Best In Class
Significant Upside
Strong Non-Dilutive Funding

- Focused on “Epigenetics” – The way cancer cells regulate gene expression
- Lead drug, Seclidemstat, focuses in areas of high unmet need
  - Lysine Specific Demethylase 1 (LSD1) – Validated target in many solid tumors
  - Ewing Sarcoma – Aggressive childhood bone cancer with no treatment
  - Related Sarcomas– Share Ewing sarcoma cell biology
  - Late Stage Prostate/Breast/Ovarian cancer are upside
- Seclidemstat specifically affects tumor cells, not healthy cells
- Texas $19M award plus ~$3M National Pediatric Cancer Foundation and ~$6M University of Utah support
Ewing Sarcoma is a Devastating Disease and Shares Biology with Other Sarcomas

• Ewing Sarcoma – Rare, Orphan Disease
  – 500 children, adolescents and adults in the U.S. per year
  – Median age of 15 years
  – Current treatment causes debilitating long-term side effects
  – 50% of patients fail to respond or relapse
  – 10% 5-year survival rate for those who fail to respond/relapse

• Up to 2,500 US annual sarcoma patients share the Ewing biology

High Clinical Unmet Need

70% localized
30% metastatic

Chemotherapy, Radiation, Disfiguring Surgeries

50% fail to respond or relapse
Our Technology Reverses Cancer Back to Normal State

Each shade represents different proteins turning on or off in the cancer cell
Red = Protein On
Blue = Protein OFF

Reverses the way proteins are expressed in cancer - this makes cancer cells die
Ewing sarcoma In Vivo Study Summary

SP2577 has been tested in five different sarcoma models and exhibited an average tumor growth inhibition of 76%.

<table>
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<tr>
<th>Study #</th>
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</table>

Note 1: Patient derived xenograft, dose was ½ dose instructed by Salarius
Seclidemstat Cures Ewing Sarcoma in Animals

Mice Afflicted with Ewing’s Model #1

- Survival %
- Days
- Seclidemstat
- Vehicle

8 out of 10 mice cured
Cured mice relapse free

Mice Afflicted with Ewing’s Study #6

- Survival %
- Days
- Seclidemstat
- Vehicle

5 out of 10 mice cured
Cured mice relapse free

P value
P value summary
Are the survival curves sig different?
< 0.0001
****
Accomplishments Since CPRIT Award

- Completion of IND enabling studies
  - Successful toxicology outcomes
- Re-formulation to enteric coated tablet
- NCI Pre-clinical Testing Consortium
- National Pediatric Cancer Foundation funding
- Huntsman Cancer Institute breast and ovarian trial funding
- FDA Orphan drug status
- Pediatric Priority Review Voucher designation
- Accepted into Jlabs@TMC
- Ewing Clinical readiness
- Compassionate use trial
- CPRIT relationship
- Pre-clinical data in Ewing sarcoma
- 11 Texas based employees
Current Series A Raise Supports Clinical Trial Program With Support From The National Pediatric Cancer Foundation And University Of Utah, Huntsman Cancer Institute

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The Next 2 Years Are An Exciting Time For Salarius
Salarius In Vivo Study Overview

• This presentation provides a broad overview of animal studies completed by Salarius.

• Salarius’ clinical candidate is SP2577 or Seclidemstat. The molecular structure of SP2577 is confidential and has not been published. This confidentiality prevents the public availability of Seclidemstat.
  • Some experiments in this presentation will use SP2509/HCl2509. This is the Salarius “tool” compound, it exhibits a similar efficacy profile and works through the same mechanism as SP2577. Salarius leverages SP2509 to publish in peer-reviewed journals keeping SP2577 protected.

• Raw data for experiments presented is available upon request.
### Ewing sarcoma In Vivo Study Summary

SP2577 has been tested in five different sarcoma models and exhibited an average tumor growth inhibition of 76%.

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Note 1: Patient derived xenograft, dose was ½ dose instructed by Salarius
In Vivo Tumor Growth Inhibition – Sarcoma

Nov 2017
Study #1: SP2577 30mg/kg IP in SK-N-MC mouse xenograft model of Ewing sarcoma

SK-N-MC represents classic Ewing sarcoma. In this study the control mice died within three weeks. 80% of the treated group survived until study end with no palpable tumors.
Study #2: SP2577 administered via IP injection or orally at various concentrations in SK-N-MC mouse xenograft of Ewing sarcoma

In this study, 2577 displayed a dose-dependent increase in efficacy, and oral administration proved nearly as efficacious as direct injection into the peritoneal cavity (IP).
Study #3: SP2509 and SP2577 in SK-NM-C Ewing Sarcoma Xenograft

In this experiment SP2577 exhibits improved efficacy compared to tool compound SP2509.

We also observed a dose-dependent increase in tumor growth inhibition with 2509.
Study #4: SP2509 30mg/kg IP in SK-N-MC mouse xenograft models of Ewing sarcoma

SP2509 exhibited similar efficacy to clinical candidate SP2577. The only difference between the two compounds is SP2577 has improved solubility and other properties that enable formulation into an orally-administered tablet for human use.

SP2509 Compounds Inhibit Lung Metastases in Ewing Sarcoma (SK-N-MC) Mouse Xenografts

Ewing’s metastases to the lung. With SP-2509, we see dramatic reduction of metastases in animals with higher blood levels.
The A673 cell line represents a more heterogeneous tumor that is more difficult to treat than classic Ewing. A673 was originally a rhabdomyosarcoma that later mutated the EWS/FLI translocation.
Study #6: SP2509 30mg/kg IP in A673 mouse xenograft models of Ewing sarcoma/rhabdomyosarcoma

SP2509 exhibited similar efficacy to clinical candidate SP2577. In the A673 model, often there is a bifurcated response with some tumors highly responding and other tumors not. Rhabdomyosarcomas represent another rare adolescent disease with a high unmet need.

Study #7: SP2509 Oral administration vs IP Administration in A673 cell line

Treatment started as soon as tumors were detectable by luciferase signal (two weeks before average tumor volume was 100mm$^3$)

The A673 cell line represents a more heterogenous tumor that is more difficult to treat than classic Ewing. A673 was originally a rhabdomyosarcoma that later mutated the EWS/FLI translocation.
Study #8: SP2509 in SK-ES-1 Ewing Sarcoma Brain Metastasis

The SK-ES-1 model represents metastatic Ewing sarcoma. Although Ewing first metastasizes to the lung, the next site of metastasis is often the brain. This model represents a more aggressive model than SK-NM-C.
Study #9: SP2577 Causes Complete Tumor Response in Clear Cell Sarcoma – Driven by EWS/ATF Fusion

In cancers with EWS translocations, such as EWS/FLI translocation in Ewing, or EWS/ATF in clear cell sarcoma, SP-2577 is very potent. This can be attributed to EWS dominating the genetic profile of cancer cells. Because EWS requires LSD1, it is possible to completely abrogate EWS effects with the right LSD1 inhibitor.

There are 500 annual cases of Ewing sarcoma in the United States, but including related sarcomas with EWS there are over 3,000 annual cases in the United States alone.
Ewing Sarcoma
PDX model
Aim: To assess the efficacy of SP-2577 (Seclidemstat) in two Ewing sarcoma PDX models.

PDX models

- Passage 4 PDX tumors from two donor mice were harvested, dissected into small tumor pieces and subcutaneously implanted (right flank) into 20 mice for each model.
- **EW5 PDX:** 16 year old Caucasian male with paraspinal Ewing sarcoma (post chemo sample)
- **EW1 PDX:** Wild-type TP53, with EWS/FLI (Type 1). Patient clinical characteristics pending.
- Experimental tumor endpoint was 2000mm³, or if >10% loss in body weight was observed.

Mouse source and strain

- Female, 6-8 week old CB17/SC-/-, purchased from Envigo (Harlan)

Dosing schedule and Seclidemstat formulation

- Seclidemstat was dissolved in 10% HP-β-CD/5%DMSO, as per Salarius instructions
- Mice were dosed with either vehicle or Seclidemstat [40mg/kg] twice daily via oral gavage (100µls per dose) for up to 55 days or until a tumor volume of 2000mm³ was reached.

*starting tumor volume = 200mm³
Study #10 Ewing PDX Model 1: 16yr male refractory to chemotherapy

- Post-doc running study initially wanted to test for resistance, so she started with a significantly lower dose of 40mg/kg BID and did not follow our instructions for dosing, which for this oral formulation should have been 100mg/kg BID.

- Significant delay in tumor growth was observed for Seclidemstat treated mice compared to vehicle control.

- Significant increase in overall survival was observed with Seclidemstat treatment (median survival 17 versus 24 days, P = 0.0056).

- Oral administration of Seclidemstat was well tolerated with no statistical significance in organ (kidney, spleen, liver) or endpoint body weight observed.

- Blood chemistry revealed no changes to hepatic markers.
Study #10 Ewing PDX Model: Tumor Growth & Mouse Survival

~35% Average Tumor Growth Inhibition

29-Day Study; 41% Extension in Median Survival
Study #10 Ewing PDX Model: Organ weight post-study

No changes in organ weight that might indicate overt toxicity
### Study #10 Ewing PDX Model: Blood Chemistry Results

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<tr>
<th>Component</th>
<th>Unit</th>
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<td>Average ± SEM</td>
<td>Range</td>
<td>Average ± SEM</td>
<td>Range</td>
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<td>Albumin</td>
<td>g/dL</td>
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<td>Alkaline Phosphatase (ALP)</td>
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<td>52.0 - 84.0</td>
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<td>Alanine Aminotransferase (ALT)</td>
<td>U/L</td>
<td>129.3 ± 48.9</td>
<td>12.0 - 345</td>
<td>22.3 ± 6.5</td>
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<td>Amylase</td>
<td>U/L</td>
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<td>699.2 - 840.3</td>
<td>726.5 ± 22.4</td>
<td>643.7 - 820.6</td>
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<td>Aspartate Aminotransferase (AST)</td>
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<td>Blood Urea Nitrogen (BUN)</td>
<td>mg/dL</td>
<td>19.1 ± 1.29</td>
<td>14.0 - 25.0</td>
<td>15.9 ± 0.97</td>
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<td>Blood Urea Nitrogen: Creatinine Ratio</td>
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<td>Calcium</td>
<td>mg/dL</td>
<td>9.84 ± 0.078</td>
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<td>9.90 ± 0.046</td>
<td>9.70 - 10.10</td>
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<td>Chloride</td>
<td>mmol/L</td>
<td>122.3 ± 0.80</td>
<td>118.6 - 124.4</td>
<td>121.1 ± 0.57</td>
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<td>Cholesterol</td>
<td>mg/dL</td>
<td>109.8 ± 5.87</td>
<td>70.6 - 122.6</td>
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<td>Creatinine</td>
<td>mg/dL</td>
<td>0.324 ± 0.030</td>
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<td>Creatinine Kinase</td>
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<td>862.4 ± 367.5</td>
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<td>Globulin</td>
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<td>Potassium</td>
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<td>Triglyceride</td>
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<td>92.0 - 204.0</td>
<td>142.8 ± 14.6</td>
<td>91.0 - 206.0</td>
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In Vivo Tumor Growth Inhibition – Prostate Cancer

Nov 2017
SP2509 Significantly Inhibits Tumor Growth in PC3 Prostate Cancer Xenograft Model

Salarius is initially targeting castration-resistant prostate cancer (CRPC). This encompasses patients whose tumors are unresponsive to standard hormonal therapies like enzalutamide and abiraterone.

The PC3 cell line represents one type of aggressive CRPC that no longer expresses the Androgen Receptor (AR). Hormonal therapies target AR, so prostate cancer frequently develops methods to work around AR dependence.

In this model, Salarius compounds are effective where there are no other treatment options available.
SP2577 Significantly Inhibits Tumor Growth in Aggressive AR Splice Variant 7 Xenograft Model

Docetaxel is a toxic chemotherapy administered by infusion over several hours; SP2577 is administered as an oral tablet and targets cancer cells overexpressing LSD1.

Prostate cancers expressing the AR Splice Variant 7 (V7) are of particular interest to big pharma. Housed in Johnson & Johnson’s incubator, Salarius has access to J&J corporate and venture arms, who have advised us to test in models expressing V7. These tumors are particularly aggressive because they rely on a mutated AR resistant to hormone therapies.

In this model SP2577 is as effective as chemotherapy, representing a significant market opportunity with regard to safety and toxicity.
In Vivo Tumor Growth Inhibition – Breast Cancer

Nov 2017
Seclidemstat in Mouse Xenograft Model of Triple Negative Breast Cancer

MDA-MB-231 Breast CDX

This cell line represents triple negative breast cancer refractory to many standards of care.

This study was conducted by an independent third party out of the University of Pittsburgh and used for a Department of Defense grant.
Additional Preclinical Studies – Endometrial Cancer

Nov 2017
SP2509 is effective in chemo-refractory endometrial cancer


![Figure 5 HCl2509 treatment causes tumor regression in vivo. (A) Average total body weight (g) of mice in both groups, vehicle and HCl2509 treatment, starting at implantation (day 0) through the course of the study. Data points shown represent the mean and standard deviation. (B) Quantified bioluminescence measurements of both the vehicle and HCl2509 treatment groups. Data is plotted as the geometric mean of total flux (photons/second). Daily treatment was initiated on day 7 (day 0 = implantation), such that day 14 represents the first day of imaging after the start of treatment. (C) Individual mouse images from study day 42 (day 35 of treatment). All images are on the same luminescence scale from...](image-url)
Additional Preclinical Studies – Leukemia

Nov 2017
These AML models (cell-line on the left and primary patient sample on the right) represent leukemias resistant to JAK inhibitors. This is a subset of AML with a high unmet need. Here, SP2509 performs better than currently marketed HDAC inhibitor (Incyte Corp), and enables survival in combination.

Effects of SP2509 in Pediatric Leukemia (T-ALL)

A. T-ALL cell viability—IC50s

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<tbody>
<tr>
<td>CEM</td>
<td>426 nM</td>
<td>474 nM</td>
</tr>
<tr>
<td>Jurkat</td>
<td>480 nM</td>
<td>620 nM</td>
</tr>
<tr>
<td>Hs-B2</td>
<td>420 nM</td>
<td>599 nM</td>
</tr>
<tr>
<td>Molt-4</td>
<td>780 nM</td>
<td>930 nM</td>
</tr>
<tr>
<td>SUP-T1</td>
<td>423 nM</td>
<td>676 nM</td>
</tr>
</tbody>
</table>

B. Rx (day) → Day +00 → Day +03 → Day +07
Post SCT  Day +7 → Day +13 → Day +16 → Day +20

Vehicle

SP-2509

*Expired (failed anesthesia recovery)

C. Naive DMSO DOXO 2577 2509 2513

*PARP

![Image of Western blotting results](attachment:image.png)
Summary

• Salarius has validated the Ewing sarcoma space, with promising results in related sarcomas sharing similar biology

• Salarius is exploring other cancers to increase value and provide risk mitigation against sarcomas. Compelling in vivo data in
  • Castration resistant prostate cancer
  • Triple negative breast cancer, and by estrogen receptor association, late stage ovarian cancer
  • Endometrial cancer
  • Acute Myeloid Leukemia
  • T cell Acute Lymphoblastic Leukemia

• Current Clinical program includes Ewing, prostate, breast and ovarian cancers
Thank You

November 2017