September 2023 | NASDAQ: BYSI

BeyondSpring

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Such statements are subject to known and unknown risks, uncertainties, and other factors that may cause our or our industry's actual results, levels of activity, performance, or achievements to be materially different from those anticipated by such statements. In some cases, you can identify forward-looking statements by terminology such as "may," "will," "should," "expects," "plans," "anticipates," "believes," "estimates," "predicts," "potential," "intends," or "continue," or the negative of these terms or other comparable terminology. Forward-looking statements contained in this presentation include, but are not limited to, (i) statements regarding the timing of anticipated clinical trials for our product candidates and our research and development programs; (ii) the timing of receipt of clinical data for our product candidates; (iii) our expectations regarding the potential safety, efficacy, or clinical utility of our product candidates; (iv) the size of patient populations targeted by our product candidates and market adoption of our product candidates by physicians and patients; and (v) the timing or likelihood of regulatory filings and approvals.

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The market data and certain other statistical information used throughout this presentation are based on independent industry publications, governmental publications, reports by market research firms or other independent sources. Some data are also based on our good faith estimates. Although we believe these third-party sources are reliable, we have not independently verified the information attributed to these third-party sources and cannot guarantee its accuracy and completeness. Similarly, our estimates have not been verified by any independent source.

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

Investment Highlights

Plinabulin: 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: 7 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 Intellectual Property and Technology Protection

Pipeline

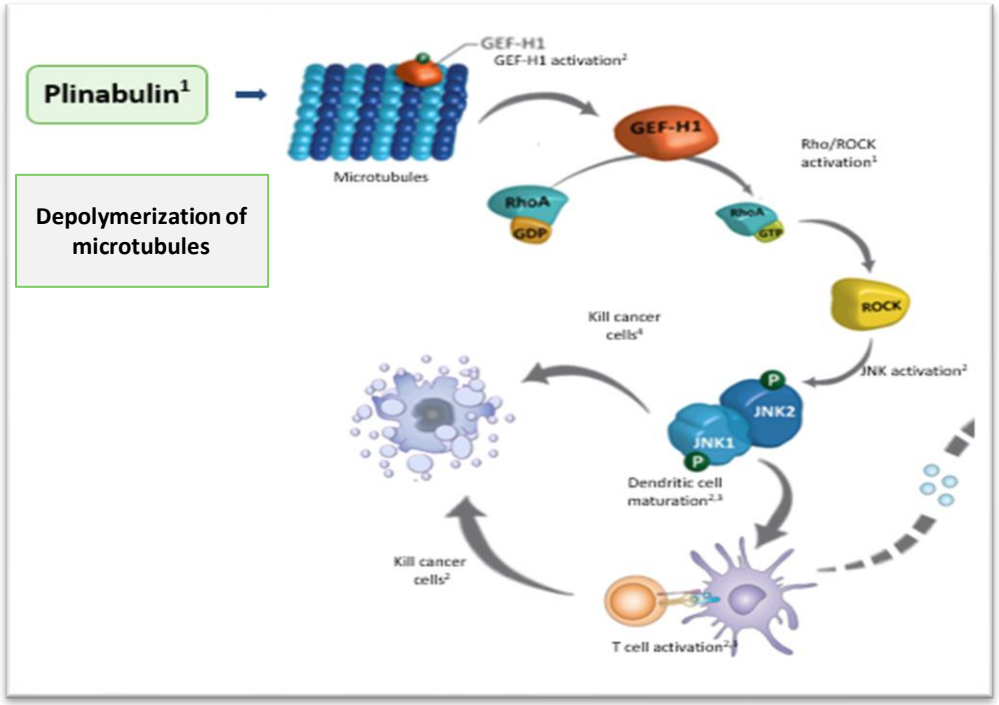
	Indication/Target	Program	Preclinical	Phase 1	Phase 2	Phase 3	Registration	Trial Name / Collaborator
Late stage	**NSCLC (2nd/3rd line)**	Plinabulin + Docetaxel						DUBLIN-3
	CIN Prevention	Plinabulin + Pegfilgrastim						PROTECTIVE-1 & PROTECTIVE-2
Investigator Initiated Trials	**SCLC (2nd/3rd line)**	Plinabulin + PD-1 + CTLA-4						Bristol Myers Squibb; Rutgers
	NSCLC (2nd/3rd line PD-1 failed)	Plinabulin + PD-1 + Docetaxel						Merck
	7 cancers (PD-1/PD-L1 failed)	Plinabulin + PD-1/PD-L1 + Radiation						The University of Texas MD Anderson Cancer Center
	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	**7 Disclosed Targets (Internal & Collaboration)**	Targeted Protein Degradation (TPD) Molecular Glue Platform						Lilly

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

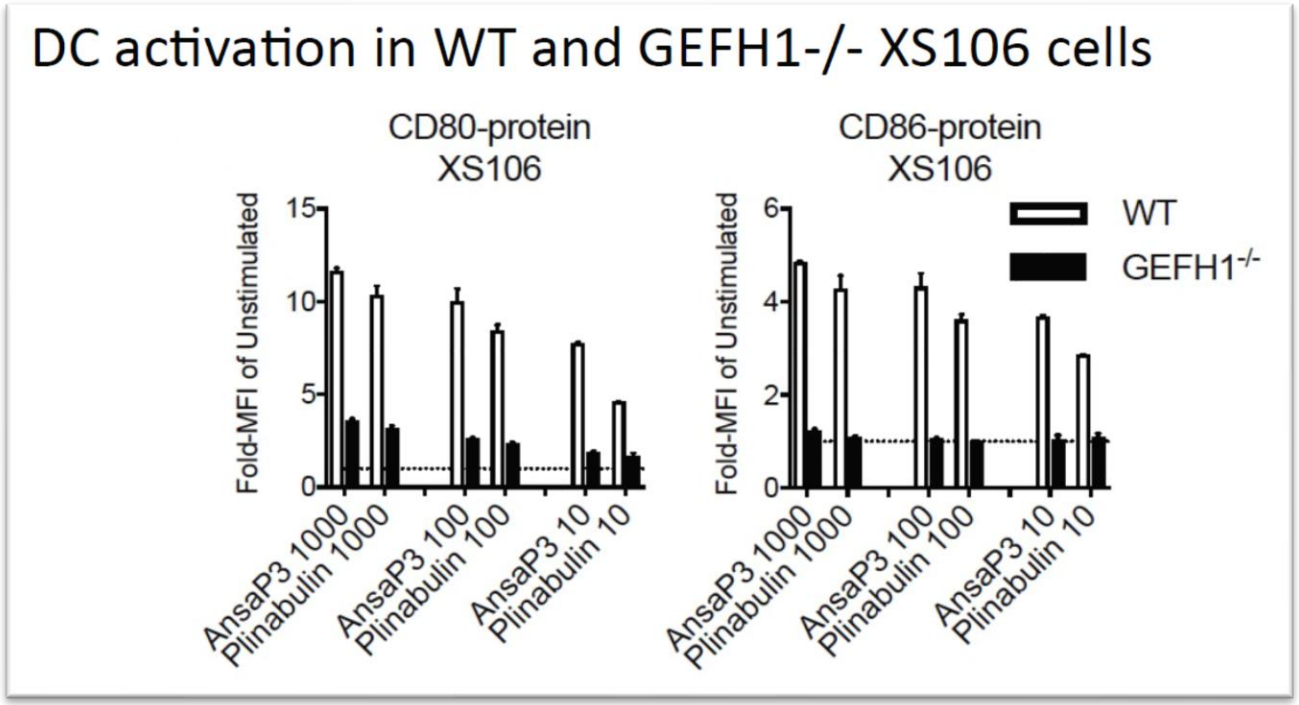
Plinabulin: Induce Innate and Adaptive Immunity

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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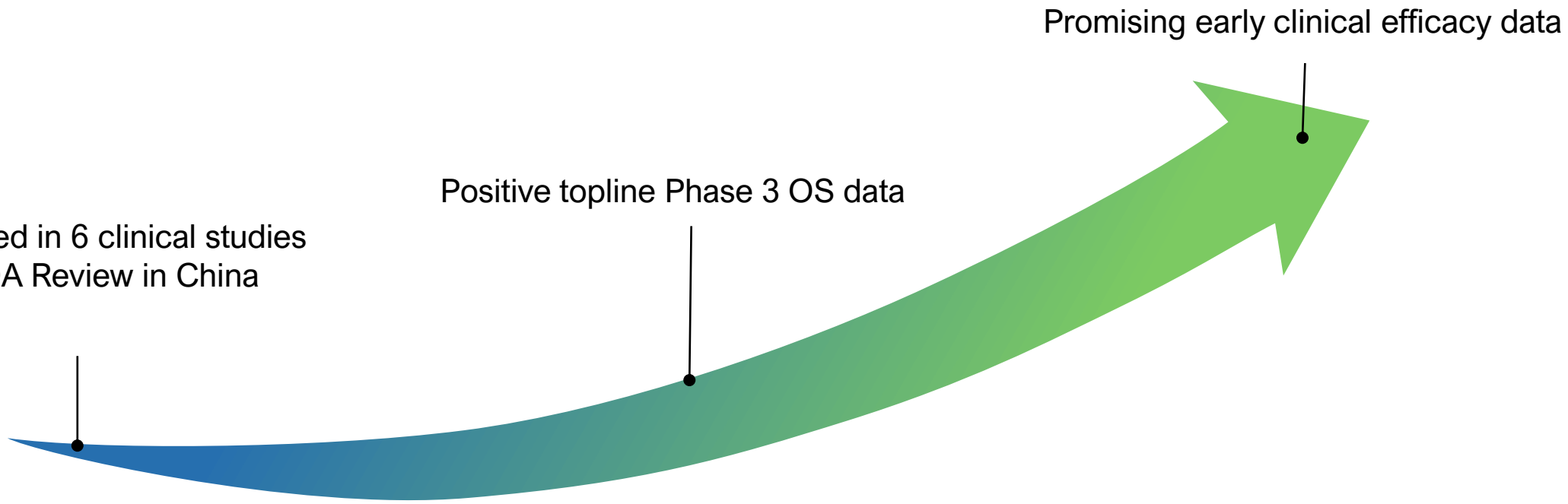
Plinabulin Franchise

Clinical Confirmation → **Expand** → **Transform**



CIN	NSCLC	Multiple Cancers (I/O Combo)

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SEED Therapeutics: Target Protein Degradation (TPD) Company

SEED Therapeutics

Founders:

- Avram Hershko, MD PhD (Nobel Prize Winner, Technion), a world expert in ubiquitin-mediated TPD research;
- Ning Zheng, PhD (HHMI, Pharmacology Dept, University of Washington), a pioneer in structural biology of ubiquitin E3 ligases;
- Michele Pagano, MD (HHMI, Chairman, Biochemistry and Molecular Pharmacology Dept, NYU School of Medicine), a pioneer in the biology of TPD research;
- Lan Huang, PhD (Scientist and entrepreneur, CEO of SEED), expert in TPD field by solving the first E3 ligase structure and biotech company operation.

Company Focus: Development of innovative precision medicine using proprietary and unique TPD platforms.

Drug Pipeline: The platform has been translated into a robust pipeline with 7 disclosed development programs including a drug candidate advancing to IND filing in 2024.

Investors and R&D Collaborator:

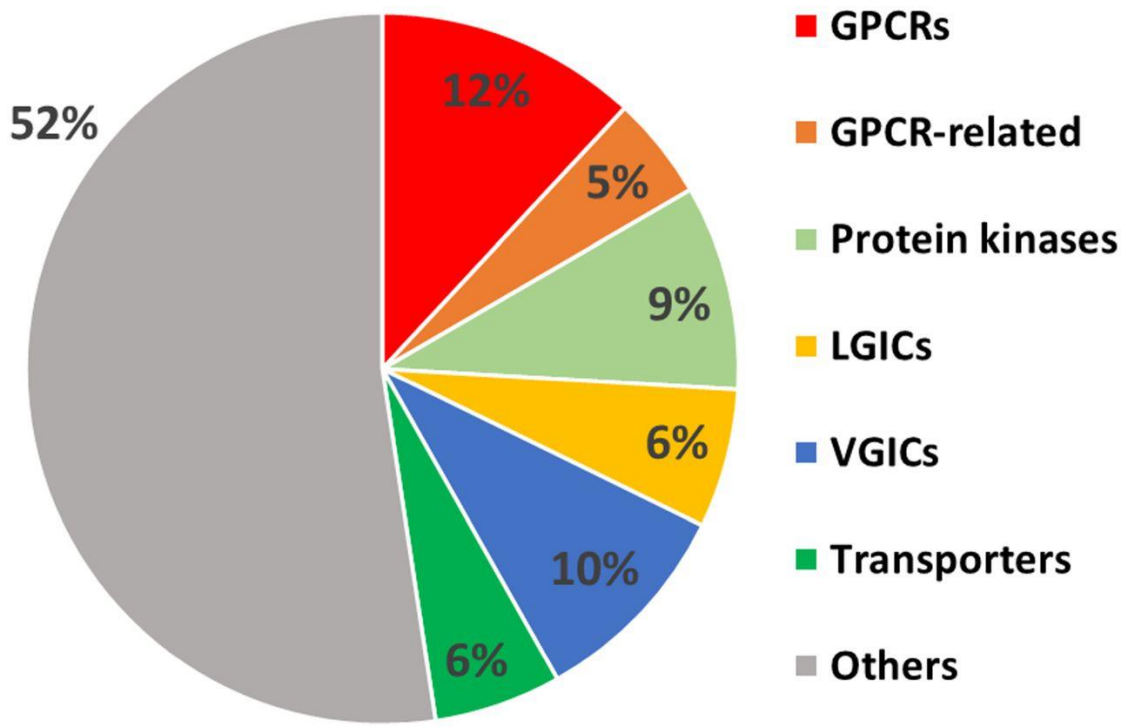
- Seed Financing: Led by Eli Lilly and Company ($10M), joined by BeyondSpring Inc. ($6M)
- Additional Eli Lilly R&D collaboration on multiple targets, with upfront $10M and up to $780M milestone payments, and tiered sales royalties.

Built Infrastructure and Organization

- 10,000 lab and office space in King of Prussia, PA
- Scientific team with precious experience of >40 IND filings and >12 drug approvals.

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

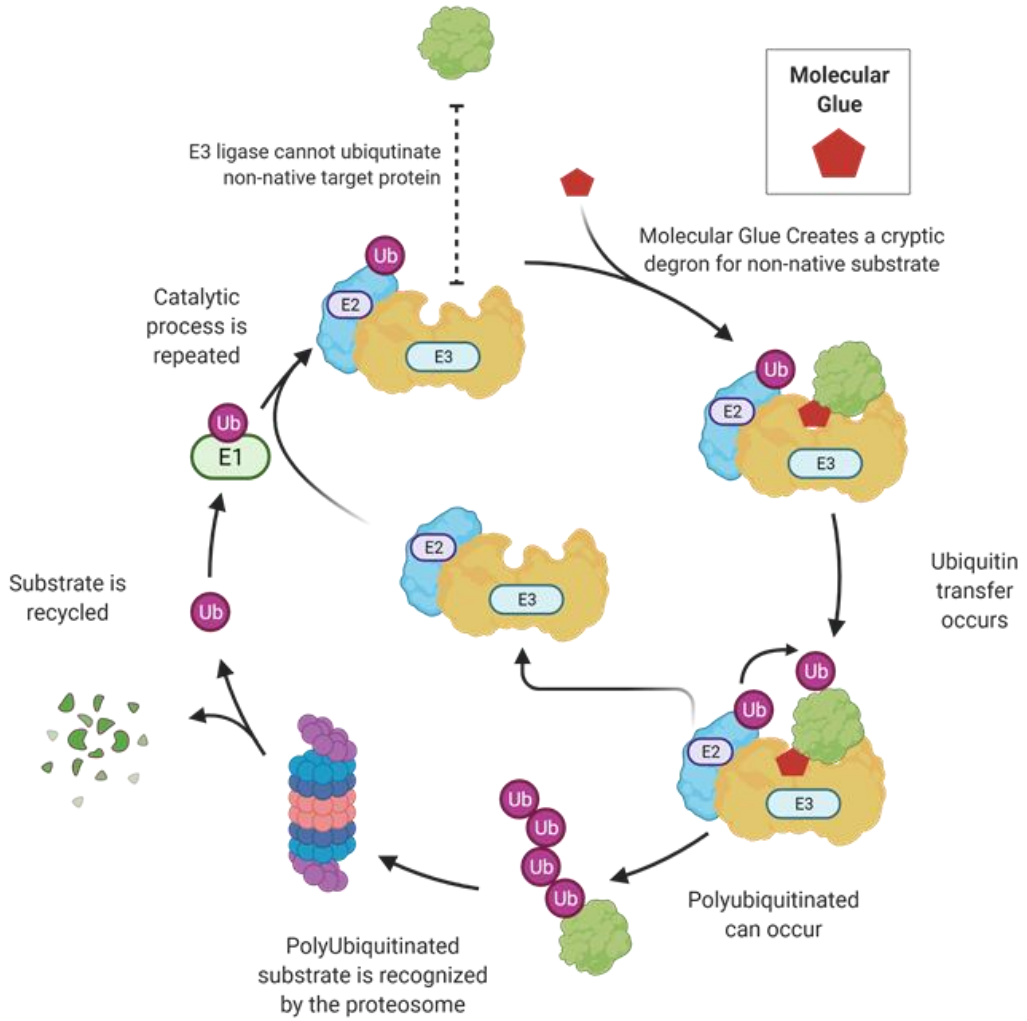
Druggable Proteins



TPD for Undruggable Proteins

https://doi.org/10.1124/mol.117.111062

Nature Derived TPD Molecular Glue Approved To Degrade Cancer-causing Proteins



Normal cell protein regulation via proteasomal degradation

- Natural method for selective protein degradation
- Nature derived IMIDs are approved molecular glues that hijack E3 ligases to degrade cancer causing proteins

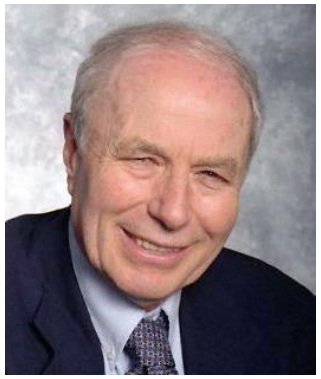
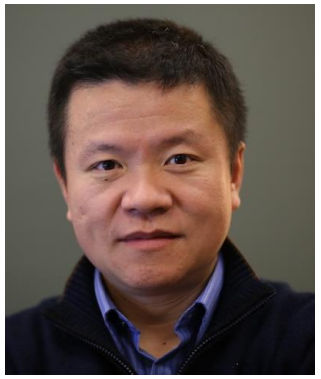
SEED opportunity to capitalize on Nobel Prize winning biology to bring de novo designed molecular glues to patients

Avram Hershko, MD, PhD
The Nobel Prize in Chemistry 2004
Prize motivation: "For the discovery of ubiquitin-mediated protein degradation."

SEED Therapeutics Co-Founder and SAB member

Strong Scientific Foundation With Proven Execution Capability to Timely Deliver Drug Pipeline for Multiple Unmet Medical Needs

World-Leading Scientific Expertise → **Translate to Drug Pipeline**

Avram Hershko MD, PhD	**Ning Zheng, PhD**	**Michele Pagano, MD**	**Lan Huang, PhD**	**James Tonra, PhD**
				
Pioneer in the ubiquitin proteasome system **Nobel Prize Recipient**	Pioneer in Molecular Glue discovery and scientific structural rationale	World leader in the discovery and application of ubiquitin ligase biology and cancer biology	Ubiquitin ligase expert and proven biotech entrepreneur at BYSI and Seed	Translating Science into the Clinic at Regeneron, Millennium, ImClone, Kadmon, and BYSI
SEED co-Founder and SAB Member	SEED co-Founder and SAB Member	SEED co-Founder and SAB Member	CEO, SAB Member and co-Founder	President and CSO

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 7 disclosed programs in various disease areas

Project X: POC in cell and animal models; lead candidate advancing to 2024 IND

Nov. 2020: SEED received investment and entered into a research collaboration and license agreement with Eli Lilly on targeted protein degradation.

Jun. 2022: Received additional investment payment

Feb. 2023: Received milestone payment for 1st R&D target

2H 2023: project to earn additional milestone payments for significant advancement for partner targets

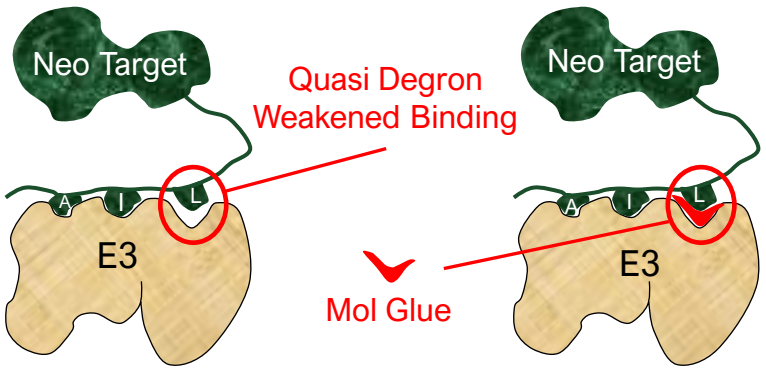
Eli Lilly Partner Program Milestones

How SEED Differentiates Itself in Surmounting Challenges

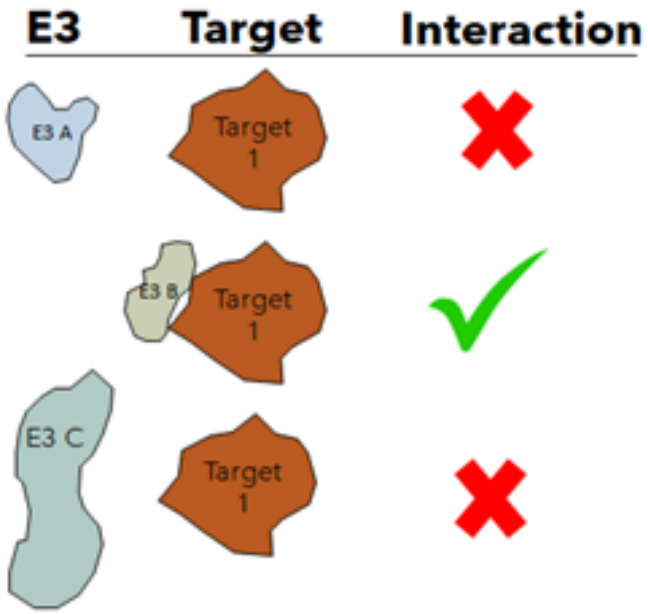
Unique Challenges	How to identify the right E3 for protein of interest (POI)?
Target-Centric Differentiation	Identify the right E3 for POI to increase the success rate in drug discovery: Platforms using **Quasi-degron, LumID, and Basal Affinity**
Pioneering Effort	World-leading scientific founding team, Talent acquisition, infrastructure development, intellectual property expansion and platform discovery
Two Prong Approach	De-risked revenue model: R&D partnership and internal program development

SEED's Multi-dimensional, Proprietary Platform for E3 Selection

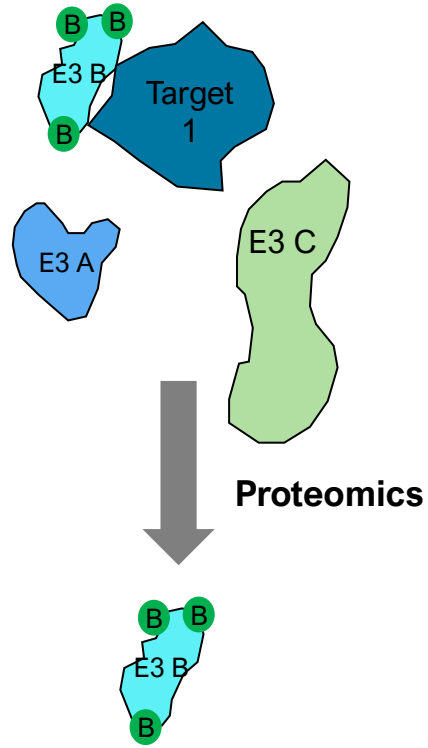
Knowledge-based Quasi Degron Approach



Direct Binding Validation

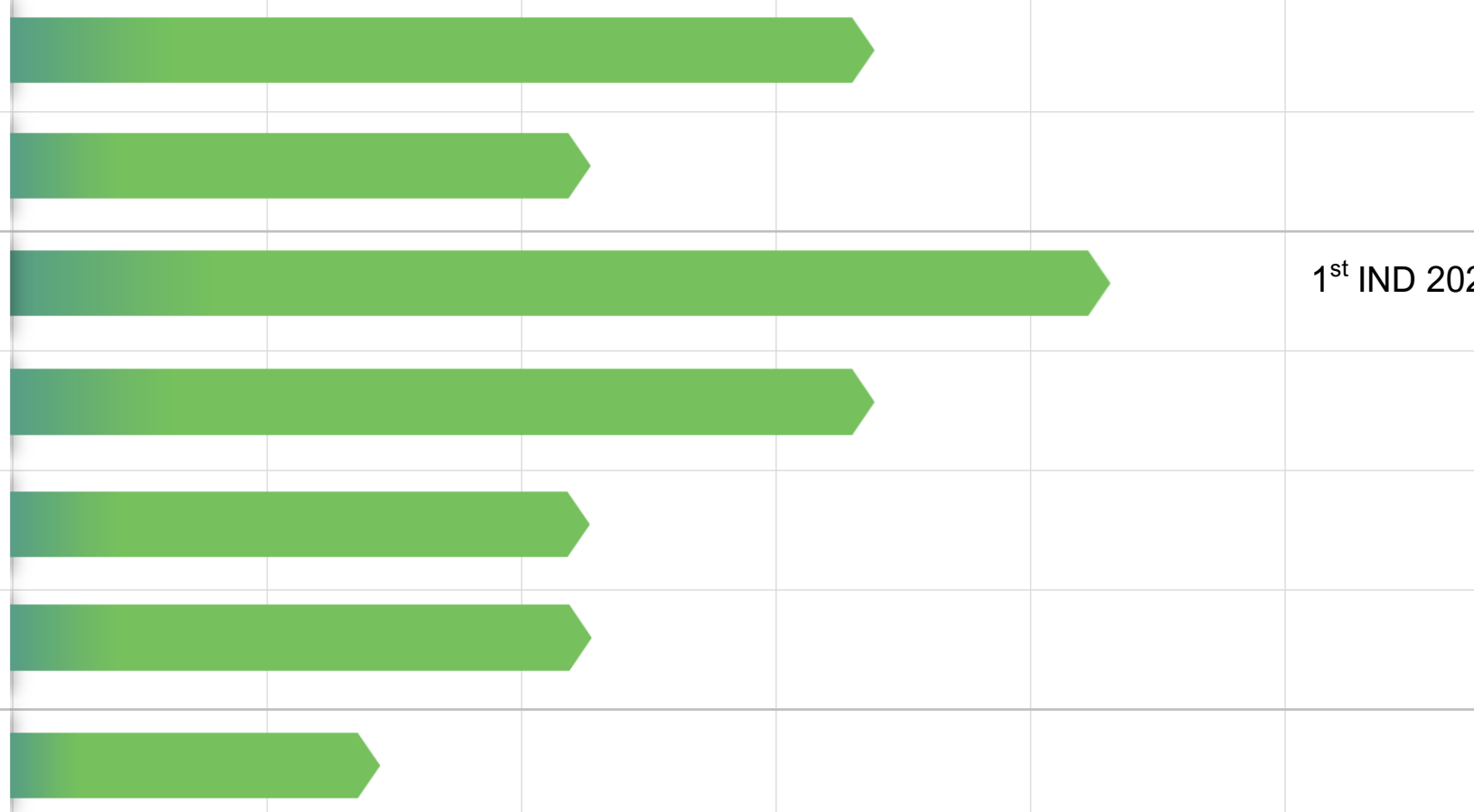


LumID: Identify the right E3 in the "living cell" near protein of interest



7 Drug R&D Pipeline in Multiple Disease Areas

Indication / Target	Target Protein Initiative*	Target Selection	E3 Ligase ID	Molecular Glue HTS	Lead ID	IND Candidate	IND Filing
NeuroD	Target Alpha						
	Tau						
Cancer	Project X						1st IND 2024
	KRAS-G12D						
	Target Beta						
	FEN1						
Antiviral	HBx						



* 6 of 7 Targets Unpartnered

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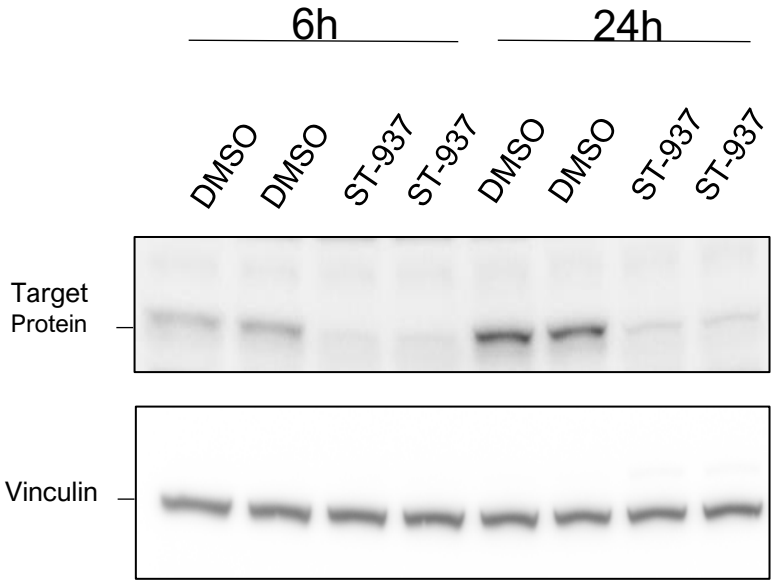
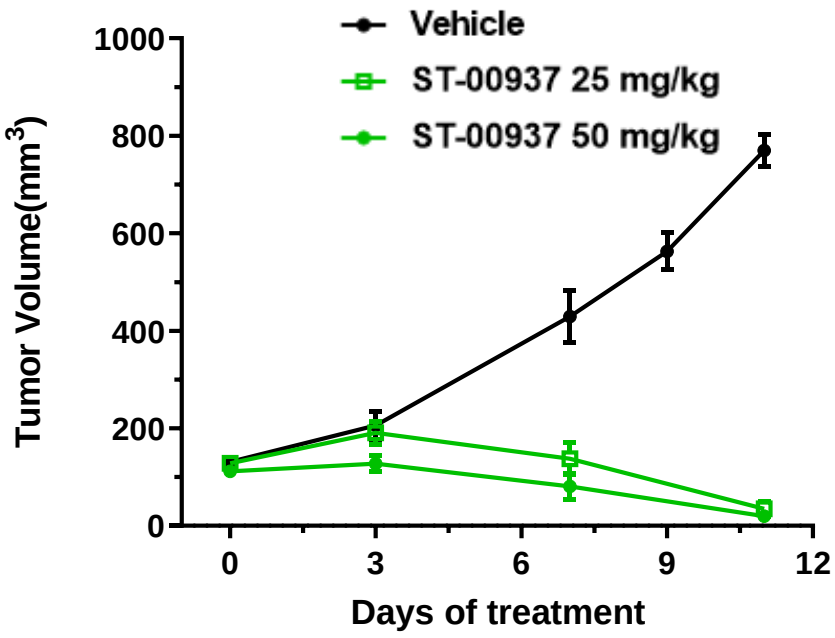
Project X: Lead ST-00973 Expected to Enter IND in 2024

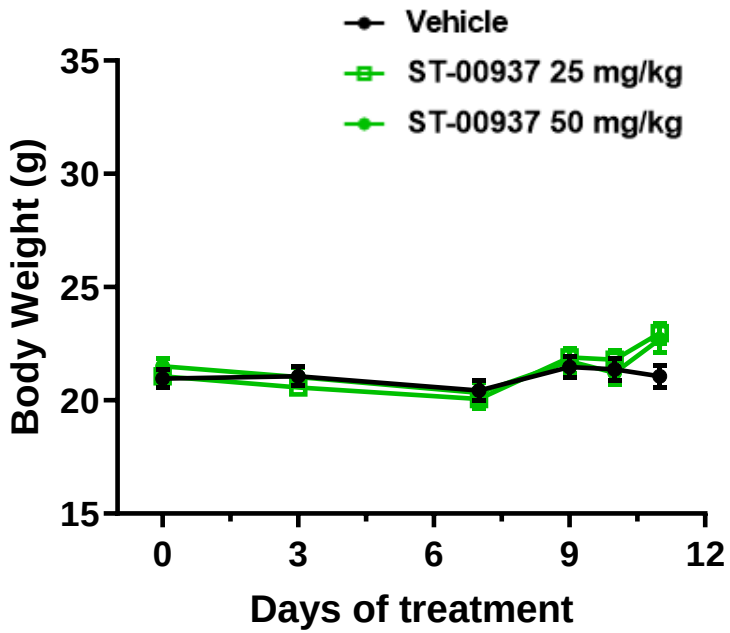
POC: Degradation of Target protein in Cell



Superb In Vivo Efficacy: Complete Tumor Regression

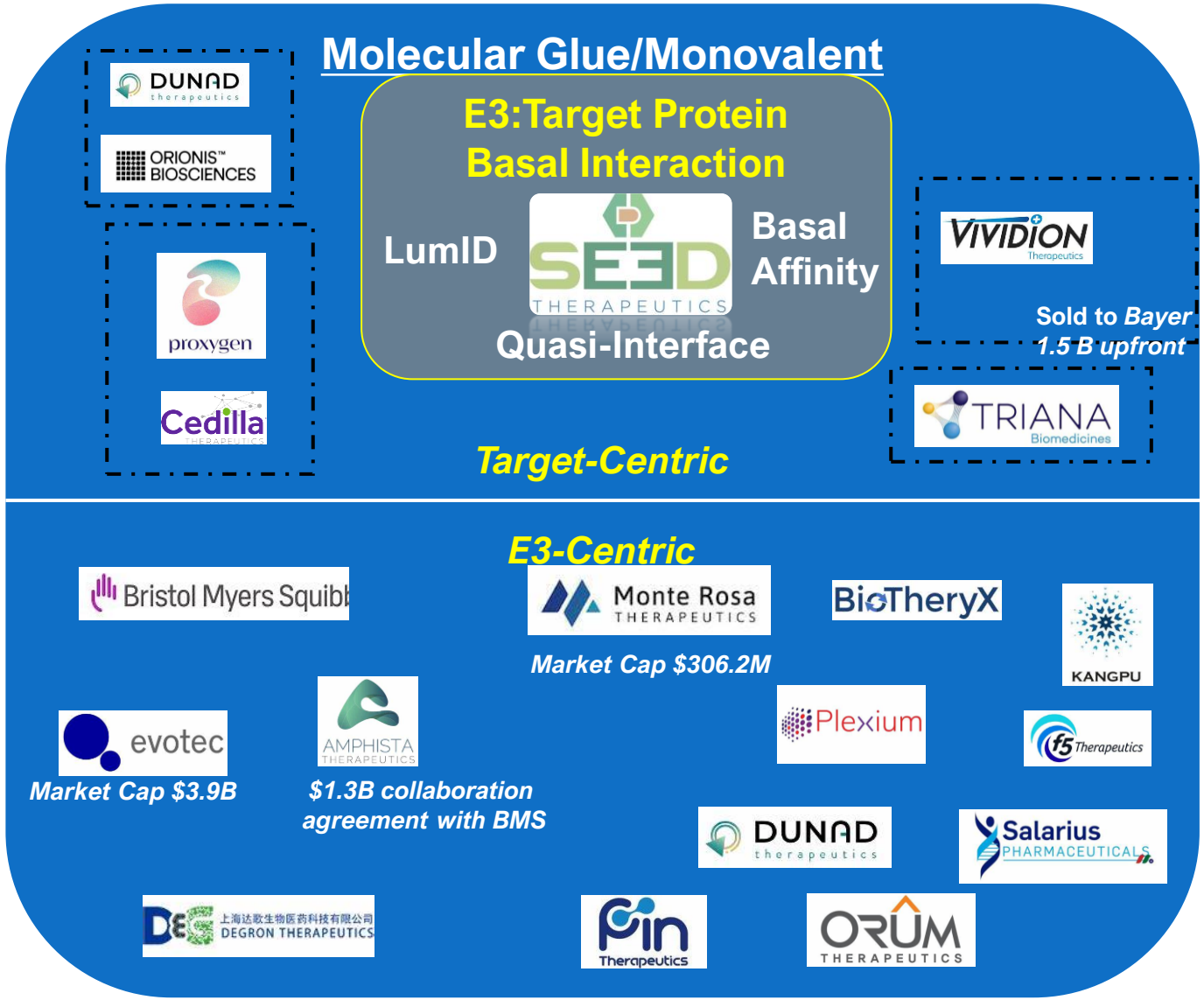


Good In Vivo Safety: No Weight Loss



Human colorectal cancer cell line, colorectal xenograft in immunodeficient mice (Oral dose, twice daily)

SEED (TPD 2.0) - Differentiated among High-value TPD Companies



Pharma Companies Licensing Deals in TPD Assets

Discovery:

- Deal Size
  - Upfront: $40 - $415 M
  - Milestone: $500 M to $5 B
- Collaborators:
  - BMS & Evotec
  - Genentech & Jemicare
  - Roche & Vividion
  - Sanofi & Nurix

Pre IND Stage:

- Deal Size:
  - Upfront: $100 - $300 M
  - Milestone: $1.3 B
- Collaborators:
  - Eli Lilly & Foghorn
  - GSK & IDEAYA

Summary: First to Market and Best in Class

1. **Target-centric SEED Breakthrough TPD Platforms Employ Multi-dimensional Proprietary Platforms for E3 Selection**
   - 80% of disease-causing proteins are undruggable, presenting a tremendous medical opportunity for SEED's new drug development platform;
   - With over 600 E3 ligases to choose from, SEED's platforms will identify and validate molecular glues with high probability of success to accelerate towards clinical testing, and to realize commercial potential in multiple disease areas, including cancer and neurodegeneration.

2. **SEED Breakthrough TPD Platforms Assure Successful Translation to Robust Pipeline with Near-term Value Creation**
   - Development candidate selected for expected 2024 IND;
   - Currently disclosed 7 programs in multiple disease areas.

3. **SEED is among the Leaders in the Industry in MG R&D for Undruggable Targets for Unmet Medical Needs**
   - Have exceeded seed financing expectations in talent attraction, infrastructure development, intellectual property, platform discovery, partner milestone achievement, and drug pipeline growth and advancement towards clinic;
   - Unmatched team of seasoned, highly motivated experts: Founders, Drug Developers, BOD.

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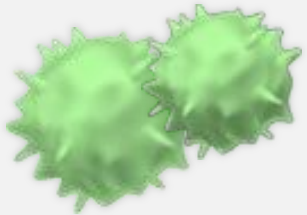
Plinabulin: Chemotherapy-Induced Neutropenia (CIN) Prevention Indication

CIN: Unmet Medical Need in Low White Blood Cell Count (depth of Neutropenia) in Week 1 After Chemotherapy

Despite widespread G-CSF use, CIN is #1 reason for FN, ER visits, hospitalization, sepsis, mortality, and chemotherapy dose reduction and disruption[1]

Short-term

G-CSF can reduce the duration of severe neutropenia, **but not depth of neutropenia**, which still happens in week 1 after chemo use.



Patients less Protected in week 1 after Chemotherapy with G-CSF

Long-term

Chemotherapy's anti-cancer effectiveness is linear to its dose exposure. **The objective to prevent CIN is to sustain optimum dose of chemo.**

Reduction in Relative Dose Intensity (RDI) of Chemotherapy

Reduction in Overall Survival[2]

The Unmet Medical Need: Neutropenia Depth or Low ANC Nadir in Week 1, "Neutropenia Vulnerability Gap" (NVP)

- >75% clinical complications occur in week 1 after chemo, which G-CSF cannot protect;
- The depth of severe neutropenia has not been improved by G-CSF, which Plinabulin can Improve.

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Source: 1 Lalami Y, Klastersky J.. Crit. Rev Oncol Hematol. 2017; 120:163-179. 2 Bonadonna G, Valagussa P, Moliterni A et al. Adjuvant cyclophosphamide, methotrexate, and fluorouracil in node-positive breast cancer. N Engl J Med 1995;332:901-906.

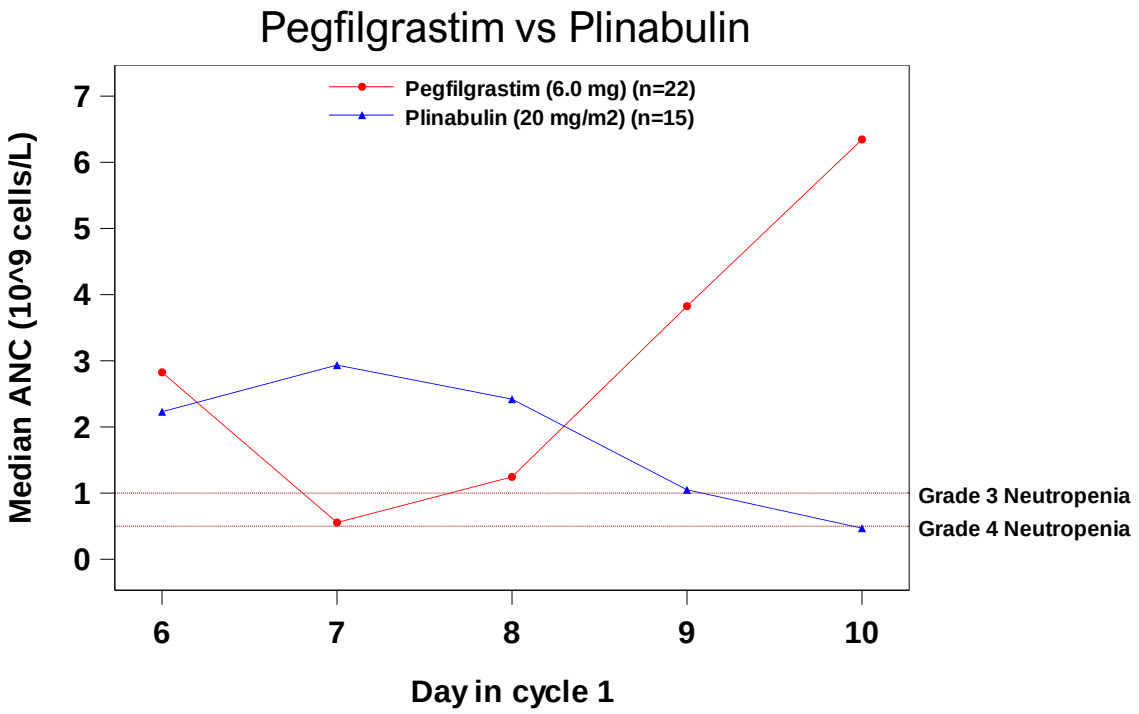
Six Clinical Studies Confirm Plinabulin's Neutropenia Prevention Benefit

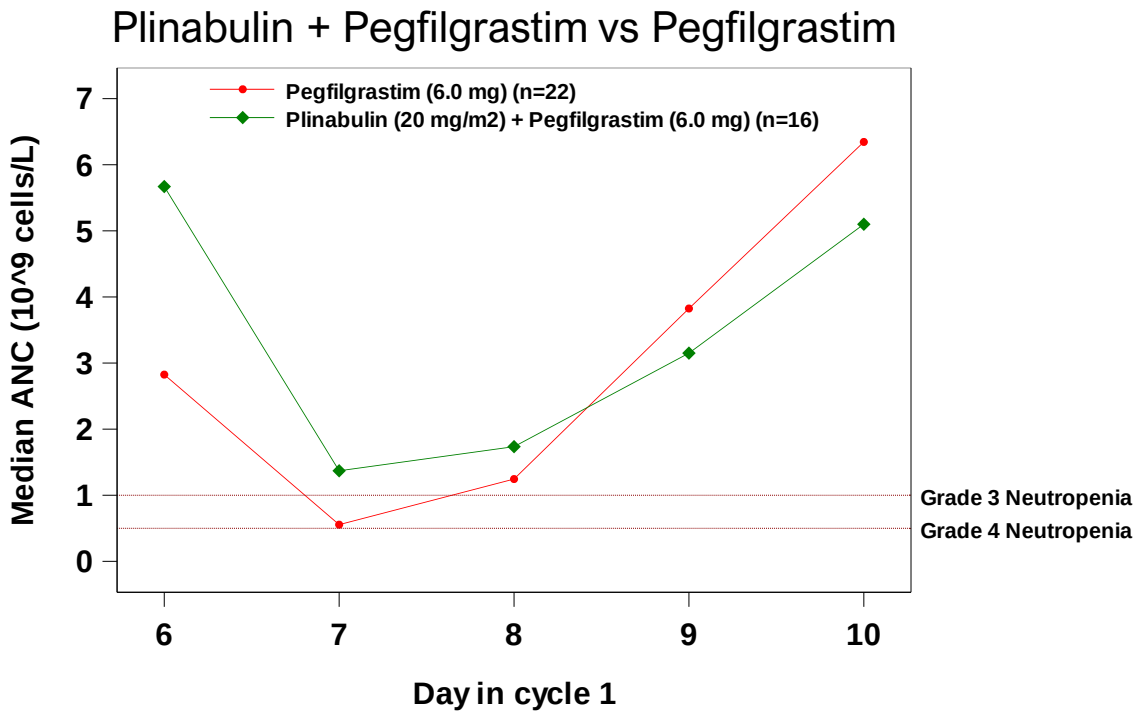
Plinabulin enables more use of chemotherapy

	ANC-based Benefit	Clinical Benefit
Study 101 (Phase 2) - Docetaxel, NSCLC	**Grade 3/4 Neutropenia (C1D8):** Plinabulin (n=90, 10% G-CSF): **7%** vs. Placebo (n=73, 30% G-CSF): **26%,** p=0.002	**Sepsis:** 0% Plinabulin vs. 3.6% Placebo **Severe Infection:** 0% Plinabulin vs. 3.6% Placebo **Docetaxel dose reduction:** 6.7% Plinabulin vs. 19.2% Placebo
Study 103 (Phase 3) - Docetaxel, NSCLC	**Grade 4 Neutropenia (SN, C1D8):** Plinabulin (n=278, 40% G-CSF): **5.3%** vs. Placebo (n=281, 50% G-CSF): **27.8%** (p<0.0001) **Grade 4 neutropenia in patients used G-CSF (C1D8):** Plinabulin + G-CSF (n=111): 7.21% vs. G-CSF (n=141): 38.3% (p<0.0001)	**FN:** 1.8% Plinabulin + G-CSF vs. 3.55% G-CSF **Infection with SN:** 0% Plinabulin + G-CSF vs. 6.38% G-CSF **More docetaxel exposure** in plinabulin vs. placebo **Overall survival (OS) extension**, double 2-year, 3-year OS rate in plinabulin vs. placebo
Study 105 (Phase 2) - Docetaxel, NSCLC	**DSN (Cycle 1):** Plinabulin (n=14): 0.36 day (non-inferior) vs. Pegfilgrastim (G-CSF): 0.15 day	**Bone pain:** minimum or no bone pain in plinabulin **Global QoL**: improvement in Plinabulin vs. G-CSF (p<0.001) **Platelet count decrease:** 0% Plinabulin vs. 35% grade 1 G-CSF
Study 105 (Phase 3) - Docetaxel, NSCLC, Breast, Prostate Cancer	**DSN (Cycle 1):** Plinabulin (n=52): 0.77 day (non-inferior) vs. Pegfilgrastim (n=53): 0.25 day	**FN:** 0% plinabulin vs. 1.9% G-CSF **Infection:** 7.7% plinabulin vs. 15.1% G-CSF **Bone pain:** less bone pain plinabulin vs. G-CSF (p=0.01)
Study 106 (Phase 2) - TAC, Breast Cancer	**Grade 4 Neutropenia (SN, Cycle 1):** Plinabulin + Pegfilgrastim (n=16): 38% vs. Pegfilgrastim (n=22): 59% **Median ANC Nadir (Cycle 1, x10^9 cells/L):** Plinabulin + Pegfilgrastim (Combo): 1.0 vs. Pegfilgrastim: 0.46	**Patients received >85% TAC treatment:** 100% combo vs. 82% G-CSF
Study 106 (Phase 3) - TAC, Breast Cancer	**Grade 4 Neutropenia (SN, Cycle 1):** Combo (n=111): 68.5% vs. Pegfilgrastim (n=110): 86.4%, p=0.0015 **Profound Neutropenia (PN, Cycle 1)** [1, 2]**:** Combo (n=111): 21.6% vs. Pegfilgrastim (n=110): 46.4%, p=0.0001	**FN**: 3.6% combo vs. 6.4% G-CSF **Duration of FN**: 1.3 day combo vs. 2.3 day G-CSF **Hospitalization for FN**: 3.8 day combo vs. 7.4 day G-CSF **Grade 4 Infection**: 0.9% combo vs. 3.6% G-CSF

PROTECTIVE-2 (106 Study, Phase 2): Rationale for the Combination

ANC in Cycle 1 After TAC for Breast Cancer
- Pegfilgrastim Unmet need in ANC protection in Week 1 - Plinabulin fills the Gap





The depth of severe neutropenia has not been improved by G-CSF, which Plinabulin can Improve.

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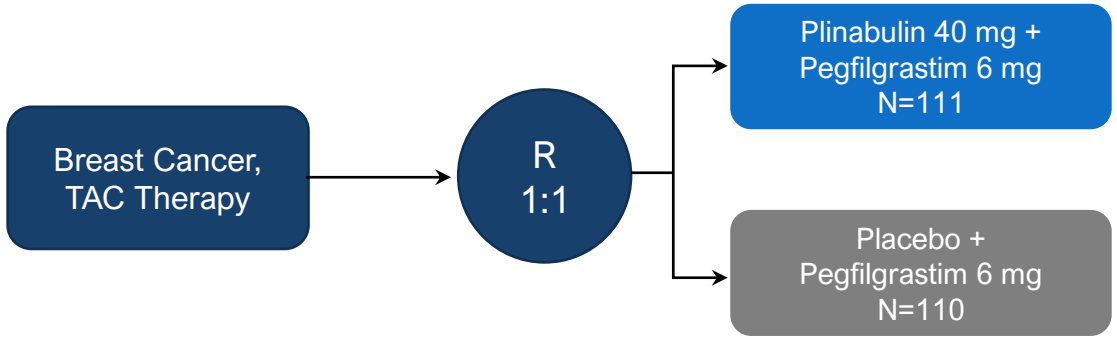
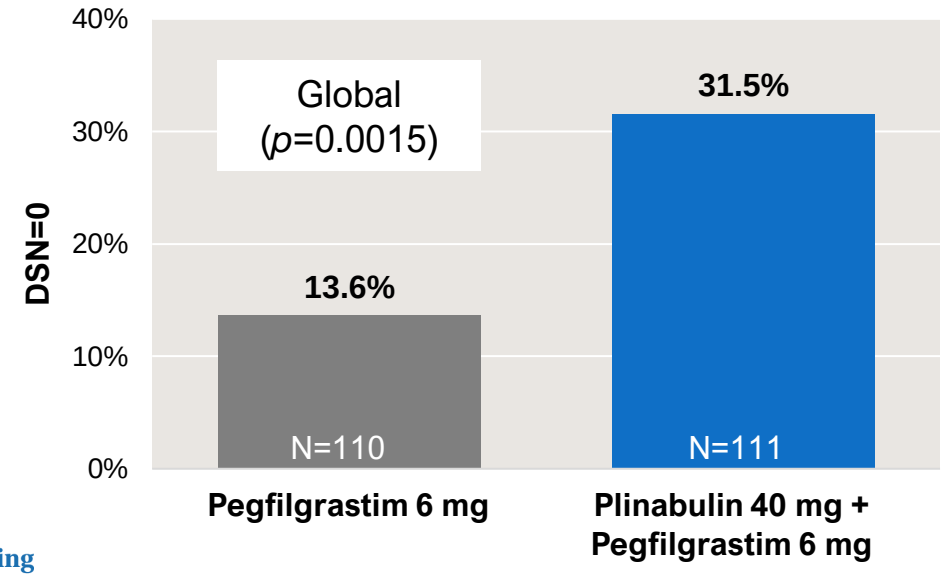
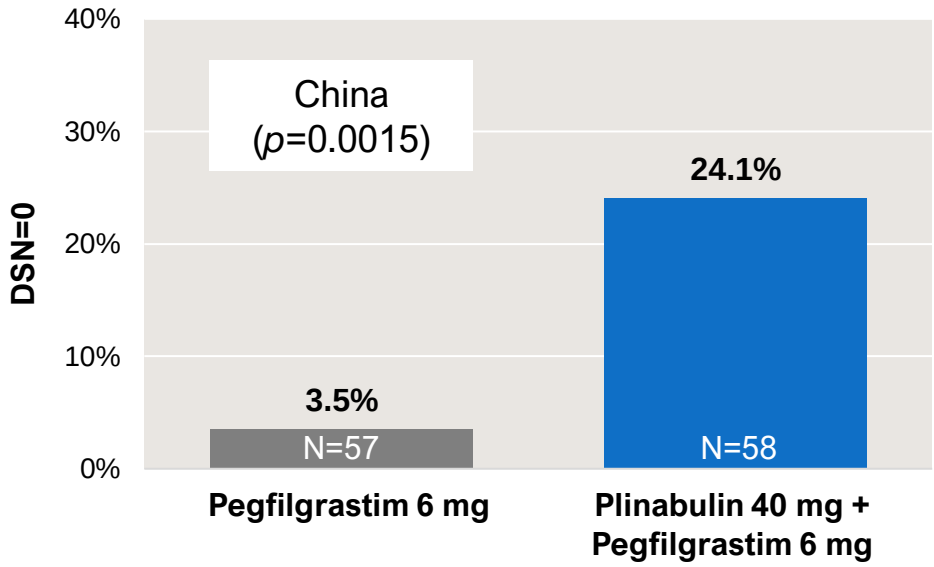
Met Primary Endpoint in PROTECTIVE-2 (106 Study) Phase 3

Design: Double blind, global study (19 centers); 4 cycles



Results: Proportion of Patients with NO Grade 4 Neutropenia (or DSN= 0 Days) in Cycle 1





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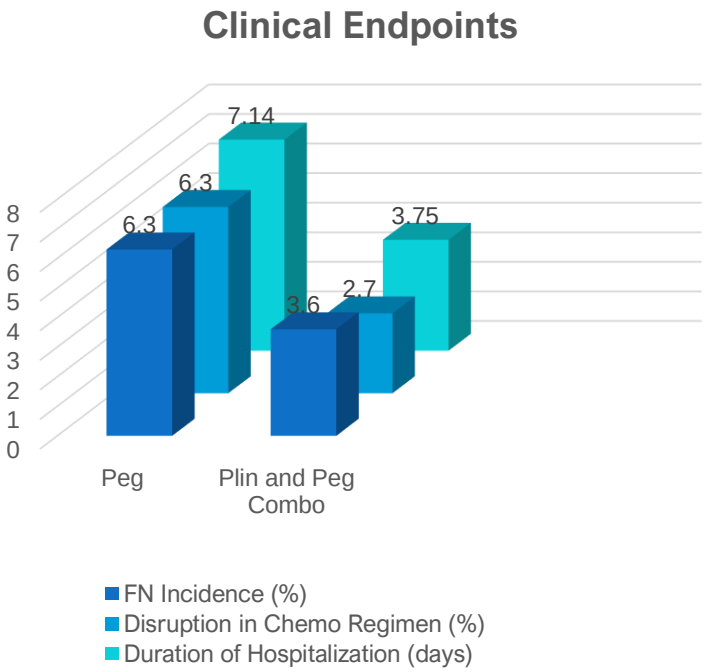
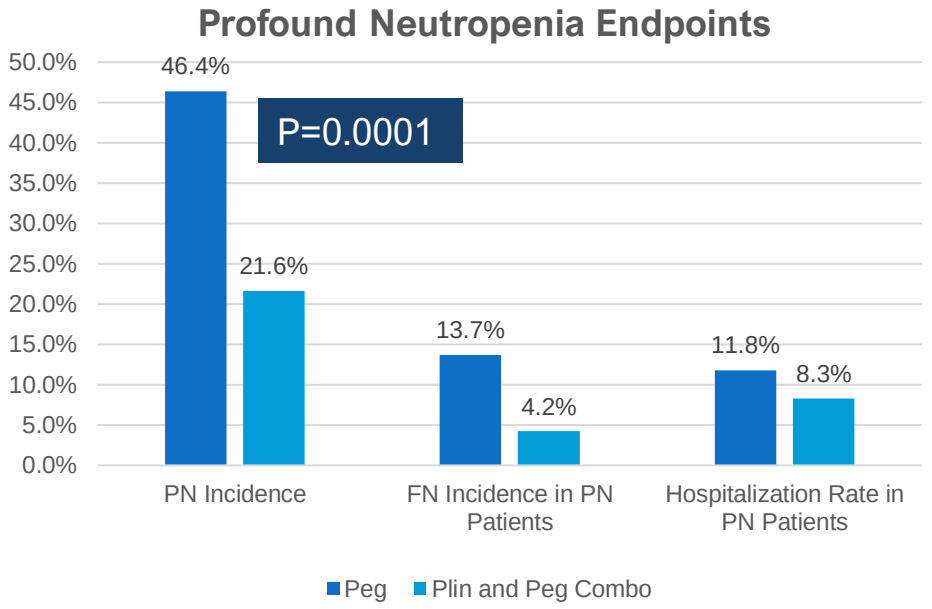
106 Phase 3: Superior Improvement Seen with the Combination in Clinically Meaningful Endpoints Compared to Pegfilgrastim Alone

Reduction of Incidence and Severity of FN and Hospitalization



Reduction of Profound Neutropenia (PN) Related Benefits

Plinabulin improves depth of neutropenia



Profound neutropenia (ANC < 0.1) is the key contributor to death from FN, which causes 15% death*.

June 2021 ASCO Presentations

*Moreira-Pinto et al. FN in Patients Undergoing Chemotherapy. Oncol Res Treat , 2020; 43:605-601

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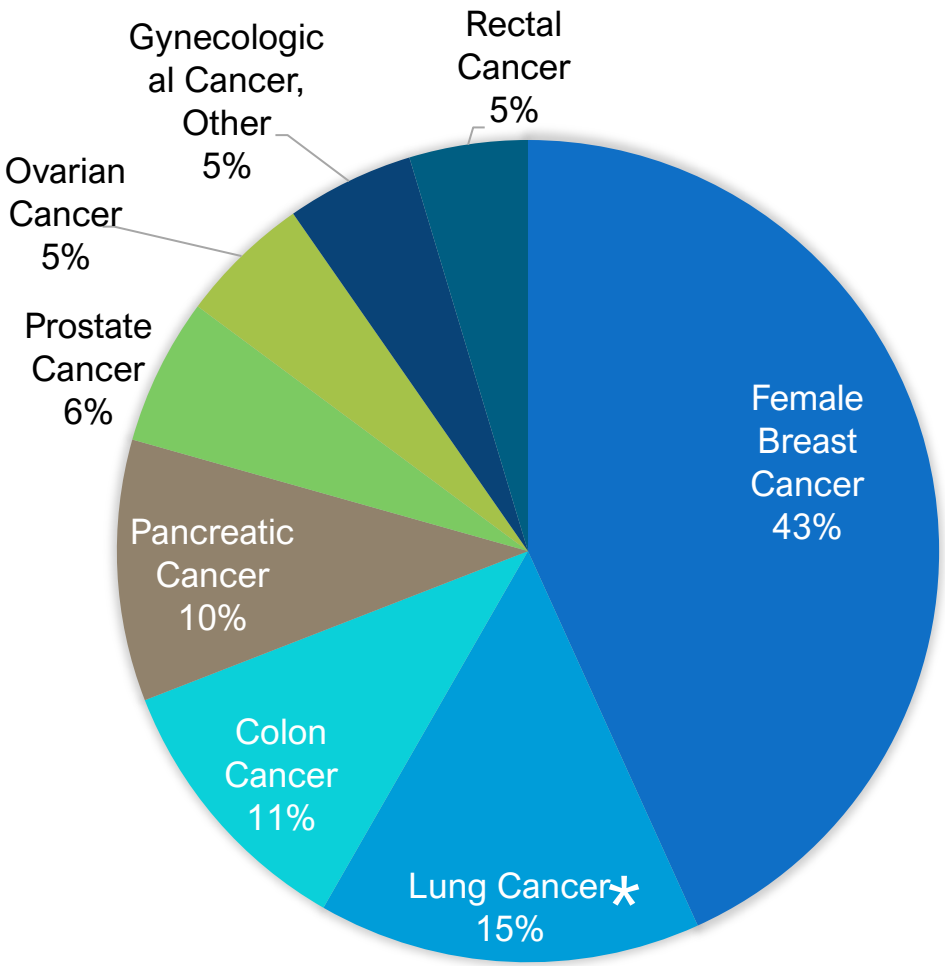
Potential for Use Across the Spectrum of Solid Tumors

G-CSF Administrations: Across Solid Tumor



Plinabulin Differentiated Benefit for CIN Prevention compared with G-CSF

- Improve depth of neutropenia;
- First day dosing;
- Limited bone pain and limited thrombocytopenia;
- Potential in combination with PD-1/PD-L1 inhibitors and chemotherapy regimen to prevent CIN and increase anti-cancer benefit

* SCLC ~15% of all lung cancer diagnoses

Source: IQVIA G-CSF Tumor Analysis; 7/19 – 6/20

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2nd/3rd Line NSCLC Indication

2L/3L NSCLC, no Driver Mutation: Docetaxel as Standard of Care (SOC)

With PD-1/PD-L1 moving to 1L, Docetaxel remains SOC for 2L/3L NSCLC

Pemetrexed and PD-1/PD-L1 Moved To First Line

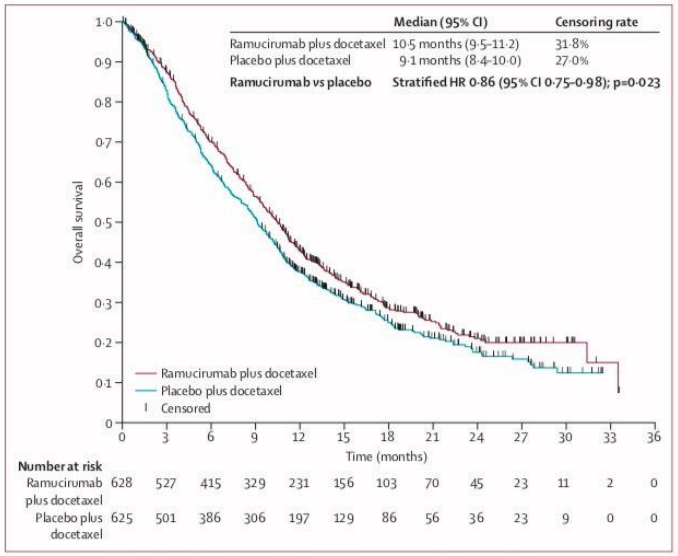
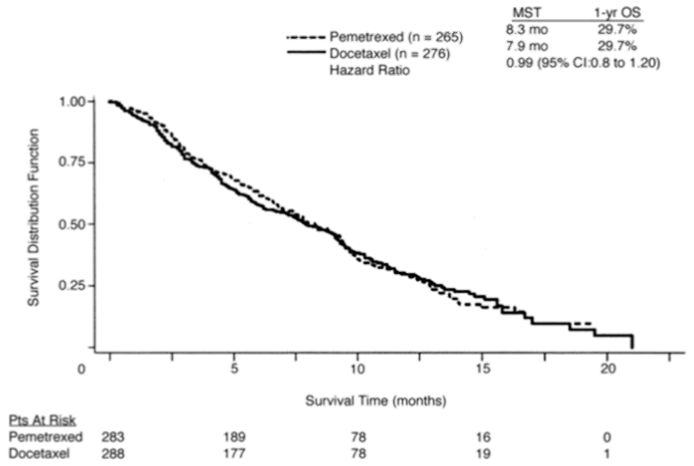
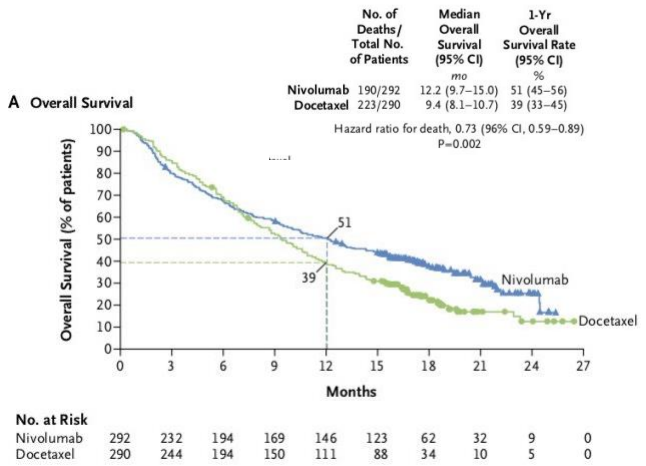


Figure 2: Kaplan-Meier estimates of overall survival in the intention-to-treat population
HR=hazard ratio.





Treatment	Ramuciramab + Docetaxel vs. Docetaxel[1]	Pemetrexed vs Docetaxel[2]	Nivolumab (PD-1 Ab) vs. Docetaxel[3]
Pros	Limited efficacy; HR for mOS: 0.86 (1.4 M mOS benefit vs. Docetaxel)	Low CIN risk	Improved efficacy; HR for OS: 0.73 (2.8 M mOS benefit vs. Docetaxel)
Cons	High CIN risk (49% severe neutropenia)	Low Efficacy, HR for mOS: 0.99 (no survival benefit vs. Docetaxel)	Moved to 1st line, current clinical data does not support its use in PD-1 failed/progressed patients

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[1] Lancet 384 (9944): 665-673 (2014). [2] JCO 22(9): 1589-1597 (2004)。 [3] NEJM 373: 1627-1639 (2015)。

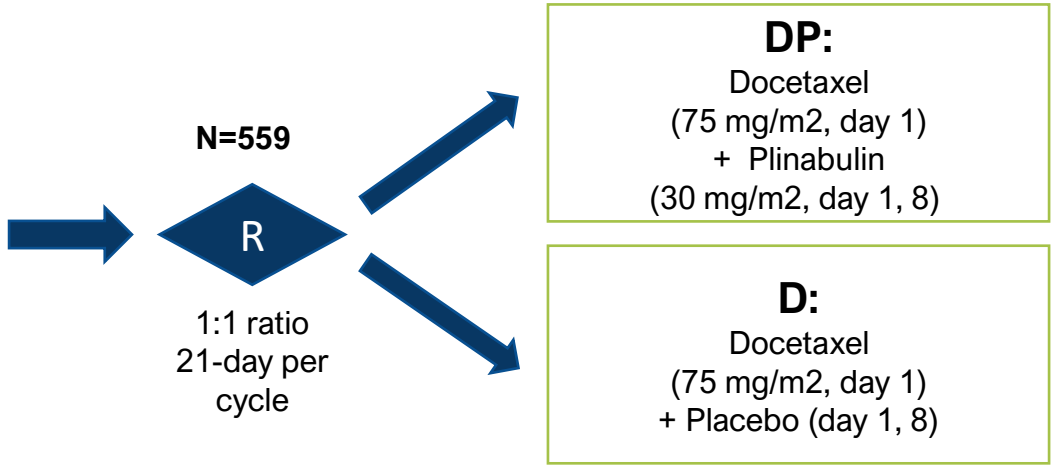
DUBLIN-3 (Study 103) Trial Design

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

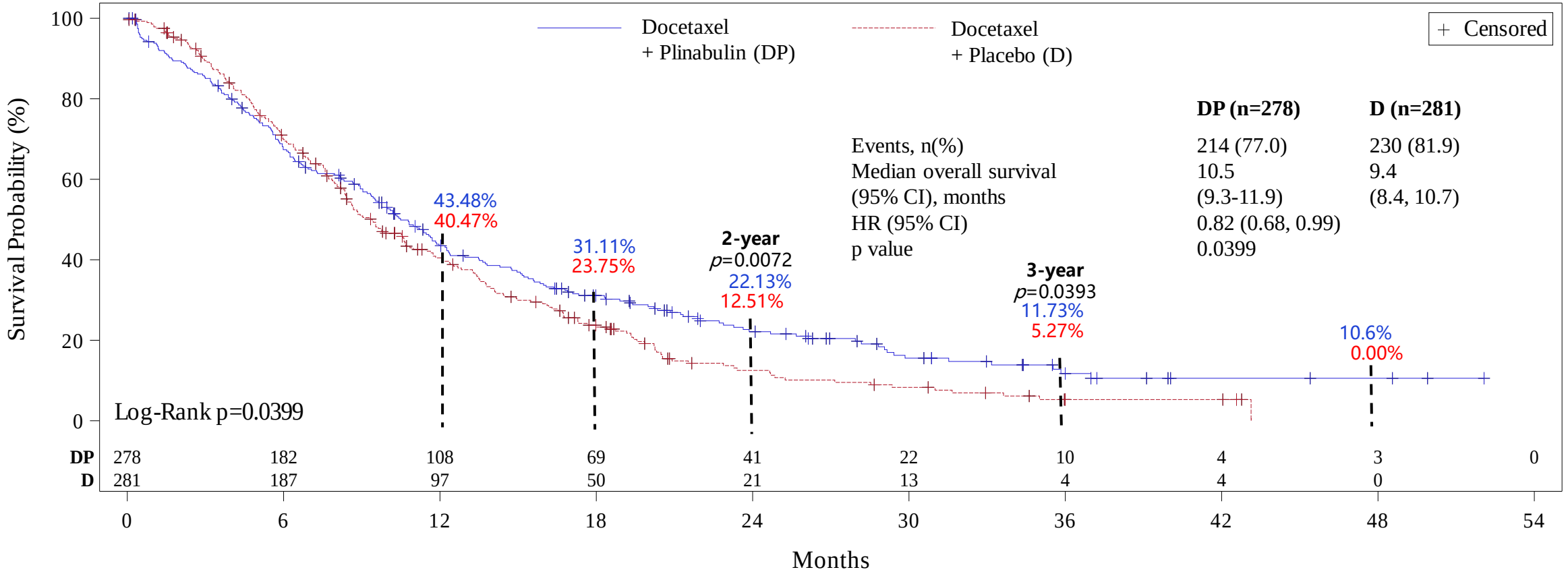
Study Plan:
- Global, randomized, single-blinded (patients only)
- Stratified by region (Asia/non-Asia), prior line(2L or 3L), ECOG (0-1/2), Prior PD-1/PD-L1 (yes/no)
- 58 sites across U.S., China and Australia
- CRO: ICON; LabCorp central lab for PK & ANC

End Points:
Primary Endpoint: OS
Secondary Endpoints:
- ORR, PFS
- Percent of patients without severe neutropenia on Day 8 of 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



Met Primary Endpoint of OS; Significantly Improve 2-, 3-year OS Rate



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

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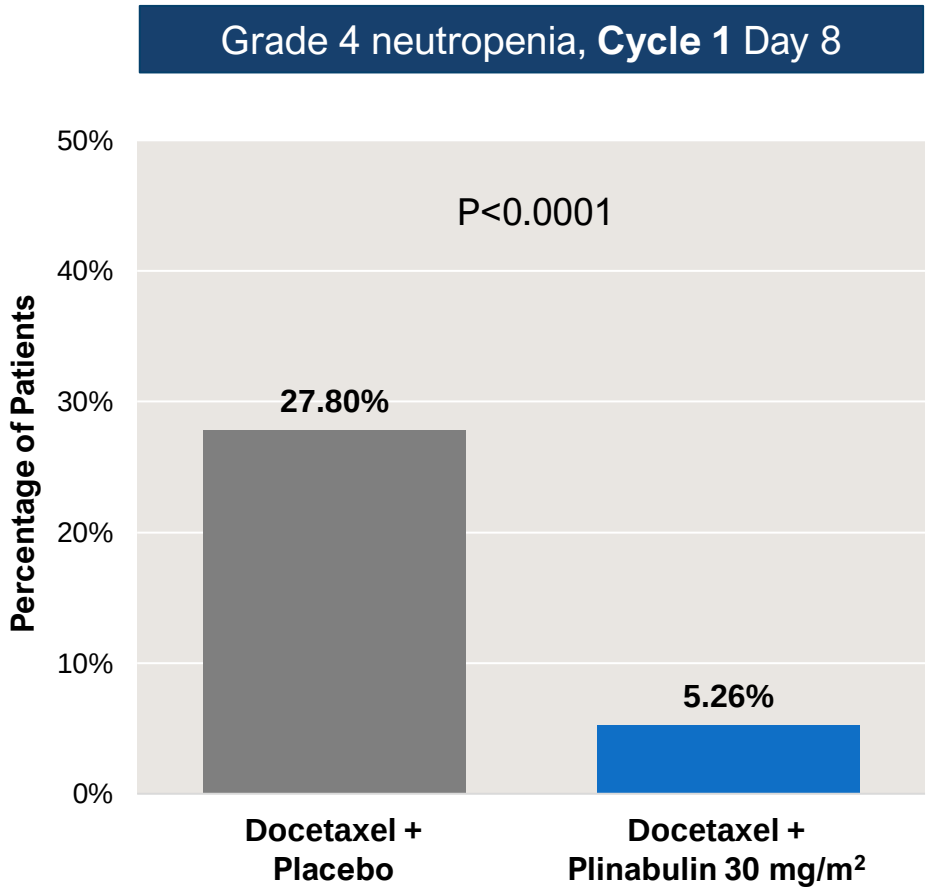
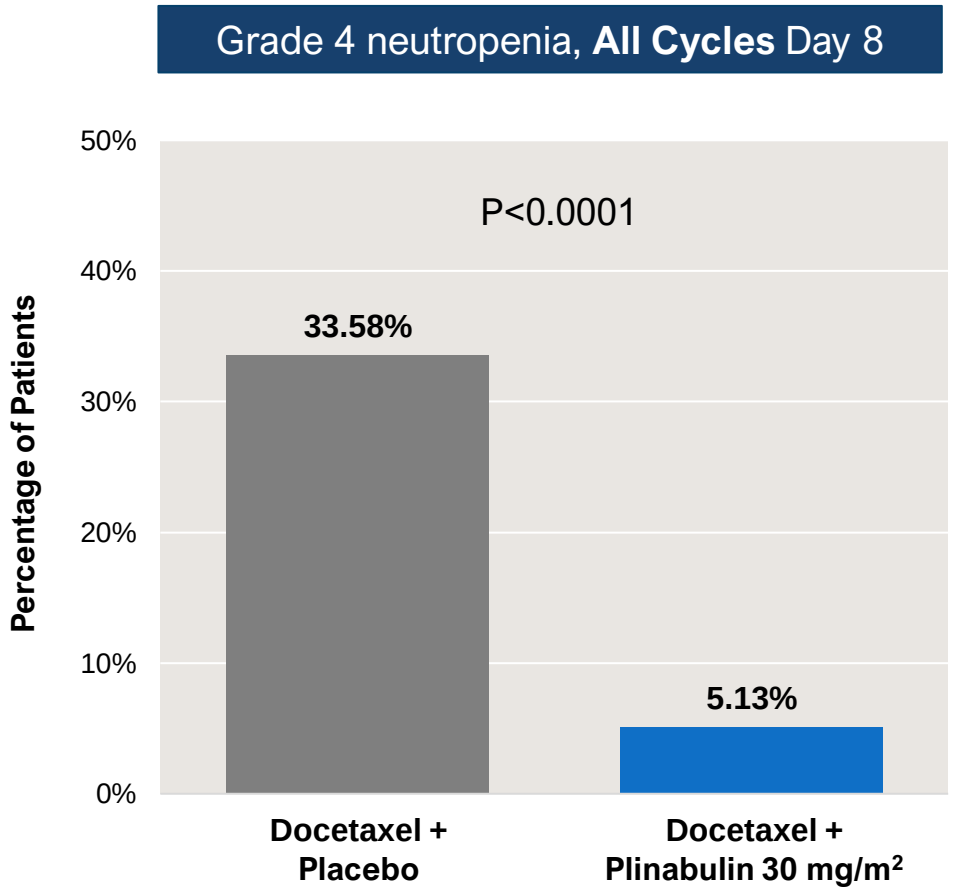
Significant Reduction in Grade 4 Neutropenia

Cycle 1 Day 8 and All Cycles Day 8

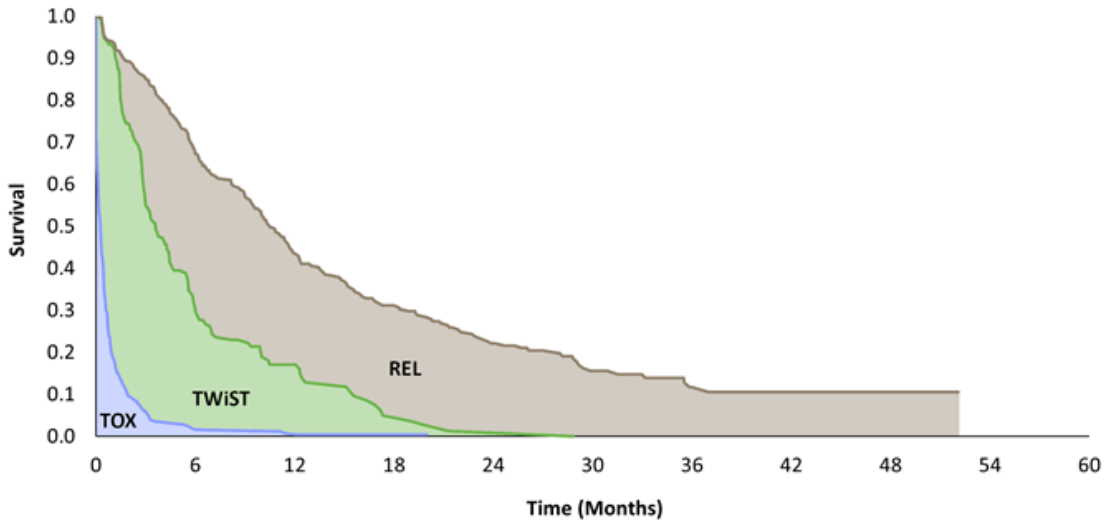




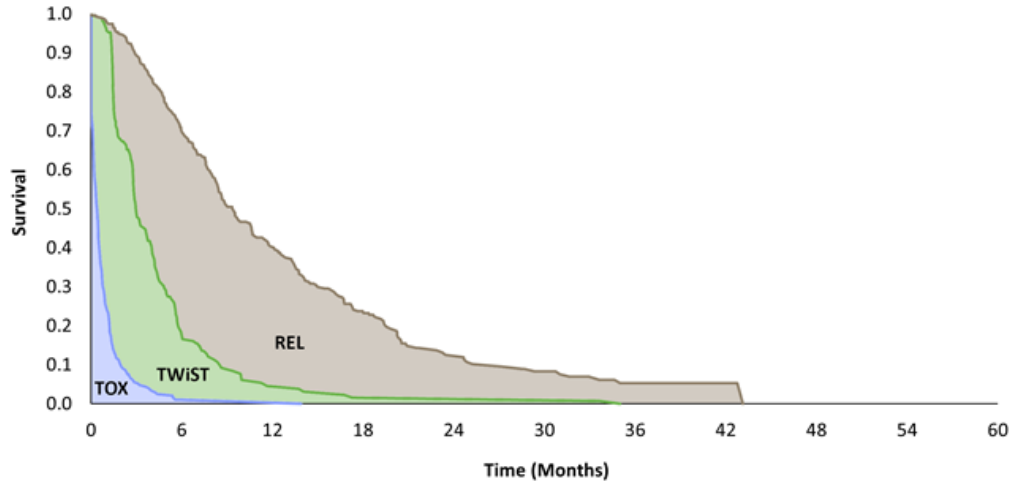
Significant Improvement in Quality of Life Benefit

- Q-TWiST (Quality-Adjusted Time Without Symptoms of Disease and Toxicity)

Plinabulin + Docetaxel



Twist alone



Q-TWiST Gain	Relative Gain to OS Restricted Mean	Relative Gain to Q-TWiST
1.93	15.11%	18.43%
	(1.72% to 30.63%)	(2.07% to 37.20%)
	p-value=0.0396	p-value=0.0393

Clinically Meaningful Improvement of >18% in Q-TWiST.

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Potential Benefit of Plinabulin + Docetaxel in 2L/3L NSCLC, No Driver Mutation

With PD-1/PD-L1 moving to 1st line NSCLC, plinabulin + docetaxel could be the potential choice.

Benefits compared to SOC Docetaxel:

Efficacy:

- Significant survival benefit in ITT (OS HR=0.82), with more pronounced survival benefit in non-squamous NSCLC (OS HR=0.76);
- Significant neutropenia reduction;
- Significant QoL benefit.

Safety:

- The regimen is well tolerated. Side effects include transient hypertension which resolves in 4-6 hours, nausea, vomiting and GI side effects.

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Immuno-Oncology Combinations

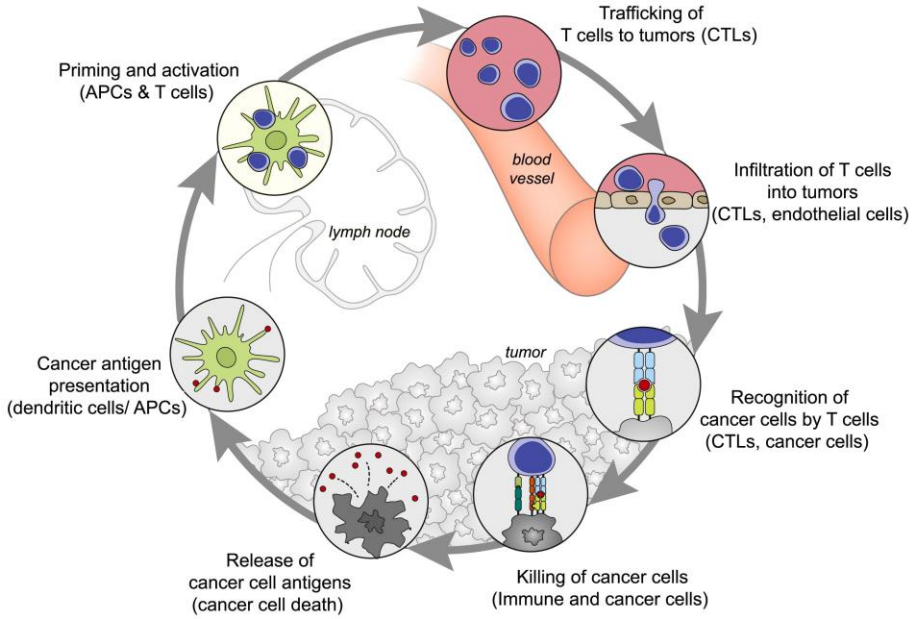
Mechanism with Broad Applications

Plinabulin Induces Dendritic Cell Maturation (the most potent APC), a Key Step in Initiating Anti-Cancer Durable Response

2 Plinabulin

Hit the Gas
Stimulates maturation of dendritic cells to increase antigen presentation

Dendritic cells are the most important antigen-presenting cells



3 Checkpoint Inhibitors

Release The Brakes
Optimize T cell response

1 Radiation/Chemotherapy

Release Tumor antigens
For more potent anti-cancer effect

1 + 2 + 3 = Optimal Immuno-Oncology Response

Clinical POC in Triple Plinabulin IO Combination Regimen

Indication / Target	Program	Preclinical	Phase 1	Phase 2	Phase 3	Collaborator
7 cancers (PD-1/PD-L1 progressed)	Plinabulin + PD-1/PD-L1 + Radiation					THE UNIVERSITY OF TEXAS MD Anderson Cancer Center
NSCLC (2L/3L, PD-1/PD-L1 progressed)	Plinabulin + PD-1 + Docetaxel					MERCK; PEKING UNION MEDICAL COLLEGE HOSPITAL

MD Anderson Phase 1 study clinical data and biomarker studies had been submitted for presentation at SITC conference in November 2023.

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

2/3rd Line: PD-1/PD-L1 resistant patients

1st Line: PD-1/PD-L1 + chemo double 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

Increase chemo dose and IO combo efficacy: Plinabulin + PD-1/PD-L1 + chemo

Turn "cold" into "hot" responding tumor: Plinabulin + PD-1/PD-L1 + chemo/radiation

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Regulatory Pathway

Regulatory Pathway

Near-Term

CIN: Ongoing discussion with NMPA in China and FDA in the US;

NSCLC: Ongoing preparation to file for NDA approval in China and the US.

Long-Term

Seek regulatory clarity and additional approvals in countries around the world.

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: 7 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