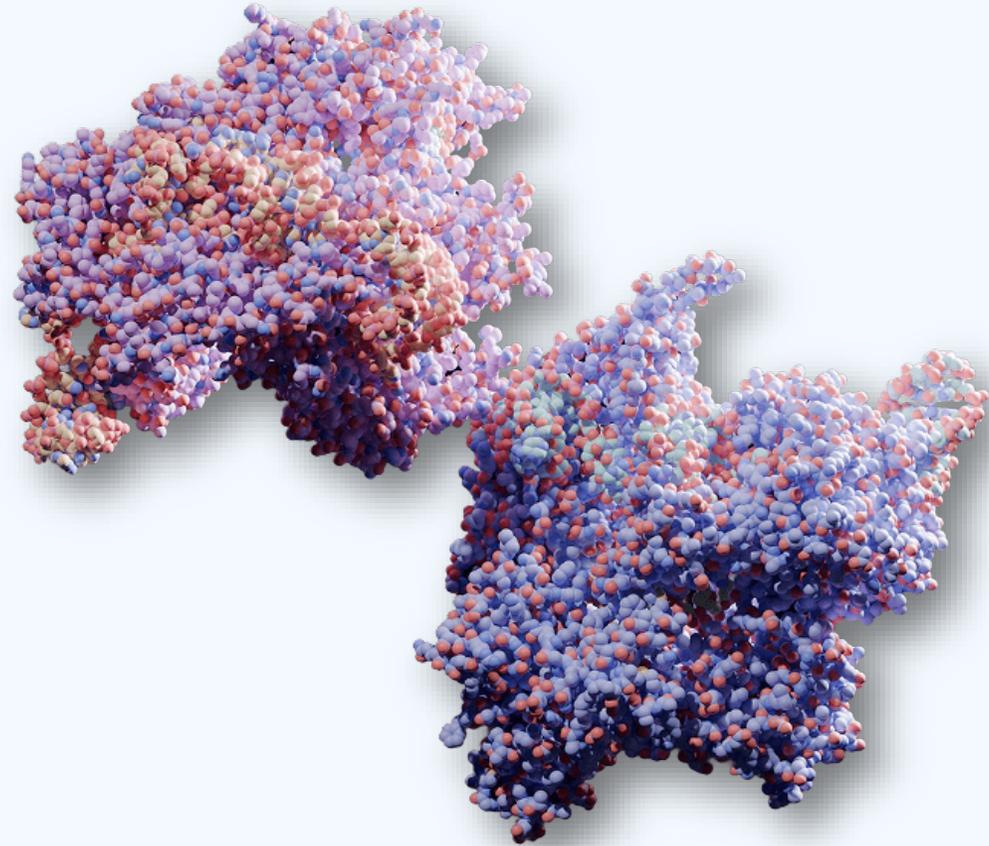
Urgent Opportunity

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

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



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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
- **Positive benefit/risk Ratio:** improve anti-cancer efficacy and tolerability in combination therapy

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



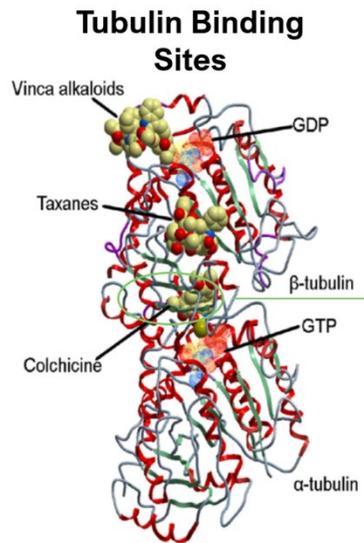
- **Robust clinical foundation:** >700 cancer patients treated with good tolerability.
- **Unique MoA:** Brain-penetrating, reversible tubulin binder driving dendritic cell maturation and T cell activation (Chem 2019, Cell Reports 2019, Med 2025); reduces chemotherapy-induced neutropenia (JAMA Oncology 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 in 2L/3L NSCLC EGFR WT (LANCET RM 2024).
- **Regulatory momentum:** Per productive discussions with US FDA and EMA, we are in late-stage clinical development in non-squamous NSCLC after progression on PD-1/L1 inhibitors (Dublin-4 study), which is MoA-targeted, homogeneous and high-need patient group.

Plinabulin Monohydrate is a Brain-Penetrant, Unique Tubulin Depolymerizing Agent which induces “Dendritic Cell or DC Maturation via GEF-H1 release”

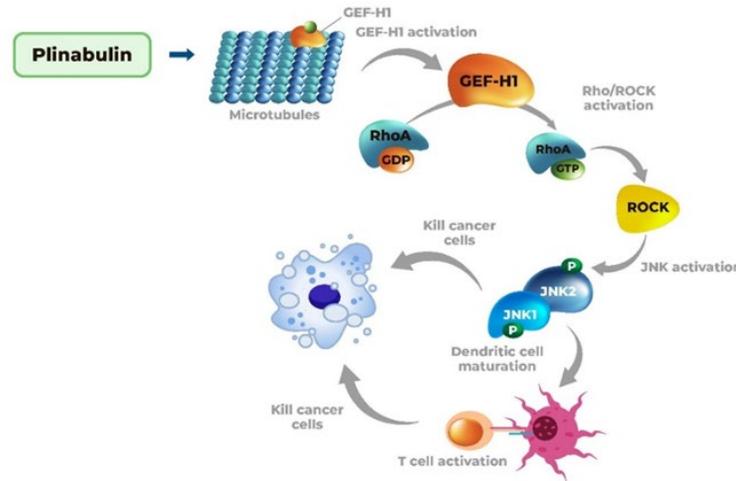
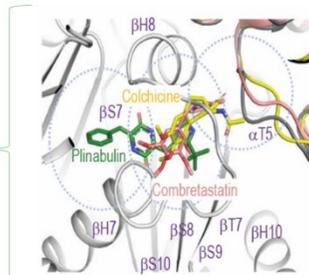
Plinabulin Monohydrate is a unique tubulin binder¹

Plinabulin release GEF-H1 from microtubule, activates RhoA/ROCK pathway, leading to DC Maturation^{2,3}

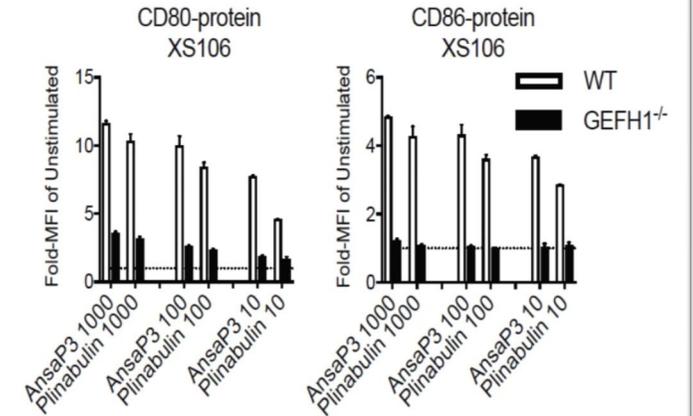
In WT DC cells, plinabulin can induce DC maturation, but not in GEF-H1 deleted DC cells²



Plinabulin Binds to β -Tubulin, Near the Colchicine Site¹



DC activation in WT and GEFH1^{-/-} XS106 cells



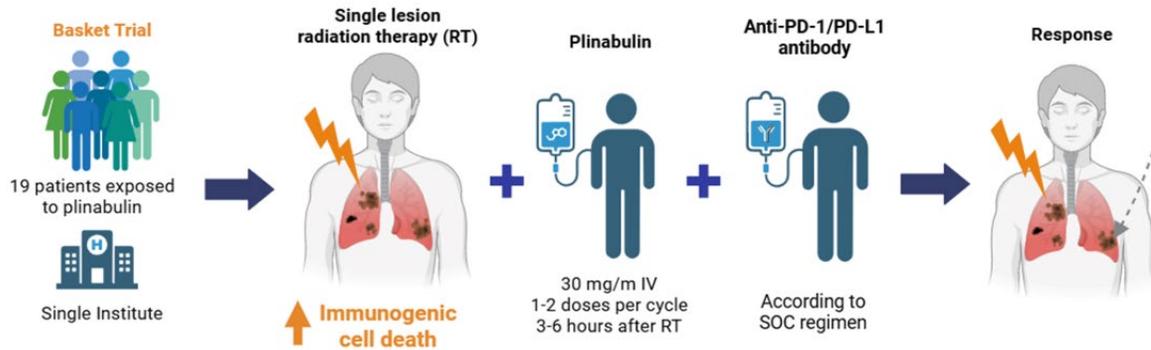
1. La Sala et al., Chem 5(11): 2969-2986 (2019); 2. Kashyap et al., Cell Reports 28(13): 3367-3380 (2019); 3. Choi et al. Cell 189 (2): 461-477 (2026)

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

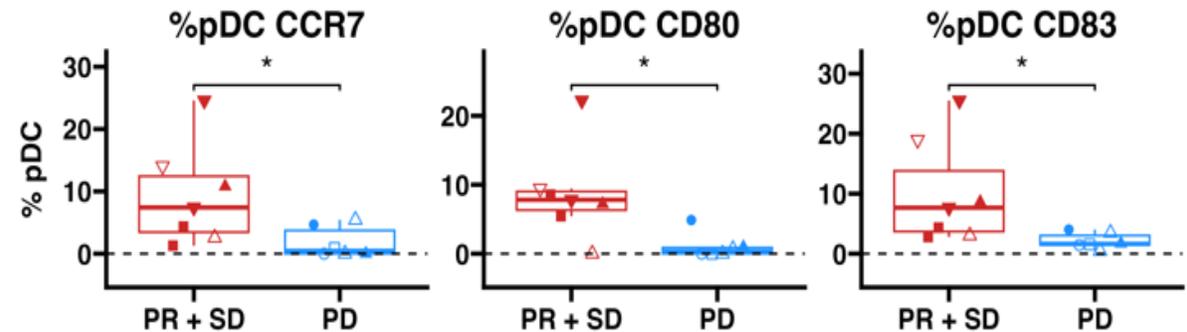


CCR7, CD80 and CD83 are rapidly upregulated at cycle 1 Day 4 in responding (PR + SD) patients¹

A Phase 1 Investigational Study Involving Plinabulin/Anti-PD-1 Antibody after Radiotherapy in ICI-Relapsed or Refractory Cancers

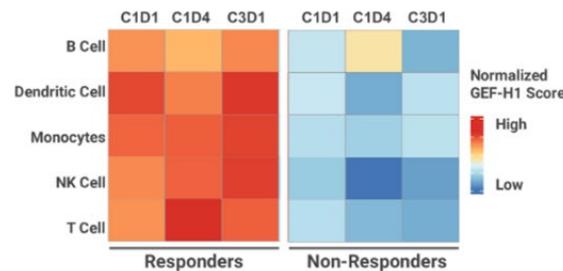


Dendritic Cell Maturation & Migration



✓ Safety
✓ Tolerance

Disease control rate: **54%**



- ▽ Fibrolamellar HCC
- Melanoma
- MSI-H CRC
- △ RCC
- Hodgkin lymphoma
- Merkel Cell Carcinoma
- ▽ NSCLC
- ▲ SCCHN

- PD
- PR + SD

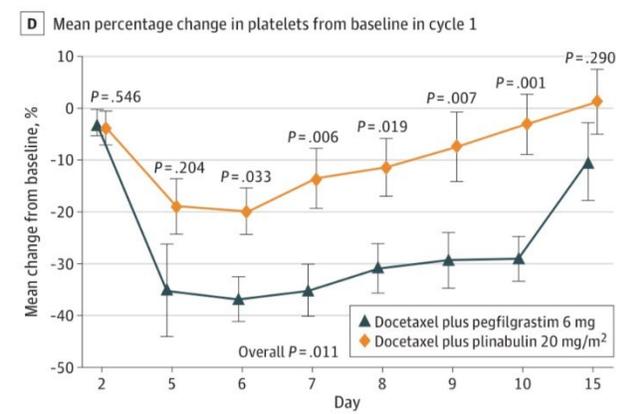
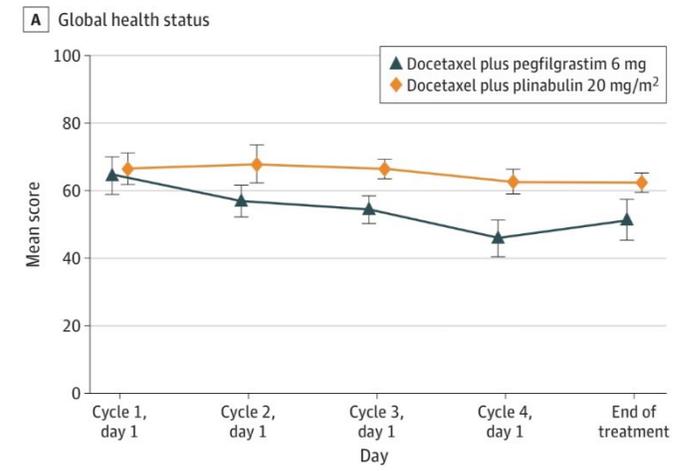
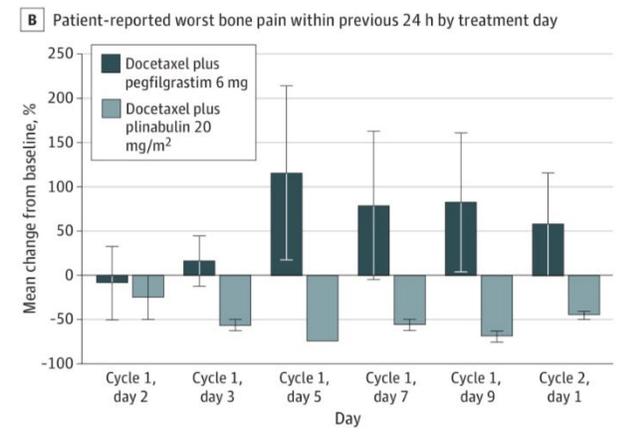
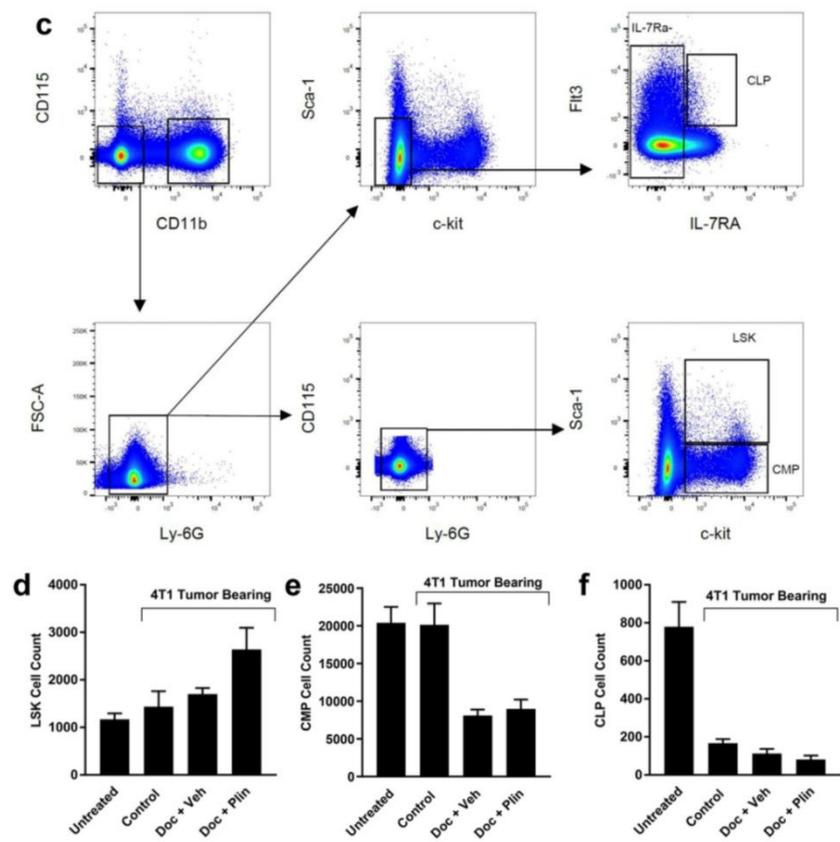
1. Lin et al., Med 6(10):100752 (2025)

Plinabulin Unique MoA in Reducing Chemotherapy-induced Neutropenia (CIN) Demonstrated in 6 clinical studies for docetaxel and other chemo with early onset action



Plinabulin: non-G-CSF based treatment for CIN¹
 - Generates HSPC, including LSK cells
 - GEF-H1 is shown to regulate HSPC proliferation²

Different from G-CSF, Plinabulin cause limited bone pain and limited platelet decrease, sustained QoL³



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 As a GEF-H1 Agonist Contribution to Its Biology Effect and Good Safety Profile

- **Good safety profile**

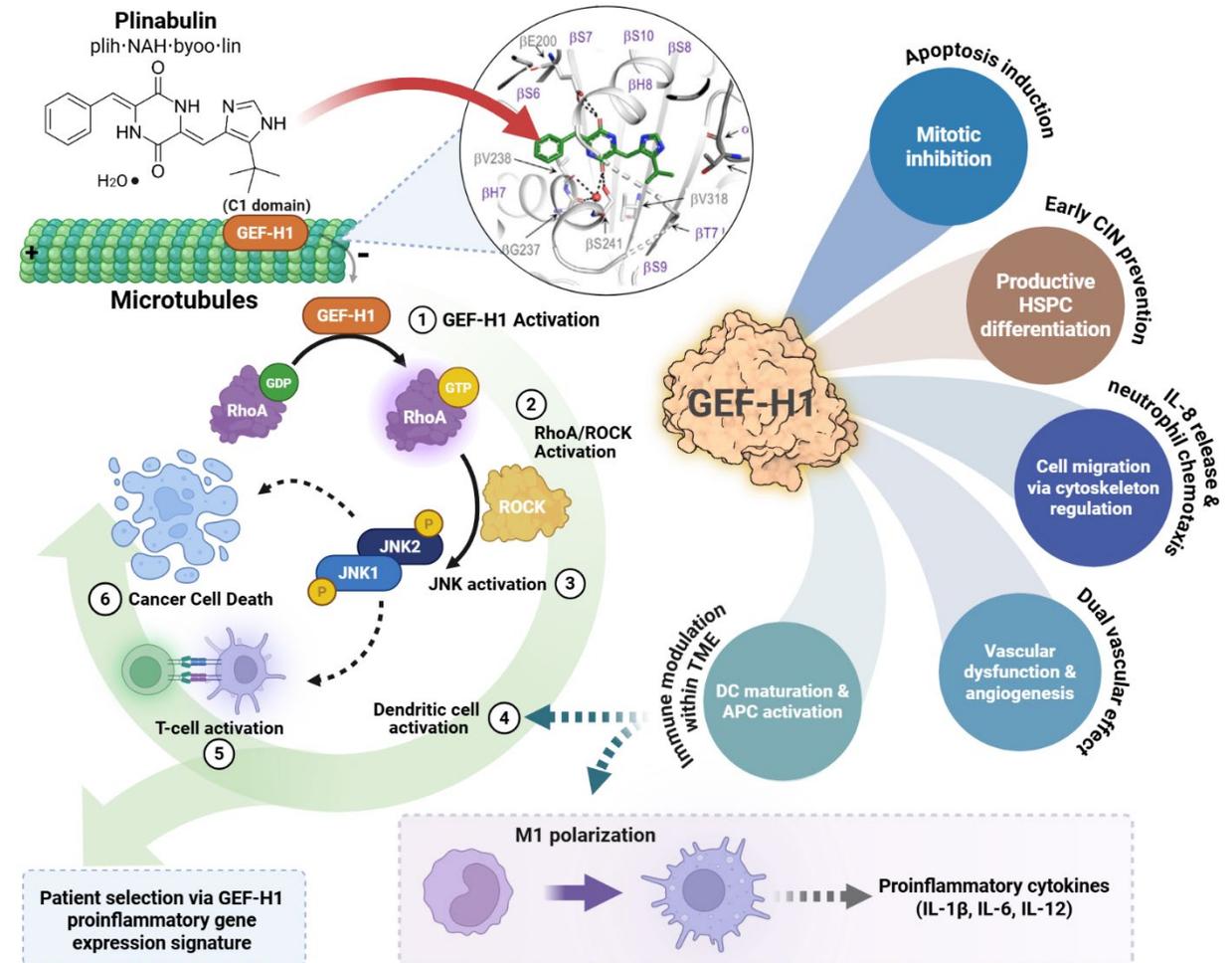
Binds in a unique pocket of β -tubulin with fast off-rate, which contributes to its good safety profile

- **First-in-class GEF-H1 agonist**

Contributes to MoA DC maturation, CIN reduction and vascular modulation

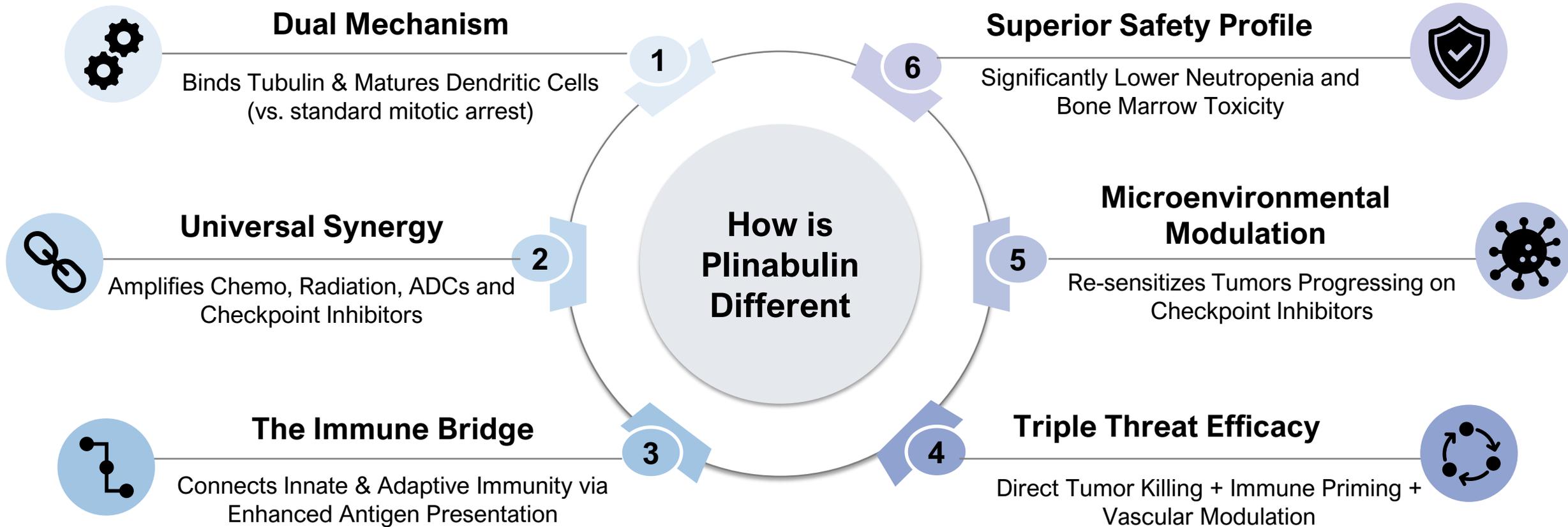
- **Synergistic anti-cancer benefit**

Capable of combining with docetaxel/other chemo, ADC, or radiation to leverage real-time tumor antigens



Plinabulin Differentiated Profile

Mechanism Supported | Clinically Validated | Favorable Safety | Scalable Opportunity



Plinabulin Clinical Studies in Multiple Cancers

>700 cancer patients treated with Plinabulin with good tolerability



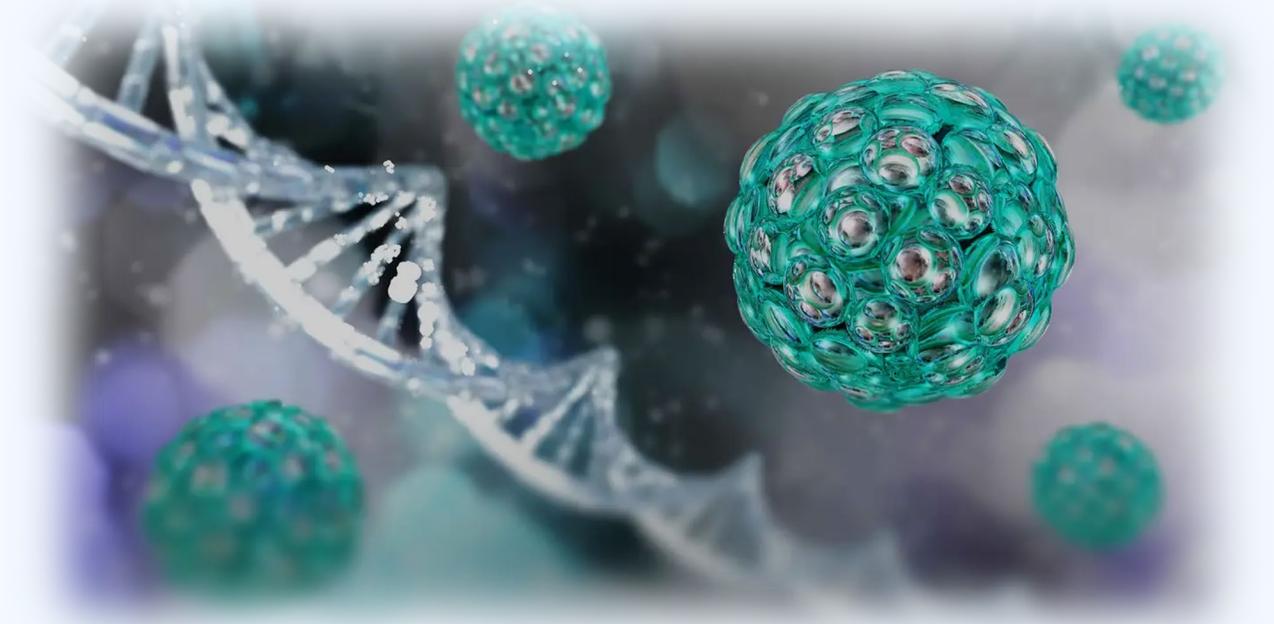
	Indication/Target	Program	Preclinical	Phase 1	Phase 2	Phase 3	Registration	Trial Name / Collaborator	
Late stage	NSCLC (2 nd /3 rd line)	Plinabulin + Docetaxel	[Progress bar]						Study 103 (DUBLIN-3) - OS, PFS, ORR benefit ¹
	CIN Prevention	Plinabulin alone or + Pegfilgrastim	[Progress bar]						Studies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

- 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)



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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 main option, with poor outcome and high toxicity
- Urgent need for differentiated therapy with immune re-sensitization

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: ~9-11 months
- 40% experience severe neutropenia

Industry-wide Phase 3 Trials Summary

11 Phase 3 Studies with no OS Benefit vs. Docetaxel

- **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 et al. 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)



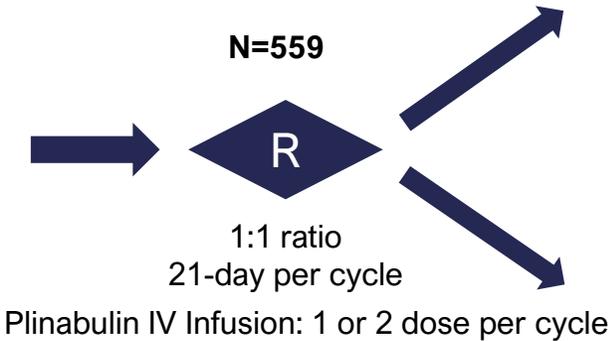
Docetaxel + Plinabulin vs. Docetaxel + Placebo in Patients with EGFR Wild-Type NSCLC

Study Plan
<ul style="list-style-type: none"> 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
<p>Overall survival (OS)</p>

Secondary endpoints
<ul style="list-style-type: none"> 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:
<ul style="list-style-type: none"> 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



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

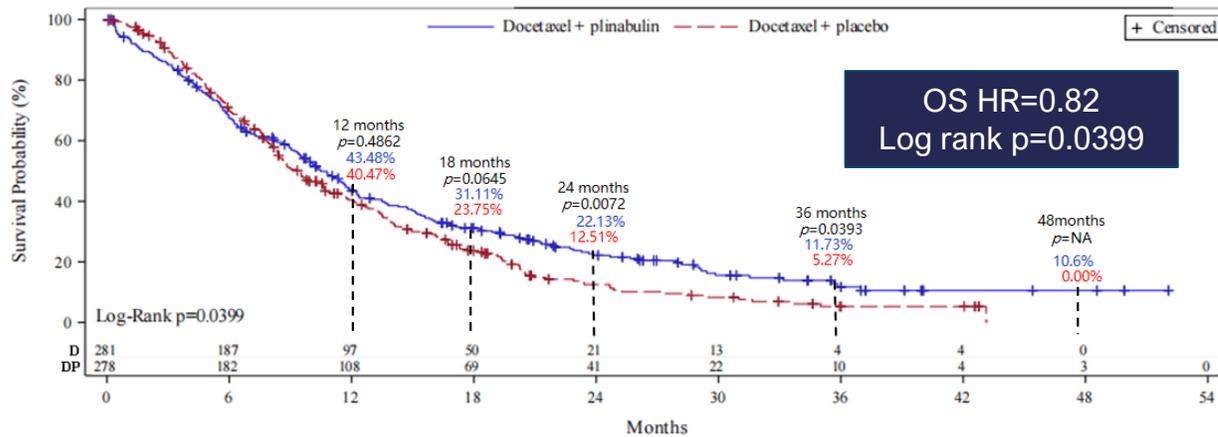
D:
<p>Docetaxel (75 mg/m², day 1) + Placebo (day 1, 8)</p>

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

Plinabulin + Docetaxel vs. Docetaxel (n=559) Met its Primary Endpoint of OS and Secondary Endpoints of PFS, ORR, and Grade 4 Neutropenia Reduction



Plinabulin and Docetaxel Showed Significant Improvement in Long-term OS Rate - Double 2-year, 3-year OS Rate



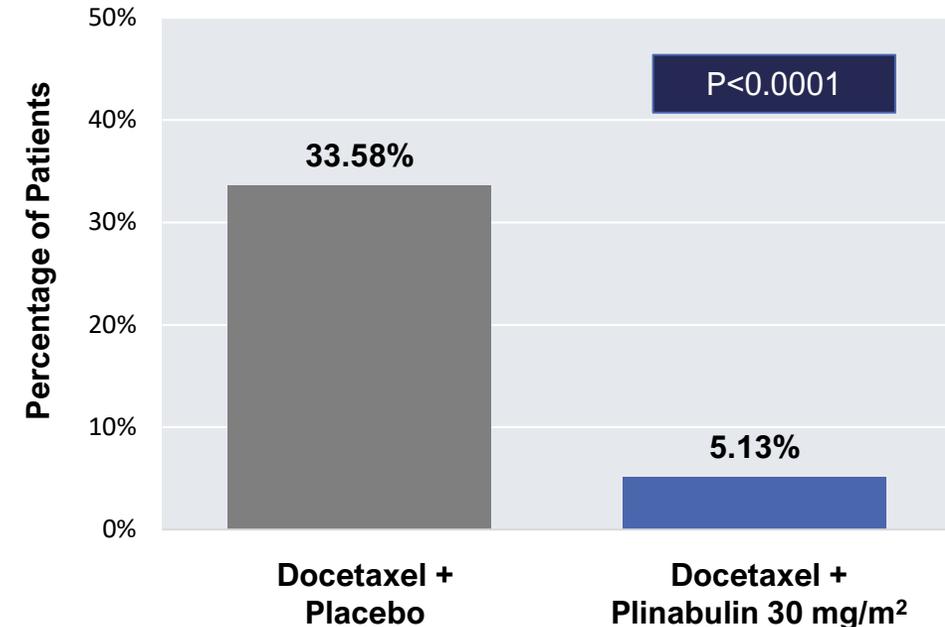
Treatment	Subjects	Event	Censored	Median (95% CI)	HR (95% CI)
Docetaxel + placebo	281	230 (81.9%)	51 (18.1%)	9.40 (8.38, 10.68)	
Docetaxel + plinabulin	278	214 (77.0%)	64 (23.0%)	10.49 (9.34, 11.87)	0.822(0.681, 0.991)

	Mean OS (SE)	Median OS (95% CI)	HR
Docetaxel	12.77 (0.676)	9.4 (8.4, 10.7)	
Plinabulin + Docetaxel	15.05 (0.848)	10.5 (9.3, 11.9)	0.82 (0.68, 0.99)

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

Plinabulin Significantly Reduced Grade 4 neutropenia of Docetaxel

Grade 4 neutropenia, All Cycles Day 8



Similar results for Grade 4 neutropenia on Cycle 1 Day 8
- Day 8 is ANC Nadir for Docetaxel

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		Grade 4 Only Event Rates	
	Adjusted by Exposure		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	

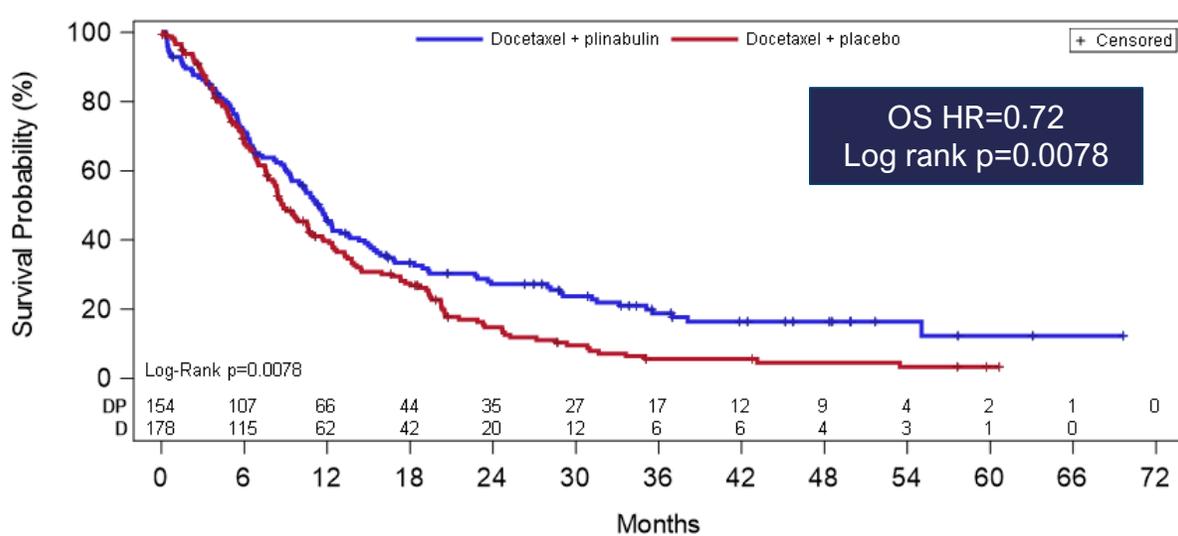
Plinabulin and Docetaxel Combo arm had more cycles of treatment vs. Docetaxel alone.

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

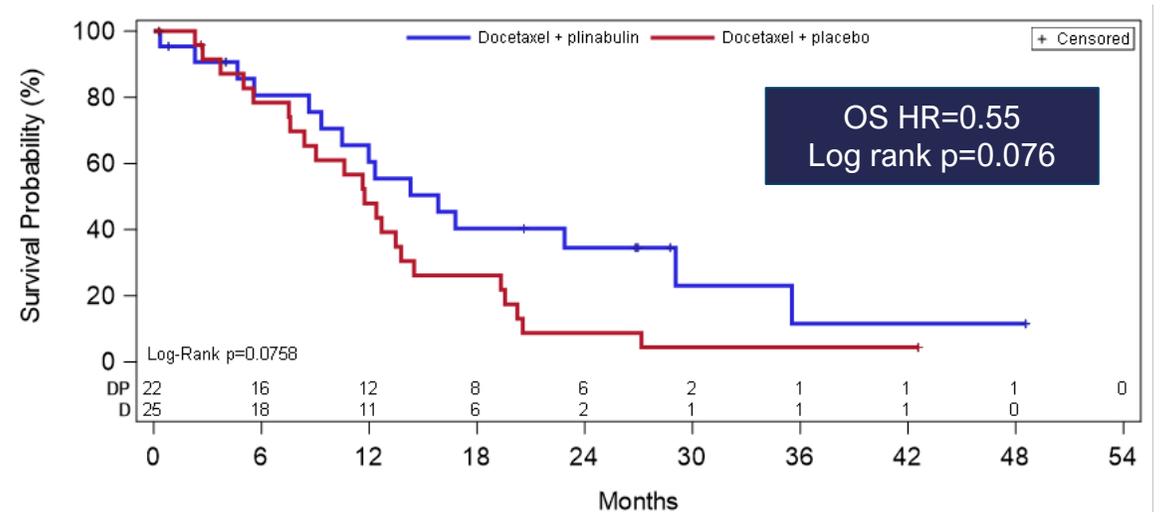
Dublin-3: Post-hoc Analysis in Plinabulin Mechanism-Based Population in 2L/3L Non-squamous EGFR WT NSCLC and Progressed on PD-1/L1 Inhibitors



Non-squamous
 mOS extension of 2.5 months
 (DP 11.4 vs. D 8.8 months, HR 0.72)



**Non-squamous, progressed on ICI
 (Plinabulin MoA-enriched population)**
 mOS extension of 4.1 months
 (DP 15.8 vs. D 11.7 months, HR 0.55)



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

Non-squamous, post-ICI	N	Median OS (95% CI)	HR	Log rank P value
Docetaxel	25	11.7 (7.59, 13.77)		
Plinabulin + Docetaxel	22	15.8 (9.34, 29.06)	0.55 (0.28, 1.07)	P = 0.076

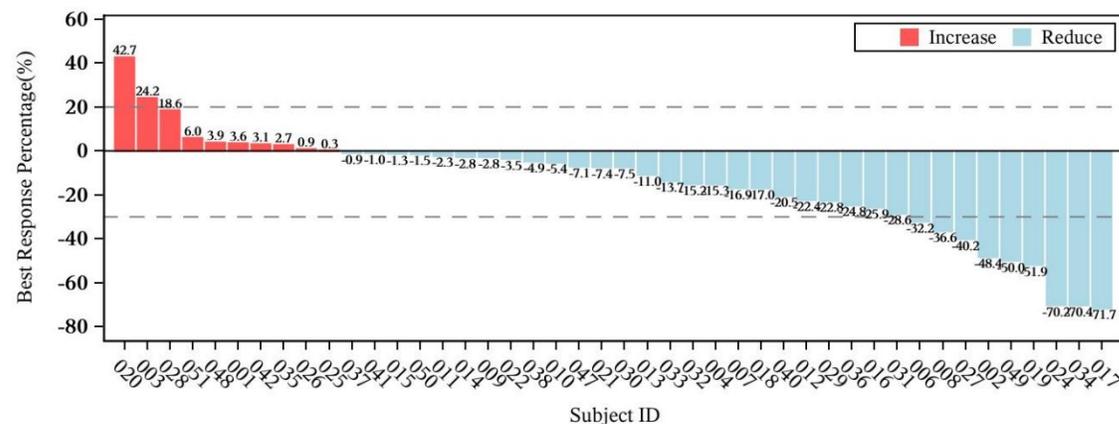
Publication: Lancet Respir Med 12(10): 775-786 (2024)

Study 303 (Plinabulin + Docetaxel + PD-1) in 2L/3L Metastatic NSCLC Progressed on PD-1/L1 Inhibitors Shows Consistent Data as Dublin-3 Analysis

Primary Endpoint (n=47) – SITC 2025	
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%

- 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.

Best Change (%) in Target Lesions



- ✓ **IO Mechanism Support:** Whole blood analysis indicated higher proportions of activated CD4+/CD8+ T-cells post treatment.
- ✓ **Clinically Meaningful Efficacy Data:** 303 study data almost **doubled efficacy** vs. historical data of **docetaxel** in similar patients from TROPOIN LUNG-01 (Thoracic Oncology 43(3): 260-272 (2024)): ORR: 12.8%; mPFS: 3.7 months; mOS: 11.8 months.

Planned Dublin-4 Study As a Confirmatory Phase 3 Study

Plinabulin Mechanism-Enriched Patients



Dublin-4: “Plinabulin + Docetaxel” vs. “Docetaxel” in 2L/3L non-squamous NSCLC, EGFR/ALK/ROS1/RET Wild Type, progressed on PD-1/L1 with PFS \geq 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 mutation¹ by dendritic-cell (DC) maturation MoA. 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

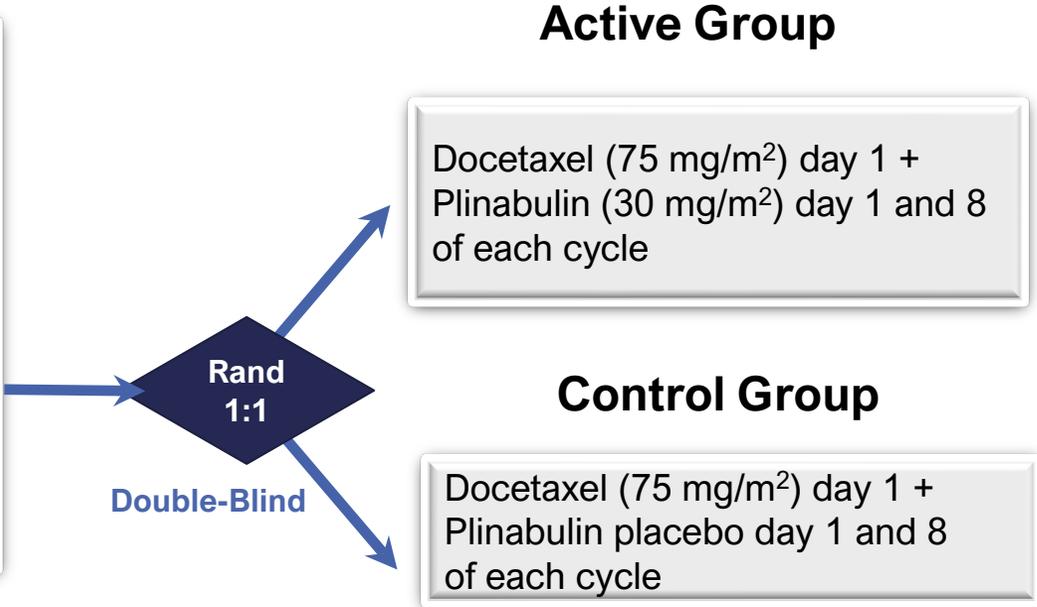
- ✓ *Dublin-3 (DP)*: OS HR 0.55, mOS: 15.8 vs 11.7 months (Plinabulin + docetaxel DP vs docetaxel D), mPFS 5.6 vs. 3.8 months (HR 0.67), ORR 18.2% vs. 8.0%.
- ✓ *303 Study (DP+PD-1)*: 24-month OS rate 66% (mOS 16 month+), mPFS 7.0 month, ORR 18.2%

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

Confirmatory Phase 3 Study Design for Dublin-4



- ✓ Advanced and Metastatic **non-squamous EGFR, ALK, ROS, RET WT NSCLC** patients who have progressed after prior immunotherapy (anti-PD-(L)1 antibody) with platinum containing chemotherapy concurrently or sequentially.
- ✓ Subjects **treated with anti-PD-(L)1 antibody** with PFS \geq 3 months and platinum doublet.
- ✓ ECOG 0 and 1.



- Primary endpoint: OS**
- Secondary endpoints:**
- ✓ PFS, ORR, Grade 4 neutropenia based on ANC,
 - ✓ 2-year and 3-year OS Rates, DoR, DCR,
 - ✓ PROs (QLQ-LC13),
 - ✓ New Brain Mets
- Safety:**
- ✓ Neutropenic Fever

	Global: N=442	
Study Group	Active	Control
Patient Numbers	221	221

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



TEAE	Docetaxel + Placebo N=278 n (%)			Docetaxel + Plinabulin N=274 n (%)		
	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)

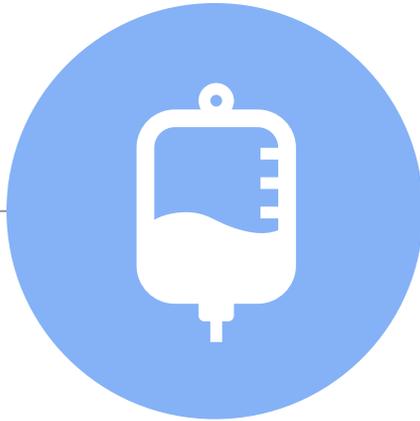
Publication: Lancet Respir Med 12(10): 775-786 (2024)

Plinabulin + Docetaxel: Addressing Unmet Needs in 2L/3L NSCLC, EGFR WT, Progressed on PD-1/L1 Inhibitor, with Potential Positive Benefit/Risk Ratio



Severity of Unmet Need	Current SOC - Docetaxel	Potential Solution - Plinabulin + Docetaxel
● ● ●	Lack of durable response post-immunotherapy	Strong immune re-priming and antigen presentation, 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 and other wild-type patients

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



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	<ul style="list-style-type: none">● Addressing 80% of disease-causing proteins considered “undruggable” by traditional methods
	Technology Platform	<ul style="list-style-type: none">● 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	<ul style="list-style-type: none">● 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	<ul style="list-style-type: none">● ~\$60M in equity, collaboration upfronts, and milestones since inception● \$30M Series A-3 closed in September 2025
	World-class team	<ul style="list-style-type: none">● 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

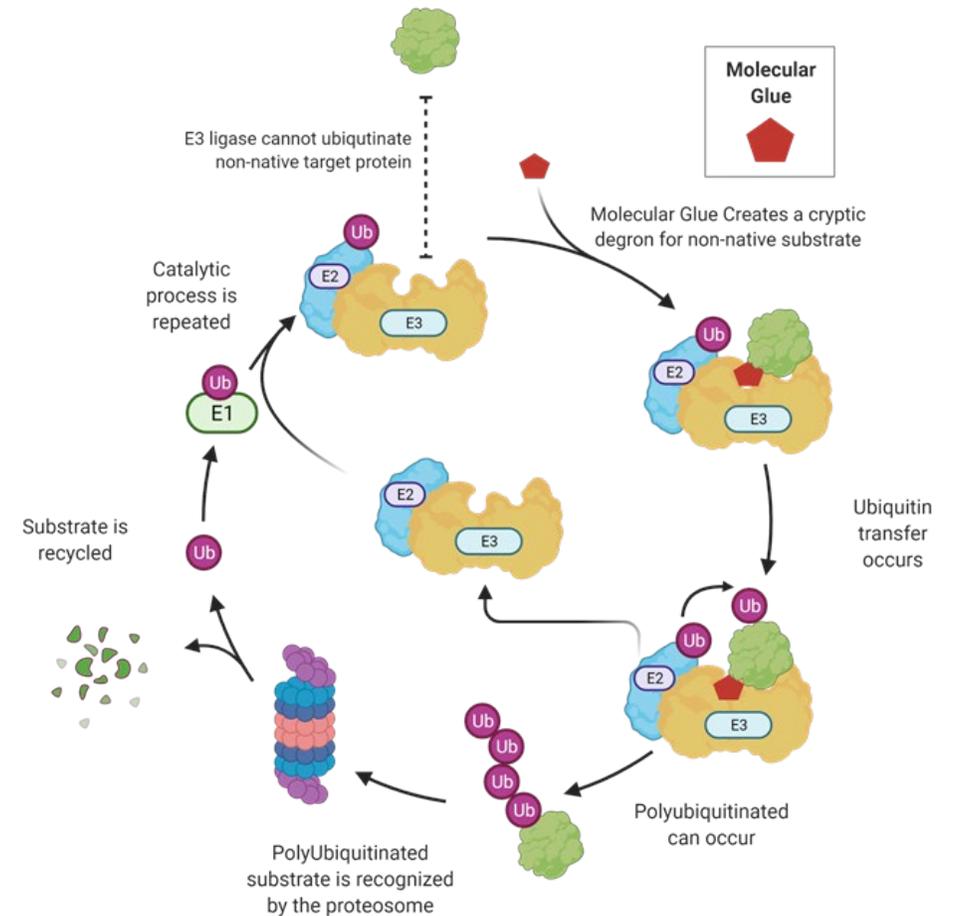
- >600 E3 ligases (only 2 structure classes: HECT & RING, both solved by SEED co-founders)
- Enables degradation of undruggable proteins

Integrated Validation

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

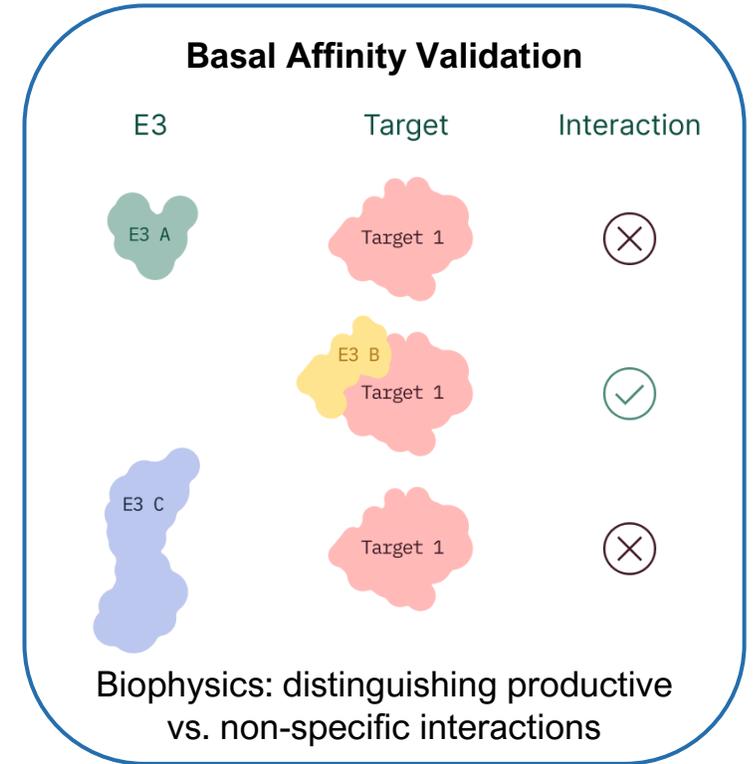
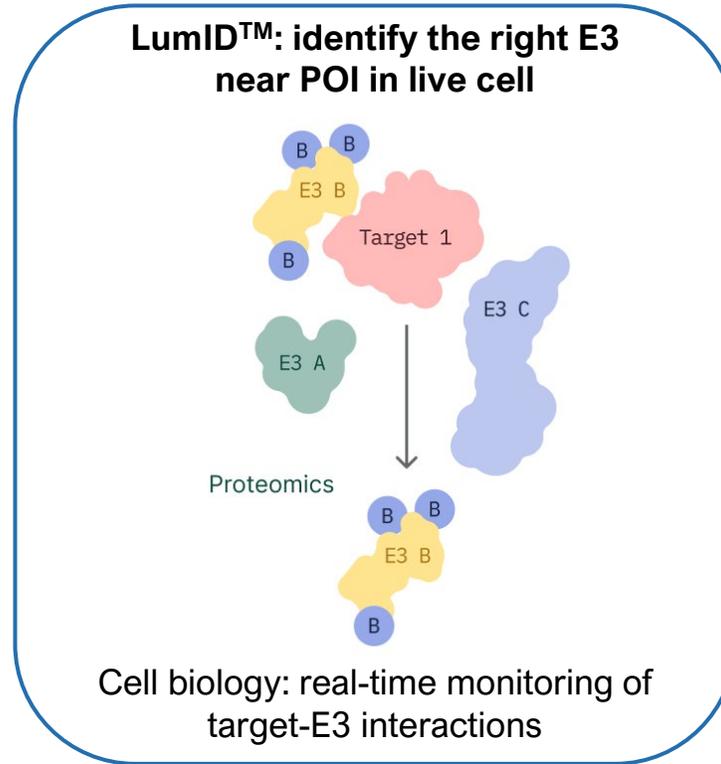
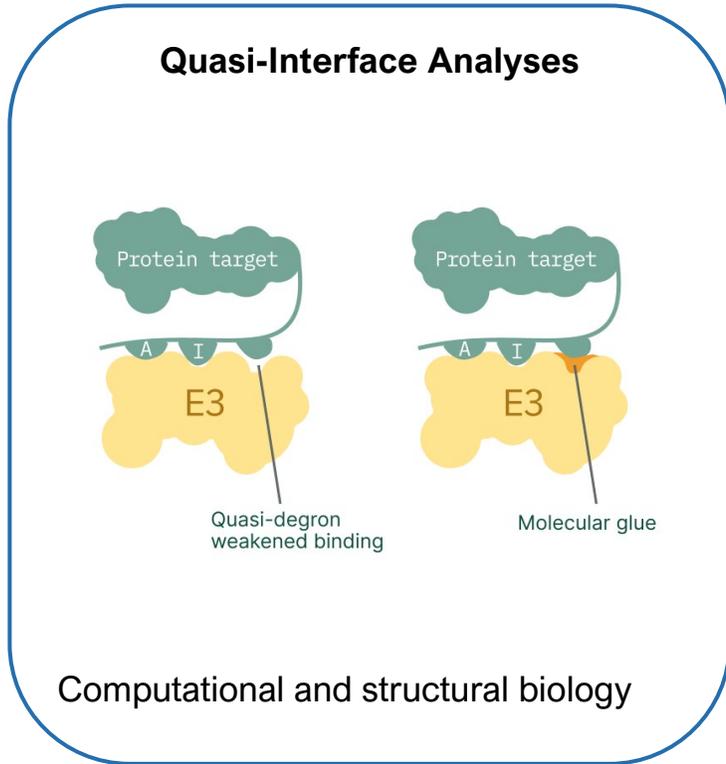
Broad & Validated Impact

- 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



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	Target Selection	E3 Ligase ID	Molecular Glue HTS	Lead ID	IND Candidate	IND Filing	Phase 1	Milestones
Oncology	RBM39	 ST-01156							<ul style="list-style-type: none"> ✓ 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



- 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



- 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



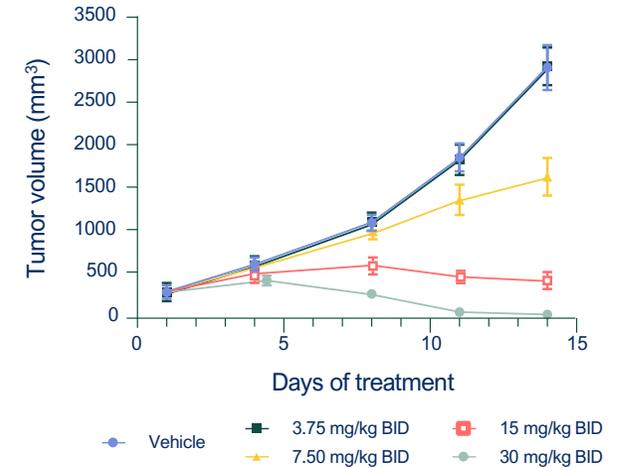
Ewing Sarcoma Is A Fusion-driven Disease with No New Drugs in the Past 30 Years

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

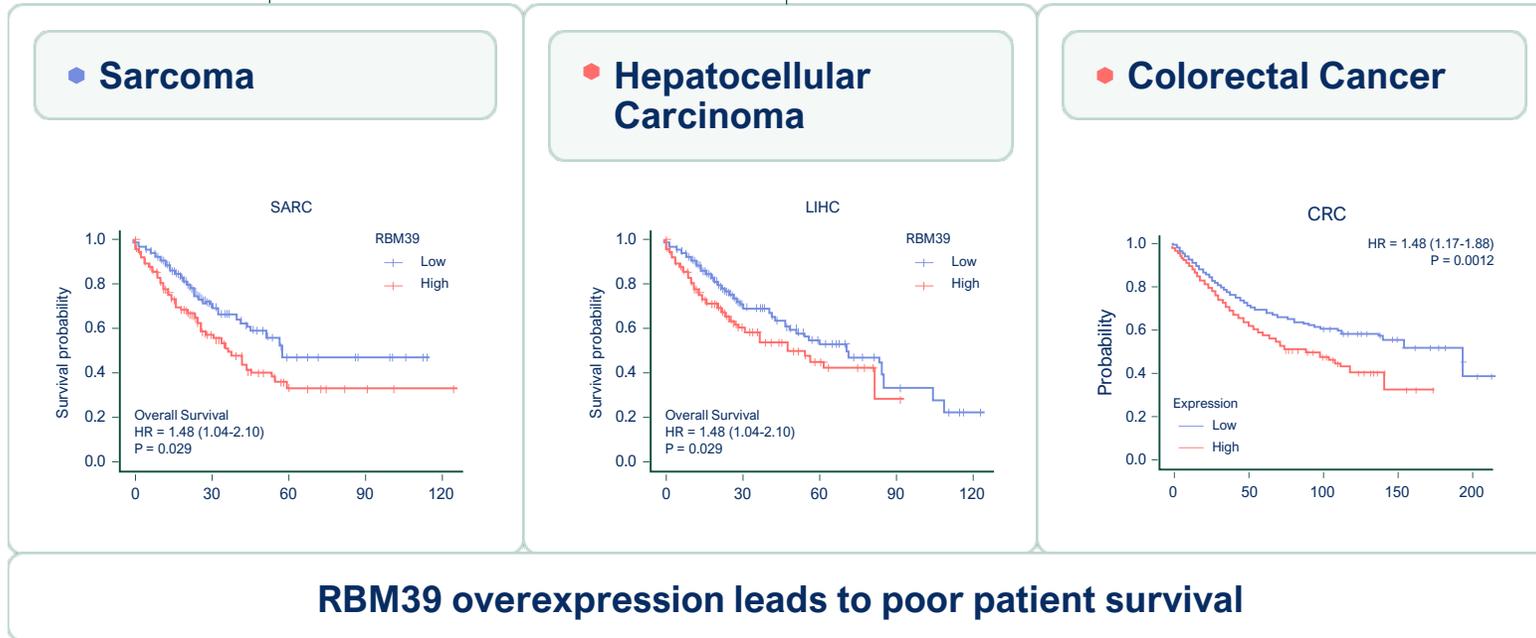


Precise target engagement

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

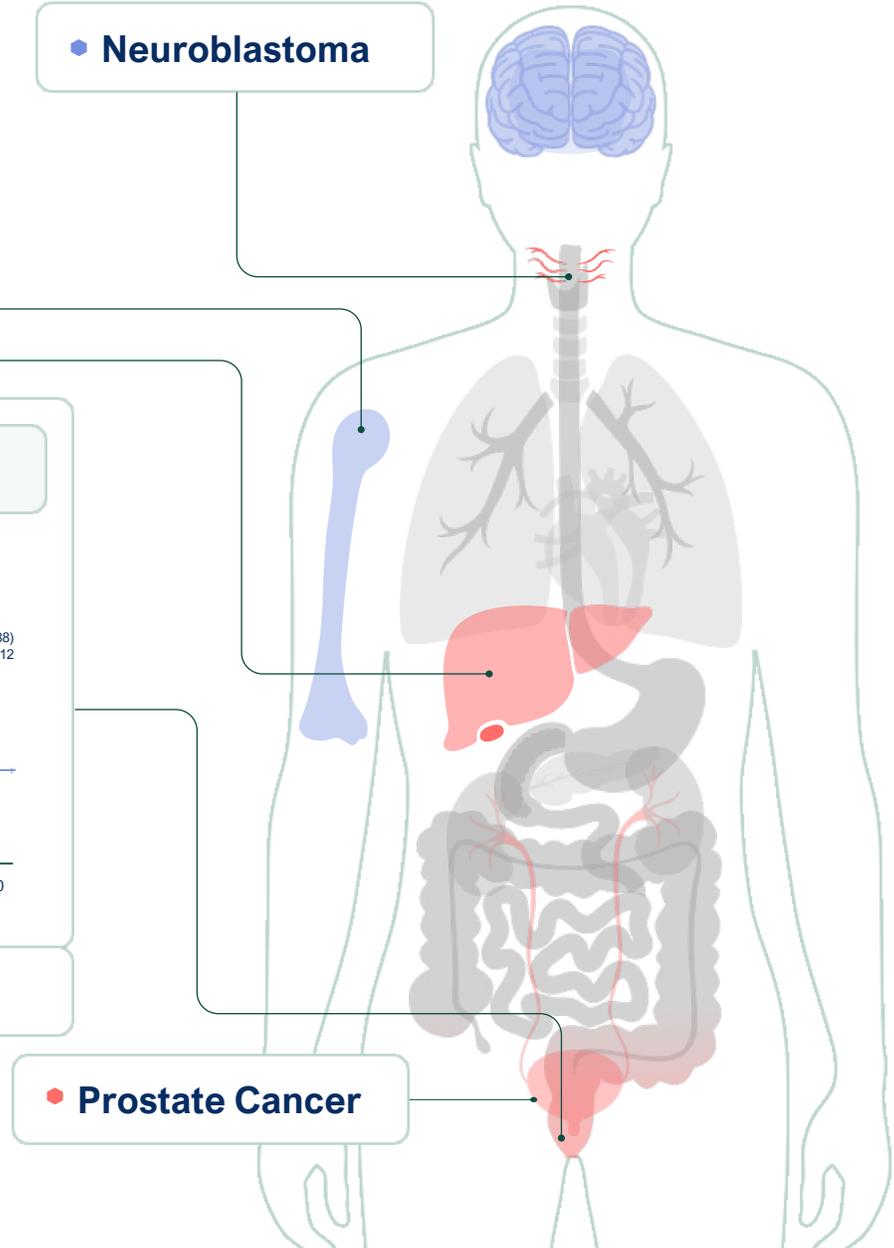


RBM39 Drives Progression in Rare and Large Tumor Indications



◆ Pediatric tumors
 ◆ High prevalence tumors

Initial total addressable market = 1,067,280 patients

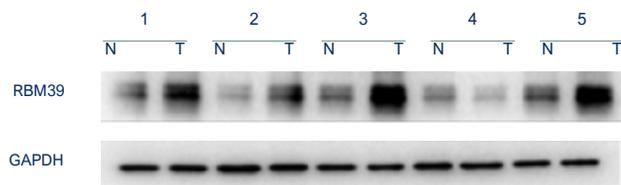


Strong Anti-tumor Activity in RBM39-dependent Liver & Colon Cancer Models



Colon Cancer

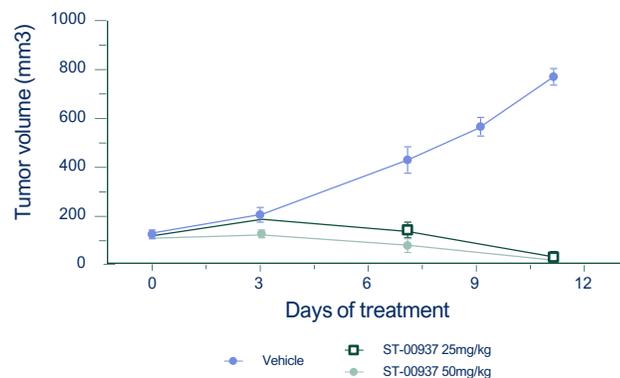
RBM39 high expression in colon cancer (T), not in normal tissue (N)



N = No Tumor
T = Tumor

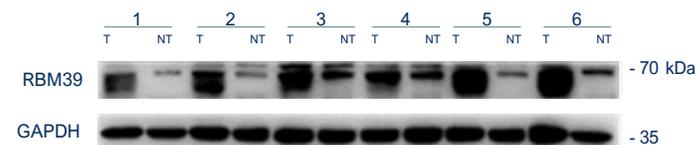
Wang et al., J of Cancer, 2025

Complete tumor regression in colon cancer model



Liver Cancer

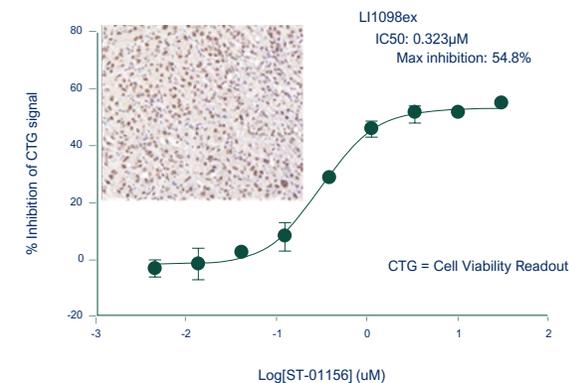
RBM39 high expression in liver cancer (T), not in normal tissue (NT)



N = No Tumor
T = Tumor

Xia et al., Cell Death Discovery, 2023

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 $>10\times$ 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.



Nurix Closes \$120 Million Financing to Support Protein Degradation Program



Pre-IND / IND stage TPD assets

\$100- \$300M in upfront payments and up to \$2B potential milestones.



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



Clinical stage TPD assets (Phases I & II)

\$150 - \$650M in upfront payments, \$350M investment and \$2.1B in potential milestones.



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
P H A R M A C E U T I C A L S

Thank You

General Inquiries: GENERAL@beyondspringpharma.com

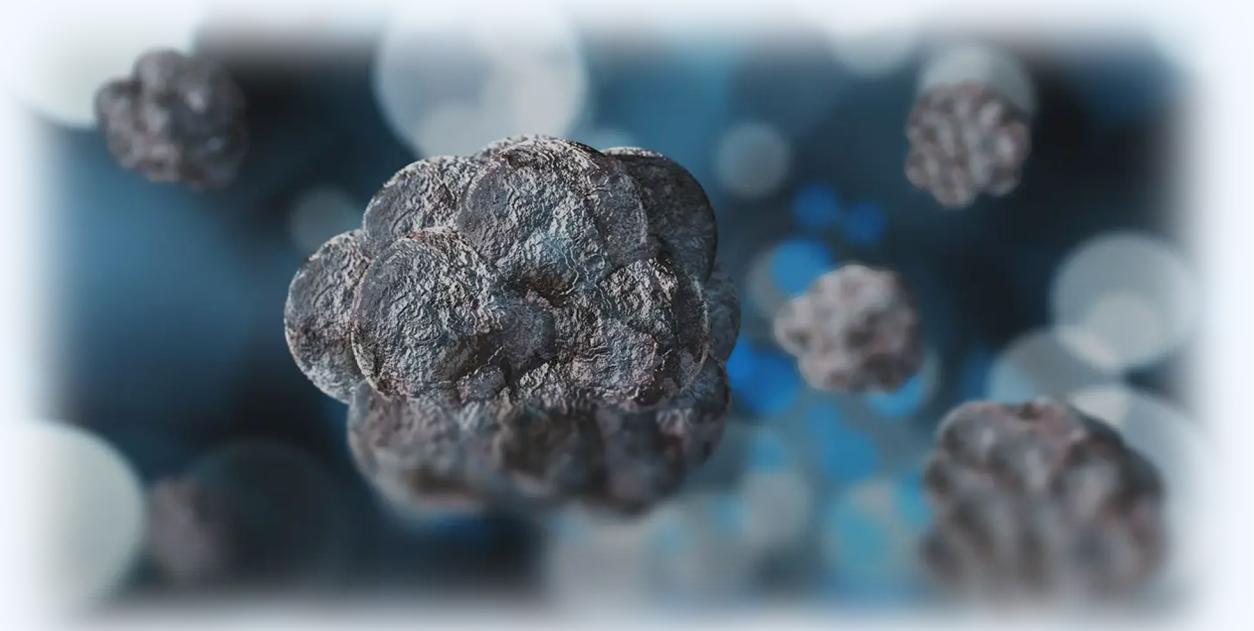
Investor: IR@beyondspringpharma.com

Media: PR@beyondspringpharma.com

www.beyondspringpharma.com



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.



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

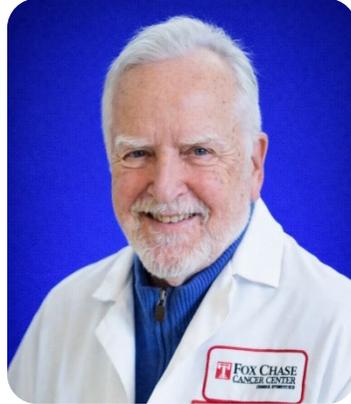
Finance executive with nearly 30 years' experience in China, CFO and board member of BOJI Health, advising numerous pre-IPO, IPO, and restructuring transactions across China and Hong Kong.

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.



Alfred Zippelius, MD
University Hospital Basel

Co-Chair of Medical Oncology and Professor at the University of Basel, leading translational cancer immunotherapy research focused on immune checkpoint resistance and innovative cellular therapies development.