STINGRAY THERAPEUTICS

Corporate Fact Sheet: May 2020

Adaptive + Innate Immunity is the Next Revolution in Immuno-Oncology = More Durable Efficacy in a Wider Range of Cancers.

We are working on an exciting new effort to fight cancer with 2nd generation immune oncology agents. Current efforts in this field are trying to do it with one hand tied behind their back. At Stingray, we are unleashing the full immune system to take this fight to an entirely new level.

The first generation of immune oncology therapies, checkpoint inhibitors, leverage adaptive immunity to counter cancer's immunosuppressive "checkpoints." However, many patients develop resistance or are non-responsive to treatment given tumors are "cold" (cancer unrecognized by adaptive immune system).

Next generation immunotherapy drugs target innate immunity to reveal cancer cells hidden from adaptive immunity.

Stingray Therapeutics is developing SR-8541A, an oral small molecule therapeutic targeting the key innate immunity pathway, STING (STimulator of INterferon Genes).

Stingray's target, Ectonucleotide Pyrophosphatase / Phosphodiesterase 1 (ENPP1), is the direct negative regulator of STING. This is what cancers dramatically upregulate to block innate immunity in the tumor microenvironment.

Key Advantages of Program:

Compelling Pre-Clinical Data

- 0.5-5 nM potency on ENPP1, with high selectivity
- Immune infiltration – lung, medulloblastoma, breast
- Single agent activity in a CT26 colon cancer model
- Strong abscopal effect in combination with radiation therapy in an MC38 colon cancer model
- Synergy with PARP inhibitors in blocking cancer resistance to many therapies
- Helps block production of adenosine, an important immunosuppressive target

Active M&A Landscape

- Acquisition of MavuPharma by Abbvie (7/19)
  - MavuPharma backed by Frazier Healthcare Partners ($20M Series A - 2017)
  - Pre-Clinical stage of development and potentially generated 10-20x on invested capital (terms undisclosed)
- Exits in immune oncology very similar to Stingray's program averaged ~$230M upfront and ~$950M in total deal value (including milestones) – see below

Sellers:

ViraTherapeutics
IFM Therapeutics
BeneVir
ADURO BIOTECH
Viralytics
MavuPharma
RIGONTEC

Buyers:

Boehringer Ingelheim
Johnson & Johnson
MERCK
NOVARTIS
abbvie

Average Upfront:
$230M

Technology:
Innate Immunity Modulators
Oncolytic Viruses

Average Milestones:
$950M

EQUITY OVERVIEW

Private

Financing Round Open: $5M Series 1 Round

Closed Financing: $2M Seed Round

INVESTMENT HIGHLIGHTS

Oral / Systemic Delivery

Inhibitor of the DIRECT NEGATIVE REGULATOR of vs. Direct Stimulation of STING Pathway

Direct stimulation has the potential to cause auto-immunity (lupus, Aicardi-Goutieres)

Also an important player in DNA damage response (synergistic with PARP inhibition, radiation therapy and checkpoint inhibition)

Speed to Market / ROI Regulatory Strategy

Medulloblastoma - "orphan" drug / Pediatric brain-stem cancer; eligible for FDA's fast-track review and review voucher

Also many solid tumors / breast cancer, lung, colon, etc.

Experienced Management / Scientific Teams

Raised >$50M of non-dilutive capital advancing two companies through phase 1 clinical studies

(Iterion Therapeutics / Salarius Pharmaceuticals)

Salarius – NASDAQ listing (SLRX) July 2019

RECENT NEWS

12/2019
Stingray Announced Clinical Candidate (SR-8541a) – ENPP1 Inhibitor: Oral, 1 Nanomolar Potency, Favorable ADME

IMMUNO-ONCOLOGY

A breakthrough in cancer arose with the development of immuno-oncology (I/O) therapeutics that incite the immune system to aggressively attack cancer. Promising classes of I/O therapeutics have been FDA approved in the last decade including checkpoint inhibitors (PD-1, PD-L1, CTLA-4) and CAR-T (Chimeric Antigen Receptor T-cell). Both classes primarily involve the activation of T-cells to target and attack tumors. As such, these therapeutics are aimed at boosting the adaptive immune system (antigen-dependent relying on the memory of a previous threat to mobilize against pathogens). While effective in certain patients, these therapeutics are limited to certain types of tumors and are expensive. Patients can develop serious adverse events (CAR-T ~cytokine storm) and tumors can develop resistance to the therapy. Despite these drawbacks, checkpoint inhibitors are predicted to generate more than $24 billion in sales in 2019.

The next wave of immuno-oncology therapeutics will involve harnessing the innate immune system (first line of defense responding immediately to threats) alongside of the adaptive system leading to more durable efficacy outcomes in a wider range of indications. The innate immunity hypothesis is to turn cold tumors hot (T-Cells recognize cancer and attack) by stimulating the STING pathway.

STING – MECHANISM OF ACTION (MoA)

STING is a signaling molecule in the endoplasmic reticulum present in both cancer and immune cells. It induces innate immunity and inflammatory responses in host defenses and plays a vital role in controlling the transcription of numerous host defense genes including type 1 interferons (IFNs) and pro-inflammatory cytokines following the recognition of aberrant DNA species or cyclic dinucleotides (CDNs) in the cell cytosol and tumor microenvironment.

STING – DIRECT vs. INDIRECT STIMULATION

Historically, biotech companies (Merck, Aduro) approached stimulating the STING pathway directly by injecting synthetic cyclic dinucleotides (CDNs) into tumors. The major downside of this approach included the possibility of overstimulating the target leading to a systemic auto-immune response. Conversely, Stingray has selected a target, ENPP1, which is the direct negative regulator of the STING pathway and is upregulated by cancers to block innate immunity. The pathway is limited by the natural production of CDNs, further eliminating the risk of systemic auto-immune response.

PARP INHIBITION / ADENOSINE PATHWAY

A second important MoA of ENPP1 is to support the cancer cell DNA damage response (DDR). Inhibition of DDR is synergistic with most anti-cancer therapies, and there is demand for more inhibitors in this pathway. This is because cancers rely on DDR to repair their genome and keep cancer cells alive longer. Innhibition of DDR is a proven effective mechanism for cancer therapy as evidenced by blockbuster sales of AstraZeneca's PARP inhibitor, Lynparza, in breast and ovarian cancer, and FDA approval of two other PARP inhibitors (Clovis Oncology's Rubraca and GSK / Tesaro's Zejula). ENPP1 works together with PARP to support DDR, and therefore ENPP1 inhibition is thought to be an effective complimentary approach to PARP inhibition. Stingray's compound, SR-8541A, has shown strong synergy with PARP inhibitors in preclinical models.

ENPP1 is also a key component in adenosine production, which works to suppress the immune system through activation of A2A receptors on the surface of immune cells. Inhibition of ENPP1 releases the brakes on tumor-killing immune cells, completing the trifecta of effective anti-tumor mechanisms afforded by an ENPP1 inhibitor.

2016 – 2018	2019				2020				2021				2022			
	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4

$250 K Raise
Completed
- Company formation
- Chemistry/biology to create best drug

$2 MM Seed
Completed
- Mouse efficacy studies
- Toxicology in rat & dog
- Bulk & drug formulation

$5 MM Series 1
(Open for Interest)
- FDA Approval for clinical program
- Start Dose escalation studies

$10 MM Series A
- Complete dose escalation study
- Expansion phases at dose

MANAGEMENT

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Chairman / CEO / Founder

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Chief Medical Officer / Founder

Scott Jordan
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