



***Using protein inhibition and
protein degradation to
develop cancer therapies for
patients in need of new
treatment options***

NasdaqCM:SLRX

April 2023

Safe Harbor Statement

This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995. All statements, other than statements of historical facts, included in this presentation are forward-looking statements. These forward-looking statements may be identified by terms such as “will,” “future,” “believe,” “developing,” “expect,” “may,” “progress,” “potential,” “could,” “look forward,” “might,” “should,” and similar terms or expressions or the negative thereof. Examples of such statements include, but are not limited to, statements relating to the following: the advantages of seclidemstat (SP-2577) as a treatment for Ewing sarcoma, Ewing-related sarcomas, and other cancers and its ability to improve the life of patients; expected cohort readouts from the Company’s clinical trials and expected therapeutic options for SP-2577 and related effects and projected efficacy, including SP-2577’s ability to inhibit LSD1; the future of the company’s Phase 1/2 trial of seclidemstat as a treatment for Ewing sarcoma and FET-rearranged sarcomas following the October 2022 suspected unexpected severe adverse reaction (SUSAR) event and resulting partial clinical hold by the U.S. Food and Drug Administration (FDA); the advantages of protein degraders including the value of SP-3164 as a cancer treatment; the timing of clinical trials for SP-3164 and expected therapeutic options for SP-3164 and related effects and projected efficacy; impact that the addition of new clinical sites will have on the development of our product candidates; the timing of our IND submissions to the U.S. Food and Drug Administration (FDA) and subsequent timing for initiating clinical trials; interim data related to our clinical trials, including the timing of when such data is available and made public; our growth strategy; whether the company will develop additional undisclosed cancer-fighting assets in the targeted protein degradation space; expanding the scope of our research and focus to high unmet need patient populations; and the commercial or market opportunity and expansion for each therapeutic option, including the availability and value of a pediatric priority review voucher for in-clinic treatments and potential for accelerated approval. We may not actually achieve the plans, carry out the intentions or meet the expectations or objectives disclosed in the forward-looking statements. You should not place undue reliance on these forward-looking statements. These statements are subject to risks and uncertainties which could cause actual results and performance to differ materially from those discussed in the forward-looking statements. These risks and uncertainties include, but are not limited to, the following: Seclidemstat’s impact in Ewing sarcoma and as a potential new and less toxic treatment; expected dose escalation and dose expansion; resolution of the FDA’s partial clinical hold on the company’s Phase 1/2 trial of seclidemstat as a treatment for Ewing sarcoma and FET-rearranged sarcomas following the SUSAR; our ability to resume enrollment in the clinical trial following its review of the available data surrounding the SUSAR; the adequacy of our capital to support our future operations and our ability to successfully initiate and complete clinical trials and regulatory submissions; the ability of, and need for, us to raise additional capital to meet our business operational needs and to achieve its business objectives and strategy; future clinical trial results and the impact of such results on us; that the results of studies and clinical trials may not be predictive of future clinical trial results; risks related to the drug development and the regulatory approval process; the competitive landscape and other industry-related risks; and other risks described in our filings with the Securities and Exchange Commission, including its Annual Report on Form 10-K for the fiscal year ended December 31, 2022, as revised or supplemented by its Quarterly Reports on Form 10-Q and other documents filed with the SEC. The forward-looking statements contained in this presentation speak only as of the date of this presentation and are based on management’s assumptions and estimates as of such date. We disclaim any intent or obligation to update these forward-looking statements to reflect events or circumstances that exist after the date on which they were made. You should review additional disclosures we make in our filings with the Securities and Exchange Commission, including our Quarterly Reports on Form 10-Q, Annual Reports on Form 10-K, and Current Reports on Form 8-K. You may access these documents for no charge at <http://www.sec.gov>. This presentation shall not constitute an offer to sell or the solicitation of an offer to buy, nor shall there be any sale of these securities in any state or other jurisdiction in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or other jurisdiction.



Investment Highlights

1

SP-3164 is a next-generation cereblon-binding targeted protein degrader that is the preferred enantiomer of the drug CC-122 that has already been studied in over 400 patients across 10 clinical trials

2

SP-3164 is shown to be preclinically superior to lenalidomide and pomalidomide in Multiple Myeloma, superior to lenalidomide in Diffuse Large B-Cell Lymphoma & highly effective in Follicular Lymphoma

3

SP-3164 IND submission planned for 1H 2023 with Phase 1/2 trial beginning 2H 2023. Market opportunity for SP-3164 could exceed \$5 billion

4

SP-2577, seclidemstat, showed encouraging clinical activity in a Phase 1 hematologic cancers trial and Phase 1/2 Ewing's sarcoma trial. Plans to advance both clinical trials pending FDA discussions

5

As of Q4 2022, cash of \$12.1M and no debt



Pipeline Overview

Protein Degradation and Protein Inhibition

Degraders

Inhibitors

| | Discovery | IND-Enabling | Phase 1 | Phase 2 | Phase 3 | Next Milestones |
|---|-----------|--------------|---------------------------|---------|---------|--|
| Hematologic cancers (SP-3164; A/I molecular glue) | ▶ | | | | | Preclinical data in 2H22 Submit IND in 1H23 |
| Undisclosed (SP-3204; GSPT1 molecular glue) | ▶ | | Announced January 5, 2023 | | | Preclinical data in 1H23 |
| Ewing sarcoma (Seclidemstat + TC ¹) | ▶ | | | | | Re-start enrollment and data update 2H 2023 |
| Hematologic cancers² (Seclidemstat + azacytidine) | ▶ | | | | | Re-start enrollment and data update 2H 2023 |
| Select gynecologic cancers³ (Seclidemstat + pembrolizumab) | ▶ | | | | | Trial activation |
| Hematologic and solid tumors NCE second-generation LSD1 | ▶ | | | | | Nominate clinical candidate |



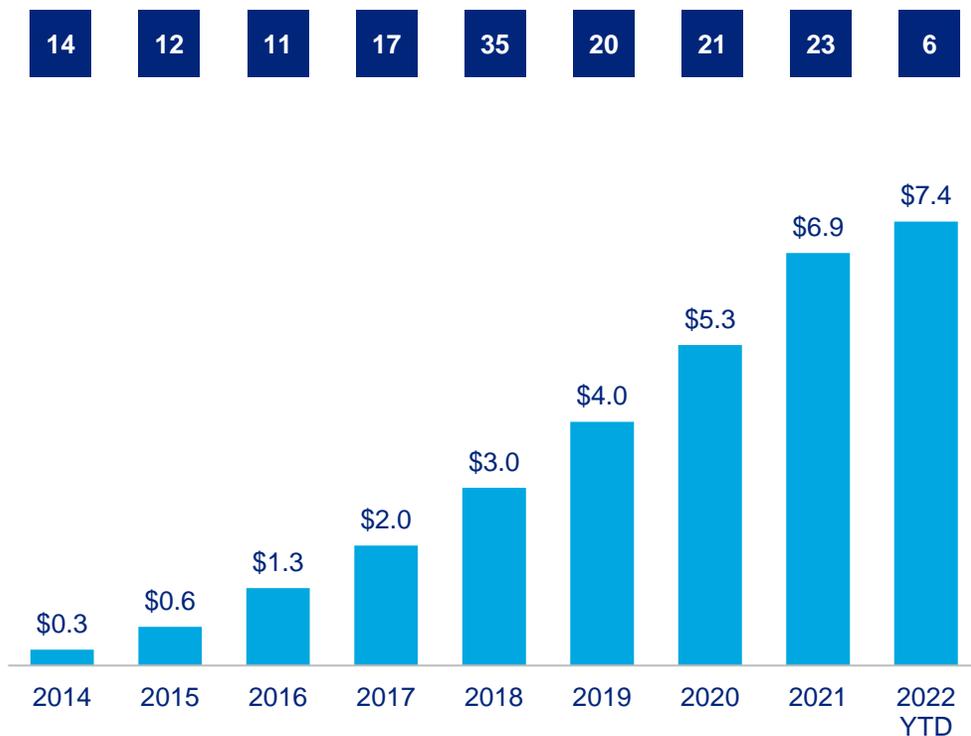
¹ Topotecan and cyclophosphamide ² Investigator initiated trial active at MD Anderson Cancer Center treating patients with Myelodysplastic Syndromes and Chronic Myelomonocytic Leukemia ³ Investigator initiated trial – Clinical trial agreement not yet finalized.

Targeted Protein Degradation Space Has Witnessed Tremendous Growth

Cumulative Capital Invested in Development of Targeted Protein Degradator Therapies^{1,2}

(\$B)

Number of Investments



Large Biopharma Companies Have Moved Aggressively to Gain Exposure³

Selected targeted protein partnerships and strategic collaborations since 2015

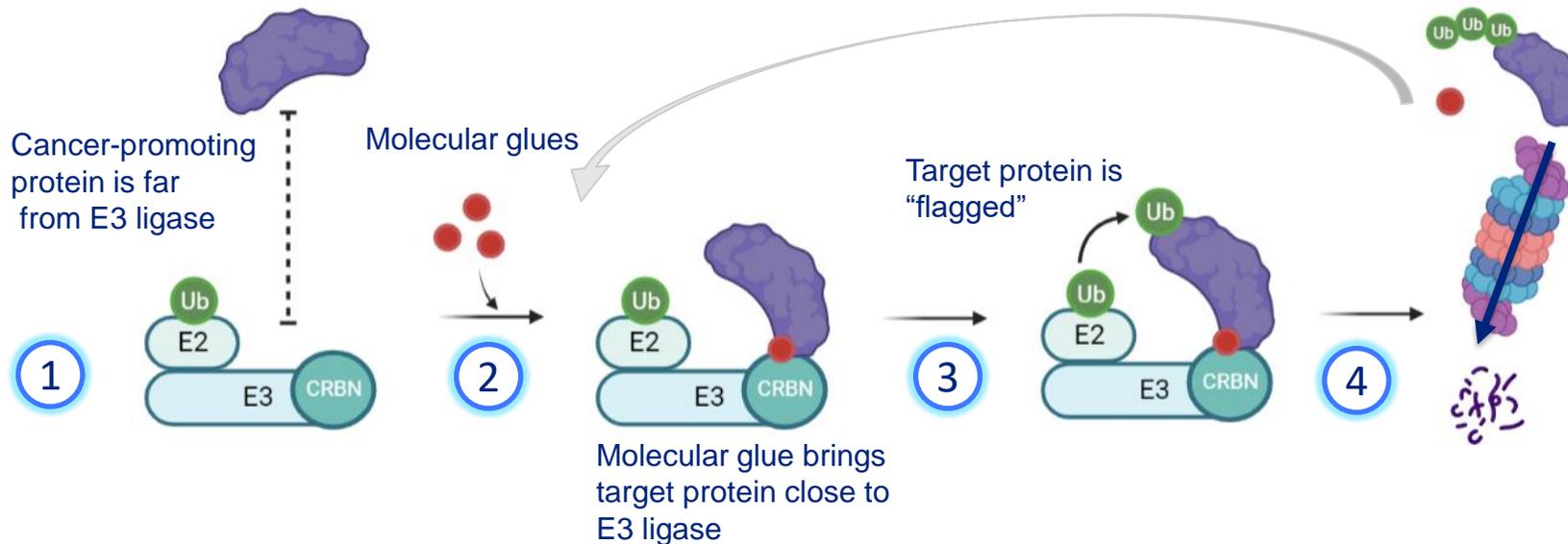


.... and many others

¹ Roots Analysis, ² Nature.com, ³ Cortellis.

CRBN-Binding Molecular Glues Induce Proteasomal Degradation

Targeted Protein Degradation (TPD) utilizes the **body's own** degradation system to **selectively eliminate** cancer-promoting proteins AND provide the ability to pursue historically **undruggable** cancer-promoting targets



Advantages

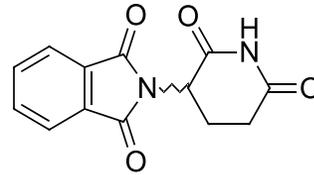
- ✓ Low doses
- ✓ Undruggable targets
- ✓ Enzymatic/scaffolding inactivation



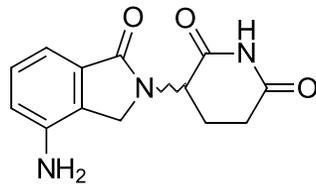
First-Generation Protein Degraders

IMiDs[®] (Immunomodulatory Drugs) – Approved for hematological malignancies

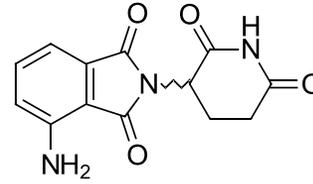
- \$16B in sales in 2021
- All exist as racemic mixtures



Thalidomide



Lenalidomide

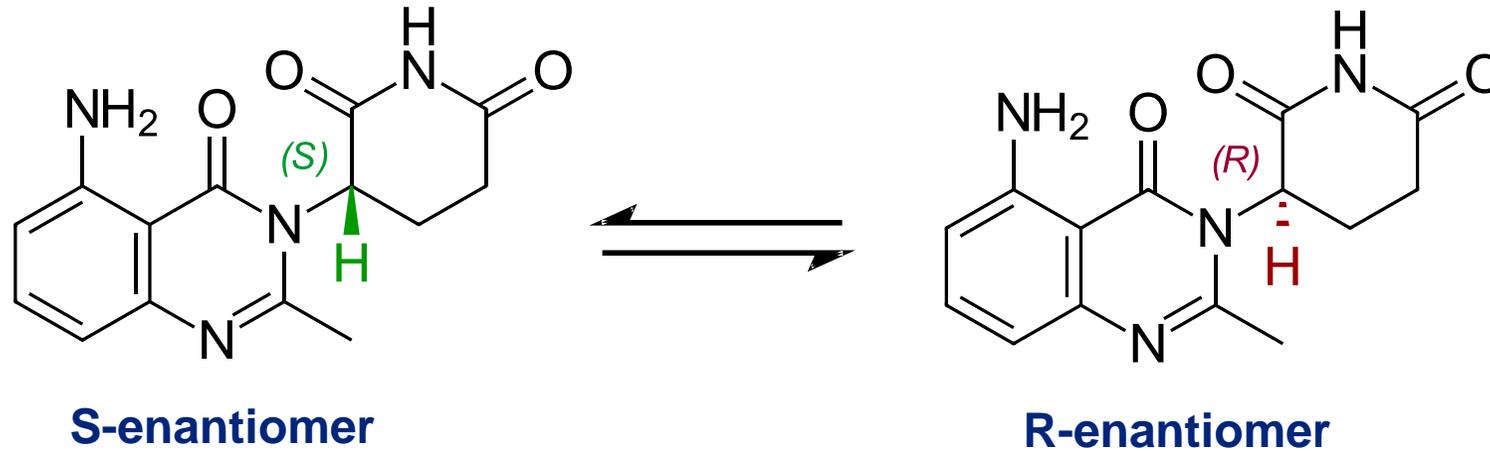


Pomalidomide

First-generation degraders validated the concept, but there is room for improvement



Avadomide, a 2nd Generation Extensively Studied Degradable *Exists as a Mixture of 2 Species*



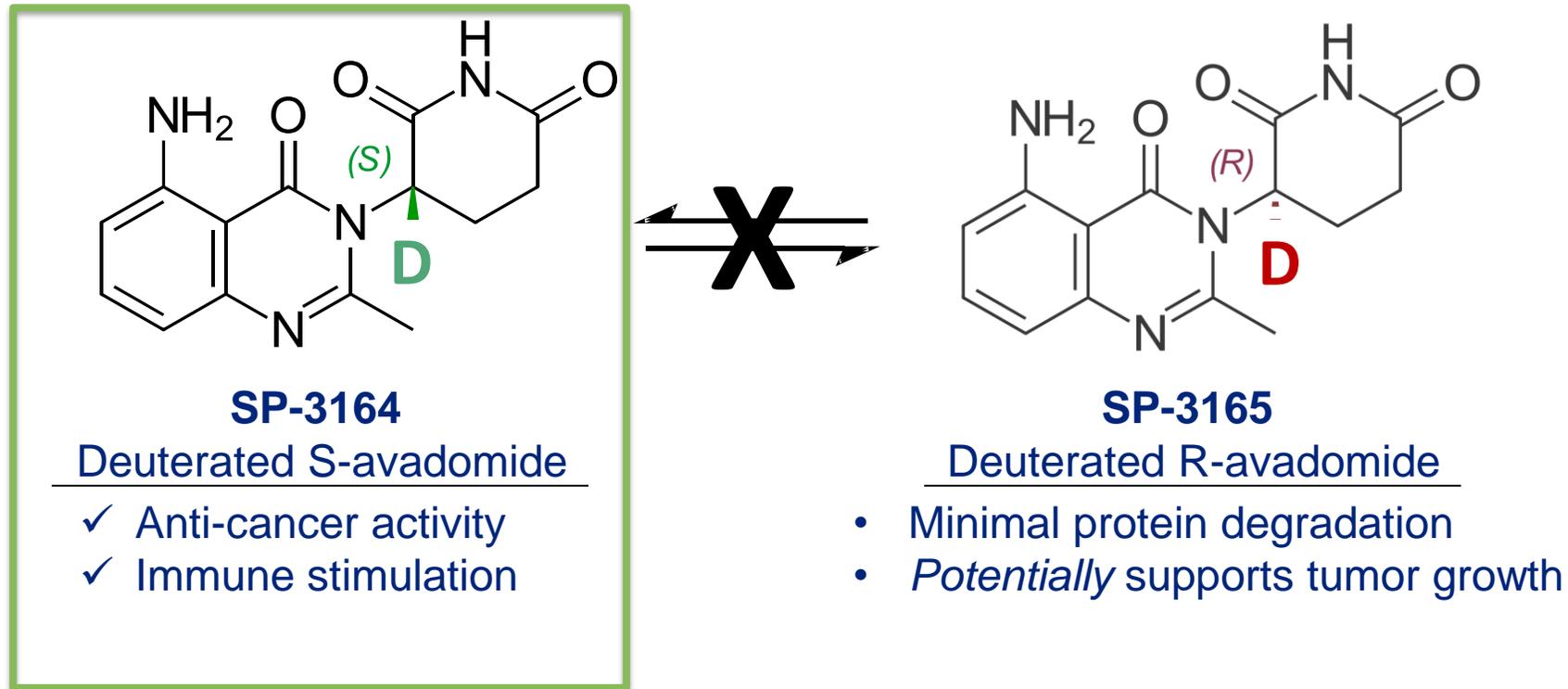
Avadomide (CC-122)

a 1:1 mixture of 2 interconverting species

- ✓ Studied in 10 clinical trials in >400 patients
- ✓ 28% Overall Response Rate as a monotherapy for lymphomas

SP-3164: The Deuterium-Stabilized S-Enantiomer of Avadomide

- Stabilization of avadomide enantiomers with deuterium blocks interconversion



- A new chemical entity with its own, issued composition of matter patent
- Potential for **improved efficacy and safety compared to avadomide**



Clinical Development Of SP-3164, A Targeted Protein Degradator With Potential In Hematological Indications Of High Unmet Need

The Problem

Initial target indication:

- **3rd line (3L+) R/R DLBCL¹**

Difficult to cure with relapse often occurring and most patients having overall survival of <6 months.

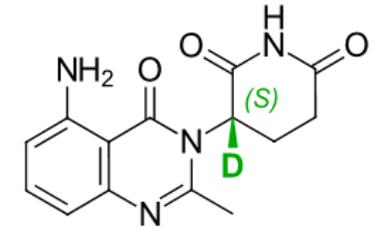
Launch indications:

1. First indication: ~3,200 R/R DLBCL (3L+) patients ineligible for stem cell transplant or CAR T therapy with survival outcomes as low as 6 mos.
2. Second indications: Select² 1st line DLBCL and 1st line FL pts, and ~1,000 R/R FL (3L+) patients seeking novel, chemotherapy-free treatment options.

Salarius's Solution

Salarius is developing SP-3164, a targeted protein degrader (molecular glue) to improve lymphoma patient outcomes.

- Potential \$1B+ market



SP-3164

SP-3164 is differentiated from other degraders

Only degrader w/ stabilized active species resulting in:

1. Improved activity in preclinical models
2. Potential for an improved therapeutic window
3. Precision medicine for improved responses

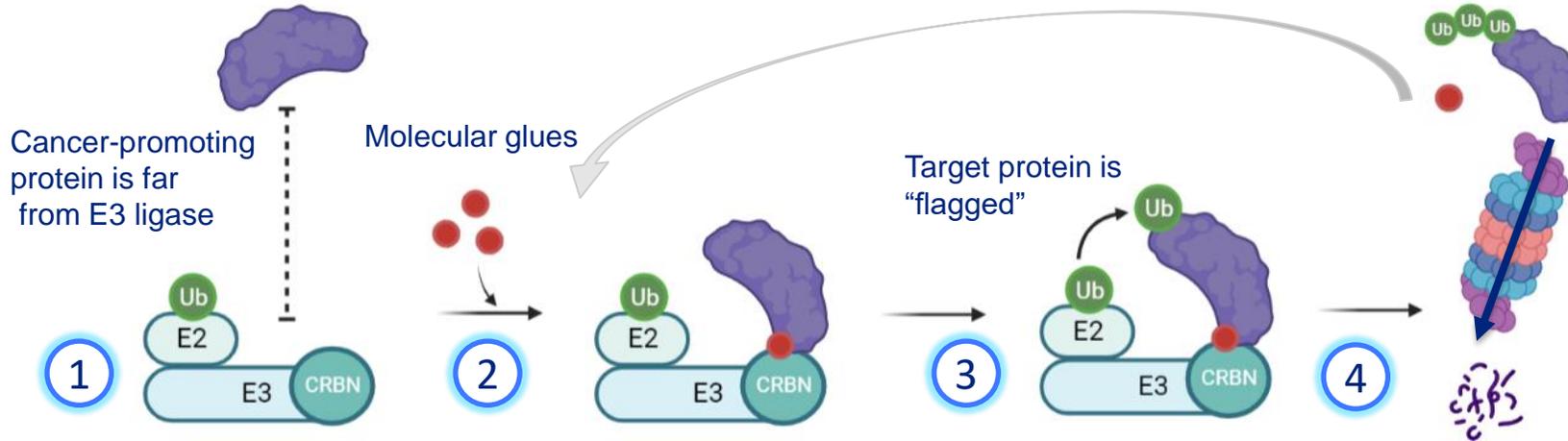
¹ Due to accelerated registration path and more aggressive disease, 3L+ DLBCL will precede 3L+ FL. ² Utilizing prospectively defined gene signatures, develop precision targeting for high responder patients
Abbreviations: NHL - Non-Hodgkin's lymphomas, R/R - relapsed/refractory, DLBCL - Diffuse Large B-cell Lymphoma, FL - Follicular Lymphoma



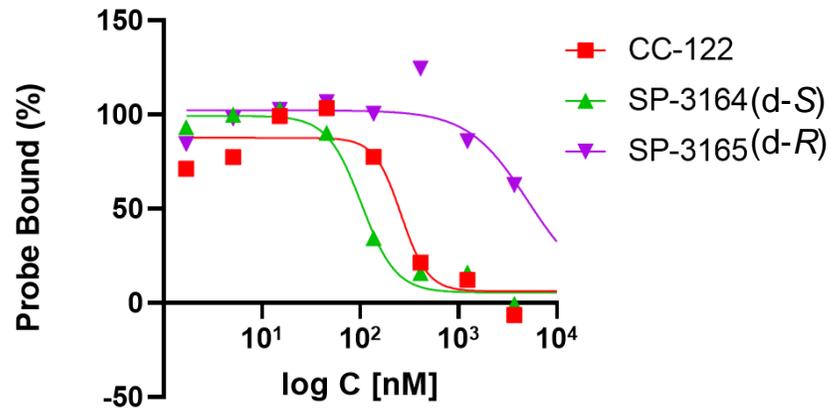
SP-3164 Preclinical Data



SP-3164 Demonstrates Improved Protein Degradation Characteristics Compared to Avadomide (CC-122)



Cereblon Binding



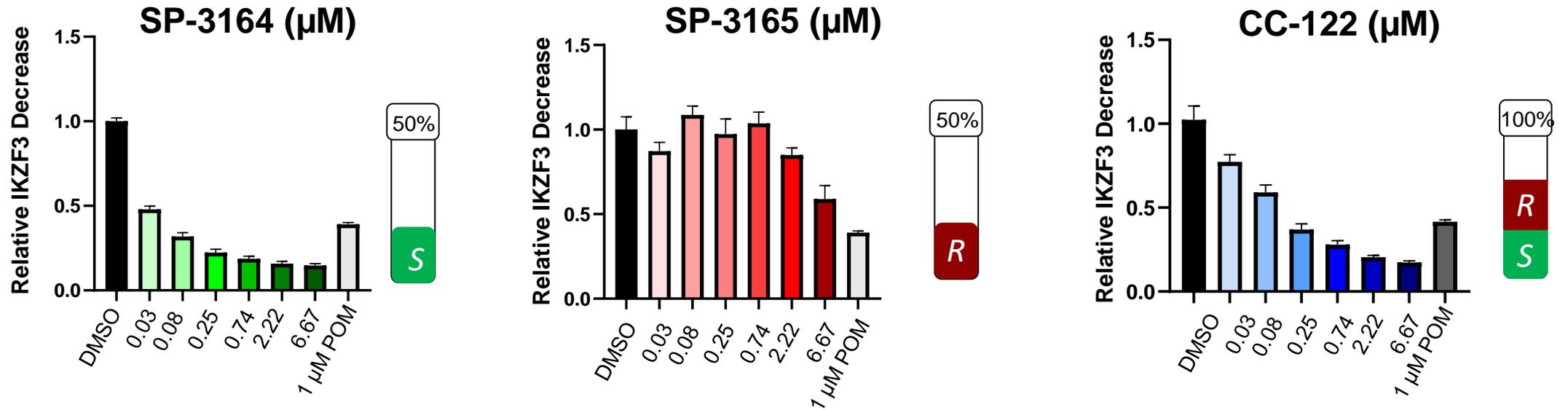
| Compound | Kd (nM) |
|---------------|---------|
| CC-122 | 330 |
| SP-3164 (d-S) | 110 |
| SP-3165 (d-R) | 14000 |

SP-3164 binds more potently to cereblon than the racemate (avadomide, CC-122) while SP-3165 (d-R-enantiomer) does not bind at meaningful concentrations.



SP-3164 Rapidly Degrades IKZF3 (Aiolos) at Low Concentrations

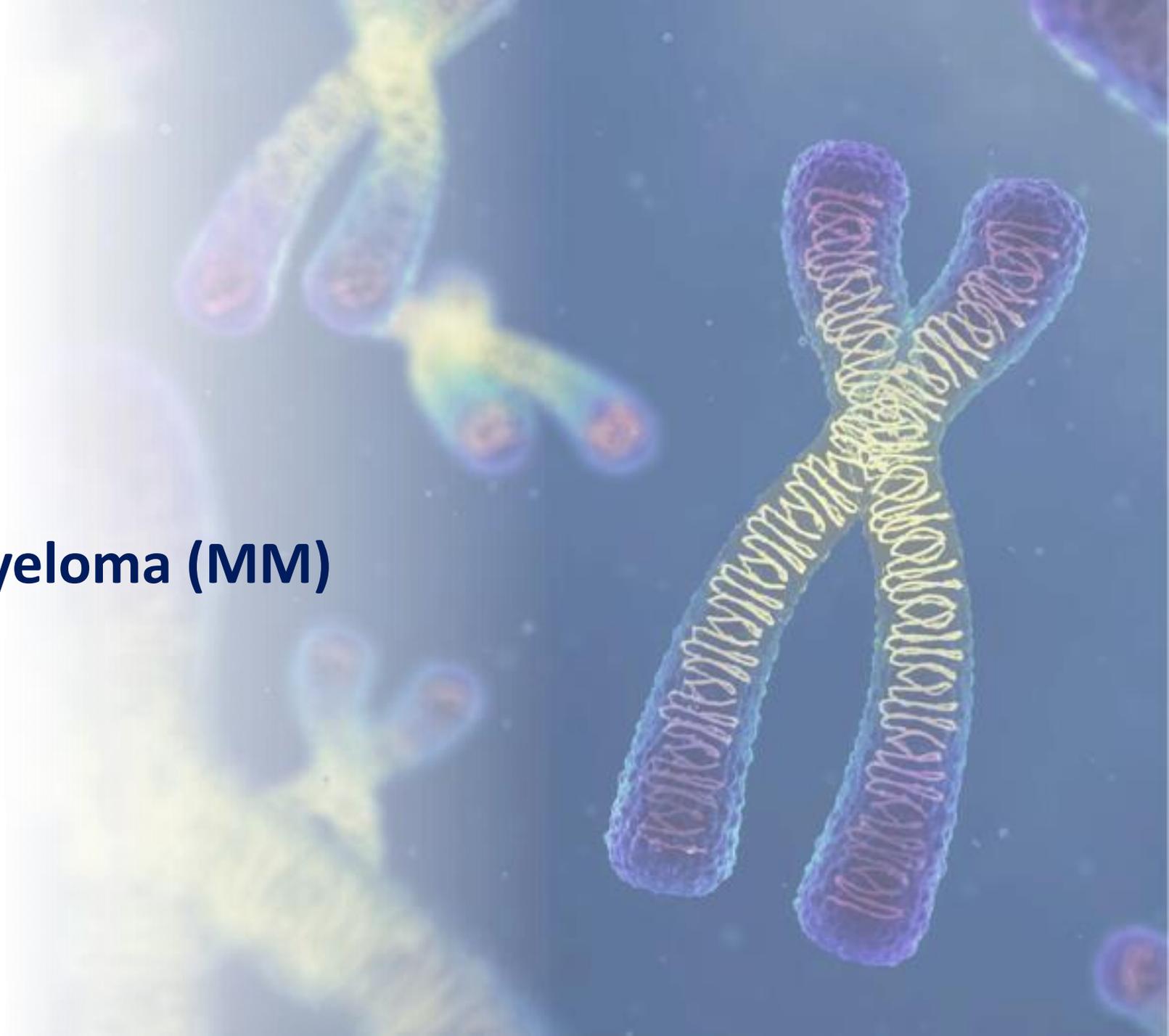
HiBiT-IKZF3 MM.1S Degradation (2 hours)



- Treatment with SP-3164 for 2 hrs results in deep and rapid degradation of the target protein, IKZF3.
- SP-3165 does not result in protein degradation except for at high concentrations.
- At comparable concentrations, SP-3164 induced more degradation of IKZF3 compared to CC-122



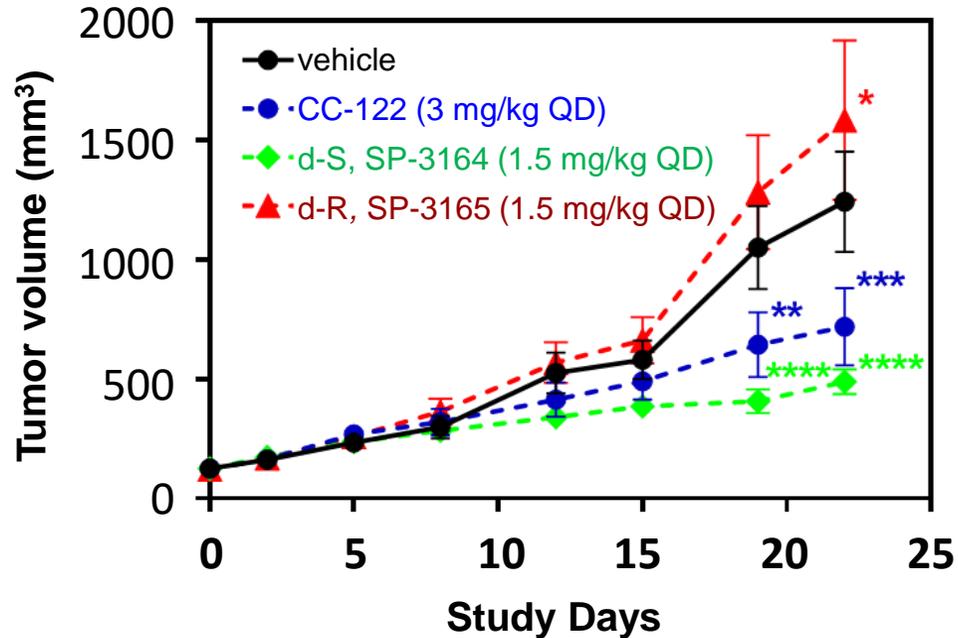
SP-3164 – Multiple Myeloma (MM)



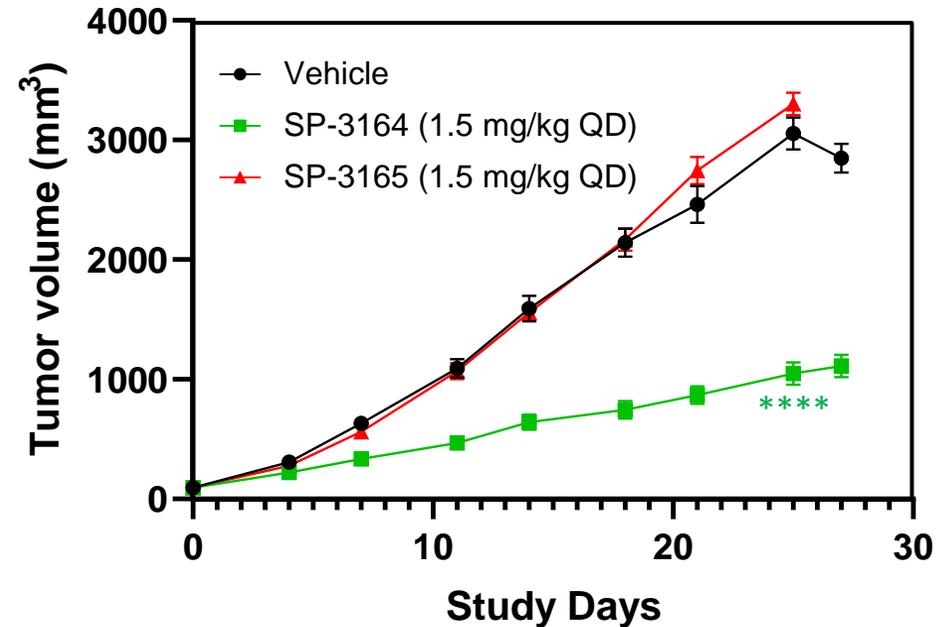
SP-3164 Shows Significant Activity in MM H929 Xenograft Model

R-Enantiomer (SP-3165) is Inactive

NCI-H929 Xenograft Study



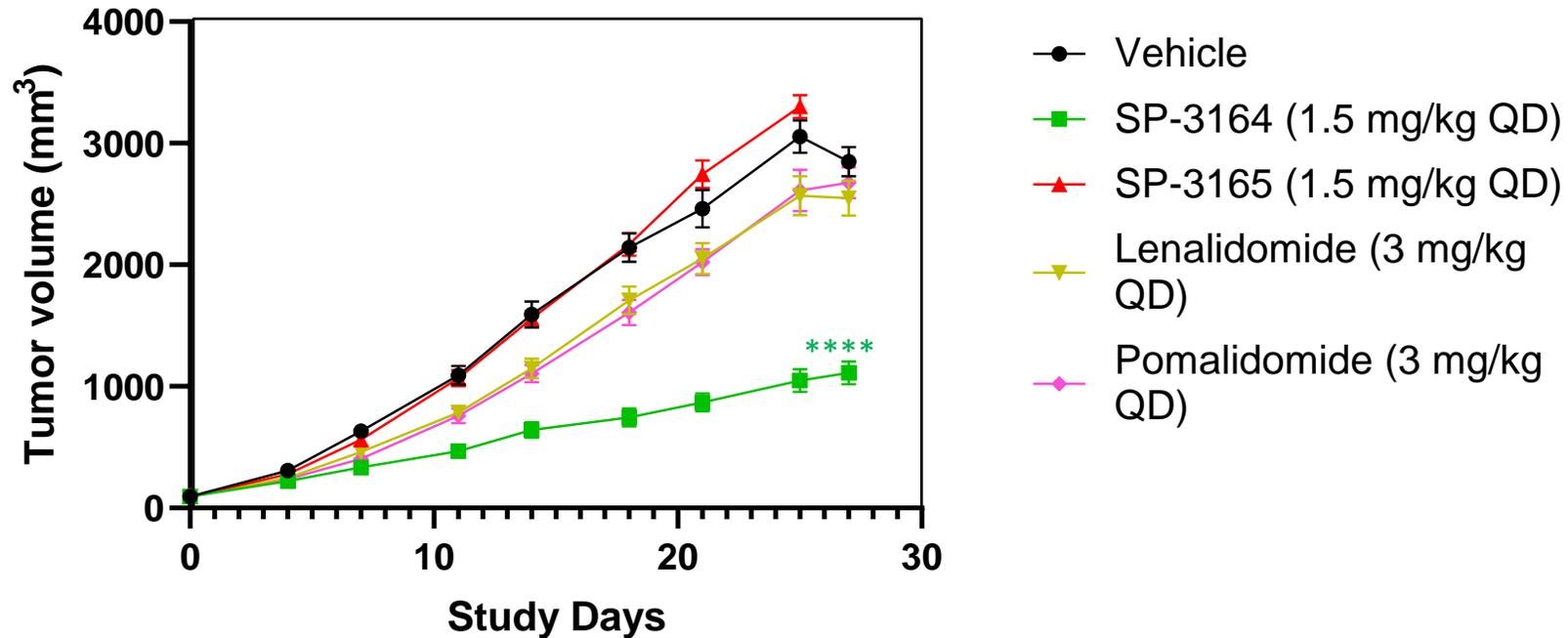
NCI-H929 Xenograft Study (repeat)



- SP-3164: Significant tumor growth inhibition (TGI) compared to vehicle
- SP-3164: Trended towards more TGI compared to CC-122
- SP-3165: No significant TGI, rather a trend towards supporting tumor growth

SP-3164 Shows Significant TGI Compared to Other IMiDs[®] In MM H929 Xenograft Model

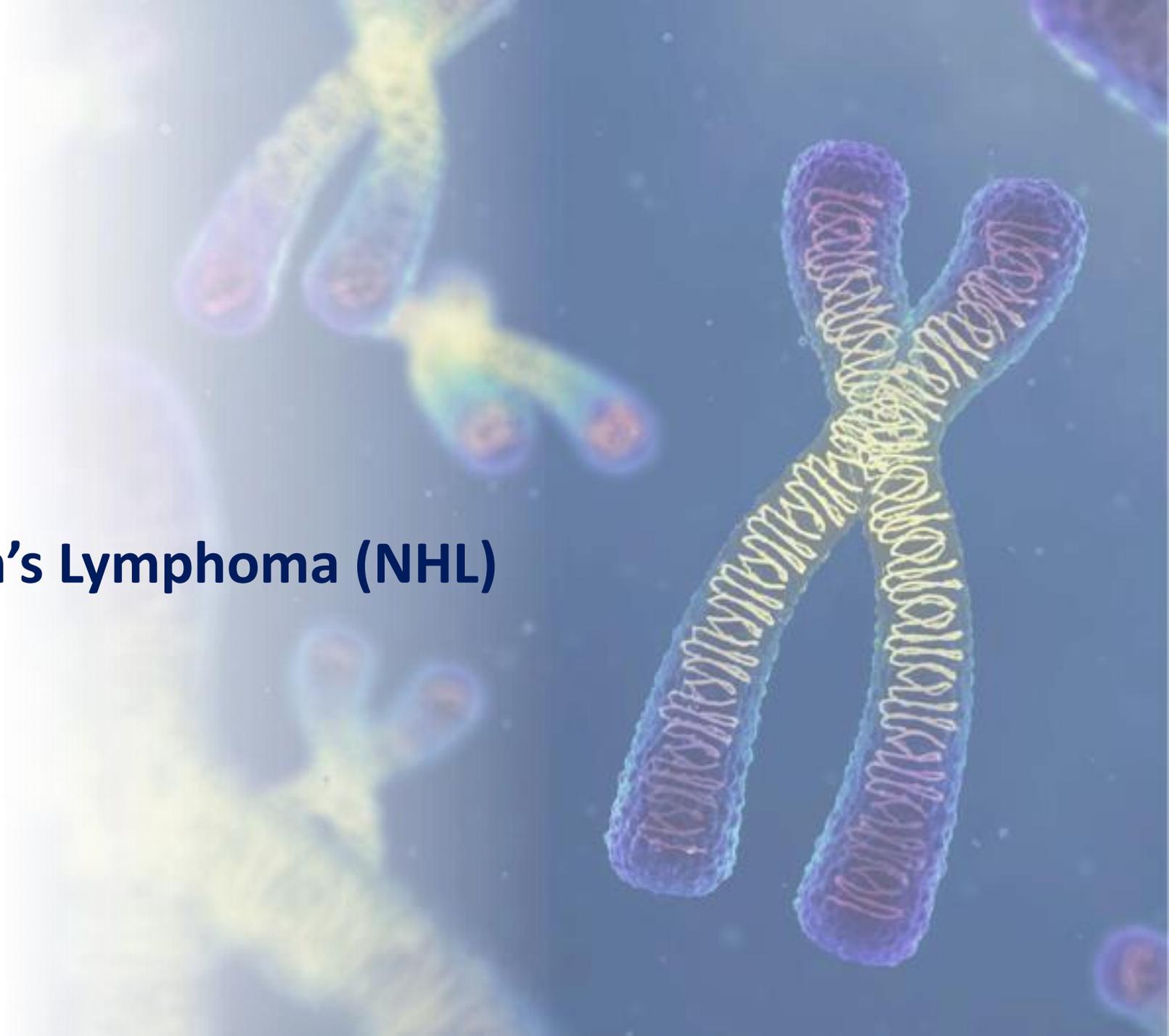
NCI-H929 Xenograft Study



- SP-3164 exhibits significant TGI compared to approved IMiDs for MM¹
- Future studies will evaluate SP-3164 in IMiD-refractory MM cell lines



SP-3164 – Non-Hodgkin's Lymphoma (NHL)

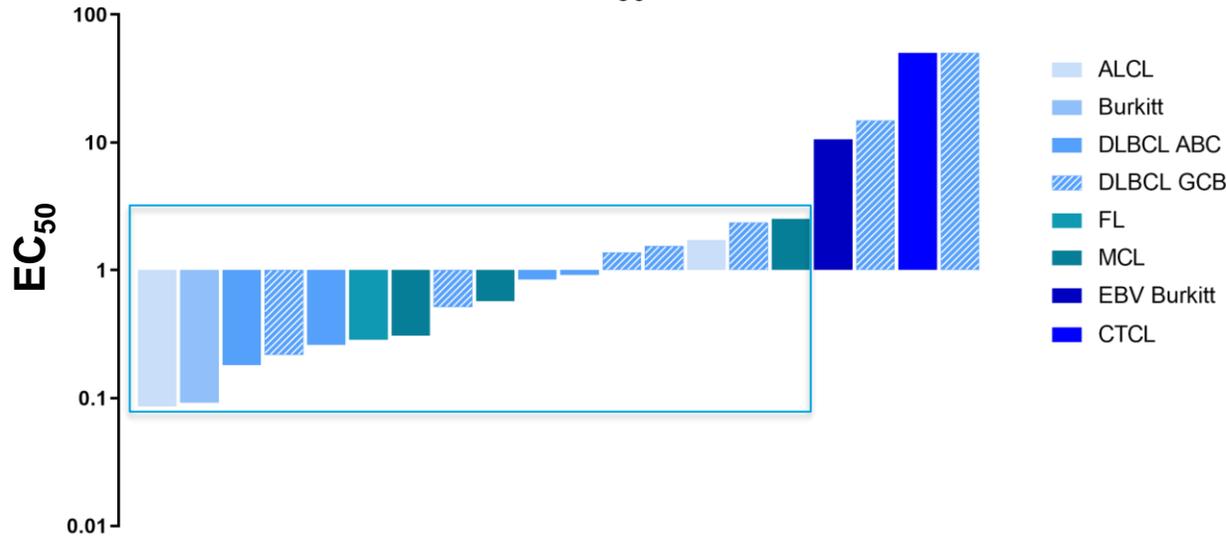


SP-3164 *in vitro* Activity in NHL

Superior to Lenalidomide in Diffuse Large B Cell Lymphoma (DLBCL)

Antiproliferative Activity of SP-3164 in Lymphoma Cell Lines

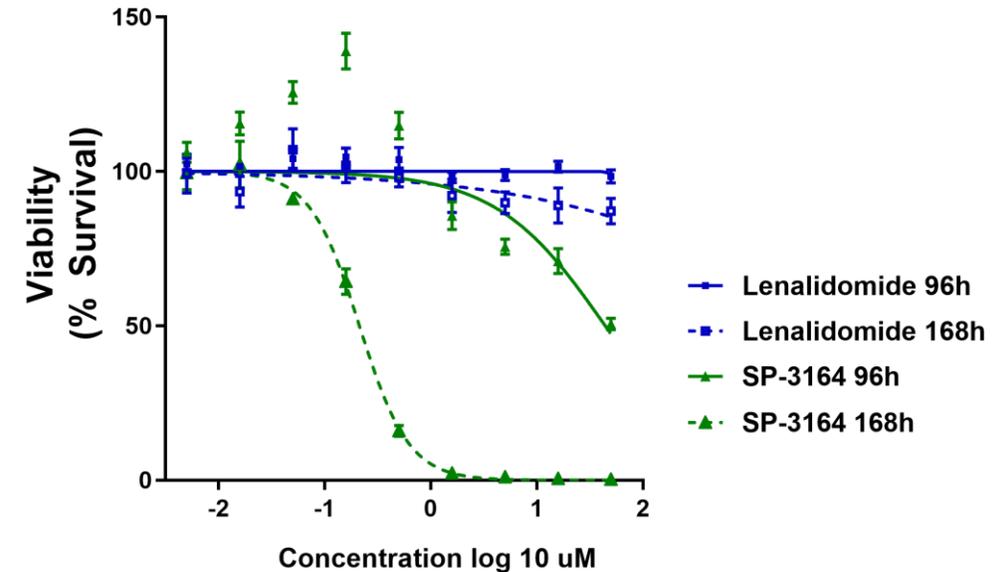
EC₅₀ after 96 hr



In a panel of 20 lymphoma cancer cell lines representing various subtypes, SP-3164 demonstrated potent antiproliferative activity within 96 hrs of dosing in 16 cell lines (average EC₅₀ < 1 μM, range 0.092-2.523 μM).

DLBCL (WSU-DLCL2) Cell Viability (IC₅₀)

% Survival after 96 hr and 168 hrs



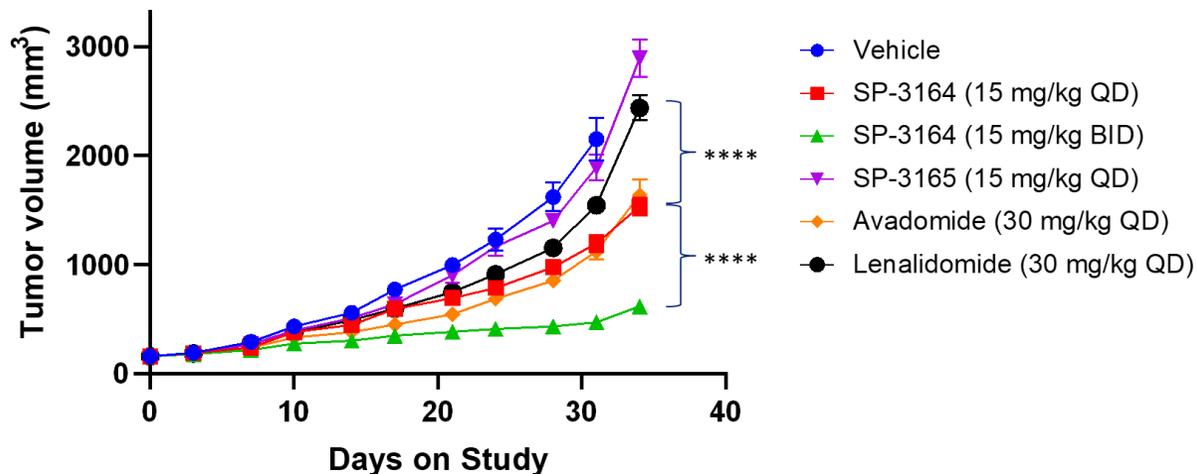
In WSU-DLCL2 cells, increased duration of treatment (168 hr vs 96 hr) revealed increased sensitivity to SP-3164 (IC₅₀ 0.217 μM), but not lenalidomide.



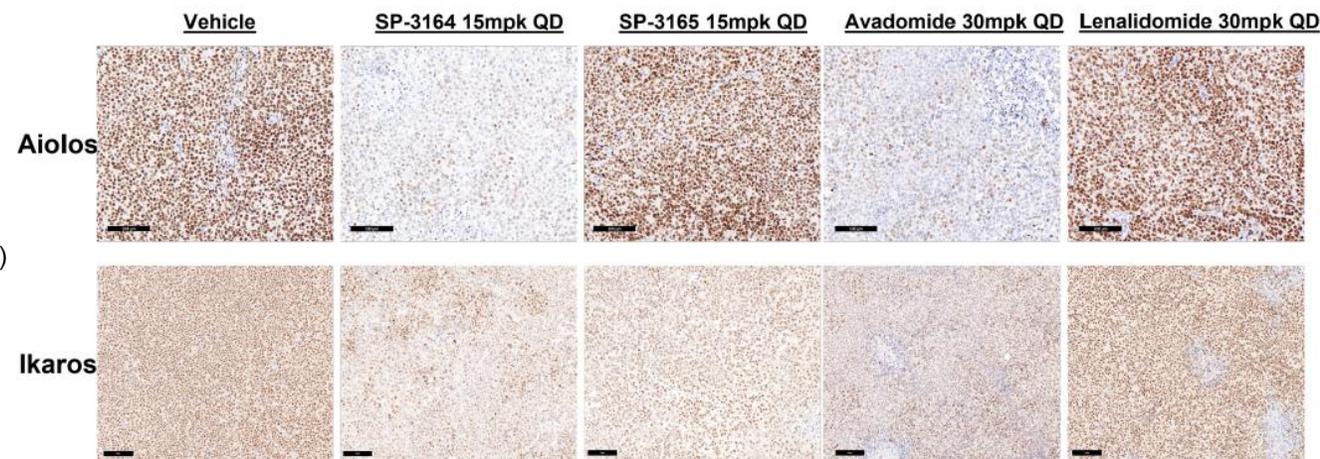
SP-3164 Demonstrates Single-Agent Activity in DLBCL

Mouse DLBCL (WSU-DLCL2) Xenograft Model

Tumor Volume \pm SEM



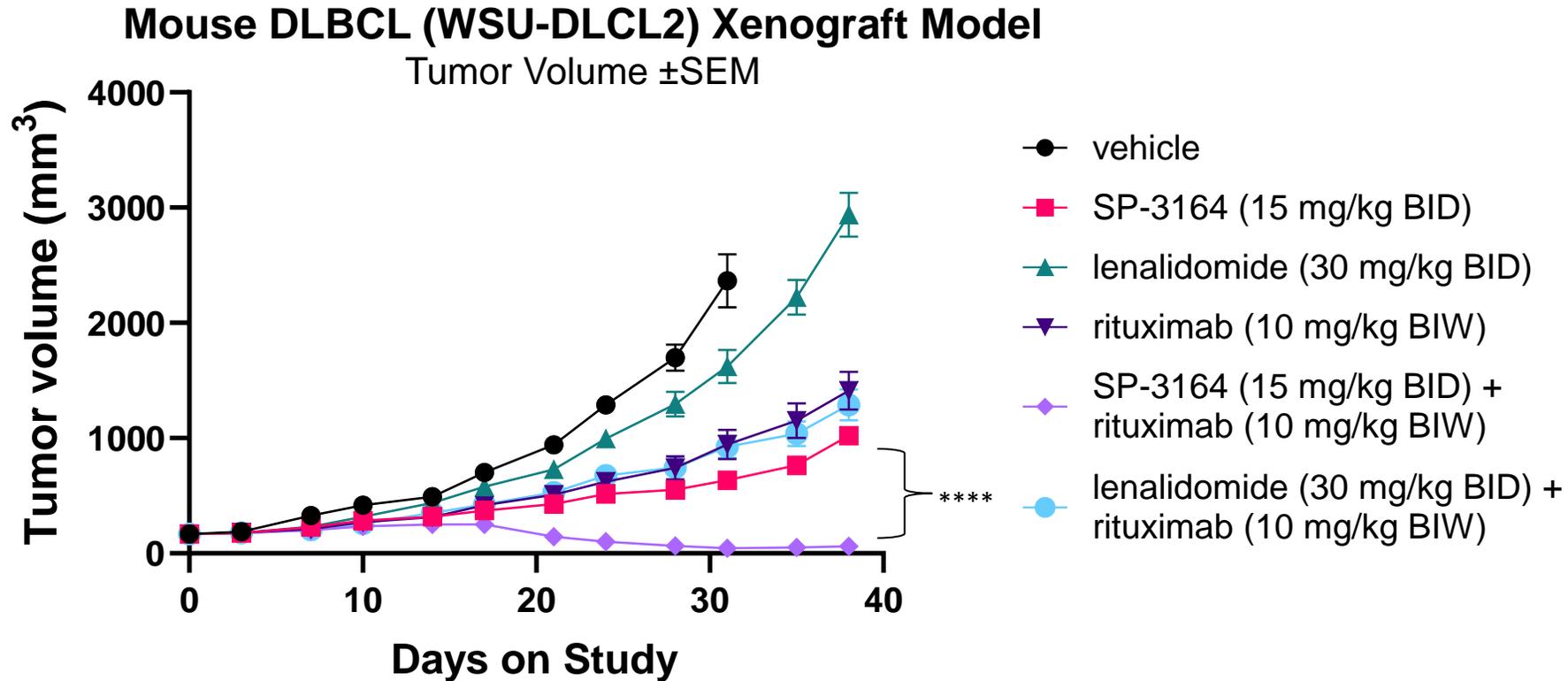
Degradation of Aiolos and Ikaros in tumors



- SP-3164 demonstrated pronounced antitumor activity as single agent outperforming lenalidomide and comparable to avadomide while SP-3165 lacked significant antitumor activity (**** $p \leq 0.0001$).
- Due to SP-3164's shorter $t_{1/2}$ vs. avadomide, SP-3164 was studied BID resulting in the largest inhibitory effect.
- Treatment with SP-3164 caused degradation of Aiolos and Ikaros in tumors (representative IHC images at $t=6hr$).



SP-3164 Shows Synergistic Activity with Rituximab in DLBCL

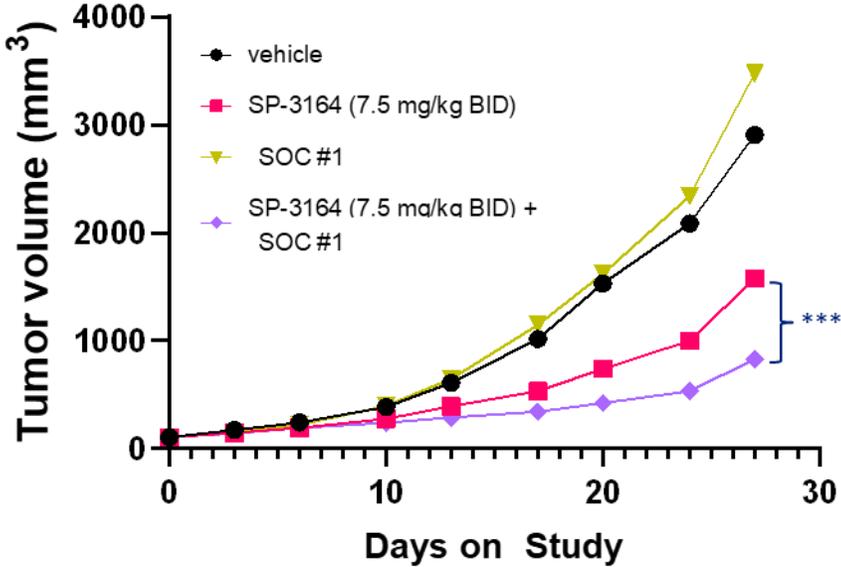


- SP-3164 combination with rituximab was compared an approved regimen, lenalidomide and rituximab, in the WSU-DLCL2 DLBCL model.
- Combination of SP-3164 and rituximab resulted in sustained regressions with 50% of mice being tumor-free, significantly better than the lenalidomide and rituximab regimen (**** $p \leq 0.001$).



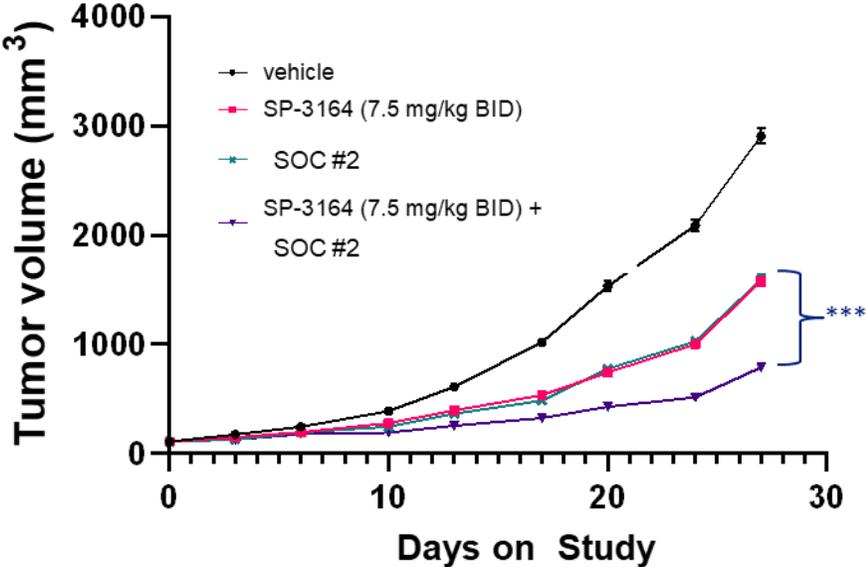
SP-3164 Shows Improved Activity Over Other I/A Degraders And Significant Synergy With SOC Agents in Follicular Lymphoma (FL)

Mouse FL (DOHH2) Xenograft Model
SP-3164 Mono and Combo Treatment
 Tumor Volume ±SEM



- SP-3164 shows single agent TGI in a FL mouse model that is significantly better than the approved agent¹ at significantly lower doses
- SP-3164 helps sensitize to the approved SOC² agent

Mouse FL (DOHH2) Xenograft Model
SP-3164 Mono and Combo Treatment
 Tumor Volume ±SEM



- SP-3164 shows single agent TGI in a FL mouse model that is equivalent to an approved agent²
- In combination with the approved drug¹, SP-3164+SOC results in improved TGI

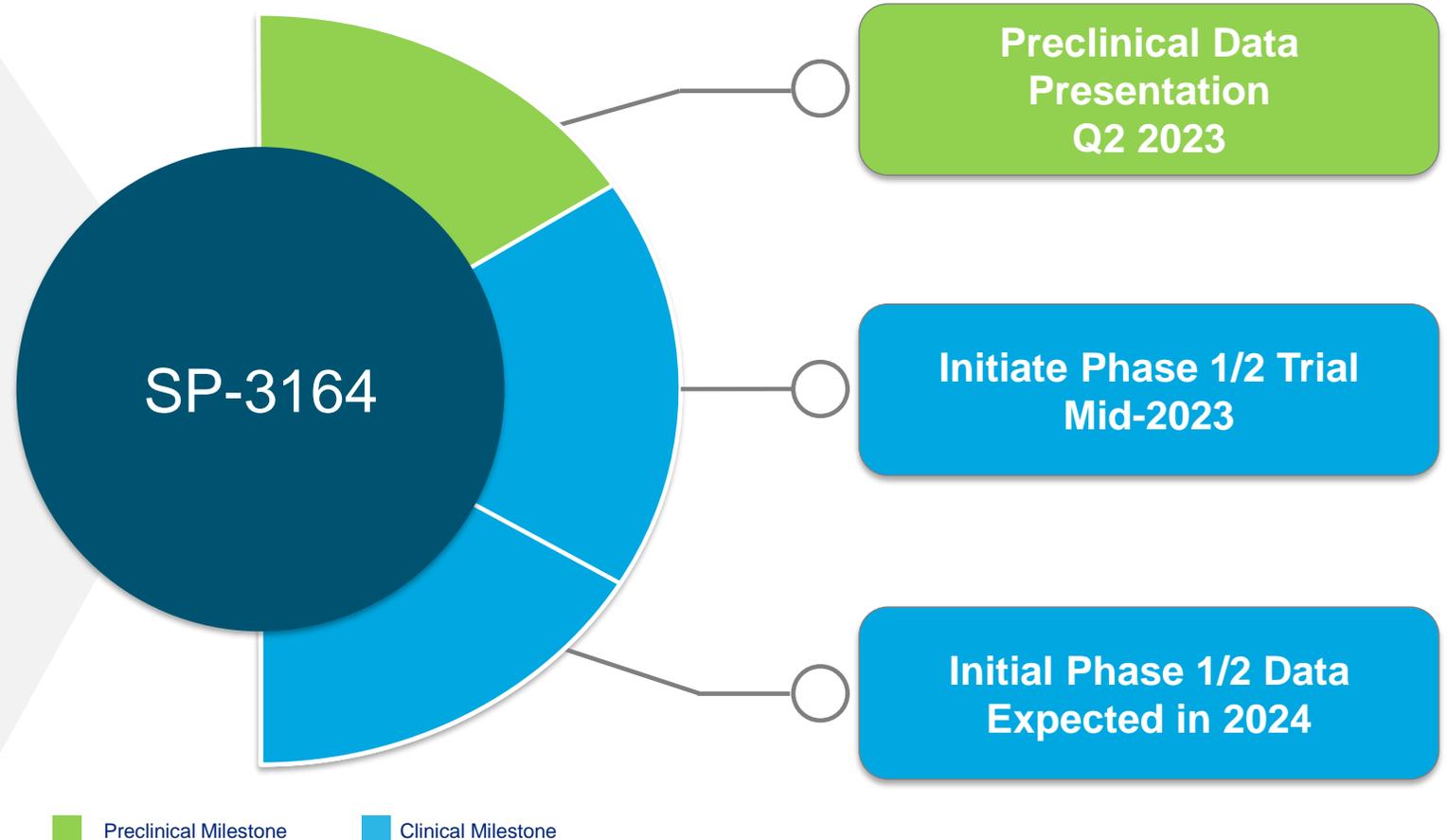
SP-3164 Current Status and Upcoming Milestones

Complete

- FDA Pre-IND meeting process
- GMP API batch – on stability
- Preclinical in vitro and in vivo studies MoA and efficacy
- Dose ranging and in life GLP toxicology
- Multiple Myeloma and Lymphoma Advisory boards

Ongoing and Upcoming

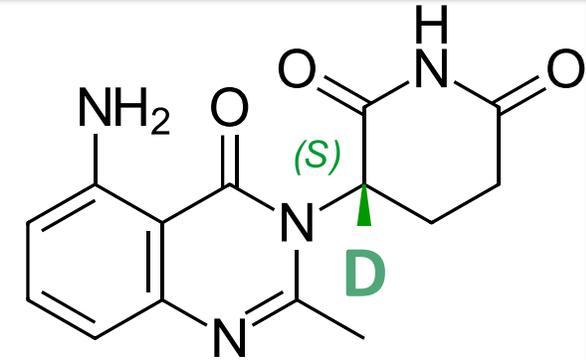
- Drug product formulation development
- Extensive in vivo single agent, combination therapy and comparator studies
- IND submission and activation
- Phase 1/2 First Patient Enrolled



SP-3164 Is The Preferred Enantiomer Of Avadomide And Is The Next-generation Cereblon-binding Targeted Protein Degradator

SP-3164 Highlights

- ✓ An NCE with its own, issued composition of matter patent (exp. 2039 w/ extensions)
- ✓ Differentiated cereblon-binding molecular glue
 - Only glue with stabilized active enantiomer
 - Improved activity in preclinical models
 - Potential for an improved therapeutic window (PK and safety advantages)
 - Precision medicine for improved responses
- ✓ Clear, de-risked clinical strategy builds upon established avadomide data
 - Target indications with high likelihood of PoC monotherapy activity and quickly move up in treatment line with appropriate combinations



SP-3164

Deuterated S-avadomide

- ✓ Anti-cancer activity
- ✓ Immune stimulation

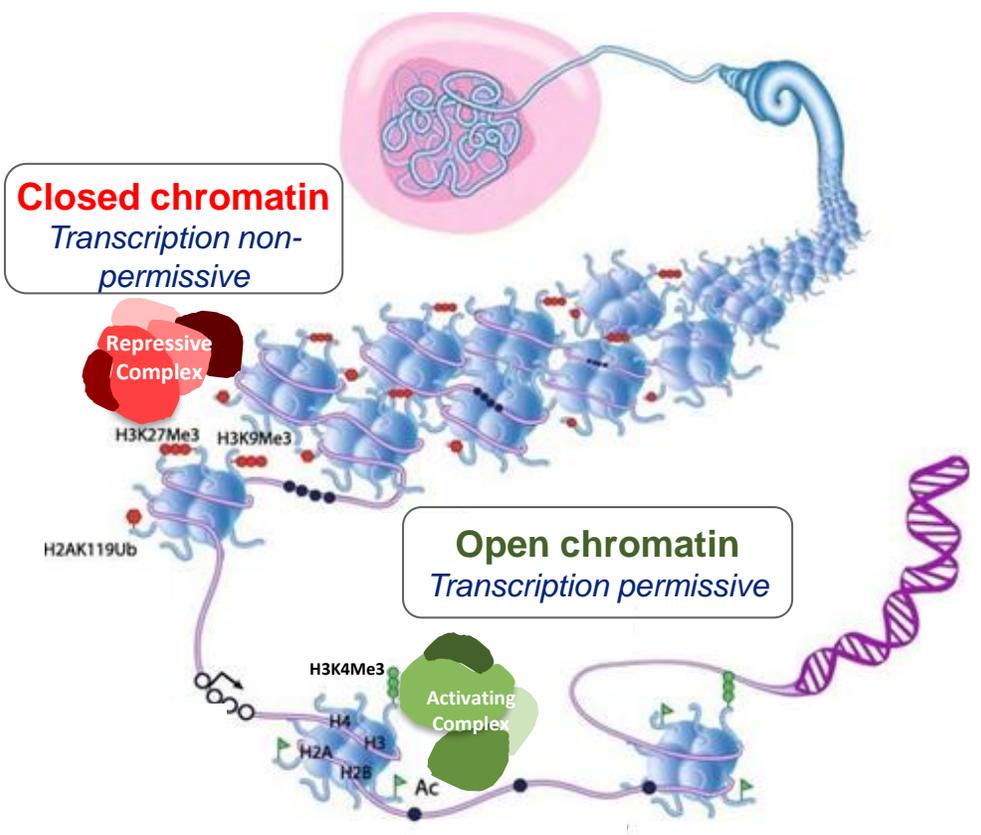
Seclidemstat

Overview

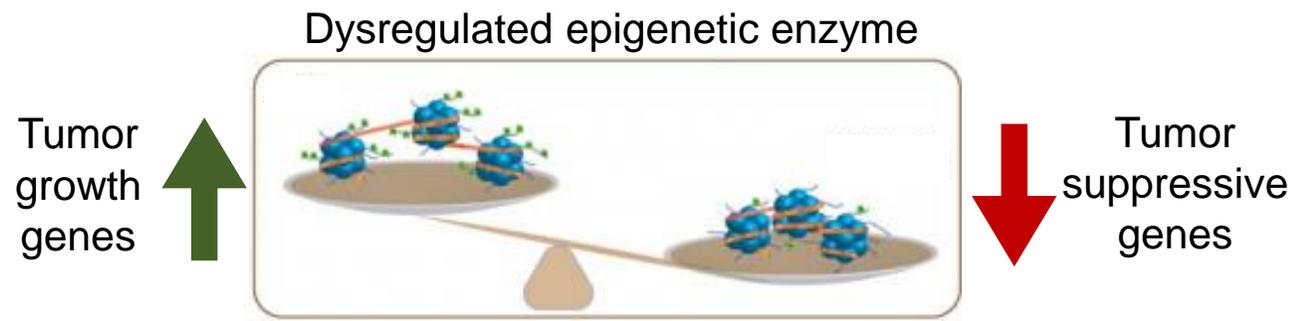


Epigenetic Enzymes Are Attractive Targets For Cancer Therapy

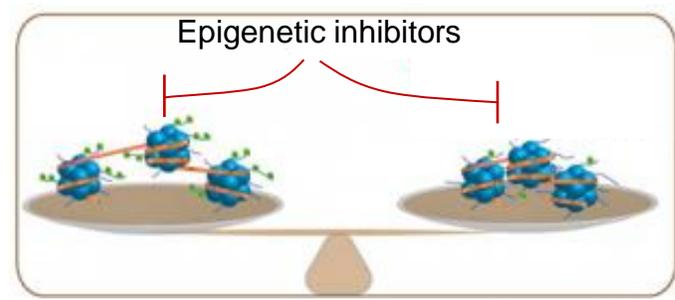
Epigenetic modifying enzymes affect gene expression by manipulating the chromatin structure



Dysregulated epigenetic enzymes can disrupt the transcriptional balance and lead to cancer development



Drugs that correct dysregulated epigenetic enzymes can help treat cancer by restoring to a balanced transcriptional state



Adapted from Holliday, H. Breast Cancer Research 2018

Adapted from Marcin et al. Biomed Intel 2018.

LSD1 - A Validated Target For Cancer Therapy

Lysine Specific Demethylase 1 (LSD1) affects gene expression through enzymatic activity and scaffolding properties (protein-protein interactions), making it an attractive target for solid tumors and hematological cancers.

| LSD1 in Normal Cells and Cancer Cells ¹ | |
|--|---|
| Normal Cells | <ul style="list-style-type: none">• LSD1 is necessary for stem cell maintenance and cell development processes (e.g., blood cells) |
| Cancer Cells | <ul style="list-style-type: none">• LSD1 is over expressed• LSD1 acts incorrectly to silence or activate genes leading to disease progression• Validated target: LSD1 CRISPR deletion often detrimental to cancer cells |



Seclidemstat (SP-2577) reversibly inhibits LSD1

- Reverses incorrect gene expression, killing or preventing the growth of cancer cells
- Inhibits both the enzymatic and scaffolding activity

Companies with LSD1 inhibitors in clinic:



¹Majello, B. *Cancers* 2019. ²Appendix B

More Comprehensive Inhibition of LSD1 Positively Impacts Therapeutic Activity



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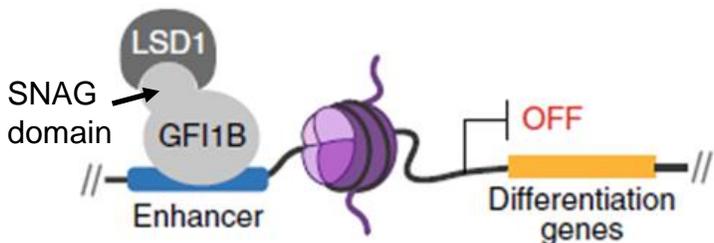
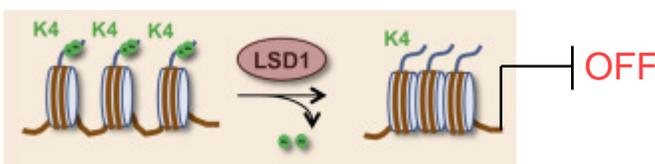
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Enzymatic activity – Demethylation
Impact: Moderately alter gene expression

Partial scaffolding* inhibition of LSD1 – protein interaction
Impact: Alter gene expression in cancers (AML, SCLC) driven by SNAG domain proteins (e.g. GFI1B)

Broader scaffolding inhibition of LSD1 – protein interaction
Impact: Potential efficacy in broader range of cancer types, destabilize LSD1 and complexes





- ✓ Differential activity
- ✓ Reduces LSD1 expression
- ✓ Favorable Toxicology Profile

 and competitors

 and competitors

*scaffolding properties – protein to protein interactions



A microscopic image of Ewing Sarcoma cells, showing several rod-shaped cells with characteristic blue-purple staining and internal structures. The cells are arranged in a cluster, with some showing a distinct X-shaped structure. The background is a light blue gradient.

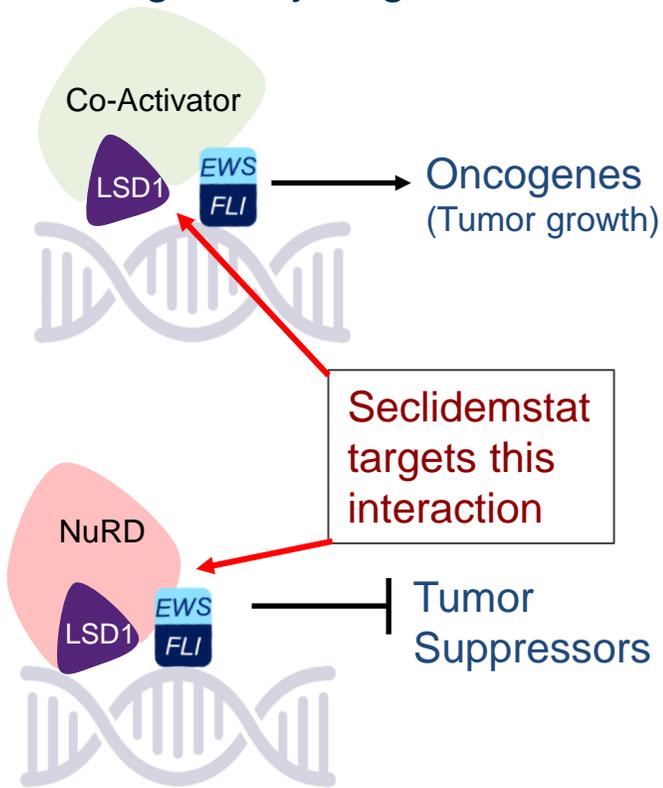
SPEED TO MARKET

Seclidemstat in Ewing Sarcoma

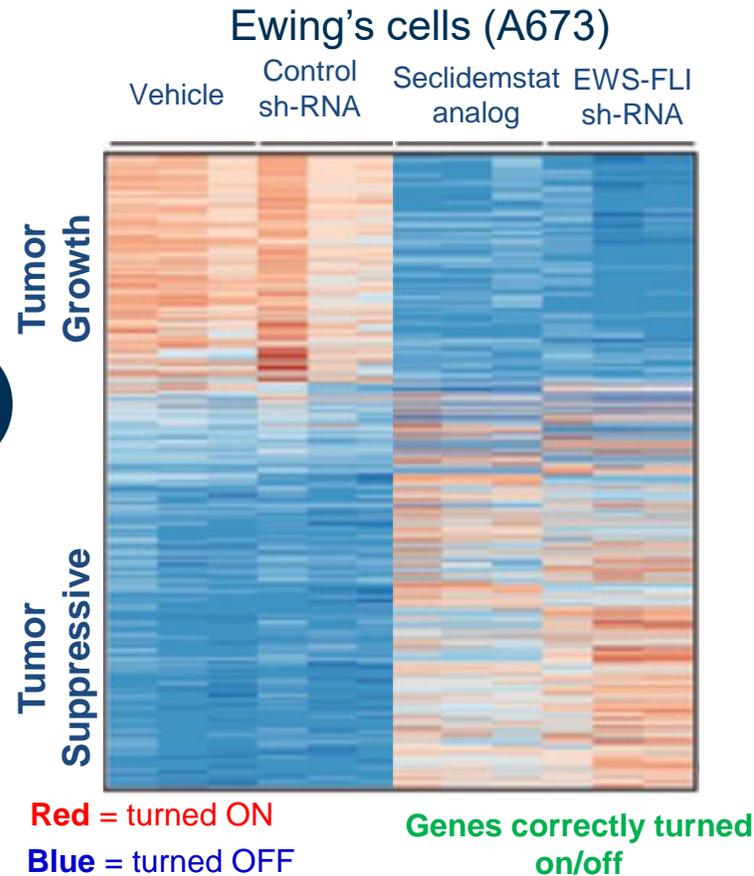
Targeting The Root Cause Of Ewing Sarcoma Via LSD1 Inhibition

Ewing sarcoma is driven by an easily diagnosed chromosomal translocation, i.e., EWS-FLI

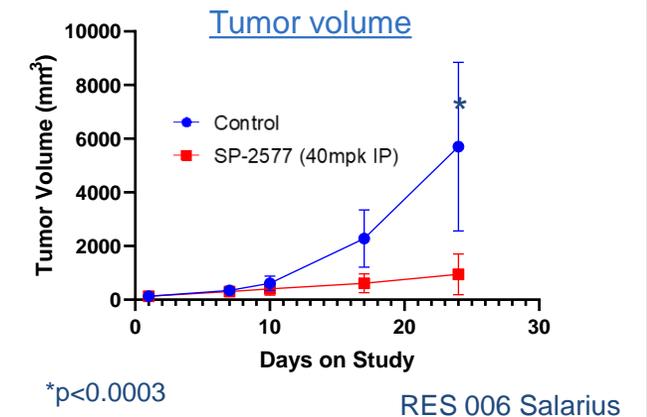
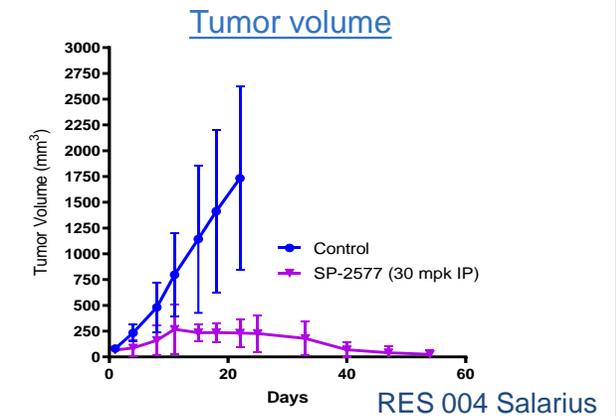
Incorrect transcription factor leads to gene dysregulation



Seclidemstat corrects gene expression



Potent anti-tumor activity in SKNMC (Ewing sarcoma cells) *in vivo* studies



Ewing's Sarcoma: Unmet Need, Meaningful Opportunity

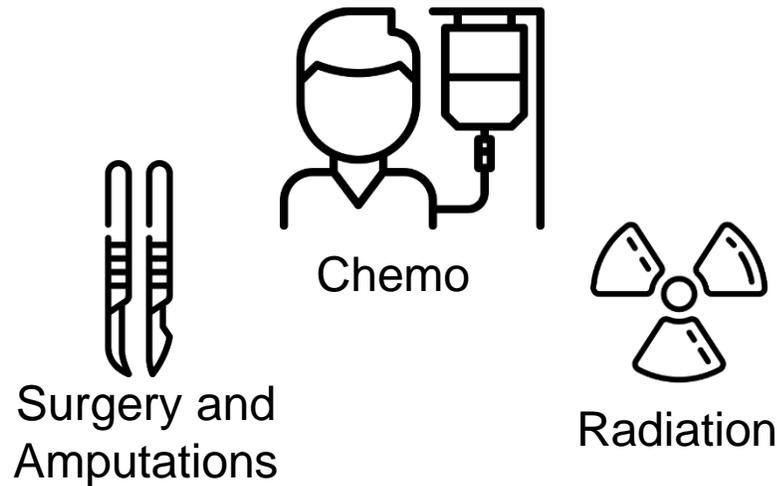
Diagnosis



~500 US patients diagnosed each year with a median age of 15 at the time of diagnosis

- 75% localized¹
- 25% with metastasis¹

Standard-of-Care



- ~40% of patients are refractory or relapse²
- 70-90% 5-year mortality rate²
- No standardized second-line treatment

Salarius' Vision

An effective, non-toxic, oral treatment

- Accelerated U.S. approval
- Rapid market uptake
- \$200M+ global sales³ (est.)
- Possible PRV worth \$80M-\$150M

- ✓ Fast track designation
- ✓ Orphan drug designation
- ✓ Rare pediatric drug designation

² Van Mater, et al. Oncotargets (2019)



1st Relapse Patients Doubled rEECur Progression Comparator And Patients with Disease Control Had No Observed Disease Progression

Results of Salarius Sponsored Phase 1/2 Salarius Trial for Treatment Ewing Sarcoma (10/31/2022)
Sarcoma clinical trial currently on partial clinical hold

| | CRC ¹ | PRc ¹ | ORR | SDc ¹ | DCRc | PD | mTTP Months | Range Months |
|---|------------------|------------------|---------|------------------|---------|----|-------------|--|
| 1 st Relapse Pts (5) | 1 | 1 | 2 (40%) | 1 | 3 (60%) | 2 | 7.4 | 1.4 to 13.8 |
| 2 nd Relapse Pts (8) | | 1 | 1 (13%) | 1 | 2 (25%) | 6 | 1.5 | 0.7 to 5.1 |
| 1 st and 2 nd Relapse Pts (13) | 1 | 2 | 3 (23%) | 2 | 5 (38%) | 8 | 1.6 | 0.7 to 13.8 |
| 1 st and 2 nd Relapse Pts w/ DCRc (5) | | | | | 5 (38%) | | 7.4 | 3.1 to 13.8 No Observed PD ² |
| rEECur (primarily a 1 st relapse Ewing sarcoma data set ³) | | | | | | | 3.5 mPFS | 95% CI 2.5 to 5.1 |
| Salarius (1 st Relapse Patients) | | | | | | | 7.4 mTTP | |

¹ Patient status confirmed (c) by both C2 and C4 scans. ² Among 5 patients with DCRc while on study: 1 pt WD at 3.1 months with 32% PRc due to partial clinical hold; 1 pt WD at 5.1 months with 11% reduction SDc due to a nondrug unrelated SAE; 1 patient WD at 7.4 months with CRC; 1 patient WD at 12.8 months with 80% PRc (elected RT consolidation treatment); 1 patient at 13.8 months continues treatment with SDc. ³ ~80% Primary Refractory or 1st Relapse Patients and ~20% 2nd Relapse Patients.



A microscopic view of chromosomes, showing several X-shaped structures in various colors (purple, blue, yellow, green) against a dark blue background. The chromosomes are arranged in a somewhat circular pattern, with one large X-shaped chromosome in the foreground and several smaller ones in the background.

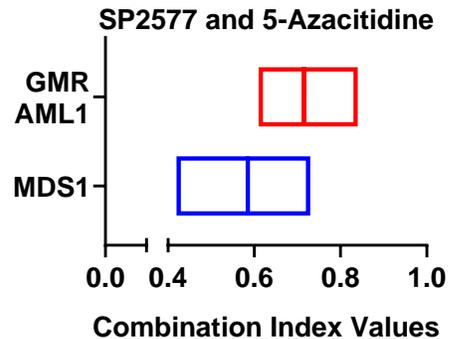
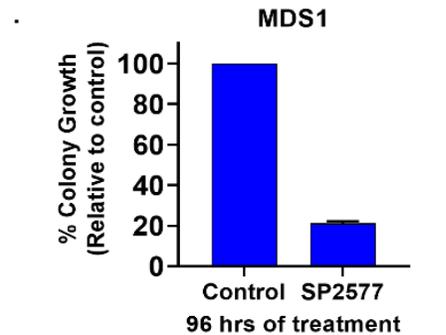
MARKET EXPANSION

Hematologic Cancers

Seclidemstat + Azacitidine Shows Activity In Hematologic Or Blood Cancers Cell Lines

Hematologic Cancers¹

Seclidemstat inhibits MDS cell growth and shows synergy with azacitidine



Phase 1/2 investigator-initiated study enrolling patients at MD Anderson Cancer Center in myelodysplastic syndromes & chronic myelomonocytic leukemia

Clinicaltrials.gov Identifier: NCT04734990

Clinical data update provided at ASH 2022

Clinical trial currently on partial clinical hold

Primary Objectives

- Safety, tolerability and maximum tolerated dose
- Overall response rate

Secondary Objectives

- Overall survival, duration of response, relapse-free survival, leukemia-free survival and safety
- Correlative studies including correlation of response with disease subtypes, genomic profile and *in vitro* studies

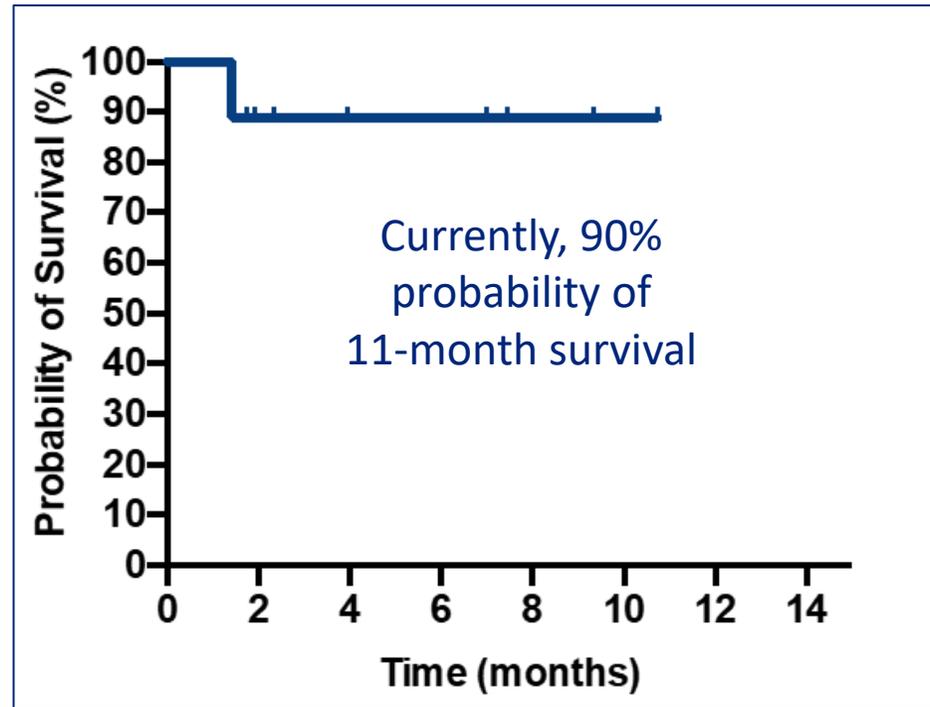
1. Seclidemstat + azacitidine trial is open for enrollment



The Combination of Seclidemstat with Azacitidine Shows Initial Signs of Potential Activity Treatment of MDS and CMML

ASH Poster Presentation Results from the Investigator Sponsored MD Anderson Trial for Treatment of MDS and CMML¹ with prior HMA² treatment

| Dose | Outcome |
|------------|------------------------|
| 150 mg BID | PD |
| 150 mg BID | No Response |
| 150 mg BID | mCR with SCT |
| 300 mg BID | mCR |
| 300 mg BID | mCR + HI + PI |
| 300 mg BID | NR |
| 450 mg BID | mCR + HI |
| 450 mg BID | SD |
| 450 mg BID | Too early for response |



- 50% ORR (4/8) including curative transplant
- Patient Disposition:
 - 3 patients on study
 - 1 to undergo allo-SCT
 - 3 off due to no response
 - 2 patients off study due to progression
- Note: Overall Survival in HMA failure patients is 4-6 months

¹ Patients previously failed azacitidine or decitabine. SCT: stem cell transplant, CMML: chronic myelomonocytic leukemia, MDS: myelodysplastic syndrome, T-MDS: therapy related MDS, mCR: marrow complete response, pCyR: partial cytogenetic response, SD: stable disease, PD: progressive disease, BM: bone marrow; HI: Hematologic Improvement; PI: Platelet improvement

² HMA Hypomethylating Agent (azacitidine, decitabine)



Financial Overview and Management



Financial Overview



Sufficient Cash Position

- Cash position of \$12.1M as of end of Q4 2022
- Sufficient cash to fund operations through near term milestones through Q3, 2023



Capitalization Structure

- No debt or structured obligations on the balance sheet



Low Fixed Costs

- Low head-count and associated overhead costs
- R&D costs maintained at healthy levels relative to company cash position



Corporate Snapshot

- Ticker: NasdaqCM:SLRX
- Common Shares Outstanding: 2.5M



Seasoned Leadership Team



David J. Arthur
Chief Executive Officer

Lilly  **Boehringer Ingelheim**



Nadeem Q. Mirza, MD, MPH
Senior VP Clinical Development

SANOFI  **abbvie**



Stephen Horrigan, PhD
Consulting Chief Scientific Officer

 **Iteron**  **NOBLE LIFE SCIENCES**



Mark Rosenblum
Chief Financial Officer

ADVAXIS
 **Deloitte.**



Rebecca Griffith-Eskew
VP Clinical Operations

 **WORLDWIDE CLINICAL TRIALS**  **Boehringer Ingelheim**



Daniela Y. Santiesteban, PhD
Director Protein Degradation Development

 **CRFP**  **Georgia Tech**

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Omeros Corporation
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Thank You