Salarius PHARMACEUTICALS

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

**NasdaqCM:SLRX**

July 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 activated in July 2023. A Phase 1/2 trial is planned to begin in the 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 March 31, 2023, cash of $9.3M and no debt

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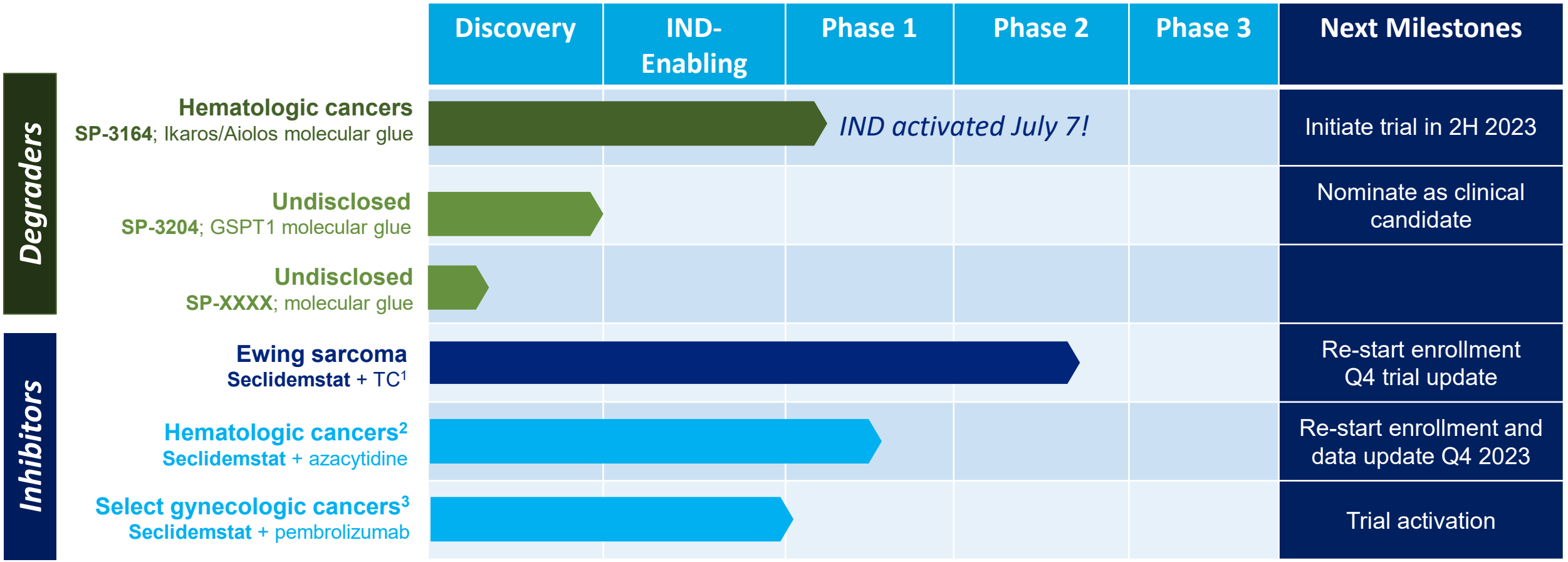
# Pipeline Overview

## *Protein Inhibition and Protein Degradation*







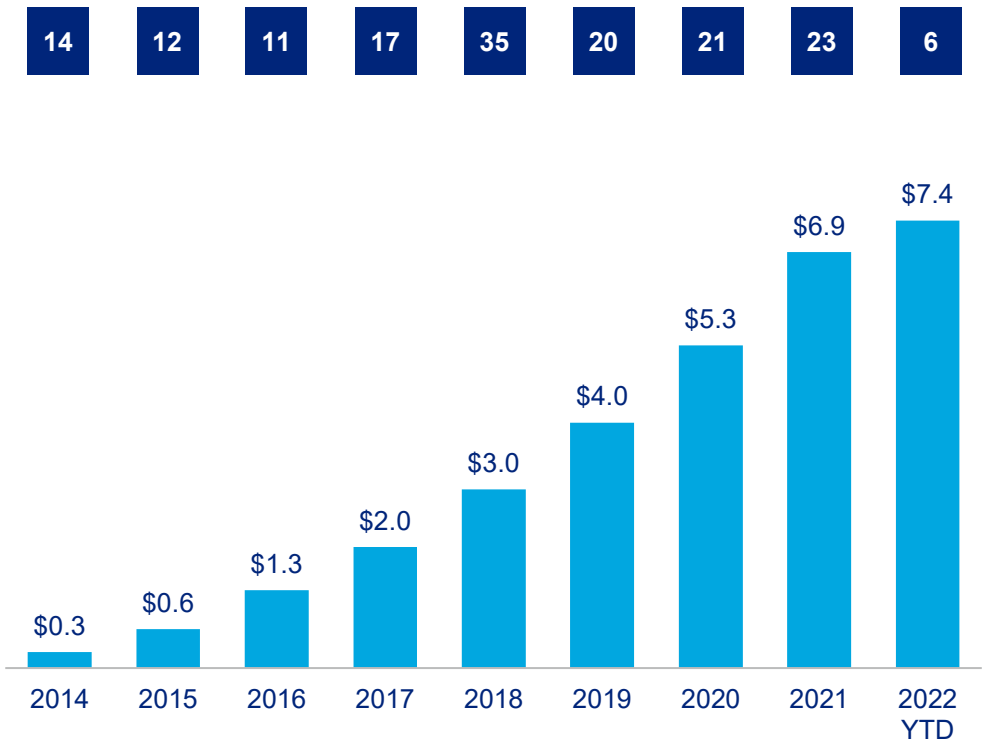
[1] Topotecan and cyclophosphamide [2] Investigator initiated trial active at MD Anderson Cancer Center treating patients with Myelodysplastic Syndromes and Chronic Myelomonocytic Leukemia [3] Investigator initiated trial – Clinical trial agreement not yet finalized.

# Targeted Protein Degradation Space Has Witnessed Tremendous Growth

## Cumulative Capital Invested in Development of Targeted Protein Degrader Therapies[1,2]

*($B)*

*Number of Investments*




## Large Biopharma Companies Have Moved Aggressively to Gain Exposure[3]

*Selected targeted protein partnerships and strategic collaborations since 2015*

SANOFI: nurix, KYMERA

BAYER: ARVINAS, VIVIDION Therapeutics

Pfizer: RANOK Therapeutics, ARVINAS

AMGEN: Plexium

Roche: ARVINAS, BAYER, VIVIDION Therapeutics, C4 Therapeutics

gsk: IDEAYA BIOSCIENCES, KYMERA

janssen: evotec

Lilly: FOGHORN THERAPEUTICS, LYCIA THERAPEUTICS, SEED THERAPEUTICS

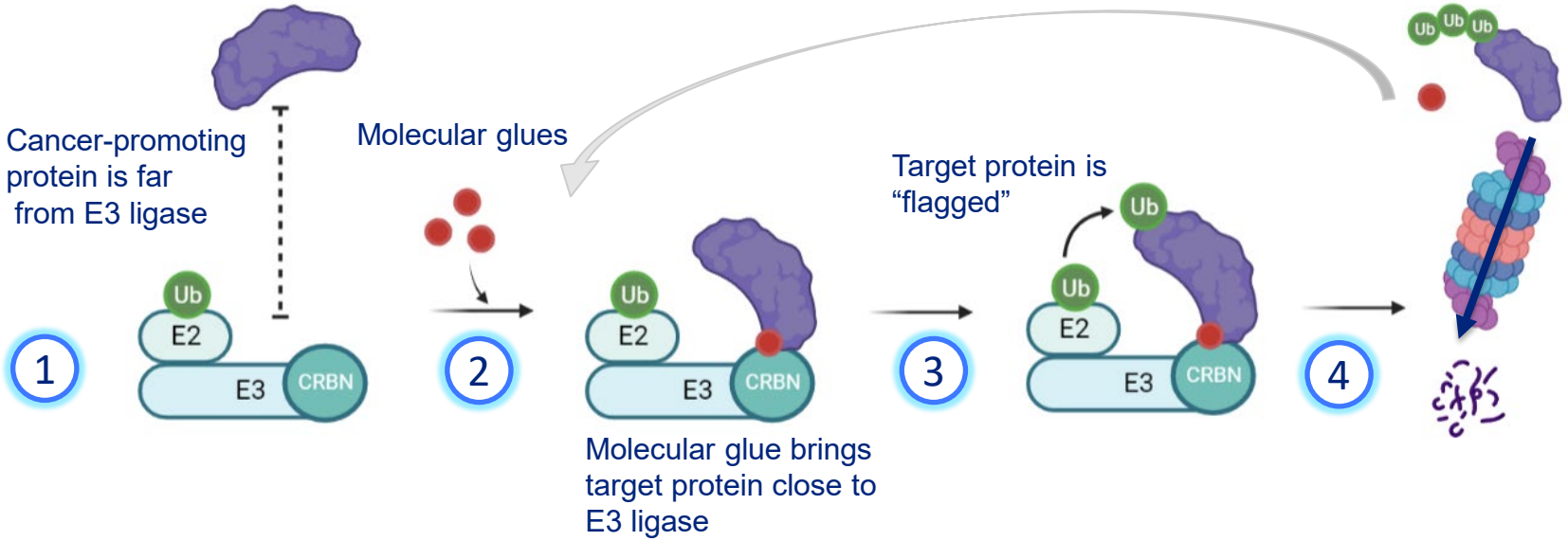
NOVARTIS: DUNAD therapeutics, ORIONIS™ BIOSCIENCES

…. 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



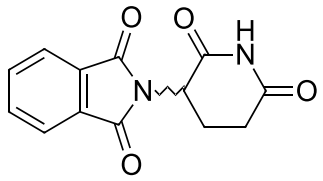

## Advantages

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

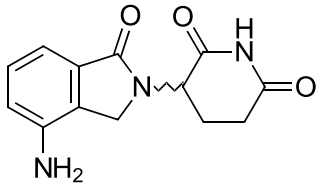
# First-Generation Protein Degraders

**IMiDs®** (Immunomodulatory Drugs) – Approved for hematological malignancies

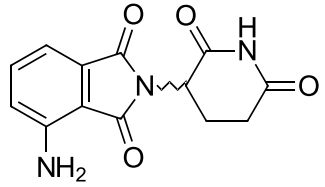
- $16B in sales in 2021
- All exist as racemic mixtures



Thalidomide



Lenalidomide

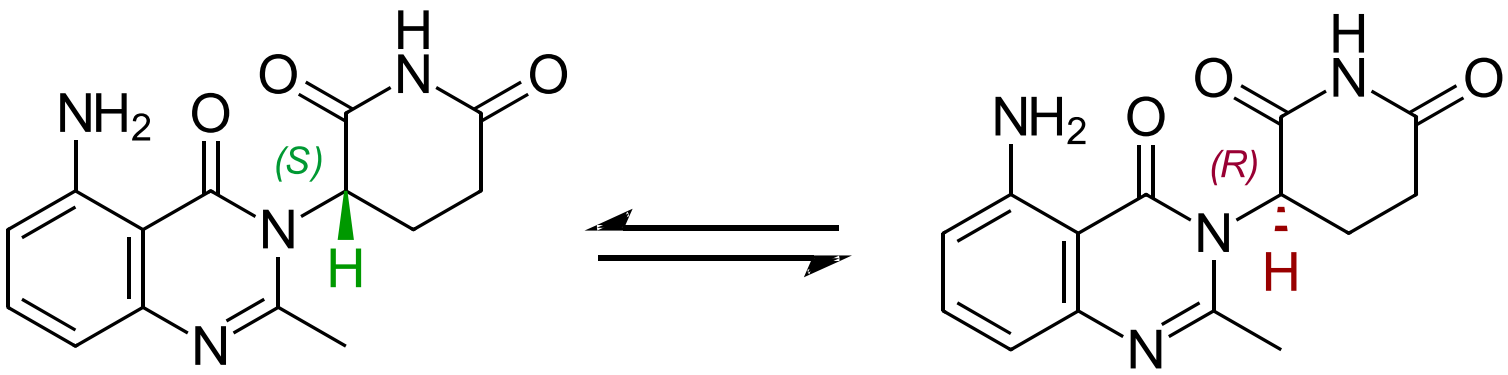


Pomalidomide

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

# Avadomide, a 2nd Generation Extensively Studied Degrader

*Exists as a Mixture of 2 Species*




**S-enantiomer** **R-enantiomer**

## Avadomide (CC-122)

a 1:1 mixture of 2 interconverting species

✓ Studied in 10 clinical trials in >400 patients

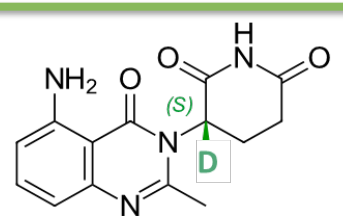
✓ 28% Overall Response Rate as a monotherapy for lymphomas

CELMoD – Cereblon E3 Ligase Modulation Drugs, a registered trademark of Celgene / BMS

# SP-3164 is New Chemical Entity that Shows Potential to Improve Upon Avadomide's Efficacy and Safety

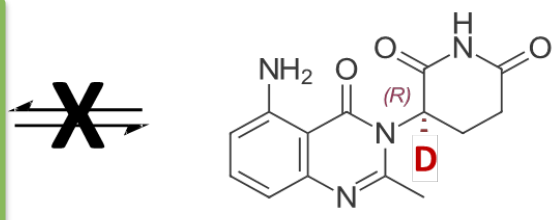
## *Development of SP-3164:*

*SP-3164 utilizes our deuterium-enabled chiral switching (**DECS**) platform to lock the enantiomer in place and therefore exists as only the active, S-enantiomer with minimal interconversion*




**SP-3164**
Deuterated S-avadomide

- ✓ Protein degradation
- ✓ Anticancer activity
- ✓ Immune stimulation




**SP-3165 (inactive)**
Deuterated R-avadomide

- No protein degradation or anticancer activity
- *Potentially* supports tumor growth

Given avadomide's compelling clinical data, Salarius developed SP-3164, which is the stabilized, active enantiomer of avadomide, with potential for improved therapeutic properties

- SP-3164 is a new chemical entity with its own composition of matter patent (expires in 2039[1])
  - Developed independently of Celgene/Bristol Myers Squibb (no relationship with Salarius)
- SP-3164 can build off of avadomide's validated clinical development plan with improved efficacy, as supported by preclinical data, and potentially improved safety




1. Includes potential patent term extension (PTE) of up to 5 years in the US.

# SP-3164 Addresses a High Unmet Need in DLBCL and Has a Clear Development Path To A Launch Indication

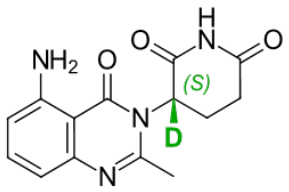
## The Problem

**About 3,200 R/R DLBCL patients are ineligible for intensive treatments and face survival outcomes as low as six months**

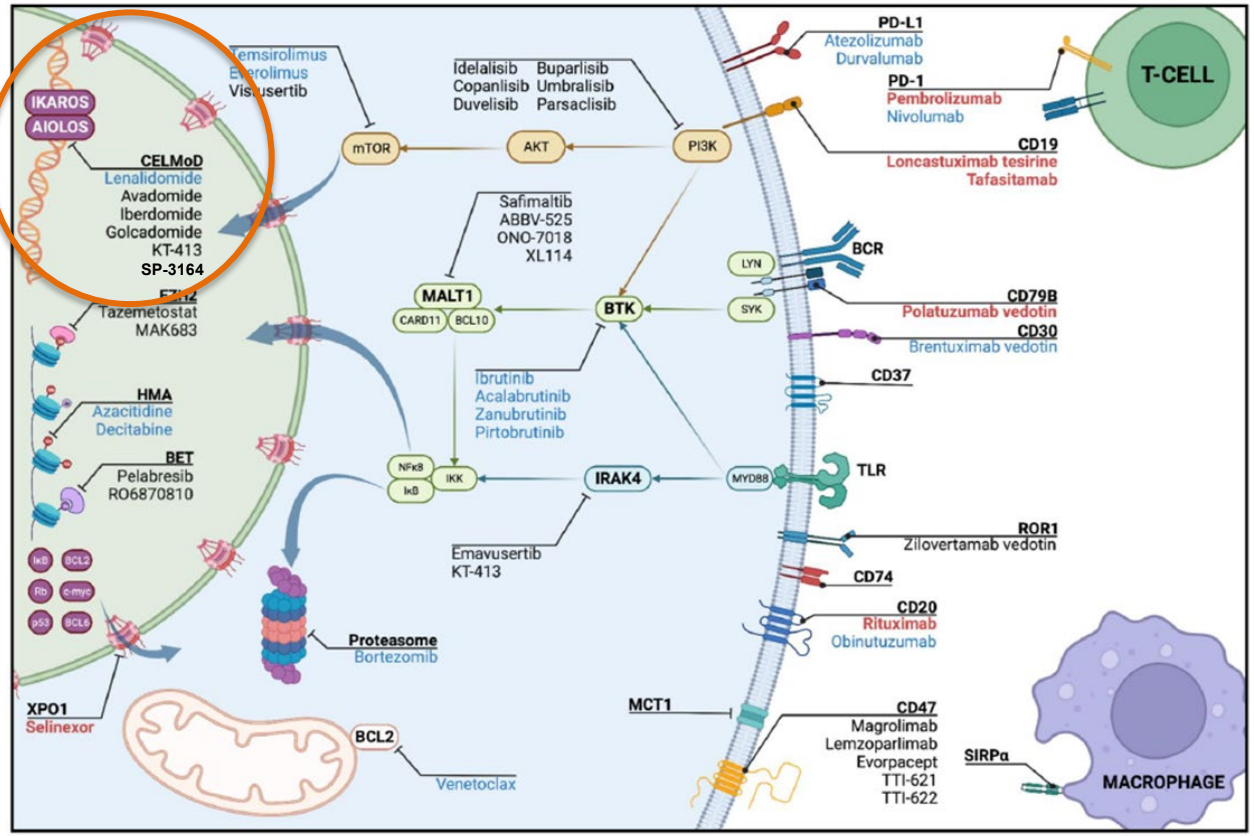
- Elderly patients may not tolerate SOC treatments
- Not all patients are eligible for curative treatments, e.g. CAR T
- Bispecifics have shown durable CRs, but >50% progress quickly or do not respond

## Salarius's Solution

Salarius has developed **SP-3164**, a targeted Ikaros/Aiolos protein degrader (molecular glue) that shows compelling data in NHLs and **complete regressions in DLBCL animal models**[1]




SP-3164




Adapted from Varma, G. et al. Heme Onc. 2023

Abbreviations: R/R - relapsed/refractory, DLBCL - Diffuse Large B-cell Lymphoma, SOC – Standard of Care

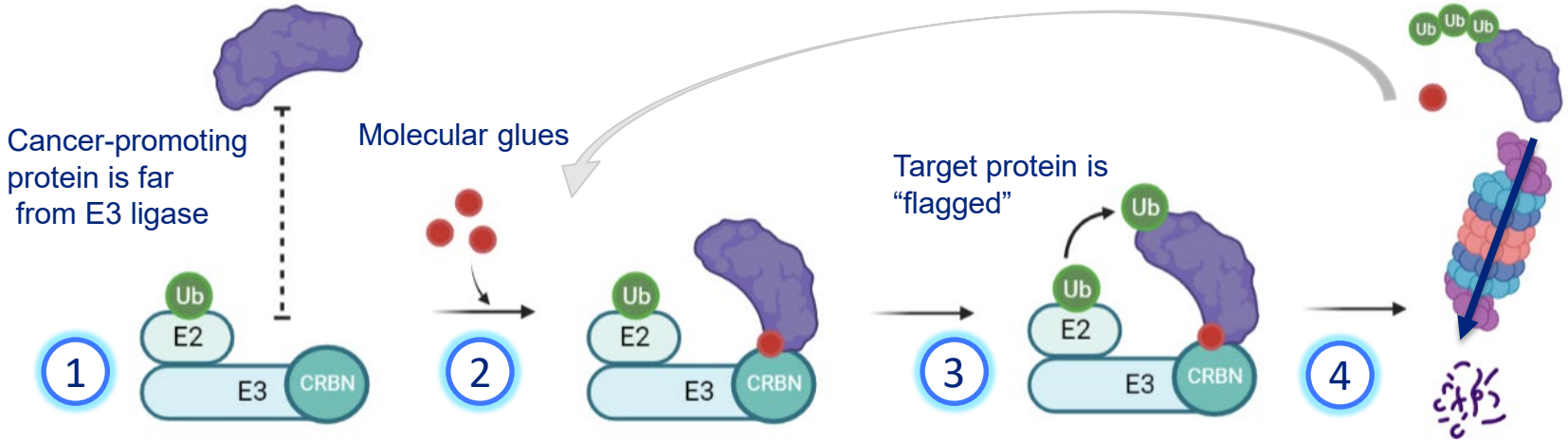


[1] SP-3164 in combination w/ rituximab (SOC) resulted in complete regressions

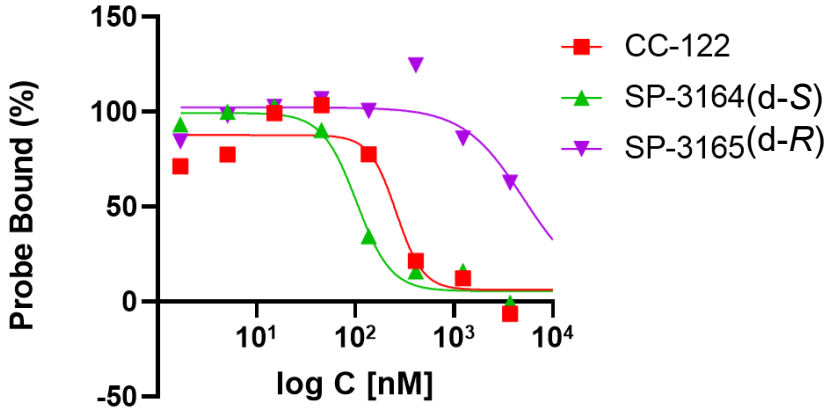
# SP-3164 Preclinical Data

# SP-3164 Demonstrates Improved Protein Degradation

## *Characteristics Compared to Avadomide (CC-122)*




**Cereblon Binding**

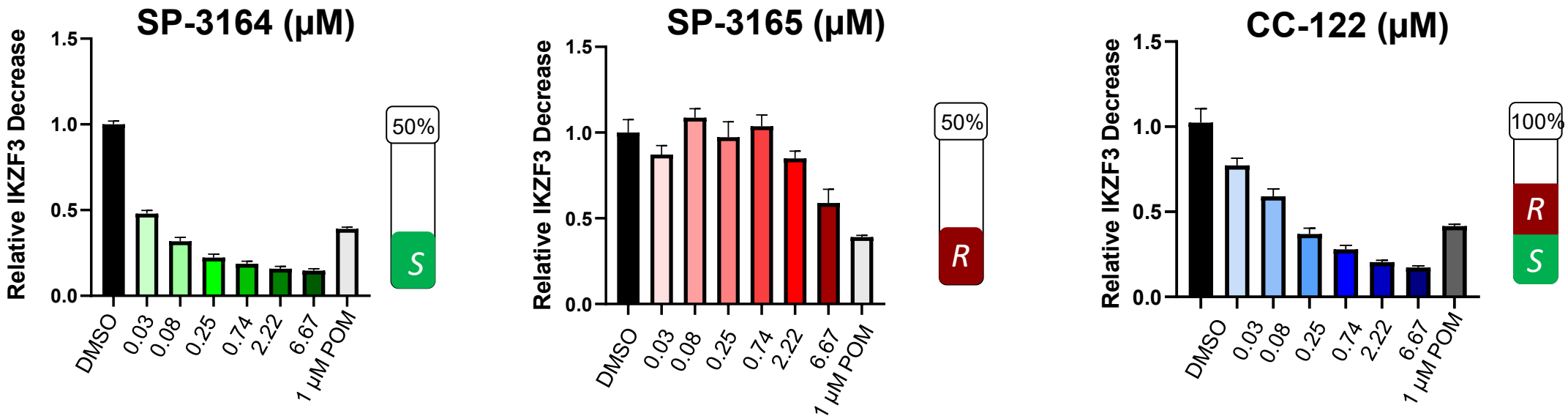



| Compound | Kd (nM) |
|---|---|
| CC-122 | 330 |
| SP-3164 (d-S) | 110 |
| SP-3165 (d-R) | 14000 |

**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)



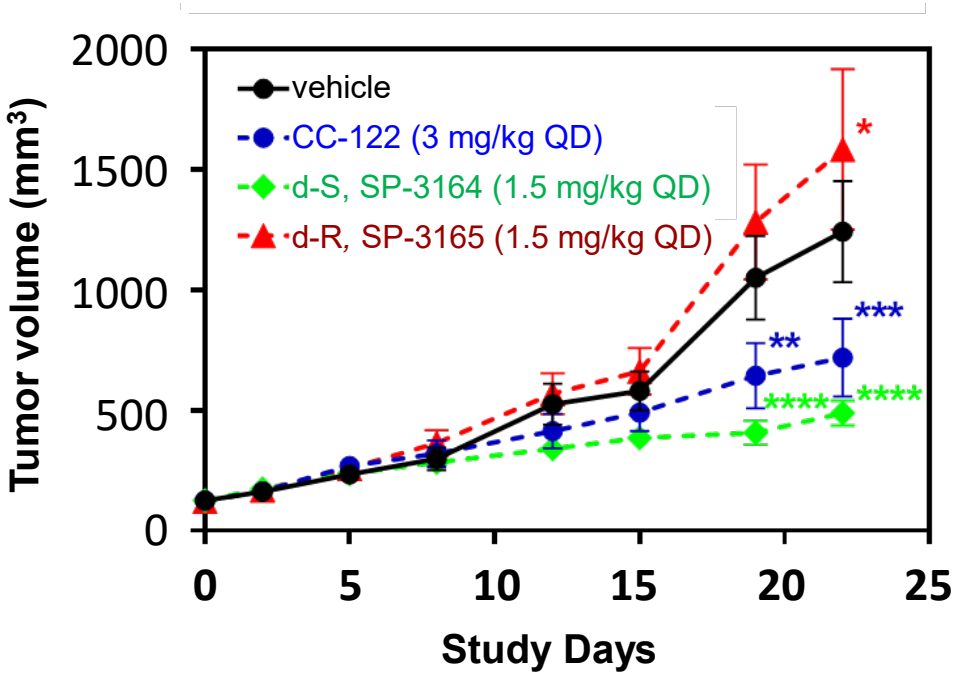

- Treatment with SP-3164 for 2 hrs results in deep and rapid degradation of the target protein, IKZF3.
- SP-3165 does not result in protein degradation except for at high concentrations.
- At comparable concentrations, SP-3164 induced more degradation of IKZF3 compared to CC-122

# SP-3164 – Multiple Myeloma (MM)

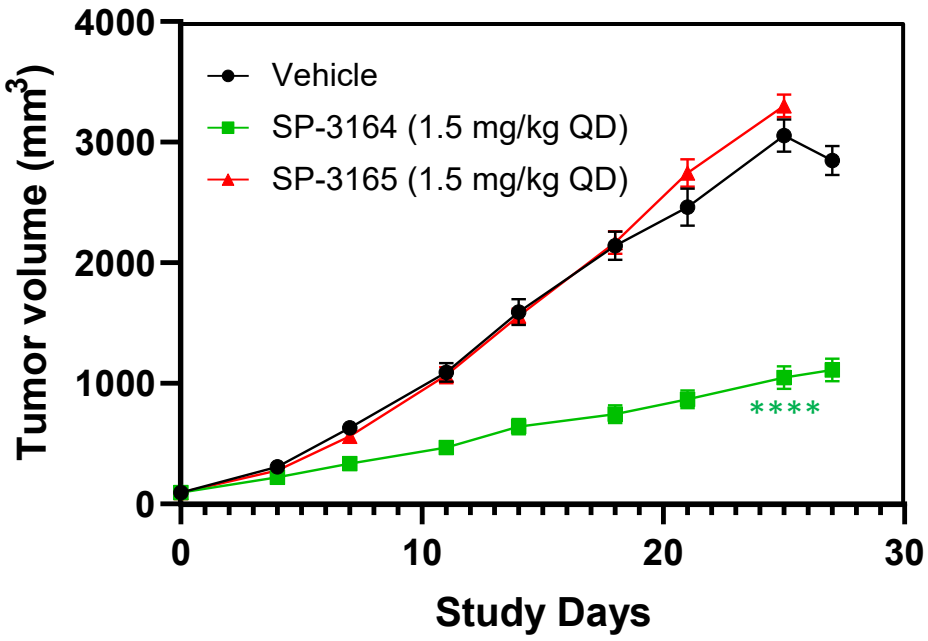
# SP-3164 Shows Significant Activity in MM H929 Xenograft Model

## *R-Enantiomer (SP-3165) is Inactive*

**NCI-H929 Xenograft Study**




**NCI-H929 Xenograft Study (repeat)**



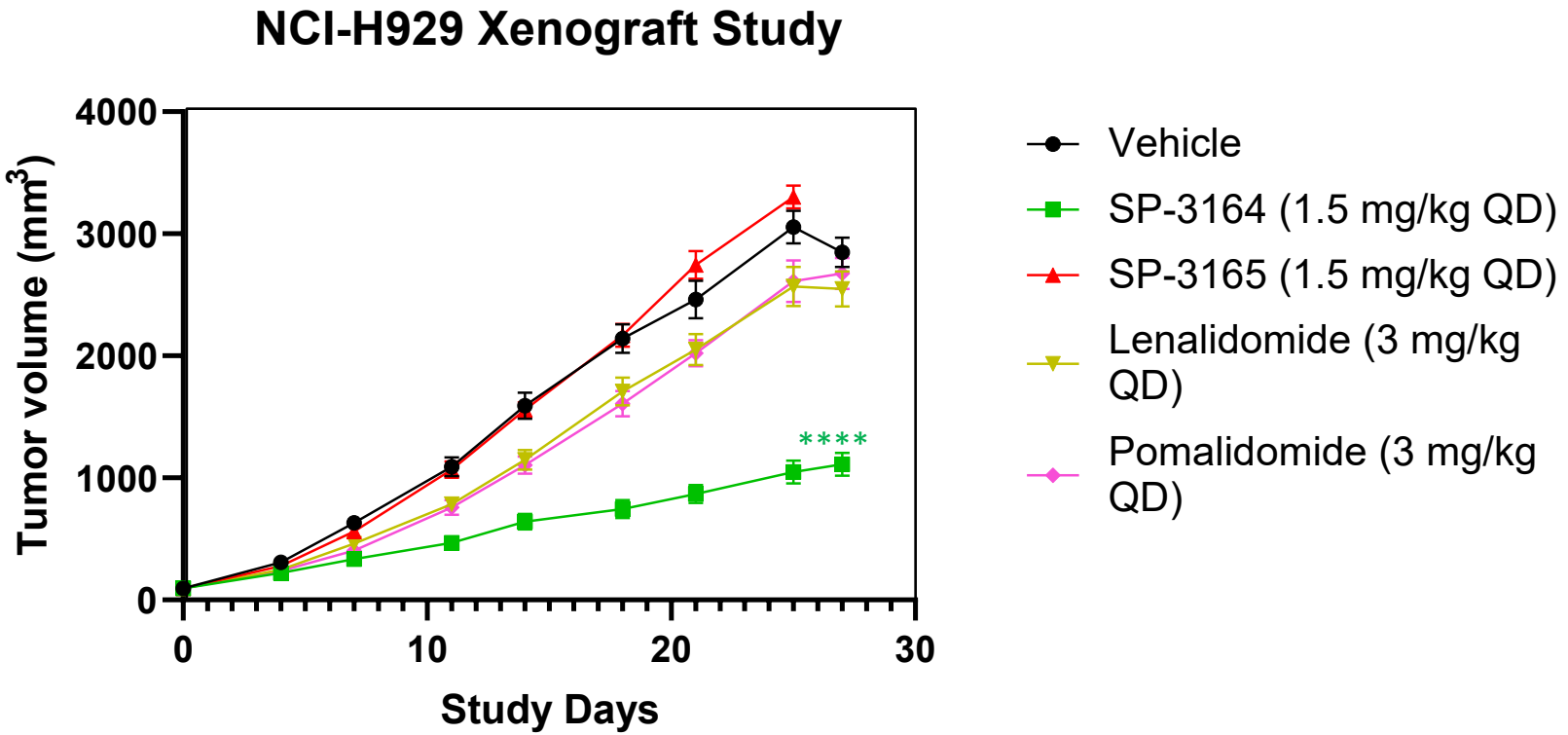

- SP-3164: Significant tumor growth inhibition (TGI) compared to vehicle
- SP-3164: Trended towards more TGI compared to CC-122
- SP-3165: No significant TGI, rather a trend towards supporting tumor growth

Abbreviations: Multiple Myeloma (MM)

Compared to vehicle, *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$; ****$p < 0.0001$

# SP-3164 Shows Significant TGI Compared to Other IMiDs®

*In MM H929 Xenograft Model*




- SP-3164 exhibits significant TGI compared to approved IMiDs for MM[1]
- Future studies will evaluate SP-3164 in IMiD-refractory MM cell lines

1. Revlimid® (lenalidomide) and Pomalyst® (pomalidomide)

Abbreviations: Tumor Growth Inhibition (TGI), Multiple Myeloma (MM)

Compared to vehicle, ****$p < 0.0001$

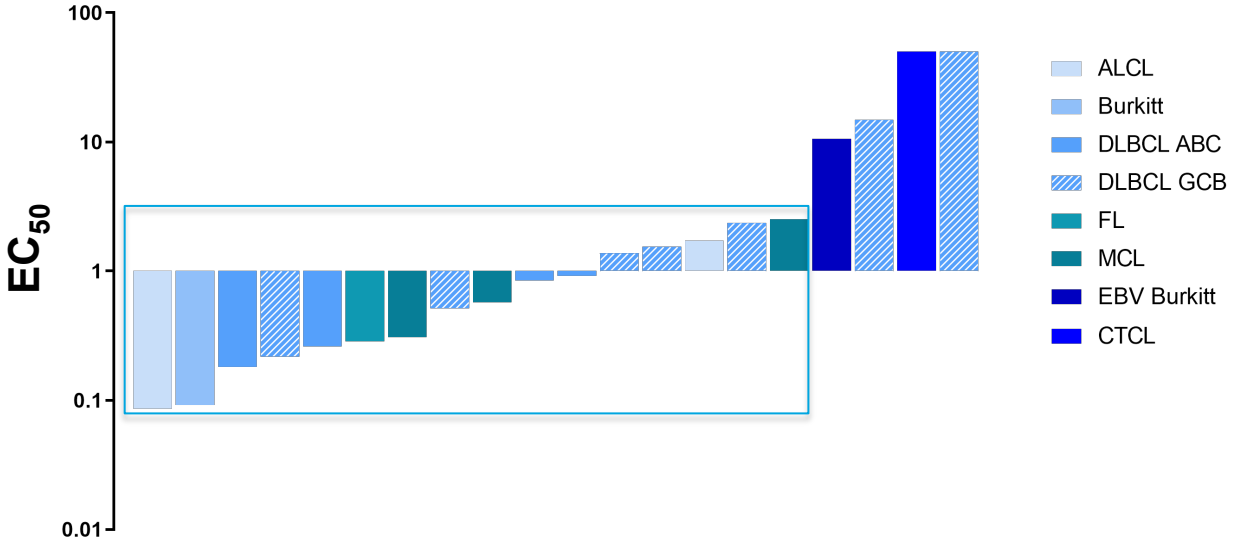
# SP-3164 – Non-Hodgkin’s Lymphoma (NHL)

# SP-3164 *in vitro* Activity in NHL

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

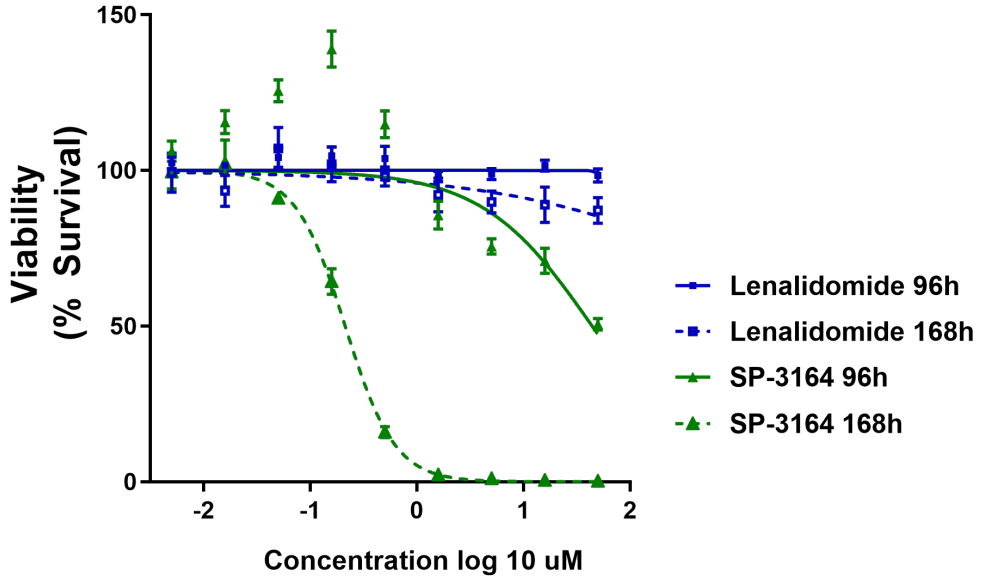
**Antiproliferative Activity of SP-3164 in Lymphoma Cell Lines**

$EC_{50}$ after 96 hr




**DLBCL (WSU-DLCL2) Cell Viability ($IC_{50}$)**

% Survival after 96 hr and 168 hrs



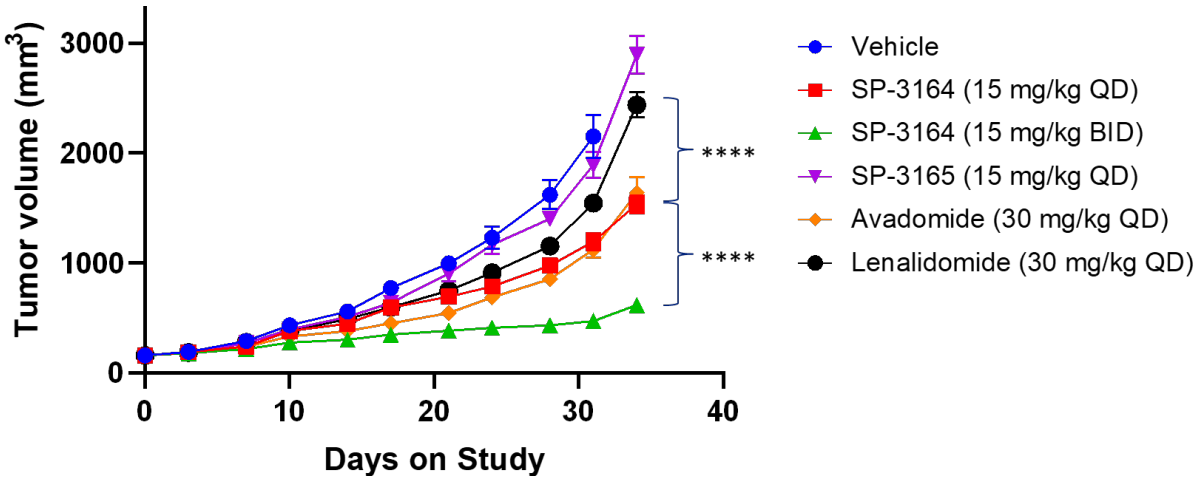

In a panel of 20 lymphoma cancer cell lines representing various subtypes, SP-3164 demonstrated potent antiproliferative activity within 96 hrs of dosing in 16 cell lines (average $EC_{50}$ <1 µM, range 0.092-2.523 µM).

In WSU-DLCL2 cells, increased duration of treatment (168 hr vs 96 hr) revealed increased sensitivity to SP-3164 ($IC_{50}$ 0.217 µM), but not lenalidomide.

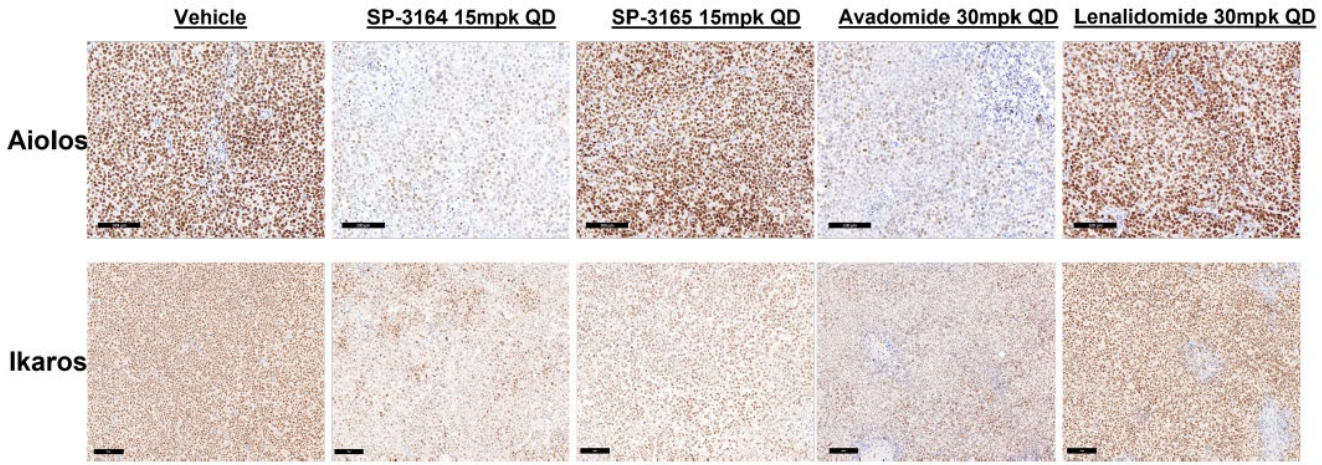
# SP-3164 Demonstrates Single-Agent Activity in DLBCL

## Mouse DLBCL (WSU-DLCL2) Xenograft Model

Tumor Volume ±SEM

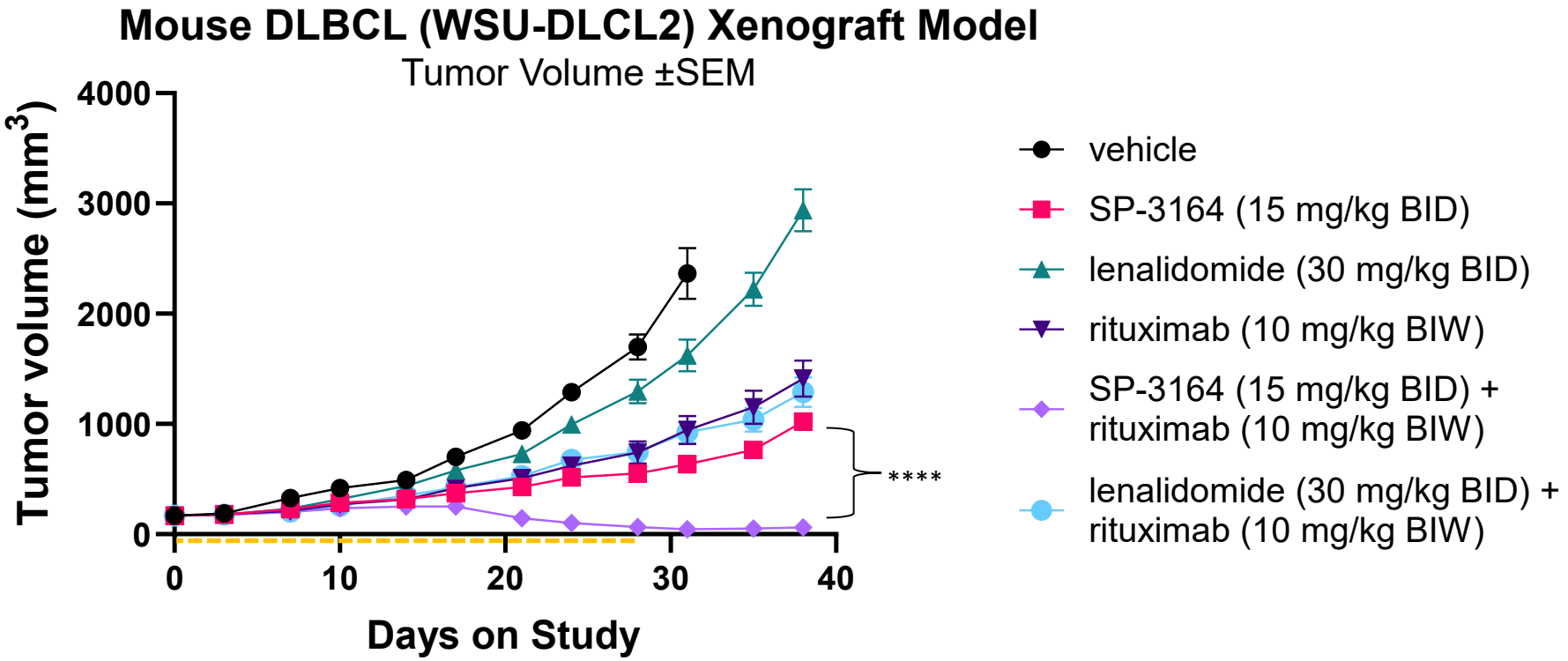



## Degradation of Aiolos and Ikaros in tumors



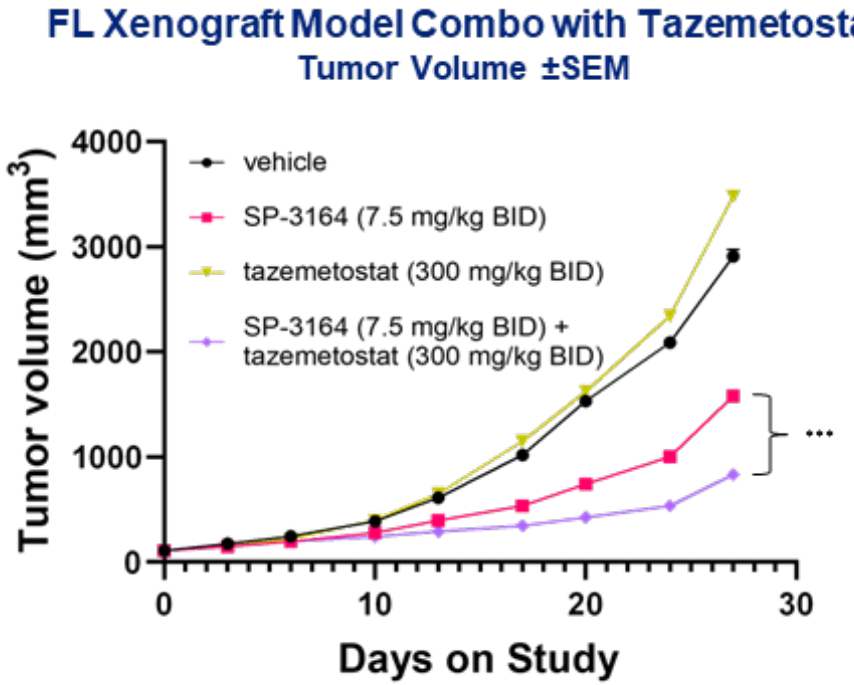

- SP-3164 demonstrated pronounced antitumor activity as single agent outperforming lenalidomide and comparable to avadomide while SP-3165 lacked significant antitumor activity (**** p≤ 0.0001).
- Due to SP-3164's shorter $t_{1/2}$ vs. avadomide, SP-3164 was studied BID resulting in the largest inhibitory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

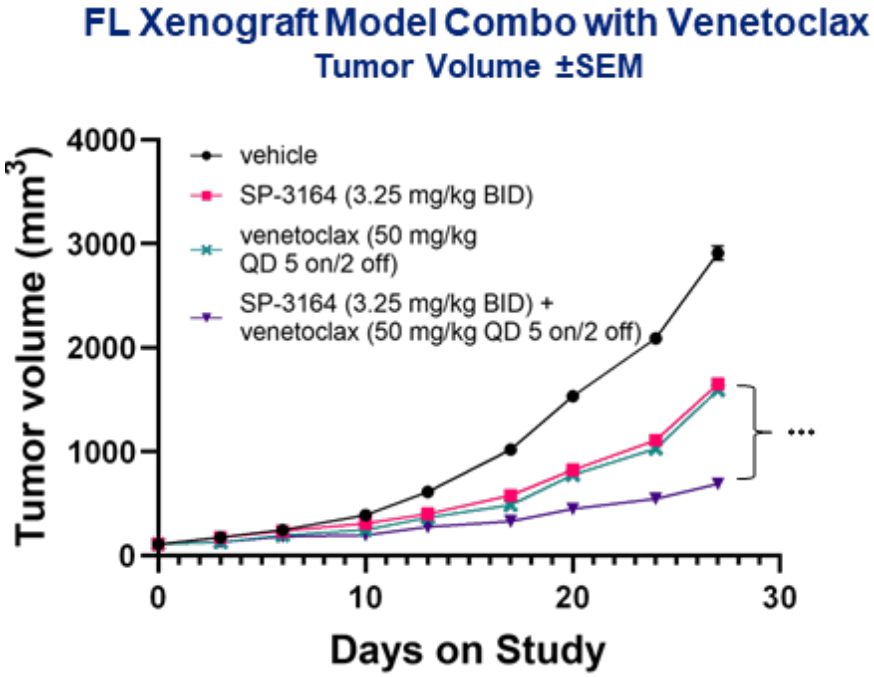
# SP-3164 Shows Synergistic Activity with Rituximab in DLBCL




- SP-3164's activity in combination with rituximab was compared to lenalidomide and rituximab in the WSU-DLCL2 DLBCL model (GCB subtype).
- Mice were treated for 28 days, and tumor volume was measured twice weekly.
- The combination of SP-3164 and rituximab resulted in regressions in 50% of treated mice, significantly better than approved regimen of lenalidomide and rituximab for NHLs.

# SP-3164 Shows Monotherapy and Combination Efficacy in FL Xenograft Model (DOHH-2)




SP-3164 performed better than the approved agent, tazemetostat (TAZ), which had no effect in the EZH2 wild-type model. SP-3164 appeared to sensitize to TAZ (***p≤0.001).

In combination with venetoclax, SP-3164 showed improved tumor growth inhibition (TGI) compared to either agent alone. (***p≤0.001)

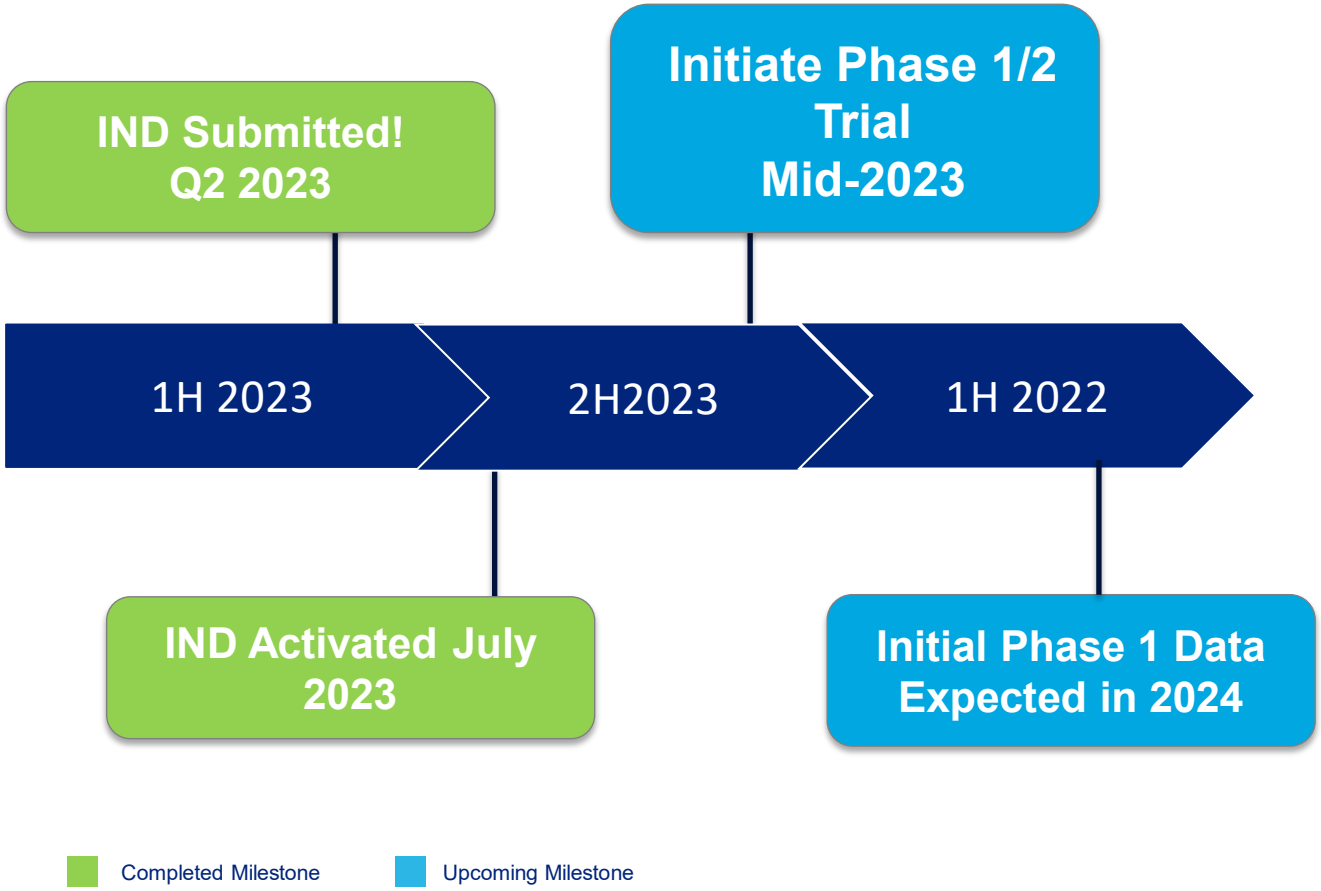
# SP-3164 Current Status and Upcoming Milestones

## *Rapidly advancing SP-3164 with upcoming IND submission*

### *Completed SP-3164 Studies Establish Preclinical Efficacy and Safety*

| | |
|---|---|
| **MoA/ Efficacy** | **In vitro**: Cereblon binding, *in vitro* immunomodulation on T cells, transcription factor proteomics, global proteomics, Western blots (I/A) in lymphoma cells, $EC_{50}$ in 93 cell lines<br><br>**In vivo**: Monotherapy and combination efficacy studies in DLBCL (2 studies) and follicular lymphoma (FL) (2 studies), efficacy in multiple myeloma models (3 studies) |
| **PK/TK** | Bioavailability (mouse and rat), single dose PK (mouse, rat, primate), pharmacodynamic study in primates, Dose Range Finding (DRF) primate TK, 28-day rat and primate TK |
| **ADME** | Bioavailability (mouse and rat), single dose PK (mouse, rat, primate), metabolism and met ID, CYP inhibition and induction, Caco-2 permeability, plasma protein binding, red blood cell partitioning |
| **Safety/ Toxicology** | Safety screen, Ames, hERG, Micronucleus test, DRF study (rate and primate), GLP 28-day studies (rat and primate) |

### *Completed and Upcoming Milestones*



# SP-3164 Addresses a High Unmet Need in DLBCL and Has a Clear Development Path To A Launch Indication

## Rationale for SP-3164 Potential Success

1. Improves upon successful 1st generation protein degraders LEN and POM
   - SP-3164 shows superior preclinical activity to LEN and POM, successful drugs with:
     - Validated activity in hematological cancers
     - Combined 2021 global sales of $16B

2. Clear clinical development plan driven by insights from its predecessor compound, AVA
   - SP-3164 is the preferred half of AVA, with improved potency and pharmacokinetic properties, preclinically. Shorter half-life may allow for dosing schedules that result in improved efficacy
   - AVA clinical data supports label expansion into 1st line DLBCL and into other NHLs and MM

3. Builds off AVA's Precision Medicine Approach[2]:
   - Ability to select for high responders differentiates SP-3164 from other degraders used in DLBCL
   - Potential to show superior ORR (44% in gene signature positive patients)[3] → if SP-3164 replicates AVA data, SP-3164 could be the most active monotherapy I/A degrader in select R/R DLBCL patients
   - Physician/Patient> Right drug for the right patient  Payors> First $ spent is the best $ spent

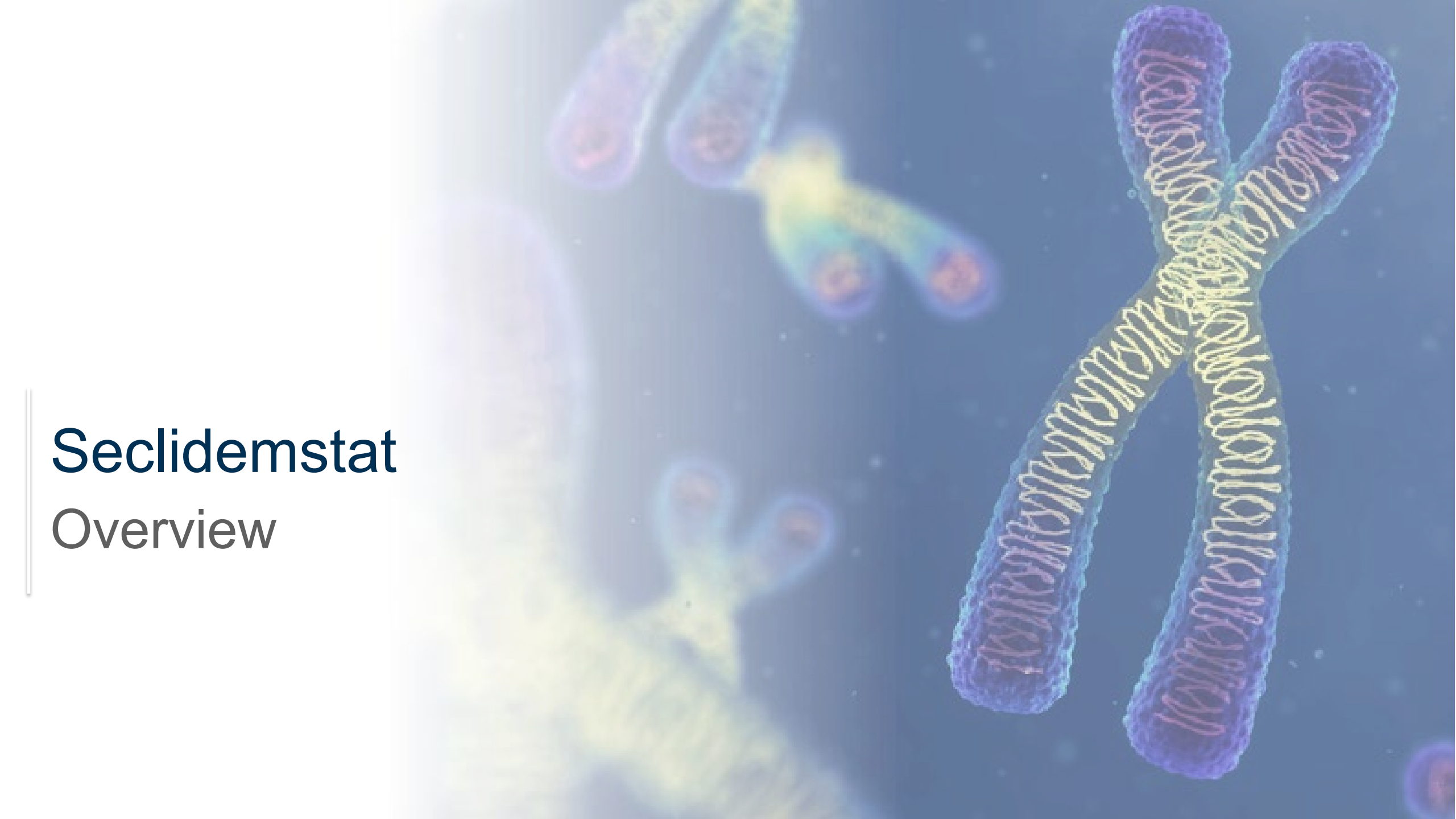
Abbreviations: R/R - relapsed/refractory, DLBCL - Diffuse Large B-cell Lymphoma, MM – Multiple Myeloma SOC – Standard of Care, LEN – lenalidomide, POM pomalidomide, AVA – avadomide, ORR – Overall Response Rate




[1] SP-3164 in combination w/ rituximab (SOC) resulted in complete regressions. 2. In clinic, AVA demonstrated that a subset of patients (~45%) have a high chance of responding. 3. 44% (gene classifier-positive) and 19% (gene classifier-negative)

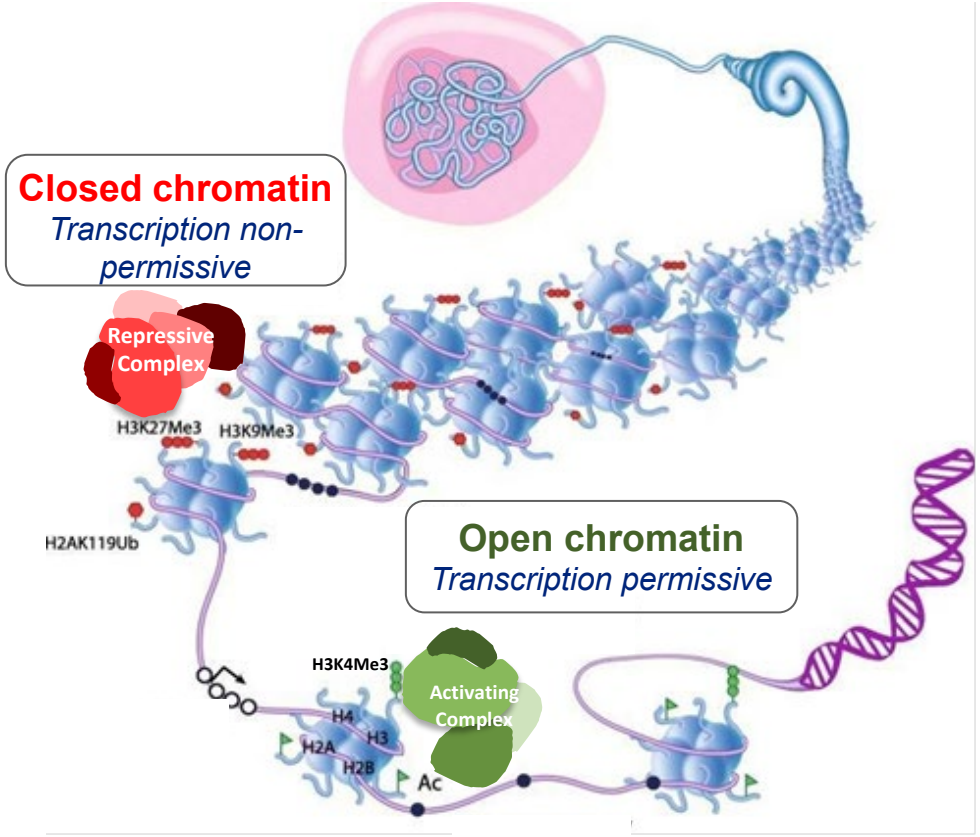
# Seclidemstat

## Overview



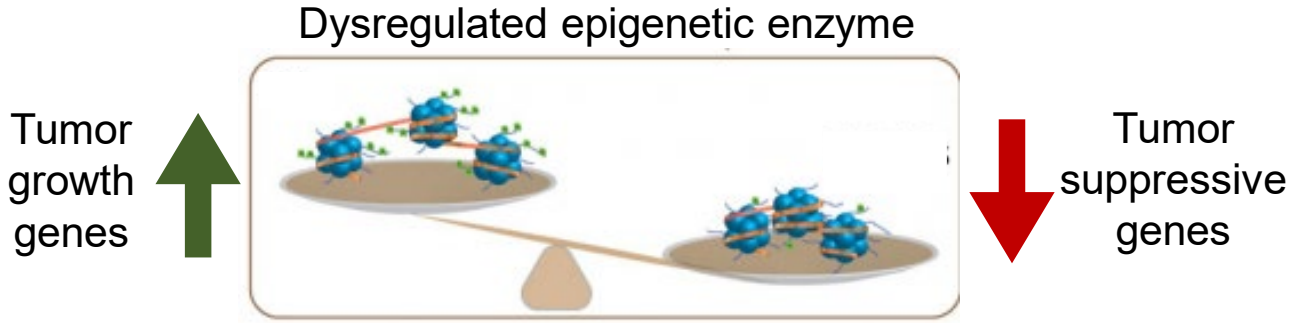
# Epigenetic Enzymes Are Attractive Targets For Cancer Therapy

Epigenetic modifying enzymes affect gene expression by manipulating the chromatin structure

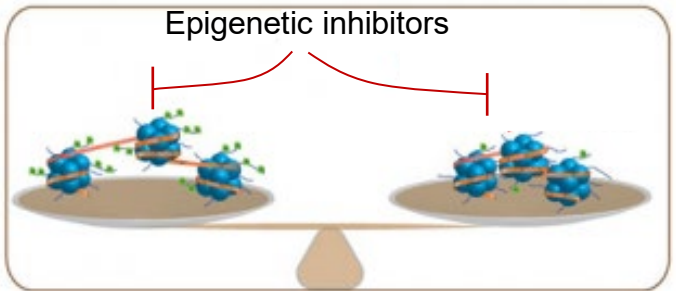



Adapted from Holliday, H. Breast Cancer Research *2018*

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 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 | • LSD1 is necessary for stem cell maintenance and cell development processes (e.g., blood cells) |
| Cancer Cells | • LSD1 is over expressed<br>• LSD1 acts incorrectly to silence or activate genes leading to disease progression<br>• 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:

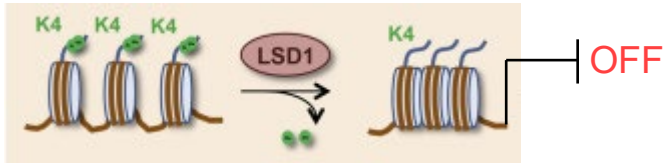
Salarius Pharmaceuticals, Oryzon, Celgene, Bristol Myers Squibb, Imago, Merck, Jubilant Therapeutics

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

# More Comprehensive Inhibition of LSD1 Positively Impacts Therapeutic Activity

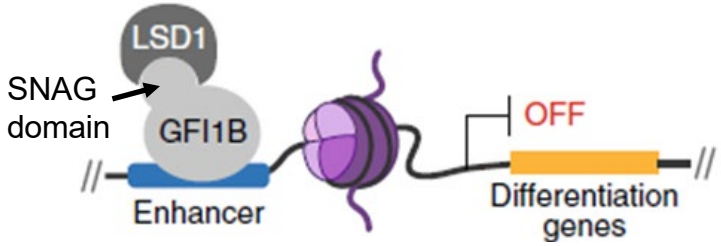
Degree of LSD1 inhibition

**Enzymatic activity – Demethylation**
Impact: Moderately alter gene expression




Salarius PHARMACEUTICALS and competitors

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




Salarius PHARMACEUTICALS and competitors

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

Salarius PHARMACEUTICALS

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

*scaffolding properties – protein to protein interactions

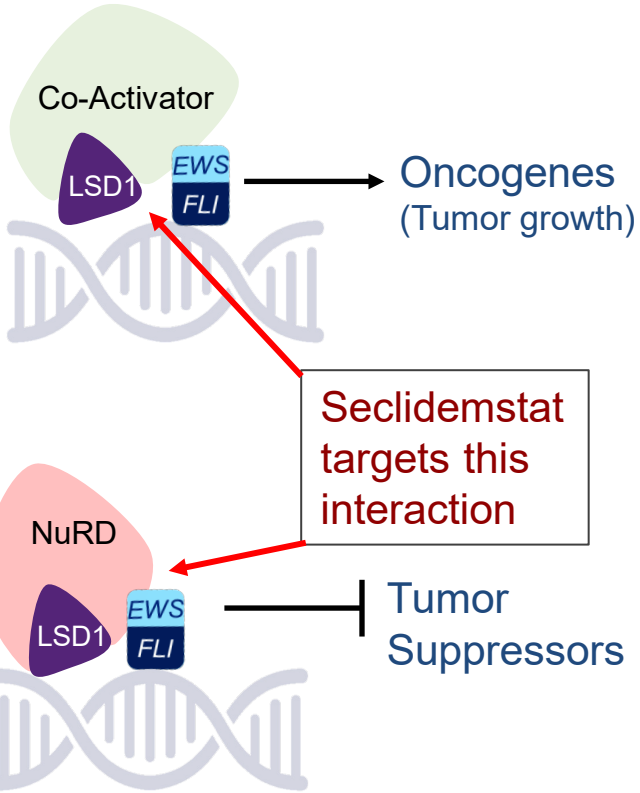
# SPEED TO MARKET

## Seclidemstat in Ewing Sarcoma

# Targeting The Root Cause Of Ewing Sarcoma Via LSD1 Inhibition

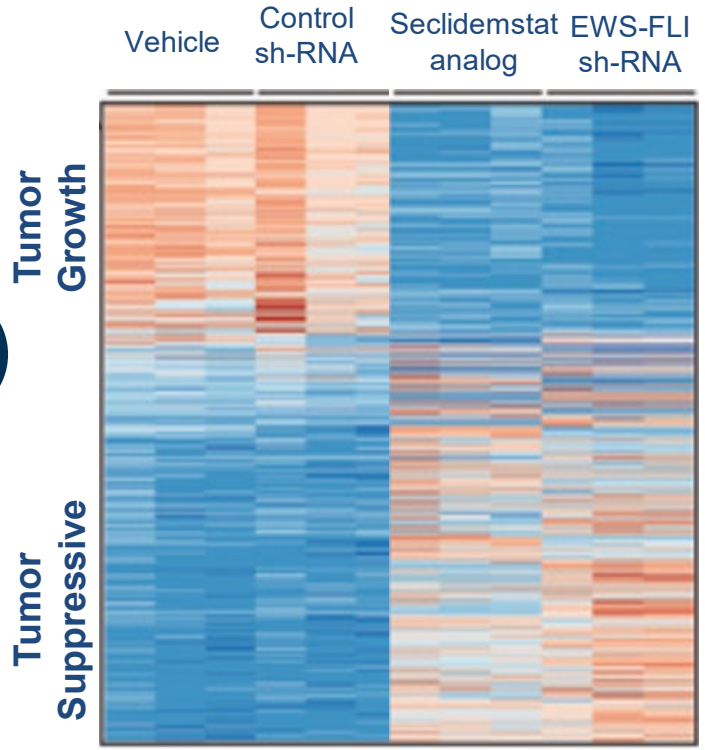
Ewing sarcoma is driven by an easily diagnosed chromosomal translocation, i.e., EWS-FLI

## Incorrect transcription factor leads to gene dysregulation




## Seclidemstat corrects gene expression

Ewing's cells (A673)



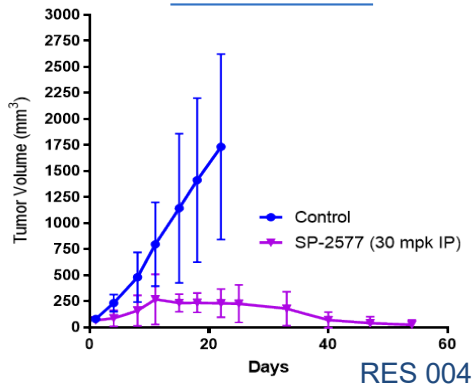

**Red** = turned ON

**Blue** = turned OFF

**Genes correctly turned on/off**

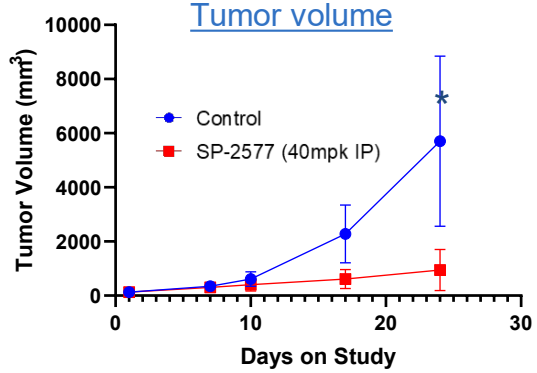
## Potent anti-tumor activity in SKNMC (Ewing sarcoma cells) *in vivo* studies

Tumor volume




RES 004 Salarius

Tumor volume




*p<0.0003

RES 006 Salarius



Sankar et al. *Clinical cancer research* 20.17 (2014)

# Ewing's Sarcoma: Unmet Need, Meaningful Opportunity

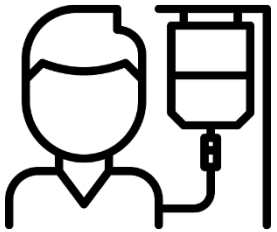
## Diagnosis

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

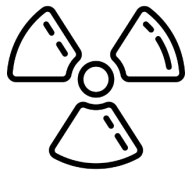
- 75% localized[1]
- 25% with metastasis[1]

## Standard-of-Care

Surgery and Amputations



Chemo



Radiation

- ~40% of patients are refractory or relapse[2]
- 70-90% 5-year mortality rate[2]
- No standardized second-line treatment

## Salarius' Vision

An effective, non-toxic, oral treatment

- Accelerated U.S. approval
- Rapid market uptake
- $200M+ global sales[3] (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 |
|---|---|---|---|---|---|---|---|---|
| 1st Relapse Pts (5) | 1 | 1 | 2 (40%) | 1 | 3 (60%) | 2 | 7.4 | 1.4 to 13.8 |
| 2nd Relapse Pts (8) | | 1 | 1 (13%) | 1 | 2 (25%) | 6 | 1.5 | 0.7 to 5.1 |
| 1st and 2nd Relapse Pts (13) | 1 | 2 | 3 (23%) | 2 | 5 (38%) | 8 | 1.6 | 0.7 to 13.8 |
| 1st and 2nd Relapse Pts w/ DCRc (5) | | | | | 5 (38%) | | 7.4 | 3.1 to 13.8 No Observed PD[2] |
| rEECur (primarily a 1st relapse Ewing sarcoma data set[3]) Salarius (1st Relapse Patients) | | | | | | | 3.5 mPFS 7.4 mTTP | 95% CI 2.5 to 5.1 |

[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.




Abbreviations CR complete response; PR partial response; SD stable disease; DCR disease controlRate (CR, PR, SD); TTT Time to Tumor Progression; PFS Progression Free Survival

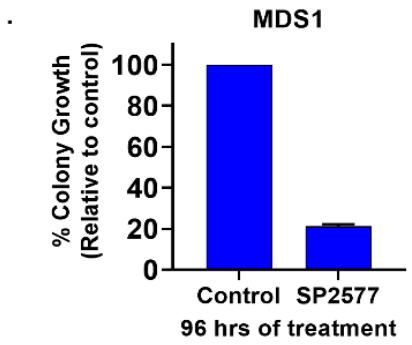
# MARKET EXPANSION

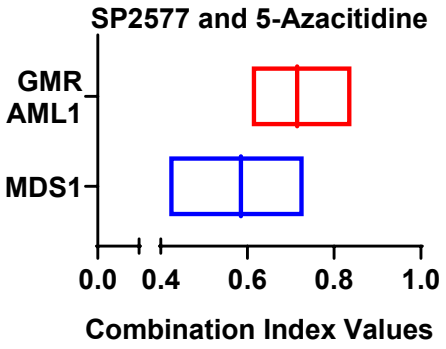
## Hematologic Cancers

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

## Hematologic Cancers[1]

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

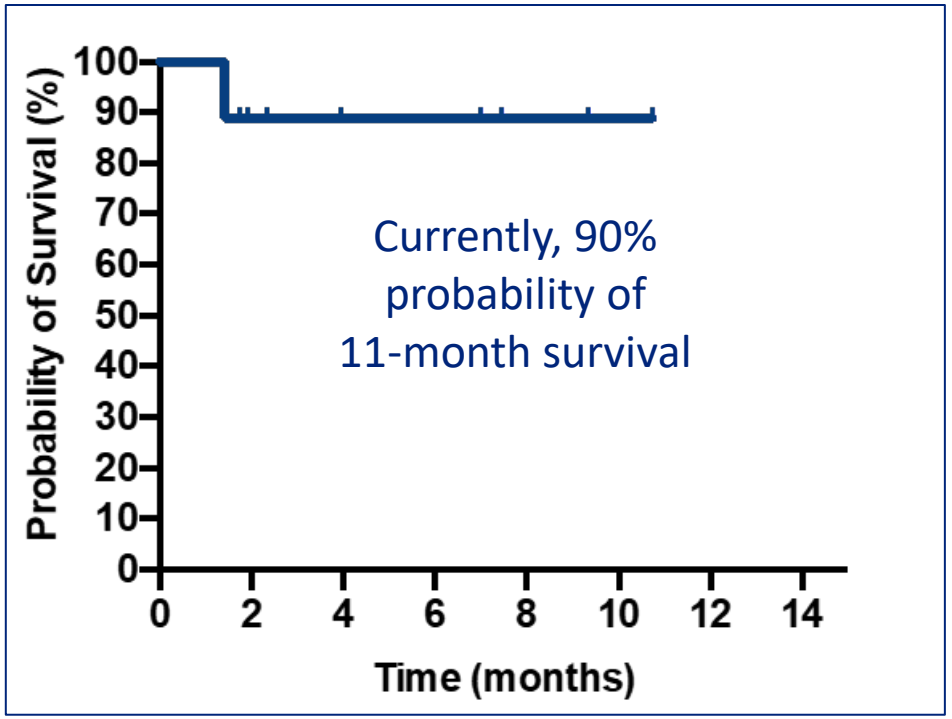
- Overall survival, duration of response, relapse-free survival, leukemia-free survival and safety
- Correlative studies including correlation of response with disease subtypes, genomic profile and *in vitro* studies

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 CMML[1] with prior HMA[2] 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 (azacytidine, decitabine)

A Phase I/II Study of Seclidemstat, an LSD1 Inhibitor, and Azacitidine for Patients with Myelodysplastic Syndromes and Chronic Myelomonocytic Leukemia, American Society of Hematology Annual Meeting December 11-14, 2022

# Financial Overview and Management

# Financial Overview

**Sufficient Cash Position**

- Cash position of $9.3M as of March 31, 2023
- $6M financing completed in May 2023
- Sufficient cash expected to fund operations through near-term milestones through Q1 2024

**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: 3.4M

# 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**

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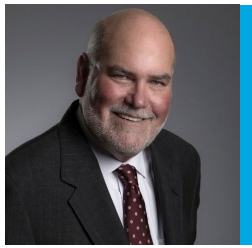

**Mark Rosenblum**
**Chief Financial Officer**

ADVAXIS · WELLMAN, INC. · Deloitte.

**Aundrietta Duncan, PhD**
**Director Non-clinical Development**

FANNIN — INNOVATION STUDIO — · THE UNIVERSITY OF TEXAS MD Anderson Cancer Center

**Daniela Y. Santiesteban, PhD**
**Director Protein Degradation Development**

GRFP · Georgia Tech

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| | Histogenics | | | | Novartis | |

# Thank You