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BeyondSpring

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Investment Highlights

Plinabulin: Safety & Efficacy	Lead Asset Plinabulin: >700 cancer patients treated with good tolerability; Positive Phase 3 data in 2 indications
Plinabulin Potential	Plinabulin: Dual Benefit in anti-cancer and prevention of CIN; Potential in re-sensitizing in PD-1/PD-L1 progressed patients in multiple cancers
SEED: Novel TPD Platform & Pipeline	SEED: 8 Disclosed Pipeline Assets with 1 expected to enter First Human Dose in 2025
Premier Partnerships	SEED: Investment and R&D Collaboration from Eli Lilly
Intellectual Property	Strong Intellectual Property and Technology Protection

Pipeline

	Indication/Target	Program	Preclinical	Phase 1	Phase 2	Phase 3	Registration	Trial Name / Collaborator
Late stage	**NSCLC (2nd/3rd line)**	Plinabulin + Docetaxel						DUBLIN-3
Late stage	**CIN Prevention**	Plinabulin + Pegfilgrastim						PROTECTIVE-1 & PROTECTIVE-2
Investigator Initiated Trials	**SCLC (2nd/3rd line)**	Plinabulin + Nivolumab + Ipilimumab						Rutgers
Investigator Initiated Trials	**NSCLC (2nd/3rd line PD-1 failed)**	Plinabulin + Pembrolizumab + Docetaxel						Peking Union Medical College Hospital
Investigator Initiated Trials	**ES-SCLC (1st line)**	Plinabulin + Pembrolizumab + Etoposide / Platinum						Wuhan Union Hospital of China
Investigator Initiated Trials	**Multiple cancers (PD-1/PD-L1 failed)**	Plinabulin + PD-1/PD-L1 + Radiation						The University of Texas MD Anderson Cancer Center
Investigator Initiated Trials	**AHCT (hematopoietic stem cell transplantation) in Multiple myeloma**	Plinabulin + Pegfilgrastim						Memorial Sloan Kettering Cancer Center
Early Stage	**Preclinical assets**	BPI-002, BPI-003, BPI-004						
SEED	**8 Targets in Oncology, Neurodegeneration, Immunology and Antiviral**	Targeted Protein Degradation Molecular Glue Platform						SEED THERAPEUTICS, Lilly

BeyondSpring Subsidiaries: 1) Dalian Wanchunbulin Pharmaceuticals Ltd., which owns Greater China rights to Plinabulin, and 2) Seed Therapeutics, a target protein degradation company.

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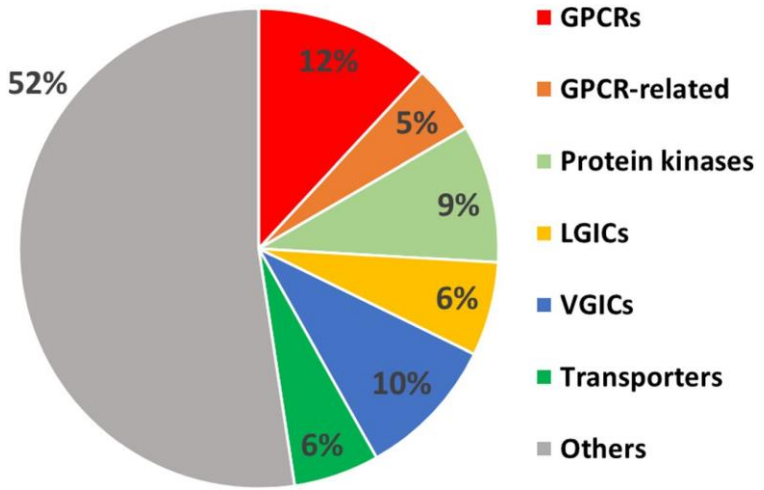
SEED Therapeutics: Target Protein Degradation (TPD 2.0) Company

TPD Targets 80% of Disease-Causing Proteins That are Currently Undruggable

Targeted Protein Degradation (TPD) Addresses 80% of Disease-Causing Proteins That are were Undruggable

TPD for Undruggable Proteins

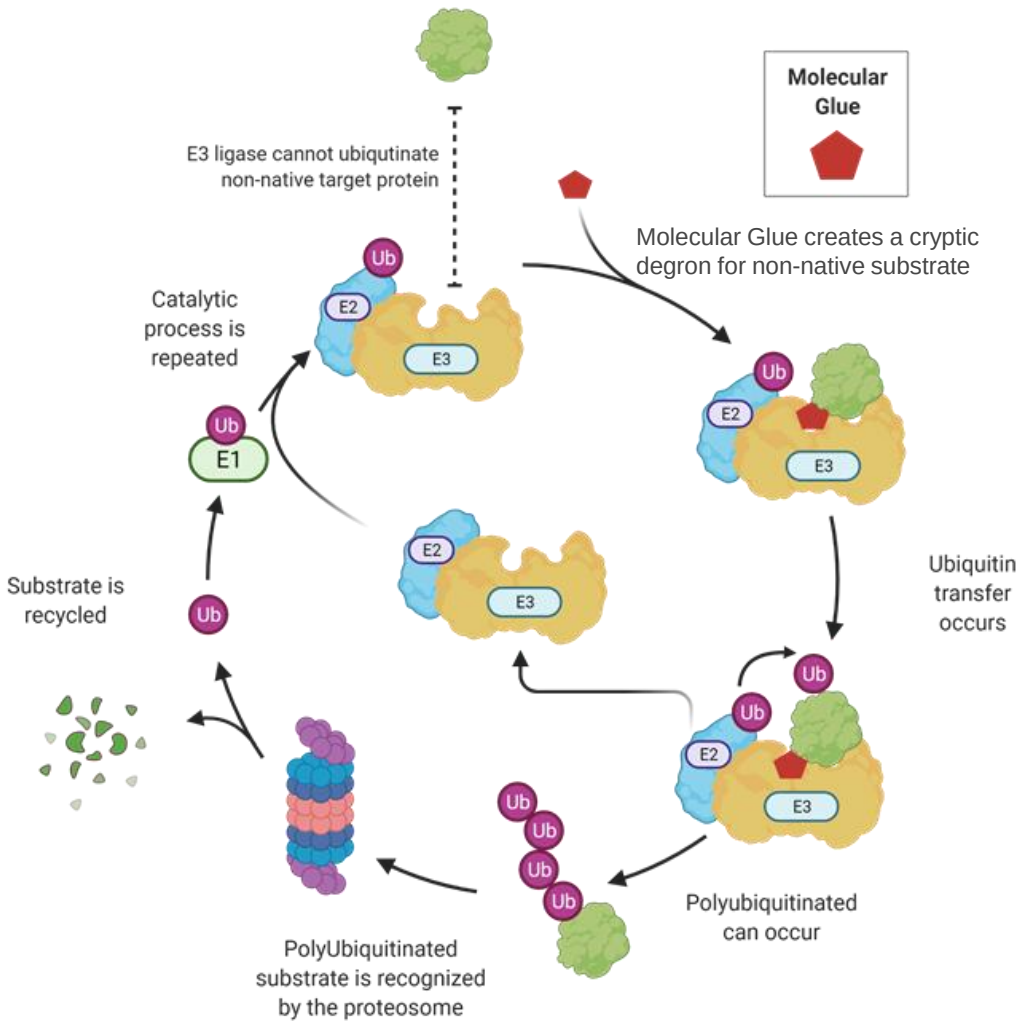
Druggable Proteins



Sriram et al., Molecular Pharmacology, 2018

TPD Development History and Recent Renaissance

TPD Process

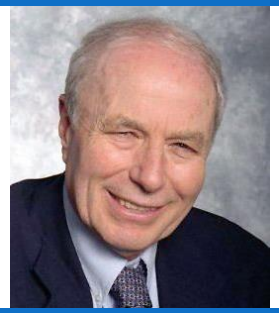


SEED Co-founders played pivotal roles in the advancement of TPD field

- 1996: **Dr. Michele Pagano (SEED co-founder)** discovered cell cycle regulation by TPD, including E3 ligases; published in *Science*
- 1999: **Dr. Lan Huang (SEED co-founder and CEO)** solved the 1st of the two E3 structures (HECT domain E3); published in *Science*
- 2002: **Dr. Ning Zheng (SEED co-founder)** solved the 2nd of the two E3 structure (Ring-finger E3); published in *Nature*
- 2003: US FDA approved **Velcade**, the first proteasome inhibitor for multiple myeloma. **Dr. Avram Hershko (SEED co-founder)** advised on Velcade development. Other companies started to develop new E3 inhibitors with no success
- 2004: **Dr. Avram Hershko won Nobel Prize** for his pioneering work in discovering all essential enzymes for TPD, including E1, E2, E3, and proteasome
- 2007: **Dr. Ning Zheng coined the term "Molecular Glue (MG)"** after solving TIR1 E3 structure and discovering the true function of Auxin, a plant hormone and the **first natural MG to be identified**; published in *Nature*
- 2010-2014: Revolutionary discovery of the mechanism of action of **Revlimid** (for treating multiple myeloma, had peak global annual sale of $12.8b), a derivative of thalidomide, is in fact a MG, that binds to Cereblon (a E3) to degrade Ikaros (a mutated POI). This discovery, published in *Nature,* ushered in the **renaissance of TPD drug discovery**.

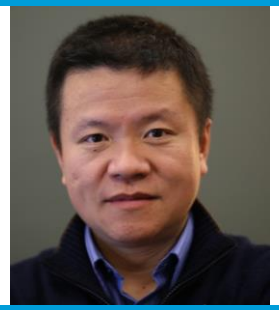
World Class Leadership Team and Exceptional Insights in TPD Drug Development

Avram Hershko MD, PhD+



"Godfather" of TPD;
2004 Nobel Laureate;
Advisor to Millennium on developing **Velcade**

Ning Zheng, PhD+



Howard Hughes Professor, University of Washington; World's foremost **thought leader on E3 and MG**

Michele Pagano, MD +

Howard Hughes Professor, NYU Medical School;
Global **thought leader on TPD biology and application**

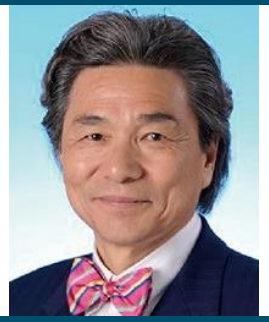
Lan Huang, PhD + *
(Chairman & CEO)

E3 structural expert; Serial biotech entrepreneur with **20+ years** of drug development experience, including assets that are NDA-ready

James Tonra, PhD*
(President & CSO)

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

Ko-Yung Tung, JD*



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

Linus Lin, PhD*

Global head of Lilly Chorus. Ex GM of Lilly China R&D Center, Head of Chemistry at WuXi AppTec, and led multiple drug discovery teams at Merck

Jackson Tai*

Wuxi Biologics Audit Committee Chair; retired board members for Eli Lilly, HSBC, Mastercard; former DBS Bank CEO, former J.P. Morgan & Co, investment banker

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+SEED Co-founder and Scientific Advisory Board Member
*Board Member

Exceeding Seed Financing Expectations

SEED Internal Program Milestones

Development of SEED's unique TPD platforms and filed patents

- Multi-dimensional platforms to select the right E3 for any target;
- HTS screening and medicinal chemistry platforms which incorporate AI-predicted blood brain barrier penetration properties for CNS drug development,
- Proprietary statistical learning algorithms and neural networks (AI)

Infrastructure and Organization Building

- Renovated and occupied 10,000 sq ft SEED Headquarter, with 7,000 sq ft lab space;
- Hired full time drug R&D personnel, with significant focus on expertise in early-stage drug discovery and development

Translation of SEED Platforms into Drug Pipeline of 8 disclosed programs in various disease areas

RBM39 Degrader: POC in cell and animal models; lead candidate in oncology advancing to FHD around 1H 2025

2020 | 2021 | 2022 | 2023 and beyond

Nov. 2020: SEED received $10 M investment and entered into a research collaboration and license agreement with Eli Lilly on multiple targets in TPD (upfront $10 M, up to $780 M milestone payments and tiered sales royalties)

Jun. 2022: Received additional investment upon achieving 1st milestone

Feb. 2023: Received 2nd milestone payment

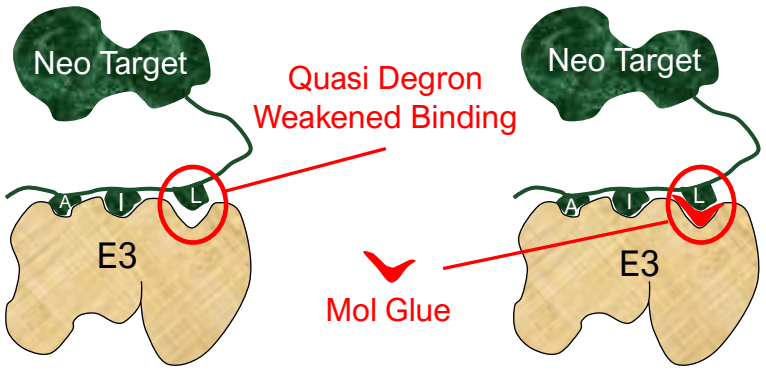
Mar. 2024: Received 3rd milestone payment

2024-2025: Target meaningful milestone payments

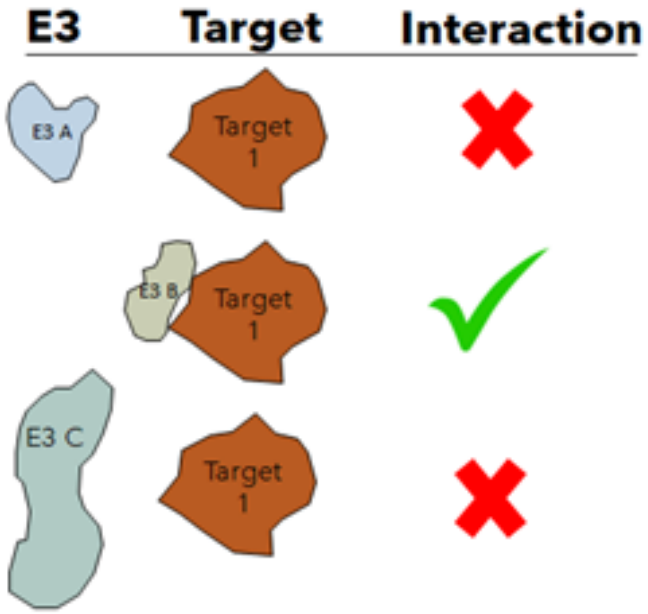
Eli Lilly Partner Program Milestones

SEED's Differentiation: Multi-dimensional Platforms for E3 Selection

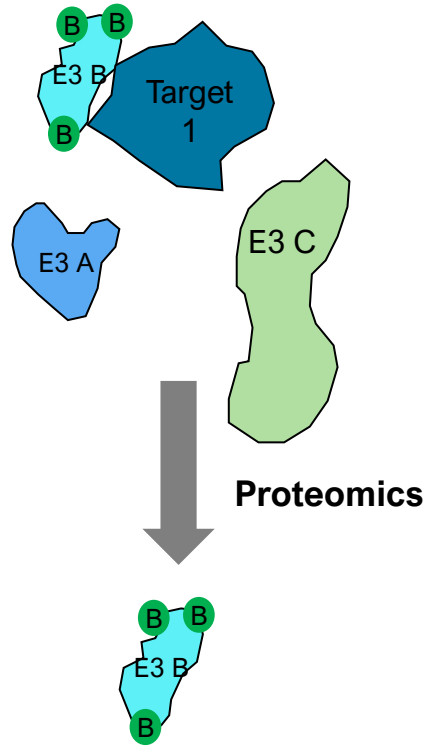
Knowledge-based Quasi Degron Approach



Direct Binding Validation

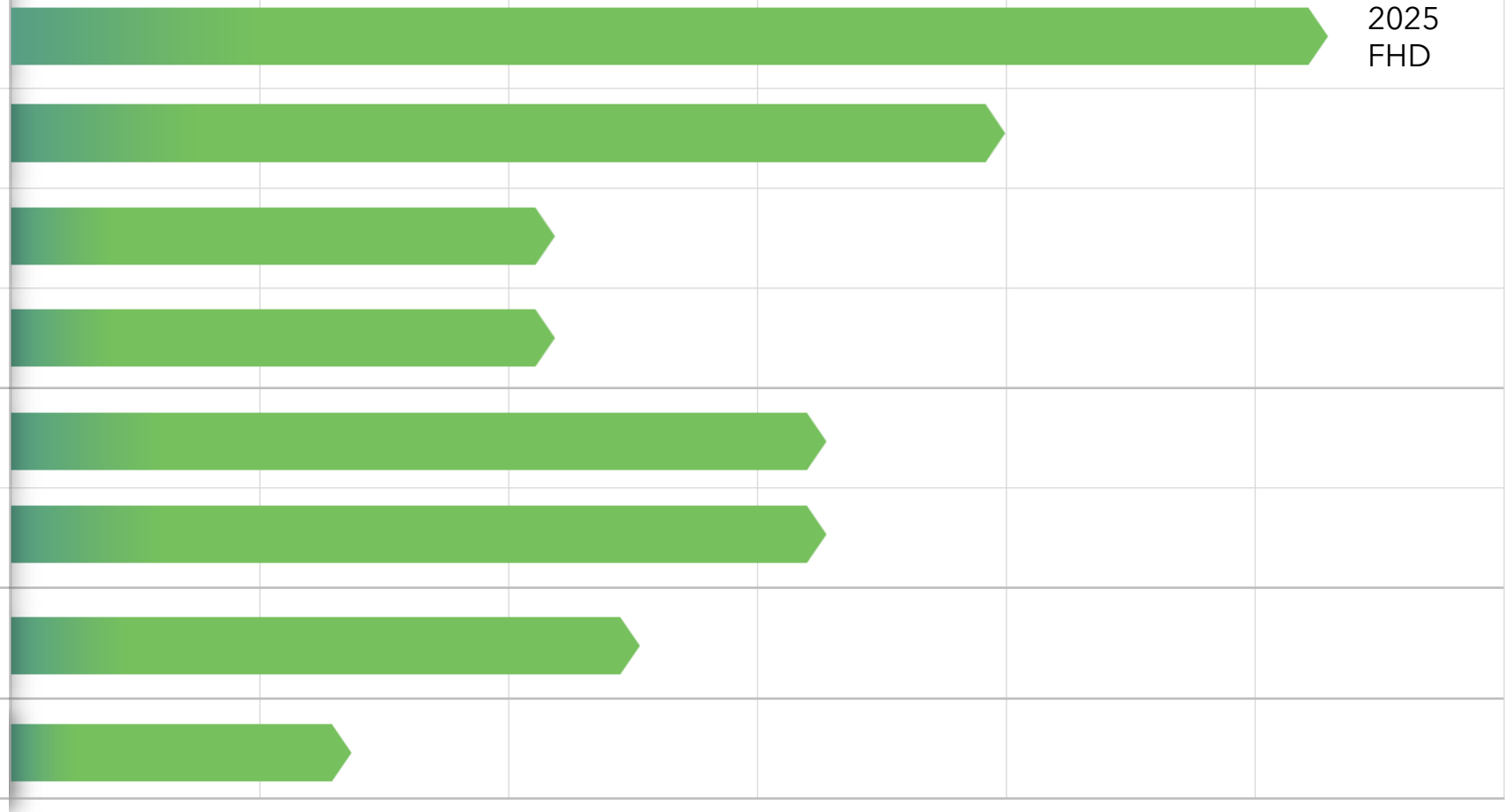


LumID: Identify the right E3 in the "living cell" near protein of interest



8 Drug R&D Pipeline in Multiple Disease Areas

Indication	Target Protein	Target Selection	E3 Ligase ID	Molecular Glue HTS	Lead ID	IND Candidate	IND Filing
Oncology	RBM39						2025 FHD
	KRAS-G12D						
	Target Beta						
	FEN1						
Neurodegeration	Target Alpha						
	Tau						
Immunology	Target Gamma						
Antiviral	HBx						



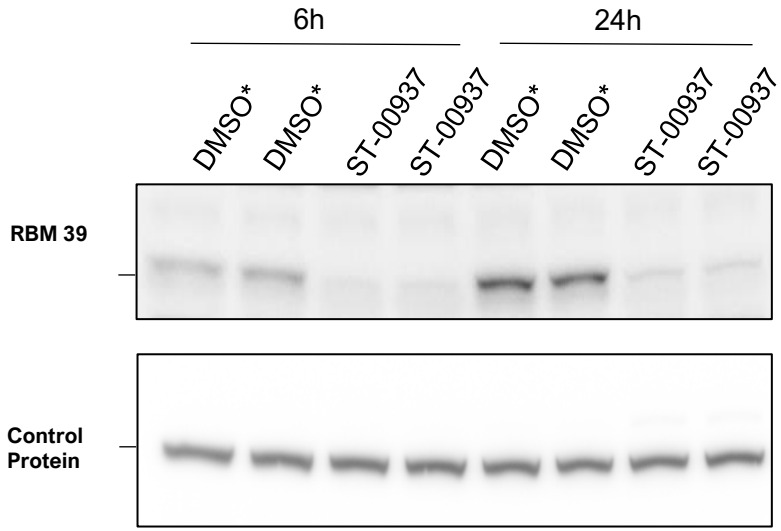
* SEED owns global IP on all programs except for two joint programs with Eli Lilly

Our RBM39 Degrader Class: Potentially Best-in-Class and First-to-Market

Program Summary

- **POI**: RNA splicing factor RMB39; **E3**: DCAF15; **MOA**: RMB39 degrader MG
- **Target indications**: Biomarker selected colon cancer, prostate cancer, neuroblastoma, and others
- **Development stage**: IND candidate; 1H 2025 FHD
- **Differentiation**: Our novel degrader demonstrates superior anticancer potency in cell line, improved pharmacokinetics and brain permeability, improved metabolic stability and absent hERG activity vs. comparators
- **Preclinical POC**: Animal data demonstrates its potential to have powerful anticancer effects with excellent safety profile
- SEED owns **global rights**

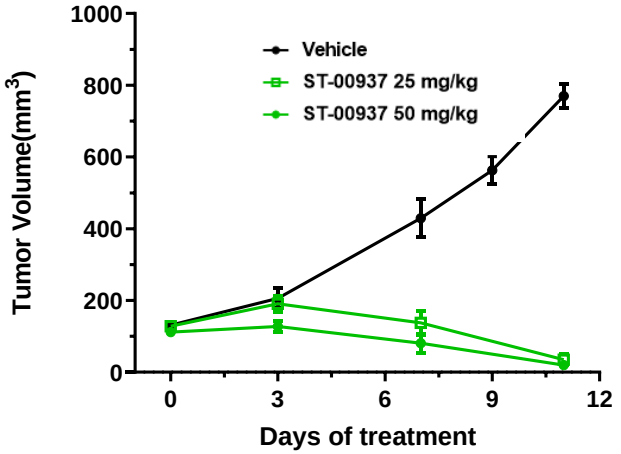
POC: RBM39 Degradation in Cell Line



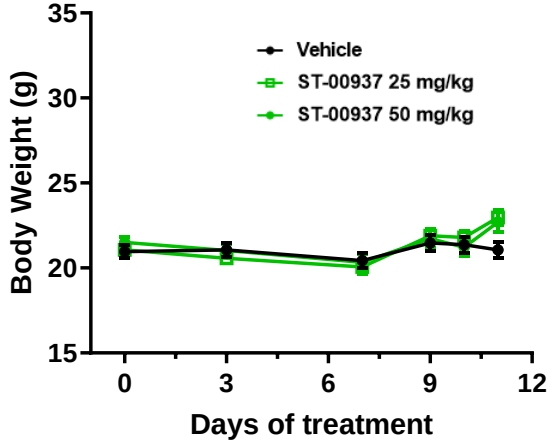
*Vehicle

Human colorectal cancer cell line

Superb In Vivo Efficacy: Complete Tumor Regression



Good In Vivo Safety: No Weight Loss

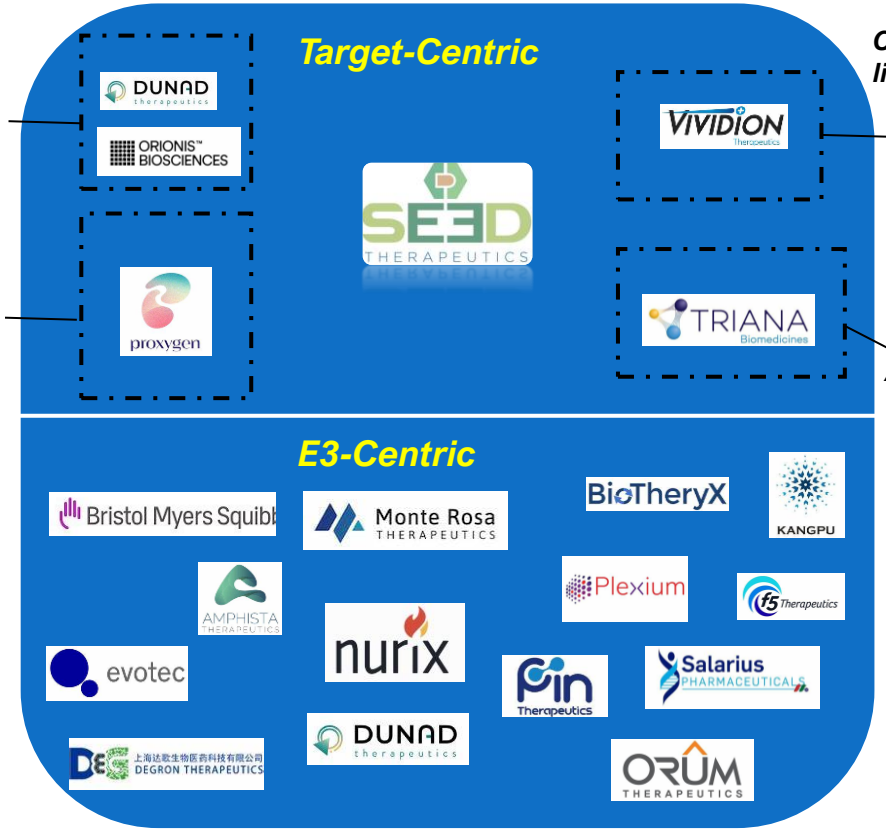


Colorectal xenograft in immunodeficient mice (Oral dose, twice daily)

TPD: a High Value and Novel Therapeutic Modality

Allosteric effect based
- Not MG selective
- Lack of evidence for TPD through small molecule-induced allosteric changes in protein structure

Cell-based HTS assays
- May not be MG selective
- Difficult to screen at higher compound concentrations that may be required



Covalent binder libraries
- Lack of evidence of target specificity
- No consideration for importance of Basal Interaction

AI-based approach
- Lack of evidence for applicability to Molecular Glue discovery from scratch and E3 selection

All top 20 global pharma have TPD programs internally and / or through collaboration

- **Discovery stage TPD assets** has been commanding **$35 - $60 million** upfront and **$500 million - $5 billion** milestone payment. Notable transactions include licensing and R&D collaboration deals between
  - ✓Genentech and Orionis; Genentech and Monte Rosa
  - ✓Astellas and Cullgen
  - ✓BMS and Evotec
  - ✓Genentech and Jemincare
  - ✓Bayer's acquisition of Vividion for **$1.5 billion** in 2021
  - ✓Merck's acquisition of Peloton for **$1.05 billion** in 2019

- **Pre-IND/ IND stage TPD assets** has been commanding **$100 - $300 million** upfront and **up to $2 billion** milestone payment. Notable transactions include licensing deals of
  - ✓Eli Lilly from Foghorn
  - ✓Sanofi from Kymera
  - ✓GSK from IDEAYA
  - ✓BMS and Orum

- **Clinical stage TPD asset** (early Phase II) has commanded **$650 million** upfront and **$350 million** equity investment in
  - ✓ Pfizer/ Arvinas' collaboration

Summary: First to Market and Best in Class

Scalable Business Model	TPD has potential to target 80% of undruggable disease-causing proteins. > 600 E3 ligases to be used for multiple disease areas.
Target-Centric Differentiation	Identify the right E3 for POI to increase the success rate in drug discovery: Platforms using **Quasi-degron, LumID, and Basal Affinity**
Pioneering Effort	World-leading scientific founding team and experienced development team in successful translation of TPD platforms to 8 disclosed Assets.
Two Prong Approach	De-risked revenue model: 1) R&D partnership for non-diluting financing (Eli Lilly); and 2) internal program development for value generation

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Plinabulin: First-in-class Late Stage Clinical Asset for Cancer Treatment

First-in-class Asset: Plinabulin

Advanced Clinical Stage Agent	• Positive Phase 3 study in 2L/3L NSCLC, successfully demonstrated significant OS benefit, doubling 2-year, 3-year OS rate – proving plinabulin's clinical utility
Enhances Cancer-Immunity Cycle	• Promising efficacy data in combination with PD-(L)1 and radiation, in IO-refractory patients across various cancers • Dual-acting IO MOA enhances the cancer immunity cycle, with potential to increase the efficacy and durability of checkpoint inhibitors and other immunotherapy agents
Favorable Safety Profile	• **700 cancer patients treated with good tolerability** • Clinically-proven to significantly reduce chemotherapy-induced neutropenia, enabling extended dosing of neutropenia-limited regimens such as chemotherapy and potentially ADCs
Ease of Use	• Intravenous (IV) Infusion: 1 or 2 doses per cycle
Strong Global Patent Protection	• 170 Granted/Allowed Patent to 2038 in 48 jurisdictions • Strong combination utility with potential to extend patent life of an approved asset

Plinabulin is a Differentiated First-in-Class Tubulin Binder with a Uniquely Favorable Safety Profile

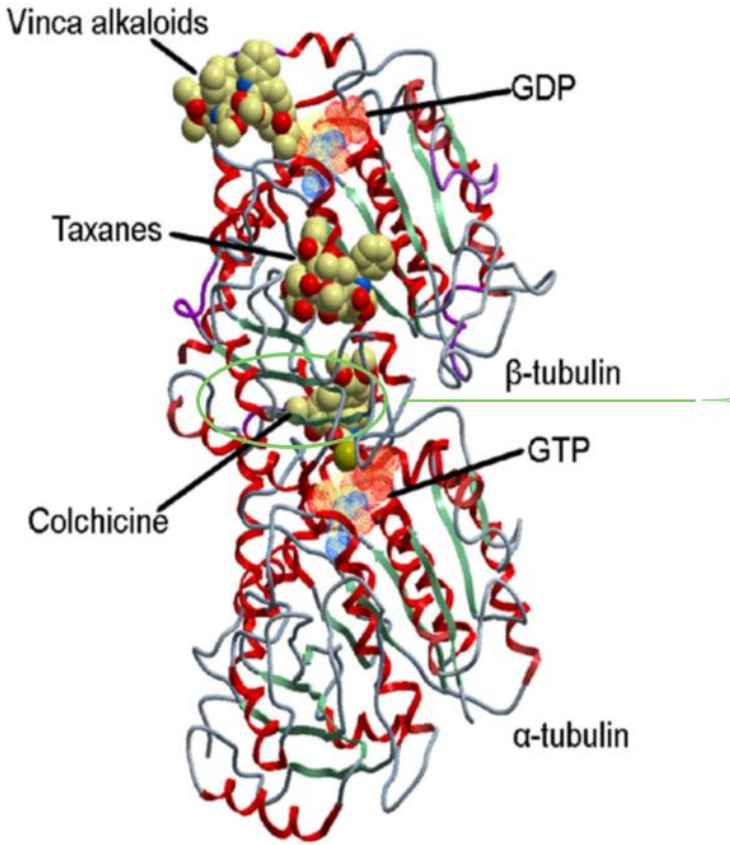
Plinabulin is a reversible tubulin binder and does not change tubulin dynamics

Conventional tubulin binding agents (such as taxanes, vinca alkaloids, and colchicine) alter tubulin dynamics upon binding, resulting in neutropenia and cardiac side effects.

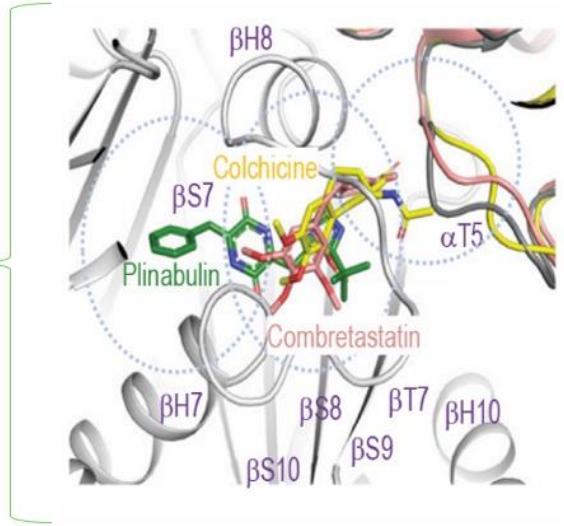
Plinabulin's tubulin binding site is distinct from that of these other agents (first in class). Because **binding is reversible**, plinabulin does not change tubulin dynamics.

Consequently, **plinabulin exhibits a favorable safety profile**, differentiating it clinically from other tubulin binding agents with concerning side effects that restrict their clinical utility.

Tubulin Binding Sites



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

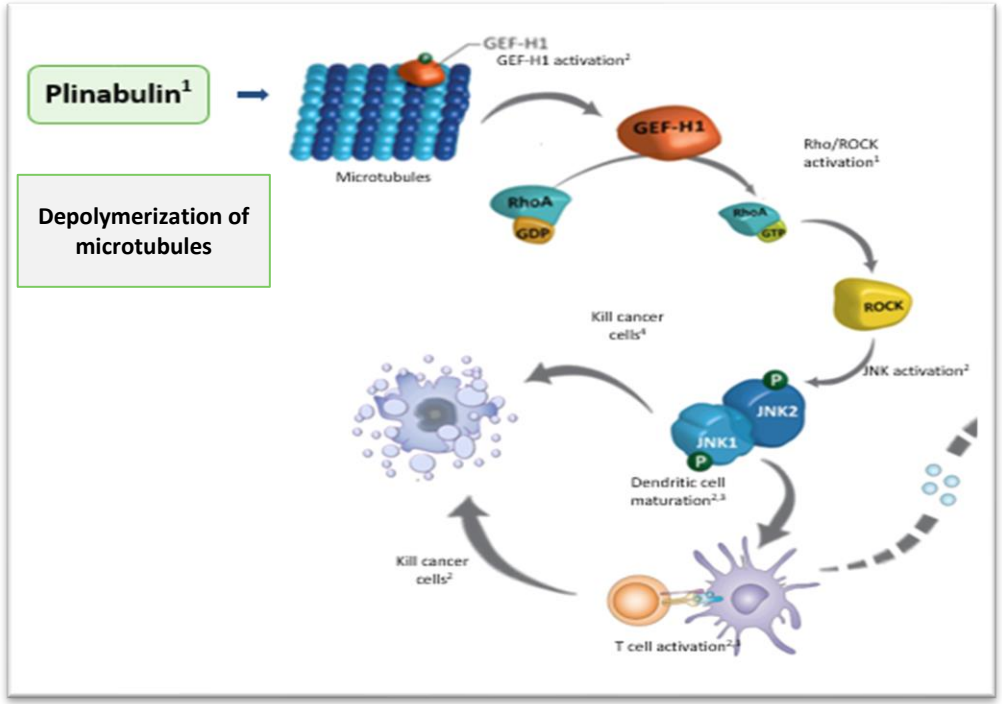


[1] La Sala et al., 2019 Chem

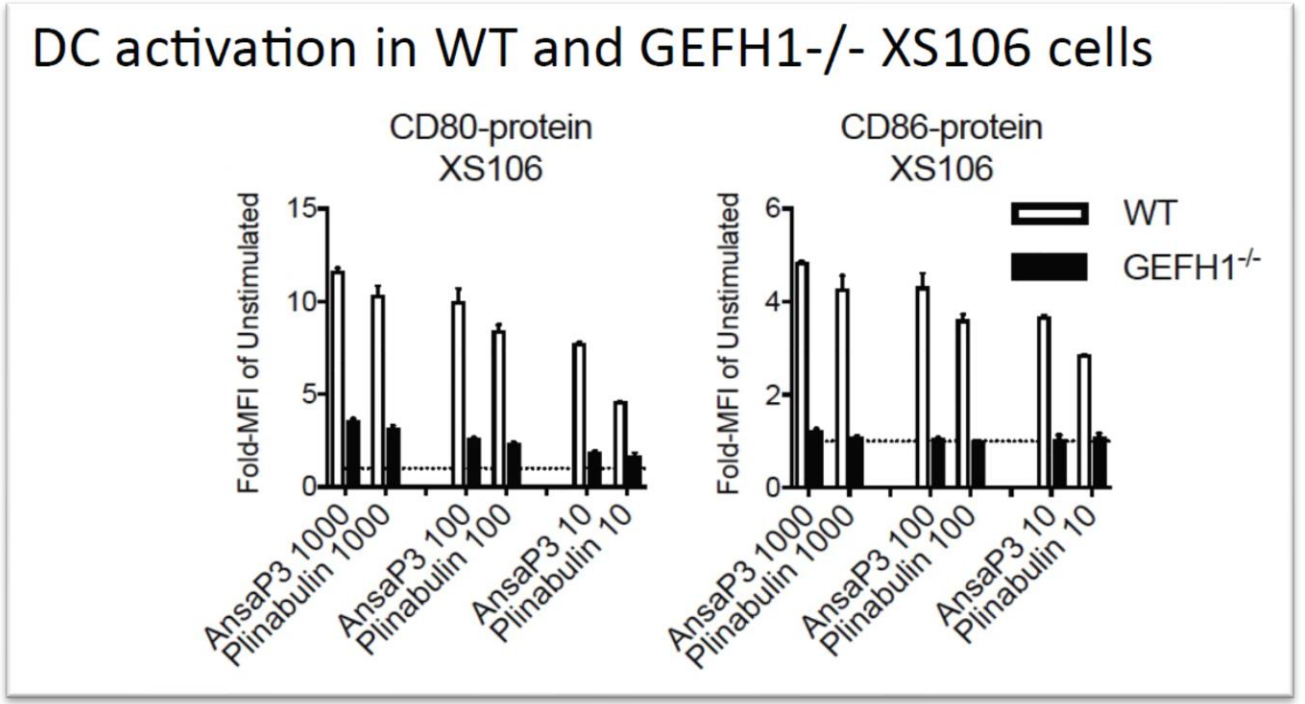
Plinabulin: Induce Innate and Adaptive Immunity

By depolymerizing microtubules, plinabulin releases, or activates, immune-defense protein GEF-H1. This leads to dendritic cell maturation (the most potent APC).

Plinabulin Novel Target: Immune Defense Protein GEF-H1[1]



In WT DC cells, plinabulin can induce DC maturation, but not in GEF-H1 deleted DC cells[2]
CD80 and CD86 up-regulation are biomarkers for DC maturation



AnsaP3, Maytansinoid cytotoxic (positive control compound), too toxic for human study

[1] La Sala et al., Chem 5(11): 2969-2986 (2019)
[2] Kashyap et al., Cell Reports 28(13): 3367-3380 (2019)

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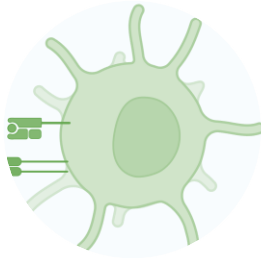
Plinabulin's Immunomodulation and Neutropenia-Mitigating Activities Position it as a Valuable Partner for Immuno-Chemotherapy Combination Regimens

These aspects result in an optimal scenario of added efficacy with potentially reduced toxicity.



Dendritic Cells

Plinabulin induces

dendritic cell maturation

Enhanced antigen presentation and T cell priming

M1-like Macrophages

Plinabulin stimulates

M1-like macrophage polarization and proliferation

Increased tumor cell killing and cytotoxic T cell recruitment

Improves Safety*

Plinabulin **reduces chemotherapy-induced neutropenia**

Improved therapeutic index of chemotherapy-based regimens

Collaborates with PD1/PD-L1 targeting agents to enhance T cell function and kill tumor cells

Extends therapeutic duration of CPI + chemo combinations

*To date, over 700 cancer patients have been treated with good tolerability.

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Plinabulin improves overall survival and enhances safety in 2L/3L NSCLC (Dublin-3 Study)

The EGFR-wild Type 2L/3L NSCLC Have Been a Historically Difficult Space in Which to Develop

Treatment options in 2L/3L NSCLC are limited

Docetaxel-based therapies are the mainstay therapy in 2L/3L NSCLC (EGFR wt).

However, **docetaxel-based therapies (SOC) demonstrate limited efficacy and are associated with >40% severe (grade 3/4) neutropenia.**

Other approved agents:

- Ramuciramab + Docetaxel vs. Docetaxel: OS HR=0.86, severe neutropenia 49% vs. 40%;
- Pemtrexed vs. Docetaxel: OS HR=0.99, severe neutropenia 5% vs. 40%.

Additionally, with immunotherapies moving to first line NSCLC, **there is a growing population of 2L/3L patients that are refractory to immunotherapy.**

Attempts to address treatment needs have been challenging

Since Nivolumab's approval 8 years ago, no new agent with a novel mechanism has been approved in this indication.

Multiple Phase 3 studies (PD-1/PD-L1 failed patients, 2L/3L NSCLC), did not meet OS endpoint vs. docetaxel:

1. BMS' Nivolumab (PD-1 antibody) + Mirati's Sitravatinib (TKI) combination
2. Roche's Atezolizumab (PD-L1 antibody) + Exelixis's Cabozantinib (TKI)
3. Merck's Pembrolizumab (PD-L1 antibody) + Eisai's Lenvima (TKI)
4. Novartis' Canakinumab (IL-1b antibody) + docetaxel
5. Gilead's sacituzumab govitecan-hziy (ADC - antibody drug conjugate)

Recent successful phase 3 studies with mixed results:

- Lunar (TTfields vs. docetaxel): OS benefit (HR=0.74), but no PFS and ORR benefit;
- TROPION-Lung01 (Datopotamab deruxtecan vs. docetaxel): OS benefit (HR=0.90) in ITT population, with better OS (HR=0.75) in non-squamous NSCLC.

Plinabulin Has Been Evaluated in Combination with Docetaxel in a Phase 3 Study with advanced, Pre-treated NSCLC Patients

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

Study Plan

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

Primary endpoint

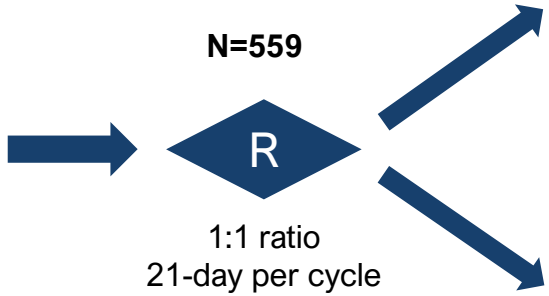
Overall survival (OS)

Secondary endpoints

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

Inclusion Criteria:

- Non-squamous or squamous NSCLC
- Stage IIIb/IV
- ECOG ≤ 2
- Progression during or after treatment with one or two treatment regimens containing a platinum
- Must have at least one measurable lung lesion
- **Prior checkpoint inhibitor therapy allowed**[1]



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)

[1] 85% CPI naïve; **15% failed PD-(L)1 blockade**

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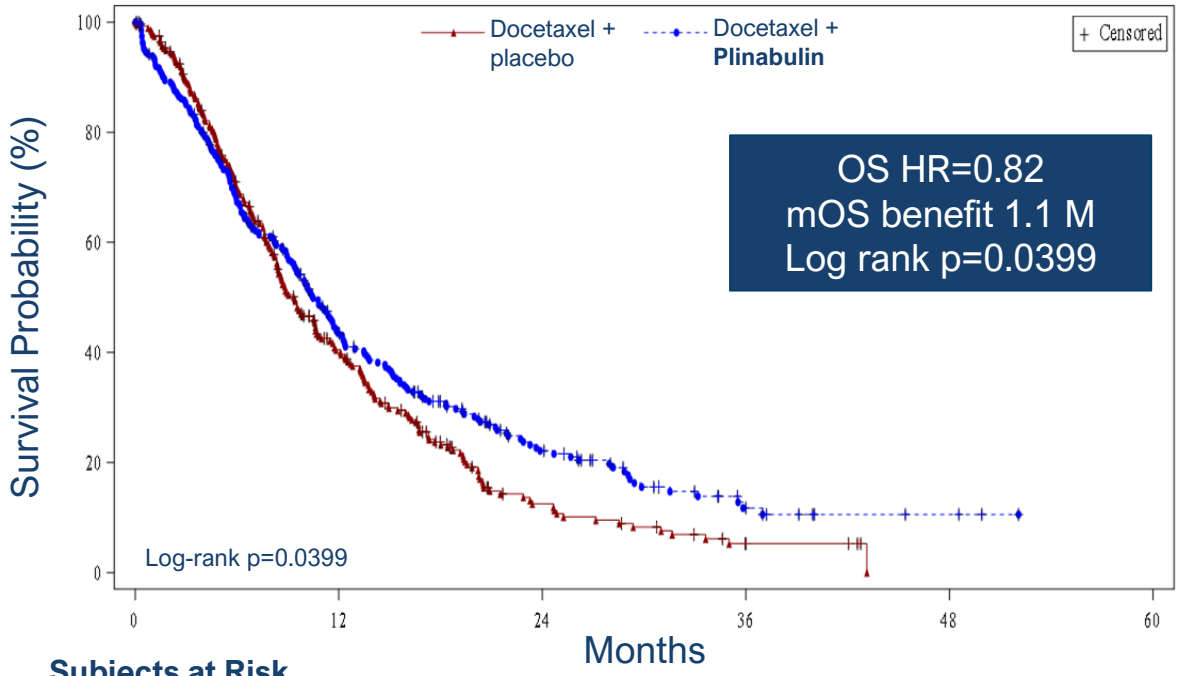
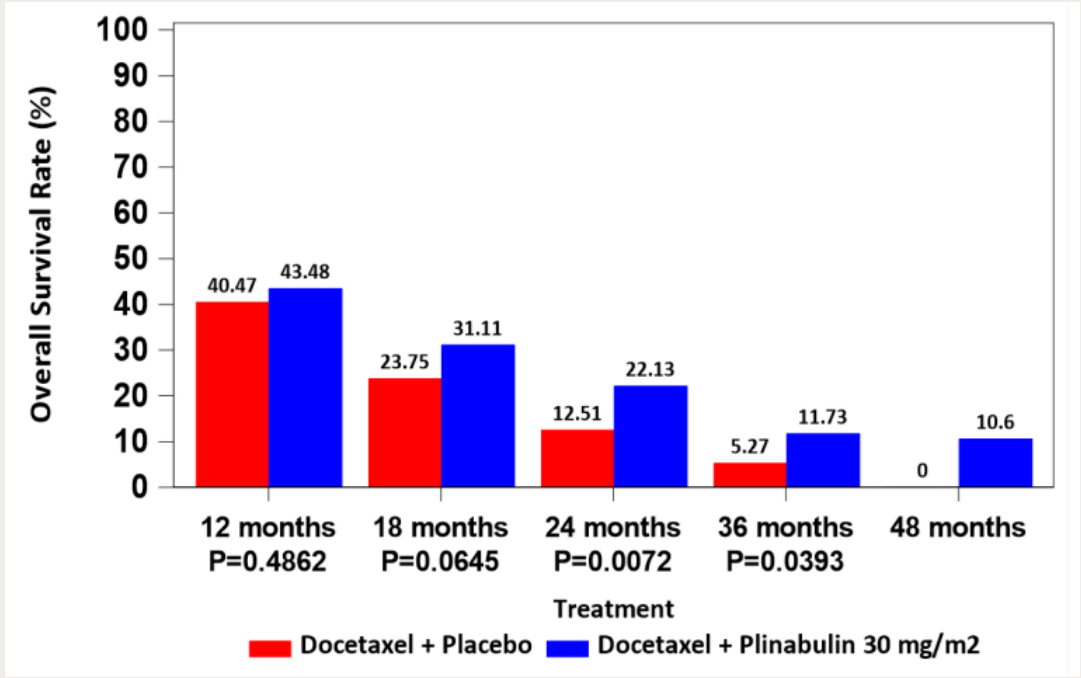
Plinabulin + Docetaxel Met its Primary Endpoint (OS) and Showed Significant Improvement in Long-term OS Rate



Subjects at Risk

Docetaxel (75mg/m2) + placebo					
281	97	21	4	0	0
Docetaxel (75mg/m2) + Plinabulin (30mg/m2)					
278	108	41	10	3	0

	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)

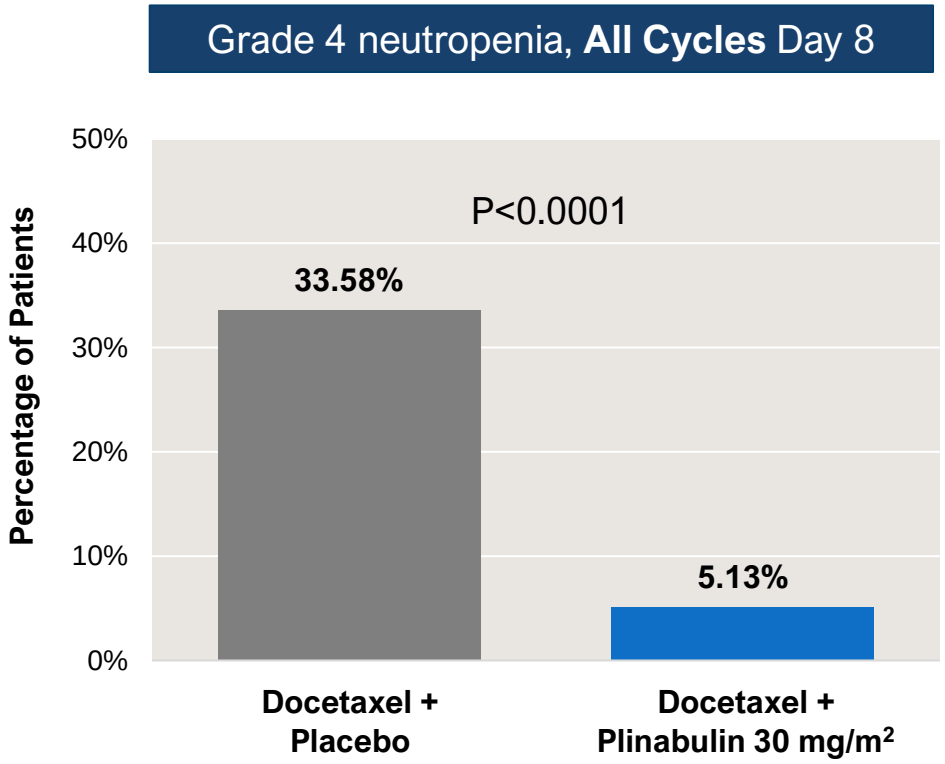


OS Rate Increase Results

- Significantly increased OS rate in 24 months, and 36 months (doubling benefit)
- 48m OS rate: D + Product X (10.6%) vs D (0%)

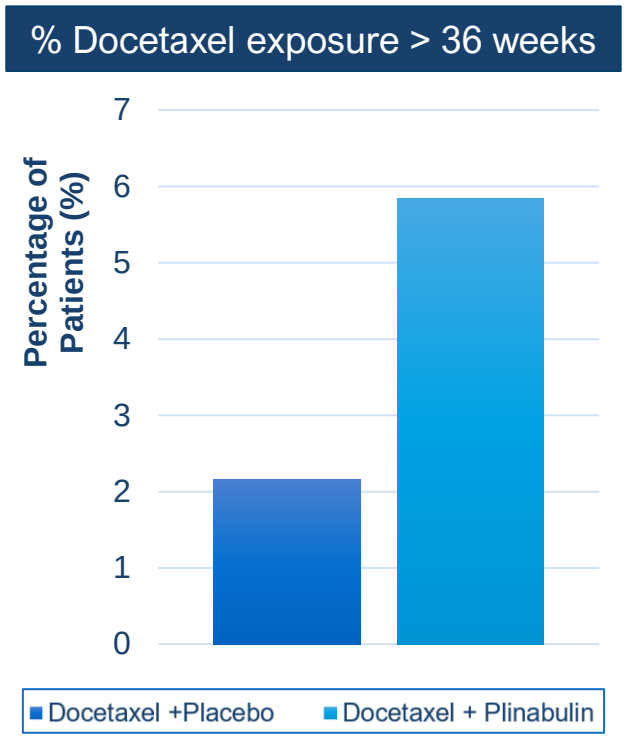
Plinabulin Not Only Slows Progressive disease, but Also Increased the Tolerability of Docetaxel and Increased Duration of Treatment

Use of plinabulin significantly reduced Grade 4 neutropenia



Similar results were observed for Grade 4 neutropenia on Cycle 1 Day 8

…allowing more patients to remain on docetaxel for a longer duration



Addition of plinabulin to docetaxel also increased docetaxel exposure by mean dose (mg)

Plinabulin Successfully Improved Efficacy of SOC in 2L/3L NSCLC, Proving its Clinical Utility, Despite Historical Failures in this Space

The addition of plinabulin as a single agent added to 2L/3L NSCLC standard-of-care led to improved overall survival and enhanced safety

Efficacy

- Significant survival benefit in ITT (OS HR=0.82)
- Even more pronounced survival benefit in 2L (HR=0.78), or non-squamous NSCLC (HR=0.76)

Safety and tolerability

- The regimen is well tolerated. Side effects include transient hypertension which resolves in 4-6 hours, nausea, vomiting and GI side effects.
- Significant QoL benefit
- Docetaxel-induced neutropenia was reduced, allowing increased treatment exposure

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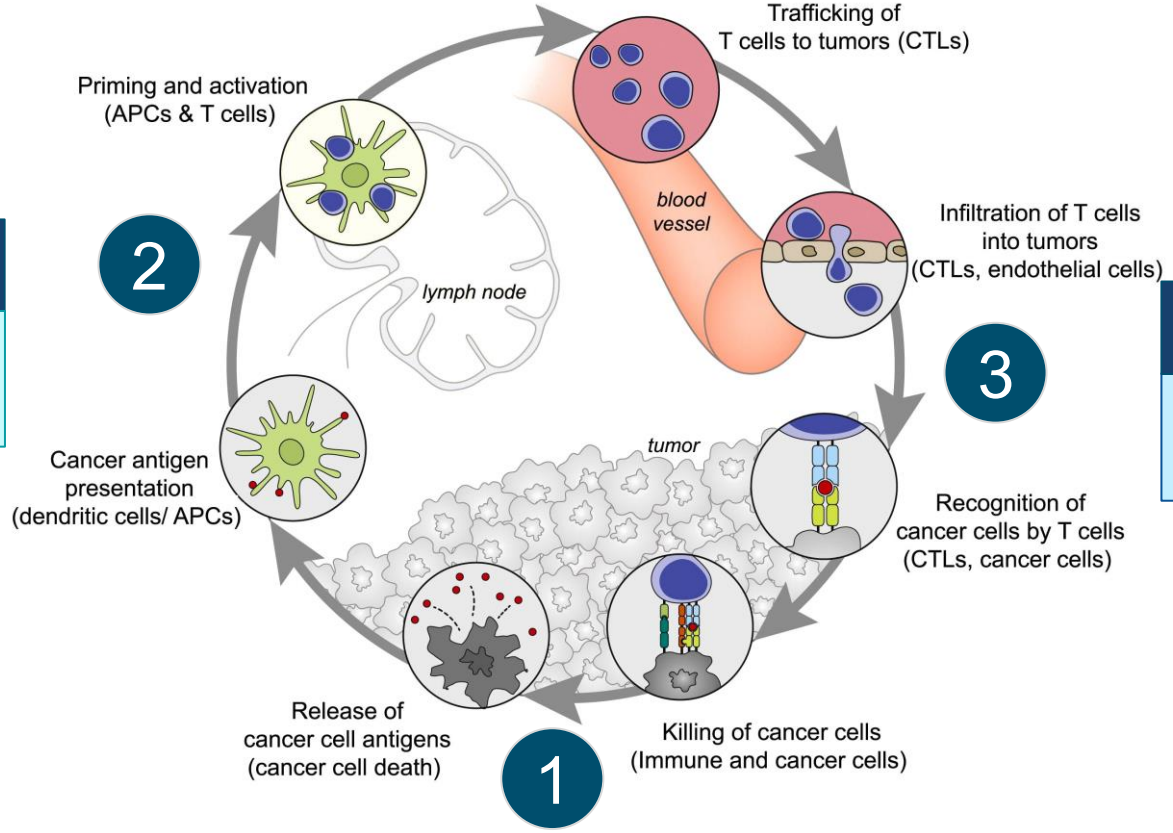
Encouraging RT+PD-1+Plinabulin clinical data demonstrates Plinabulin's partnering potential with IO agents

RT: Radiation therapy

Plinabulin Enhances the Cancer Immunity Cycle When Used with Radiation and Anti-PD1

② Plinabulin

Improved antigen presentation
Stimulate maturation of dendritic cells to increase antigen presentation.



③ Checkpoint Inhibitors

Anti-tumor T cell activation
Optimize T cell response

① Chemotherapy
Radiation Therapy
Oncolytic Viruses
Antibody Drug Conjugates
Targeted Therapy

① Radiation/Chemotherapy

Release tumor antigens
For more potent anti-cancer effect

BeyondSpring

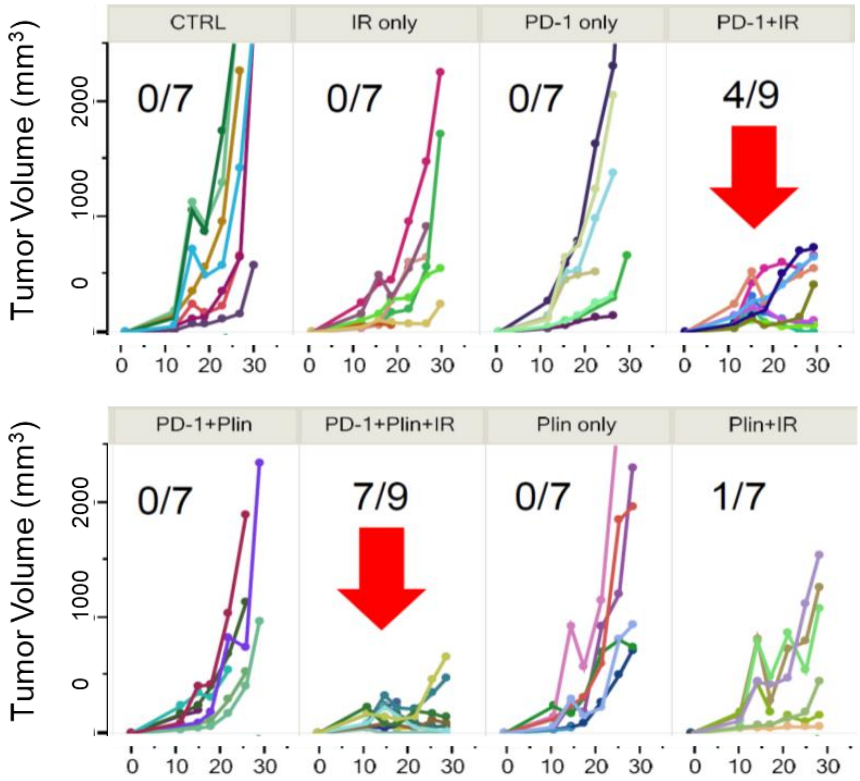
Plinabulin is Being Evaluated in Multiple Immunotherapy Combination Trials in Collaboration with Major Pharmaceutical Companies

	Indication/Target	Program	Preclinical	Phase 1	Phase 2	Phase 3	Trial Name / Collaborator
Investigator Initiated Trials	**ES-SCLC (1st line)**	Plinabulin + Pembrolizumab + Etoposide / Platinum					
	NSCLC (2nd/3rd line PD-1/PD-L1 progressed)	Plinabulin + Pembrolizumab + Docetaxel					
	Multiple cancers (PD-1/PD-L1 progressed)	Plinabulin + PD-1/PD-L1 + Radiation					

MD Anderson Phase 1 study clinical data and biomarker studies was presented at SITC conference in November 2023.

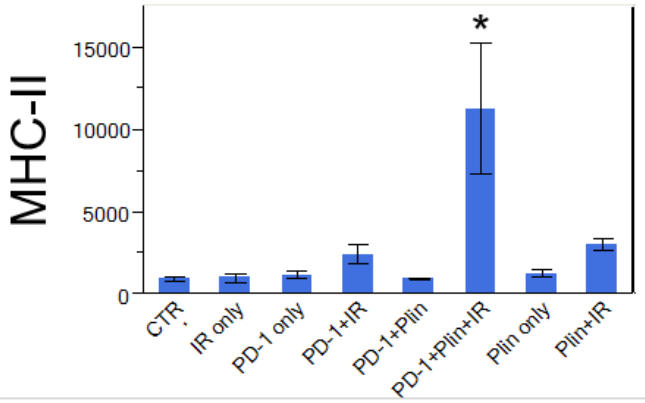
RT+anti-PD1+Plinabulin Triple Combination POC in Animals Provides Evidence of Plinabulin's Immunomodulatory activity

Tumor reduction is most robust in triple I/O combination

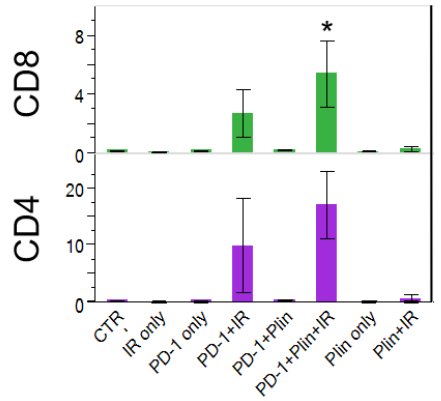


Plinabulin+RT+PD-1 induced ~80% tumor volume reduction in a preclinical cancer model

DC activation is most dramatic in triple I/O combination



T cell doubles in triple I/O combination vs. PD1 + IR



Plinabulin+ RT+PD-1

increased DC maturation and doubled CD4+ and CD8+ T cells in tumor samples 30 days after treatment

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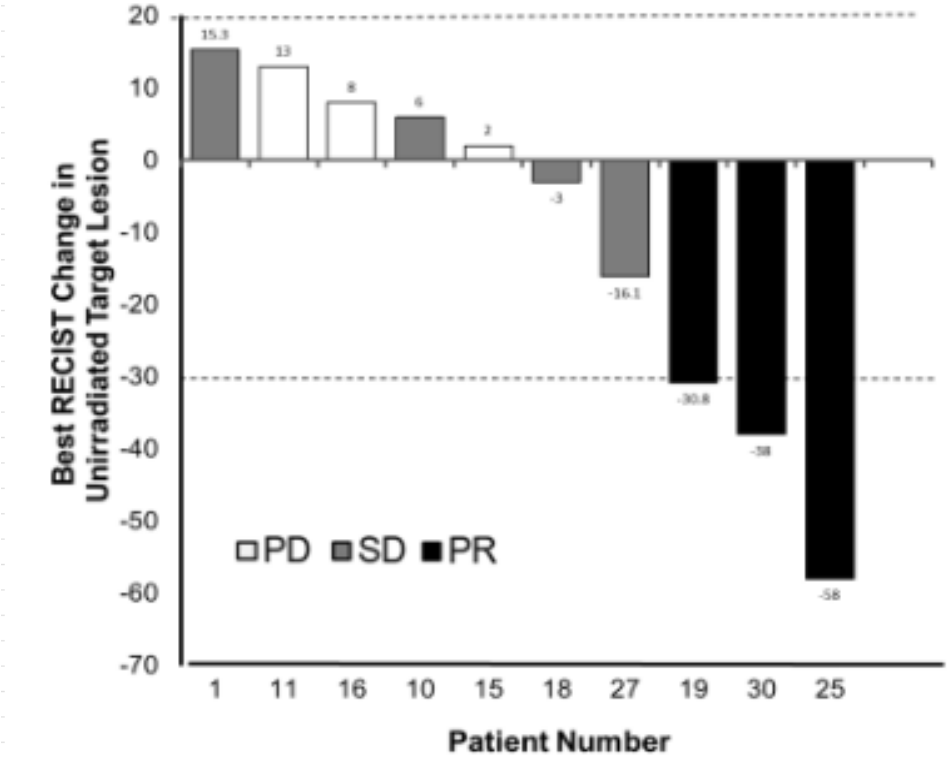
AACR 2020 (MD Anderson Collaboration)

Studies performed in TS/A Syngeneic Breast Cancer Model

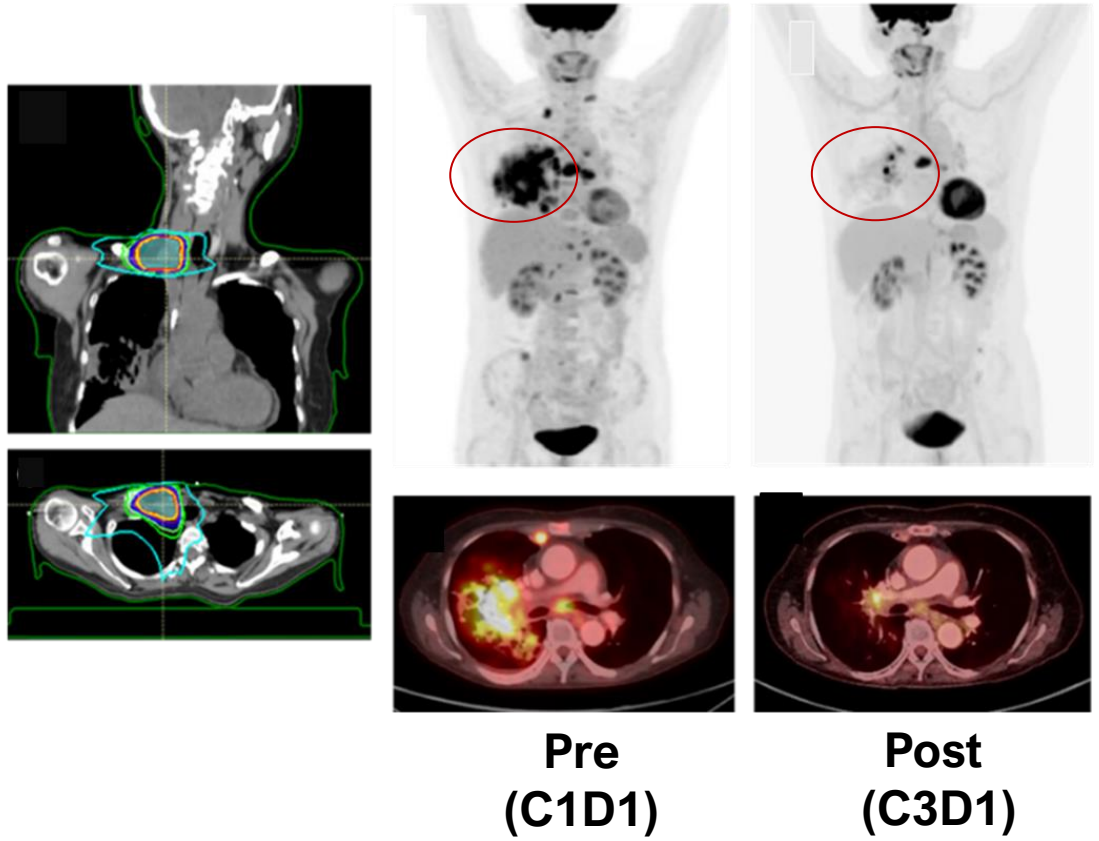
Clinical PoC in **Efficacy**: Plinabulin Triple Combo Produces Clinically Meaningful Responses in the Non-Irradiated Tumor Across Multiple Cancers after IO-failure

Plinabulin triple combination led to 70% DCR (3 PR, 4 SD) in 10 IO-failed patients with Tumor assessment



Durable response has been observed in 2 Hodgkin lymphoma patients who progressed after 12 or 16 prior lines of therapy.

Systemic abscopal effect seen comparing baseline and C3D1 for one Hodgkin lymphoma patient



Seven IO-relapsed/refractory cancers: NSCLC (Patient #1, #19); Merkel cell (#11); RCC (#16, #18); FL-HCC (#10); CRC (#15); HNSCC (#27); Hodgkin (#25, #30)

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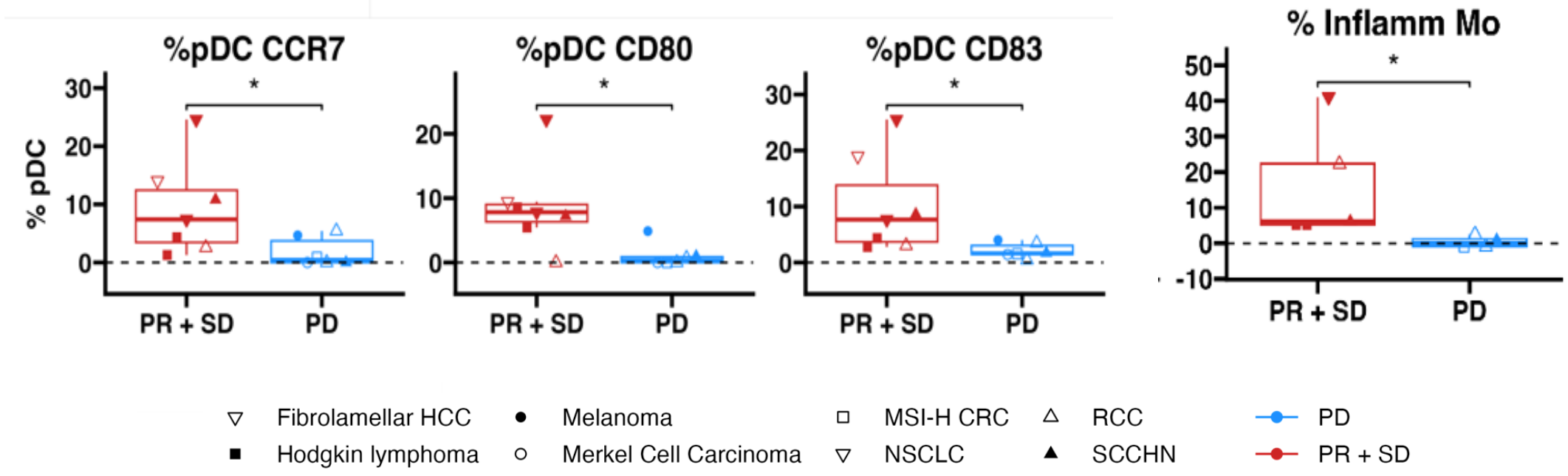
Clinical PoC in MOA: Plinabulin-Responding Patients Show Early Immune Activation Evidenced by DC Maturation and Proinflammatory Monocytes in the Peripheral Blood

In different cancers, DC maturation and anti-tumor inflammatory macrophages were observed in plinabulin-responding patients

Dendritic Cell Maturation & Migration

Proinflammatory monocyte/macrophages



Plinabulin as Potential Add-on Therapy to Current I/O Regimens to Address Severe Unmet Medical Needs

PD-1/PD-L1 Inhibitors
- >$30B global annual sales

→ Potential to greatly expand the addressable market

Current Severe Unmet Medical Needs

2L/3L: PD-1/PD-L1 relapsed/refractory patients

1L: PD-1/PD-L1 + chemo doubles anti-cancer efficacy of PD-1, but with CIN risk

"Cold" Tumor: PD-1/PD-L1 non-responsive tumor

Plinabulin:
APC Inducer with easy administration*

Plinabulin Clinical Development

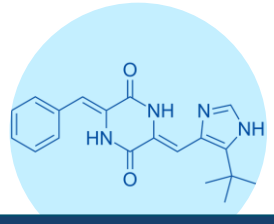
Re-sensitize: Plinabulin + PD-1/PD-L1 + chemo/radiation/ADC

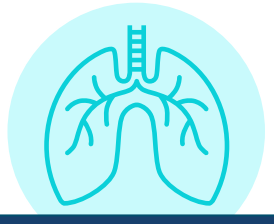
Increase Combo Anti-cancer Efficacy: Plinabulin + PD-1/PD-L1 + chemo/ADC

Turn "cold" into "hot" Responding Tumor: Plinabulin+ PD-1/PD-L1 + chemo/ADC; Plinabulin + chemo/ADC

*Plinabulin IV Infusion: 1 or 2 dose per cycle (DUBLIN-3 Trial)

Plinabulin's Multiple MoAs, Proven Clinical Activity, and Strong Global Patent Protection are Highly Favorable for Partnerships with Immunotherapy Agents…and Beyond





Lead Asset **Plinabulin displays dual IO MOAs**	**Proven clinical efficacy and safety**	**Enhances the Cancer-Immunity cycle**	Strong **global patent** protection
A first-in-class tubulin modulator that activates **dendritic cell** maturation and **M1-like macrophage** proliferation which **enables the cancer immunity cycle**	Successfully demonstrated significant **OS benefit** in 2L/3L NSCLC, as well as **reduction in severe neutropenia,** allowing **extended regimen duration**	Clinically enhanced the anti-tumor response to checkpoint inhibitors in combination with radiation or chemotherapy, even in **immunotherapy-refractory patients**	BeyondSpring (est. 2010) is a global company that has **170 Granted/Allowed Patents to 2038 in 48 jurisdictions**

Plinabulin enhances the cancer immunity cycle to increase patient survival and reduce adverse events in combination use settings with a minimal patient administration schedule.

Plinabulin's multiple mechanisms of action provide strong rationale for its combination with both immunotherapy agents as well as neutropenia-limited agents such as chemotherapy and ADCs

Investment Highlights

Plinabulin: Safety & Efficacy	Lead Asset Plinabulin: >700 cancer patients treated with good tolerability; Positive Phase 3 data in 2 indications
Plinabulin Potential	Plinabulin: Dual Benefit in anti-cancer and prevention of CIN; Potential in re-sensitizing in PD-1/PD-L1 progressed patients in multiple cancers
SEED: Novel TPD Platform & Pipeline	SEED: 8 Disclosed Pipeline Assets with 1 expected to enter IND in 2024
Premier Partnerships	SEED: Investment and R&D Collaboration from Eli Lilly
Intellectual Property	Strong IP and technology protection

www.beyondspringpharma.com