BeyondSpring PHARMACEUTICALS

Transforming Cancer Care with Immune-modulating Therapies

NASDAQ: BYSI

Aug 2026

Non-Confidential

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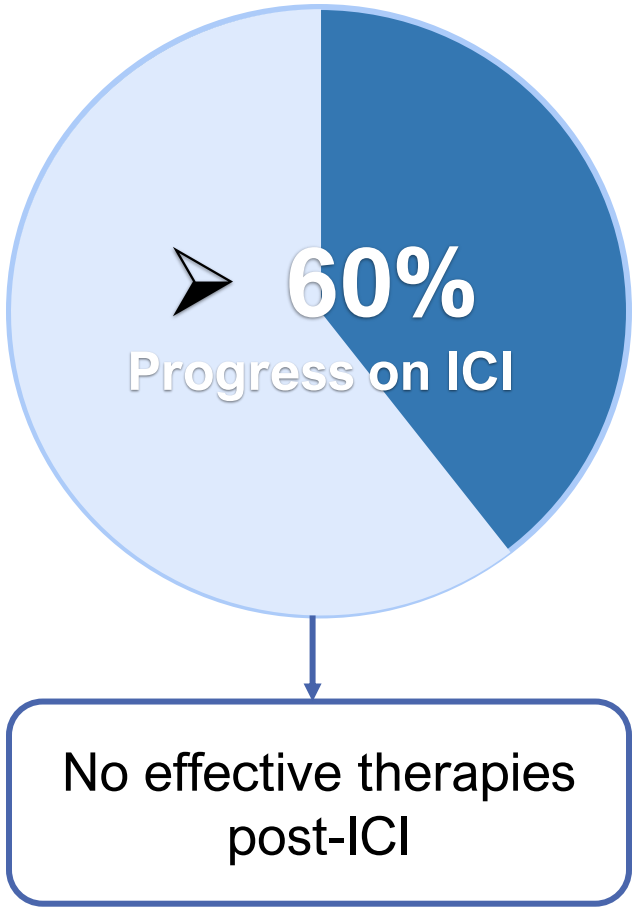
Investment Highlights

- Focused on developing transformative medicine for patients with unmet medical needs
- Plinabulin: Unique immune modulator with durable anti-cancer OS Efficacy and Safety in reducing CIN
- Plinabulin Dublin-4 Confirmatory Phase 3 study: 2/3L non-squamous EGFR WT NSCLC post-ICI
- Equity owner in SEED: Clinical stage company, TPD RITE3 Platform & Robust Pipeline, Eli Lilly & Eisai Investment
- Strong Intellectual Property

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.

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” to ICI 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
- Plinabulin’s DC maturation mechanism could target “acquired resistance” to ICI

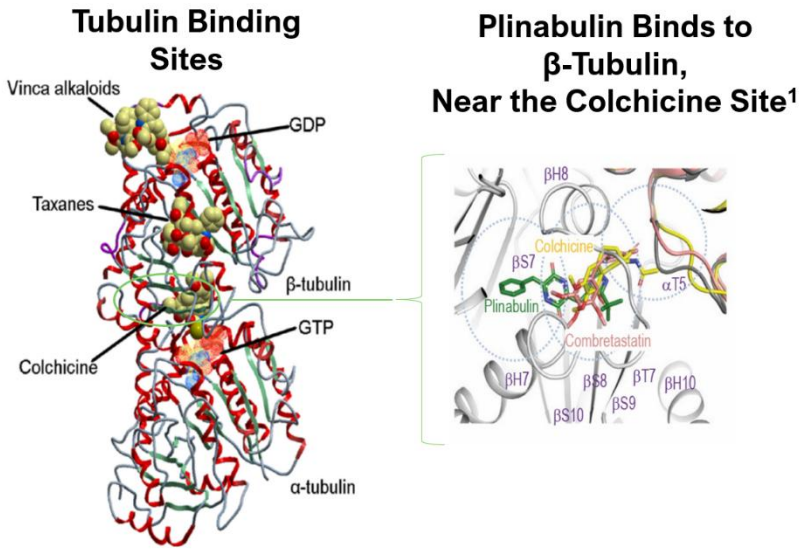
1. Memon et al. Cancer Cell 42, 209–224 (2024).

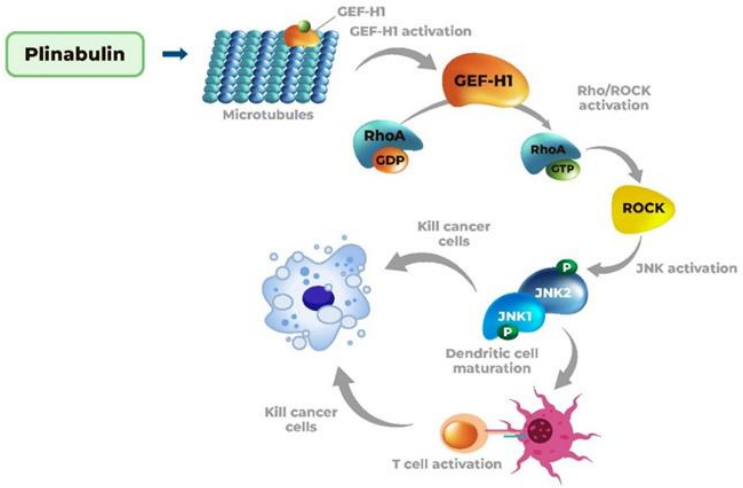
Plinabulin Monohydrate is a Brain-Penetrant, Unique Tubulin Depolymerizing Agent which induces “Dendritic Cell or DC Maturation via GEF-H1 release”

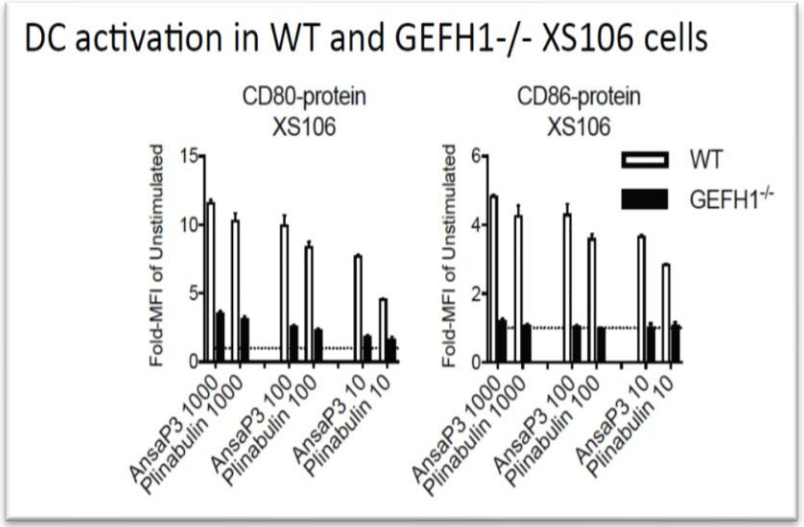
Plinabulin Monohydrate is a unique tubulin binder[1]

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]

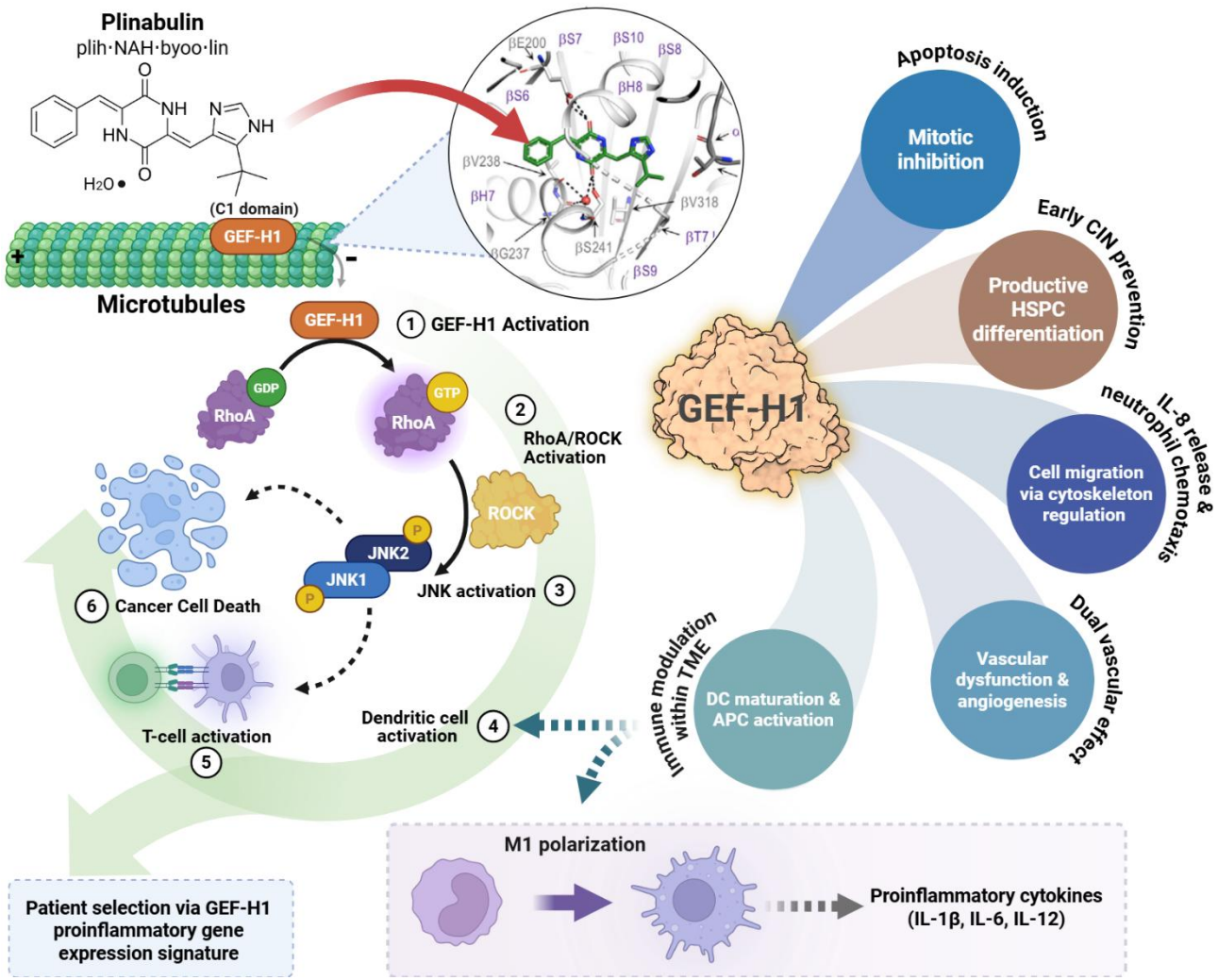






1. La Sala et al., Chem 5(11): 2969-2986 (2019); 2. Kashyap et al., Cell Reports 28(13): 3367-3380 (2019); 3. Choi et al. Cell 189 (2): 461-477 (2026)

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:**

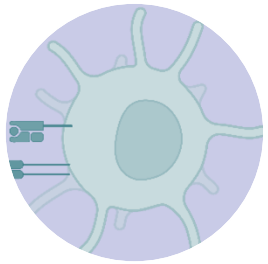
a) GEF-H1 activates RhoA/ROCK signaling pathway and leads to **DC maturation/M1 polarization and T-cell activation**, which has been validated in preclinical and clinical studies.[2-4]

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.

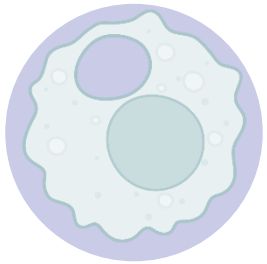
Plinabulin's Immunomodulation and Neutropenia-Mitigating Activities Position it as a Valuable Addition for Combination Regimens



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: Potential "Pipeline in a Drug"

	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 EGFR WT NSCLC:
A Large, Growing and Unmet Clinical Challenge

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

12 Phase 3 Studies with No OS Benefit vs. Docetaxel in Post-ICI EGFR WT NSCLC

Phase 3 study	Patient Population	Study Design	median OS (M)	OS HR	P value
SAPPHIRE	2/3L，non-squamous	Sitravatinib (TKI) + Opdivo (PD-1) vs. docetaxel	12.2 vs. 10.6	Non-squamous HR=0.86	p=0.144
LEAP-008[1]	2L, non-squamous and squamous	Lenvima (TKI) + pembrozulimab (PD-1) vs. docetaxel	11.3 vs. 12.0	HR=0.98 Non-squamous：HR=0.96	p=0.434
CONTACT-01	2L，non-squamous and squamous	Cabozantinib (TKI) + Atezolizumab (PD-L1) vs. docetaxel	10.7 vs. 10.5	HR=0.88	p=0.3668
EVOKE-01	3L，non-squamous and squamous	Sacituzumab govitecan (ADC) vs. docetaxel	11.1 vs. 9.8	HR=0.84	p=0.0534
CARMEN-LC03	2L，non-squamous	Tusamitamab Ravtansine (ADC) vs. docetaxel	12.8 vs. 11.5	Non-squamous HR=0.85	p=0.112
CANOPY-2	2L，non-squamous and squamous	Canakinumab(IL-1b Ab)+docetaxel vs. docetaxel	10.6 vs. 11.3	HR=1.06	p=0.633
TROPION-Lung01	2L，non-squamous and squamous	Dato-DXd (ADC) vs. docetaxel	12.9 vs. 11.8 Non-squamous：14.6 vs. 12.3	HR=0.94 Non-squamous：HR=0.84	p=0.530
PRAGMATICA-LUNG[2]	2L，non-squamous and squamous	Ramuciramab (VEGFR Ab)+pembrozulimab (PD-1) vs. SOC	10.1 vs. 9.3	HR=0.99	p=0.46
COSTAR[3]	2L，non-squamous and squamous	TIM-3+PD-1+docetaxel vs. PD-1+docetaxel vs. docetaxel	11.9 vs. 11.8 vs. 11.3	HR=0.85 (triple vs. D); HR=0.96 (double vs. D)	p=0.08; p=0.37
LATIFY[4]	2L，non-squamous and squamous	Durvalumab (PD-L1) +Ceralasertib (ATR inhibitor) vs. docetaxel	11.1 vs. 10.0	HR=0.90	p=0.287
ABBIL1TY	2L，non-squamous and squamous	Acasunlimab (Biospecific PD-L1x4-1BB)+pembrozulimab (PD-1) vs. docetaxel	No OS benefit vs. Docetaxel	-	-
SigVie-002	2L/3L, non-squamous	Sigvotatug vedotin (ADC) vs. docetaxel	No OS benefit vs. Docetaxel	-	-

1. Malinou J et al., ASCO 2024; 2. Dragnev KH etal. ASCO 2025; 3. ESMO IO and Technology 2025; 4. ELCC 2026

2/3L EGFR WT NSCLC post ICI with severe unmet medical needs with docetaxel remains SOC; Dublin-3 study demonstrated OS benefit vs. docetaxel

Docetaxel + Plinabulin vs. Docetaxel + Placebo in Patients with EGFR WT 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

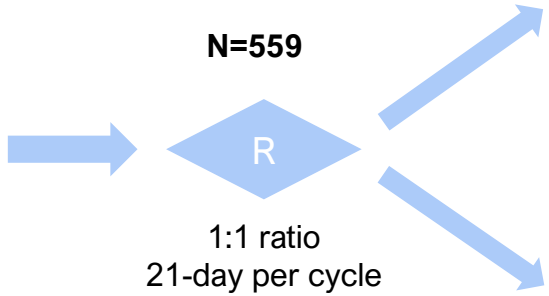
Overall survival (OS)

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-based Chemo
- Must have at least one measurable lung lesion
- Prior checkpoint inhibitor therapy allowed



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

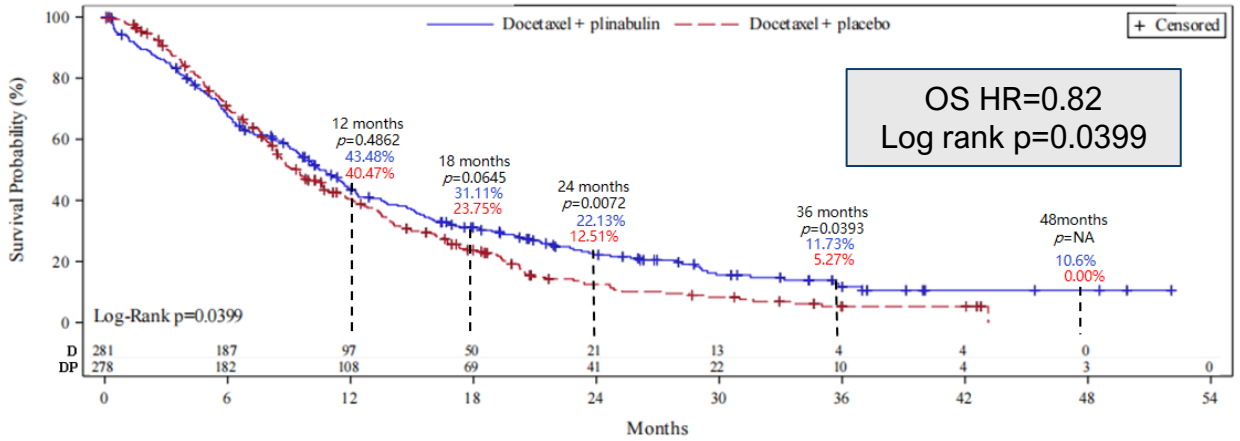
D:
Docetaxel
(75 mg/m2, day 1)
+ Placebo (day 1, 8)

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



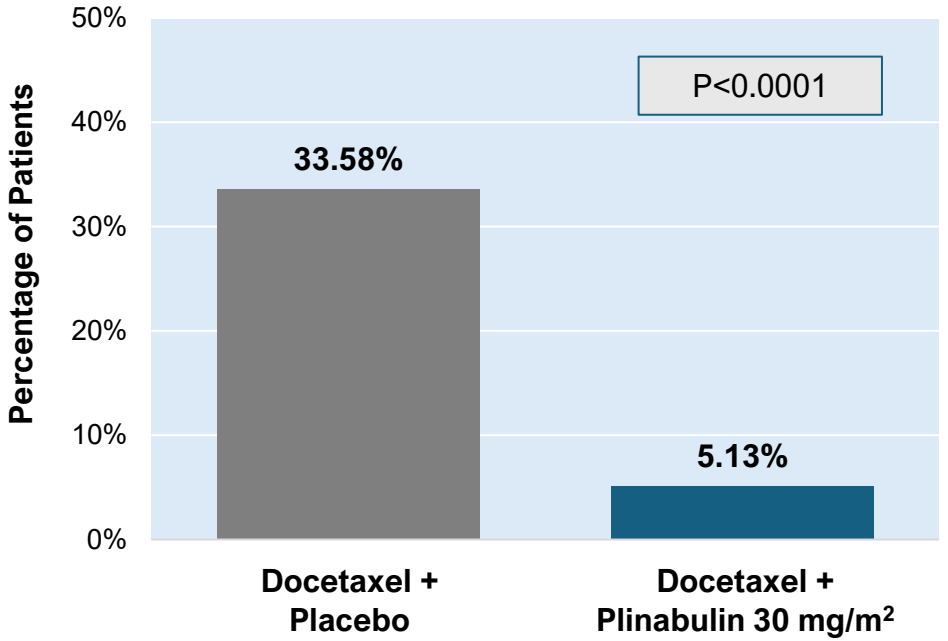
Treatment	Subjects	Event	Censored	Median (95% CI)	HR (95% CI)
Docetaxel + placebo	281	230 (81.9%)	51 (18.1%)	9.40 (8.38,10.68)	
Docetaxel + plinabulin	278	214 (77.0%)	64 (23.0%)	10.49 (9.34,11.87)	0.822(0.681,0.991)

	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

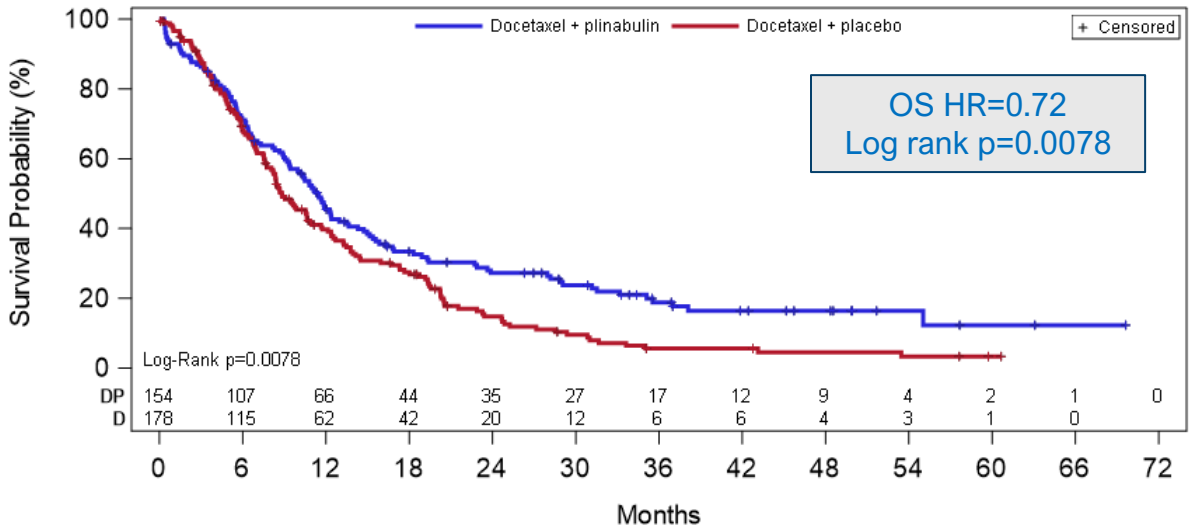
	Grade 3 or 4 Event Rates Adjusted by Exposure		Grade 4 Only Event Rates Adjusted by Exposure	
	DP (n=274)	D (n=278)	DP (n=274)	D (n=278)
Descriptive Statistics				
Number of patients with events, n (%)	203 (74·1)	212 (76·3)	61 (22·3)	121 (43·5)
Total number of years of dose regimen	**83·43**	**71·65**	**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)

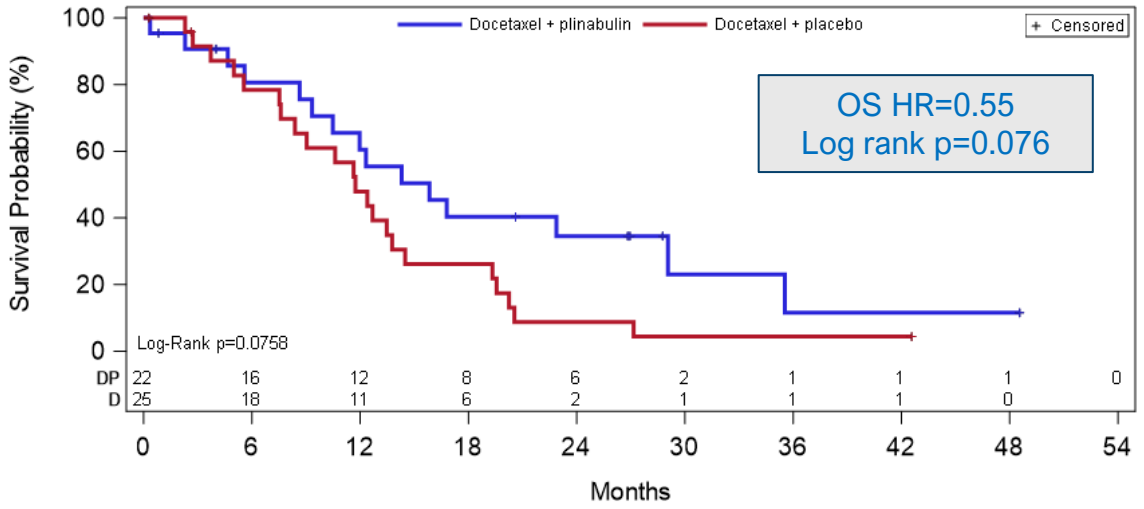
Dublin-3: Post-hoc Analysis in Plinabulin Mechanism-Based Population in 2L/3L Non-squamous EGFR WT NSCLC and Progressed on PD-1/L1 Inhibitors

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)



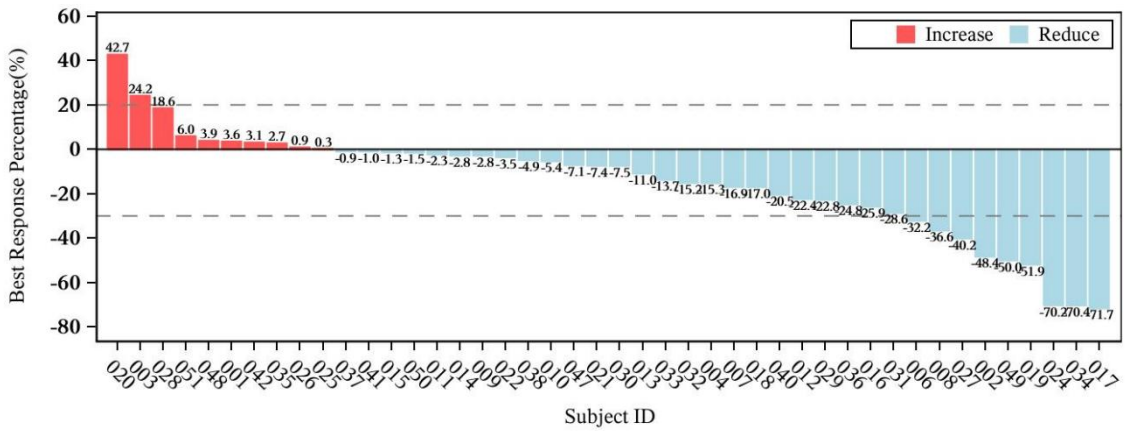
Non-squamous, post-ICI	N	Median OS (95% CI)	HR	Log rank P value
Docetaxel	25	11.7 (7.59, 13.77)		
Plinabulin + Docetaxel	22	15.8 (9.34, 29.06)	**0.55 (0.28, 1.07)**	**P = 0.076**

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

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

Plinabulin + Docetaxel + PD-1 Inhibitor (n=47) ASCO 2026	
Primary endpoint: 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.

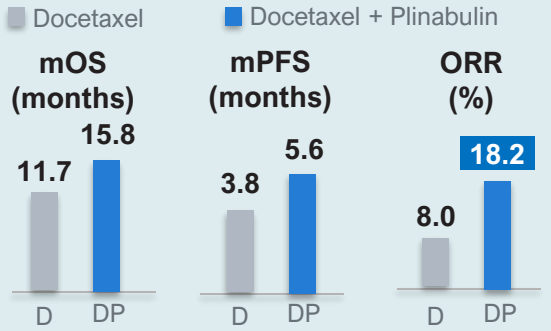
Plinabulin Clinical Efficacy Supports Dublin-4 Patient Selection

Dublin-3 efficacy with the post-ICI subgroup signal and Study 303 efficacy provide the clinical rationale for Dublin-4 for Plinabulin-mechanism targeted population:

- **Non-squamous NSCLC + Progressed on ICI with "acquired resistance"**

Dublin-3 Post-hoc

- Non-squamous, post-ICI EGFR WT NSCLC (DP, n=22, D, n=25)



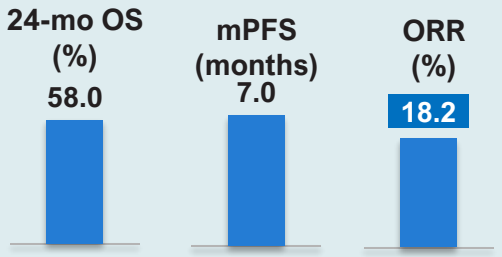
mOS +4.1 months

OS HR 0.55

MoA-enriched population aligned with Dublin-4

Study 303 prospective

- Plinabulin + Docetaxel + PD-1
- post-ICI NSCLC (n=47)



2-year OS rate 58%

DCR 79.5%

Consistent with Dublin-3 Post-hoc

Scientific Rationale for Dublin-4 Patient Selection

✓ **Tumor Vasculature Targeting to select non-squamous patients:**

Avastin, a vasculature targeting agent, only approved in non-squamous NSCLC patients.

✓ **Immune Re-sensitization MoA to select patients with "acquired resistance" to ICI**

"Acquired resistance" from ICI due to T-cell exhaustion / APC pathway mutation[1] which can be targeted by Plinabulin with dendritic-cell (DC) maturation MoA, as DC is the most potent APC.

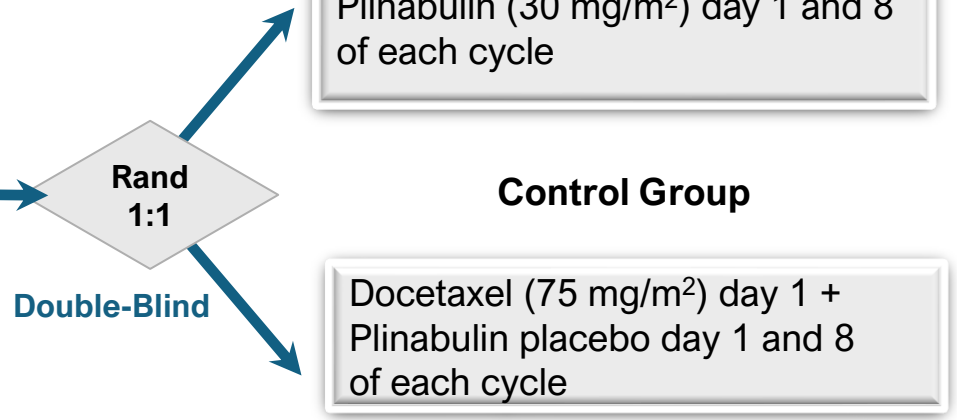
Dublin-3: *Lancet Respir Med 2024*

303 Study: ASCO 2026

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, ROS1, 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.



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),

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

	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

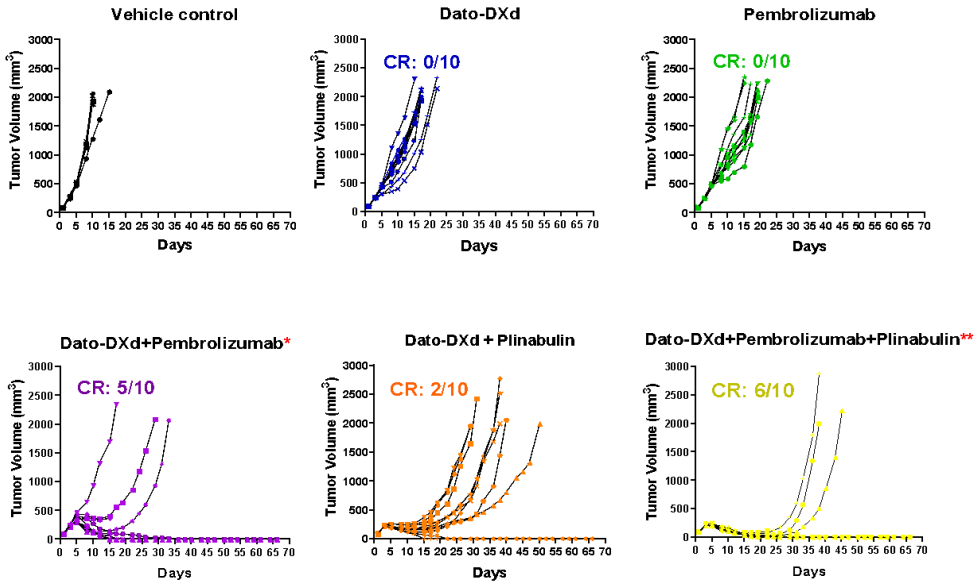
Severity of Unmet Need	Current SOC - Docetaxel	Potential Solution - Plinabulin + Docetaxel
●●●	Lack of durable response post-immunotherapy	Strong immune re-priming and antigen presentation, **durable OS** benefit
●●●	Immune exhaustion, “cold” tumors	**Converts tumors to “hot”**
●●	Limited efficacy with 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

Plinabulin Future Development: ADC Combination, IO Combination

Preclinical ADC Combination Data

Dato-DXd ± PD-1 in hTROP2-MC38 / hPD-1 mice

CR 6/10	Survival ↑	CD8/Treg ↑
Dato-DXd + Pembro + Plinabulin	Sustained survival curve in triple combo	Immune-active TME signal



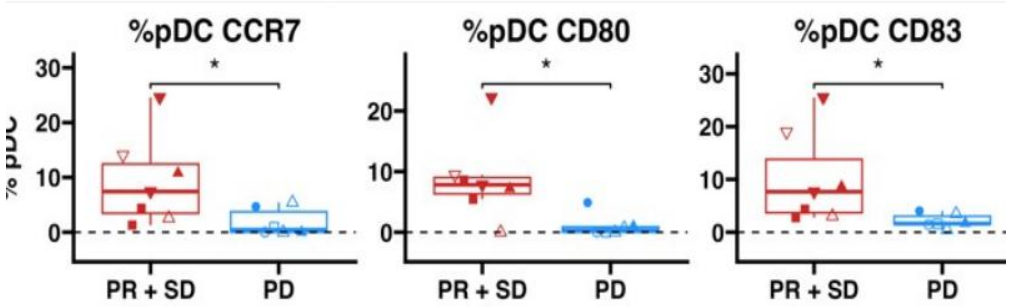
*4/10 animals found dead No animal death **1/10 animals found dead

Human Evidence After ICI Failure

8 cancers failed prior ICI – NSCLC, Head & Neck, Hodgkin lymphoma

DCR 54%	C1D4	DC Mature
Plinabulin combination across ICI-failed cancers	Rapid marker upregulation	CCR7 / CD80 / CD83

pDC maturation markers in responding patients



Responding patients showed rapid upregulation of CCR7, CD80 and CD83, supporting Plinabulin-mediated dendritic-cell maturation and migration.

Sources: *AACR 2026 preclinical ADC combination data*; *Lin et al., Med 6(10):100752 (2025).*

First-in-Class Agent Plinabulin: Potentially Transforming Oncology Treatment 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

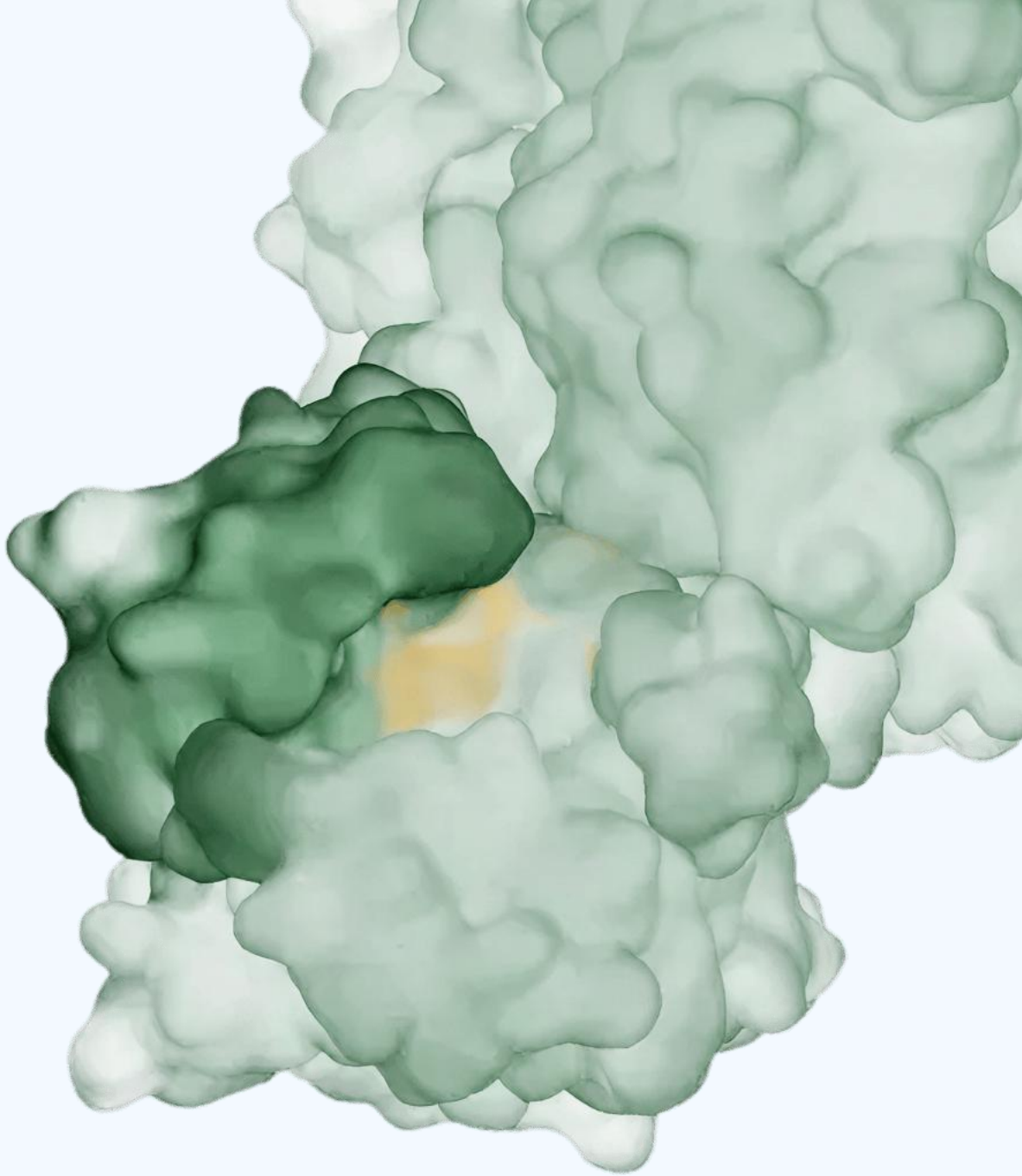
DURABLE

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 Therapeutics – Category-defining Molecular Glue Company

1 Molecular glue field with clinical validation + value recognition

Molecular glue and TPD validation keeps building: Revolution Medicines (~$38B market cap), the Orionis–Novartis collaboration at discovery stage ($40M upfront, up to $1.4B), and the Nurix–Roche transaction for clinical stage BTK degrader ($700M upfront, up to $2.3B).

2 Best-in-class RITE3™ platform for multiple molecular glues

Validated by global pharma collaborations and investments following multi-year diligence. Beyond ST-01156, the RITE3™ platform has already produced additional clinic-bound programs, the wholly-owned Tau degrader and a second oncology program, creating multiple future value inflections.

3 Experienced team with NDA and M&A track record

Founders are pioneers in targeted protein degradation, and the development team has a track record of 40 INDs and 12 NDAs. A deep board and advisory bench add finance, legal, drug-development, M&A, and commercialization expertise.

4 Clinical proof of mechanism in RBM39 target engagement

ST-01156, novel RBM39 degrader: Rapid enrollment with leading US institutions, such as Dana Farber, MSK and MD Anderson. Enrolling the third ST-01156 dose cohort, with target engagement already confirmed below the dose range where anti-tumor activity is expected. Tumor responses are anticipated in cohort 4 and are possible in the current cohort.

5 Efficient path to speedy approval + commercial upside

ST-01156's Ewing sarcoma or neuroblastoma offers an efficient route to human proof of concept and a potential Priority Review Voucher, while colorectal, hepatocellular, and endometrial cancers represent large long-term markets.

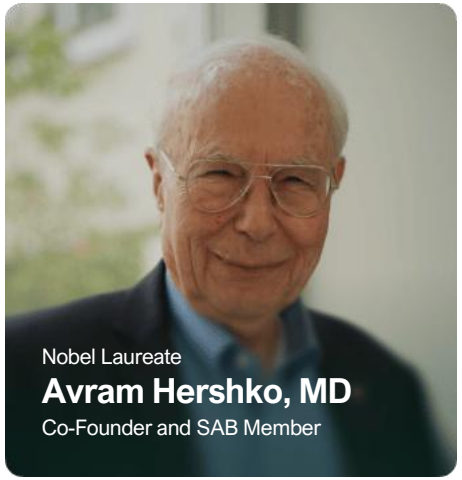
The model echoes Parabilis' record $670M biotech IPO: a rare-disease beachhead with parallel large-indication development.

6 Time and cost-efficient global clinical development

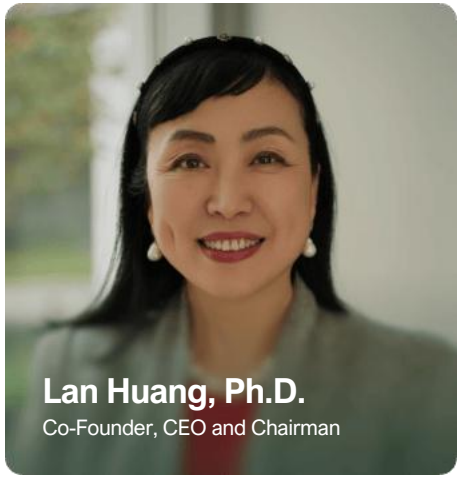
Global clinical synergies across the U.S. and China to accelerate development with cost efficiency.

SEED Was Co-founded By Pioneers in Targeted Protein Degradation and Eli Lilly

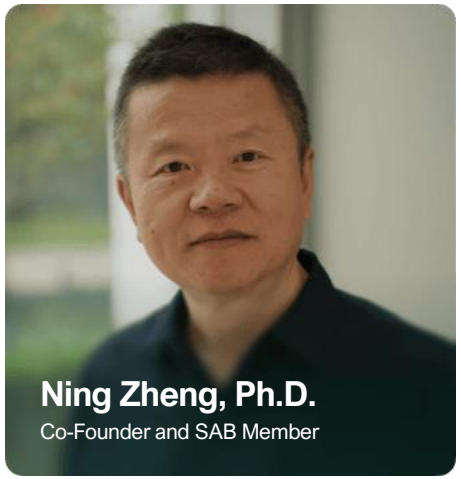
- Scientific capability to use >600 E3 ligases, which consists only 2 structural classes of HECT and RING, solved first by co-founders.
  - Novel E3s or RITE3 use can increase tissue selectivity and scale to reach full therapeutic potential of molecular glues.
- Combined leadership record of 40 INDs and 12 NDAs
- Balanced by an experienced board across finance, risk, legal, and drug development



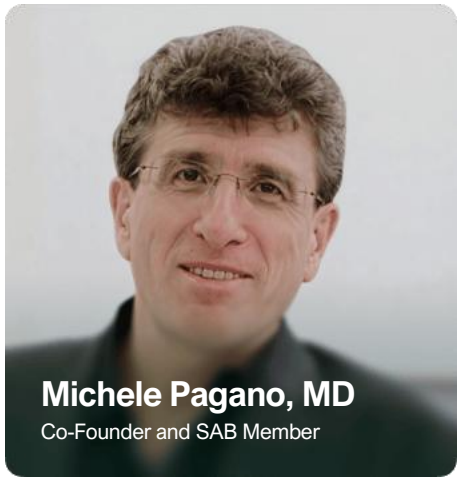
Nobel Laureate who discovered the ubiquitin–proteasome system



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



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



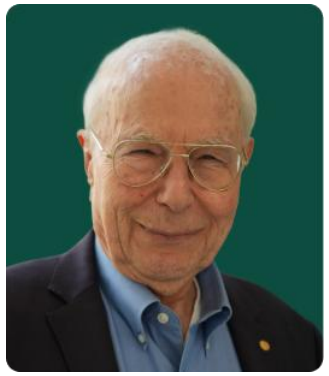
Defined SCF ubiquitin ligase and core cell-cycle ubiquitin biology

SAB with Deep Expertise in Science, Clinical, AI and Business

George Demetri, MD

SAB Chair, clinician with track record for NDA

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, and co-founder of IDRX, which was acquired by GSK with $1B upfront.



Avram Hershko MD

Co-Founder and SAB Member

"Godfather" of TPD; 2004 Nobel Laureate; Distinguished Professor at the Technion; Advisor to Millennium on developing Velcade; Co-discoverer of ubiquitin-mediated protein degradation.

Ning Zheng, PhD

Co-Founder and SAB Member

Howard Hughes Medical Institute investigator; Professor of Pharmacology at the **University of Washington** School of Medicine. Pioneer in RING Finger E3 structure and coin the phrase of "molecular glue" in 2007.

Michele Pagano, MD

Co-Founder and SAB Member

Howard Hughes Medical Institute investigator, **Chair of Biochemistry at NYU Medical School;** Global thought leader on TPD biology, ubiquitin-mediated proteolysis, E3 ligase regulation, cell-cycle control, and cancer therapeutic applications.

Mansuo Shannon, PhD

SAB Member, Neuro-expert

Seasoned pharmaceutical executive and drug hunter with 18+ years at Bayer, Eli Lilly, Roche/Chugai, and Merck; **AskBio Chief Scientific Office** with a track record of **developing CNS drugs**, including Lilly's approved Alzheimer disease drug.

Alan Roemer, MBA, MPH

AB Member, Business

Entrepreneurial life sciences executive and board leader with a record launching multibillion-dollar biotechs, **enabling nine drug approvals, raising ~$2B**, leading five IPOs, and holding senior finance, operations, and corporate development roles across biopharma companies.

Pranam Chatterjee, PhD

Assistant Professor at the **University of Pennsylvania and AI-driven molecular design** pioneer developing generative models for functional biologics; Biotech co-founder translating computational design into fertility solutions, RNA medicines, and cancer therapeutics.

Highly Experienced Board of Directors

Lan Huang, PhD
Co-Founder, CEO and Chairman

Pioneer in E3 structure; Serial biotech entrepreneur with 20+ years of drug development experience, including NDA-ready assets

James Tonra, PhD
President, CSO and Director

20+ years of drug discovery experience that led to 5 NDAs; Ex leadership role in Regeneron, Millennium, ImClone, Kadmon, and BYSI

Linus Lin, PhD
Director

AVP of Molecular Discovery Capabilities at Lilly Global; Ex leadership role in Lilly Chorus, Lilly China R&D Center, WuXi AppTec, and Merck

Yoshiharu Mizui, PhD
Director

Founder and President of Eisai Innovations, Inc.; former Global Business Development and Strategy Head in Eisai's Oncology Business Group

Jackson Tai
Director

Retired board member for Lilly, HSBC Holdings, Mastercard; Former DBS Bank CEO, former J.P. Morgan & Co. investment banker; Expert in finance and risk

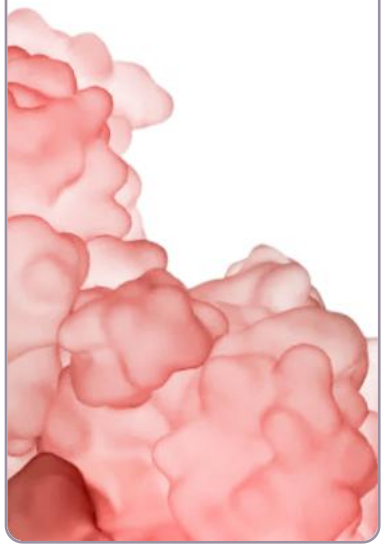
Ko-Yung Tung, JD
Director

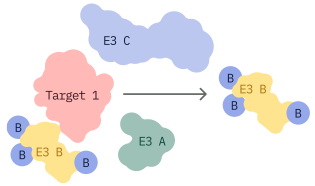
Former Eisai director, World Bank General Counsel, and lecturer at Harvard and Yale Law School; Expert in law and international business

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

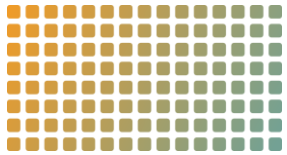
SEED's RITE3™ platform unlocks access to more than 600 ligases encoded in the human genome.
Then designs custom molecular glues to bind them. More ligases. Better matches. Higher hit rates.

Pathogenic Protein

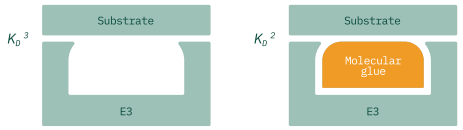




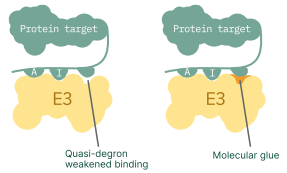
LumID™ Live-cell assays
Functional Degradation



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 Internal Pipeline Spans Three Therapeutic Areas

SEED Therapeutics is advancing 5 wholly-owned programs in oncology, neurodegeneration, and immunology, powered by 4 novel E3 ligases discovered on our proprietary platform.

ONCOLOGY

RBM39

Current: Phase 1

2H 2026: Preliminary data readout

ONCOLOGY

Undisclosed

Current: Lead ID

NEURODEGENERATION

Tau

Current: Lead ID

1H 2027: In vivo activity

NEURODEGENERATION

Undisclosed

Current: Lead ID

IMMUNOLOGY

Undisclosed

Current: Lead ID

5

novel E3 ligases discovered on our platform

5

wholly-owned internal programs advancing

2027

target for our 2nd IND candidate nomination

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

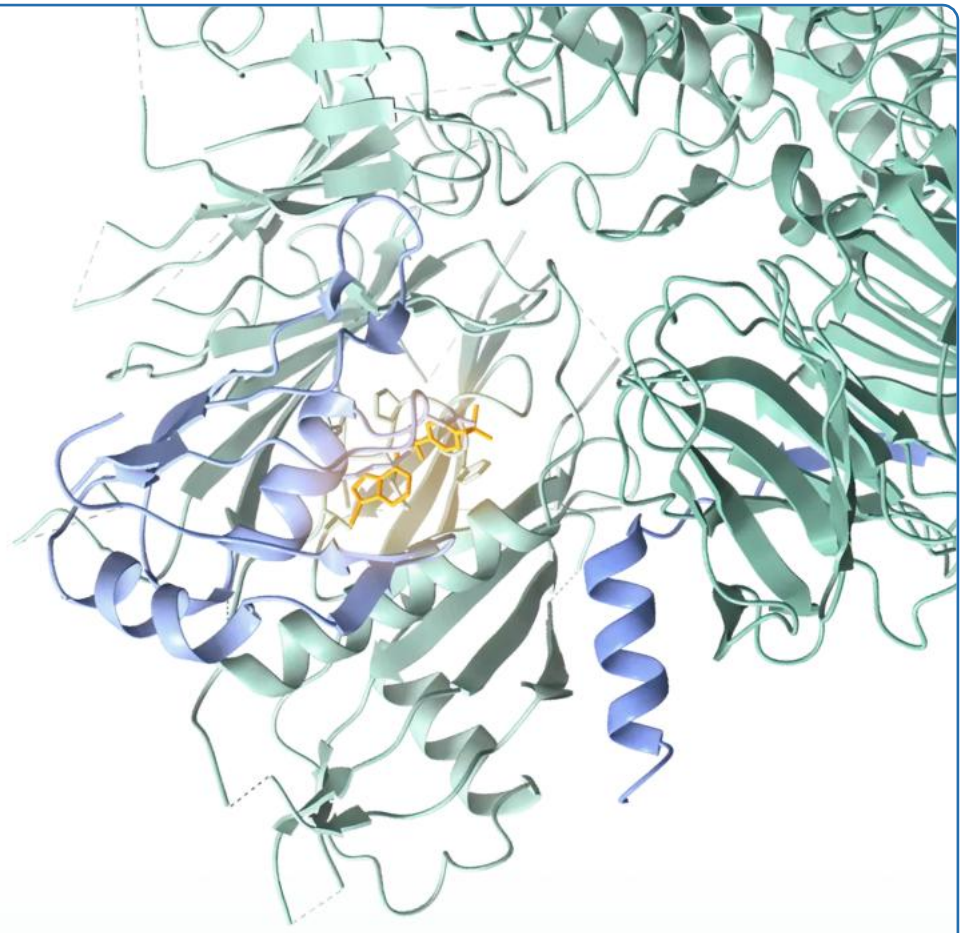
Why it works

- 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

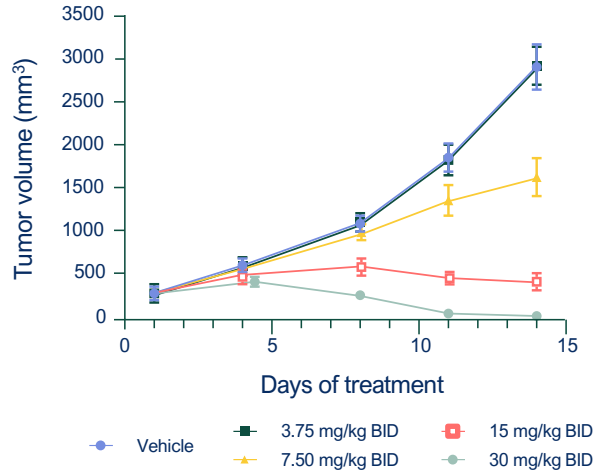
ST-01156: Total Tumor Regression In Vivo. FDA Orphan + Rare Pediatric Disease Designation

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)

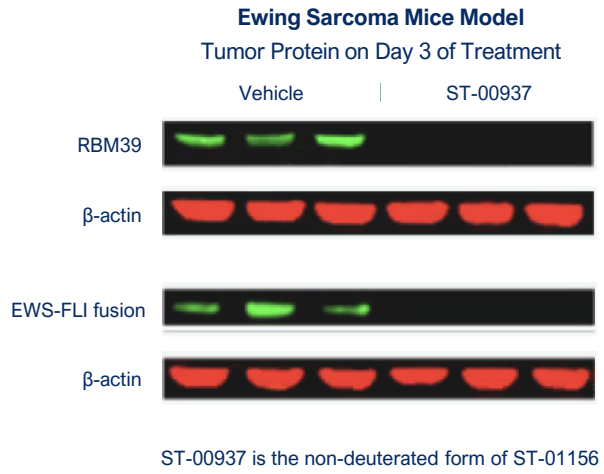
Complete tumor regression in Ewing Sarcoma

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

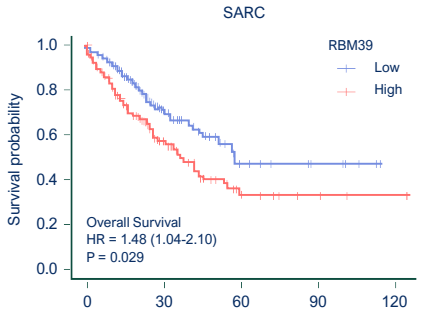


ST-00937 is the non-deuterated form of ST-01156

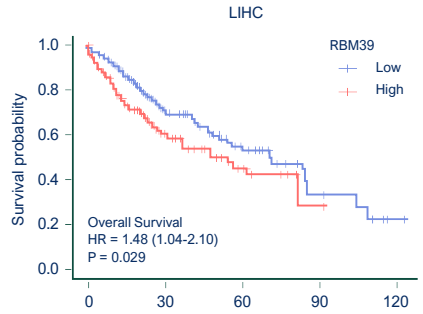
RBM39 Drives Progression For Rare and Large Cancer Indications with >1 Million Addressable Patients

Neuroblastoma

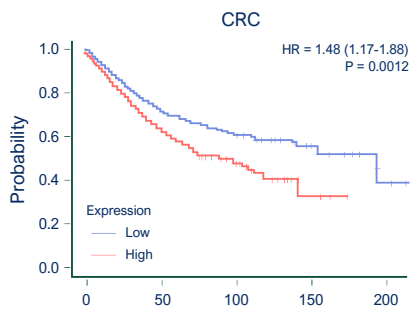
Sarcoma



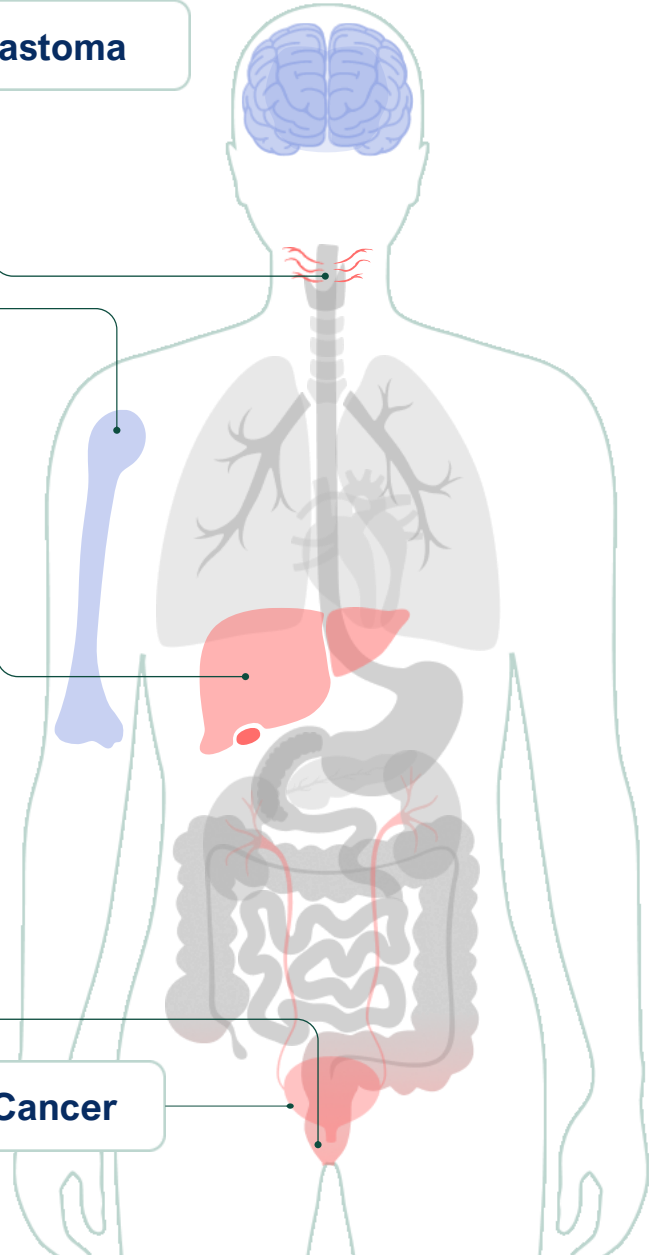
Hepatocellular Carcinoma



Colorectal Cancer



RBM39 overexpression leads to poor patient survival



Prostate Cancer

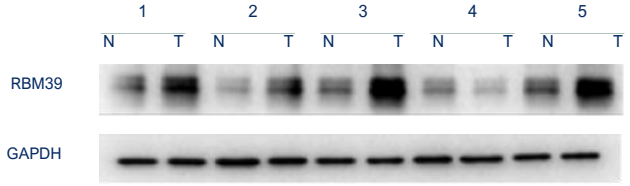
Pediatric tumors High prevalence tumors

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

Ewing Sarcoma Proves The Mechanism.
Liver Cancer And Colon Cancer — More Than 700,000 Patients — Are The Market.

Colon Cancer

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

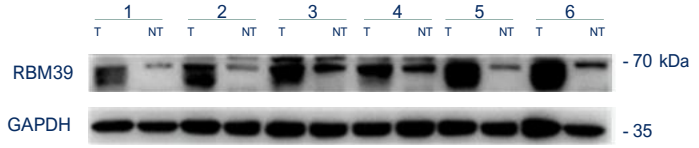


N = No Tumor
T = Tumor

Wang et al., J of Cancer, 2025

Liver Cancer

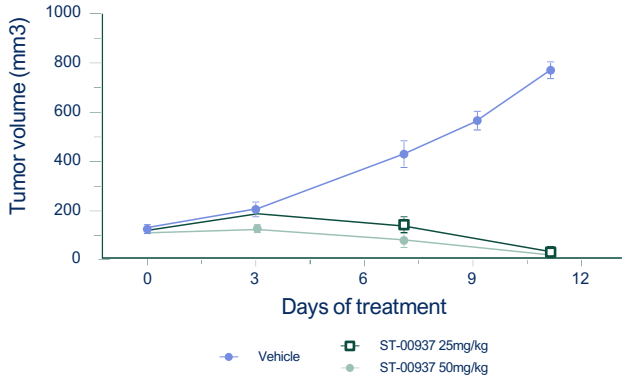
RBM39 high expression in liver cancer (T), not in normal tissue (NT)



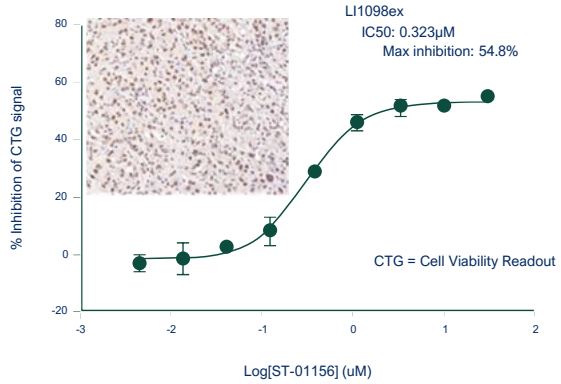
N = No Tumor
T = Tumor

Xia et al., Cell Death Discovery, 2023

Complete tumor regression in colon cancer model



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



Quality And Efficient Execution of Phase 1a Clinical Study

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

Third Cohort Dosed

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

Encouraging First-in-human Safety, Pharmacokinetics, And Proof of Mechanism

ST-01156 CLINICAL PROGRAM

Oral, CNS-penetrant RBM39 molecular glue degrader • Phase 1 in advanced solid tumors • Dose escalation ongoing

Safety & Tolerability

No DLTs

OBSERVED TO DATE

- Treatment-related adverse events low grade to date
- No clinically significant cardiac (QT/QTc) or laboratory findings
- Dose escalation continuing across ascending cohorts

Pharmacokinetics

On Track

TOWARD EFFICACIOUS EXPOSURE

- Rapid absorption across subjects
- Exposure increases with continued dose escalation
- Profile supports advancement toward projected efficacious exposure

Target Engagement

RBM39

DEGRADED IN PATIENTS

- First-in-human proof of mechanism for the molecular glue platform
- RBM39 degradation confirmed in patient blood cells (PBMCs)
- Rapid onset with a reversible, on-target degradation

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

Novartis Sticks With Orionis in Second Deal: $40 M upfront with total value up to $1.4 B

Pharmaceutical Technology

Gilead eyes Kymera's 'adhesive' cancer drug in up to $750 M deal and $85 M upfront

BioSpace

Nurix and Roche deal: $700 M upfront for Nurix's BTK degrader with total value up to $2.3 B

BeyondSpring PHARMACEUTICALS

Thank You

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Media: PR@beyondspringpharma.com

www.beyondspringpharma.com