MAXIM GROUP

Equity Research
Initiation

Biotechnology

TLSI – NASDAQ	December 7, 2023
Closing Price 12/6/23	**$4.08**
Rating:	Buy
12-Month Target Price:	$12.00
52-Week Range:	$3.32 - $16.24
Market Cap (M):	$107.4
Shares O/S (M):	26.3
Float:	80.0%
Avg. Daily Volume (000):	38.2
Debt (M):	$0.0
Dividend:	$0.00
Dividend Yield:	0.0%
Risk Profile:	Speculative
Fiscal Year End:	December

Total Revenues ('000)

	2023E	2024E	2025E
1Q	2,984A	6,436	10,142
2Q	4,612A	7,355	11,591
3Q	5,193A	7,968	12,556
4Q	5,453	8,887	14,005
CY	18,242	30,645	48,294

Total Expenses ('000)

	2023E	2024E	2025E
1Q	13,105A	15,965	17,839
2Q	16,046A	16,563	18,765
3Q	23,670A	17,119	20,329
4Q	14,600	17,676	21,256
CY	67,421	67,324	78,189

On 8/11/23, TriSalus Life Sciences began trading on NASDAQ under the symbol TLSI following completion of its merger with MedTech Acquisition Corporation.

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TriSalus Life Sciences, Inc.

Buy

Commercial-Stage Oncology Company with a Potential High Value TLR9-Asset – Initiating Coverage with a Buy, $12 PT

Summary

- TriSalus is a commercial-stage oncology company currently marketing the TriNav Infusion System for liver tumors, leveraging the company's proprietary "Pressure-Enabled Drug Delivery" (PEDD) approach.
- TriNav is on track to generate $18M-$20M in revenue in 2023 and reach $30M+ in 2024, likely achieving gross profitability.
- SD-101 is a class C TLR9 being developed for liver and pancreatic tumors, which are difficult to penetrate given the higher pressure environment; but we believe it is a challenge that TriNav may overcome.
- A P1 study is ongoing in uveal melanoma (UM) liver metastases as are P1s in pancreatic cancer and intrahepatic cholangiocarcinoma. The company is also moving forward with hepatocellular carcinoma (HCC) and other areas with metastases to the liver.
- TriSalus went public via SPAC merger in August 2023, and as of end of 3Q23, had $21.4M in cash, which should provide runway into mid-2024. At a ~$100M market cap, we believe TLSI shares are undervalued on TriNav alone. In addition, the longer-term, higher-value play is SD-101, and we see catalysts ahead as more clinical data emerges in 2024.

Details

Proprietary PEDD, targeting liver and pancreas.

- Two FDA-cleared Pressure-Enabled Drug Delivery **(**PEDD) systems; TriNav Infusion System (TriNav) for liver tumors, and a system with pancreatic retrograde venous infusion (PRVI) for pancreatic tumors.
- TriNav is penetrating the transarterial chemoembolization (TACE) and transarterial radio embolization (TARE) market for liver tumor treatment.
- TriNav has distinct advantages with reflux and pressure control over standard methods.
- On track to generate $18M-$20M in 2023, growing to $30M+ in 2024, likely reaching gross profitability.
- Salesforce is expanding, including additional clinical specialists.
- Additional TriNav products to follow on.

High-value, longer-term play is in SD-101 immune therapy, a TLR9 agonist.

- TLR9 agonists as a class have demonstrated clinical proof-of-concept, including SD-101, but have challenges, not including SD-101, mainly from requiring direct needle injection to the tumors.
- Injection is not practical in liver and pancreatic tumors; thus, PEDD-based delivery presents a unique opportunity for TriSalus.
- P1 in uveal melanoma liver metastases for SD-101 + anti-PD1 has demonstrated positive data thus far, most recently at SITC (Society for Immunotherapy of Cancer) in November 2023. Early stage pancreatic cancer P1 data was also presented.
- Also targeting HCC and ICC as well as other indications with liver metastases.

Valuation. We model sales of TriNav in liver tumor procedures with a revenue risk adjustment of 10%-30% based on commercial risk. We model SD-101 for liver tumors in UM, HCC, and colorectal cancer (CRC) in 2028, 2029 and 2030, respectively, as well as pancreatic cancer in 2030 with an 80% revenue risk adjustment based on stage of development and clinical trial risk. A 25% discount is then applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month PT of $12.

CORPORATE PROFILE

TriSalus Life Sciences
6282 W 91st Ave
Westminster, CO 80031
www.trisaluslifesci.com

Investment Risk(s):

- The TriNav Delivery System product is commercially available, but may not reach profitability.
- Development-stage products may not be successful in clinical trials
- Products are entering competitive spaces in immune therapy and drug delivery
- Regulatory and commercial risk
- Need for additional capital could result in dilution risk.

Ownership:

Institutional ownership:	< 2%
Insider ownership:	20%
Short interest:	< 1%

Balance Sheet Summary:
(as of Sept 30, 2023)

Cash:	$21.4M
Debt:	$0M
Shareholders' equity:	$8.5M
Total assets:	$33.6M

Analysts Covering the Co.: 0
(*Excluding Maxim Group LLC*)

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Company Background. TriSalus Life Sciences was created to develop and commercialize an innovative approach for the treatment of liver and pancreatic tumors. The company's platform approach addresses immune dysfunction in liver and pancreatic tumors by combining immunotherapy drugs with highly effective drug delivery technology. TriSalus' heritage is in the development of devices for its Pressure-Enabled Drug Delivery™ (PEDD™) method. Building on this history, the company launched its new platform in 2020 with the acquisition of SD-101, an investigational toll-like receptor 9 (TLR9) agonist with the potential to modulate the immune system to enable immunotherapy. TriSalus currently markets the TriNav Delivery System for use in treating liver tumors with TASE/TARE procedures.

Senior Management:

Mary Szela, Chief Executive Officer—Mary Szela has nearly 35 years of experience driving growth and creating value for patients and investors in both the commercial and clinical pharmaceutical arenas. Ms. Szela has served as CEO and President of TriSalus Life Sciences since January 2018. Prior to joining TriSalus, she served as Chief Executive Officer of Novelion Therapeutics, a rare disease company, where she led the company through regulatory compliance and legal difficulties, ultimately orchestrating a successful merger and expansion of Aegerion Pharmaceuticals and QLT Therapeutics. Prior to Novelion, Ms. Szela was Chief Executive Officer of Melinta Therapeutics, leading the company's revitalization effort and accelerating clinical development of its lead asset for treatment of MRSA (methicillin-resistant Staphylococcus aureus). Previously, she held progressive leadership roles at Abbott Laboratories including that of President of the company's $8 billion U.S. pharmaceutical business. She developed global brands such as Humira® and served as Senior Vice President for global strategic marketing and services. Ms. Szela currently serves as a member of the Board of Directors for Kura Oncology, Prometheus Biosciences, Omega Therapeutics, and Senda Biosciences.

Sean Murphy, Chief Financial Officer—Sean Murphy has served as CFO of TriSalus since June 2022. Mr. Murphy has also been a Director of TriSalus since August 2020 and served as the Chairman of the audit committee, from August 2020 through June 2022. Previously, he served as Executive Vice President at Malin PLC, a publicly listed company investing in life sciences companies, from April 2016 through June 2021. Mr. Murphy was a senior advisor at Evercore, an independent investment banking advisory firm, from August 2011 to June 2018. Prior to that, he held numerous positions over a 30-year career with Abbott Laboratories, a multinational medical devices and healthcare company, culminating as Vice President of Business Development and Licensing. Mr. Murphy has had extensive Board experience as well. He currently serves on the Boards of Immucor, Xenex, and Prenosis. In addition, he previously served on the public company Board of Directors of Radius Health, where he sat on the audit committee, and Poseida, where he was a member of the compensation and governance committee.

Steven C. Katz, M.D., FACS, Chief Medical Officer, SAB Chairman—Dr. Steven Katz has been the Chief Medical Officer at TriSalus since September 2020 and is Chairman of the Scientific Advisory Board, which includes leadership of TriSalus' Translational Immunotherapy Laboratory. Previously, Dr. Katz served as an advisor to TriSalus, from June 2014 to August 2020, and Chief Medical Advisor, from January 2019 to August 2020. Since 2016, Dr. Katz has also served as a consultant for several companies developing cell therapies for solid tumors. In Dr. Katz's academic work, he is an Associate Professor of Surgery at Brown University and has been with Brown Surgical Associates in a part-time role since February 2022. From 2009–2021, Dr. Katz led the creation of a solid tumor immunotherapy program at CharterCare Health Partners, where he served as the Director of the Office of Therapeutic Development and Complex Surgical Oncology Program Director.

Additional members from TriSalus Life Sciences can be found HERE.

INVESTMENT SUMMARY

SPAC Merger. In August 2023, TriSalus completed its merger with MedTech Acquisition Corporation and began trading under the symbol TLSI.

TriSalus is an oncology company with a disruptive drug delivery technology; Pressure-Enabled Drug Delivery (PEDD). Liver and pancreatic cancers, including both primary tumors and secondary tumors, have very high rates of mortality. While immune-based therapies such as checkpoint inhibitors and CAR-T have revolutionized cancer management, tumors of the liver and pancreas are less likely to respond. This is due in part to intratumoral pressure, which is characterized by constricted and/or collapsed blood vessels, that limits drug uptake. This also limits uptake of conventional, standard-of-care options like chemotherapy and radiation, even with TACE/TARE procedures. In addition, from an immune therapy perspective, tumors of the liver and pancreas also have immune suppressive tumor microenvironments. TriSalus addresses both of these issues. The company has two FDA-cleared devices to enhance drug delivery to liver and pancreatic tumors; TriNav (liver) and the TriSalus Infusion System with pancreatic retrograde venous infusion (PRVI; pancreas). The company is leveraging its PEDD platform for the development of a clinical-stage TLR9 agonist, SD-101, which it acquired from Dynavax Corporation (DNAX—NR) in 2020. Combined, TriNav, as well as the pancreatic system, address both biological (immunosuppressive environment of the tumor) and mechanical (high pressure, collapsed vessels etc.) barriers associated with treatment.

TriNav Infusion System, overcoming challenges in TACE/TARE treatment. For inoperable tumors of the liver such as hepatocellular carcinoma (HCC) or metastases from other cancers, catheter-based local treatments transarterial chemoembolization (TACE) and transarterial radioembolization (TARE) are commonly used. However, TACE and TARE have multiple challenges including intratumoral pressure and blood flow issues (collapsed/constricted, leaky vessels) that TriNav solves to improve delivery of chemotherapy or radiotherapy, as well as other therapies including SD-101 and CAR-T (prior PEDD work successfully delivered CAR-T to the liver, with positive outcome). TriNav has a one-way microvalve, or “SmartValve”, that overcomes intratumoral pressure, is able to control that pressure, and has reflux control that combines to deliver therapeutics deeper into the tumor tissue while also avoiding damage to healthy tissue and/or systemic toxicities. In clinical studies, TriNav was shown to improve TACE delivery by 60% and TARE by 33%–90%. The system also has successfully delivered high concentrations with low systemic exposure of the company’s immunotherapy candidate SD-101. TriNav, as well as the pancreatic device, were both FDA-cleared, with TriNav currently being actively marketed. The company is also developing large vessel TriNav (FDA-cleared; launch expected in 1H24), small vessel TriNav, a next-gen pancreatic infusion system, and a next-gen TriNav with integrated sensing.

TriSalus is already a revenue generating company with TriNav and is on the path to gross profitability in 2024. TriNav commercialization was launched in 2020. In the U.S. there are 41,000 cases annually of liver cancer and nearly 100,000 cases of liver metastases. Of these, ~40% are eligible for TACE/TARE procedures, which use a standard catheter-based approach and 25%–30% may be appropriate candidates for TriNav. This translates to ~37,000 TriNav units, and at a price of $7,750 per unit, represents a ~$300M market opportunity. TriNav generated $8.4M and $12.4M in revenue in 2021 and 2022, respectively, and is on track to generate $18M–$20M in 2023. In 3Q23, which was reported on 11/14/23, TriSalus reported TriNav revenue of $5.2M, up 32% y/y, and gross margin expansion to 89%, from 82%, given increased factory production and operational efficiencies. The company is expanding its reps to 30+ in 2024, including the addition of 7–10 clinical specialists, which should help accelerate revenue growth to ~$30M–$35M in 2024 and upwards of $100M over the next 4–5 years. The revenue generated by TriNav also substantially reduces costs associated with R&D, including new PEDD devices/options and for its lead therapeutic candidate SD-101, which is currently the subject of several early-stage clinical programs. The addition of the large vessel TriNav in 2024 to the portfolio, as well as additional options after that, should help accelerate revenue growth further.

SD-101 with PEDD is potentially a high-value play. Tumors of the liver and pancreas remain areas of significant unmet need, in large part due to an inability to deliver therapeutics adequately. As such, with two FDA-cleared delivery platforms in TriNav and PRVI, TriSalus is uniquely positioned to develop its immune therapy candidate SD-101 for these indications, and potentially other therapeutic candidates. Liver and pancreatic cancer treatments have high costs despite continued high rates of mortality. In liver cancer, 1L treatment now includes the combination of Tecentriq (anti-PD1) and Avastin (anti-VEGF), which can cost $200K+. Treatment with Lenvima, the leading tyrosine kinase inhibitor (TKI) can cost $150K+ annually. There are no approved 2L therapies, and 2L+ includes liver metastases from other cancers. In pancreatic cancer, depending on stage, monthly costs associated for chemotherapy alone can range from $8K to $15K per month. For SD-101, given the unmet need in these indications and the opportunity for checkpoints to have success in the SD-101 setting to extend survival, it’s reasonable to assume pricing of SD-101 could be in the $200K+ range. In addition, the use of systemic checkpoint therapy and dosing requirements point to the need to deliver SD-101 multiple times and this likely requires 5+ delivery devices per patient at a cost of $7,750 each that would be independent of the SD-101 cost. Combined, this points to a potential multi-billion-dollar market opportunity, the largest of which would be in the HCC space.

TLR9 agonists have established clinical proof-of-concept, which we view as derisking. The TLR9 agonist space, including with SD-101 while it was with Dynavax, has established clinical proof-of-concept in other indications, using a direct needle injection into the tumors and combining that with checkpoint therapies. However, this does not include the liver or pancreas given the challenges and risks associated with trying to inject tumors in these organs, setting up an opportunity for TriSalus with its PEDD options. TriSalus has demonstrated SD-101’s unique dual mechanism as a class C TLR9 agonist (as opposed to class A and/or B where others in the space have focused) of action to reduce immune suppressive myeloid-derived suppressor cells (MDSCs) and drive infiltration of T cells, to create a more favorable tumor microenvironment (turning “cold” tumors “hot”) for checkpoint therapies. This was also demonstrated in the clinic, most recently at the SITC (Society of Immune Therapy for Cancer) meeting in November, in uveal melanoma liver metastases (UMLM, PERIO-01 study). SD-101 PEDD alone, or in combination with systemic anti-PD1 therapy, induced MDSC and Treg cell reductions, a shift from M2 to M1 macrophages, increases

in inflammatory cytokines in the tumors and in the periphery, and trafficked T cells and NK cells to the tumor. In the combination groups, there was an overall disease control rate (DCR) of 58%, and in the identified ideal SD-101 dose of 2mg, when combined with anti-PD1, the results were even more pronounced, including a DCR of 81%. The trial update was for 56 enrolled patients and the company is moving to enroll more in the 2mg group. Additionally, there were positive signals of immune changes in the pancreatic trial (PERIO-03), though it was for three patients and its early in the SD-101 monotherapy dose escalation phase. More data is expected from these programs in 2024. The company is also active in hepatocellular carcinoma (HCC) and intra-hepatic cholangiocarcinoma (ICC) and may expand to liver metastases arising from colorectal cancer as well as other indications.

Bottom line—TriSalus is an emerging oncology company active in both drug delivery via its PEDD platforms and drug development with its class C TLR9 agonist SD-101. The company is already generating revenue with TriNav at a ~50% CAGR that could substantially accelerate in 2024 with the expansion of its reps and clinical specialist base, as well as new products. The benefits of having a commercial product are multi-fold and interconnected. The two FDA-cleared devices, TriNav and PRVT, have been demonstrated to be sufficient in delivering therapeutics to both the liver and pancreas, which is beneficial to the development of SD-101 and creates a unique opportunity for TriSalus, in our view. Revenue growth, which is expected to reach over $30M in 2024 and possibly $100M+ over the next 4–5 years, substantially reduces costs associated with R&D, including for new/expanded PEDD devices and the development of SD-101. This may reduce or limit the need for other sources of capital. There are catalysts ahead on both the commercial and development sides of the company. The SD-101 clinical programs in UMLM and pancreatic cancer are ongoing, and more data is expected in 2024. Updates on TriNav commercial efforts and revenue growth are expected, as are updates on new PEDD devices, including the launch of FDA-cleared large vessel TriNav in 2024. In 2023, TriNav is expected to generate ~$18M–$20M in revenue and grow to $30M+ next year. Considering the current market capitalization of ~$100M, we believe TLSI shares are undervalued from the TriNav commercial aspect alone. In addition, we see the high-value, longer-term play on the therapeutics/device combo side of the company with SD-101/PEDD, which is not priced into shares.

TriSalus Life Sciences, Finances.

- 8/10/23: TriSalus Life Sciences completed its merger with MedTech Acquisition Corporation. TriSalus' common stock and warrants began trading on NASDAQ Global Market under the symbols TLSI and TLSIW, respectively, on 8/11/23.

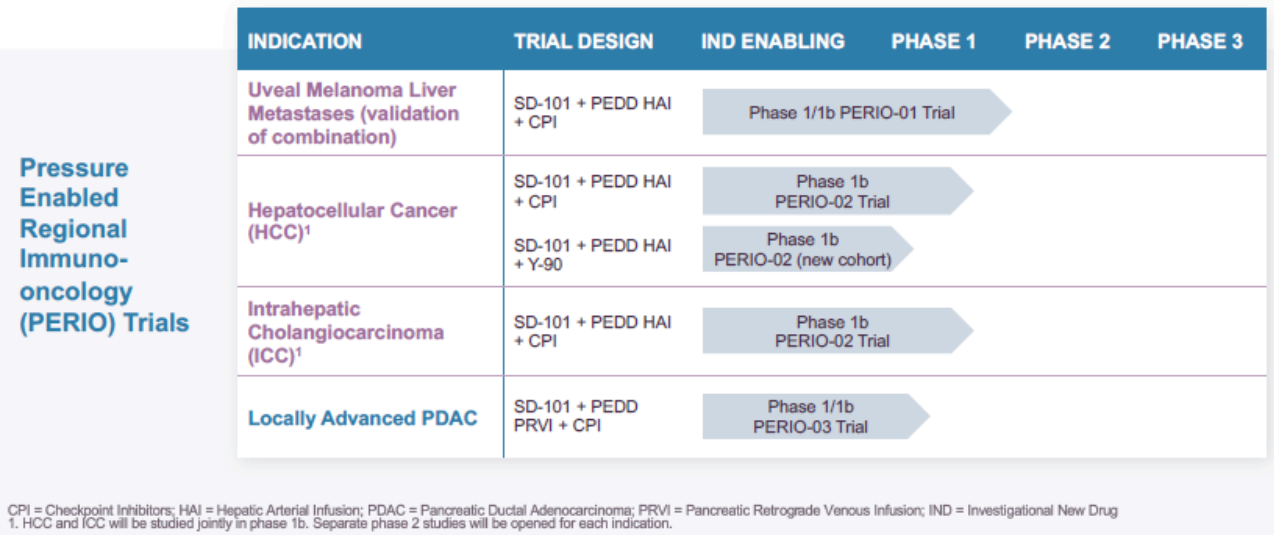
- 11/13/23, TriSalus reported 3Q23 with a TriNav revenue of $5.2M, an increase of 32% y/y. Gross margin expanded to 89%, from 82%, due to increased factory volumes and improved operations efficiency. Operating expenses came in at $23.7M, including a one-time non-recurring expense of $4.8M related to the costs of completion of the de-SPAC process in August. R&D expenses increased to $9.4M, from $6.9M, as clinical programs advanced. A non-cash loss of ($2.8M) was related to change in fair value of warrant liabilities and there was a non-cash gain of $19.9M related to fair value of contingent earnout liability. The company ended the period with a net loss of ($1.7M) and $21.4M in cash on the balance sheet, which should fund operations into mid-2024. The runway could be extended based on trajectory of TriNav revenue, as well as non-dilutive options including partnerships and/or debt. The company does have access to a $30M stock purchase agreement as well. We factor in capital raises and associated dilution in our model as TriSalus is not profitable. We do expect, and model, gross profitability for TriNav in 2024, though this is offset by R&D costs associated with clinical development of SD-101. The company expects to have a quarterly cash burn of ~$7M–$10M in 2024.

Exhibit 1. Upcoming Catalysts for TriSalus Life Sciences, 2023–2024.

Catalyst	Indication	Anticipated Timing
Phase 1 PERIO Data	Uveal Melanoma	2H 2023
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	1H 2024
Initiation of Phase 1b Y-90 + SD-101	Hepatocellular Cancer	1H 2024
Phase 1 PERIO Data	Locally Advanced Pancreatic Cancer	2H 2024
Phase 1b with IV checkpoint	Locally Advanced Pancreatic Cancer	1H 2025

Source. TriSalus Life Sciences presentation.

Exhibit 2. TriSalus Life Sciences Pipeline (TriNav Delivery System is commercially available, but not shown)



Source. TriSalus Life Sciences presentation.

TriNav Delivery System

TriSalus's proprietary oncology delivery system is Pressure-Enabled Drug Delivery, or PEDD, which modulates pressure and flow within blood vessels to improve intravascular delivery of cancer drugs. The PEDD platform currently has two FDA-cleared devices; TriNav for liver tumors and a pancreatic retrograde venous infusion (PRVI). TriNav is currently being commercialized for use for the treatment of primary liver tumors and liver tumors arising from metastases (mets). The PRVI device is not expected to be commercialized until 2025. TriSalus' PEDD devices are also being used to deliver the company's immune therapy candidate SD-101, a class C toll-like receptor 9 (TLR9) agonist, which is in clinical development for liver and pancreatic tumors. The company is evaluating its PEDD system delivery of SD-101 in liver mets arising from uveal melanoma (UM), intrahepatic cholangiocarcinoma (ICC), as well as hepatocellular carcinoma (HCC) and locally advanced pancreatic cancer (PDAC) with potential expansion to other liver tumor mets. The trial designations are PERIO, or pressure-enabled regional immuno-oncology. The clinical programs are evaluating SD-101 with a checkpoint inhibitor (CPI), though the program in HCC is in combination with radiation therapy using Y-90 (isotope of yttrium) (see pipeline for trials and timelines on next events. The UM liver met indication is small from an incidence perspective, but will be used for validation of the SD-101 + CPI combination. P1 data is expected before YE23 with a P2 trial expected in 1H24 along with starting the P1b in HCC for SD-101 + CPI, another P1b in HCC with SD-101 + Y90, and then P1 in PDAC in 2H24 for SD-101 + CPI. There is a P1b in ICC that is awaiting P1 data from which next steps may be determined but we expect the primary focus to be in UM liver mets and HCC, followed by PDAC. There could also be movement towards liver mets arising from colorectal cancer (CRC).

Combined, there are two key aspects to TriSalus, the PEDD system itself, including ongoing commercialization of TriNav Delivery System that continues to build traction. The company expects ~$20M in revenue expected for 2023 and $30M–35M in 2024 as new reps and clinical specialists are added to the team. Growth is expected to continue and potentially reach as much as ~$100M in revenue over the next five years. The second key aspect to the story and the potential long-term high-value proposition, is the development of SD-101 delivered with PEDD. We'll start with PEDD and the commercial opportunity for TriNav Delivery System.

Why PEDD? For inoperable tumors of the liver such as that in hepatocellular carcinoma (HCC) or metastases in the liver, two commonly used loco-regional therapies are transarterial chemoembolization (TACE) and transarterial radioembolization (TARE), which are aimed at slowing tumor growth and potentially bridging patients to other therapies[1]. A TACE or TARE procedure involves putting a patient under general anesthetic and the physician inserting a catheter into the femoral artery via the groin. A dye is injected and via angiogram, the catheter is guided to the artery branches feeding the tumor in the liver. There are two types of TACE. One is conventional TACE, or cTACE, where gelatin sponge particles are injected to cut off tumor blood flow. The other type uses drug-eluting beads to deliver chemotherapy[2]. TARE also uses a transcatheter intra-arterial approach, but delivers microspheres with the radioisotope yytrium-90, or Y-90. TACE has been the standard-of-care

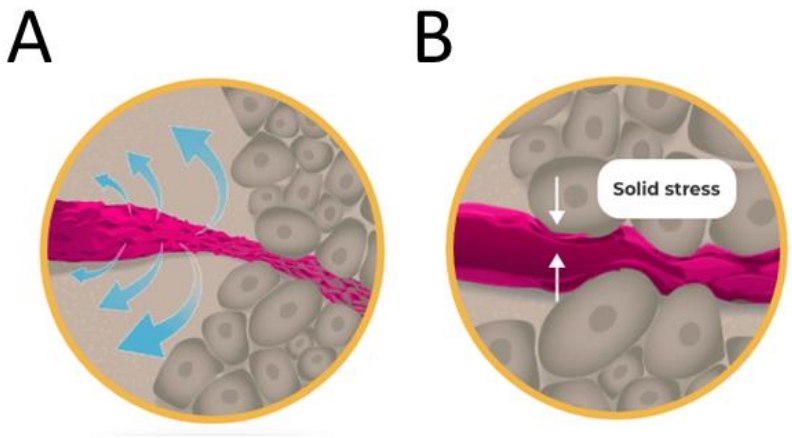
[1] Brown AM et al., Canc Med. 2022; 12:2590-2599
[2] ncbi.nlm.nih.gov

for unresectable intermediate-stage liver tumors, though there is increasing data that supports TARE. TARE has been shown to have a longer time-to-progression and a better toxicity profile, and is used more often in patients with more advanced disease, including patients with portal vein thrombosis where TACE is contraindicated[3].

While commonly used, both TACE and TARE have multiple challenges that TriNav solves. In particular tumors in the liver and the pancreas, which are the targets of TriNav (commercial) and PRVI (FDA-cleared), respectively, are high-pressure environments, or high-pressure tumors. The pressure in the tumor can be higher than the patient's blood pressure, with constricted or collapsed blood vessels, which can significantly limit drug uptake. There are several aspects around the tumor vasculature for this to consider. Elevated interstitial fluid pressure, or IFP, limits the ability of drugs to leave blood vessels and deeply penetrate the tumor. There are also leaky blood vessels that cause fluid to seep into the interstitial space with no way out and the lymphatic system within tumors is often underdeveloped and cannot drain fluids away. Then there is actual physical collapse of blood vessels from lots of extracellular matrix fibrous material that creates solid stress, where blood flow may be reduced or halted to many parts of the tumor.

The limited therapeutics delivered includes chemotherapy drugs, radiotherapy like Y-90, and essentially anything else including immune-based therapies like the CPIs. CPIs have not had significant success in terms of response rates in liver and pancreatic tumors, though there are some CPIs approved, including Tecentriq (anti-PD-LI) + Avastin in 1L HCC, both Optivo (anti-PD1) and Keytruda (anti-PD1) are used in 2L HCC, as well as Keytruda in pancreatic cancer (w/certain mutations). In addition, the tumor microenvironment (TME) in the liver and pancreas is immune suppressive with presence of high amounts of myeloid-derived suppressor cells (MDSCs), which limits immune-based therapeutics if they can reach the tumor(s). The TME and immune status may be addressed by TriSalus' SD-101, which seems to knock down MDSCs and make a more hospitable environment for CPIs. This is discussed below. Back to TACE and TARE.

Exhibit 3. High intratumoral pressure limits drug uptake. (A) Interstitial fluid pressure, leaky vessels, and poor lymphatic drainage impact therapy uptake in tumors. **(B).** Solid stress, largely from fibrous extracellular matrix in the tumors, can squeeze and collapse tumoral blood vessels, limiting therapeutic delivery.



Source: TriSalus Life Sciences

TACE and TARE need to use the vascular biology of the liver tumor, which derives its blood supply from the hepatic artery. This delivers drug and drives both oxygen and nutrient depletion to induce necrosis. However, a limited number of lesions demonstrate complete or extensive necrosis, often leading to the presence of viable tumor cells adjacent to the necrotic lesions. Additionally, there are issues with chemotherapy impacting healthy tissue. There are similar challenges with TARE. Overall, the goal is to deliver more drug or radiotherapy accurately to treat the tumors while sparing normal liver and extrahepatic tissues. TriNav (and PRVI) solves this issue.

TriNav is a flexible microcatheter that can be used to deliver diagnostic and therapeutic agents into the vasculature, with its main uses being for TACE or TARE for liver tumors. TriNav has a one-way microvalve, or "SmartValve", capable of generating infusion pressure greater than mean arterial pressure to help overcome intratumoral pressure and improve delivery of therapeutics. The SmartValve is designed to provide reflux protection and to maintain centroluminal position during infusion. The increase in pressure, and control of that pressure, is what allows TriNav to deliver therapeutics deeper into solid tumors. The device itself is made of ultra-thin nitinol fibers laid out in a precise braid geometry, that is then overlaid with nanofilaments made of composite polymers. This creates a filter valve that allows particles >10µm (e.g., red blood cells) to pass

[3] Kallini JR et al., Adv Ther. 2016; 33(5):699-714

through. The exact geometry of the braid and composition of the polymers have been calibrated to create a soft, pliable valve that can react dynamically to varying pressure and flow conditions in vasculatures, yet strong enough to prevent reflux of material and generate sufficient pressure without imposing too much radial force on the vessel walls.

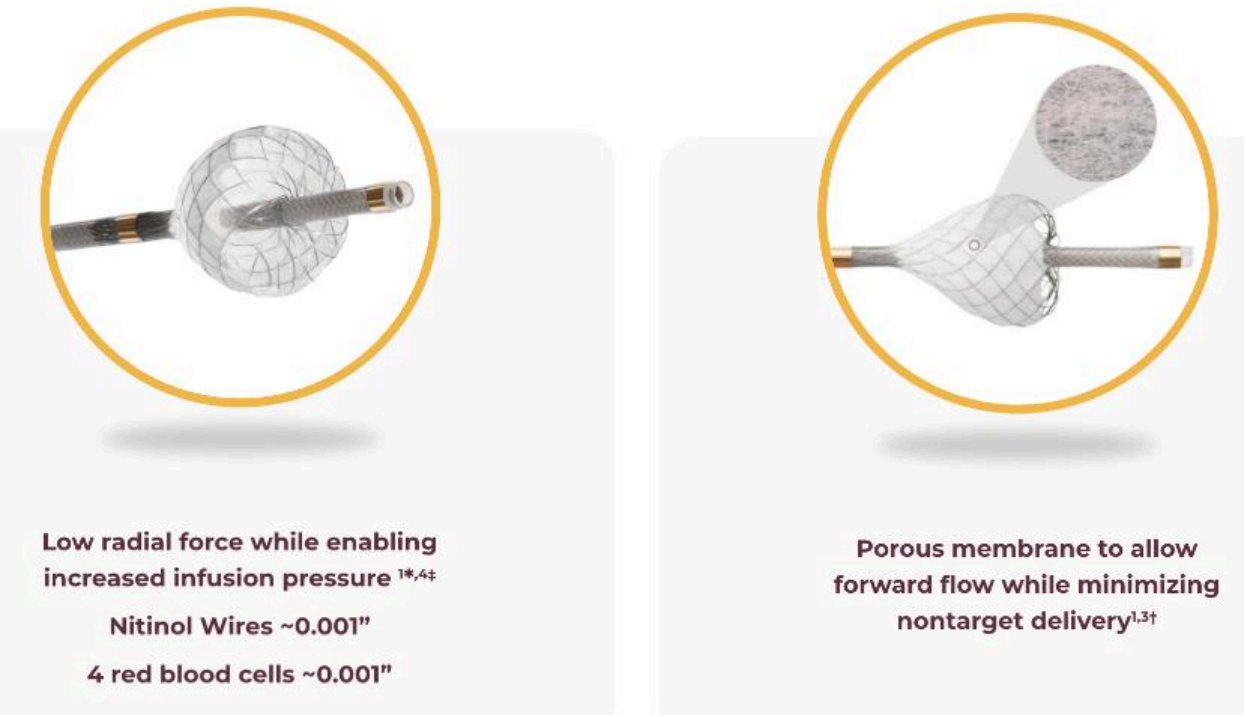
The catheter shaft is made of composite polymer (Pebax) segments of varying softness and reinforced with stainless-steel braid. The design and material of the shaft have been optimized to provide strength, kink resistance, ease of tracking, and flexibility — all of which are important to enable navigation of the catheter over microwires in tortuous vasculature. At the distal end of the catheter, there are two radiopaque marker bands to help physicians locate the distal end of the catheter as it is being threaded through the vasculature. The inner lumen of the catheter shaft is lined with polytetrafluoroethylene, a highly inert and lubricious polymer, to minimize friction and maximize compatibility with microwires, chemotherapy, cell therapy products, and other agents used during the procedure. Finally, the device is coated with a hydrophilic formulation that is thin yet durable, making it even more trackable and capable of accessing the most tortuous vasculature.

Exhibit 4. TriNav dimensions, compatible with standard base catheters.

Specifications	LENGTH 120 cm	LENGTH 150 cm
Product Code	TNV-21120-35	TNV-21150-35
Inner Diameter	0.021"	0.021"
Vessel Size	1.5-3.5 mm	1.5-3.5 mm
Base Catheter Compatibility	0.035"	0.035"
Proximal Outer Diameter	2.4 F	2.4 F
Dead Space	0.37 mL	0.44 mL
Maximum Infusion Pressure	1200 psi	1200 psi
Bead Size Compatibility	Hydrogel ≤500 µm Glass ≤110 µm Resin ≤60 µm	Hydrogel ≤500 µm Glass ≤110 µm Resin ≤60 µm

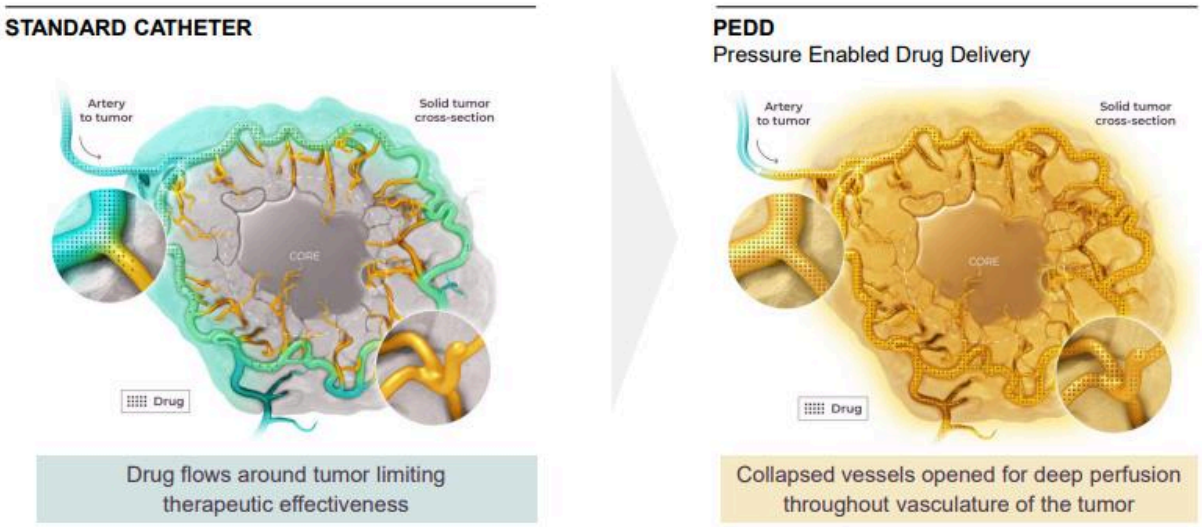
Source. TriSalus Life Sciences

Exhibit 5. TriNav Device and SmartValve. The TriNav device overcomes the limitations of traditional end-hole and balloon catheters. The device is commercially available and most commonly used for TACE or TARE procedures in liver tumors. TriNav allows for forward blood flow and can react dynamically to varying pressure and flow conditions, in sync with the cardiac cycle. TriNav also conforms to blood vessels due to its soft, flexible valve that has an expanding, self-conforming tip for therapeutic delivery and reflux protection. Lastly, the porous mesh allows red blood cells and particles >10µm to pass through, while being strong enough to minimize embolic reflux.



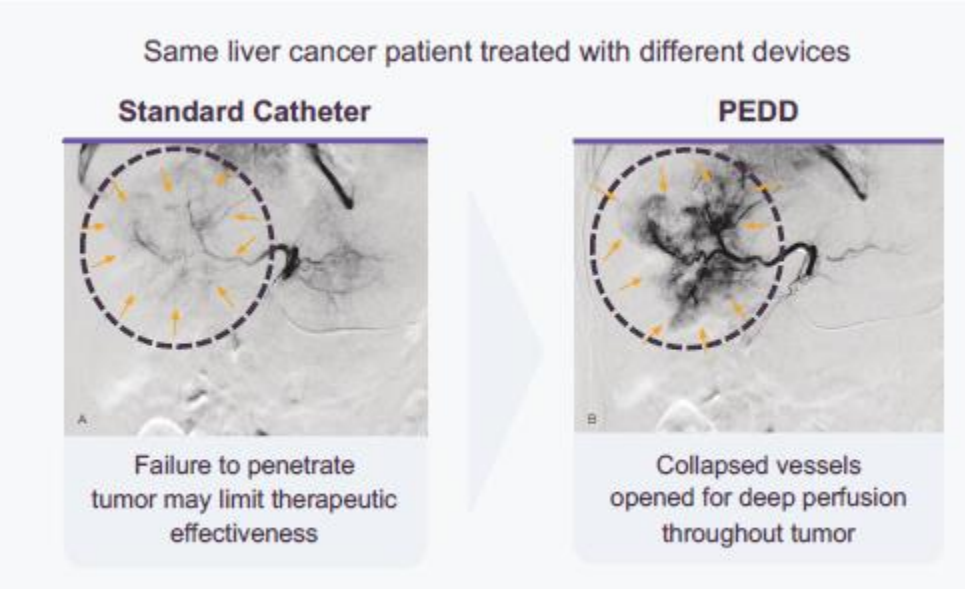
Source: TriSalus Life Sciences

Exhibit 6. PEDD vs. standard catheter.



Source: TriSalus Life Sciences

Exhibit 7. PEDD drives more therapeutic penetration into the tumors. Shown below is interventional radiology of PEDD vs. standard catheter in the liver of the same patient. What can be seen in the PEDD image on the right is the increased delivery of dye contrast into the liver tumor, opening of collapsed blood vessels and decreased reflux of the contrast dye into the normal liver.



Source: TriSalus Life Sciences

Exhibit 8. There is a substantial data supporting the use of PEDD with SmartValve to improve tumor targeting in the liver.

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 (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

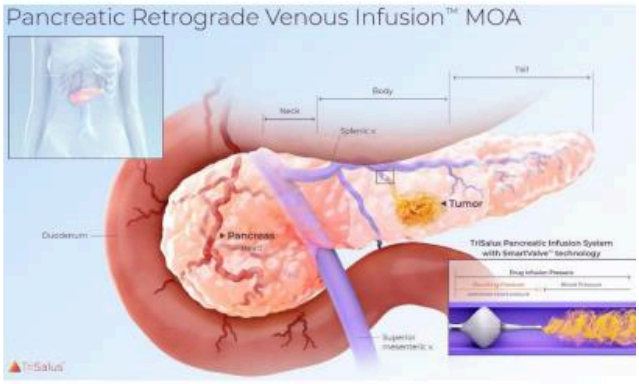
TACE = Transarterial chemoembolization, TARE = Transarterial radioembolization

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

Source: TriSalus Life Sciences

Exhibit 9. FDA-cleared TriSalus infusion system with pancreatic retrograde venous infusion (PRVI). This device has 510(k) clearance as noted above, but is not yet commercially available. It's being developed with immune therapy candidate SD-101 for treatment of locally advanced pancreatic cancer.

- Poor blood flow limits drug access to the pancreas[1-3]
- Pancreatic arteries difficult to access[4,5]
- Innovative retrograde venous approach eliminates need for balloons[6,7]
- Pressure measurement included for safety and efficacy
- Leveraging PEDD and SD-101 experience from liver trials
- Phase 1 locally advanced pancreas data from MDACC to be presented at SITC 2023

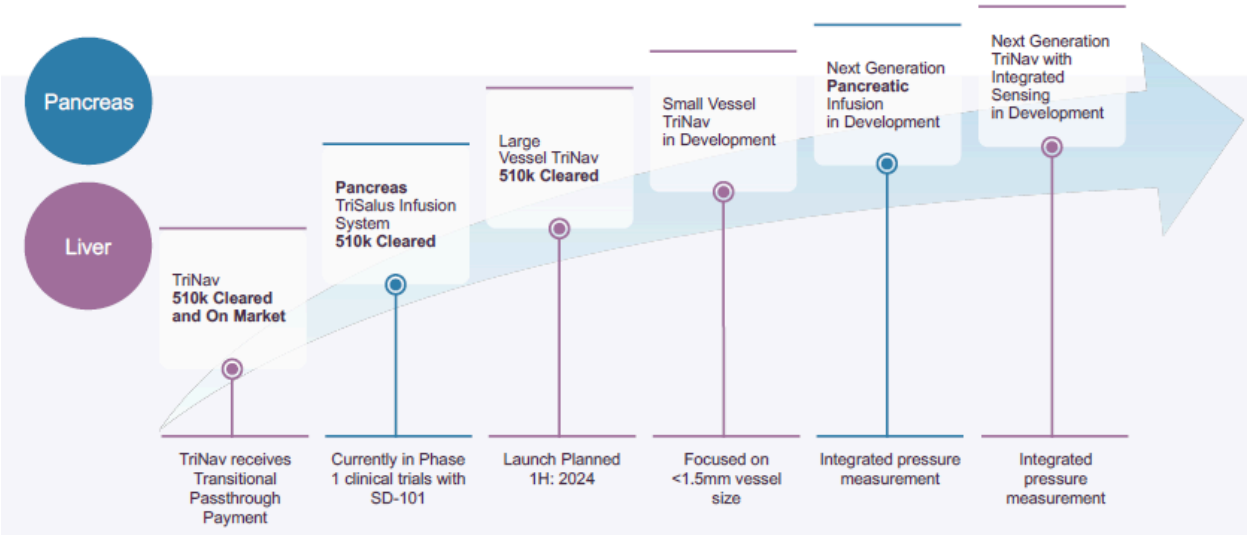


1. Rakesh Jain (2013) Normalizing Tumor Microenvironment to Treat Cancer: Bench to Bedside in Biomarkers. 31:17 2205-2218
2. DuFort et al, Interstitial Pressure in Pancreatic Ductal Adenocarcinoma Is Dominated by a Gel-Fluid Phase. Biophysical Journal 110 2106-2119
3. Soltani et al Numerical Modeling 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).

Source: TriSalus Life Sciences

Reimbursement. Both the TriNav Infusion System and the Pancreatic Retrograde Venous Infusion (PRVI) device have 510(K) clearances from the FDA, though currently only TriNav is commercialized. TriSalus also received 510(K) clearance for large vessel TriNav with commercial launch of this product planned for 1H24. Commercialization of PRVI is not anticipated before 2025. The primary purchasers of TriNav (and future devices/systems) are hospitals. Both TACE and TARE procedures have coding, coverage and payment in all settings of care. TriNav uses CPT codes 37242 and 37243, for "vascular embolization or occlusion, inclusive of all radiological supervision and interpretation, intraprocedural road mapping, and imaging guidance necessary to complete the intervention; for tumors, organ ischemia, or infarction. Of note, TriSalus received approval for TPT (transitional pass-through status) payments for TriNav in November 2019, which took effect on January 1, 2020. TPT status was extended to the end of 2023 and the company is engaging with the Centers for Medicare and Medicaid Services (CMS) to obtain permanent reimbursement for TriNav at similar rates starting on January 1, 2024. Currently TriSalus catheters are ~$7,750 vs. a standard catheter at ~$800–$1,200. The company has also filed a Category III Code with the AMA (American Medical Association), which routinely creates codes for emerging technology, services, and procedures. Our expectation is that the company should be able to maintain its pricing at $7,750 per TriNav device. The company also has 510(k) clearance for the large vessel TriNav and is developing a small vessel TriNav, as well as next-generation pancreatic infusion and next-generation TriNav with integrated sensing.

Exhibit 10. TriSalus Technology Pipeline.



Source: TriSalus Life Sciences

Commercialization of TriNav. TriNav was FDA-cleared and launched in 2020. The company's sales and marketing team, as of July 2023, had ~35 people on it, including 24 reps. The company expects to have 30+ reps in 2024, as well as the addition of 7–10 clinical specialists. There are currently around 34,000 liver TACE/TARE procedures per year that take place in 800 centers/hospitals, as well as 7,000 procedures at other locations. There are ~800 centers/hospitals that perform these procedures, though ~400 do 70% of those procedures. TriSalus believes that the 400 top higher-volume centers can be accessed with 40+ people and should be aided by the addition of clinical specialists. Currently, as of this report, TriSalus is in 170 centers. The current cost for a TriNav device is $7,750. Revenue for 2023 is expected to be in the $18M–$20M range with 50%–70% growth in 2024 to ~$30M–$35M, growing at a 50% CAGR from there. Margins are in the low-mid-80% range and could grow to 90% or higher over time. Margins as of the 3Q23 quarter report were 89% based on increased manufacturing output and efficiencies. Gross profitability for the TriNav business, excluding opex and the associated cost for development programs (PEDD and SD-101), could be reached in 2024. Our expectation is that TriNav, combined with the addition of large vessel TriNav in 1H24, followed by additional devices can drive revenues to $100M+ within five years. This should substantially reduce R&D costs associated with development of other devices and the high costs associated with oncology drug development.

PEDD + SD-101, clinical development

The PEDD approach for drug delivery is an attractive approach for drug development in "high pressure" tumors where vascular blood flow is severely limited, particularly tumors of the liver and pancreas. Additionally, tumors in these organs are notoriously difficult for immune-based therapies like checkpoint inhibition given their immune suppressive tumor microenvironments (TMEs) including the presence of high amounts of myeloid-derived suppressor cells (MDSCs). TriSalus set out to identify a drug candidate that can be delivered via its PEDD system(s) and drive immune modulation to create an environment more favorable for checkpoint inhibition. As such, TriSalus' SD-101 asset, which is a class C TLR9 (toll-like receptor 9) agonist, was acquired from Dynavax Technologies Corporation (DVAX—NR) in July 2020. TLRs are known to have broad TME modulating effects with induction of immunity at distal sites. There are key points of differentiation for TriSalus's molecule vs. comps in the space, including SD-101 as a class C vs class A TLR9 agonists, target organ/tumor, and method of delivery using PEDD vs. others using direct needle tumor injection. TriSalus is evaluating, or planning to evaluate, SD-101 in combination with a checkpoint inhibitor (CPI) in uveal melanoma (UM) liver mets, hepatocellular carcinoma (HCC, also with Y90 delivery), intrahepatic cholangiocarcinoma (ICC), colorectal cancer (CRC), liver mets, and pancreatic cancer. Let's start with some basic background on these indications and then move into SD-101 and the TLR9s. Overall, the challenges in these indications are issues with the ability to deliver drug therapies and deeply immune-suppressive environments, both of which may be addressed with TriSalus' PEDD systems (TriNav for liver, PRVI for pancreas) delivery of loco-regional SD-101 (followed by systemic checkpoint therapy).

Uveal Melanoma (UM). Melanoma is a relatively rare tumor arising from melanocytes in the skin, mucosal membranes, and ocular sites. The rarest manifestation, with only ~3,000 cases annually in the United States, is uveal melanoma (UM). UM is the most common primary intraocular tumor in adults. Although survival is high with a five-year survival rate at >80%, higher-risk UM patients often develop metastatic disease of which 90% occurs in the liver. In this setting mean survival time ranges on average from 2 to 6 months; survival is at the higher end of this range if tumors are treated. As opposed to metastatic cutaneous melanoma, which has multiple therapeutic options B-Raf and MEK inhibitors, as well as checkpoint inhibitors (CPIs) that are highly effective, metastatic UM has no effective standard treatment. There are multiple factors that contribute to this difference, such as a lower mutational load in UM, which for example, could lead to less frequent generation of neoantigens that make tumors more immunogenic and susceptible to immune-based therapies. For metastatic UM, which as noted above, primarily affects the liver, it's even more challenging, particularly given the presence of MDSCs driving an immune suppressive TME.

Hepatocellular carcinoma (HCC), intrahepatic cholangiocarcinoma (ICC)—primary liver cancer. In the United States, in 2022, it is estimated that over ~43,000 cases of primary liver cancer will be diagnosed. Of these, over 30,000 deaths are estimated, with live cancer representing the fifth most common cause of cancer death. Globally an estimated 841,000 cases of liver cancer are expected to result in over 780,000 deaths. Outcomes in HCC are poor, with a five-year survival net of ~20%, and for those with advanced disease <3%. Prognosis is determined by several factors, including alpha-fetoprotein (AFP) concentration, tumor extension, histology, liver dysfunction degree, and patient performance status. HCC prognosis is correlated to delay in diagnosis. HCC makes up 90% of primary liver cancer, while ICC is the second-most common liver cancer at ~10%. From a treatment perspective, surgery, if possible, is an option. Standard-of-care (SOC) therapies have mainly been for use of tyrosine kinase inhibitors (TKIs) and targeted biologics, including the combination of atezolizumab + bevacizumab, which has been approved for 1L liver cancer. This combo is also being used in some 2L and pembrolizumab is approved in 2L as well. Chemotherapies have not had success given that they are administered systemically, and, as such, have severe systemic toxicity and the primary filter/processor of these drugs. However, cases of inoperable tumors of the liver TACE and TARE are commonly used.

There are a number of known causes of liver cancer, with ~50% of cases stemming from hepatitis B virus (HBV) infection, as well as hepatitis c virus (HCV) infection, though for the latter, incidence rates have significantly declined due to antivirals that have been able to induce sustained

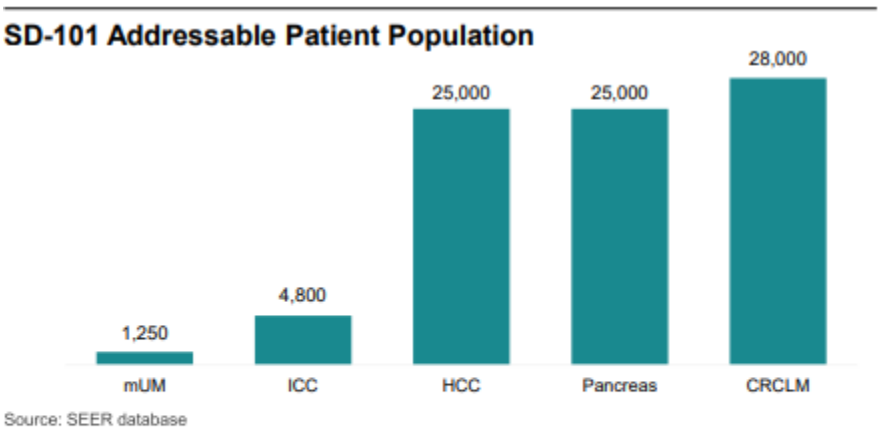
virological response.[4] Other risk factors/causes include non-alcoholic steatohepatitis (NASH), metabolic syndrome, and diabetes. There is also a smaller number of cases related to mutagenic factors like tobacco and alcohol. There have been some mutational drivers such as TERT, CTNNB1, and TP53, which have been identified in ~25% of cases though these targets to date still remain undruggable.[5] In general, liver cancer is a prototypical inflammation-associated cancer with 90% or so of the cancer burden associated with prolonged inflammation. The sources of inflammation can be due to hepatitis, excessive alcohol, NASH, or non-alcoholic fatty liver disease (NAFLD).

From an immune perspective, it should be noted that the liver harbors the largest number of immune cells in the body and has a unique immune state that makes it more tolerant than other organs. This is due to the constant flow of inflammatory signals from the gut. Essentially everything flows through the liver. However, while this state of tolerance eventually may be more permissive of liver cancer development and although the interaction of immune cells with liver malignant cells has a unique dynamic, it does point to an opportunity for immune-based therapies like checkpoint modulators to be effective. One key challenge in the immune therapy approach, in addition to actually penetrating the tumor due to vasculature issues, is the presence of MDSCs and a "cold" TME. Response rates for CPIs have been relatively poor in HCC with the combination of nivolumab (PD1) + ipilimumab (CTLA4) overall response rate (ORR) at 32% and atezolizumab (PD-L1) + bevacizumab (VEGF, not a CP) at 27% ORR. In ICC, ORR is more limited in the 20%–30% range, but like HCC, there seems to be challenges with an immune suppressive TME.

Pancreatic cancer. In the United States, there are 64,000 new cases of pancreatic cancer annually and it is the fourth-leading cause of cancer deaths. The five-year survival rate is only 12%. One of the factors that make it so fatal is the stage at which it is often diagnosed. Early symptoms are vague and not very noticeable; by the time significant symptoms appear, the cancer is likely at a locally advanced stage and has metastasized, at which point, surgical intervention is no longer a viable option. Surgery, the only potentially curable treatment, is available to only about 20% of patients, the vast majority of whom experience tumor relapse. The median overall survival for adenocarcinoma of the pancreas, accounting for ~95% of pancreatic cancer, is eight to 11 months. The needle has moved very little with respect to survival outcomes since the approval of gemcitabine, the current standard-of-care chemotherapy. Poorly perfused connective tissue in combination with high interstitial pressure leads to an avascular environment that is not receptive to systemically administered chemotherapy. Metabolism aberrations and immune system evasion are other characteristics of pancreatic adenocarcinoma.[6] This may be due to the presence of immune suppressors such as MDSCs and scarcity of effector T cells. As such, immune therapies like the CPIs have not had substantial success in treating pancreatic cancer with response rates <10%.

Other liver metastases. In addition to the above indications, TriSalus may also move into liver mets that arise from colorectal cancer. Like in UM liver mets, the objective here would be to treat the liver tumor to extend life and perhaps bridge to other therapies. The prognosis in these populations though is poor, late-stage disease.

Exhibit 11. PEDD + SD-101 addressable market. TriSalus estimates that the addressable market for combined indications (mUM, ICC, HCC, panc, CRC-LM) is ~$15B, or >80,000 cases. This estimate in cases is based on NCI SEER (National Cancer Institute- Surveillance, Epidemiology, and End Results Program) statistics.



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

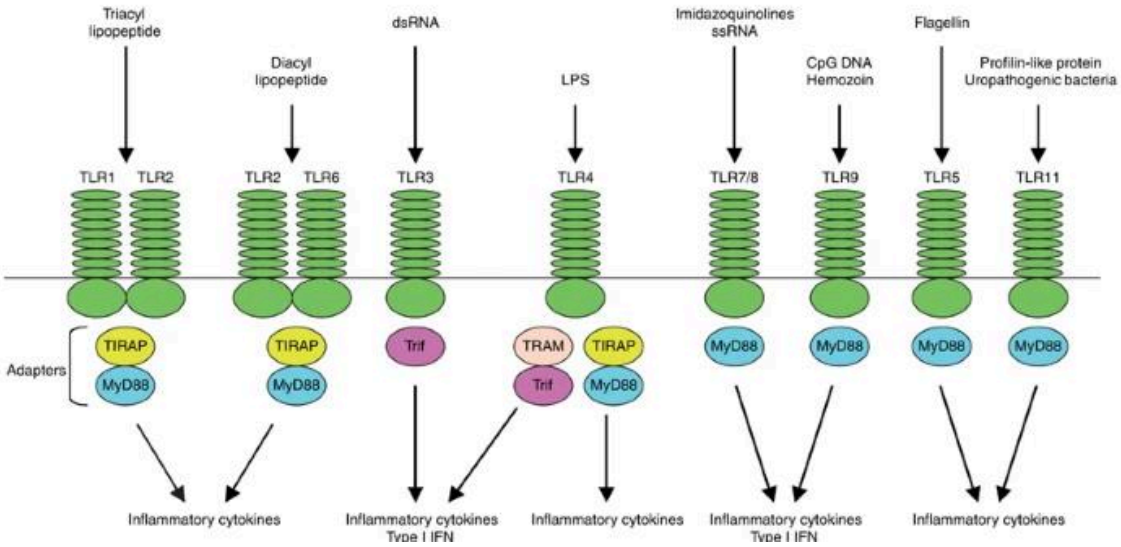
[4] Dasgupta P et al., Front Oncol. 2020; 10(171)
[5] Zucman-Rossi J et al., Gastroenterology. 2015; 149(5):1226:1239
[6] 3 Feig C, Gopinathan A, Neesse A, et al. The pancreas cancer microenvironment. Clin Cancer Res 2012;18:4266-76.

Source: TriSalus Life Sciences

SD-101 and Targeting TLR9

Toll-like receptor 9. In humans, there are 10 toll-like receptors (TLRs) that in general respond to various stimuli and trigger intracellular signaling pathways that result in the production of inflammatory cytokines, type-I interferon, and chemokines. They also induce upregulation of co-stimulatory molecules on dendritic cells (DC) as part of DC maturation, and thus act not only as key mediators of innate immunity, but also as a link between innate and adaptive responses. Essentially, these 10 TLRs have a common signaling pathway that results in induction of inflammatory cytokines, including TNFα, IL-6, IL-1β and IL-12, as well as alternate pathways to drive effector responses. While the signaling converges on induction of inflammatory response, there is variability in what is produced. The TLRs also have different molecular patterns that they recognize; ligands include triacyl lipopeptide (TLR1,2), diacyl lipopeptide (TLR 2,6), dsRNA (TLR3), lipopolysaccharide (TLR4), bacterial flagellin (TLR5) ssRNA (TLR 7,8), unmethylated CpG DNA motifs (TLR9), uropathogenic bacteria, and profilin-like protein (TLR11).

Exhibit 12. TLR family, ligands.



Source: Kawai 2006[7]

TLR9s are predominantly expressed on B cells, T cells, and dendritic cells, particularly in immune-rich tissues. TLR9 is located within the endoplasmic reticulum, and upon stimulation by its CpG-DNA motifs, translocate to endosomes. In the absence of its ligand unmethylated CpG DNA, TLR9 remains a monomer. Activation of TLR9 is driven by CpG-DNA ligand binding followed by proteolytic cleavage of TLR9 to form a heterodimer. However, CpG DNA motifs are not all the same and structure/sequence differences lead to differential downstream immune responses. There are three classes (A, B and C) of CpG-DNA that differ based on backbones and sequence motifs. These are important to differentiate since the TL9-targeting therapeutics space with synthetic TLR9 CpG agonists is differentiated along these classes. TriSalus' SD-101 is a class C. What is common between them is that TLR9s in general detect unmethylated CpG motifs with a preference for CpG hexamers (5'-GTCGTT-3 in humans)[8].

Type A CpG:

- Backbone/motif: poly G at 5', 3' or both, internal palindrome sequence with GC, partial phosphonothioate (PS) modified backbone.
- Activation: plasmacytoid dendritic cells (pDC), natural killer (NK) cells, drive pDC interferon- α production (INF-α).

Type B CpG:

- Backbone/motif: ~18-28 nucleotides, complete PS-modified backbone, contain one or more 6mer CpG motifs (5'-PuPyCGPyPu-3'). Most potent type Bs have three 6mers.
- Activation: B cells, some/little NK cells, no effect on DCs

Type C CpG:

- Backbone/motif: features of both type A and type B CpGs; complete PS-modified backbone and internal palindrome sequence.

[7] Kawai and Akira., Nature. 2006; 13:816-825
[8] Karapetyan et al., OncoTargets and Therapy. 2020; 13:10039-10060

- Activation: B cell, INF- α production by pDCs, antigen presenting cell activation/maturation, indirect NK-cell activation[9].

TLR9 agonists in cancer. The development of checkpoint inhibitors ushered in a new era in the management of cancer, particularly with clinical and commercial success of anti-PD1 (programmed cell death 1), anti-PD-L1 (programmed cell death ligand 1) and anti-CTLA4 (cytotoxic T-lymphocyte associated protein 4) therapies. In addition, the first anti-LAG3 (lymphocyte activating gene 3) was also recently approved. These CPIs have been approved across multiple indications in 16 different diseases. One key to their success has been the durability of responses in subsets of patients that have responses. However, the majority of patients do not have adequate responses (10%–35%) and/or those that do ultimately may have disease progression. A key driver is the complexity and diversity of the tumor microenvironment (TME).

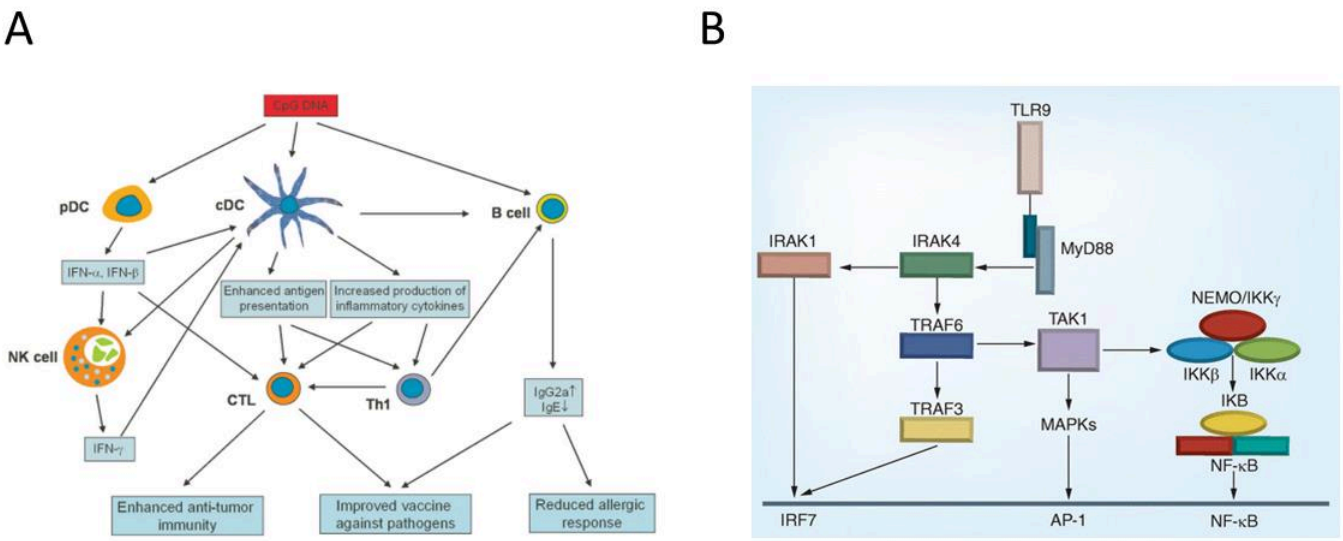
There are biomarkers associated with improved outcomes with CPI therapies including T-cell infiltrate in the tumor region, production of INFγ (interferon gamma), mutation burden, and PD-L1 expression. While the T cells are there, it doesn't always mean a CPI can work well. An inflamed T cell, or "hot" tumor, is typically characterized by the presence of C8+ T cells, CXCL9, CXCL10, and high INFγ, as well as other proinflammatory markers. Conversely, there is the TME that is "cold", characterized by the presence of immunosuppressive cells including Tregs, myeloid-derived suppressor cells (MDSCs) and tumor-associated macrophages (TAMs), vascular endothelial cells, and cancer-associated fibroblasts (CAFs). As such, alteration of the immune environment in the TME is critical to expanding the use of immune-based therapies like CPIs, and thus there is the opportunity for TLR9 agonists like SD-101.

Basic TLR9/CpG DNA signaling. In general, signaling through TLR9 ultimately drives an adaptive immune response, a key component of which for cancer is the increase in CTLs and NK cells. The intracellular signaling pathway is through TLR9-MyD88. In steady state, meaning with no CpG ODN (oligodeoxynucleotide, e.g. CpG DNA) stimuli, TLR9 is localized to the endoplasmic reticulum (ER). A CpG-ODN binds TLR9, triggering dimerization as noted above. This triggers a conformation change in TLR9 that leads to the recruitment of MyD88 (myeloid differentiation primary response 88). Myd88 is an adapter protein that receives signals, in this case from TLR9, and then relays that signal through a series of downstream steps. The next step is the activation of IRAK-4 (interlukein-1 receptor-associated kinase-4), which is critical for TLR9-mediated pro-inflammatory cytokine production. IRAK-4 then recruits TRAF6 (tumor necrosis factor receptor associated factor 6), followed by activation of TAK1 (transforming growth factor-β associated kinase 1) leading to activation of NF-kB (nuclear factor kappa light chain) and MAPKs (mitogen-activated protein kinases). The latter leads to AP-1 (activator protein-1) activation. Both NF-kB and AP-1 are important transcription factors that will drive the increased expression of the proinflammatory cytokines, IL-6, IL-12 and TNF, as well as co-stimulatory molecules CD80 and CD86.

In plasmacytoid dendritic cells (pDCs), which are important in the TLR9-CpG-DNA response, produce high levels of the INF (type 1 interferons). Interestingly, in non-pDCs like conventional dendritic cells (cDCs), the cytokines produced do not include INFs, though it's not clear exactly why yet. However, both pDCs and cDCs, as well as B-cell activation, all of which are antigen presenting cells, are stimulated through this TLR9-CpG-DNA pathway and ultimately contribute to the recruitment of CTLs and NK cells in the tumor microenvironment. This signaling is also important for driving our immune suppressive cells, like MDSCs...turning a "cold" tumor "hot". From a cancer therapeutics perspective, this sets the stage for the inclusion of a checkpoint inhibitor (CPI) combination therapy with a TLR9 agonist, like SD-101.

Exhibit 13. TLR9 signaling and cell activation. (A) CpG DNA can activate TLR9 on plasmacytoid dendritic cells (pDCs), conventional dendritic cells (cDCs), and B cells. This leads to mainly type I interferons, such as INFα and INFβ, which then act on various cell types including T cells, NK cells, and cDCs. **(B)** Basic signal propagation upon TLR9 stimulation via CpG-DNA, ultimately leading to AP-1 and NF-kβ-driven expression of proinflammatory cytokines.

[9] Gursel et al., J. Leukoc Biol. 2002; 71(5):813-820



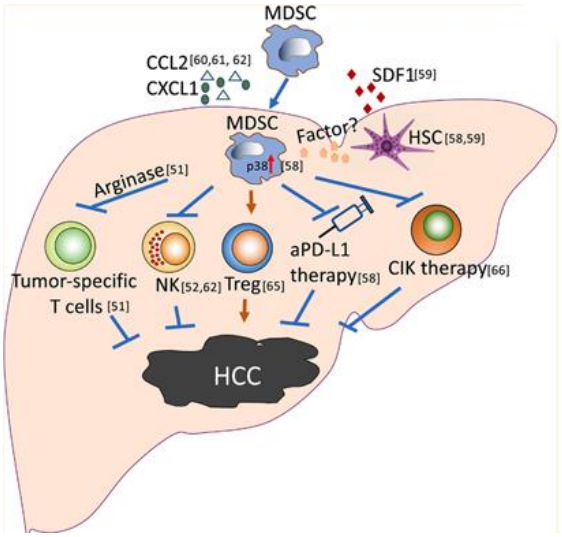
Source: (A) Huang 2010[10] (B) Farrokhi 2017[11]

SD-101, targeting liver tumors and the MDSC issue. SD-101 is a class C CpG-ODN in development for liver tumors arising either primary or from metastases of other cancers as noted above. Challenges in targeting liver tumors are multi-fold, including actually being able to reach the tumor with systemic therapy and immune suppressive environment of the organ itself. The immune suppressive properties of the liver immune cells are important as they protect the organ from excessive immune responses to foreign antigens. In other words, the liver tends to be "immune tolerant". However, while important, this also limits the ability of the liver to fight cancerous cells. Immune-suppressive MDSCs are an important component of the tumor microenvironment (TME) and promote tumor progression through a variety of mechanisms. MDSCs in general are a heterogenous population that include two primary subsets; polymorphonuclear (PMN-MDSCs) which resemble neutrophils and monocytic (M-MDSCs), which have markers in common with monocytes, macrophages, and dendritic cells. Nonetheless, these cells originate via normal myeloid differentiation and have the capacity to differentiate into DCs or macrophages[12].

MDSCs inhibit proliferation of anti-tumor immune cells in the TME, including both T cells and NK cells, as well as promote tumor invasion. This is in part mediated by L-arginine depletion by arginase 1 or reactive oxygen species. The liver is also the preferred organ for tumor MDSC accumulation. Liver MDSCs are recruited to the liver via expression of CCL2 and CXCL1. In addition, liver MDSCs expand in response to granulocyte-macrophage colony-stimulating factor (GM-CSF) secreted by tumor cells. The GM-CSF enhances capacity to immune suppress through STAT3 (signal transducer and activator of transcription 3) signaling, (indoleamine 2,3-dioxygenase) and PD-L1.

Exhibit 14. MDSCs promote liver tumors and inhibit immunotherapy.

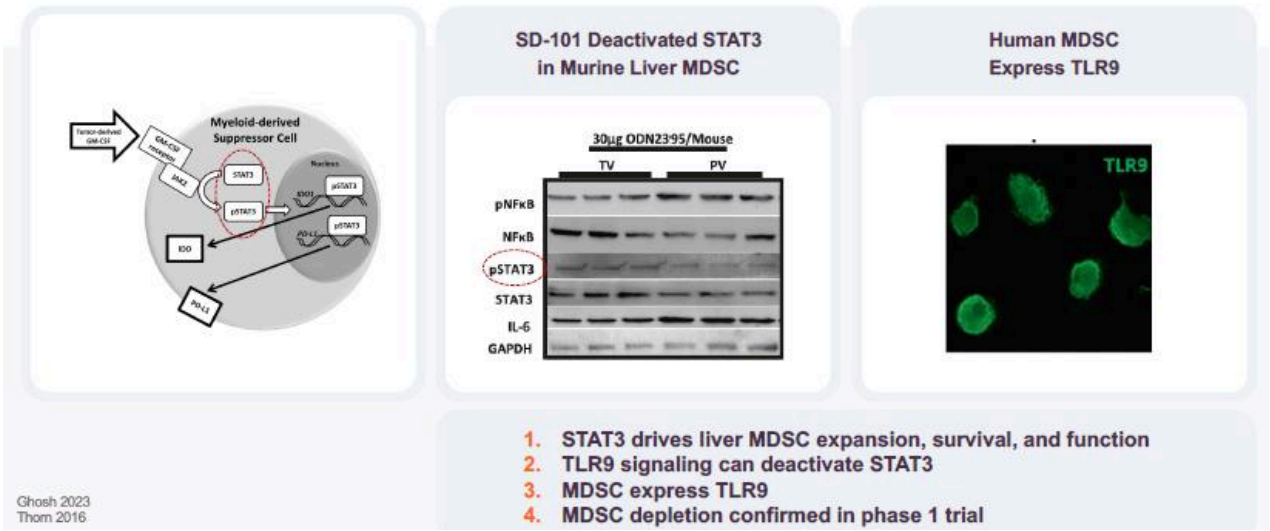
[10] Huang H., Expert Opin Ther Targets. 2010; 14(8):787-796
[11] Farrokhi et al., Immunotherapy. 2017; 9(4)
[12] Ostrand-Rosenberg S., J Immunol. 2018; 200: 422-41



Source: Ma et al., 2021[13]

Overall, as described in a paper titled **"MDSC; the most important cell you have heard of"** published in 2019, the MDSC can be characterized as the 'queen bee' of the tumor microenvironment, protecting the cancer from the patient's immune system and making the tumor resistant to immunotherapy. Being able to eliminate MDSCs should improve response rates[14]. For liver cancer and liver mets therapeutics, the challenge is two-fold; dealing with the hostile cold TME driven largely by MDSCs, and drug delivery. For the latter, as described above, liver tumors are high pressure, with leaky and/or collapsed vasculature, making drug delivery extremely difficult. SD-101 delivered via PEDD can potentially solve both challenges and set the stage for checkpoint inhibitors or other therapies to be more effective.

Exhibit 15. SD-101 deactivates STAT3, a key signaling molecule in MDSCs that drives immunosuppression.



Source: TriSalus Life Sciences presentation.

TriSalus acquires SD-101 oncology program from Dynavax. On August 3, 2020, TriSalus announced it had entered into an asset purchase agreement with Dynavax Technologies Corporation under which TriSalus purchased SD-101, a proprietary investigational, second-generation, TLR9 agonist CpG ODN Class C. The asset, prior to TriSalus acquiring it, had been studied in advanced cutaneous melanoma and was in development for high-risk stage II/III breast cancer. The purchased assets included SD-101-related intellectual property, clinical data, regulatory filings, and inventory. Under the terms of the agreement, TriSalus made a $5M upfront payment with an additional $4M payment on 12/30/20 for

[13] Ma C., Cell Immunol. 2021; 361
[14] Tesi RJ., Trends in Pharm Sci. 2018; 40(1):4-7

reimbursement of research and development expenses, up to $250M in biobucks (development, regulatory, commercial milestones) and low double-digit royalties on future commercial sales.

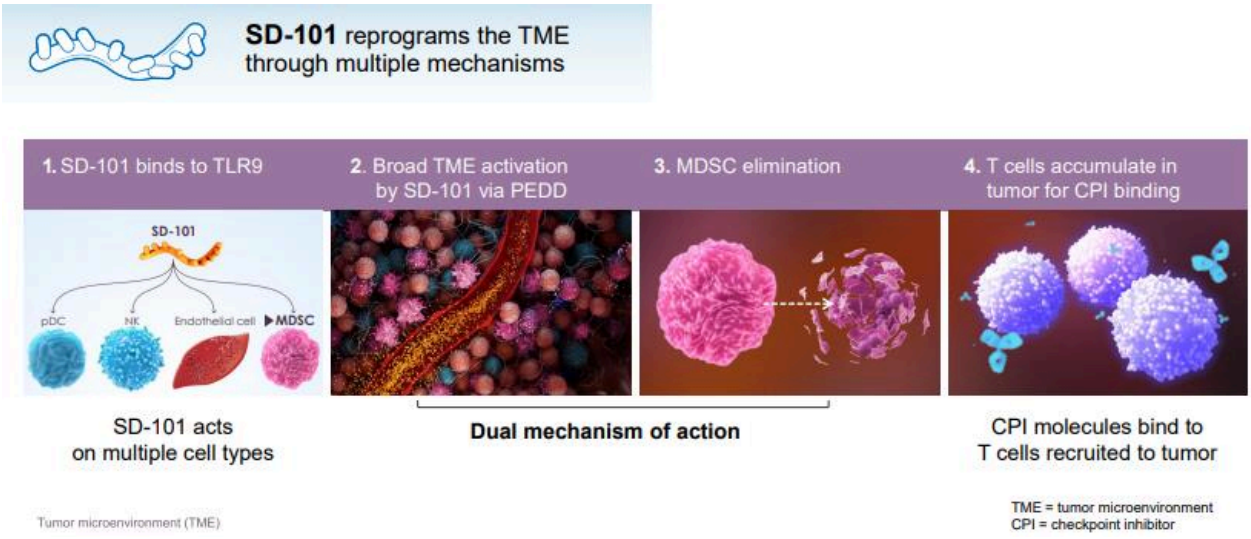
Prior clinical data for SD-101; advanced melanoma, head and neck cancer. The data for SD-101 while it was with Dynavax was quite compelling and it's important, in our view, to note that the SD-101 delivery, like it has been with other CpD-ODNs, is by intratumoral injection. This alters the TME to make it more favorable for CPIs to be more effective. In addition, there are some abscopal effects. Intratumoral injection is the approach used generally in the CpG-ODN space, including for Checkmate Pharmaceuticals ([CMPI—NR] was acquired by Regeneron [REGN—NR] in 2022 for $250M). We would also point out that Checkmate/Regeneron's drug, vidutolimod is a class A CpG-ODN that does not impact MDSCs. From a delivery perspective, the liver and pancreas are particularly challenging to perform intratumoral injection; thus, the approach by TriSalus to use PEDD as an alternative option. Now let's look at some prior SD-101 data using just intratumoral injection.

Exhibit 16. TLR9 agonists, combination therapies in development.

TLR9 agonist	Interventions	Conditions	Phase	Status	NCT number
CMP-001	Drug: Pembrolizumab	Carcinoma, Squamous cell of head and neck	Phase 2	Recruiting	NCT04633278
	Drug: Nivolumab	Melanoma, Advanced melanoma, Metastatic melanoma, Unresectable melanoma	Phase 2	Recruiting	NCT04698187
	Drug: Cemiplimab-rwlc	Advanced cancer, Metastatic cancer	Phase 2	Recruiting	NCT04916002
	Drug: Nivolumab	Melanoma, Advanced melanoma, Metastatic melanoma, Unresectable melanoma	Phase 2, 3	Recruiting	NCT04695977
	Radiation: Stereotactic body radiotherapy	Triple negative breast cancer	Phase 2	Recruiting	NCT04807192
	Biological: Nivolumab	Melanoma, Lymph node cancer	Phase 2	Completed	NCT03618641
	Biological: Agonistic Anti-OX40 Monoclonal Antibody INCAGN01949	Locally advanced malignant solid Neoplasm, Metastatic pancreatic Adenocarcinoma, Stage IV pancreatic cancer AJCC v8, Unresectable malignant solid Neoplasm	Phase 1, 2	Recruiting	NCT04387071
	Biological: Nivolumab, Other: [18 F]F-AraG PET/CT	Melanoma	Phase 2	Recruiting	NCT04401995
	Biological: Pembrolizumab, Procedure: Surgical Procedure	Clinical stage III cutaneous Melanoma AJCC v8, Melanoma of unknown primary, Pathologic stage IIIB cutaneous Melanoma AJCC v8, (and 3 more...)	Phase 2	Recruiting	NCT04708418
	Drug: Pembrolizumab	Lymphoma	Phase 1, 2	Recruiting	NCT03983668
	Radiation: Liver radiation therapy Drug: Nivolumab Injection [Opdivo] Drug: Ipilimumab Injection [Yervoy]	Colorectal neoplasms malignant, Liver metastases	Phase 1	Unknown	NCT03507699
CpG-7909	Biologica: ELI-002 immunotherapy peptide-based antigens	Minimal residual disease, KRAS G12D, KRAS G12R, (and 9 more...)	Phase 1	Recruiting	NCT04853017
SD-101	Biological: Nivolumab, Ipilimumab	Metastatic uveal melanoma in the liver	Phase 1	Recruiting	NCT04935229
	Biological: Anti-OX40 Antibody BMS 986178	Advanced malignant solid neoplasm, Extracranial solid neoplasm, Metastatic malignant solid neoplasm	Phase 1	Active, not recruiting	NCT03831295
	Biological: Anti-OX40 Antibody BMS 986178 Other: Laboratory Biomarker Analysis Radiation: Radiation Therapy	B-cell non-Hodgkin lymphoma, Grade 1 follicular lymphoma, Grade 2 follicular lymphoma, (and 5 more...)	Phase 1	Active, not recruiting	NCT03410901
	Biological: Nivolumab, Radiation: Radiation Therapy	Metastatic pancreatic Adenocarcinoma, Refractory pancreatic Adenocarcinoma, Stage IV pancreatic cancer AJCC v8	Phase 1	Active, not recruiting	NCT04050085
	Drug: Epacadostat Radiation: Radiotherapy	Advanced solid tumours, Lymphoma	Phase 1, 2	Completed	NCT03322384
	Biological: Nivolumab Biological: Ipilimumab Biological: Pembrolizumab	Hepatocellular carcinoma, Intrahepatic cholangiocarcinoma	Phase 1, 2	Recruiting	NCT05220722

Source. Dongye et al 2022[15]

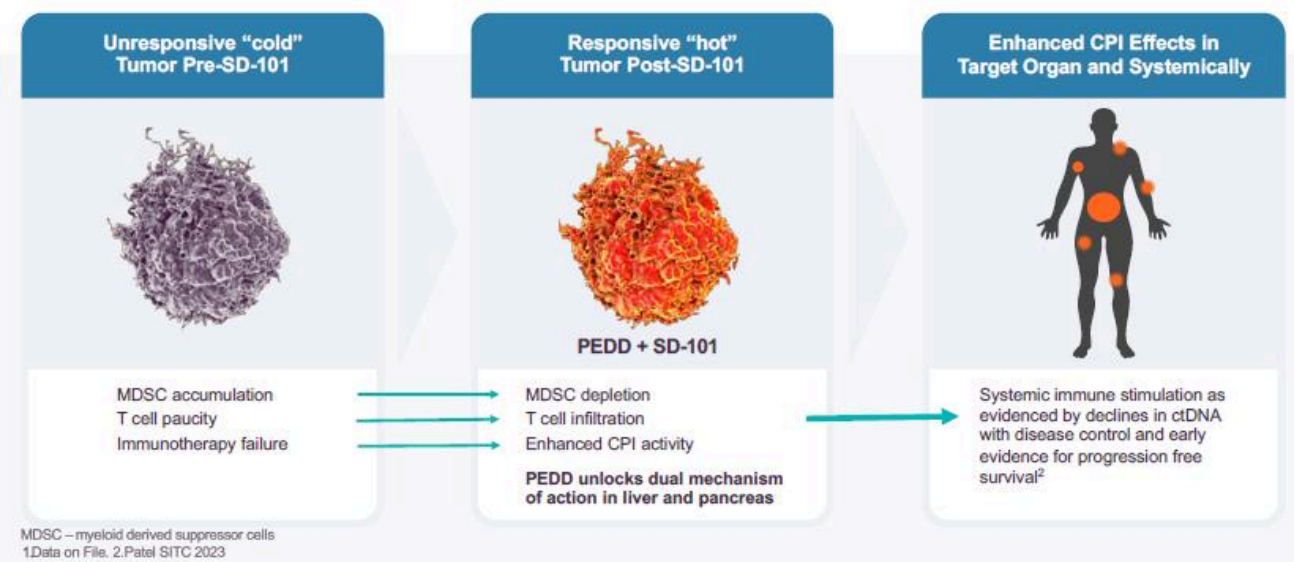
Exhibit 17. SD-101 dual mechanism of action (MOA); MDSC elimination, drives T-cell accumulation.



Source: TriSalus Life Sciences presentation.

Exhibit 18. SD-101 dual MOA with potential to enhance checkpoint activity in the TME and systemically.

[15] Dongye et al. British Journal of Cancer. 2022; 127:1584-1594



Source: TriSalus Life Sciences presentation

SD-101 in advanced melanoma. In June 2019, updated data from a P1b/2 study (SYNERGY-001/KEYNOTE-184) of SD-101 + pembrolizumab ("pembro", Keytruda) was presented at ASCO (American Society of Clinical Oncology). In the study, SD-101 was delivered by intratumoral injection with 8mg in 1 lesion (n=45) or 2mg in lesions 1–4 combined (n=41) with 200mg pembro intravenous. The overall response rate (ORR) in the SD-101 2mg/lesion group was 76% vs. 49% in the 8mg/lesion group. 18-month progression-free survival (PFS) was 72% vs. 36% and there were similar rates of responses in patients with PD-L1 negative and PD-LI positive tumors. Tumor shrinkage was observed in both injected and non-injected lesions, such as in the liver and lung. These tumors are "cold", with PD-L1 negative, low INFγ and T-cell signature at baseline, and were converted into "hot" tumors. Positive response rates were also shown in a P1b/2 combo study in advanced/metastatic melanoma that was resistant to CPI therapy. In this setting, the ORR was 19.4% in the 2mg/lesion group and 13.3% in the 8mg/lesion group. Infiltration of the TME with activated T cells, NK cells, and B cells was observed demonstrating conversion of the tumor to an inflamed state. This points to the dual mechanism of action (MOA) of SD-101 to eliminate the MDSCs and alter the TME.

Exhibit 19. SD-101 + pembrolizumab, response rates in advanced melanoma (Data presented by Dynavax at ASCO 2019.)

Best Overall Response Rate (ITT)	2 mg/lesion (N=45)	8 mg/lesion (N = 41)
Objective response rate (ORR), n (%) (95% CI)	**34 (76)** (61, 87)	**20 (49)** (33, 65)
Complete response	8 (18)	4 (10)
Partial response	26 (58)	16 (39)
Stable disease	2 (4)	7 (17)
Progressive disease	5 (11)	9 (22)
Not evaluable†	4 (9)	5 (12)
Time to response, median (months)	2.2	2.3
Duration of response (DOR), median (months) (95%CI)	not reached (NE, NE)	not reached (14.2, NE)

† Patients discontinued prior to first scan: 2 mg—clinical progression (n=3), consent withdrawn (n=1); 8 mg—clinical progression (n=2), irAE/AE (n=2), withdrew consent (n=1). NE, not estimable; ITT, intent to treat

Note: The concordance between blinded central assessment and investigator assessment on a subset of the 2 mg group (n=38) was 89%

- ORR in patients with BRAF mutant tumors who received 2 mg/lesion (n=18) was 61%
- ORR in patients with PD-L1 negative tumors who received 2 mg/lesion (n=14) was 79%

Source. Dynavax ASCO 2019

SD-101 in recurrent/metastatic head and neck cancer. Also presented at ASCO 2019 by Dynavax was the SD-101 + pembro combo from a P1b/2 study (open-label, multicenter) in CPI treatment naïve patients with recurrent or metastatic head and neck squamous cell carcinoma (HNSCC), n=50. In the study, SD-101 was delivered by intratumoral injection with 8mg in 1 lesions or 2mg in 1–4 lesions combined with IV pembro. The ORR was 24% in the intent-to-treat (ITT) population. In the 2mg/lesions group and 8mg/lesion group the ORR was 22.2% and 26.1%, respectively. In patients with low PD-L1 expression, the ORR was 33.3% and disease control rate (DCR) was 41.6%, which points to

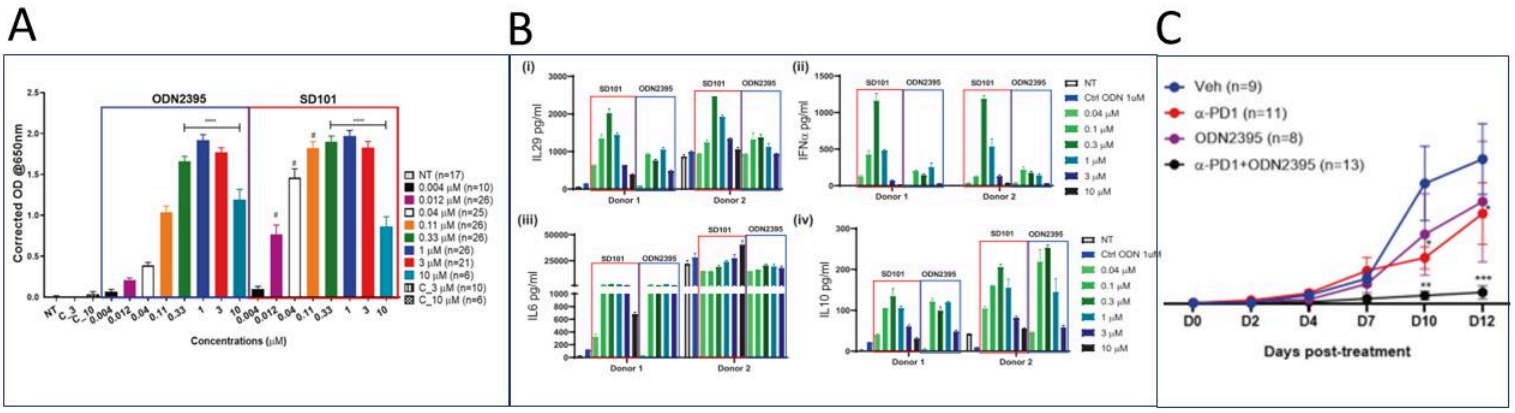
increased tumor inflammation with SD-101 to support use of pembro in combination. The ORR in HPV-positive tumors was 36%. The biomarker data demonstrated infiltration of active T cells and upregulation of type 1 and type II interferon. As in the melanoma study above, in patients that displayed "cold" tumors, SD-101 was driving response to pembro.

TriSalus delivers SD-101 via PEDD. With its Pressure-enabled Drug Delivery (PEDD) technologies TriSalus is targeting liver and pancreatic tumors, which are notoriously "cold" tumors and there has been little success with CPI therapies. Most of the work done with TLR9 agonists has focused on more superficial tumors that are more amenable to injection. The application of TLR9 agonists, including SD-101 (while it was with Dynavax), for liver (or pancreatic) tumors has been limited by the challenges of direct injection into the liver tumor and the high intratumoral pressure where there is limited access via circulation. Using PEDD to deliver SD-101 is a differentiated approach in the TLR9 space.

Preclinical data supports ongoing clinical program(s). In a study published by Ghosh et al. in 2022 PEDD delivery of ODN2395, a class C TLR9 against available from InvivoGen, and SD-101, was evaluated for the impact on the TME and changes in responsiveness to CPI. This was done via regional intravascular infusion of ODN2395 into mice with liver metastases. This study compared multiple dose levels of ODN2395 (1, 3, 10, and 30µg) delivered via PEDD using the portal vein or 30µg via the tail vein: regional to the liver vs. systemic delivery of a class C TLR9 agonist. It was found that there was improved tumor control at 30ug via the portal vein and not via the tail vein, which suggested it's more ideal for regional delivery. In harvested liver mets, researchers found that in portal vein delivered ODN2395, there was enhanced activity of proinflammatory markers NF-kB, STAT3, and IL6. In addition, there were significantly reduced MDSCs. M1 macrophage increases were observed in the 30ug doses delivered by either the portal vein or tail vein, but significantly increased liver M1 polarization with portal vein delivery. Note, M1 macrophages mediate anti-tumor immune responses.

This was with the ODN2395, so what about the potency of SD-101 in this model? The rationale, in part, in this paper, is in support of SD-101 given its ongoing development for treating liver metastases from uveal melanoma. Similar responses in TLR9 signaling activity were observed and there were similar patterns in cytokine production (IL6, IL10, IL29, INFα) as well as reduced MDCSs. It was also shown that TLR9 is being expressed on the surface of mouse liver met samples, which is supportive of the use of regional delivery of SD-101 to the liver. Given that MDSCs arise from peripheral blood mononuclear cells, human PBMCs were induced via IL6 + GMCF to transform into MDSCs. However, the presence of SD-101 reduced the MDSC population and significantly increased the M1 macrophage population. Combined, these data point to a potent class C TLR9 agonist in SD-101 and are supportive of this class of CpG ODN to alter the TME. The next step was to model the ongoing P1/1b study in uveal melanoma liver mets of SD-101 + CPI. To show this, mice with liver mets were treated with ODN2395 regionally in the liver and then treated with or without systemic anti-PD1 CPI.

Exhibit 20. Preclinical data supporting class C TLR9 agonists. Shown below are select images from preclinical work conducted by Ghosh et al. published in 2022. In these experiments TLR9 agonists ODN2395 and SD-101 are used to evaluate delivery (regional vs. systemic), cytokine induction/pattern, TLR9 expression, TME changes, control of liver met tumor burden, and combination with anti-PD1. **(A)** Effects of ODN2395 and SD101 on NF-kB activity. **(B)** Both ODN2395 induced reductions in MDSCs (not shown) and increased production of cytokines IL6, IL29, IL10, INFα. **(C)** In mouse model of liver mets, the combination of ODN2395 + anti-PD1 enhanced tumor control.



Source. Modified from Ghosh et al., 2022[16]

In preclinical work that was presented in November 2023 at SITC, a mouse model of liver mets was used to demonstrate the potential of the combination of SD-101 delivered regionally with checkpoint therapy given either systemically or by subcutaneous injection. In the data presented, SD-101 in combination with checkpoint delivered by either route, induced reduction in MDSCs and increased B cells, T cells, and M1 macrophages in the liver mets. In addition, there was an increase in proinflammatory cytokines INFγ and IP10 in the peripheral circulation

[16] Ghosh et al., Cancer Gene Therapy. 2022; 19: 1854-1865

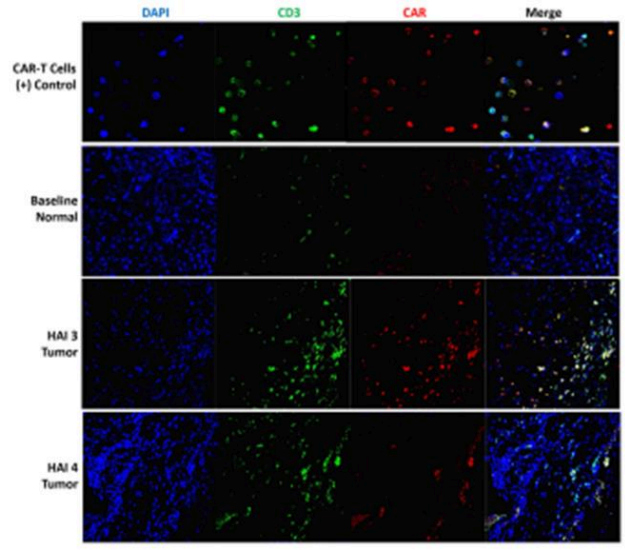
demonstrating immune activation in the periphery where there is little exposure to SD-101. These observations were further supported by gene transcript analysis and in mouse models where there was increased survival for liver met-bearing mice. These observations have been replicated in the clinical development of SD-101 PEDD in combination with checkpoint therapy in the PERIO-01 study, which was also presented at SITC and is shown in detail below. The poster for the preclinical work at SITC can be found HERE.

PEDD can deliver CAR-T cells too, clinical. The preclinical work shown above in the Ghosh paper was led by principal investigator Steven Katz, M.D., FACS, TriSalus' Chief Medical Officer. Dr Katz has also demonstrated the utility of PEDD for the delivery CAR-T cells to solid tumors in the liver. CAR-T therapies, while having success in hematological cancers, have challenges with solid tumors. Tumors of the liver are particularly challenging due to a combination of factors including increased intratumoral pressure, poor T-cell trafficking and immune suppressive TME. As such, regional delivery may be an attractive approach.

In 2015, a P1 study was published demonstrating the utility of regional delivery via hepatic artery infusion (HAI) for delivery of anti-CEA (carcinoembryonic antigen) CAR-T cells as immunotherapy for adenocarcinoma liver metastases. These tumor types have high levels of CEA expression which makes them an attractive target for CAR-T therapy. In addition, CEA levels can be measured in the serum. CEA-targeting CAR-T cells had been used previously via systemic administration and demonstrated hopeful results but had dose limiting toxicities. By delivering CEA CAR-T cells regionally, toxicity may be reduced while achieving enhanced CAR-T cell delivery to, and killing of, tumor cells. Eight patients were enrolled in the study and six completed the protocol. These patients had unresectable CEA+ adenocarcinoma liver mets (four having more than 10 lesions) and a mean of 2.5 lines of prior systemic therapies. Of the six patients that completed protocol, three were a part of dose escalation, receiving 10^8, 10^9, and 10^10 cells, while another three were treated with three doses of the higher 10^10 cells along with systemic IL-2. Post-CAR-T cell infusion biopsies showed that the CAR cells were more abundant in the tumor than the normal liver; 5/6 patients. In 4/6 patients, the CAR cells were not found in peripheral blood, and only transiently in two other patients. From a safety perspective, there were no grade 4 or 5 adverse events. Five of the six patients at last follow-up died from disease progression, and one patient was alive at 23 months. Overall, these were very sick, heavily treated patients with high tumor burden. However, what the study showed was that the delivery approach was feasible and there were early signs of clinical activity.

In 2020, a P1b study was published that presented a case-study patient with liver mets from pancreatic cancer that received CEA-CAR-T therapy via PEDD, in combination with systemic IL-2 support. The CEA-CAR-T cells were delivered via hepatic artery infusion with PEDD to the site of the tumors. At six weeks follow-up there was no evidence of metabolically active disease in the liver or elsewhere in the body. This response continued at 3.7 months and the response was durable for 13 months before progression. The response was also evident via biomarker with 81% and 68% decrease in CEA and CA19-9, respectively. This was with three doses of cells. A further dose was administered after the progression point, 13.8 months. There were also marked changes in the tumor. Importantly, there was no evidence of systemic toxicity related to CAR-T therapy, which is further supportive of the HAI-PEDD delivery approach and presents future opportunities for TriSalus in the cell therapy space using its PEDD platform, in our view.

Exhibit 21. Liver tissue from P1b case study, demonstration of CAR-T cell penetration following HAI- PEDD delivery.



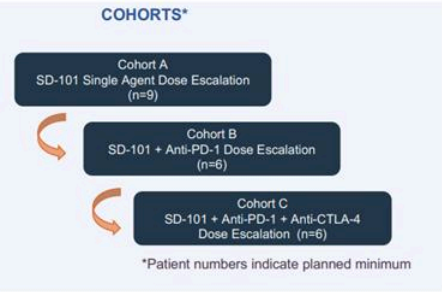
Source. Katz et al., 2020[17]

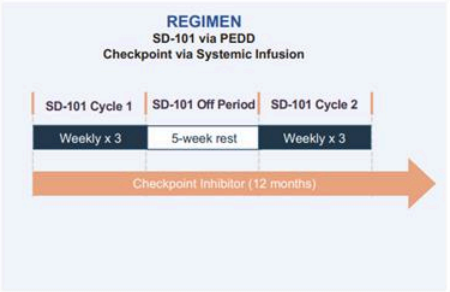
[17] Katz et al., J Immunother Cancer. 2020; 8

Clinical development of SD-101

PERIO-01 (NCT04935229) Targeting uveal melanoma liver metastases. This is an ongoing open-label P1/1b study of SD-101 via intrahepatic delivery by pressure-enabled regional immuno-oncology, PERIO-01. The study is evaluating SD-101 regional delivery combined with systemic administration of a checkpoint inhibitor across three cohorts; SD-101 alone (cohort A), SD-101 + anti-PD1 (cohort B), and SD-101 + anti-PD1/CTLA4 (cohort C). The study evaluated dose escalation cohorts of SD-101 at 2mg, 4mg, and 8mg delivered alone or with checkpoint therapy. In cohorts A–C and for P1b, patients will receive two cycles of SD-101, where one cycle consists of three consecutive weekly infusions followed by intravenous checkpoint therapy. An update on the program was presented at the Society of Immunotherapy for Cancer (SITC) meeting in November (2023). At the data cutoff of 9/29/23, there were 56 patients enrolled, with each having received at least one dose of SD-101; cohort A had n=13, cohort B had n=26, and cohort C had n=17.

Exhibit 22. PERIO-01 trial design, SD-101 via PEDD regimen, dose escalation.





	N = 56 (%)
Cohort A	13 (23)
2 mg	4
4 mg	5
8 mg	4
Cohort B	26 (46)
2 mg	7
4 mg	8
8 mg	11
Cohort C	17 (30)
2 mg	10
4 mg	7

Source. TriSalus Life Sciences, SITC 2023

Of the patients with available data, 16 patients (29%) were treatment-naïve and 40 (71%) had failed at least one prior line of therapy, including eight patients (14%) at 3L+. SD-101 infused via PEDD in combination with systemic checkpoint therapy was well tolerated, with an overall serious grade 3/4 adverse event rate related to treatment of 11% (n=56), and no such events at the optimal SD-101 dose level of 2mg in combination with anti-PD1 (n=7). The most common adverse events overall were gastrointestinal (41%), fatigue (30%), and skin toxicity (27%), with the majority being minor (Exhibit 21). PK data also demonstrated high levels of SD-101 in the liver region it was delivered to with transient drug levels in the peripheral circulation (not shown).

Exhibit 23. PERIO-01 safety data.

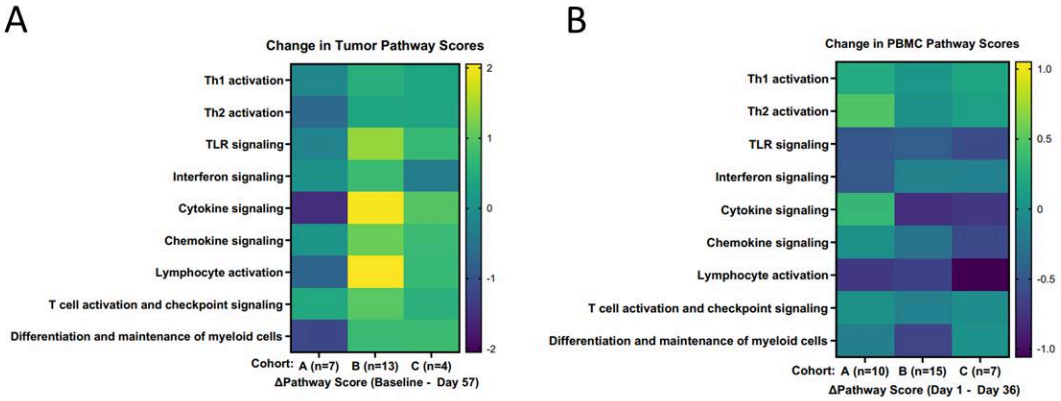
Phase 1: Cohorts A, B, C

All listed events are related to SD-101, ipi, and/or nivo	N=13 Cohort A Summary n (%)	N=4 Cohort A 2mg SD-101 n (%)	N=5 Cohort A 4mg SD-101 n (%)	N=4 Cohort A 8mg SD-101 n (%)	N=26 Cohort B Summary n (%)	N=7 Cohort B 2mg SD-101 + Nivo n (%)	N=8 Cohort B 4mg SD-101 + Nivo n (%)	N=11 Cohort B 8mg SD-101 + Nivo n (%)	N=17 Cohort C Summary n (%)	N=10 Cohort C 2mg SD-101 + Ipi/Nivo n (%)	N=7 Cohort C 4mg SD-101 + Ipi/Nivo n (%)	N=56 All cohorts n (%)
Treatment-related AEs (TRAE) (any grade)	**5 (38)**	0	3 (60)	2 (50)	**19 (73)**	5 (71)	6 (75)	8 (73)	**15 (88)**	10 (100)	5 (71)	**39 (70)**
DLTs	**0**	0	0	0	**2 (8)**	1 (14)	0	1 (9)	**0**	0	0	**2 (4)**
SAEs	**1 (8)**	0	0	1 (25)	**1 (4)**	0	1 (13)	0	**6 (35)**	4 (40)	2 (29)	**8 (14)**
≥G3 events (%)	**1 (8)**	0	1 (20)	0	**5 (19)**	1 (14)	2 (25)	2 (18)	**8 (47)**	5 (50)	3 (43)	**14 (25)**
Serious G3/G4 TRAEs	**0**	0	0	0	**1 (4)**	0	1 (13)	0	**5 (29)**	4 (40)	1 (14)	**6 (11)**
Most common AEs												
-Gastrointestinal Disorders	**3 (23)**	0	2 (40)	1 (25)	**8 (31)**	2 (29)	3 (38)	3 (27)	**12 (71)**	8 (80)	4 (57)	**23 (41)**
-Fatigue	**2 (15)**	0	2 (40)	0	**5 (19)**	2 (29)	2 (25)	1 (9)	**10 (59)**	7 (70)	3 (43)	**17 (30)**
-Skin Disorders	**0**	0	0	0	**7 (27)**	3 (43)	1 (13)	3 (27)	**8 (47)**	6 (60)	2 (29)	**15 (27)**
-Fever	**2 (15)**	0	1 (20)	1 (25)	**1 (4)**	0	0	1 (9)	**6 (35)**	3 (30)	3 (43)	**9 (16)**
-Administration Site Conditions	**1 (8)**	0	0	1 (25)	**4 (15)**	2 (29)	0	2 (18)	**4 (24)**	3 (30)	1 (14)	**9 (16)**
-Increased ALT	**0**	0	0	0	**4 (15)**	1 (14)	2 (25)	1 (9)	**4 (24)**	2 (20)	2 (29)	**8 (14)**
-Increased AST	**0**	0	0	0	**4 (15)**	1 (14)	2 (25)	1 (9)	**4 (24)**	2 (20)	2 (29)	**8 (14)**

Source. TriSalus Life Sciences, SITC 2023

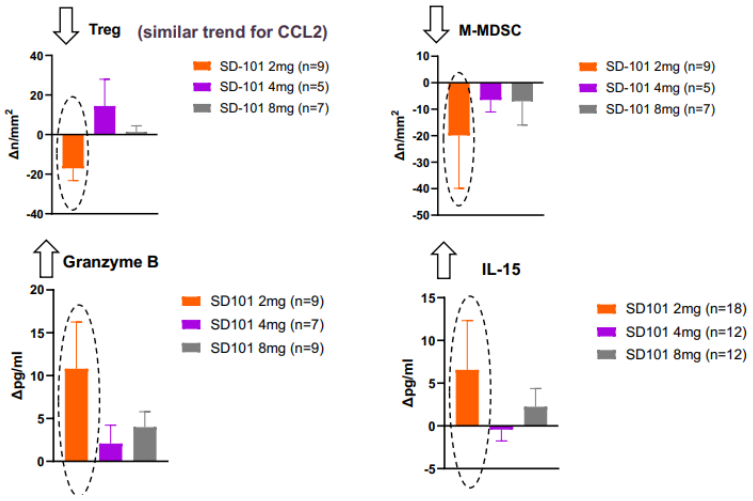
Increased levels of CD8+ T cells and NK cells were also observed in the liver tumors at day 57 (not shown). Gene expression analysis demonstrated increased TLR signaling, cytokine signaling, Th1 T-cell activation, and lymphocyte activation. This included increases in CXCL10, INFγ, TNFα, IL-2R, IL-15, and IL-18, pointing to SD-101 driving an inflamed tumor. This was further supported with demonstration of reduced MDSCs, Tregs, and M2 macrophages in the liver mets. Given the data, the optimal dose of 2mg SD-101 was identified.

Exhibit 24. Immune activation in the liver tumor and peripheral immune signature. (A) Increased immune activation within the liver mets at day 57. SD-101 delivery via PEDD into the liver tumor region drives T-cell activation and cytokine signaling. **(B)** In the periphery there is immune cell activation and cytokine signaling despite the low levels of SD-101 outside the liver.



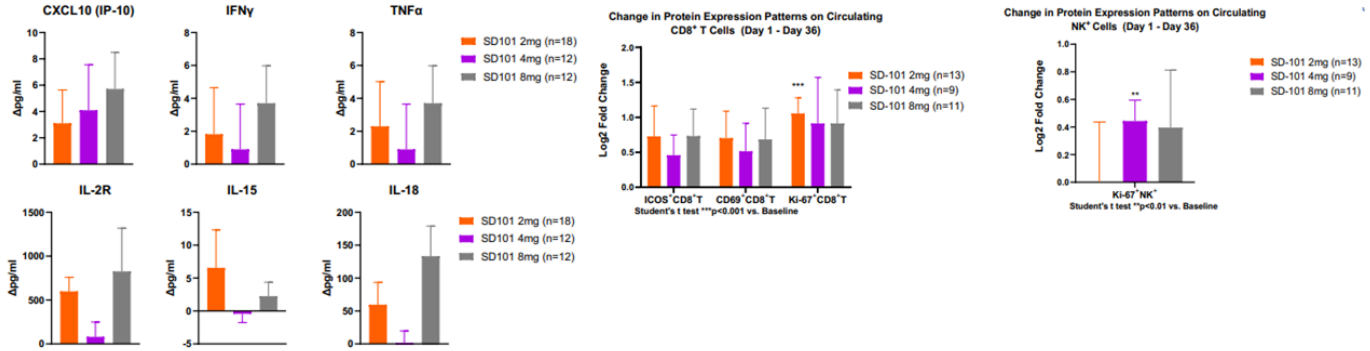
Source. TriSalus Life Sciences, SITC 2023

Exhibit 25. SD-101 reduces MDSCs and Tregs. As demonstrated in prior work and again in the clinical work presented at SITC, the dual mechanism of action of SD-101 as a class C TLR9 agonist drives the reduction of immune suppressive MDSCs and Tregs while increasing key molecules such as granzyme B which is used by T cells to kill tumor cells and IL-15, which stimulates anti-tumor responses of both T cells and NK cells. The best results seem to be with the 2mg SD-101 dose, which is being selected for further enrollment for SD-101 + anti-PD-1.



Source. TriSalus Life Sciences, SITC 2023

Exhibit 26. Peripheral immune signatures for SD-101 via PEDD to liver mets. Even with low SD-101 exposure in the periphery, there is still increased pro-inflammatory cytokines and increased activation of both T cells and NK cells.

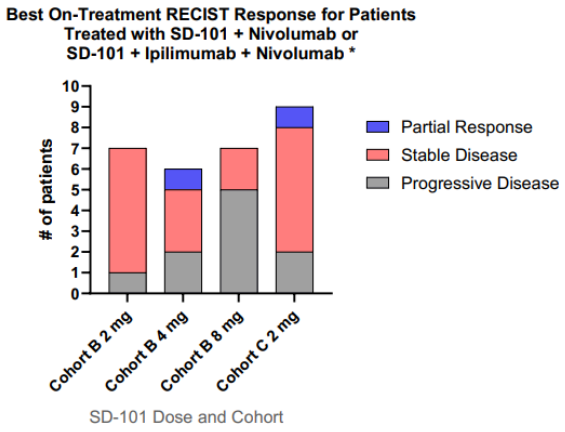


Source. TriSalus Life Sciences, SITC 2023

Efficacy signals in PERIO-01. Early efficacy signals were noted in patients treated in PERIO-01. Overall, the ctDNA (ct: circulating tumor) molecular response rate was 65% using specified time points (n=20), and 82% when analyzing the best on-treatment response (n=26). Clearance of ctDNA was noted in 59% of subjects when assessing the best on-treatment response. There was a 58% disease control rate (DCR) across all SD-101 doses (2mg, 4mg, 8mg). However, there was an 81% DCR at 2mg SD-101 via PEDD with anti-PD1 (n=7). Across all subjects, two partial responses (≥30% decrease) and five minor responses (10%–29% decrease) were documented as the best on-treatment response. The median progression-free survival at the optimal dose of SD-101 via PEDD (2mg) in combination with nivolumab was 11.7 months with a 1-year overall survival rate of 86% (n=7).

Exhibit 27. PERIO-01 efficacy. The combination of SD-101 PEDD and checkpoint therapy demonstrated a disease control rate (DCR) of 58%, which increased to 81% in the 2mg dose. The 2mg dose, as noted above, was selected as the optimal dose and there are plans to further enroll at 2mg SD-101 + anti-PD1.

- **58% DCR across all SD-101 doses** in combination with ICI
- Delayed responses noted in some subjects
- ***81% DCR at 2 mg***

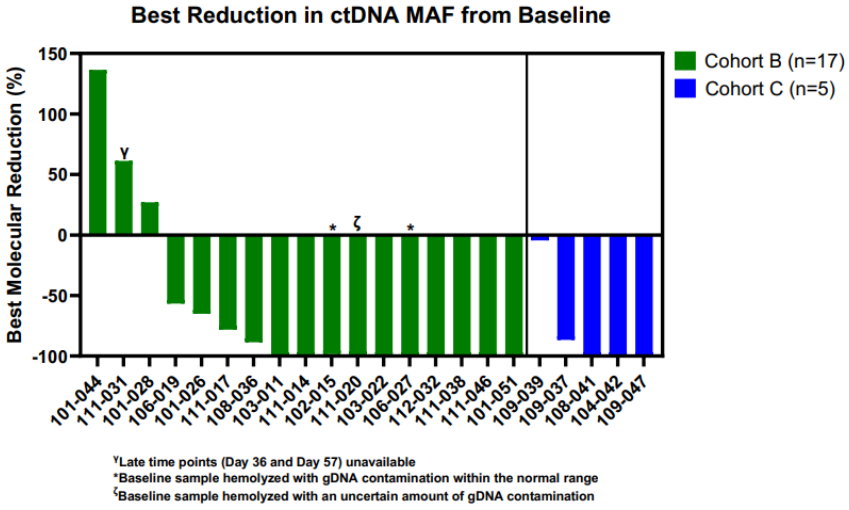


***5 SD subjects with 10-29% target lesion shrinkage**

DCR: Disease Control Rate
ICI: Immune Checkpoint Inhibitor
SD: Stable Disease

Source. TriSalus Life Sciences, SITC 2023

Exhibit 28. PERIO-01 efficacy, ctDNA reduction.



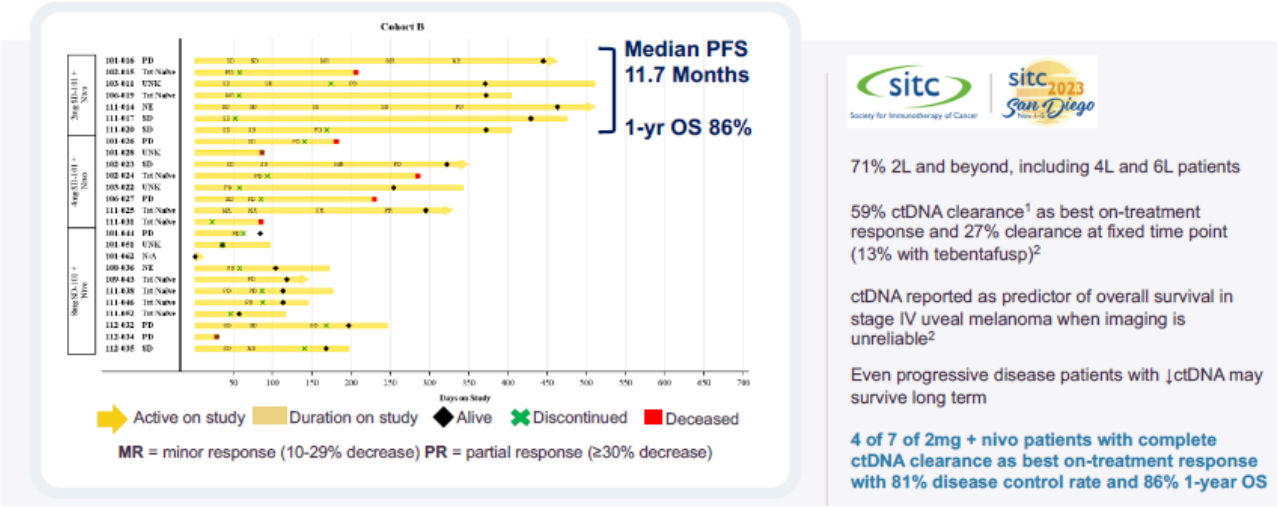
- **59%** ctDNA clearance
- **86%** reduction of any rate
- At 2 mg + nivo:
  - **5 of 7 with >50%** decrease in ctDNA
  - 2 patients with ctDNA clearance

ctDNA response correlates with overall survival consistent with published data[1,2,3]

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

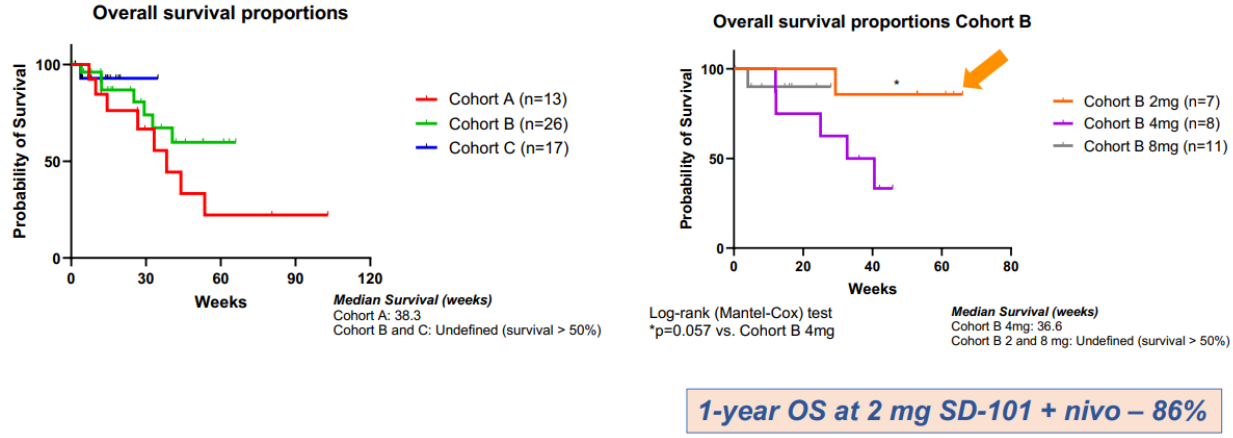
Source. TriSalus Life Sciences, SITC 2023

Exhibit 29. PERIO-01; Progression-free survival (PFS) and durable disease control. Shown below is cohort B across SD-101 PEDD 2mg, 4mg and 8mg groups, each with anti-PD1 therapy. Cohort C 2mg PR not shown due to timing. Next phase will further explore PFS conversion into OS benefit as ctDNA levels may predict.



Source. TriSalus Life Sciences, SITC 2023

Exhibit 30. Overall survival (OS) by cohort and dose level.



Source. TriSalus Life Sciences, SITC 2023

PERIO-02 (NCT05220722), Targeting hepatocellular carcinoma (HCC) and intrahepatic cholangiocarcinoma (ICC). This is an open-label P1b/2 study evaluating PEDD SD-101 alone or in combination with checkpoint inhibitor. All patients will receive two cycles of SD-101 where each cycle consists of three consecutive weekly infusions and cycle 1 and 2 were separated by one month. Escalating doses of SD-101 will be administered alone (cohort A), with pembrolizumab (Keytruda, cohort B) and with nivolumab/ipilimumab (Opdivo/Yervoy, cohort C). Cohort C will start with one dose below the maximum tolerated dose (MTD) or optimal dose from Cohort B. Following identifying the optimal dose and which checkpoint works best, the study will progress to P2.

PERIO-03 (NCT05607953), Targeting locally advanced pancreatic adenocarcinoma. This is an open-label P1/1b study evaluating PEDD SD-101 alone or in combination with checkpoint inhibitor. In the P1 portion, escalating doses of S-101 will be administered alone via pancreatic retrograde venous infusion, or PRVI. Recall that this delivery device is already cleared by the FDA, but unlike TriNav (for liver), is not commercialized yet as it's being developed with SD-101 in pancreatic cancer. The first three patients in the study will be part of a safety run-in and then MTD will be determined or the optimal dose. In P1b there will be the combination with PRVI SD-101 and pembrolizumab. Initial safety and feasibility data was presented at the SITC (Society for Immunotherapy of Cancer) in November 2023.

In the study, the PRVI infusion system is inserted into the portal venous system and then tracked into the pancreatic vein. Three patients were enrolled at the lowest SD-101 dose of 0.5mg. The infusions were well tolerated and there was demonstration of potentially favorable immune changes in the periphery and tumors. The single-agent dose escalation is still ongoing. At SITC the update was provided via poster presentation which can be viewed HERE.

MODELING ASSUMPTIONS

TriNav Delivery System

1. We assume there are 34,000 TASE/TARE procedures in the U.S. for liver cancer treatment with an annual growth rate ~2% y/y. These procedures take place in ~800 centers nationwide, though 70% occur in ~400 centers. The company, as of 2023, approximates sales in 17 centers with only 10 reps, and has plans to expand to 30–40+ reps, as well as 7–10 clinical specialists to work at the sites (training etc.) over 2023–2025. This is expected to enable targeting of the top-400 centers performing 70% of procedures.
2. Pricing is $7,750 per TriNav device, which is a premium to standard catheters used in TACE/TARE procedures at $800–$1,2000. Each device is only used once per procedure. TriSalus already has pricing and reimbursement (public and private) for TriNav in liver procedures. The company does not expect substantial price fluctuation and modest discounts for volume accounts were used, but not substantial.
3. We assume a CAGR of 35%–50% over the next 5 years with sales in 2023 in the range of $17M–$19M with margins ~80%. Growth is expected to accelerate in 2024 with the added reps and clinical specialists, with margin expansion over the next 5 years to the 90% range. We expect TriNav to reach break-even and be cashflow positive in 2025.
4. A revenue risk adjustment is factored in with 10% in 2024 and 2025, and growing to 15%–30% over out years.

SD-101 with TriNav Delivery, combination drug/device

1. We assume SD-101 TLR9 Class C agonist delivered via TriNav device will gain approval for uveal melanoma with liver metastases in 2028, followed by the larger indication in hepatocellular carcinoma (HCC) and liver metastases associated with colorectal cancer (CRC) in 2029 and 2030, respectively. Additionally, we assume SD-101 could gain approval in combination with a different Pressure Enabled Drug Delivery device (PEDD) in 2030.
2. We assume pricing for SD-101 in all indications to be ~$250,000 with y/y price increase of 3% and pricing of the PEDD or TriNav device separate from drug in the $7,750 range as with the TriNav pricing model currently.
3. For TriNav/PEDD device use with SD-101 therapy, the assumption is each patient would need 6–8 devices over the course of therapy and our model uses a median of 7 devices per patient.
4. A revenue risk adjustment of 80% is factored into the therapeutic models based on stage of development and clinical trial risks.

Exhibit 31. TriNav Infusion System Market Model (U.S.)

TriNav Infusion System, liver tumor procedures (US)	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E
TARE/TASE liver procedures per year (hospital-based)	34,000	34,680	35,374	36,081	36,803	37,539	38,290	39,055	39,836	40,633	41,446	42,275	43,120
TARE/TASE liver procedures per year (other centers)	7,000	7,140	7,283	7,428	7,577	7,729	7,883	8,041	8,202	8,366	8,533	8,704	8,878
Total TARE/TACE procudures	41,000	41,820	42,656	43,510	44,380	45,267	46,173	47,096	48,038	48,999	49,979	50,978	51,998
Market Penetration	3.20%	5.50%	10.00%	15.00%	20.00%	25.00%	30.00%	35.00%	38.00%	39.00%	40.00%	40.00%	40.00%
Total procedures using TriNav delivery	1,312	2,300	4,266	6,526	8,876	11,317	13,852	16,484	18,254	19,110	19,992	20,391	20,799
Cost of TriNav unit (one unit per patient procedure)	7,750	7,750	7,983	8,222	8,469	8,723	8,984	9,254	9,532	9,817	10,112	10,415	10,728
Increase in Cost	3%	3%	3%	3%	3%	3%	3%	3%	3%	3%	3%	3%	3%
Total revenue ('000)	$ 10,168	$ 17,826	$ 34,050	$ 53,660	$ 75,167	$ 98,713	$ 124,450	$ 152,538	$ 173,993	$ 187,607	$ 202,154	$ 212,383	$ 223,130
Risk adjustment	0%	0%	10%	10%	15%	15%	15%	20%	20%	25%	25%	30%	30%
Total Revenue ('000)	**$ 10,168**	**$ 18,242**	**$ 30,645**	**$ 48,294**	**$ 63,892**	**$ 83,906**	**$ 105,782**	**$ 122,030**	**$ 139,194**	**$ 140,705**	**$ 151,615**	**$ 148,668**	**$ 156,191**

Source: Maxim Estimates

Exhibit 32. SD-101/TriNav Combination, Liver Cancer and Liver Metastases Market Model (U.S.)

SD-101 + TriNav : Liver tumors (US)	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E
Uveal melanoma (UM)	3,000	3,060	3,121	3,184	3,247	3,312	3,378	3,446	3,515	3,585	3,657	3,730	3,805
UM w/liver metastases (45%)	1,350	1,377	1,405	1,433	1,461	1,491	1,520	1,551	1,582	1,613	1,646	1,679	1,712
Hepatocellular carcinoma (HCC) incidence	41,210	42,034	42,875	43,732	44,607	45,499	46,409	47,337	48,284	49,250	50,235	51,239	52,264
HCC, single tumor (60%)	24,726	25,221	25,725	26,239	26,764	27,300	27,845	28,402	28,970	29,550	30,141	30,744	31,359
Colorectal cancer (CRC) incidence	153,020	156,080	159,202	162,386	165,634	168,946	172,325	175,772	179,287	182,873	186,531	190,261	194,066
Colorectal cancer (CRC) w/ liver metastases	27,544	28,094	31,840	32,477	33,127	33,789	34,465	35,154	35,857	36,575	37,306	38,052	38,813
Market Penetration, UM liver mets							3.00%	5.00%	7.00%	10.00%	12.00%	15.00%	20.00%
Market Penetration, HCC								0.50%	1.00%	3.00%	5.00%	7.00%	10.00%
Market Penetration, CRC liver mets									1.00%	3.00%	5.00%	7.00%	10.00%
Total addressable patients							46	220	759	2,145	3,570	5,067	7,360
Cost of TriNav							$ 8,984	$ 9,254	$ 9,532	$ 9,817	$ 10,112	$ 10,415	$ 10,728
Number of TriNav devices per patient (6-9)							7	7	7	7	7	7	7
Cost of SD-101 therapy (excluding TriNav device)							$ 250,000	$ 257,500	$ 265,225	$ 273,182	$ 281,377	$ 289,819	$ 298,513
Increase y/y in SD-101 cost							3%	3%	3%	3%	3%	3%	3%
Cost of treatment per patient, total							$ 312,891	$ 322,277	$ 331,946	$ 341,904	$ 352,161	$ 362,726	$ 373,608
Total revenue ('000)							$ 14,271	$ 70,755	$ 251,947	$ 733,409	$ 1,257,154	$ 1,838,113	$ 2,749,606
Risk adjustment							80%	80%	80%	80%	80%	80%	80%
Total Revenue ('000)							**$ 2,854**	**$ 14,151**	**$ 50,389**	**$ 146,682**	**$ 251,431**	**$ 367,623**	**$ 549,921**

Source: Maxim Estimates

Exhibit 33. SD-101/PEDD Combination, Pancreatic Cancer Market Model (U.S.)

SD-101 + PEDD: Locally advanced pancreatic cancer (US)	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E
Pancreatic cancer incidence	64,050	65,331	66,638	67,970	69,330	70,716	72,131	73,573	75,045	76,546	78,077	79,638	81,231
Locally advanced PC (25%)	16,013	16,333	16,659	16,993	17,332	17,679	18,033	18,393	18,761	19,136	19,519	19,910	20,308
Market penetration									1.00%	3.00%	5.00%	10.00%	15.00%
Total addressable patients									188	574	976	1,991	3,046
Cost of TriNav									$ 9,532	$ 9,817	$ 10,112	$ 10,415	$ 10,728
Number of TriNav devices per patient (6-9)									7	7	7	7	7
Cost of SD-101 therapy (excluding TriNav device)									$ 265,225	$ 273,182	$ 281,377	$ 289,819	$ 298,513
Increase y/y in SD-101 cost									3%	3%	3%	3%	3%
Cost of treatment per patient, total									$ 331,946	$ 341,904	$ 352,161	$ 362,726	$ 373,608
Total revenue ('000)									$ 62,277	$ 196,285	$ 343,694	$ 722,170	$ 1,138,068
Risk adjustment									80%	80%	80%	80%	80%
Total Revenue ('000)									**$ 12,455**	**$ 39,257**	**$ 68,739**	**$ 144,434**	**$ 227,614**

Source: Maxim Estimates

VALUATION

We model sales of TriNav in liver tumor procedures with a revenue risk adjustment of 10%–30% based on commercial risk. We model SD-101 for liver tumors in UM, HCC, and colorectal cancer in 2028, 2029, and 2030, respectively, as well as pancreatic cancer in 2030, each with an 80% revenue risk adjustment based on stage of development and clinical trial risk. A 25% discount is applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month PT of $12.

Exhibit 34. Free Cash Flow Model.

Average	12

Price Target	13
Year	2024

DCF Valuation Using FCF (mln):

	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034
units ('000)	**2022E**	**2023E**	**2024E**	**2025E**	**2026E**	**2027E**	**2028E**	**2029E**	**2030E**	**2031E**	**2032E**	**2033E**	**2034E**
EBIT	-	(32,649)	(36,678)	(29,895)	(26,482)	(21,946)	(4,562)	15,111	69,011	175,514	300,218	464,044	703,214
Tax Rate	0%	0%	0%	0%	0%	0%	5%	5%	8%	12%	15%	20%	25%
EBIT (1-t)	-	(32,649)	(36,678)	(29,895)	(26,482)	(21,946)	(4,334)	14,356	63,490	154,452	255,185	371,235	527,411
CapEx	-	-	-	-	-	-	-	-	-	-	-	-	-
Depreciation	-	-	-	-	-	-	-	-	-	-	-	-	-
Change in NWC													
FCF	-	(32,649)	(36,678)	(29,895)	(26,482)	(21,946)	(4,334)	14,356	63,490	154,452	255,185	371,235	527,411
PV of FCF	-	(40,811)	(36,678)	(23,916)	(16,948)	(11,237)	(1,775)	4,704	16,643	32,391	42,813	49,826	56,630

Discount Rate	25%	
Long Term Growth Rate	3%	
Terminal Cash Flow	1,738,055	
Terminal Value YE2033	233,278	
NPV	304,920	
NPV-Debt		
Shares out ('000)	24,034	2033E
NPV Per Share	13	

Source: Maxim estimates

Exhibit 35. Discounted-EPS Model.

Current Year	2024
Year of EPS	2034
Earnings Multiple	5
Discount Factor	25%
Selected Year EPS	21.86
NPV	12

Source: Maxim estimates

	Discount Rate and Earnings Multiple Varies, Year is Constant						
	11.73	**5%**	**10%**	**15%**	**20%**	**25%**	**30%**
Earnings Multiple	**0**	0	0	0	0	0	0
	5	67.09	42.13	27.01	17.65	11.73	7.93
	10	134.18	84.27	54.03	35.30	23.47	15.85
	15	201.27	126.40	81.04	52.95	35.20	23.78
	20	268.36	168.53	108.05	70.60	46.94	31.71
	25	335.45	210.67	135.07	88.25	58.67	39.64
	30	402.54	252.80	162.08	105.90	70.41	47.56
	35	469.63	294.93	189.09	123.55	82.14	55.49

Exhibit 36. Sum-of-the-Parts Model.

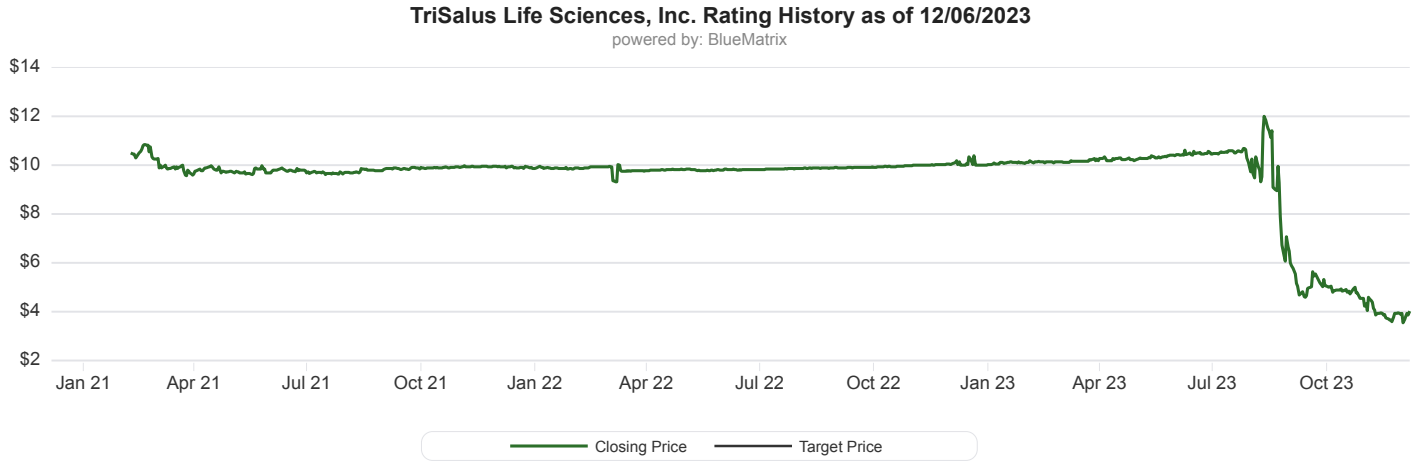
TriSalus Life Sciences	LT Gr	Discount Rate	Yrs to Mkt	% Success	Peak Sales (MM's)	Term Value
TriNav Infusion System, liver tumor procedures (US)	3%	15%	0	40%	$156	$1,302
NPV						$9.75
SD-101 + TriNav : Liver tumors (US)	3%	30%	6	25%	$550	$2,037
NPV						$2
SD-101 + PEDD: Locally advanced pancreatic cancer (US)	3%	30%	8	25%	$228	$843
NPV						$0.5
PEDD platform technology	2%	30%	8	25%	$50	$179
NPV						$0.1
Net Margin						45%
MM Shrs OS (2033E)						24
Total						$12

Source: Maxim estimates

TriSalus Life Sciences, TLSI.: Income Statement ($000)																			
YE December 31	1Q23A	2Q23A	3Q23A	4Q23E	2023E	1Q24E	2Q24E	3Q24E	4Q24E	2024E	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E
Revenue:																				
TriNav Infusion System, liver tumor procedures (US)	2,984	4,612	5,193	5,453	18,242	6,436	7,355	7,968	8,887	30,645	48,294	63,892	83,906	105,782	122,030	139,194	140,705	151,615	148,668	156,191
SD-101 + TriNav : Liver tumors (US)														2,854	14,151	50,389	146,682	251,431	367,623	549,921
SD-101 + PEDD: Locally advanced pancreatic cancer (US)																12,455	39,257	68,739	144,434	227,614
Net revenue	2,984	4,612	5,193	5,453	18,242	6,436	7,355	7,968	8,887	30,645	48,294	63,892	83,906	108,636	136,182	202,039	326,644	471,785	660,725	933,726
Collaborative revenue:																				
Revenues					-					-										
Other Income					-					-										
Total Collaborative Revenue	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Revenue	**2,984**	**4,612**	**5,193**	**5,453**	**18,242**	**6,436**	**7,355**	**7,968**	**8,887**	**30,645**	**48,294**	**63,892**	**83,906**	**108,636**	**136,182**	**202,039**	**326,644**	**471,785**	**660,725**	**933,726**
Gross Margins:																				
Cost of Goods Sold	662	772	589	600	2,623	965	1,103	1,195	1,333	4,597	7,244	7,667	8,391	10,864	13,618	20,204	32,664	47,179	66,072	93,373
%Gross Margin	78%	83%	89%	89%	89%	85%	85%	85%	85%	85%	85%	88%	90%	90%	90%	90%	90%	90%	90%	90%
Gross Profit	2,322	3,840	4,604	4,853	15,619	5,470	6,252	6,773	7,554	26,049	41,050	56,225	75,516	97,773	122,563	181,835	293,980	424,607	594,652	840,353
Operating Expenses:																				
Research and Development	5,642	6,862	9,367	4,000	25,871	4,500	4,750	5,000	5,200	19,450	23,340	30,342	42,479	44,603	46,833	49,175	51,633	54,215	56,926	59,772
Growth rate										10%	20%	30%	40%	5%	5%	5%	5%	5%	5%	5%
%R&D																				
Selling, General and Administrative	6,801	8,412	13,714	10,000	38,927	10,500	10,710	10,924	11,143	43,277	47,605	52,365	54,983	57,732	60,619	63,650	66,833	70,174	73,683	77,367
G&A										20%	10%	10%	5%	5%	5%	5%	5%	5%	5%	5%
%SG&A																				
Total Expenses	13,105	16,046	23,670	14,600	67,421	15,965	16,563	17,119	17,676	67,324	78,189	90,374	105,853	113,199	121,070	133,028	151,130	171,568	196,681	230,511
Operating Income (Loss)	(10,121)	(11,434)	(18,477)	(9,147)	(49,179)	(9,530)	(9,208)	(9,152)	(8,789)	(36,678)	(29,895)	(26,482)	(21,946)	(4,562)	15,111	69,011	175,514	300,218	464,044	703,214
					-															
Change in fair value of warrant liabilities	2,421	1,070	(2,812)		679															
Interest income	35	36	116		187															
interest expense	(5)	(4)	(4)		(13)															
Change in fair value of contingent earnout liability			19,904		19,904															
Loss on equity issuance	(585)	(3,604)			(4,189)															
Other income, expense (net)		(25)	(13)		(38)															
Total Other income	**1,866**	**(2,527)**	**17,191**	-	**16,530**	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Pretax Income	(8,255)	(13,961)	(1,286)	(9,147)	(32,649)	(9,530)	(9,208)	(9,152)	(8,789)	(36,678)	(29,895)	(26,482)	(21,946)	(4,562)	15,111	69,011	175,514	300,218	464,044	703,214
Taxes on income	5	(13)			(8)									(228)	756	5,521	21,062	45,033	92,809	175,804
Tax Rate														5%	5%	8%	12%	15%	20%	25%
GAAP Net Income (Loss)	(8,250)	(13,974)	(1,286)	(9,147)	(32,641)	(9,530)	(9,208)	(9,152)	(8,789)	(36,678)	(29,895)	(26,482)	(21,946)	(4,334)	14,356	63,490	154,452	255,185	371,235	527,411
Deemed dividend related to series B-2 preferred	(959)	(2,022)	(458)		(3,439)															
Total comprehensive loss	(9,209)	(15,996)	(1,744)	(9,147)	(36,080)	(9,530)	(9,208)	(9,152)	(8,789)	(36,678)	(29,895)	(26,482)	(21,946)	(4,334)	14,356	63,490	154,452	255,185	371,235	527,411
GAAP-EPS	(0.74)	(0.89)	(0.13)	(0.69)	(2.29)	(0.72)	(0.51)	(0.50)	(0.48)	(2.16)	(1.48)	(1.27)	(0.96)	(0.18)	0.61	2.67	6.48	10.66	15.45	21.86
GAAP-EPS (Dil)	(0.74)	(0.89)	(0.13)	(0.69)	(2.29)	(0.72)	(0.51)	(0.50)	(0.48)	(2.16)	(1.48)	(1.27)	(0.96)	(0.18)	0.61	2.67	6.48	10.66	15.45	21.86
Wgtd Avg Shrs (Bas) - '000s	12,526	18,057	13,173	13,187	14,236	13,200	18,213	18,231	18,249	16,973	20,172	20,880	22,840	23,559	23,653	23,748	23,843	23,938	24,034	24,131
Wgtd Avg Shrs (Dil) - '000s	12,526	18,057	13,173	13,187	14,236	13,200	18,213	18,231	18,249	16,973	20,172	20,880	22,840	23,559	23,653	23,748	23,843	23,938	24,034	24,131

Source: Company reports and Maxim

DISCLOSURES



Maxim Group LLC Ratings Distribution			As of: 12/06/23
		% of Coverage Universe with Rating	% of Rating for which Firm Provided Banking Services in the Last 12 months
Buy	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to outperform its relevant index over the next 12 months.	**82%**	**48%**
Hold	Fundamental metrics are currently at, or approaching, industry averages. Therefore, we expect this stock to neither outperform nor underperform its relevant index over the next 12 months.	**18%**	**56%**
Sell	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to underperform its relevant index over the next 12 months.	**0%**	**0%**
	**See valuation section for company specific relevant indices*		

I, **Jason McCarthy, Ph.D.**, attest that the views expressed in this research report accurately reflect my personal views about the subject security and issuer. Furthermore, no part of my compensation was, is, or will be directly or indirectly related to the specific recommendation or views expressed in this research report.

I, **Michael Okunewitch**, attest that the views expressed in this research report accurately reflect my personal views about the subject security and issuer. Furthermore, no part of my compensation was, is, or will be directly or indirectly related to the specific recommendation or views expressed in this research report.

The research analyst(s) primarily responsible for the preparation of this research report have received compensation based upon various factors, including the firm's total revenues, a portion of which is generated by investment banking activities.

Maxim Group makes a market in TriSalus Life Sciences, Inc.

Maxim Group expects to receive or intends to seek compensation for investment banking services from TriSalus Life Sciences, Inc. in the next 3 months.

TLSI: For TriSalus Life Sciences, we use the BTK (ARCA Biotechnology Index) as the relevant index

Valuation Methods
TLSI: We model commercialization of TriNav Delivery System for use in liver-related TARE/TASE procedures with a revenue risk adjustment based on commercial risk. We model SD-101 immune therapy delivered with TriNav in liver-related tumors primary and/or metastases) and Pressure-Enabled Drug Delivery (PEDD) in pancreatic cancer by indication (uvea melanoma, hepatocellular carcinoma, colorectal cancer and pancreatic cancer). A discount rate is then applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target.

Price Target and Investment Risks
TLSI: Aside from general market and other economic risks, risks particular to our price target and rating for TriSalus Life Sciences, Inc. include: (1) the regulatory and clinical risk associated with product development; (2) the ability to access capital and the very high likelihood that company will need to raise additional capital, the terms of which may not be favorable based on the outcome of commercial sales, clinical data and other factors, and if the company is unable to raise capital, this may hinder the company's ability to continue operations; (3) the rate and degree of progress of product development; (4) the rate of regulatory approval and timelines to potential commercialization of products in development; (5) the level of success achieved in clinical trials; (6) the requirements for marketing authorization from regulatory bodies in the United States and other countries; (7) the liquidity and market volatility of the company's equity securities; (8) regulatory and manufacturing requirements and uncertainties; (9) product and technology developments by competitors, potentially with more resources and commercial infrastructure; (10) inability, to reach profitability with currently commercialized products and other products, should they be approved, they may not gain adequate market share.

RISK RATINGS

Risk ratings take into account both fundamental criteria and price volatility.

Speculative – Fundamental Criteria: This is a risk rating assigned to early-stage companies with minimal to no revenues, lack of earnings, balance sheet concerns, and/or a short operating history. Accordingly, fundamental risk is expected to be significantly above the industry. Price Volatility: Because of the inherent fundamental criteria of the companies falling within this risk category, the price volatility is expected to be significant with the possibility that the investment could eventually be worthless. Speculative stocks may not be suitable for a significant class of individual investors.

High – Fundamental Criteria: This is a risk rating assigned to companies having below-average revenue and earnings visibility, negative cash flow, and low market cap or public float. Accordingly, fundamental risk is expected to be above the industry. Price Volatility: The price volatility of companies falling within this category is expected to be above the industry. High-risk stocks may not be suitable for a significant class of individual investors.

Medium – Fundamental Criteria: This is a risk rating assigned to companies that may have average revenue and earnings visibility, positive cash flow, and is fairly liquid. Accordingly, both price volatility and fundamental risk are expected to approximate the industry average.

Low – Fundamental Criteria: This is a risk rating assigned to companies that may have above-average revenue and earnings visibility, positive cash flow, and is fairly liquid. Accordingly, both price volatility and fundamental risk are expected to be below the industry.

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