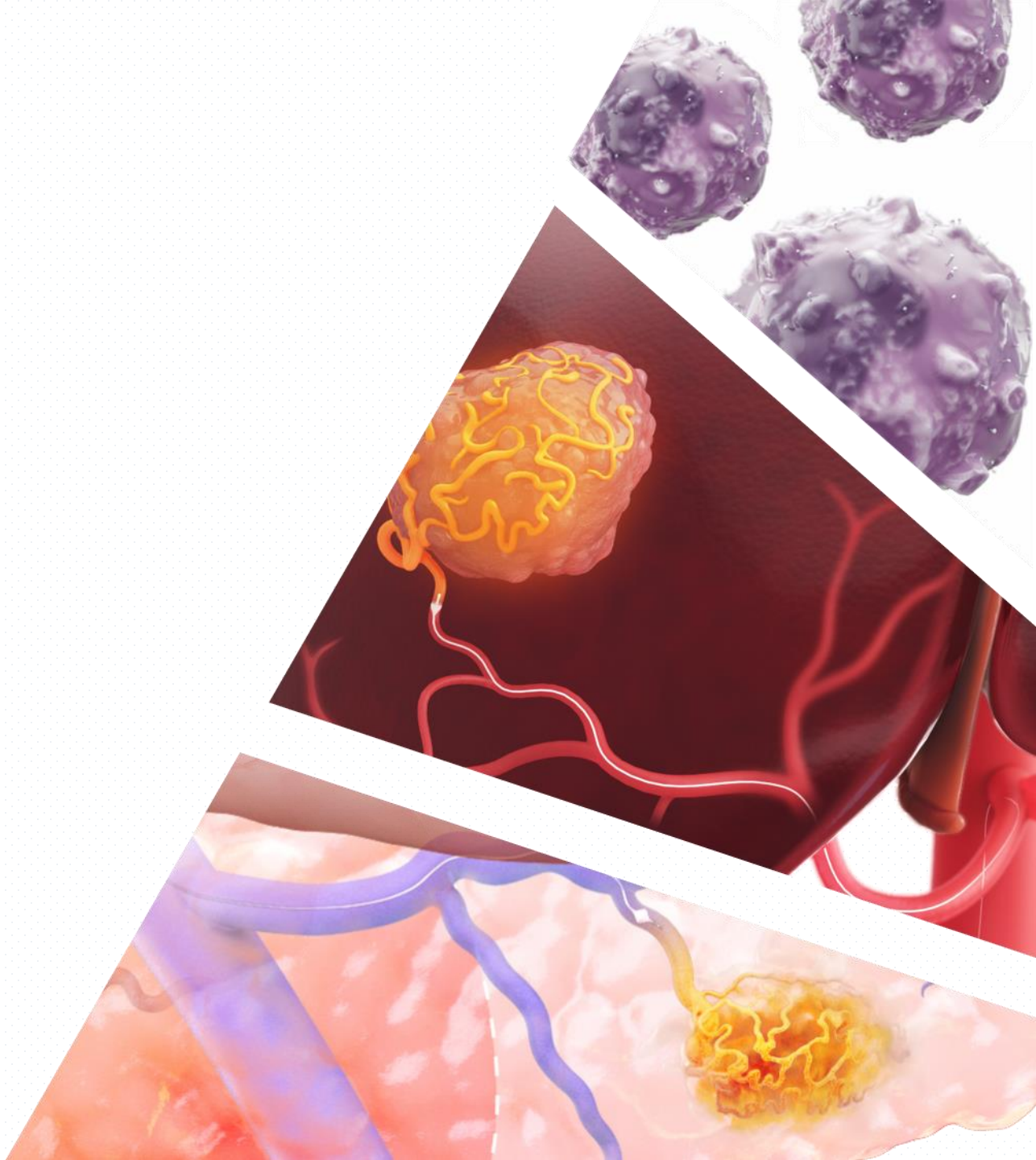
TriSalus Life Sciences

June 2024



Disclaimer

Certain statements in this presentation may constitute “forward looking statements” within the meaning of applicable United States federal securities laws. Forward looking statements include, but are not limited to, statements regarding TriSalus’s expectations, hopes, beliefs, intentions or strategies regarding the future including, without limitation, statements regarding: TriSalus’s business strategy and clinical development plans; the safety and efficacy of TriSalus’s product candidates; TriSalus’s plans and expected timing with respect to clinical trials, clinical trial enrolment and clinical trial results; the size and growth potential of the markets for TriSalus’s products and TriSalus’s ability to serve those markets; TriSalus’s ability to compete with other companies; TriSalus’s expected financial results as of and for the year and quarter ended December 31, 2023;TriSalus's projected financial results and expected cash runway; TriSalus’s ability to partner with other companies; and TriSalus’s products continuing to be subject to a favorable reimbursement environment. In addition, any statements that refer to projections, forecasts, or other characterizations of future events or circumstances, including any underlying assumptions, are forward looking statements. The words “anticipate,” “continue,” “could,” “estimate,” “expect,” “may,” “might,”“ plan,” “possible,” “potential,” “predict,” “project,” “should,” “strive,” “would” and similar expressions may identify forward looking statements, but the absence of these words does not mean that statement is not forward looking. Forward looking statements are predictions, projections and other statements about future events that are based on current expectations and assumptions and, as a result, are subject to risks and uncertainties.

Such statements are subject to a number of known and unknown risks, uncertainties and assumptions, and actual results may differ materially from those expressed or implied in the forward looking statements due to various important factors, including, but not limited to: changes in business, market, financial, political and legal conditions; unfavorable changes in the reimbursement environment for TriSalus’s products; TriSalus’s product candidates not achieving success in preclinical or clinical trials or not being able to obtain regulatory approval, either on a timely basis or at all; future clinical trial results/data may not be consistent with interim, initial or preliminary results/data or results/data from prior preclinical studies or clinical trials; TriSalus’s ability to maintain and grow its market share; the size of the addressable markets for TriNav and TriSalus’s product candidates being less than TriSalus estimates; TriSalus’s ability to successfully commercialize any product candidates that are approved; TriSalus’s ability to continue to fund preclinical and clinical trials for its product candidates; future economic and market conditions; the effects of competition on TriSalus’s business; risks relating to the uncertainty of the projected financial information with respect to TriSalus; the ability of the company to raise money to finance its operations in the future; and the outcome of any potential litigation, government and regulatory proceedings, investigations and inquiries. You should carefully consider the risks and uncertainties described in the “Risk Factors” section of TriSalus’s Quarterly Report on Form 10-Q for the quarter ended September 30, 2023, and other documents filed by TriSalus from time to time with the SEC. These filings identify and address other important risks and uncertainties that could cause actual events and results to differ materially from those expressed or implied in the forward-looking statements. Forward looking statements speak only as of the date they are made. Readers are cautioned not to put undue reliance on forward looking statements, and TriSalus and its representatives assume no obligation and do not intend to update or revise these forward-looking statements, whether as a result of new information, future events, or otherwise. Neither TriSalus or any of its representatives give any assurance that TriSalus will achieve its expectations.

Certain financial information and data contained in this presentation may be unaudited and may not conform to Regulation S-X promulgated under the Securities Act of 1933, as amended. Accordingly, such information and data may not be included in, may be adjusted in, or may be presented differently in, any documents filed with the SEC.

Overcoming Key Mechanical & Biological Bottlenecks in the Treatment of Solid Tumors

- Commercial high growth MedTech business with potential upside from device + immunotherapeutic combination
- Integrating unique device and therapeutic to overcome key challenges with drug delivery to liver and pancreatic tumors
- Lead program: Nelitolimod (SD-101), a TLR9 agonist: phase 1 data provides POC for mechanism, well tolerated safety profile, and encouraging clinical outcomes
- Exclusive worldwide rights on all intellectual property related to overcoming mechanical and biologic barriers within the Tumor Microenvironment (TME)[1]
- Multiple value-creating opportunities (clinical data, sales growth and new product launches) anticipated over the next 18 months

TriSalus™

1. TriSalus has entered into an exclusive distribution agreement for PEDD devices in China.

Novel approach to overcome key treatment barriers in liver and pancreatic tumors

Novel combination approach of innovative Pressure Enabled Drug Delivery™ (PEDD™) method with a promising therapeutic, nelitolimod (SD-101), a TLR9 agonist, to overcome mechanical and biologic TME barriers to immunotherapy success

PEDD Technology On-market

Chemoembolization and Radioembolization for Hepatocellular Cancer

Increased Therapeutic Delivery

\+

Therapeutic Nelitolimod (SD-101), a TLR9 Agonist, in Clinical Development

Uveal Melanoma Liver Metastases	Hepatocellular Carcinoma	Pancreatic Cancer
5-YEAR SURVIVAL 16%[1]	5-YEAR SURVIVAL 21%[1]	5-YEAR SURVIVAL 12%[1]

1. American Cancer Society, National Cancer Institute SEER Database as of August 2023

Two important barriers to immunotherapy success in liver and pancreatic tumors

1. Mechanical Barrier [1-3]

High intra-tumoral pressure in solid tumors limits efficient drug delivery to tumor

2. Biologic Barrier [4]

Immunosuppression in TME limits activity of therapeutic agents

1. Wilhelm et al. (2016) Nature Reviews Materials 1.5:16014.
2. Sheth, et al. (2013). Journal of Vascular and Interventional Radiology 24 (8): 1201–7.
3. TriSalus data on file from pre-clinical and clinical studies.
4. Guha, Katz, et al. Cancer Gene Ther. 24, 114–120 (2017)

SECTION 1

PEDD: pressure enabled drug delivery

Overcoming the Mechanical Barrier for Drug Delivery to Liver and Pancreatic Tumors

Addressing mechanical and biologic barriers in the TME of liver and pancreatic tumors

- **Mechanical Barriers**: high pressure within solid tumors
  - Limits delivery of sufficient therapeutic agent by standard catheters or by intravenous delivery
- **Biologic Barriers**: immunosuppressive TME limits efficacy of therapies
  - Limits effectiveness of checkpoint inhibitors and other immune modulating agents within the TME

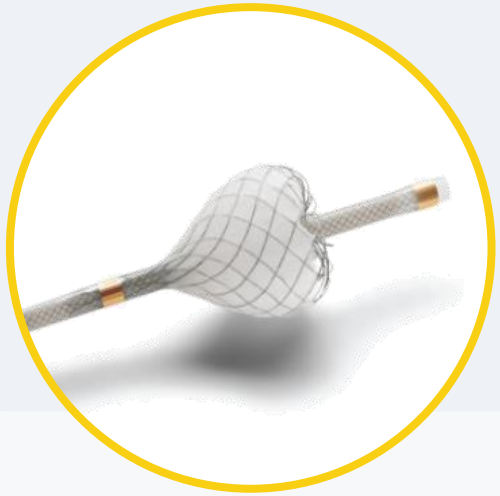
PROPOSED TRISALUS SOLUTION

A novel drug-device combination

- PEDD method to overcome high intratumoral pressure, allowing for potential unprecedented drug delivery to tumor
- TLR9 agonist to overcome immunosuppressive TME, enhancing therapeutic effects

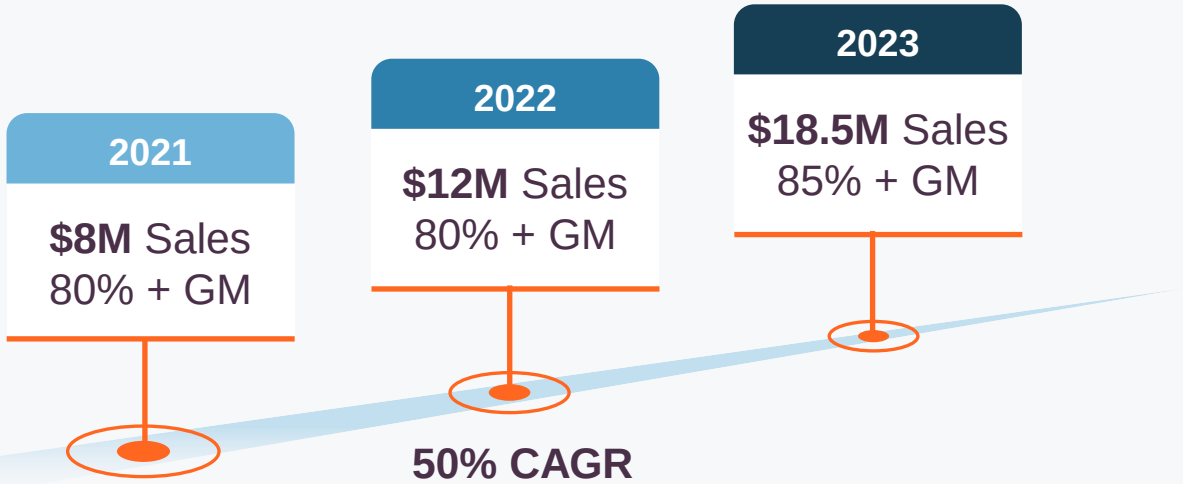
TriNav® Infusion System: a better solution for drug delivery

- FDA: 510(k) cleared device
- CMS: Unique HCPCS reimbursement code for procedures involving the TriNav Infusion System
- Drug delivery technology to overcome mechanical barriers of the high-pressure TME
- Atraumatic, self-expanding/collapsing SmartValve® technology
- Clinically validated in multiple studies
- Additional technology expansion opportunities with potential immunotherapy partners

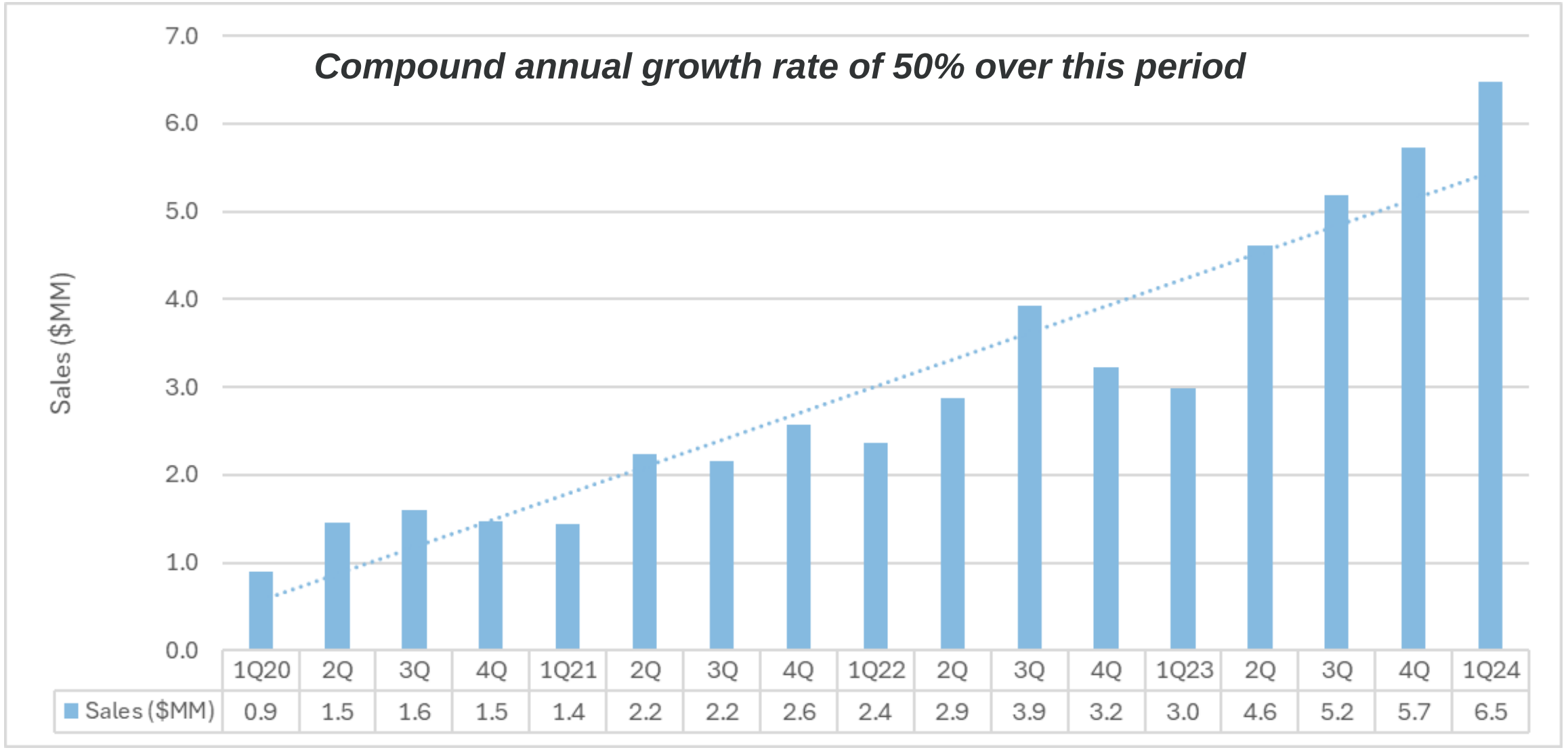


TriNav Infusion System

Commercial-stage, FDA-cleared technology using the proprietary PEDD method



TriSalus Life Sciences, Inc. – Historical Quarterly Sales 1Q-2020 to 1Q-2024

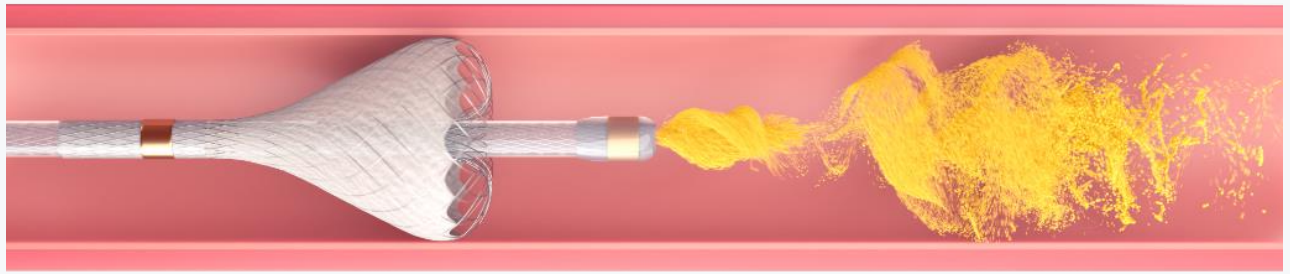


PEDD Method: How it Works

Overcomes barriers in the tumor microenvironment to increase therapeutic delivery to tumors

TriNav PEDD with SmartValve Technology

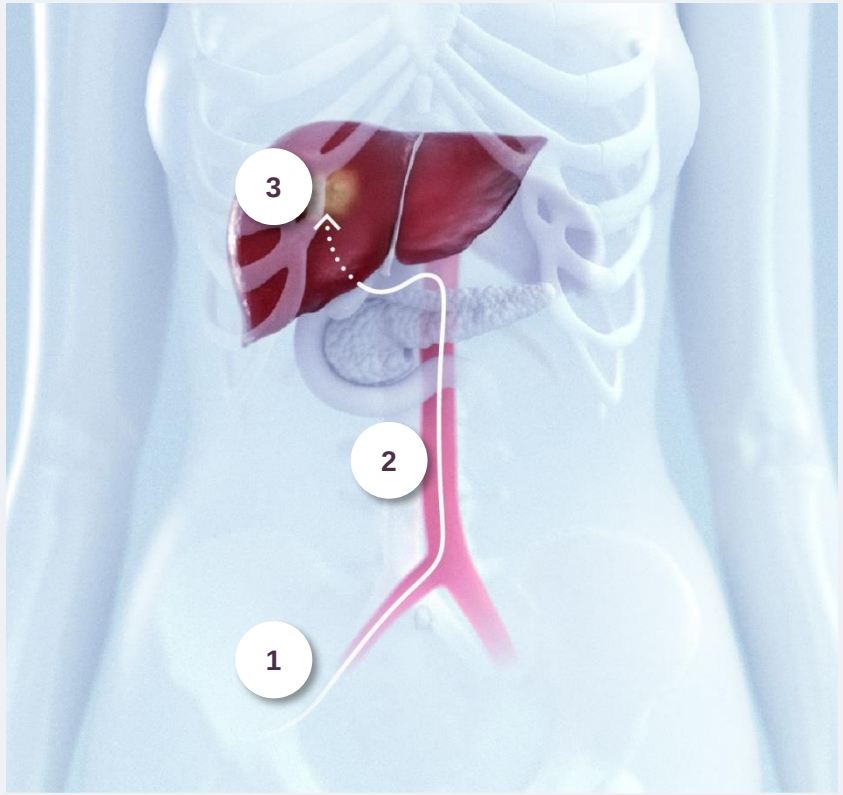
- **Enhances perfusion by modulating the pressure gradient**
- **Improves target delivery by redirecting blood flow to the tumor and away from normal liver**
- **Creates turbulence to mix therapeutic with the blood for reliable distribution**
- **Reduces reflux to protect normal tissue outside the liver**



- SmartValve can modulate intravascular pressure for regional delivery of nelitolimod (SD-101) directly to the site of the disease.

PEDD = Pressure-Enabled Drug Delivery

TriNav is used in embolization procedures - to deliver either chemotherapy or radiation therapy beads directly to liver tumors - to destroy cancer cells



Performed for tumors that cannot be surgically removed or resected:

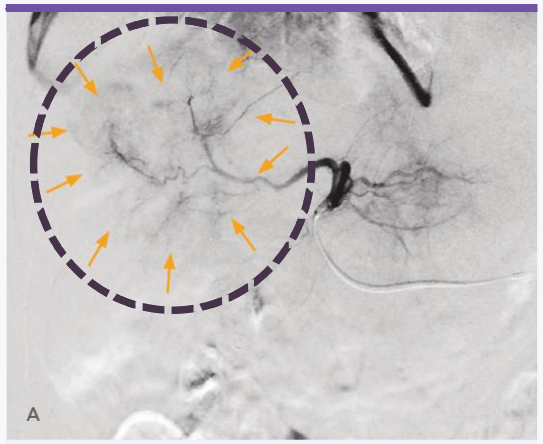
1. Insertion through small puncture in artery
2. X-rays are used to guide device into liver
3. Enhanced pressure and flow maximize dose to tumor

PEDD = Pressure-Enabled Drug Delivery.

PEDD opens collapsed vessels and improves delivery into high pressure tumors[1]

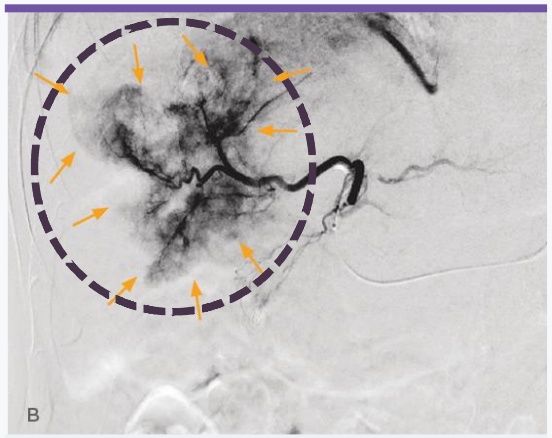
Same liver cancer patient treated with different devices

Standard Catheter



Failure to penetrate tumor may limit therapeutic effectiveness

PEDD



Collapsed vessels opened for deep perfusion throughout tumor

Angiogram of tumor vessels demonstrating that PEDD:

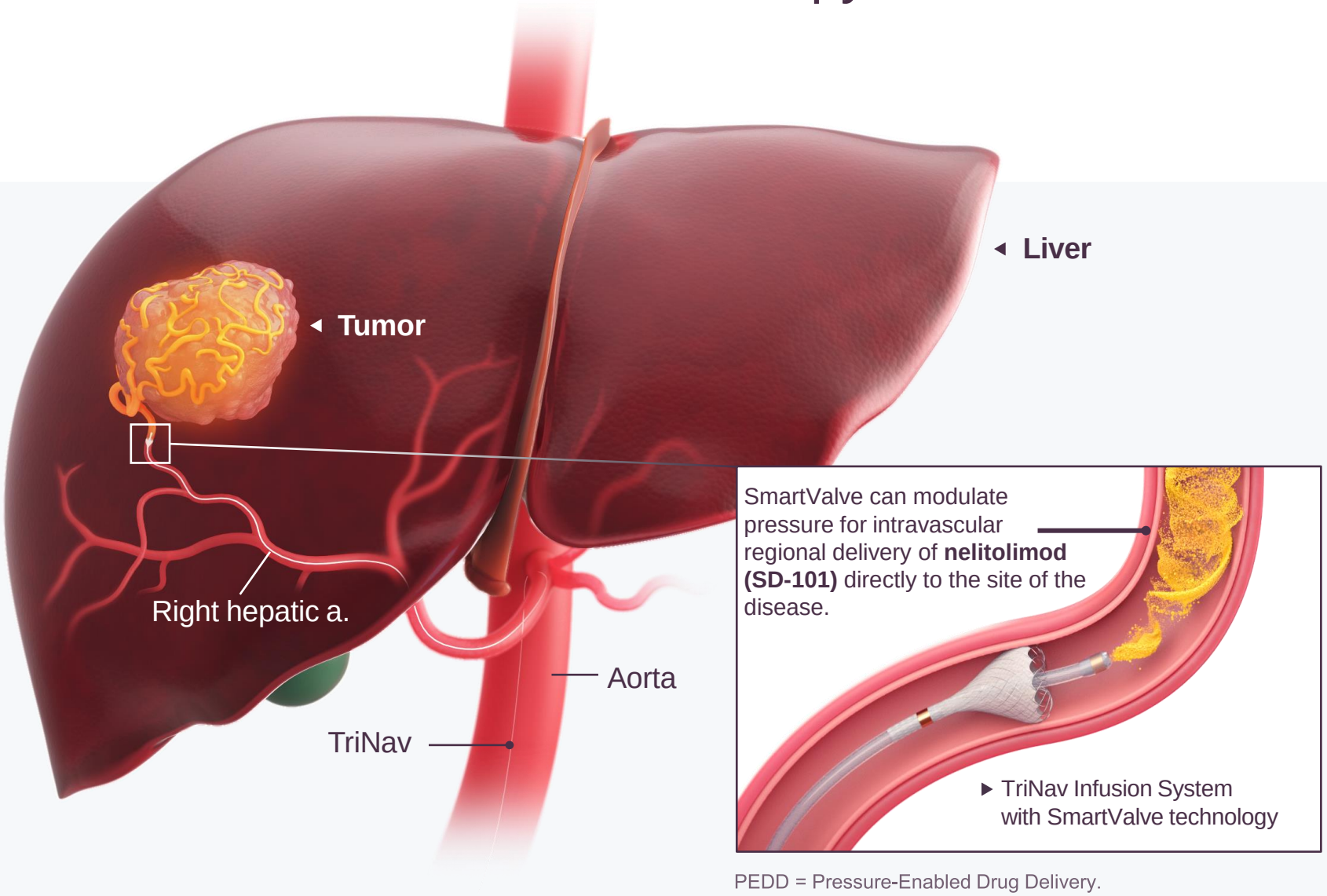
- ↑ Delivers contrast dye into liver tumor
- ↑ Opens collapsed tumor vessels
- ↓ Reflux of contrast dye into normal liver

1. TriSalus images and data on file

TriNav also overcomes infusion barriers for immunotherapy

TriNav infuses therapeutics into the vessels that supply blood to the organs and tumors.

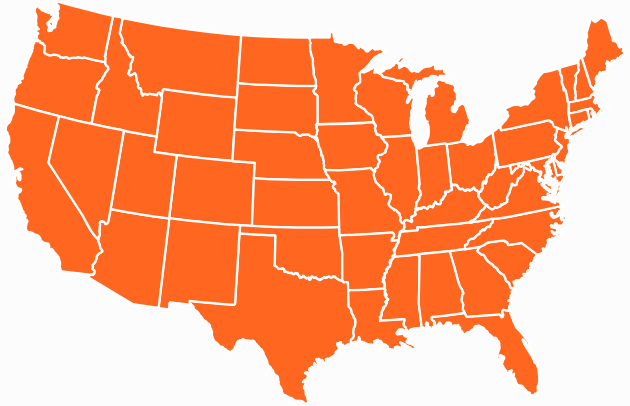
This ensures targeted drug delivery inside the organ with minimal systemic toxicity.



PEDD = Pressure-Enabled Drug Delivery.

A recently published real-world evidence study[1] provides evidence that TriNav successfully treats complex liver cancer patients

Population/Setting



- 300 million patient lives
- > 98% of US payers
- Representative of the entire US population

Study Design

- Study compared 258 TriNav patients to 8,940 non-TriNav patients

TriNav Patient Type - Key Findings

TriNav patients are more complex:

- They have more comorbidities, and liver-related adverse events
- They have had prior embolization's and/or prior systemic therapy
- TriNav patients are sicker and showed a higher burden of disease

onfid

Key Comparitive Findings

- In chemoembolization's:
  - TriNAv delivered 40% more doxorubicin
- Higher disease burden patients receiving TriNav had outcomes similar to healthier non-TriNav patients
- In matched cohort analyses, TriNav patients did better
  - 48% increase in liver transplantation
  - 50% reduction in 30-day inpatient admissions
  - 17% reduction in complications
  - 40% reduction in fatigue

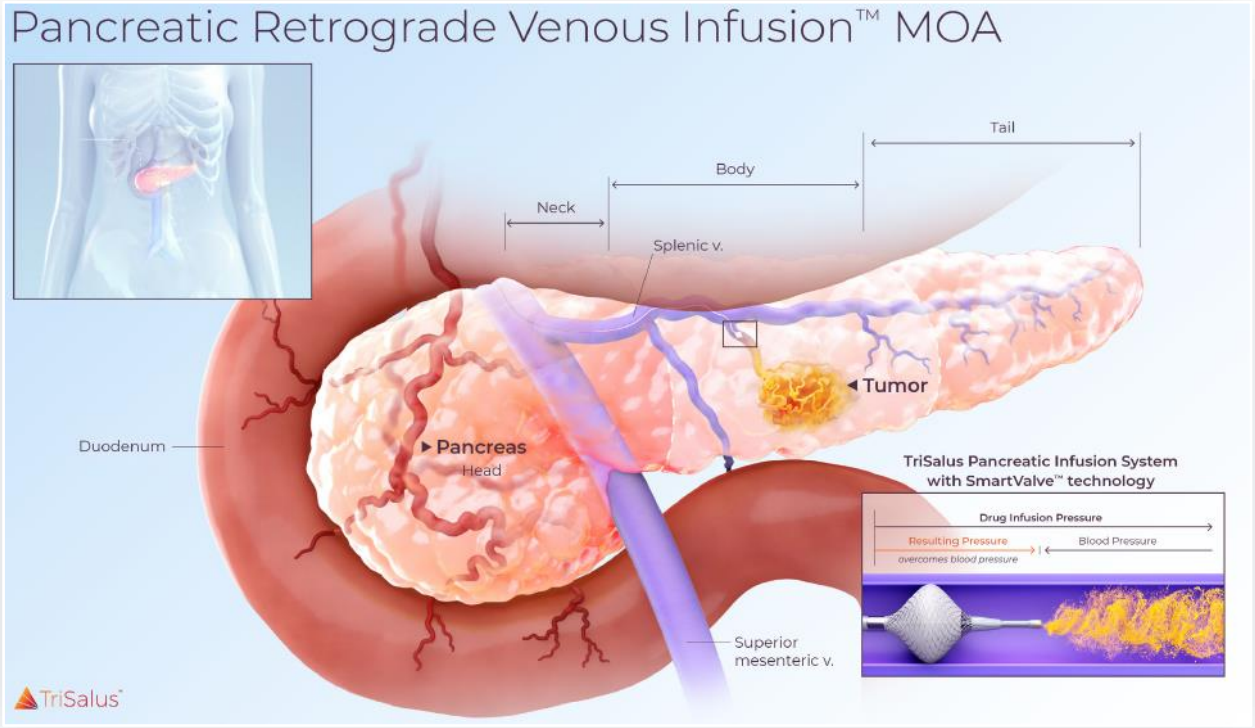
TriSalus

1. Cook et al, Real-world evidence of Pressure-enabled Drug Delivery, *Current Medical Research and Opinion*, March 2024

TriSalus developed a separate, novel PEDD method for the Pancreas

FDA-cleared and in phase 1 clinical trials with Nelitolimod

- Poor blood flow limits drug access to the pancreas[1,2,3]
- Pancreatic arteries difficult to access[4,5]
- Innovative retrograde venous approach eliminates need for balloons that eliminate blood flow[6,7]
- Target vessel pressure monitoring for safety, efficacy, and consistency
- Leveraging PEDD and nelitolimod (SD-101) data from liver trials
- Phase 1 locally advanced pancreas data from MDACC was presented at SITC 2023



1. Rakesh Jain (2013) Normalizing Tumor Microenvironment to Treat Cancer: Bench to Bedside in Biomarkers. 31:17 2205-2218.
2. DuFort et al, Interstitial Pressure in Pancreatic Ductal Adenocarcinoma Is Dominated by a Gel-Fluid Phase. Biophysical Journal 110 2106-2119.
3. Soltani et al Numerical Modeling of Fluid Flow in Solid Tumors. PLoS ONE 6:6 e20344
4. Homma, H. et al. Cancer 89, 303–313 (2000).
5. Rosemurgy, A. S. et al. J Pancreat Cancer 3, 58–65 (2017).
6. Piras, C., Paulo, D. N. S., Paulo, I. C. A. L., Rodrigues, H. & Silva, A. L. da. Acta Cirurgica Brasileira 25, 105–110 (2010).
7. Moody, A. R. & Poon, P. Y. American Journal of Roentgenology 158, 779–783 (1992). 5. Okahara, M. et al. Abdom Imaging 35, 134–142 (2010).

Clinical and preclinical data demonstrate superiority of PEDD method

Therapeutic Modality	TriNav Improvement vs. Standard Catheter		
TACE	**60% ↑**	in therapeutic delivery to liver tumors[1] vs. standard catheter	Clinical liver study
TARE (Y-90)	**33% -90% ↑**	in MAA deposition in liver tumors[2] vs. standard catheter	Clinical liver study
Immunotherapy (nelitolimod, SD-101)	**High concentrations in liver tissues with low serum exposure**	undetectable in serum after 4 hours in 97% of patients[3]	Clinical liver study
Chemotherapy	**6.7 – 10.1** fold ↑	improved delivery vs. systemic infusion[4]	Preclinical pancreas study

TACE = Transarterial chemoembolization
TARE = Transarterial radioembolization

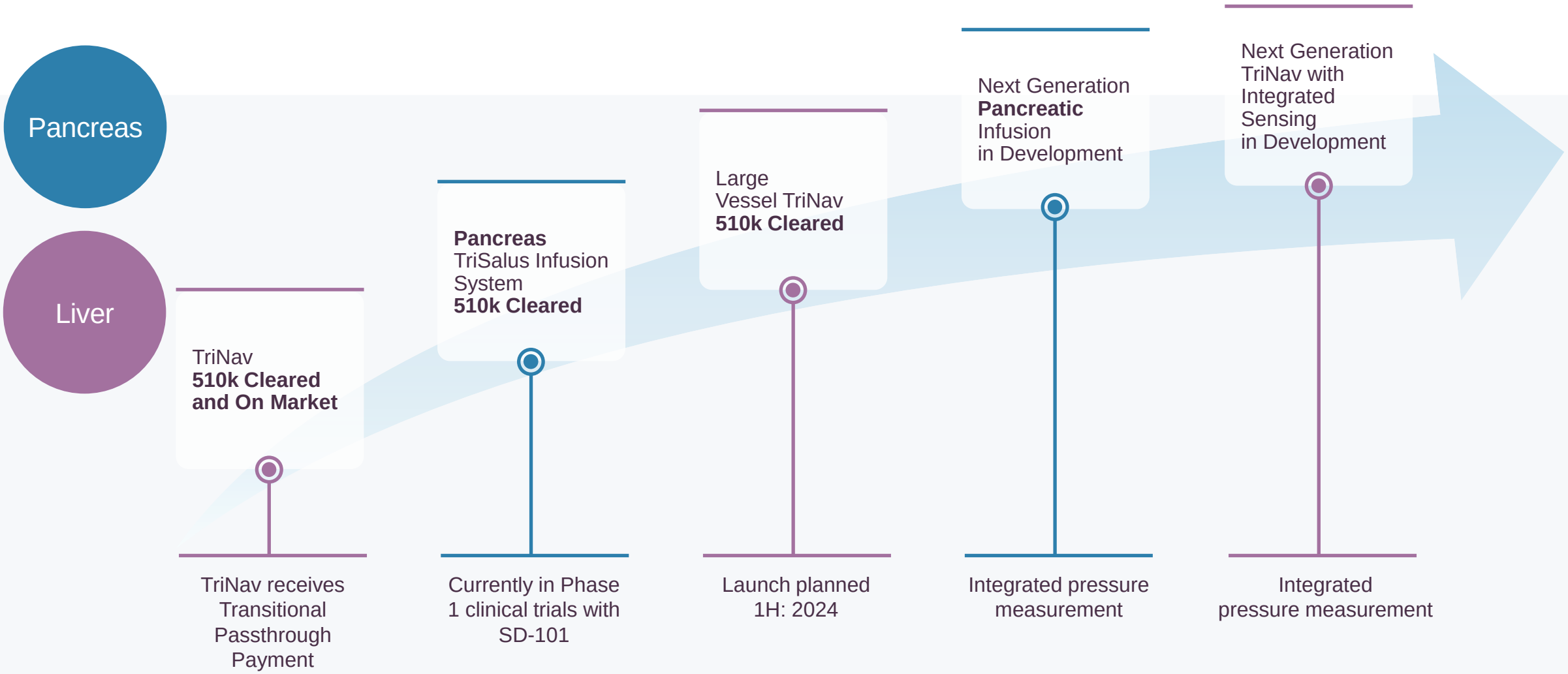
1. Titano JJ, et al. Cardiovasc Intervent Radiol. 2019;42:560-568

2. Pasciak AS, et al. J Vasc Interv Radiol. 2015;26:660-669

3. TriSalus clinical data on file

4. Pressure-enabled delivery of gemcitabine in an orthotopic pancreatic cancer mouse model. Surgery 2020;168(3):448-456. Data on file, Porcine Animal Model, TriSalus Life Sciences, 2019

TriSalus technology pipeline: opportunities for further expansion



SECTION 2

Nelitolimod (SD-101): Class C TLR9 Agonist

Overcoming the Biologic Barrier in Liver & Pancreatic Tumors

Pipeline: potential commercial opportunities across range of liver and pancreatic tumors

PRESSURE ENABLED REGIONAL IMMUNO-ONCOLOGY (PERIO) TRIALS

INDICATION	TRIAL DESIGN	IND ENABLING	PHASE 1	PHASE 2	PHASE 3
Uveal Melanoma Liver Metastases (validation of combination)	Nelitolimod + PEDD HAI + CPI	Phase 1/1b PERIO-01 Trial			
Hepatocellular Cancer (HCC)[1]	Nelitolimod + PEDD HAI + CPI	Phase 1b PERIO-02 Trial			
Intrahepatic Cholangiocarcinoma (ICC)[1]	Nelitolimod + PEDD HAI + CPI	Phase 1b PERIO-02 Trial			
Locally Advanced PDAC	Nelitolimod + PEDD PRVI + CPI	Phase 1/1b PERIO-03 Trial			

CPI = Checkpoint Inhibitors; HAI = Hepatic Arterial Infusion; PDAC = Pancreatic Ductal Adenocarcinoma; PRVI = Pancreatic Retrograde Venous Infusion; IND = Investigational New Drug
1. HCC and ICC will be studied jointly in phase 1b. Separate phase 2 studies will be opened for each indication.

Clinical trials leverage established biology of nelitolimod (SD-101)

PRESSURE ENABLED REGIONAL IMMUNO-ONCOLOGY (PERIO) TRIALS

Enrollment criteria

- Liver or pancreas main site of disease
- Failure or refusal of standard treatment
- Good performance status

Trial Design

- Cohorts with SD-101/PEDD alone
- Cohorts with SD-101/PEDD + IV checkpoint
- Six outpatient SD-101 infusions in IR suite

Endpoint

- Safety and dose determination
- Efficacy – progression free survival
- ctDNA – strong correlate for overall survival[1,2,3]
- Immune assays to confirm MoA

1. Carvajal Nat Med 2022
2. Dawson NEJM 2013
3. Al-Showbaki JITC 2023

MoA – Mechanism of action
HCC – hepatocellular carcinoma
ICC – intrahepatic cholangiocarcinoma
PDAC – pancreatic ductal adenocarcinoma

Nelitolimod (SD-101) dual mechanism of action overcomes biological TME barriers

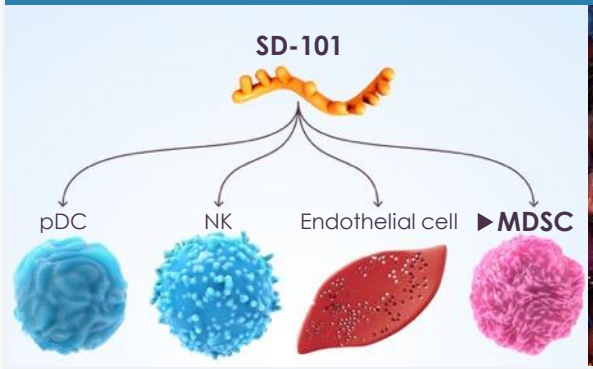
SD-101 reprograms the TME through multiple mechanisms

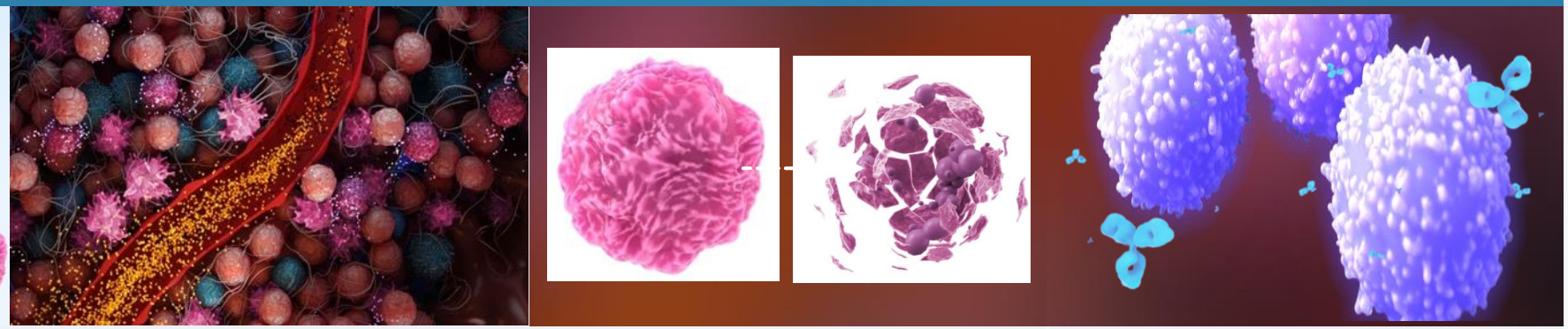
1 SD-101 binds to TLR9

2 PEDD allows sufficient SD-101 delivery into TME

3 Eliminates immunosuppressive MDSCs

4 T cells accumulate in tumor for CPI binding





SD-101 with immune adjuvant effect on multiple cell types

Dual mechanism of action
to prime tumor for checkpoint inhibitor (CPI)

T cells recruited to tumor, which may enable durable disease control and longer survival **without** need for early tumor size reduction

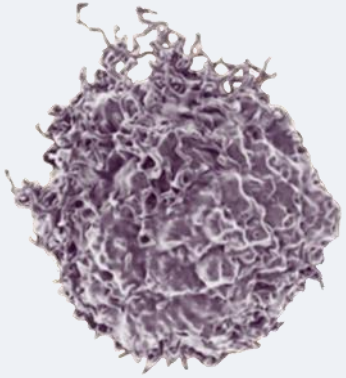
SD-101 Target (TLR9) Present Across Cancer Type

TME = tumor microenvironment
MDSC = myeloid derived suppressor cell

Clinical proof-of-concept

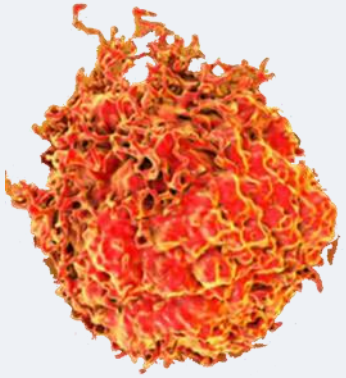
DUAL MOA WITH POTENTIAL TO ENHANCE CHECKPOINT ACTIVITY BOTH IN TME AND SYSTEMICALLY

Unresponsive "Cold" Tumor Pre-SD-101



- MDSC accumulation
- T cell paucity
- Immunotherapy failure

Responsive "Hot" Tumor Post-SD-101

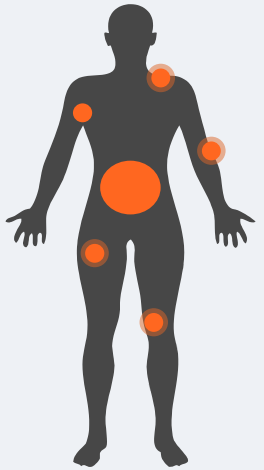


PEDD + Nelitolimod (SD-101)

- MDSC depletion
- T cell infiltration
- Enhanced CPI activity

PEDD unlocks dual mechanism of action in liver and pancreas[1]

Enhanced CPI Effects in Target Organ and Systemically



Systemic immune stimulation as evidenced by declines in ctDNA with disease control and early evidence for progression free survival[2]

MDSC – myeloid derived suppressor cells
1.Data on File. 2.Patel SITC 2023

Nelitolimod (SD-101) clinical data consistent with established drug Mechanism of Action

1. **Optimal dose range** predicted by preclinical models and known Mechanism of Action (MoA) – approach consistent with FDA Project Optimus, which supports role for Optimal Biological Dose-based (OBD-based) decisions
   - Accommodates non-linear dose effects
2. Immune markers and **liquid biopsies** (ctDNA) used to confirm nelitolimod MoA as conventional, scan-based RECIST ORR are assessments less reliable (when immune cell infiltration distorts tumor size)
   - ctDNA levels in blood shown to be highly predictive of PFS and OS when imaging unreliable[1,2,3]

- Nelitolimod relieves immunosuppression in TME, may yield PFS/OS benefit in absence of robust RECIST ORR
- Liquid biopsy (ctDNA levels) more accurate predictor of survival than imaging (RECIST ORR)[1,2,3]
- Optimal dose determination for nelitolimod to be driven by drug's biological effects and not by MTD/DLT

1. Carvajal Nat Med 2022
2. Dawson NEJM 2013
3. Al-Showbaki JITC 2023

RP2D: recommended phase 2 dose
MTD: maximum tolerated dose
DLT: dose limiting toxicity

Nelitolimod (SD-101) is highly distinct from other TLR9 agonists

PEDD ENABLES MOA THAT ALIGNS WITH LIVER AND PANCREAS BIOLOGY

Nelitolimod induces significantly greater gene expression changes[1] compared with class A or B TLR9 in liver metastasis model along with depletion of immunosuppressive MDSC[2]

1. Activated STAT3 drives liver MDSC expansion, survival, and function
2. MDSC (key immunosuppressive cells in TME) express TLR9
3. TLR9 signaling triggered by SD-101 can **deactivate** STAT3

- Liver MDSC depletion and enhanced CPI effect seen in liver metastasis model[3]
- MDSC **depletion (relieving TME immunosuppression)** seen in phase 1
- Dual MoA predicted by preclinical model and supported by phase 1 data[4, 5]

Optimal dose selection to be based on desired immune effects

MDSC – myeloid derived suppressor cells

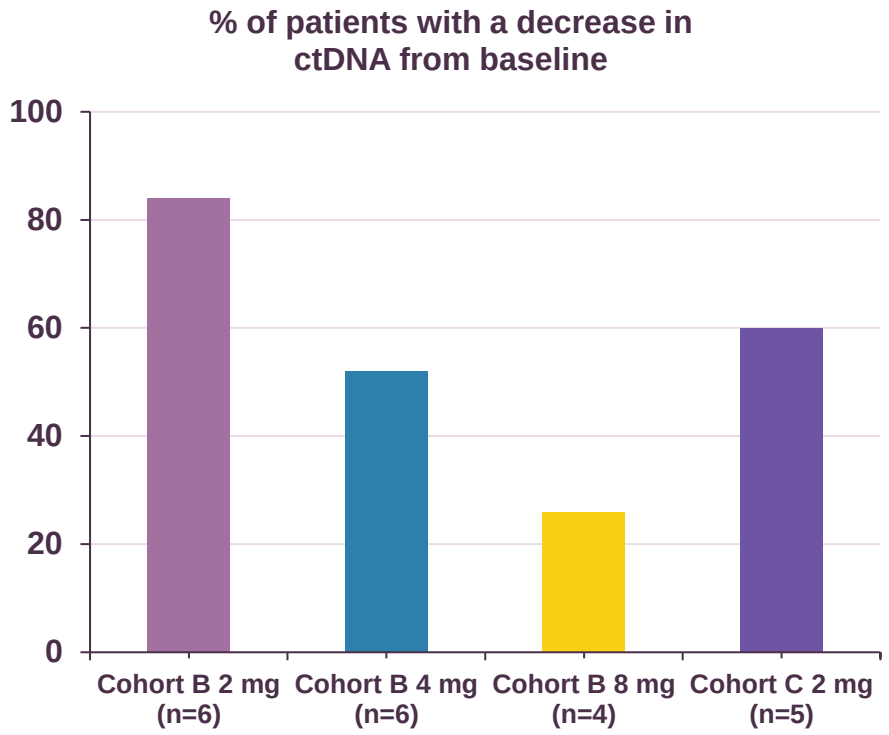
1. TriSalus unpublished
2. Ghosh SITC 2022
3. Ghosh Cancer Gene Therapy 2022
4. Montazeri ASCO 2023
5. Patel SITC 2023

Liquid biopsy showing ctDNA reduction predicts survival in uveal melanoma

PHASE 1 DATA CONSISTENT WITH CLINICAL ACTIVITY IN PREDICTED ACTIVE DOSE RANGE

- Nelitolimod (SD-101) causes substantial tumor inflammation and cell infiltration - tumor size increase unrelated to tumor cell proliferation and complicates RECIST imaging for ORR
- Even progressive disease patients with ctDNA decrease may survive long term (role for PFS endpoint)
- ctDNA recently emerged as better predictor of disease control and survival than ORR
- Not yet a regulatory endpoint, but well-validated predictor of OS in UM and other indications[1,2,3]

68% MOLECULAR RESPONSE RATE WITH 28% CR (total elimination of ctDNA) [4]



1.Carvajal Nat Med 2022
2. Dawson NEJM 2013
3. Al-Showbaki JITC 2023
4. Patel SITC 2023

Cohort B – SD-101/PEDD + nivo
Cohort C – SD-101/PEDD + nivo/ipi

Dose optimization guided by clinical and immune signals

DOSE WITHIN PREDICTED RANGE ELICITS EXPECTED IMMUNE SIGNALS WITHIN LIVER METASTASES, PHASE 1

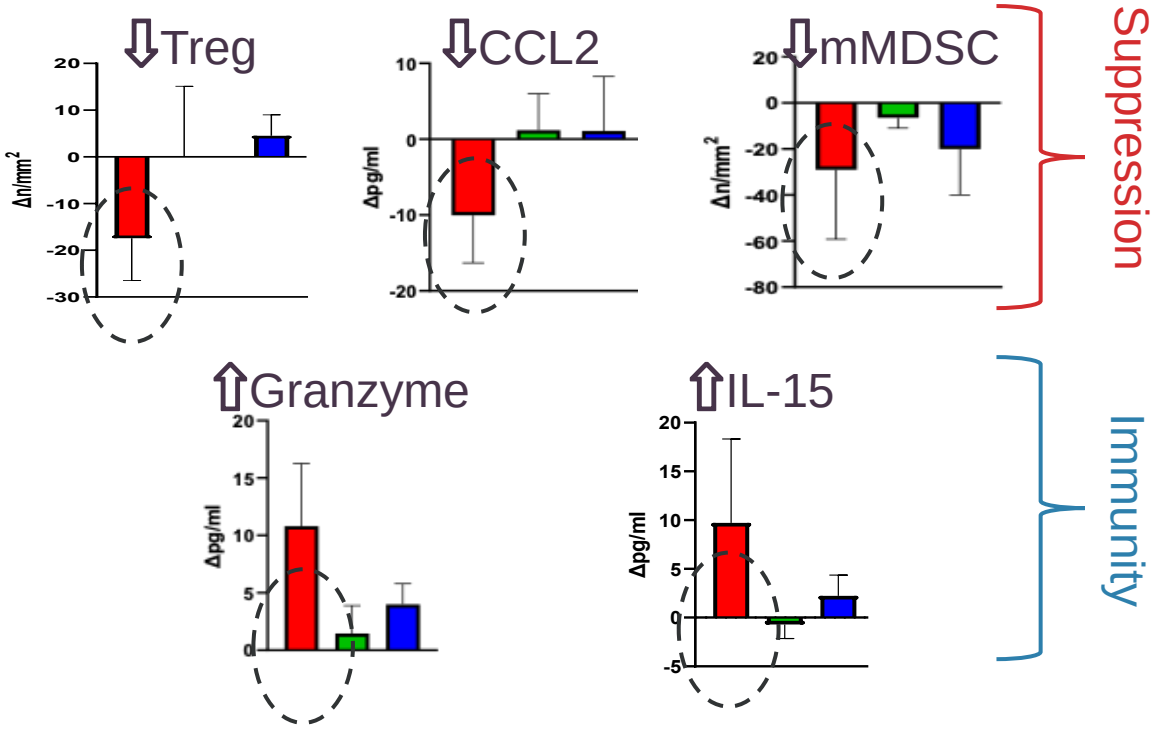
At 2 mg SD-101 via PEDD + nivolumab:

- ✓ > 80% ctDNA response rate
- ✓ > 80% disease control rate
- ✓ 11.7-month progression free survival (PFS)
- ✓ Immune signals predictive of clinical effect:

⇧ Granzyme B – protein used by T cells to kill tumor cells

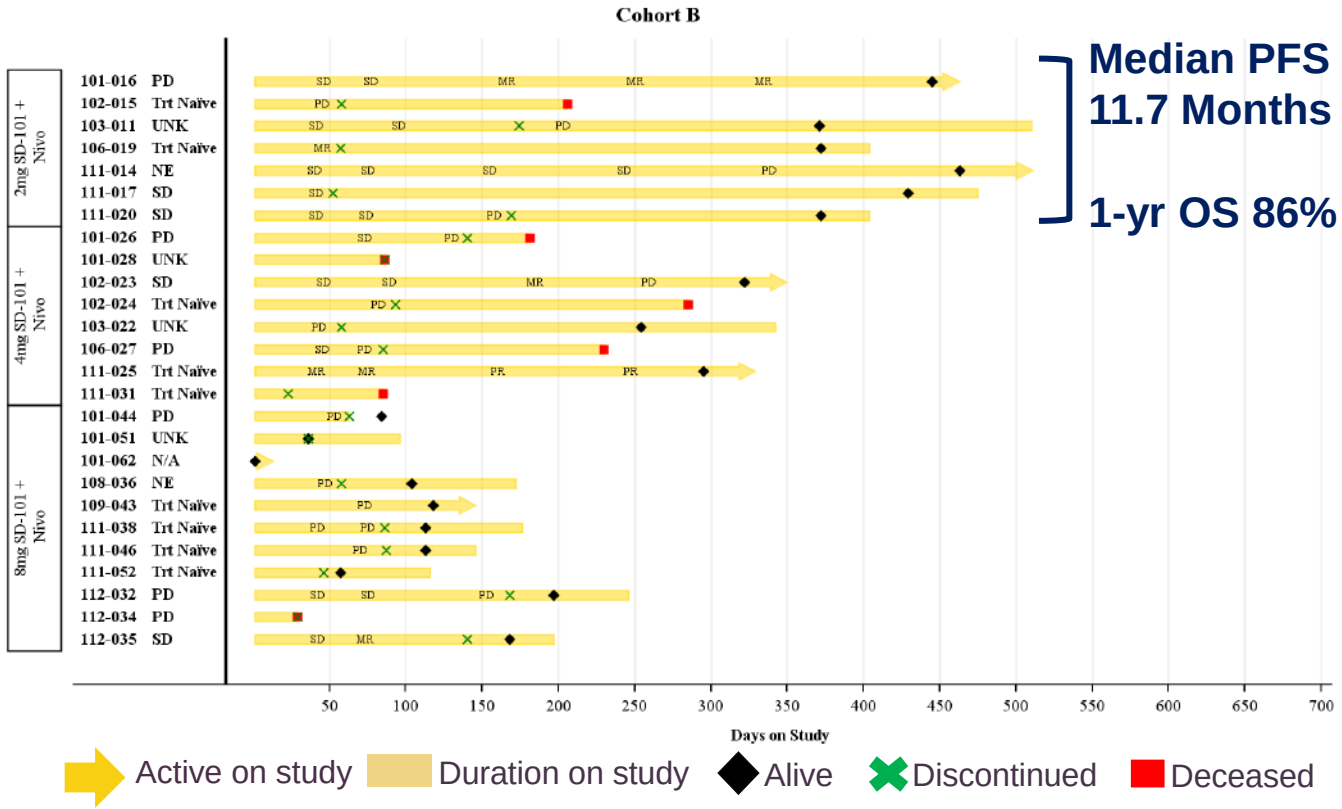
⇧ IL-15 – cytokine stimulating anti-tumor T + NK cell immune responses

⇩ MDSC and Treg in liver tumors – fewer cells that drive CPI failure



Nelitolimod (SD-101) durable disease control and PFS in phase 1[1]

NEXT PHASE WILL FURTHER EXPLORE PFS CONVERSION INTO OS BENEFIT AS CIRCULATING TUMOR DNA LEVELS MAY PREDICT[2,3,4]



MR = minor response (10-29% decrease) **PR** = partial response (≥30% decrease)

71% 2L and beyond, including 4L and 6L patients

59% ctDNA clearance[1] as best on-treatment response and 27% clearance at fixed time point (13% with tebentafusp)[2]

ctDNA reported as predictor of overall survival in stage IV uveal melanoma when imaging is unreliable[2]

Even progressive disease patients with ↓ctDNA may survive long term

6 of 7 of 2mg + nivo patients with > 50% decrease in ctDNA including 4 ctDNA clearance as best on-treatment response

1. Patel SITC 2023
2. Carvajal Nat Med 2022
3. Dawson NEJM 2013
4. Al-Showbaki JITC 2023

Nelitolimod (SD-101) well tolerated with low level of serious adverse events

TS-PERIO-01 Phase 1 (1L if Kimmtrak ineligible; 2L+ if Kimmtrak eligible)	
sitc Society for Immunotherapy of Cancer | sitc 2023 San Diego Nov. 1–5	**TriSalus (nelitolimod)** N=56 (phase 1)[1]
Stage IV UM LM population eligible	100%
Grade 3 or 4 treatment related serious adverse events	11% (4% at optimal dose)
Grade 2 or higher cytokine release syndrome	2%

- PEDD concentrates nelitolimod in liver with well tolerated systemic immune effects
- Nelitolimod undetectable in serum after 4 hours in 97% of subjects[2]
- Kimmtrak is approved for stage IV UM but <50% of the population is eligible based on HLA type
- Grade 3/4 adverse event rates with immunotherapy in this population are typically >30%[4]

1. Patel SITC 2023, 2. Montazeri ASCO 2023 4.Nathan NEJM 2021

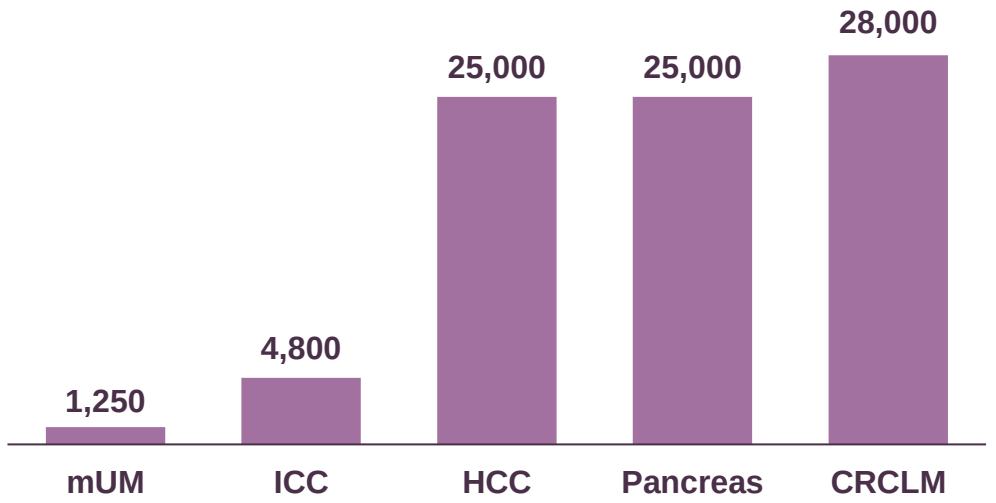
Unmet needs create broad market opportunities across multiple indications

- Addressing unresectable disease in liver and pancreas

- Target indications all areas of high unmet need with poor overall survival
- Total available market > 80,000[1] in the U.S.
- Dual mechanism of action and unique route of administration bring potential safety and efficacy advantages
- TriSalus retains worldwide commercial rights

1. SEER Database 2023

NELITOLIMOD (SD-101) ADDRESSABLE PATIENT POPULATION



Source: SEER database

Available market includes **uveal melanoma liver metastases, intrahepatic cholangiocarcinoma, HCC, PDAC,** and **CRC with liver metastases**

2023 – 2024: Anticipated Key Milestones

Catalyst	Indication	Anticipated Timing
Phase 1 PERIO Data	Uveal Melanoma	2H 2023 (Complete)
Confirmation of optimal dose	Uveal Melanoma	1H 2024
Phase 1b PERIO Data	Hepatocellular Cancer and Intrahepatic Cholangiocarcinoma	1H 2024
Launch of TriNav Large	Hepatocellular Cancer and liver metastases	2H 2024
Phase 1 PERIO Data	Locally Advanced Pancreatic Cancer	2H 2024
Phase 1b with IV checkpoint	Locally Advanced Pancreatic Cancer	1H 2025

Executive Team

Mary Szela
CEO & President

Sean Murphy
Chief Financial Officer

Steven Katz, MD, FACS
Chief Medical Officer, Chairman of SAB

Jodi Devlin
President, Therapeutics

Jennifer Stevens
Chief Regulatory Officer

Bryan Cox, PHD
Chief of Research

Richard Marshak, VMD
Senior Vice President, Business Development and Strategy

Jim Young
Senior Vice President, Investor Relations and Treasurer

Abbott
HUMIRA adalimumab

MALIN
EVERCORE
Abbott

Memorial Sloan Kettering Cancer Center
NYU School of Medicine
BROWN Alpert Medical School
Takeda

Baxter
ALTATHERA PHARMACEUTICALS
Abbott

MERCK
FDA

Abbott
Nephraegis Therapeutics

Abbott
Nephraegis Therapeutics

Abbott
EY

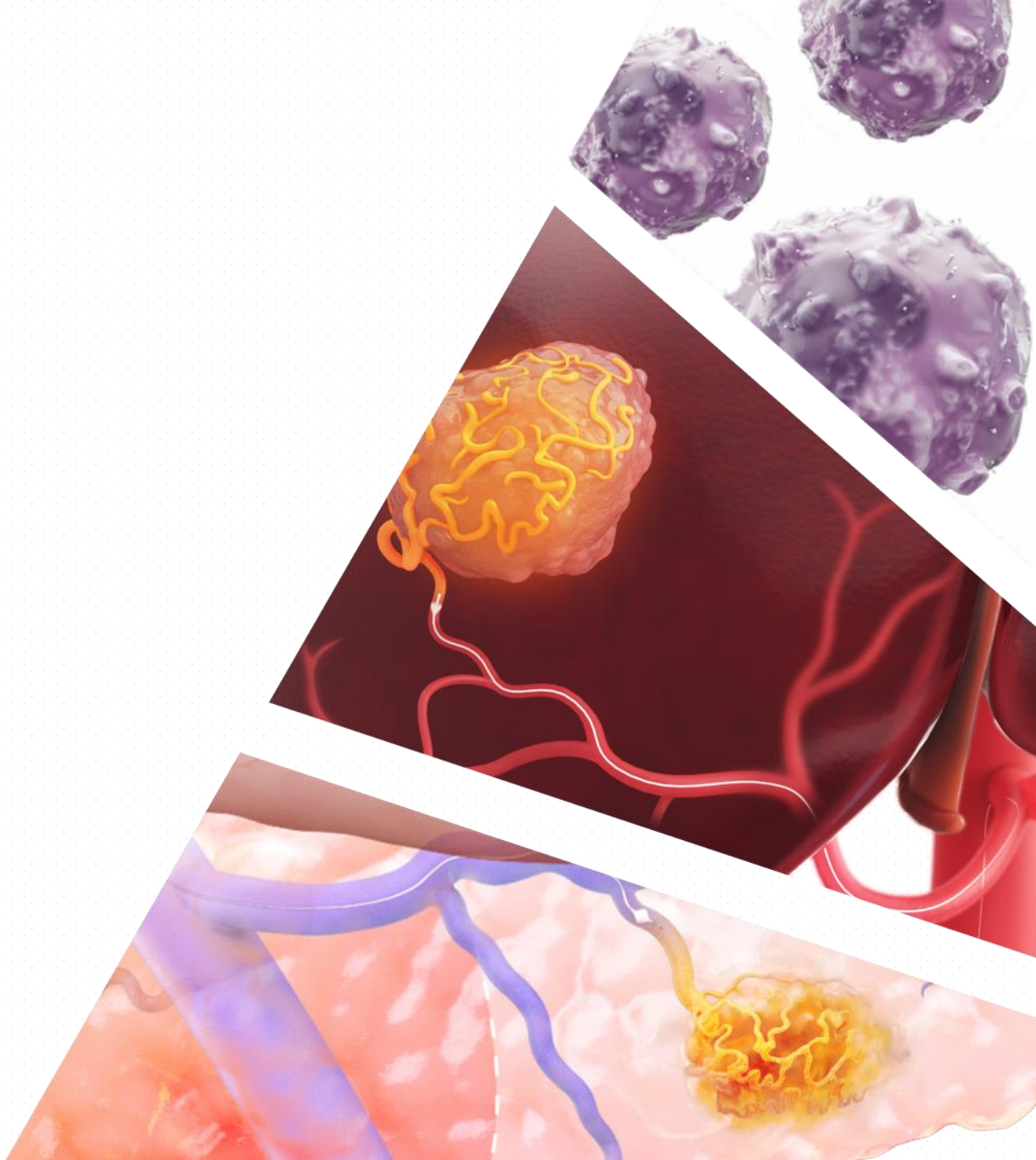
Overcoming Key Mechanical & Biological Bottlenecks in the Treatment of Solid Tumors

- Commercial MedTech business with consistent growth and potential upside from device + immunotherapeutic combination
- Integrating unique device and therapeutic to overcome key challenges with drug delivery to liver and pancreatic tumors
- Lead program: Nelitolimod, (SD-101), a TLR9 agonist: phase 1 data provides proof of concept for mechanism and well tolerated safety profile
- Exclusive worldwide rights on all intellectual property related to overcoming mechanical and biologic barriers within the Tumor Microenvironment (TME)[1]
- Multiple value-creating opportunities (clinical data, sales growth, and new product launches) anticipated over the next 18 months

TriSalus™

1. TriSalus has entered into an exclusive distribution agreement for PEDD devices in China

TriSalus LIFE SCIENCES®



Argot Partners

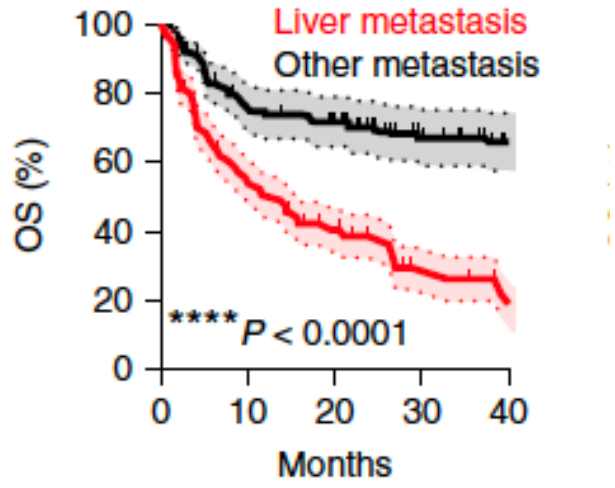
New York

767 Third Avenue, 34th Floor
New York, NY 10017

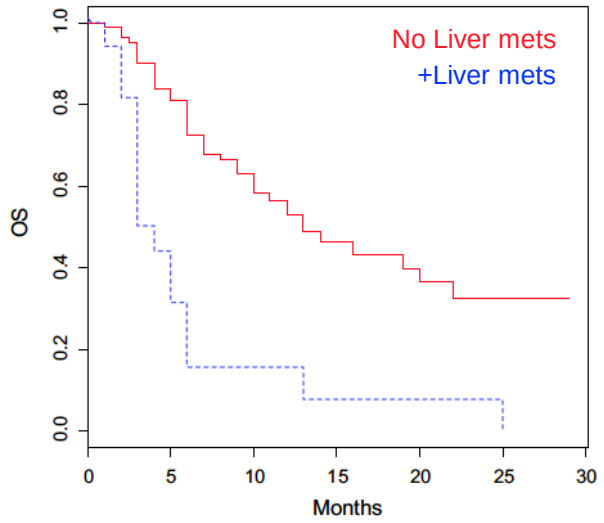
212.600.1902
trisalus@argotpartners.com

Do liver tumors drive immunotherapy failure?

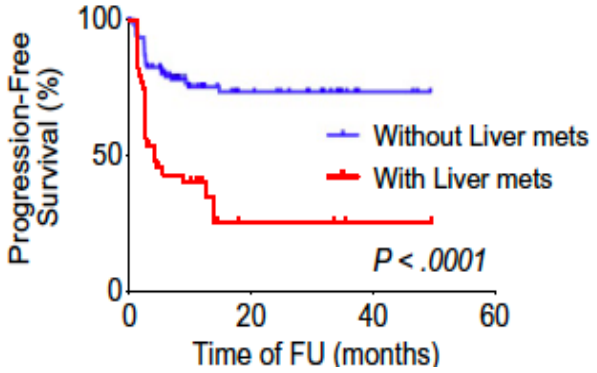
In **multiple indications,** liver mets predicted CPI failure **in association with myeloid cell driven suppression**[1]



In **lung carcinoma** patients, the presence of liver mets was an independent predictor of CPI failure[2]



In **cutaneous melanoma** patients, liver mets predicted inferior PFS and OS[3]



1. Yu J, Green MD, Li S, et al. Liver metastasis restrains immunotherapy efficacy via macrophage-mediated T cell elimination. Nat Med. 2021;27:152-164. https://doi.org/10.1038/s41591-020-1131-x
2. Botticelli A, Salati M, Di Pietro FR, et al. A nomogram to predict survival in non-small cell lung cancer patients treated with nivolumab. J Transl Med. 2019;17:99. https://doi.org/10.1186/s12967-019-1847-x
3. Silva I, Lo S, Quek C, González M, Carlino M, Long G, and Menzies A. Site-specific response patterns, pseudoprogression, and acquired resistance in patients with melanoma treated with ipilimumab combined with anti–PD-1 therapy. Cancer. 2019;126: 10.1002/cncr.32522

SD-101 program design: key considerations and takeaways

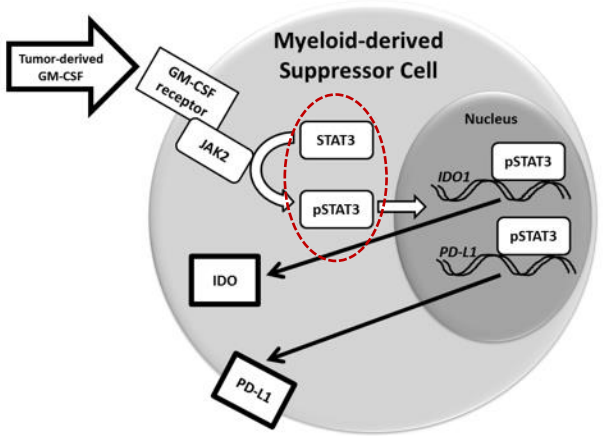
METHODICAL AND DATA-DRIVEN DEVELOPMENT PLAN

Preclinical Foundation	Trial Design	Pharmacokinetics + Pharmacodynamics	Early Clinical POC	Efficacy and Potential Regulatory Endpoints
Predicted SD-101 dose range with PEDD approach[1]	Single agent SD-101 dose-escalation	Liver tissue and serum SD-101 measurements	De-emphasis of ORR given DCR and PFS	PFS based on early disease control rate and possible basis for accelerated approval
Porcine PEDD of SD-101 with TriNav supported dose range	SD-101 re-escalation with single CPI and dual CPI	Liver tumor T cell, MDSC, and Treg levels signals align with pre-clinical data	ctDNA response favorable	OS for full approval supported by ctDNA data which has been reported as predictor of survival in UM[1,2,3]
Defined MoA for MDSC depletion by SD-101	Dose expansion at promising OBD	Systemic immune activation signals	Tolerable safety profile	

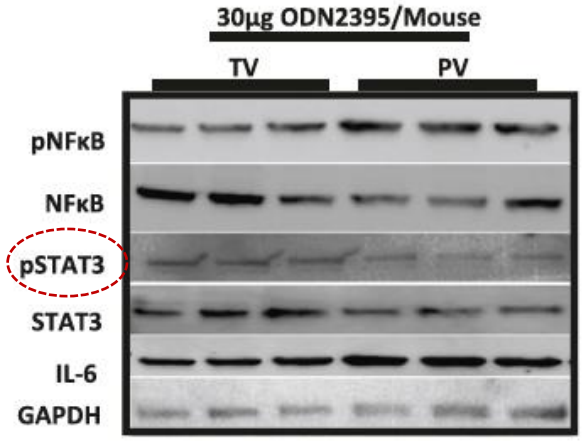
PFS – progression free survival
OS – overall survival
CPI – checkpoint inhibitor

1. Ghosh Cancer Gene Ther 2022
2. Carvajal Nat Med 2022
3. Dawson NEJM 2013

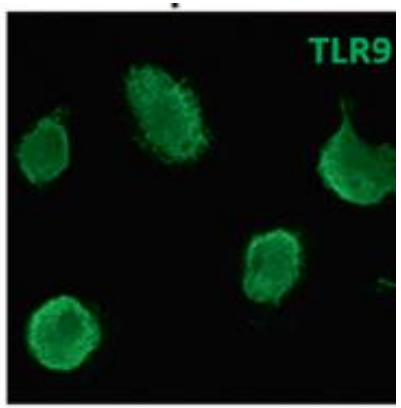
Nelitolimod (SD-101) deactivated key signaling molecule in MDSC to align with immunosuppression biology in liver and pancreas



SD-101 Deactivated STAT3 in Murine Liver MDSC



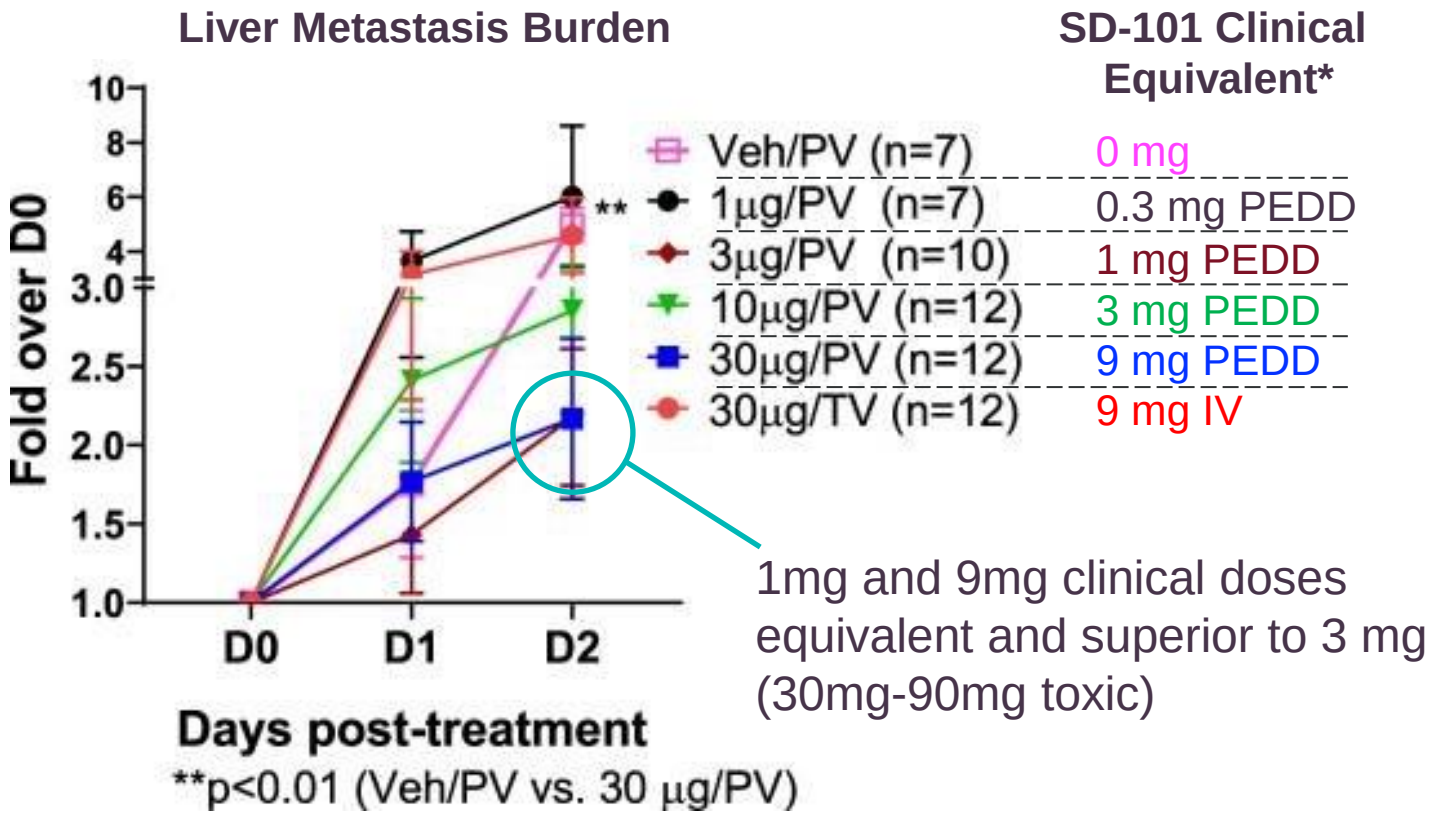
Human MDSC Express TLR9



1. **STAT3 drives liver MDSC expansion, survival, and function**
2. **TLR9 signaling can deactivate STAT3**
3. **MDSC express TLR9**
4. **MDSC depletion confirmed in phase 1 trial**

Ghosh 2023
Thorn 2016

Preclinical PEDD liver metastasis model enabled rational selection of clinical dose range of 2-8 mg and support for use of PEDD



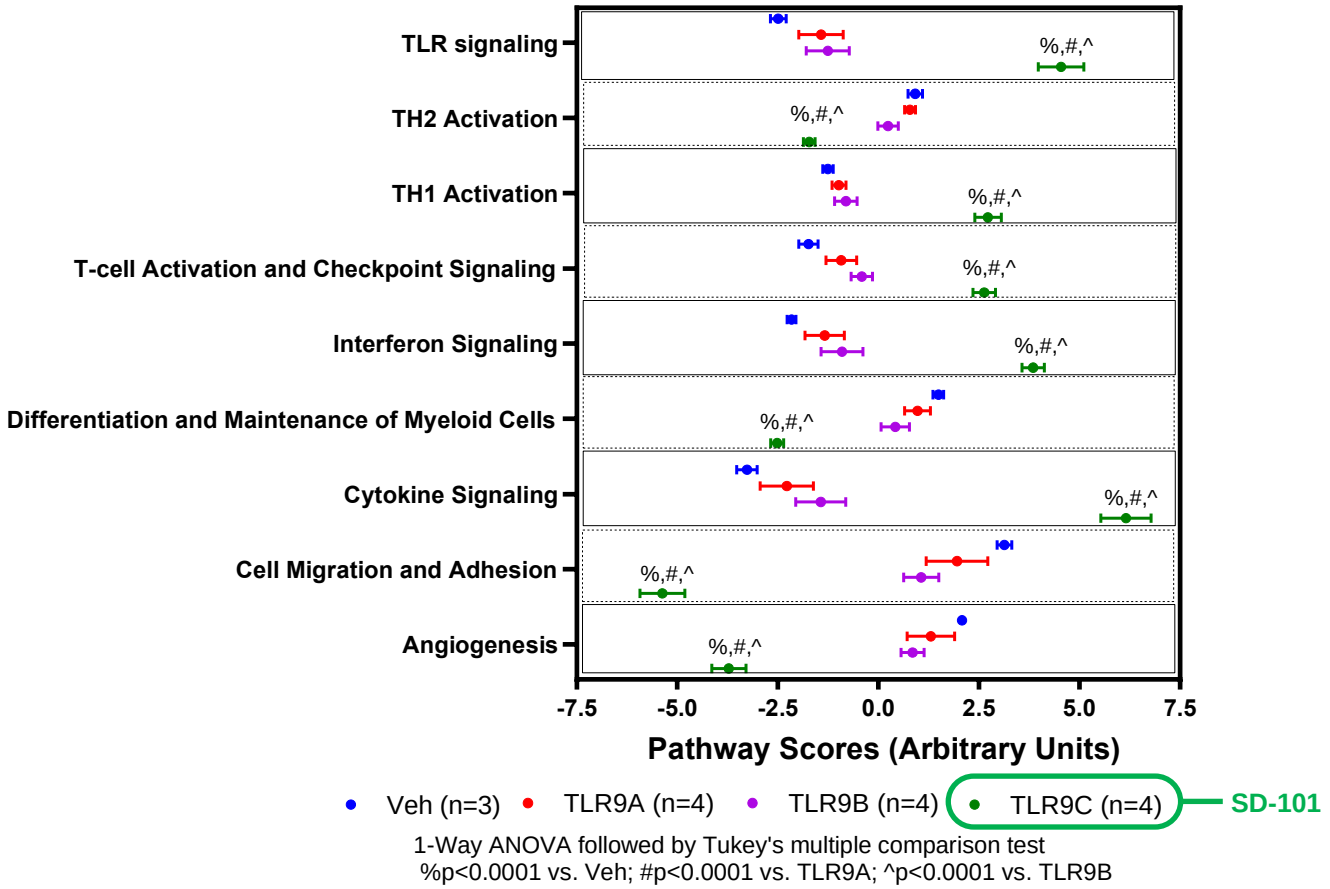
Existence of effective dose **range** expected for TLR9 agonist, with lower doses potentially optimal.

Optimal dose selection to be based on desired immune effects.

1. Ghosh. Cancer Gene Therapy 2023
2. Montazeri. ASCO 2023

Nelitolimod (SD-101) is distinct from other TLR9 agonists in murine liver tumor model PEDD

PEDD ENABLES MOA THAT ALIGNS WITH BIOLOGY IN LIVER AND PANCREAS



SD-101 (class C) Impacts 263 Genes Uniquely vs other TLR9 Classes (<20 for class B or C)

- ↑ Direct TLR9 activation
- ↓ Th2 T cell signal (immunosuppression)
- ↑ Th1 T cell signal (anti-tumor killing T cells)
- ↑ T cell activation
- ↑ Interferon signals
- ↓ MDSC associated genes
- ↑ Cytokine signaling
- ↓ Genes associated with metastases
- ↓ Genes associated with tumor progression

1. TriSalus unpublished
2. Ghosh SITC 2022