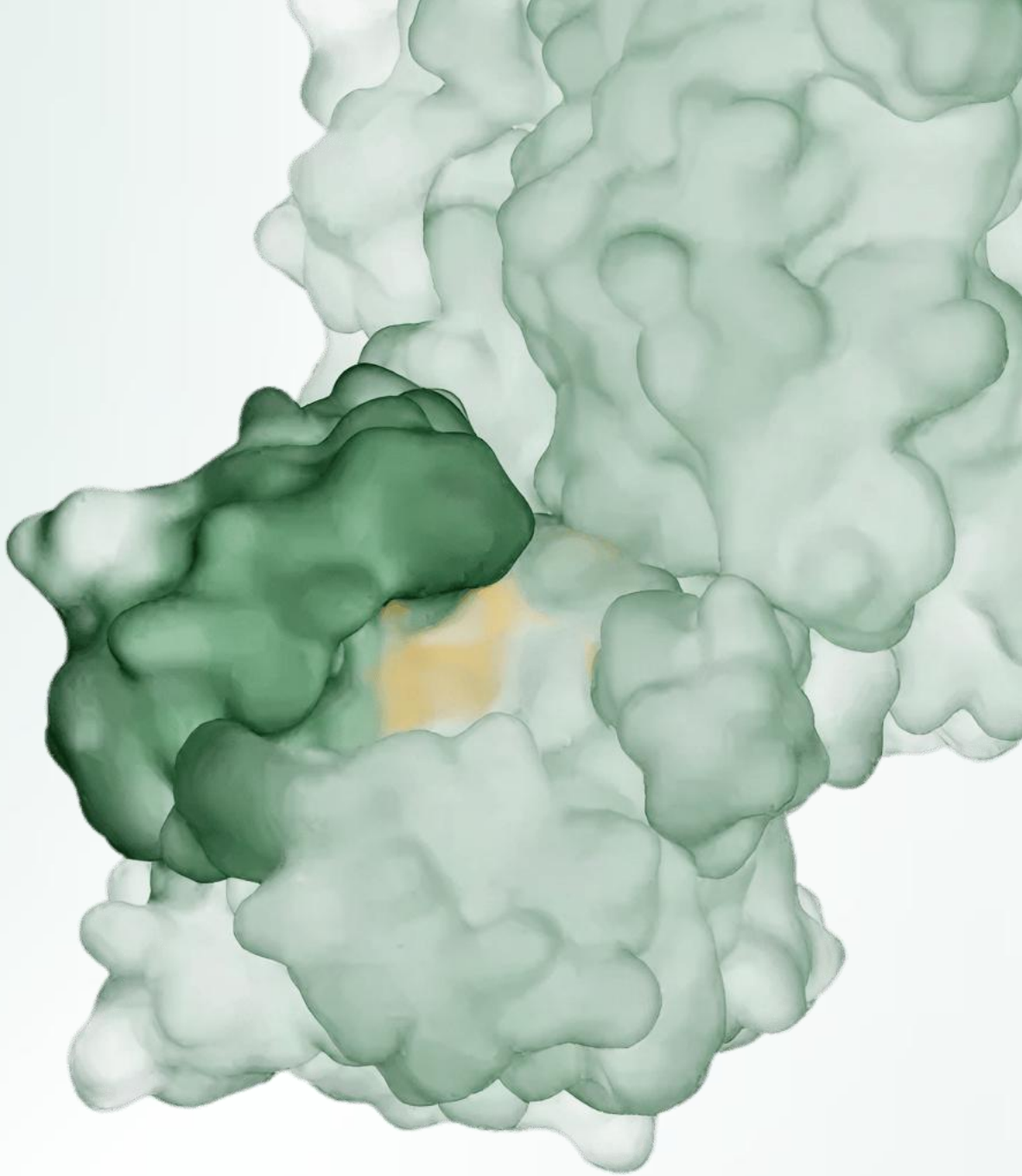
SEED THERAPEUTICS

Create the glue. Capture the protein. Eliminate disease.

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



January 2026

SEED: A clinic stage molecular glue company with validated pharma partnerships and near-term data

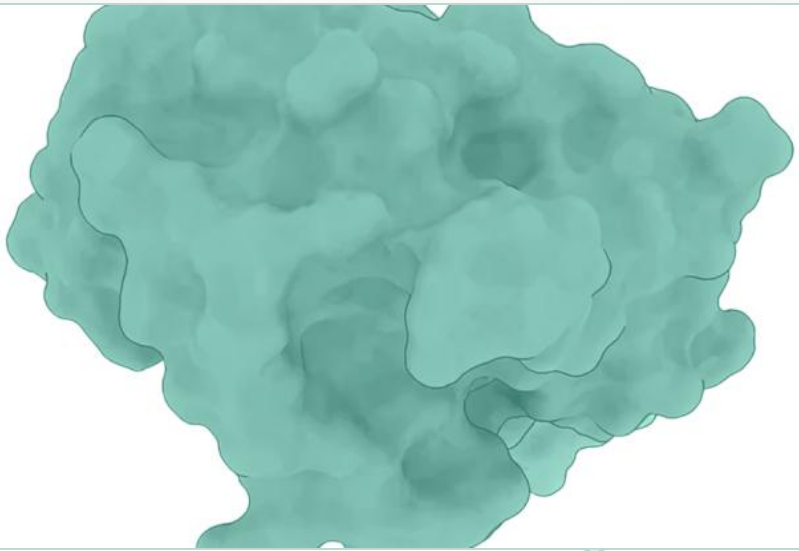
Targeted protein degradation (TPD) focused on developing novel “molecular glues”

TPD Potential	• Addressing 80% of disease-causing proteins considered “undruggable” by traditional methods
Technology Platform	• Target-centric: Differentiation in using novel E3 ligases among 640 E3s for protein of interest, featured as one of leading TPD companies by two Nature review articles in 2024 • R&D collaborations with Lilly and Eisai with potential value exceeding $2.3 billion plus royalties
Robust Pipeline	• 6 Key Programs (3 internal; 3 partnered) across oncology, neurodegeneration, and immunology • ST-01156, an RBM39 degrader (oncology): preliminary Phase 1 clinical data in 2H 2026 • Oral Tau degrader (neurodegeneration): current cell activity; in vivo PK expected in 2H 2025
Finance	• ~$60M in equity, collaboration upfronts, and milestones since inception • $30M Series A-3 closed in September 2025
World-class team	• World-class founding team: Co-founders are scientific leaders in TPD E3 ligase structures and ubiquitin biology, including Nobel prize Winner Dr. Avram Hershko.

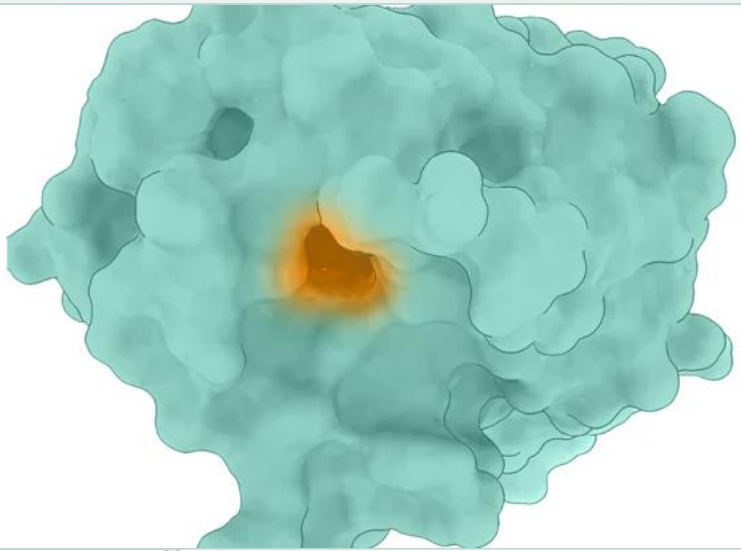
Modern medicine was built for proteins with druggable pockets. Most of disease-causing proteins do not have them

Small molecule inhibition depends on stable binding pockets. The majority of pathogenic proteins are structurally inaccessible, with cancers evolving faster than we can produce inhibitors.

Pocketless protein



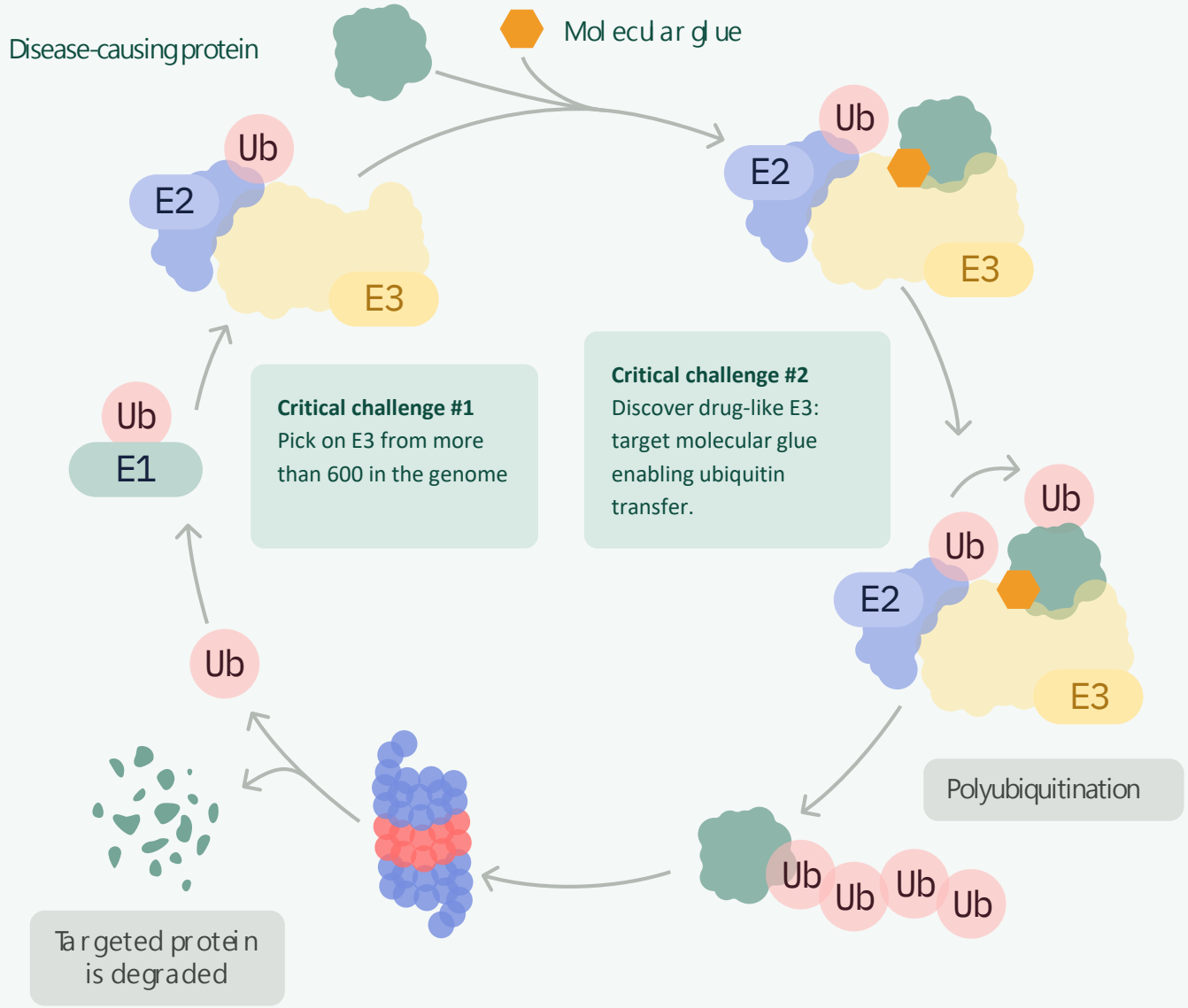
Pocketed protein



- **~80% of disease-causing proteins lack druggable pockets**
- **Most are intracellular, disordered, or fusion-driven**
- **Many mutate rapidly under selective drug pressure**

Elimination, not inhibition, is the next therapeutic paradigm

The ubiquitin-proteasome system (UPS) evolved to tag and destroy aberrant proteins with exquisite selectivity, compounded by molecular glues for precision



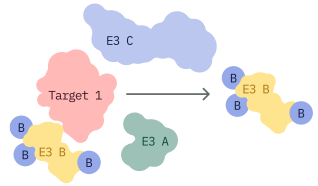
Molecular glues proved the modality, but the scientific scope is constrained

- Revlimid revealed that small molecules can induce targeted protein degradation in patients ($12.8B peak sales), before its mechanism was even understood.
- The industry defaulted to just two E3 ligases: Cereblon and VHL.
- Humans encode 600+ E3 ligases, yet the vast majority remain untapped by existing TPD approaches.
- This bottleneck essentially limits selectivity, scale & the full therapeutic reach of molecular glues.

SEED built the TPD 2.0 engine to find the optimal ligase and degrade any disease-causing protein

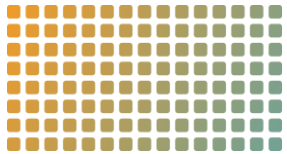
SEED's RITE3™ platform unlocks access to more than 600 ligases encoded in the human genome. Then designs custom molecular glues to bind them. More ligases. Better matches. Higher hit rates.

Pathogenic Protein

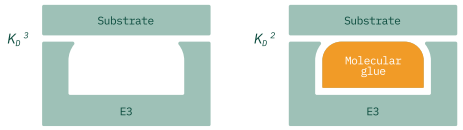


LumID™ Live-cell assays

Functional Degradation

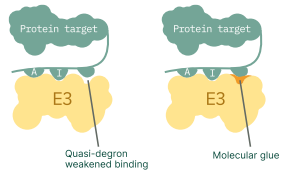


Screen and select across 600+ human ligases



Proteomics

proximity and tissue relevance



Structural Biology

ligase-target complementarity

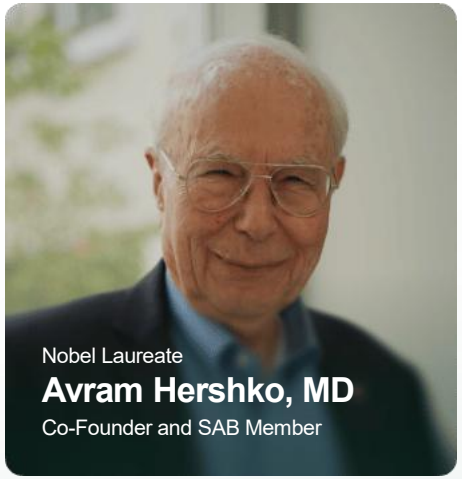
Design the Glue

>1% Hit Rate

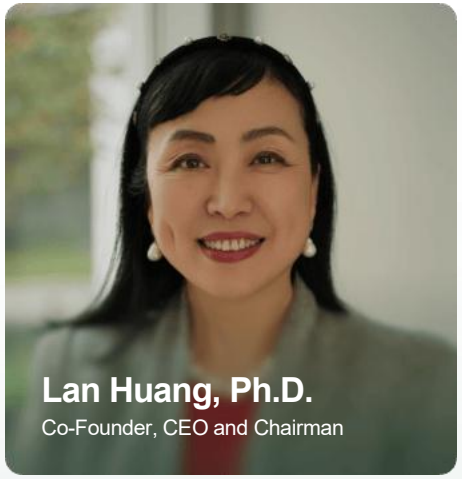
Eliminate Disease

SEED is led by the scientists who discovered targeted protein degradation

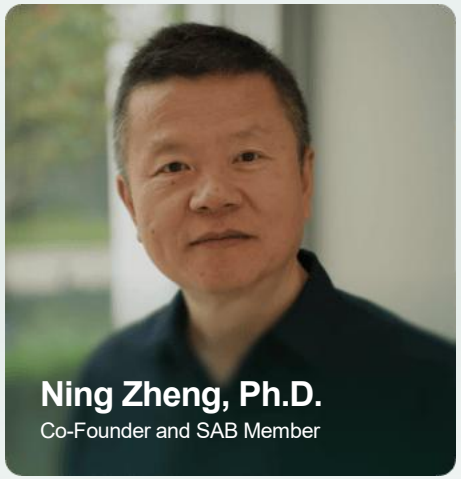
- Combined leadership record of 40 INDs and 12 NDAs
- Balanced by an experienced board across finance, risk, legal, and drug development



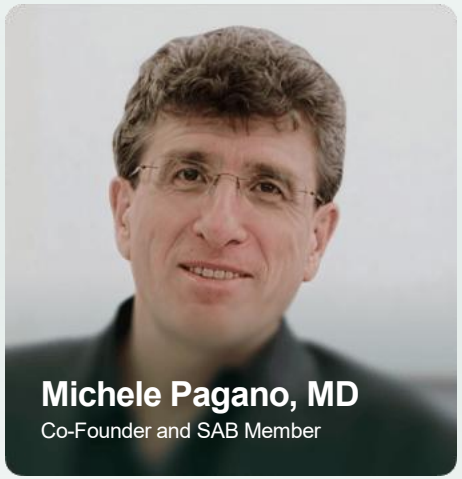
Nobel Laureate who discovered the ubiquitin–proteasome system



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



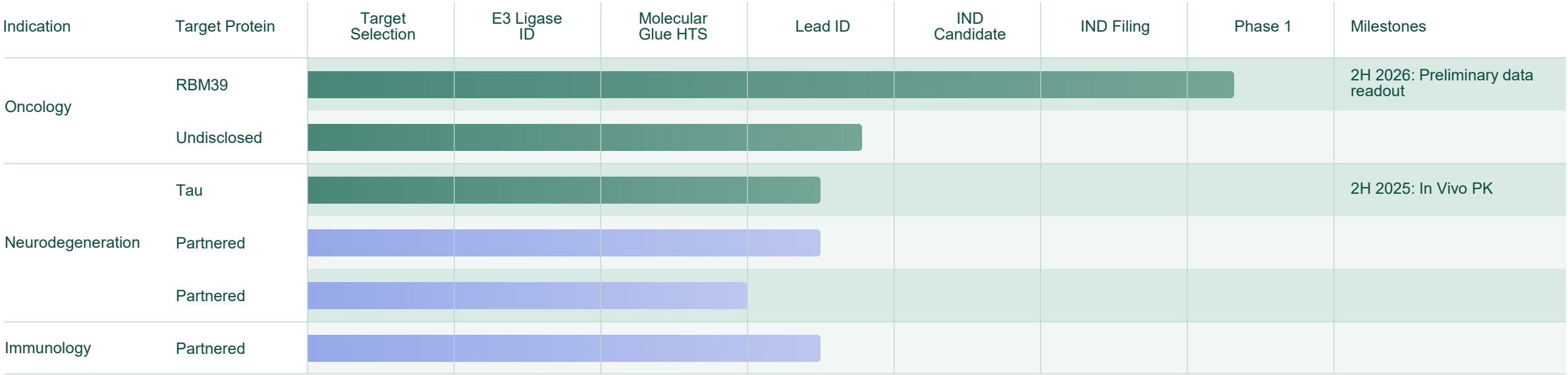
Solved the first RING E3 ligase and coined the term “molecular glue”



Defined SCF ubiquitin ligase and core cell-cycle ubiquitin biology

SEED's platform is already generating clinical assets alongside world-class partners

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



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

- Series A-3 financing: first close of $24 million from investors led by Eisai in August 2024.
- SEED–Eisai Collaboration: SEED receives upfront and milestone payments of up to $1.5 billion, plus tiered royalties upon Eisai's exercise of their exclusive rights under the strategic research collaboration.

RBM39: A clinically validated splicing dependency across multiple cancers

BIOLOGY

Why it matters

- Master regulator of oncogenic RNA splicing programs essential for tumor survival
- Splicing machinery is non-enzymatic and historically undruggable

MECHANISM

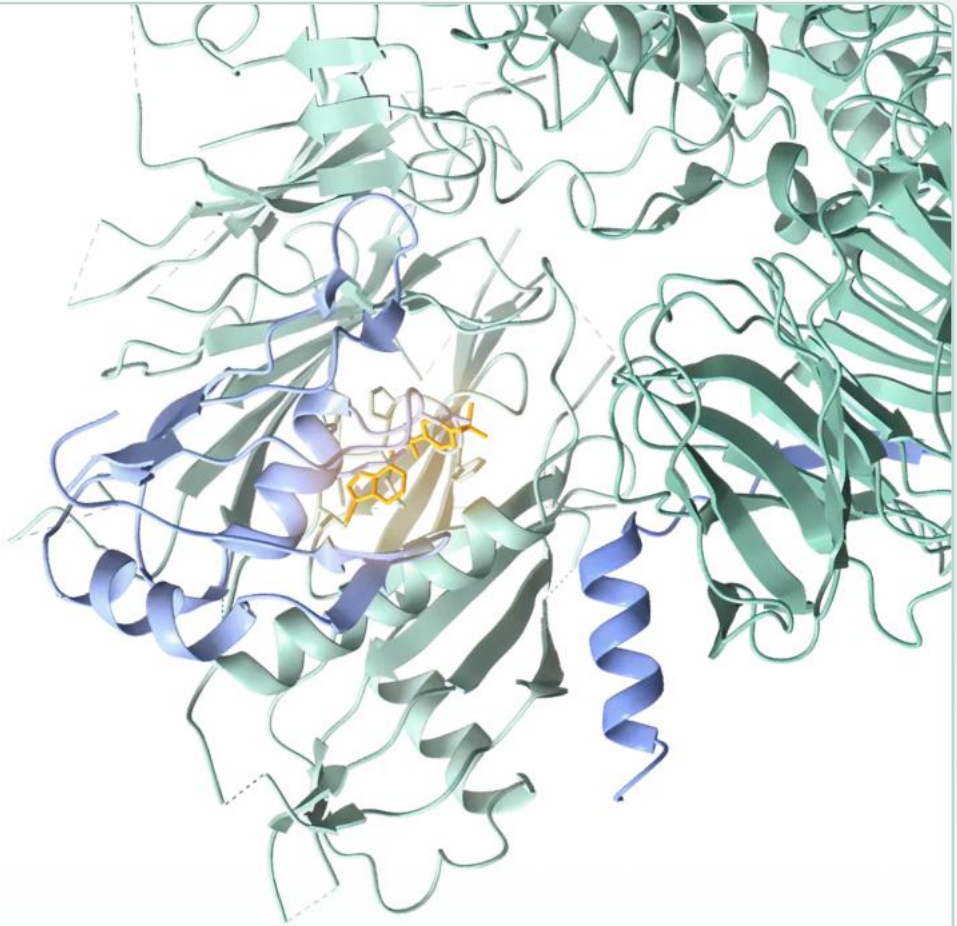
Why it works

- Molecular glue-mediated degradation via DCAF15 selectively eliminates RBM39
- Induces lethal mis-splicing in tumors while sparing normal cells via redundancy

VALIDATION

Why it's de-risked

- Genetic + pharmacologic degradation drives strong anti-tumor effects (incl. indisulam)
- Broad dependency across Ewing sarcoma and multiple solid tumors



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

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

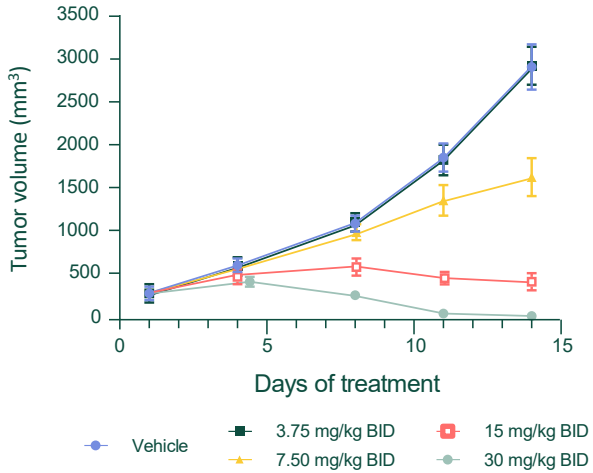
Ewing sarcoma is a fusion-driven disease with no new drugs in the past 30 years

The cleanest biological proof point for RBM39 degradation.

- ST-01156 IND candidate eliminates EWS-FLI1 which causes 90% of ES cases
- Total tumor regression in vivo with precise target engagement
- FDA Orphan + Pediatric Rare Disease designation (2025)

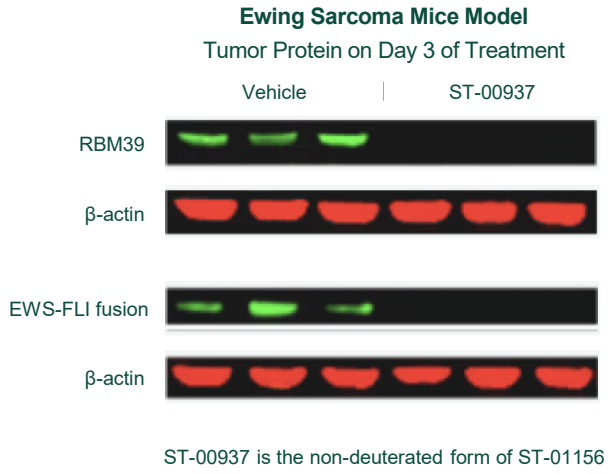
Complete tumor regression in Ewing sarcoma

Rare pediatric and orphan cancer designation by US FDA



Precise target engagement

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

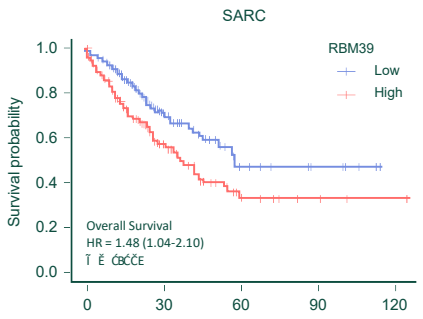


ST-00937 is the non-deuterated form of ST-01156

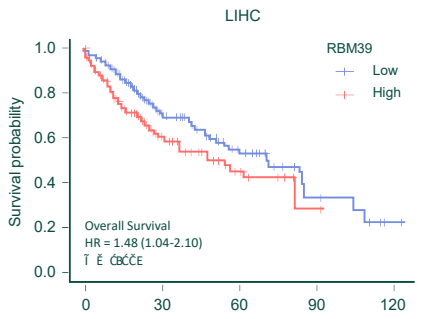
RBM39 drives progression for rare and large cancer indications with >1 million addressable patients

Neuroblastoma

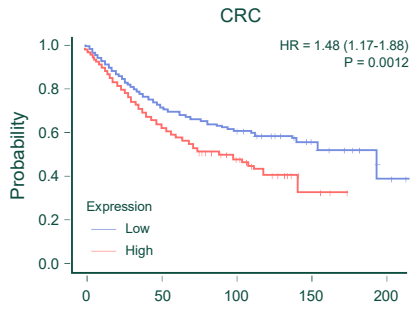
Sarcoma



Hepatocellular Carcinoma



Colorectal Cancer

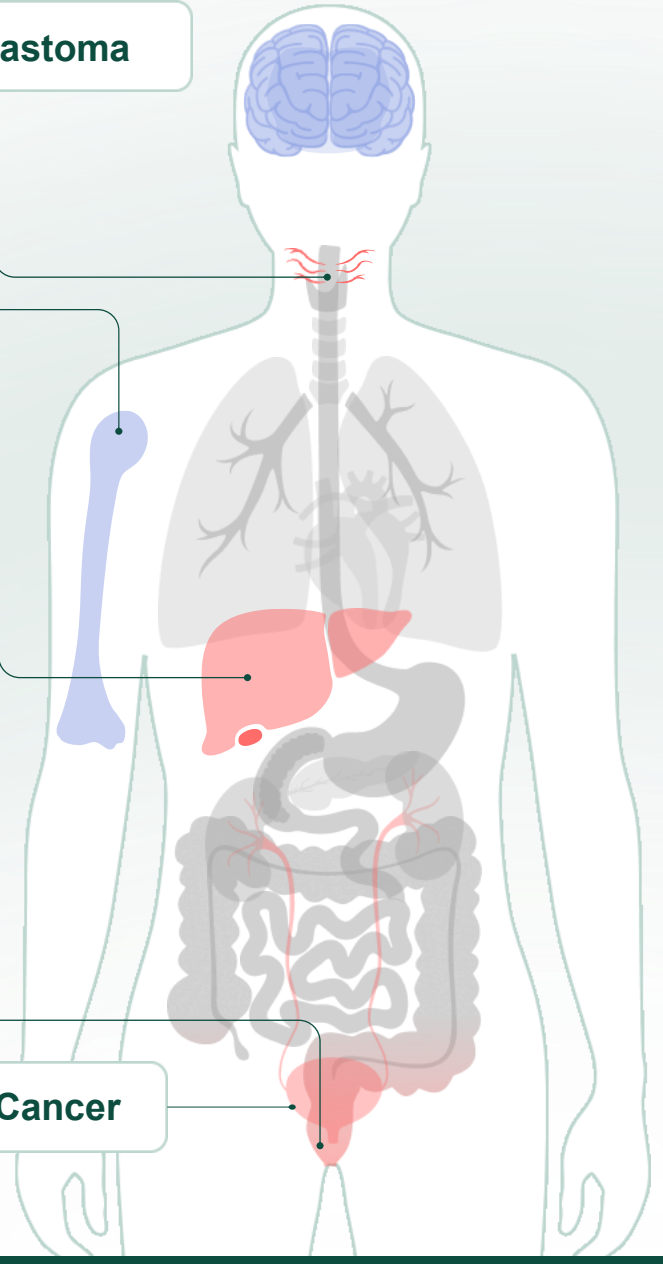


RBM39 overexpression leads to poor patient survival

Pediatric tumors

High prevalence tumors

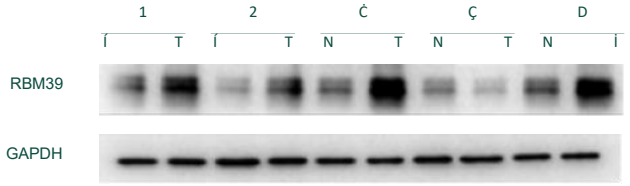
Prostate Cancer



Strong anti-tumor activity in RBM39-dependent liver & colon cancer models

Colon Cancer

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

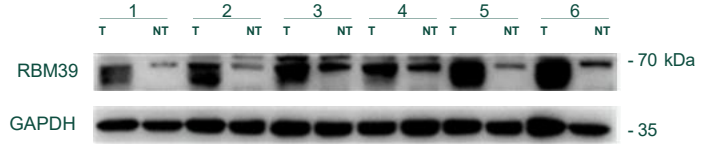


N = No Tumor
T = Tumor

Wang et al., *J of Cancer*, 2025

Liver Cancer

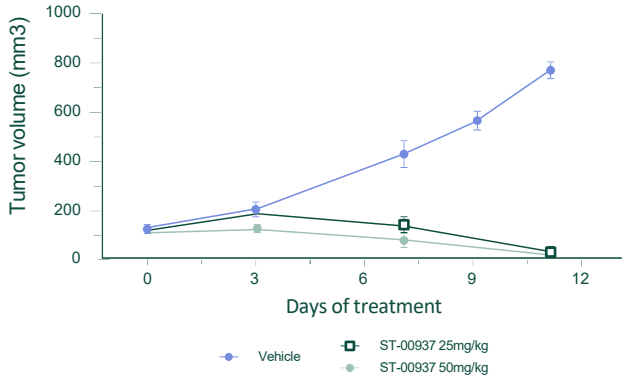
RBM39 high expression in liver cancer (T), not in normal tissue (NT)



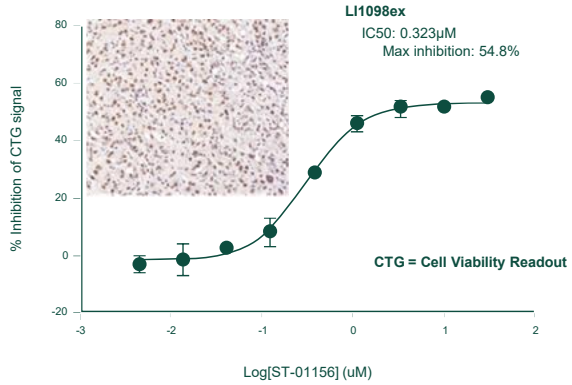
N = No Tumor
T = Tumor

Xia et al., *Cell Death Discovery, 2023*

Complete tumor regression in colon cancer model

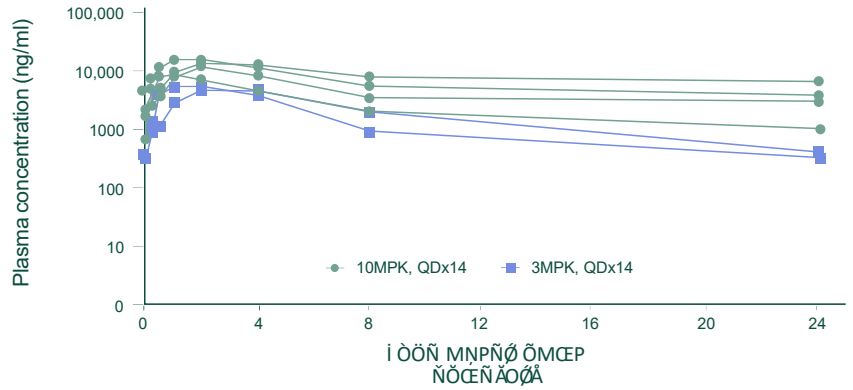


ST-01156 targets RBM39-expressing patient-derived hepatocellular carcinoma cells



RBM39 degrader safety dose at >10 times plasma PK for anticancer activities

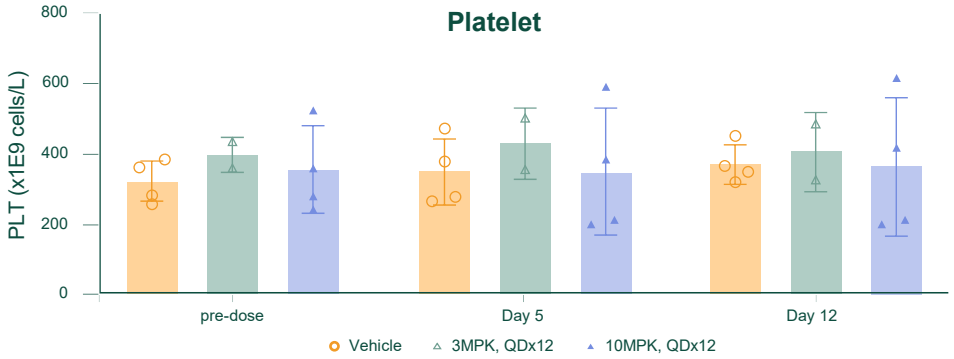
RBM39 degrader concentration in monkey plasma

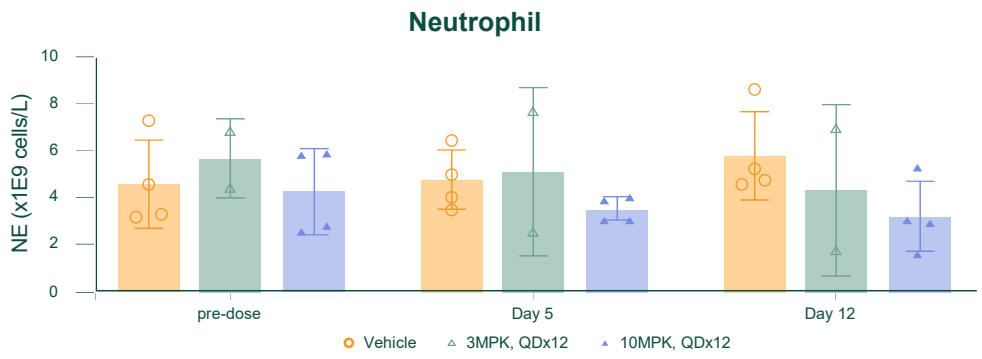


Mean plasma PK parameters

Dose	C_{min} (ng/ml)	C_{max} (ng/ml)	$AUC_{0\text{-}24hr}$ (h*ng/ml)
3MPK	321	4615	37902
10MPK	2560	11093	116813

Good hematological safety





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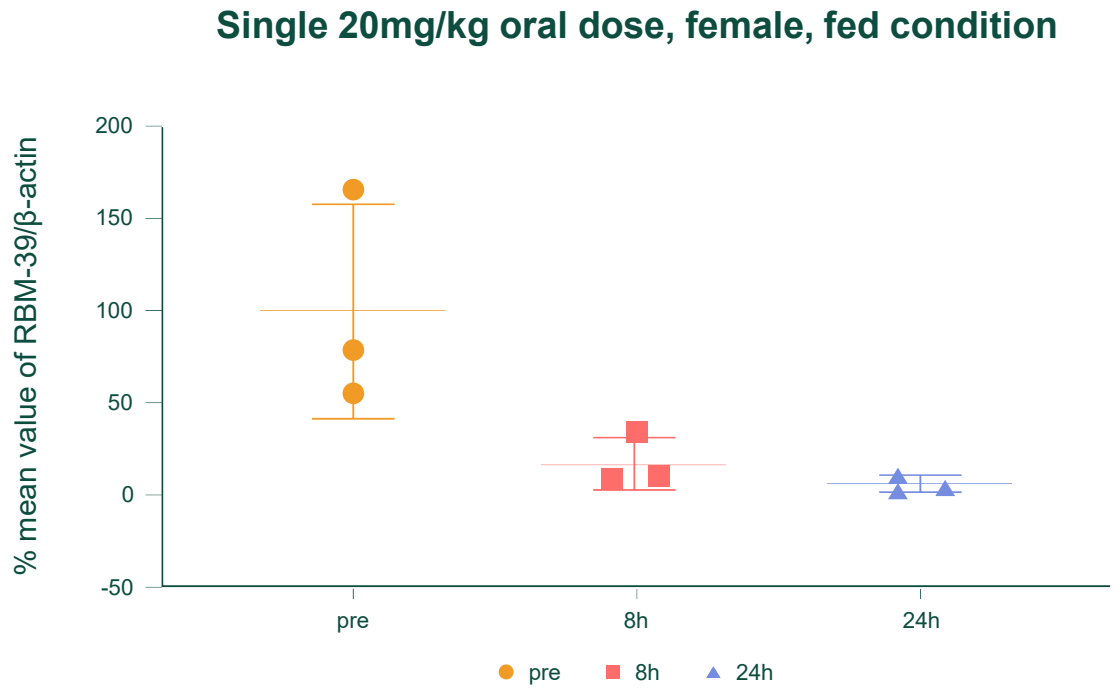
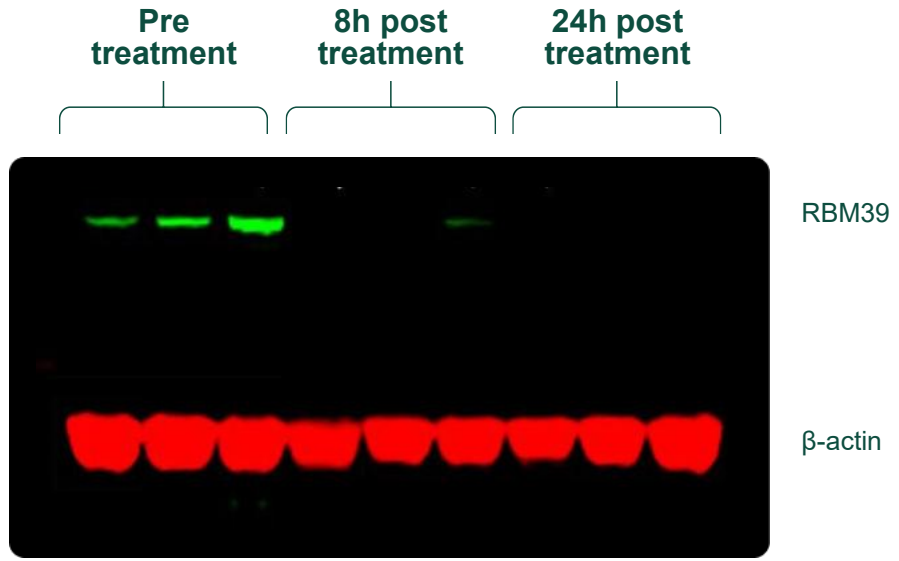
ST-00937 is the non-deuterated form of ST-01156

Rapid RBM39 degradation in PBMCs after oral dosing in monkey studies





Plan for RBM39 target engagement assay in PBMC for phase 1 to achieve rapid RP2D dose

ST-01156: Phase 1a clinical development plan for dose escalation

Objectives

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

Treatment Arms

Single-arm, open-label

N = 30–50 subjects

3 patients per cohort

Treatment Regimen & Timing

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

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

Multiple ascending doses

First Patient Dosed January 2026

Key Eligibility

Age 18+ all solid cancers

Age 16+ for Ewing sarcoma

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

Primary Endpoints

Safety, tolerability, MTD/MAD, RP2D

Secondary Endpoints

PK/PD, preliminary efficacy

Sites

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

ST-01156: Phase 1b clinical development plan for dose expansion

Cohorts at recommended RP2D

N for each cohort =
16–35 subjects

Single-group open-label

Presence and quantification of relevant antitumor activity
Priority indications

- **Ewing sarcoma (Age 12+)**
- **Hepatocellular carcinoma**
- **KRAS mutated solid cancers, including colon cancer**
- **Biliary tract cancer, uterine cancer, and DNA repair mutations (BRCA and others)**
- **Pediatric dose escalation (Age 12+) (staggered with adult study)**

Simon 2-stage–like design with initial futility
Expand to 10–15 global centers (US, Asia, Europe)

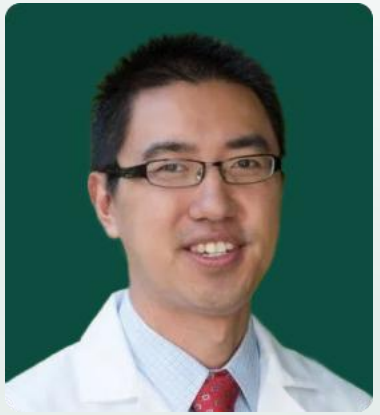
Leading oncologists guiding development of ST-01156

Dr. George D. Demetri

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

Dr. Robert Maki

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



Dr. Daneng Li

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

Dr. Jordi Rodón

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

Dr. Monica Mita

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

Expert leadership to support ST-01156 first-in-human development

James Tonra, PhD
President & CSO

20+ years of drug discovery experience that led to 5 NDAs

Former leadership roles at Regeneron, Millennium, ImClone, Kadmon, and BYSI

Eric K. Rowinsky, MD
Clinical and Medical Lead

Veteran oncologist and drug developer with leadership roles across early- and late-stage clinical development.

Former CMO of ImClone Systems; led approvals of cetuximab, ramucirumab, necitumumab and clinical development of erlotinib, gefitinib, panitumumab, temsirolimus, ridaforolimus, trabectedin, paclitaxel, docetaxel, irinotecan, and topotecan.

Scott L. Spector
Clinical Operations Management Lead

Clinical development and operations leader with deep US/EU regulatory expertise. Former

Head of EU Operations at Quintiles, overseeing global trial execution, quality, biometrics, medical affairs, and regulatory strategy.

Bill Desmarais, PhD
CFO & CBO

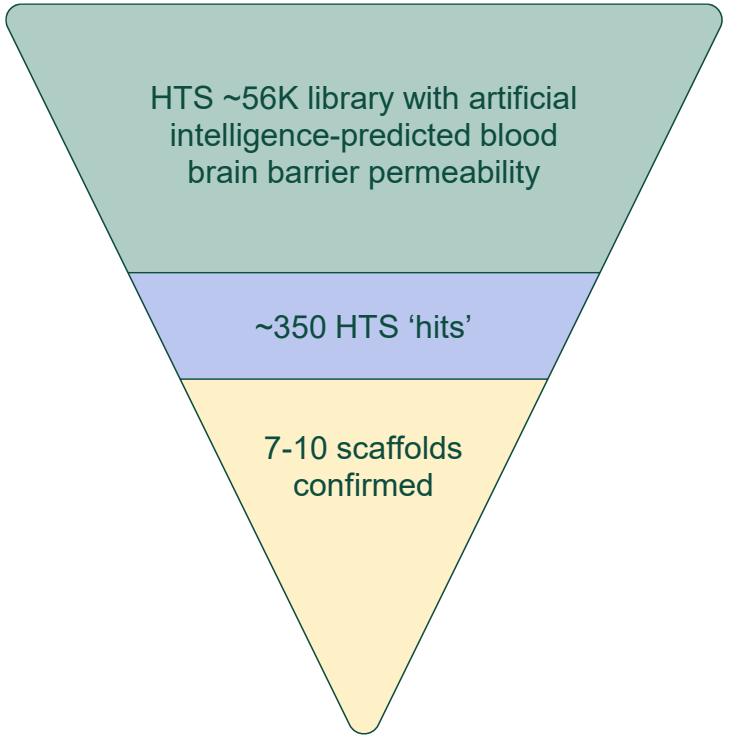
20+ years in finance, business development, and strategic operations

Former leadership roles at Alchemab, TScan, Momenta, and Lilly

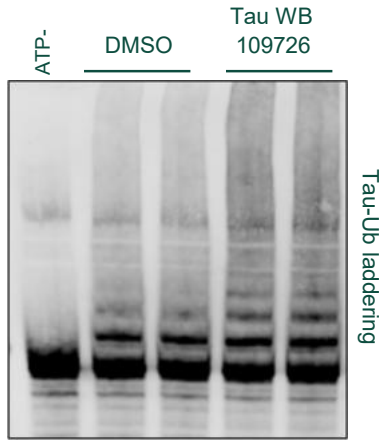
The same degradation engine is now aimed at neurodegeneration with an oral Tau degrader for Alzheimer's

✓ Novel E3 ligase selected that is highly expressed in neuron

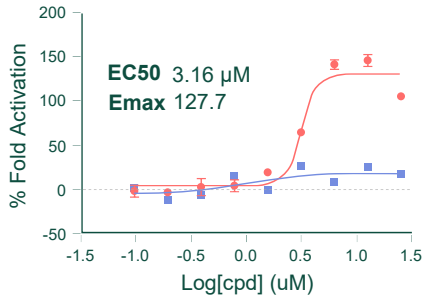
✓ Identified specific lysine residues of Tau being ubiquitinated



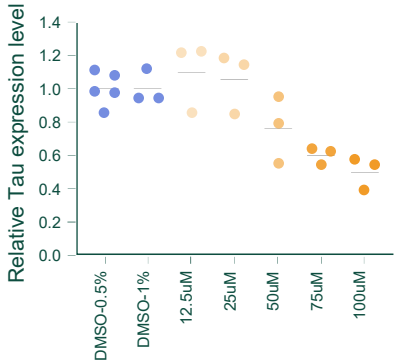
Increase Tau Poly-Ubiquitination



Increase binding between Tau/E3 (TR-FRET Assay)



Reduce Tau Protein Level in Neuron Cells (ELISA Assay)



✓ Increase binding and poly-ubiquitination

✓ Reduce total Tau level in a human neuronal cell line, not affecting cell viability

Breakthrough investments highlight the value of molecular glues and degraders

Discovery stage TPD assets

Upfront payments of $35 - $60M and $500M - $5B in potential milestones.

Pre-IND / IND stage TPD assets

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

Clinical stage TPD assets (Phases I & II)

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

BioSpace

Nurix Closes $120 Million Financing to Support Protein Degradation Program

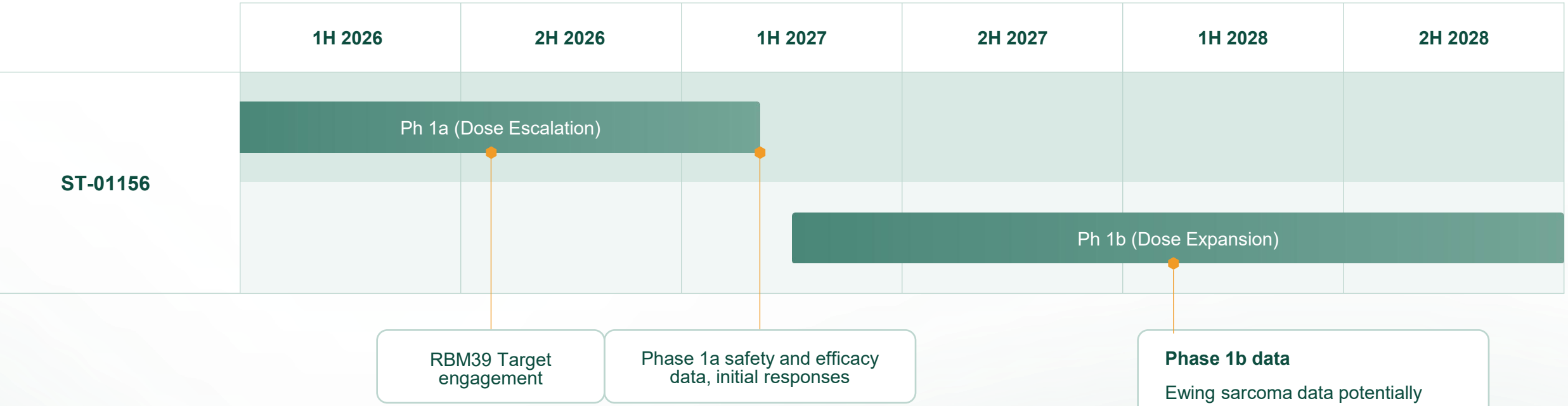
BioSpace

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

Pharmaceutical Technology

Gilead eyes Kymera's 'adhesive' cancer drug in $750m deal

SEED's Rapid Growth Trajectory



Additional Milestones Supporting Long-Term Value Creation Through 2028

- Nomination of second development candidate for IND-enabling studies
- Strengthening the RITE3™ Platform to target undruggable protein targets

SEED Therapeutics – Creating significant value in 2026 - 2028

Clinically advancing first-in-class molecular glue degrader

- ST-01156 IND cleared in the U.S. and China
- First-in-human safety and target engagement data expected in 2026

Science built on the founders' pioneering E3 structural discoveries

- Solved structural biology of both major E3 classes: HECT and CRL (RING-based) ligases
- These insights power SEED's rational E3 selection and neo-substrate design via RITE3™

Mechanism-driven clinical strategy enabling rapid proof-of-concept

- Prioritized indications: Ewing sarcoma, HCC, KRAS-mutant solid tumors
- Strong PK/PD, regression models, and biomarker strategy support early efficacy readouts

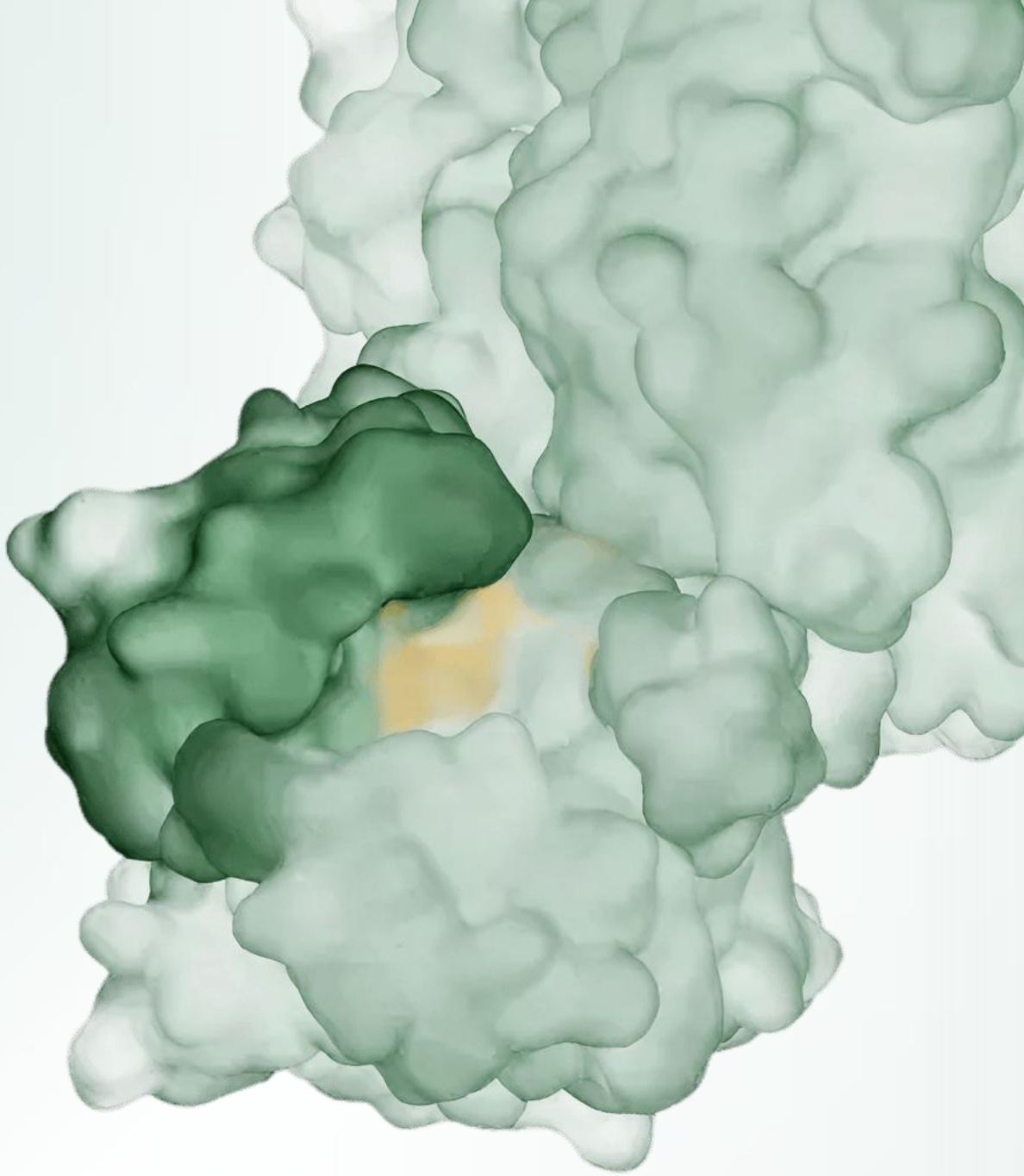
Pharma-validated platform with significant non-dilutive value

- Lilly and Eisai collaborations with >$2.3B milestone potential
- Novel program portfolio across oncology and neurodegeneration

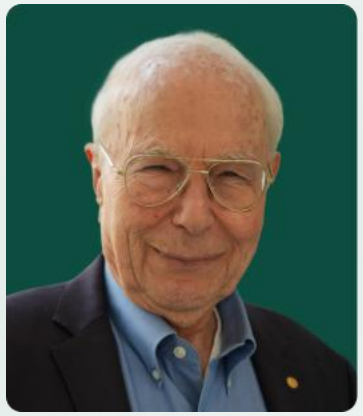
SEED THERAPEUTICS

Thank You

info@seedtherapeutics.com www.seedtherapeutics.com



Expert co-founders and leadership team



Avram Hershko MD, PhD

Co-Founder and SAB Member

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

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

Ning Zheng, PhD

Co-Founder and SAB Member

Howard Hughes Professor; 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 Professor, Chair of Biochemistry at NYU Medical School; Global thought leader on TPD biology and application

James Tonra, PhD

President, CSO and Director

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

Bill Desmarais, PhD

CFO & CBO

20+ years in finance, business development, and strategic operations; Expert leadership role at Alchemab, TScan, Momenta and Lilly

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

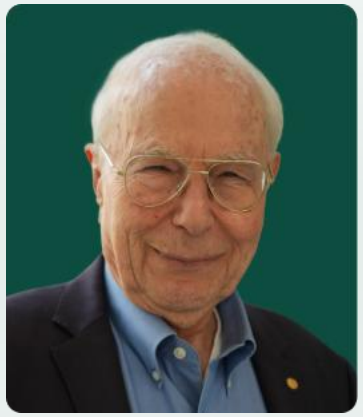
Former Eisai director, World Bank general counsel, and lecturer at Harvard and Yale Law School; Expert in law and international business

Scientific and Strategic Advisors

George Demetri, MD

SAB Chair

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



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Howard Hughes Medical Institute Investigator and Chairman of Biochemistry at NYU Medical School; Global thought leader on TPD biology and application

Mansuo Shannon, PhD

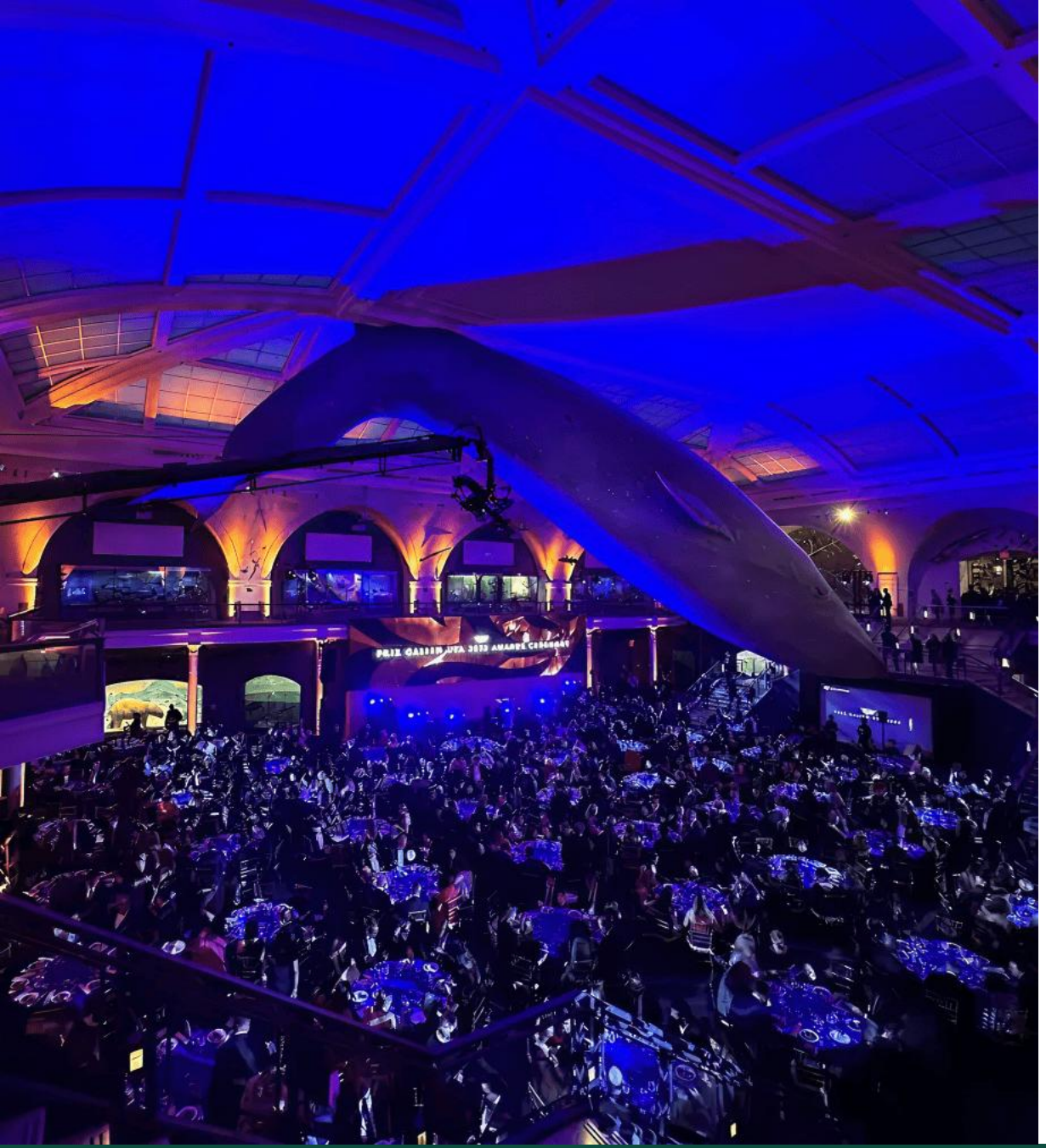
SAB Member

Seasoned pharmaceutical executive and drug hunter with over 18 years of experience at Bayer, Eli Lilly, Roche/Chugai, and Merck; Chief Scientific Officer of AskBio, Bayer's gene therapy subsidiary, with a track record of advancing multiple programs into clinical development, including Phase 2 Alzheimer's studies.

Alan Roemer, MBA, MPH

AB Member

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



SEED Therapeutics

Recognized at the highest level of biopharmaceutical innovation

SEED named finalist for the 2025 Prix Galien USA “Best Start-Up” award, the industry’s most prestigious honor.