BeyondSpring PHARMACEUTICALS

Transforming Cancer Care with Immune-modulating Therapies

NASDAQ: BYSI

June 2026

Non-Confidential

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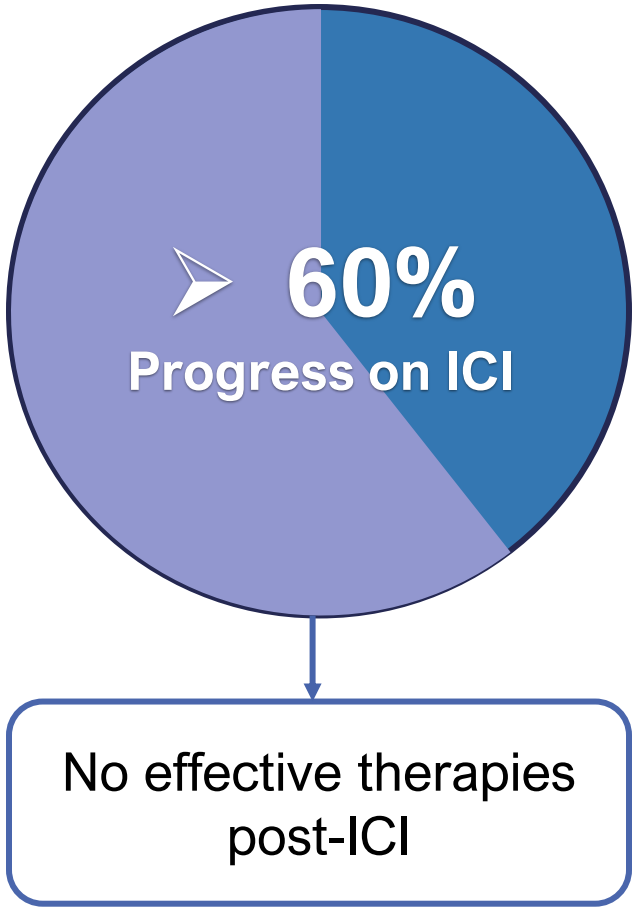
Investment Highlights

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 Wild-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

Post-ICI Landscape: Severe Unmet Medical Need



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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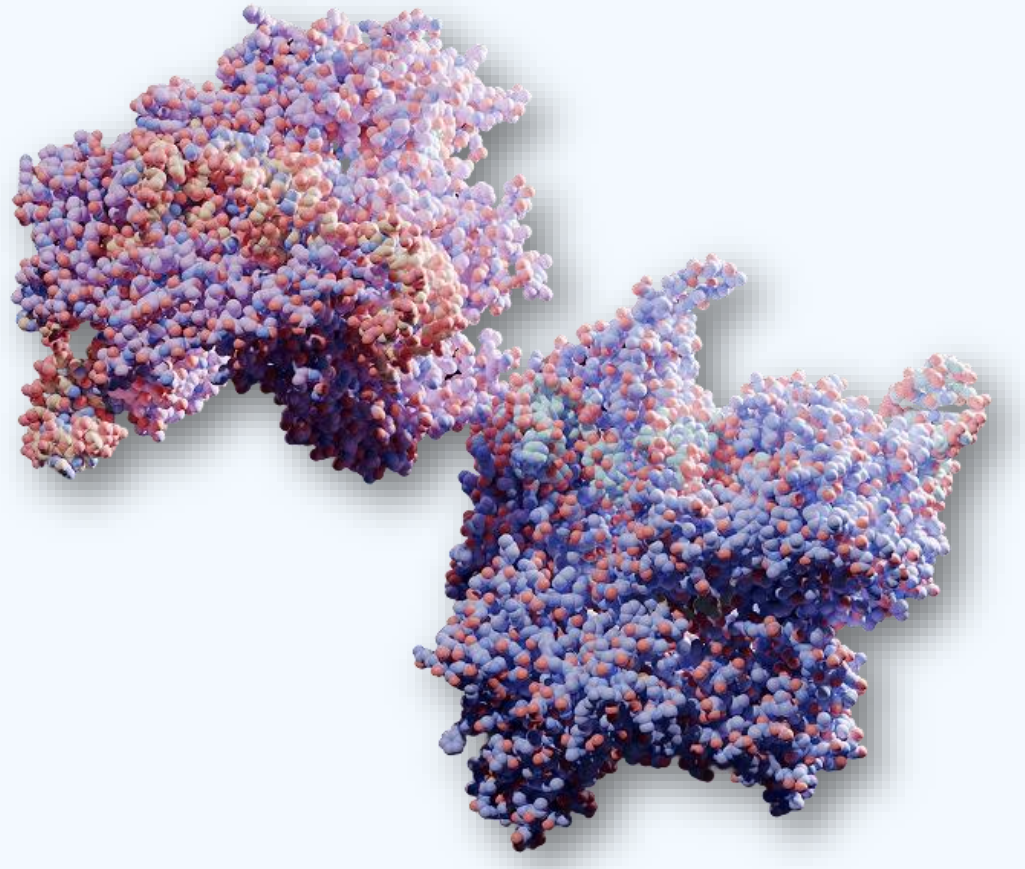


Plinabulin: Unique Dual Mechanism with Immune and Safety Benefits

- **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

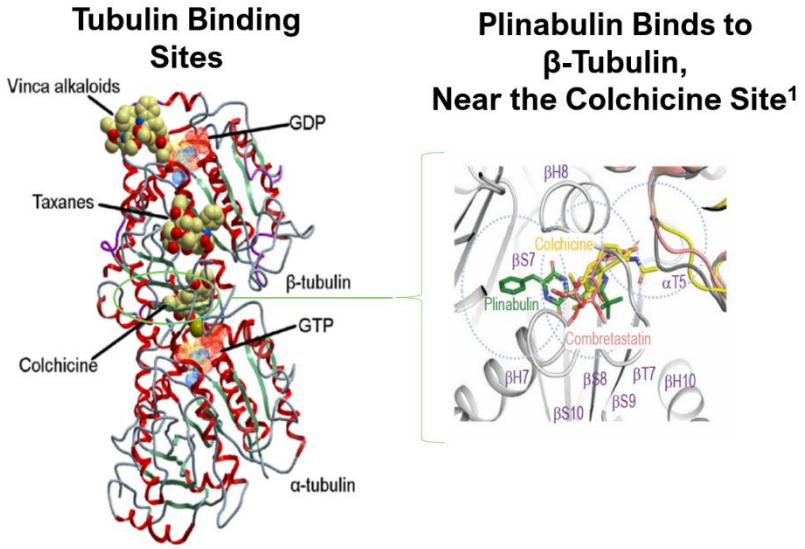
- **Robust clinical foundation**: >700 cancer patients treated with good tolerability.
- **Unique MoA**: Brain-penetrating, reversible tubulin binder driving dendritic cell maturation and T cell activation (Chem 2019, Cell Reports 2019, Med 2025); reduces chemotherapy-induced neutropenia (JAMA Oncology 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**: Based on 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.

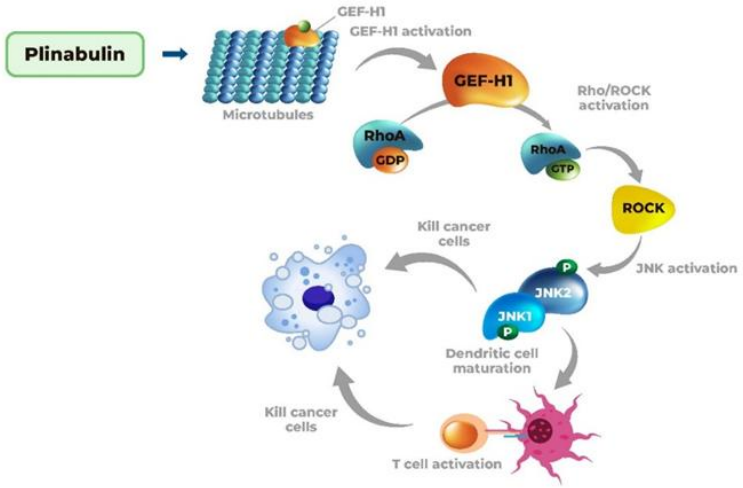
Plinabulin Monohydrate is a Brain-Penetrant, Unique Tubulin Depolymerizing Agent which induces “Dendritic Cell or DC Maturation via GEF-H1 release”

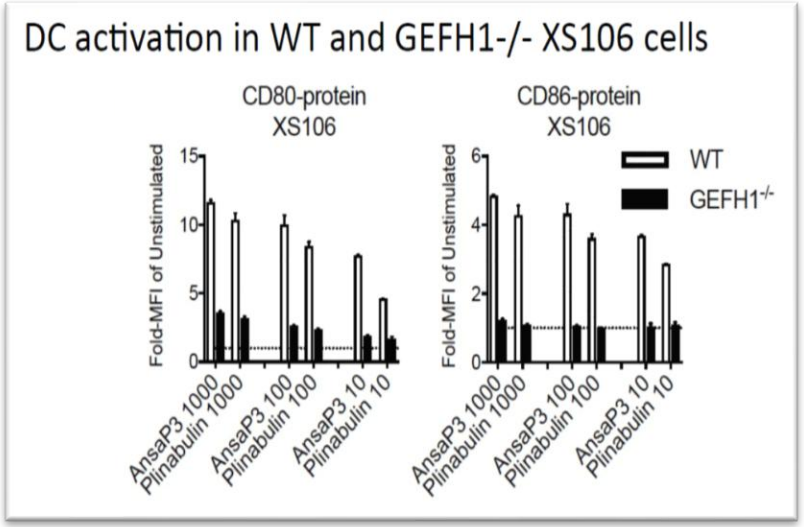
Plinabulin Monohydrate is a unique tubulin binder[1]

Plinabulin releases 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 cells[2]

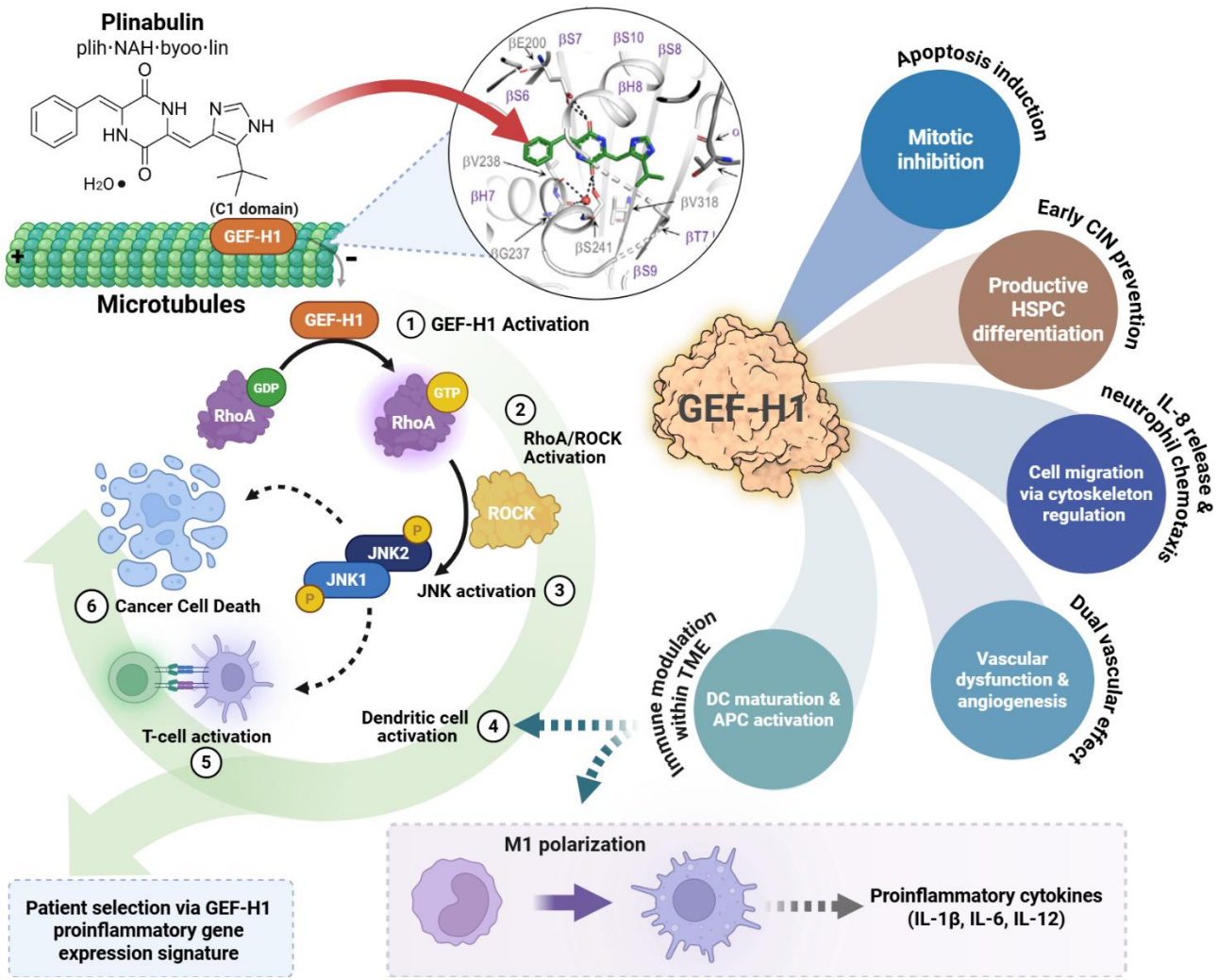






1. La Sala et al., Chem 5(11): 2969-2986 (2019); 2. Kashyap et al., Cell Reports 28(13): 3367-3380 (2019); 3. Choi et al. Cell 189 (2): 461-477 (2026)

Plinabulin Displays GEF-H1-mediated Mechanism-of-Action



- By depolymerizing microtubules, plinabulin activates **GEF-H1,** a C1 domain-associated RhoA activator**, in a highly regulated and spatio-temporal dependent manner.**[1]
- **As a GEF-H1 agonist, plinabulin has the following anti-cancer mechanism:**

a) GEF-H1 activates RhoA/ROCK signaling pathway and leads to **DC maturation/M1 polarization and T-cell activation**, which has been validated in preclinical and clinical studies.[2-4]

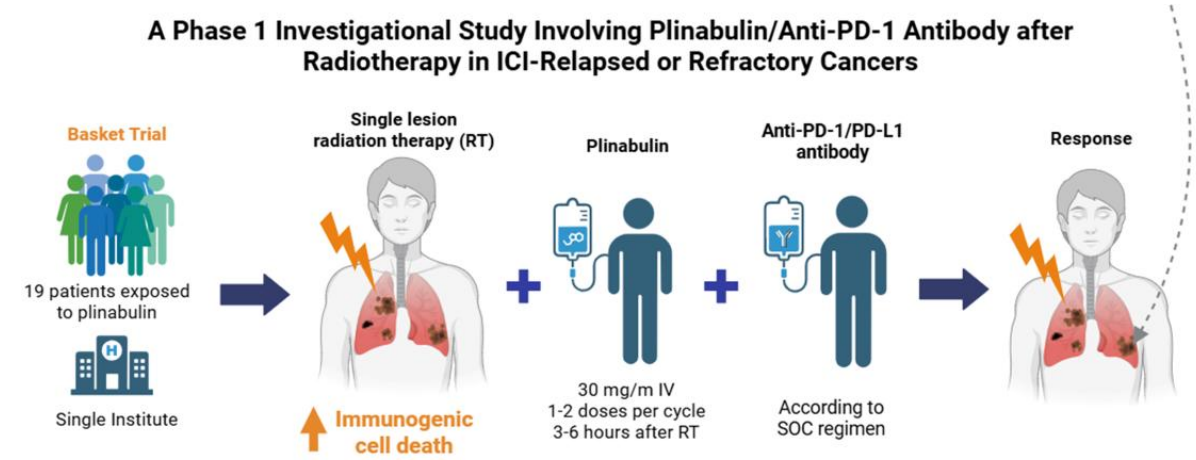
b) GEF-H1 promotes **proliferation of HSPCs** biasing towards the GMP lineage during productive hematopoiesis, contributing to Plinabulin **CIN prevention benefit.**[5-7]

c) Due to GEF-H1's role in **vasculature**, plinabulin **modulates angiogenesis.**[8-9]

1. Choi SR (2026) ***Cell*** 189(2):461; 2. La Sala G (2019) ***Chem*** 5(11):2969; 3. Kashyap AS (2019) ***Cell Rep*** 28(13):3367; 4. Lin SH (2025) ***Med*** 10(6):100752; 5. Tonra JR (2020) ***Cancer Chemother Pharmacol*** 85:461; 6. Blayney DW (2020) ***JAMA Oncol*** 6(11):e204429; 7. Chan DCH (2021) ***Blood Advances*** 5(16):3120; 8. Mita MM (2010) ***Clin Cancer Res***. 16(23)1; 9. Risinger AL (2025) ***EMBO Mol Med*** 17(5):866.

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

In 8 Cancers failed prior ICI, Plinabulin combination with disease control rate of 54%[1]

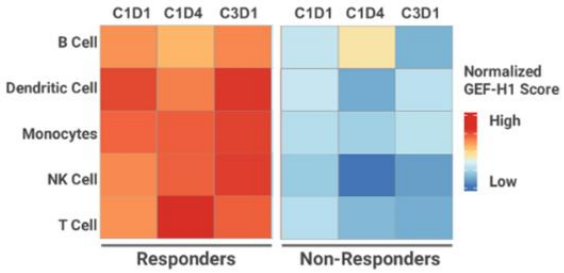


Safety

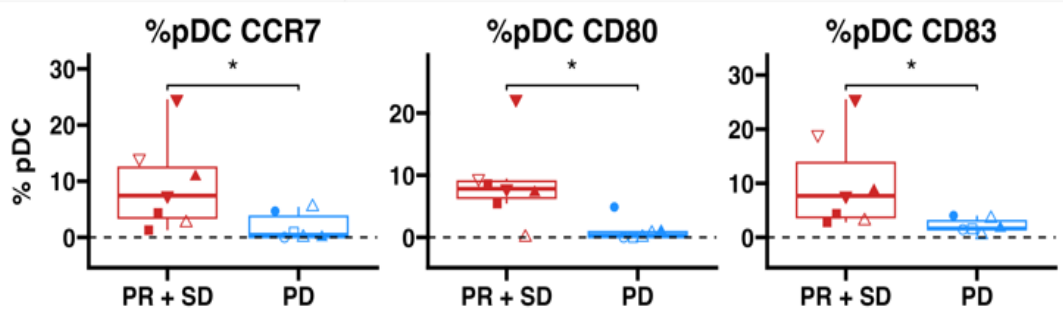
Tolerance

Disease control rate

54%



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

■ Hodgkin lymphoma ○ Merkel Cell Carcinoma ▽ NSCLC ▲ SCCHN

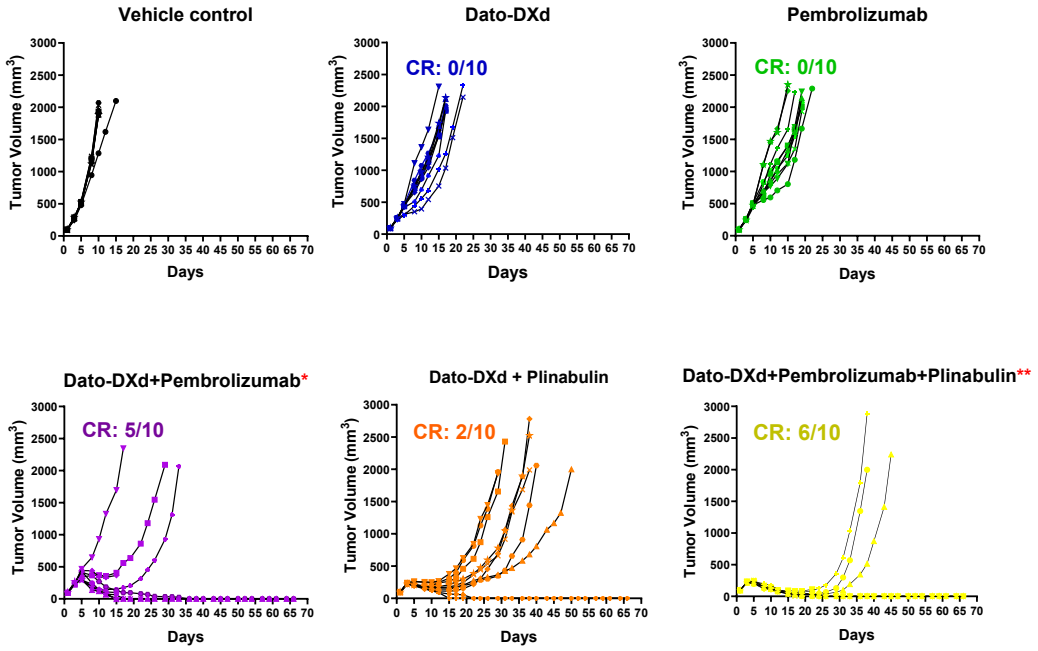
PD

PR + SD

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

Plinabulin Improves Anti-cancer Efficacy and Increases Tolerability of Dato-DXd (TROP2-TOP1 ADC) with Increased CD8+ T cell/Treg Ratio (AACR 2026)

Plinabulin significantly improves complete response rate and survival of ADC with or without PD-1 Inhibitor

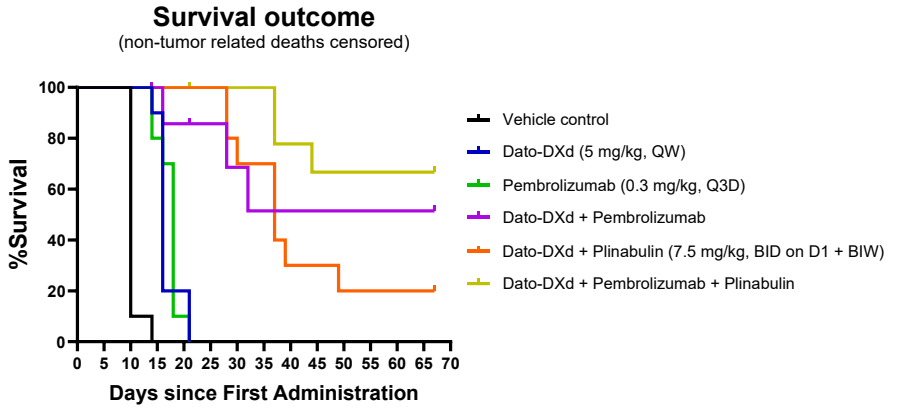


*4/10 animals found dead

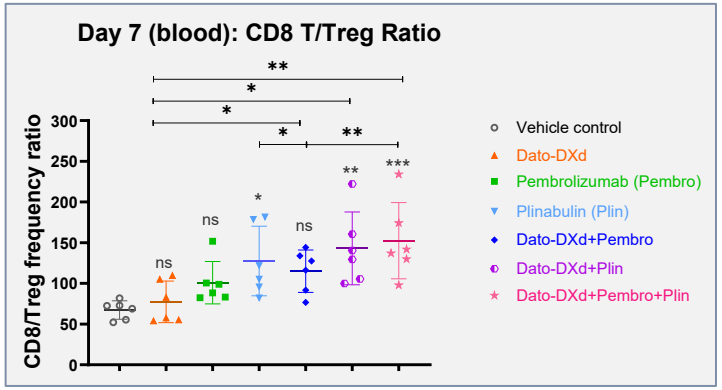
No animal death

**1/10 animals found dead

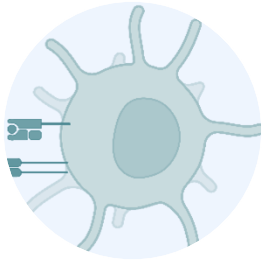
hTROP2-MC38 colorectal cancer in hPD-1 mice



Plinabulin shifts tumor microenvironment to Immune Active by increasing CD8+ T cell/T reg ratio



Plinabulin's Immunomodulation and Neutropenia-Mitigating Activities Position it as a Valuable Addition for Combination Regimens



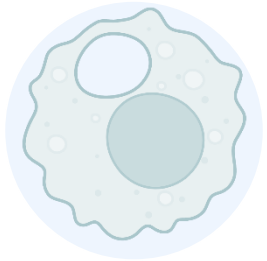
Antigen-Presenting Cell (APC)

Plinabulin induces

DC maturation +

M1 Polarization

Enhanced antigen presentation and T cell priming



Tumor Vasculature

Plinabulin targets

tumor vasculature

Vascular Normalization

Target Acquired Resistance to ICI; Antigen Presentation, Boost T cell function, Kill tumor cells, and Normalize vasculature

Improves safety*

Plinabulin **reduces chemotherapy-induced neutropenia**

Improved therapeutic index of chemotherapy-based regimens

Extends chemo therapeutic duration and improves anti-cancer benefit

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]
	Breast Cancer, NSCLC	Plinabulin + ADC with topoisomerase inhibitors (TOP1-ADC)						

- Mechanisms not restricted to lung cancer; other solid tumors may benefit, with early signals in liver, head and neck, prostate, breast, and ES-SCLC.

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

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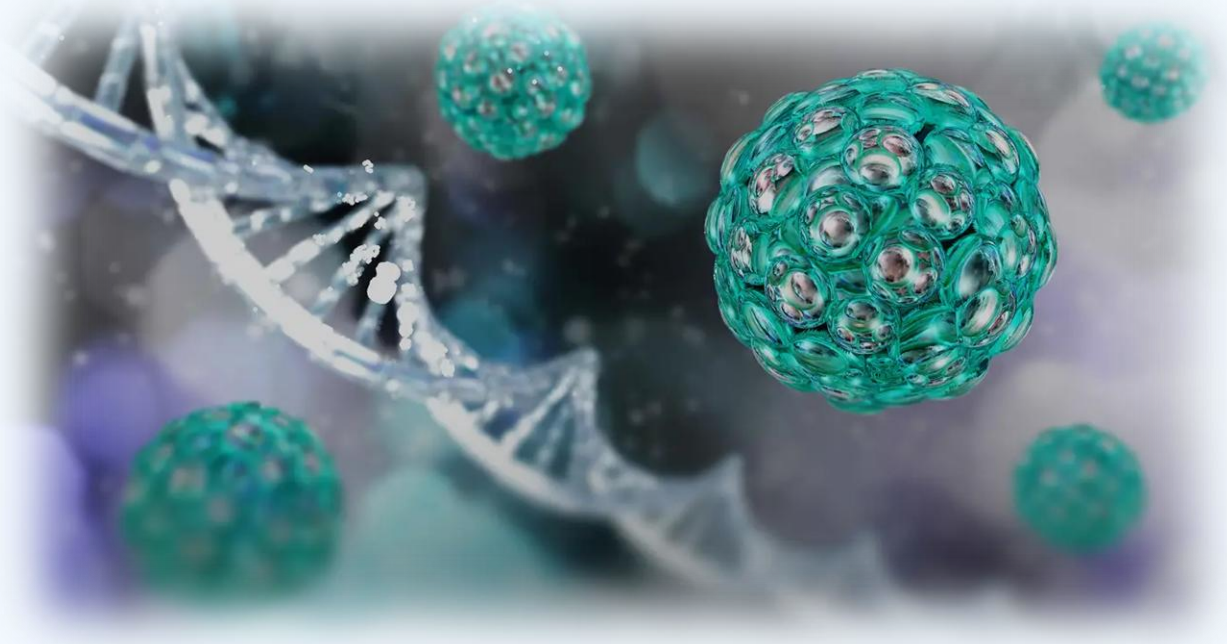


Post-ICI NSCLC: A Large, Growing and Unmet Clinical Challenge

- 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) + pembrolizumab vs Docetaxel

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

- 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

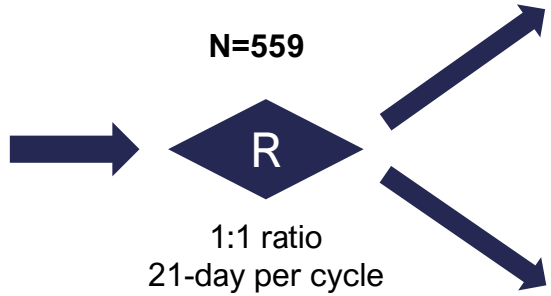
Overall survival (OS)

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



Plinabulin IV Infusion: 1 or 2 dose per cycle

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

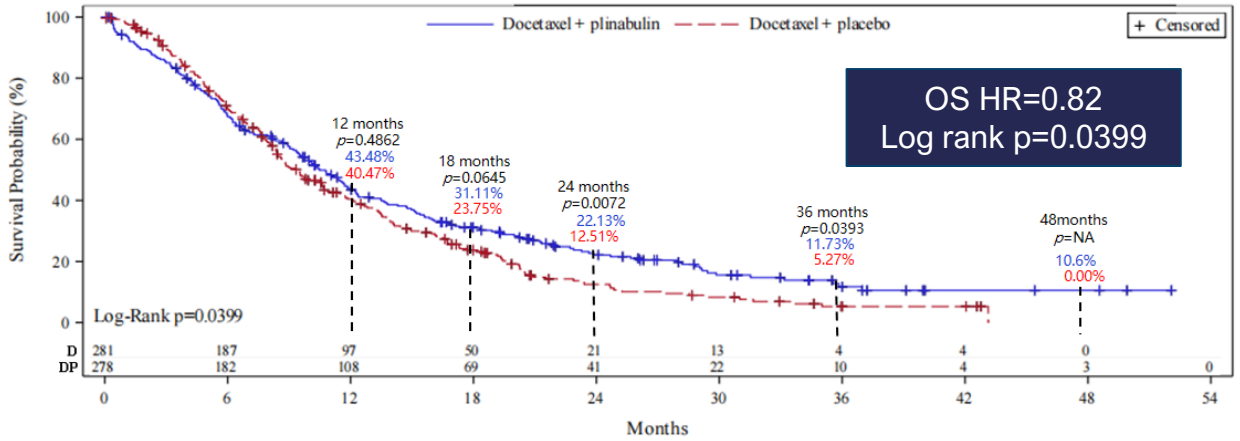
D:
Docetaxel
(75 mg/m2, day 1)
+ Placebo (day 1, 8)

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

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



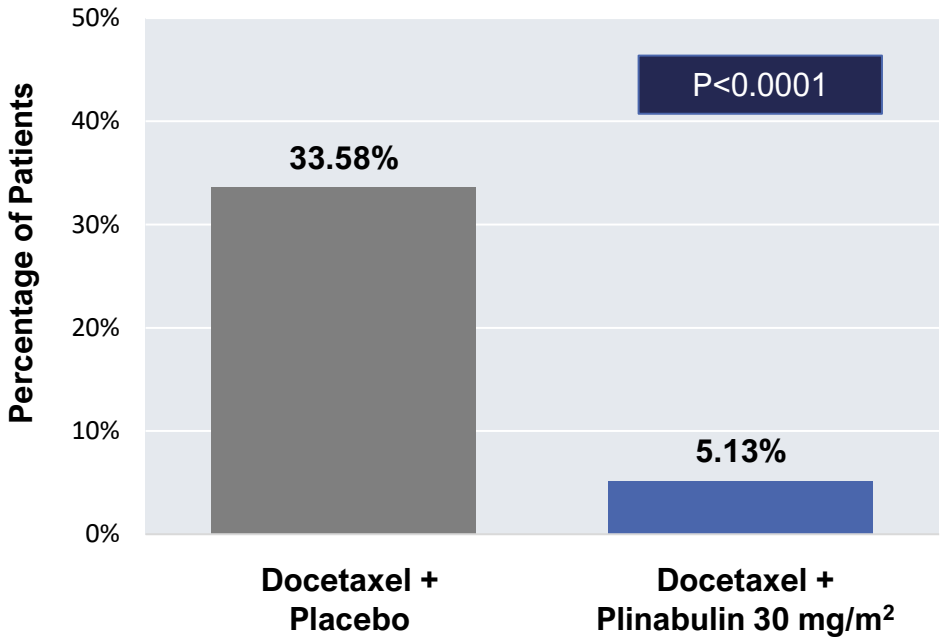
Treatment	Subjects	Event	Censored	Median (95% CI)	HR (95% CI)
Docetaxel + placebo	281	230 (81.9%)	51 (18.1%)	9.40 (8.38,10.68)	
Docetaxel + plinabulin	278	214 (77.0%)	64 (23.0%)	10.49 (9.34,11.87)	0.822(0.681,0.991)

	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 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**	

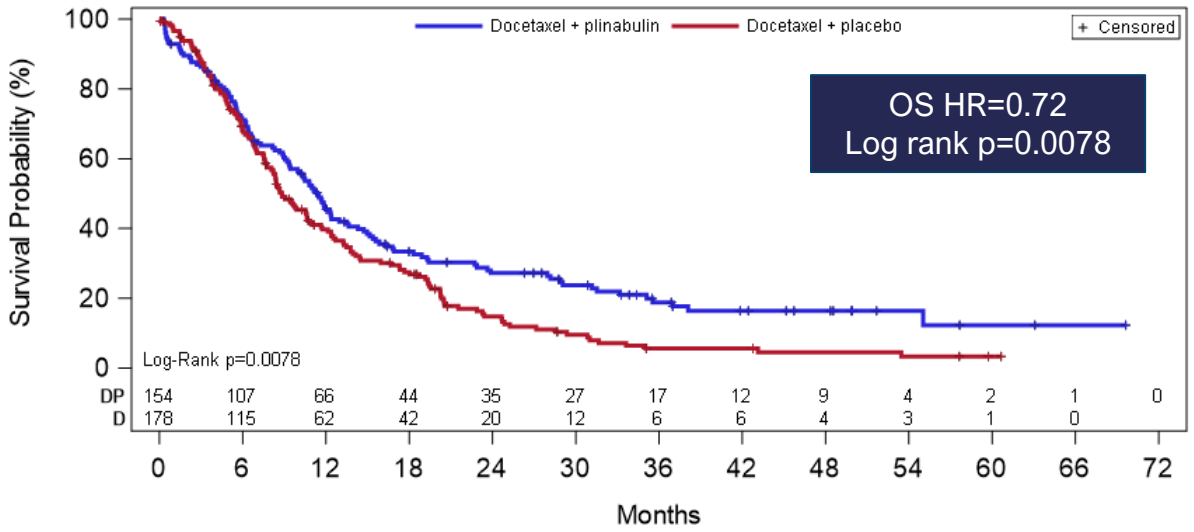
Plinabulin and Docetaxel Combo arm had more cycles of treatment vs. Docetaxel alone.

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

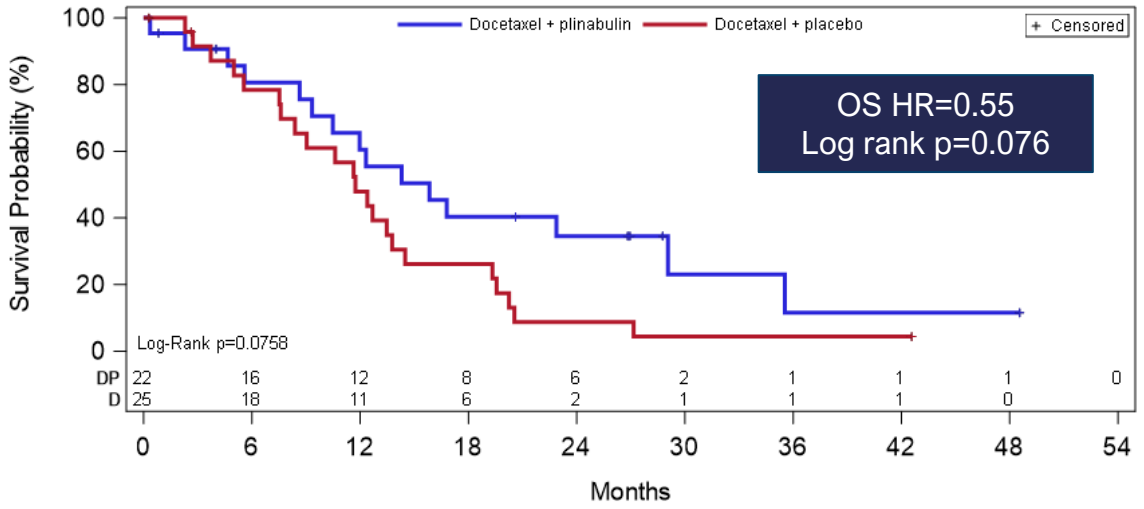
mOS extension of 2.5 months
(DP 11.4 vs. D 8.8 months, HR 0.72)



Non-squamous	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, progressed on ICI (Plinabulin MoA-enriched population)

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



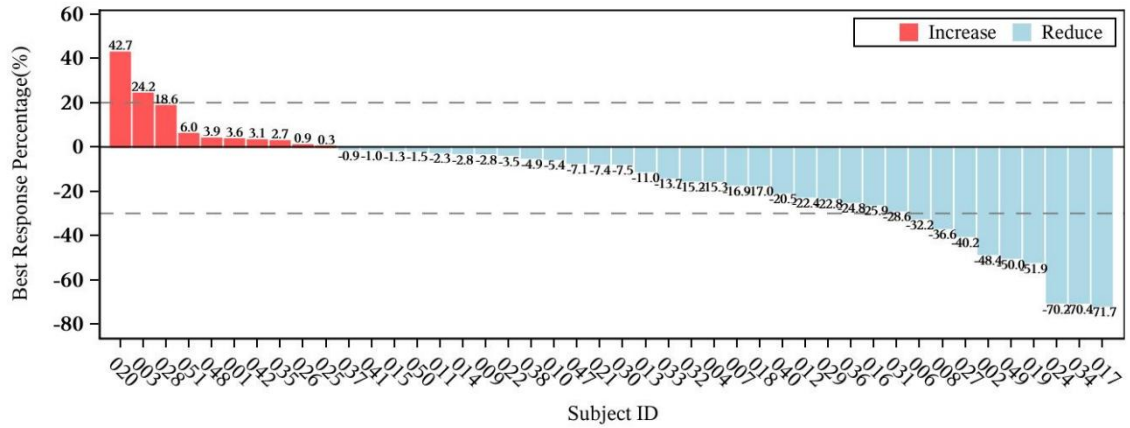
Non-squamous, post-ICI	N	Median OS (95% CI)	HR	Log rank P value
Docetaxel	25	11.7 (7.59, 13.77)		
Plinabulin + Docetaxel	22	15.8 (9.34, 29.06)	**0.55 (0.28, 1.07)**	**P = 0.076**

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) – ASCO 2026	
Confirmed ORR（RECIST 1.1）	**18.2%**
Secondary Endpoint	
Median PFS（RECIST 1.1）	**7.0 months**
Median OS	**Not reached**
Median DoR（RECIST 1.1）	9.3 months
Disease Control Rate (DCR)	**79.5%**
12 months OS%	78.1%
24 months OS%	**58.0%**

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.

- Median follow-up at data cutoff (28 February 2026) was 28.8 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

Plinabulin Mechanism-Enriched Patients

Dublin-4: "Plinabulin + Docetaxel" vs. "Docetaxel" in 2L/3L non-squamous NSCLC, EGFR/ALK/ROSm/RET Wild-Type, 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. 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

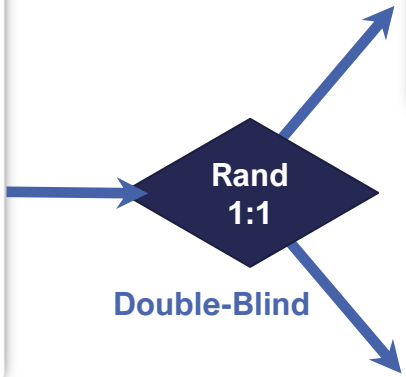
✓ *Dublin-3 (DP):* OS HR 0.55, mOS: 15.8 vs 11.7 months (Plinabulin + docetaxel DP vs docetaxel D), mPFS 5.6 vs. 3.8 months (HR 0.67), ORR 18.2% vs. 8.0%.

✓ *303 Study (DP+PD-1): 24*-month OS rate 58% (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 >= 3 months and platinum doublet.
- ✓ ECOG 0 and 1.



Active Group

Docetaxel (75 mg/m^2) day 1 + Plinabulin (30 mg/m^2) day 1 and 8 of each cycle

Control Group

Docetaxel (75 mg/m^2) day 1 + Plinabulin placebo day 1 and 8 of each cycle

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

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

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

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

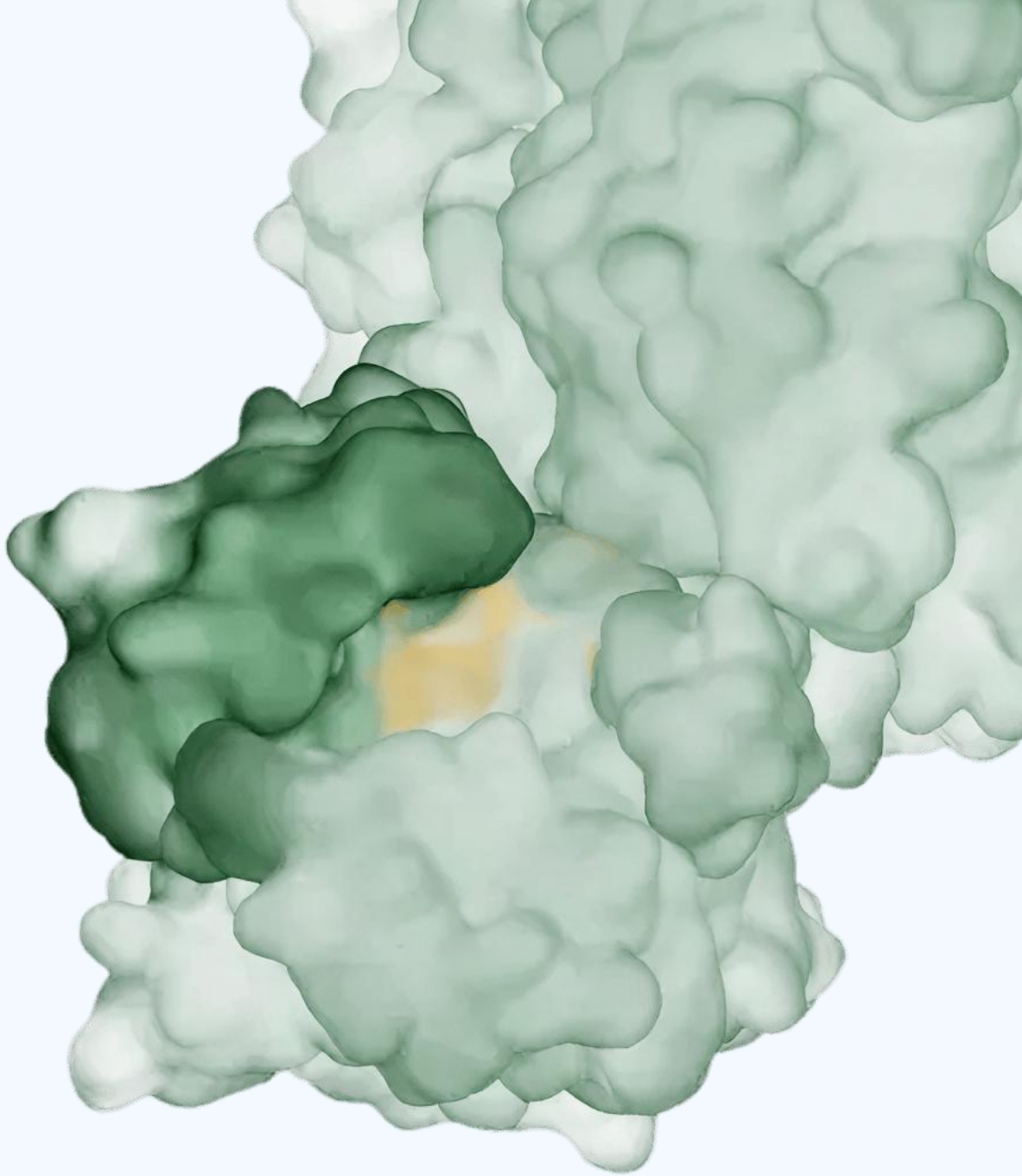
DURABLE

Clinical Benefit

Overall survival and durable response

SEED THERAPEUTICS

Create The Glue
Capture The Protein
Eliminate The Disease



De novo molecular glues for protein targets beyond the reach of traditional therapies

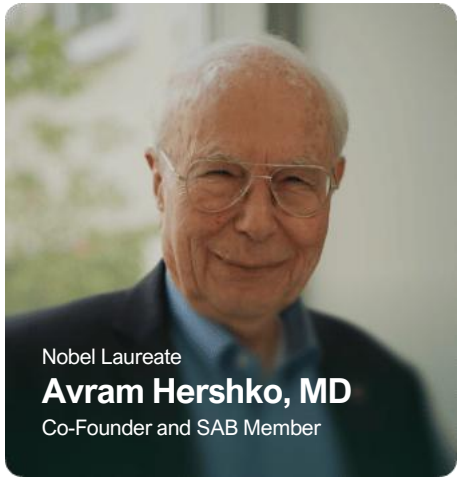
SEED: A Clinically Advancing Molecular Glue Company with Validated Pharma Partnerships and Near-term Data

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

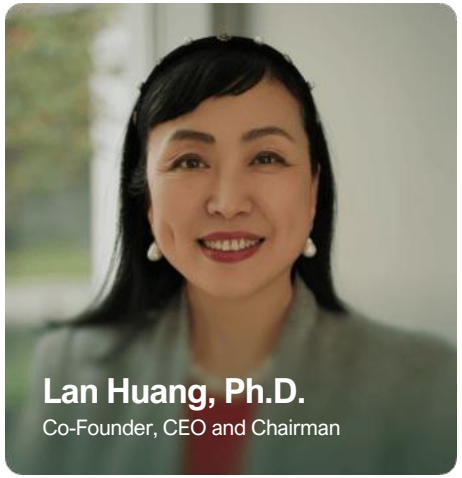
TPD Potential	• Addressing 80% of disease-causing proteins considered “undruggable” by traditional methods
Technology Platform	• Target-centric: Differentiation in using novel E3 ligases among 640 E3s for protein of interest, featured as one of leading TPD companies by two Nature review articles in 2024 • 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.

SEED Is Led By The Scientists Who Discovered Targeted Protein Degradation

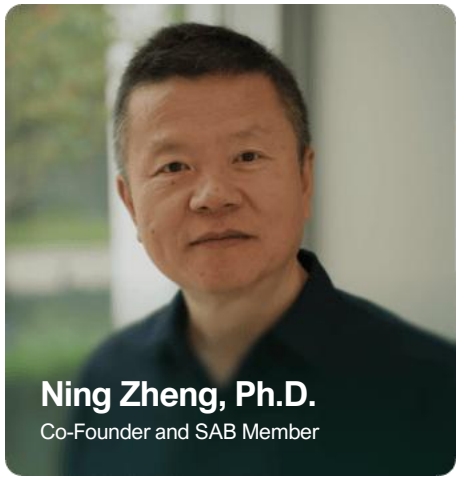
- Combined leadership record of 40 INDs and 12 NDAs
- Balanced by an experienced board across finance, risk, legal, and drug development



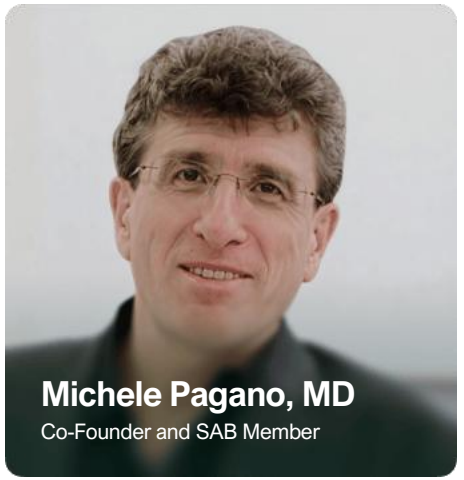
Nobel Laureate who discovered the ubiquitin–proteasome system



Solved the first HECT E3 ligase structure; 20+ years in therapeutic dev.



Solved the first RING E3 ligase and coined the term “molecular glue”

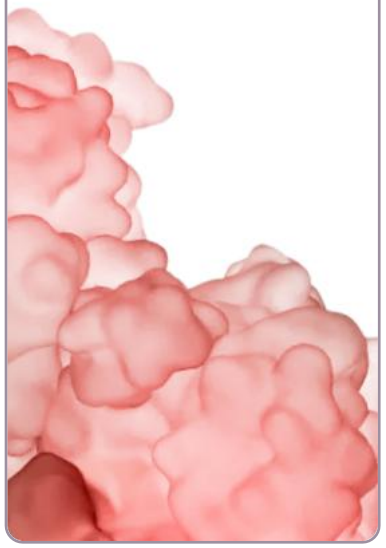


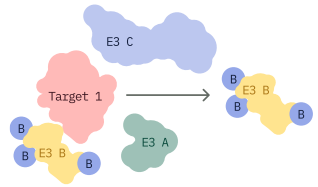
Defined SCF ubiquitin ligase and core cell-cycle ubiquitin biology

SEED Built The TPD 2.0 Engine To Find The Optimal Ligase And Degrade Any Disease-causing Protein

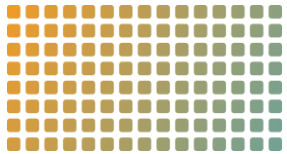
SEED's RITE3™ platform unlocks access to more than 600 ligases encoded in the human genome.
Then designs custom molecular glues to bind them. More ligases. Better matches. Higher hit rates.

Pathogenic Protein

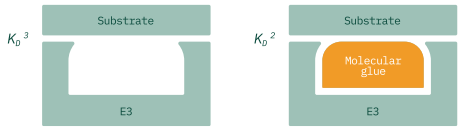




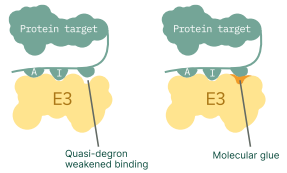
LumID™ Live-cell assays
Functional Degradation



Screen and select across 600+ human ligases



Proteomics
proximity and tissue relevance



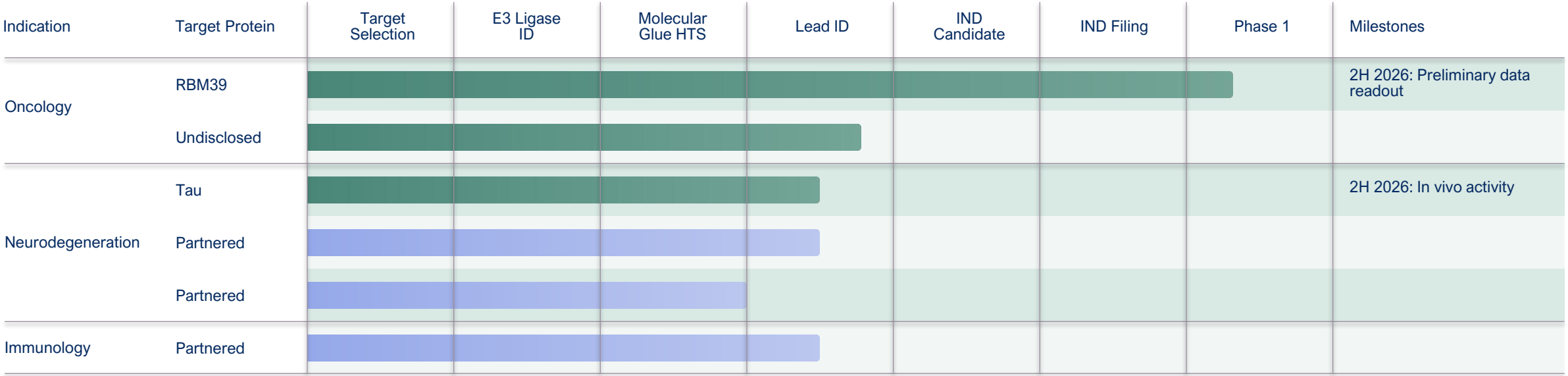
Structural Biology
ligase-target complementarity

Design the Glue
>1% Hit Rate

Eliminate Disease

SEED's Platform Is Already Generating Clinical Assets Alongside World-class Partners

SEED Therapeutics has been validated through deep diligence, long-term capital, and active R&D partnerships with Eli Lilly and Eisai, while building a broad, multi-indication internal pipeline with 6 active programs and 5 novel ligases.



- Research collaboration with Eli Lilly on TPD with multiple targets.
- $10 million upfront, and a $10 million equity investment in Series A-2.
- Eligible to receive up to $780 million in potential milestones, and tiered royalties of sales.

- Series A-3 financing: first close of $24 million from investors led by Eisai in August 2024.
- SEED–Eisai Collaboration: SEED receives upfront and 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

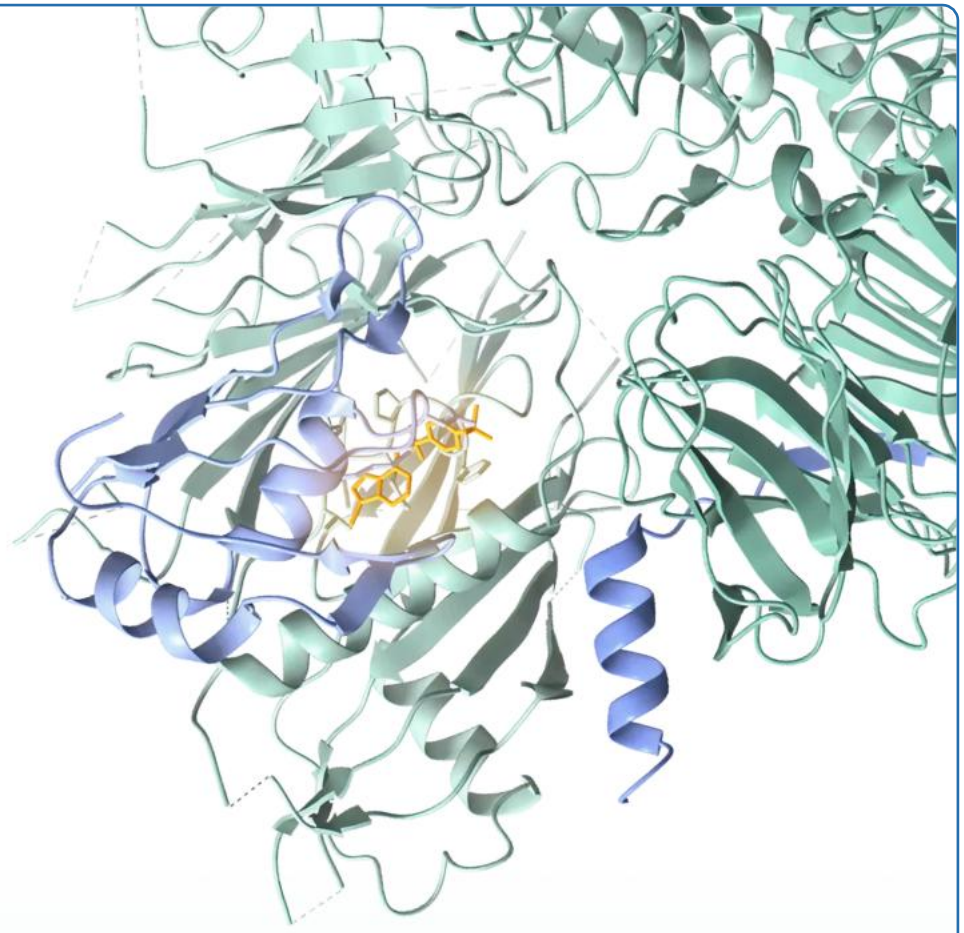
Why it works

- 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



RBM39–DCAF15 complex structurally solved → enables next-gen precision degraders

*Structure of DCAF15-RBM39 complex solved by Prof. Zheng

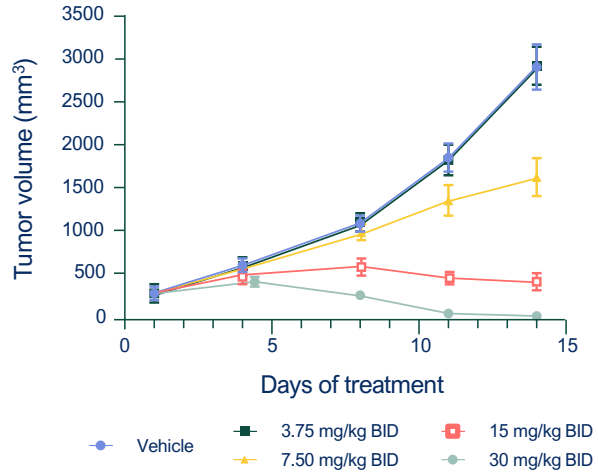
Ewing Sarcoma Is A Fusion-driven Disease with No New Drugs in the Past 30 Years

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)

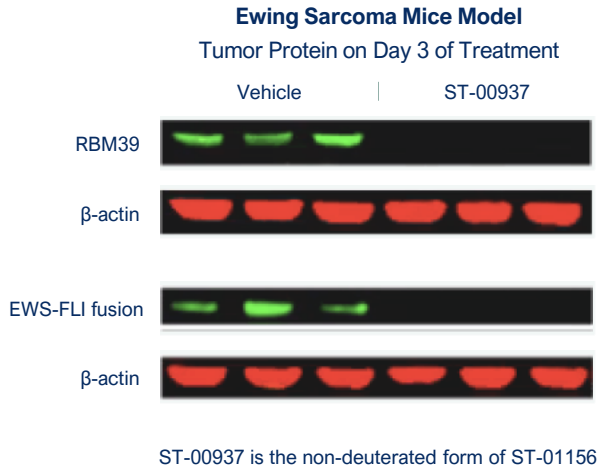
Complete tumor regression in Ewing Sarcoma

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

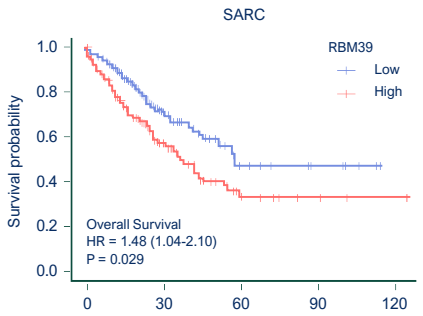


ST-00937 is the non-deuterated form of ST-01156

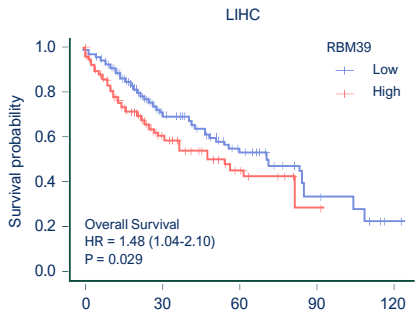
RBM39 Drives Progression For Rare and Large Cancer Indications with >1 million Addressable Patients

Neuroblastoma

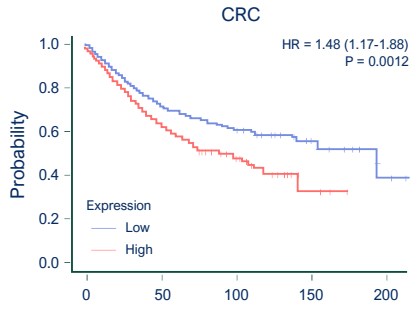
Sarcoma



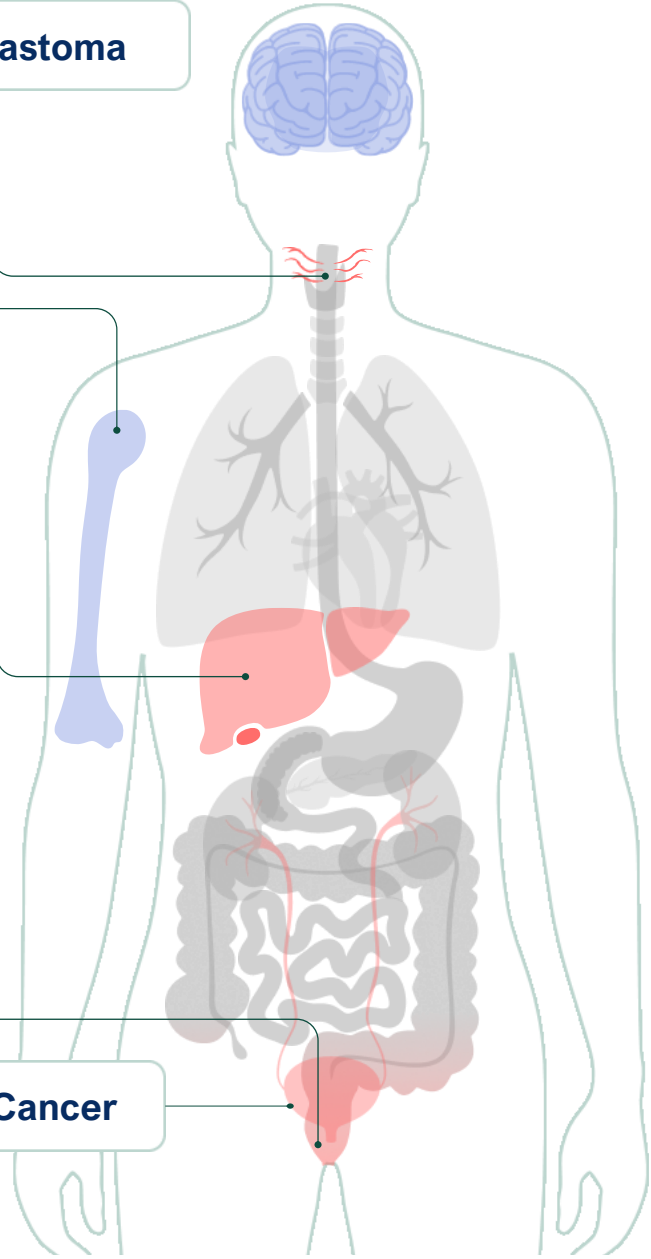
Hepatocellular Carcinoma



Colorectal Cancer



RBM39 overexpression leads to poor patient survival



Prostate Cancer

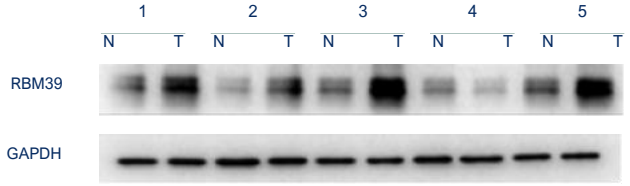
Pediatric tumors High prevalence tumors

J Cell Mol Med. 2022;26:4859–4871.
J Cancer. 2025; 16(7): 2233-2249.

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)

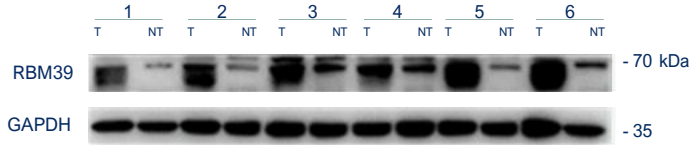


N = No Tumor
T = Tumor

Wang et al., J of Cancer, 2025

Liver Cancer

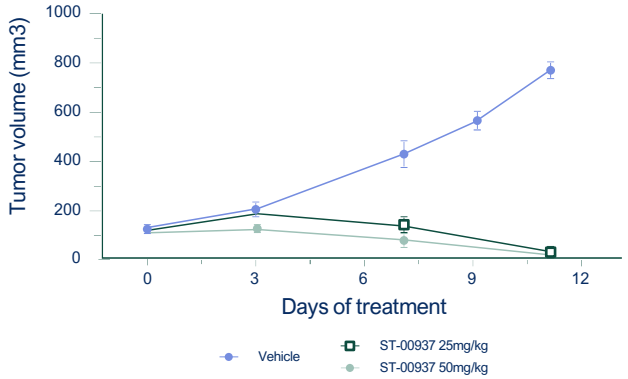
RBM39 high expression in liver cancer (T), not in normal tissue (NT)



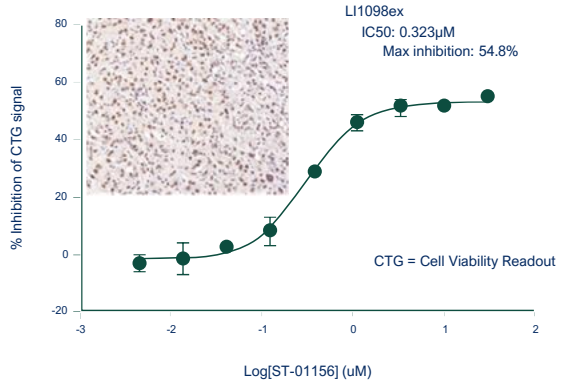
N = No Tumor
T = Tumor

Xia et al., Cell Death Discovery, 2023

Complete tumor regression in colon cancer model



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



Leading Oncologists Guiding Development of ST-01156

Dr. George D. Demetri

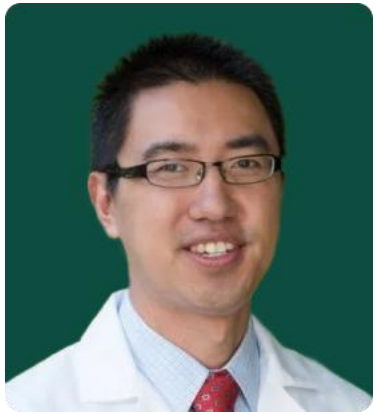
Dana Farber

Associate Professor at Harvard Medical School and Director of the Center for Sarcoma & Bone Oncology at Dana-Farber and Brigham and Women's Hospital. Global leader in sarcoma drug development, including Gleevec for GIST.

Dr. Robert Maki

MSK

Globally recognized sarcoma expert at MSK with four decades of clinical/ research leadership and 100+ publications. Leads adult and pediatric sarcoma programs integrating early-phase trials with translational research.



Dr. Daneng Li

City of Hope

Associate Professor of Medical Oncology at City of Hope and leader of the liver tumors program. Expert in gastrointestinal cancers with active roles in national cooperative groups including SWOG.

Dr. Jordi Rodón

MD Anderson

Medical oncologist at MD Anderson specializing in early-phase drug development and precision oncology. PI on 80+ phase I trials and a leader in inhibitors and key personalized-medicine studies (WINTHER and Basket of Baskets)

Dr. Monica Mita

Hoag Cancer Institute

Medical oncologist at Hoag Family Cancer Institute with deep expertise in early-phase clinical development and breast cancer. Former Cedars-Sinai leader with 100+ phase I trials and extensive experience advancing first-in-class therapies.

ST-01156: Phase 1a Clinical Development Plan for Dose Escalation

Objectives

Safety, tolerability, PK, PD, recommended phase 2 dose (RP2D) and preliminary activity / signal detection

Treatment Arms

Single-arm, open-label

N = 30–50 subjects

3 patients per cohort

Treatment Regimen & Timing

Daily × 5 days and rest for 2 days, with a cycle defined as 28 days

Variable increments (33–100%) based on incidence & severity of adverse events

Multiple ascending doses

Second Dose Cohort Completed

Key Eligibility

Age 18+ all solid cancers

Age 16+ for Ewing sarcoma

Backfilling of lower doses: Priority cancers (Ewing, hepatocellular carcinoma, KRAS mutant cancers, colon cancer, uterine/biliary / DNA repair aberrant cancers)

Primary Endpoints

Safety, tolerability, MTD/MAD, RP2D

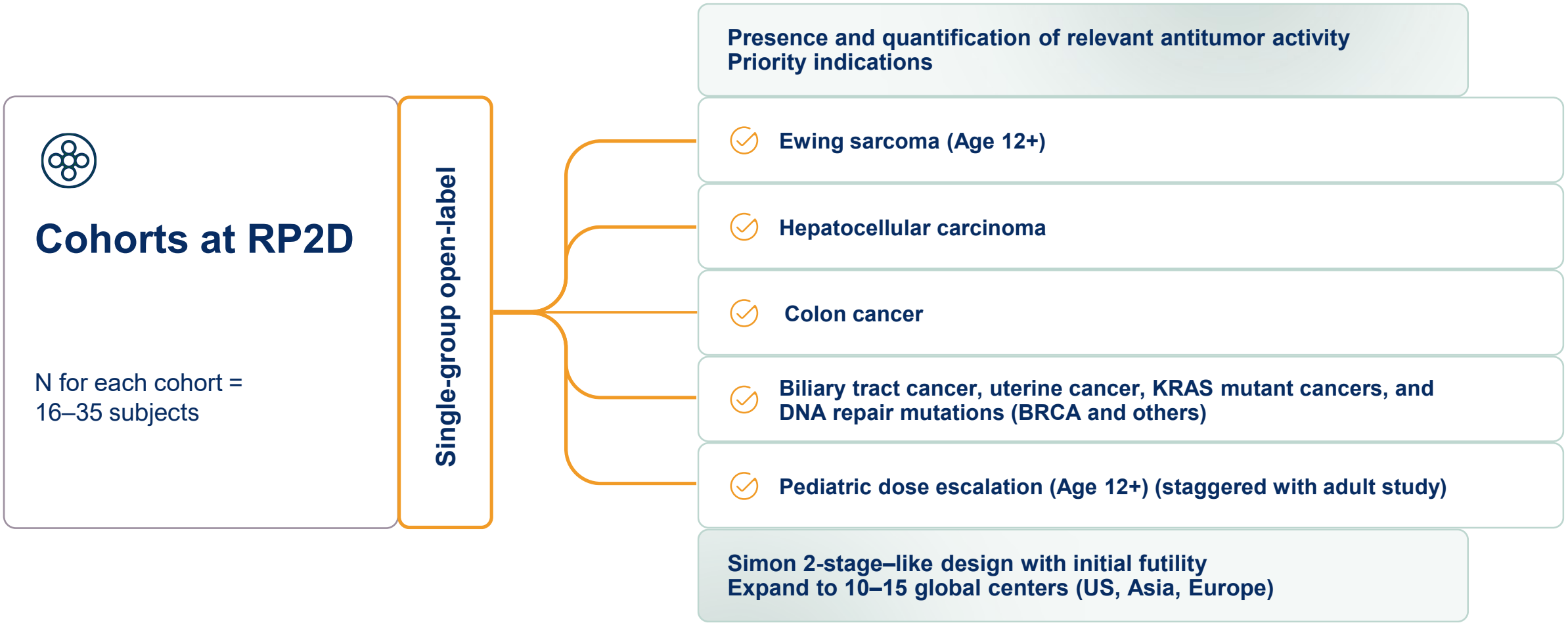
Secondary Endpoints

PK/PD, preliminary efficacy

Sites

6 top-tier U.S. centers, including Dana-Farber, MD Anderson, and MSKCC

ST-01156: Phase 1b Clinical Development Plan for Dose Expansion



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 — Creating 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
- Series B financing underway to fund 2026–28 inflection points

BeyondSpring PHARMACEUTICALS

Thank You

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Media: PR@beyondspringpharma.com

www.beyondspringpharma.com