UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): December 6, 2021

Sensei Biotherapeutics, Inc.
(Exact Name of Registrant as Specified in its Charter)

Delaware	**001-39980**	**83-1863385**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

1405 Research Blvd, Suite 125 **Rockville, MD** **(Address of Principal Executive Offices)**	**20850** **(Zip Code)**

Registrant's telephone number, including area code: (240) 243-8000

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Securities Exchange Act of 1934:

Title of each class	Trading symbol	Name of each exchange on which registered
Common Stock	**SNSE**	**The Nasdaq Stock Market LLC**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On December 6, 2021, Sensei Biotherapeutics, Inc. (the "**Company**") will be posting an updated corporate presentation on its website. A copy of the Company's presentation is furnished as Exhibit 99.1 to this Current Report on Form 8-K.

In accordance with General Instruction B.2. of Form 8-K, the information in this Item 7.01 and Exhibit 99.1 hereto shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "**Exchange Act**"), or otherwise subject to the liability of that section, nor shall it be deemed incorporated by reference in any of the Company's filings under the Securities Act of 1933, as amended, or the Exchange Act, whether made before or after the date hereof, regardless of any incorporation language in such a filing, except as expressly set forth by specific reference in such a filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Exhibit Description
99.1	Company Presentation.
104	The cover page from Sensei Biotherapeutics, Inc.'s Form 8-K filed on December 6, 2021, formatted in Inline XBRL.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Sensei Biotherapeutics, Inc.

Date: December 6, 2021

/s/ John Celebi
John Celebi
President and Chief Executive Officer

Exhibit 99.1

SENSEI BIOTHERAPEUTICS

Training the Immune System to Fight Cancer

December 6, 2021

NASDAQ: SNSE

Disclaimer

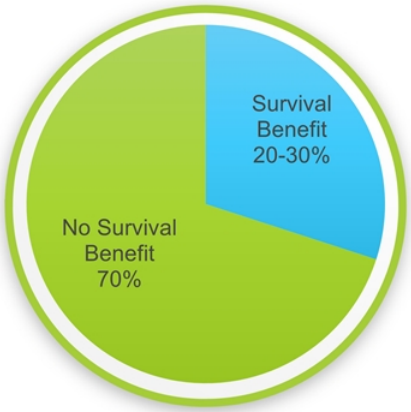
This presentation has been prepared by Sensei Biotherapeutics, Inc. (the “Company,” "we," "us") and is made for informational purposes only. The information set forth herein does not purport to be complete or to contain all of the information you may desire. Statements contained herein are made as of the date of this presentation unless stated otherwise, and neither the delivery of this presentation at any time, nor any sale of securities, shall under any circumstances create an implication that the information contained herein is correct as of any time after such date or that information will be updated or revised to reflect information that subsequently becomes available or changes occurring after the date hereof.

This presentation contains estimates and other statistical data made by independent parties and by us relating to market shares and other data about our industry. This presentation also contains "forward-looking" statements as that term is defined in the Private Securities Litigation Reform Act of 1995 that are based on our management's beliefs and assumptions and on information currently available to management. These forward-looking statements include, without limitation, statements regarding our industry, business strategy, plans, the preclinical and clinical development of our product candidates, and other financial and operating information. When used in this presentation, the words "may," "believes," "intends," "seeks," "anticipates," "plans," "estimates," "expects," "should," "assumes," "continues," "could," "will," "future" and the negative of these or similar terms and phrases are intended to identify forward-looking statements. Forward-looking statements involve known and unknown risks, uncertainties and other factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Risks and uncertainties that may cause actual results to differ materially include uncertainties inherent in the development of therapeutic product candidates, such as preclinical discovery and development, conduct of clinical trials and related regulatory requirements, our reliance on third parties over which we may not always have full control, and other risk and uncertainties that are described in our Annual Report on Form 10-K filed with the SEC on March 30, 2021 and our other Periodic Reports filed with the SEC. Forward-looking statements represent our management's beliefs and assumptions only as of the date of this presentation and include all matters that are not historical facts. Our actual future results may be materially different from what we expect. Except as required by law, we assume no obligation to update these forward-looking statements publicly, or to update the reasons actual results could differ materially from those anticipated in the forward-looking statements, even if new information becomes available in the future.

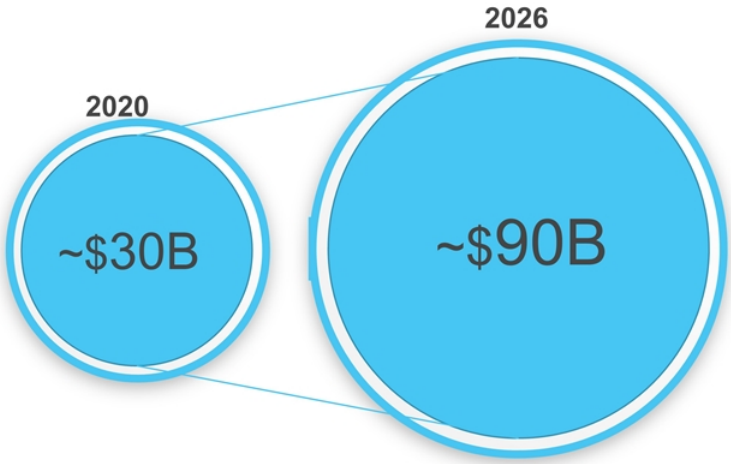
Certain information contained in this presentation relates to, or is based on, studies, publications, surveys and other data obtained from third-party sources and the Company's own internal estimates and research. While the Company believes these third-party sources to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while we believe our own internal research is reliable, such research has not been verified by any independent source.

The Modern-Day Challenge in Immuno-Oncology

Majority of patients don't respond to PD-1/PD-L1 monotherapy[1]

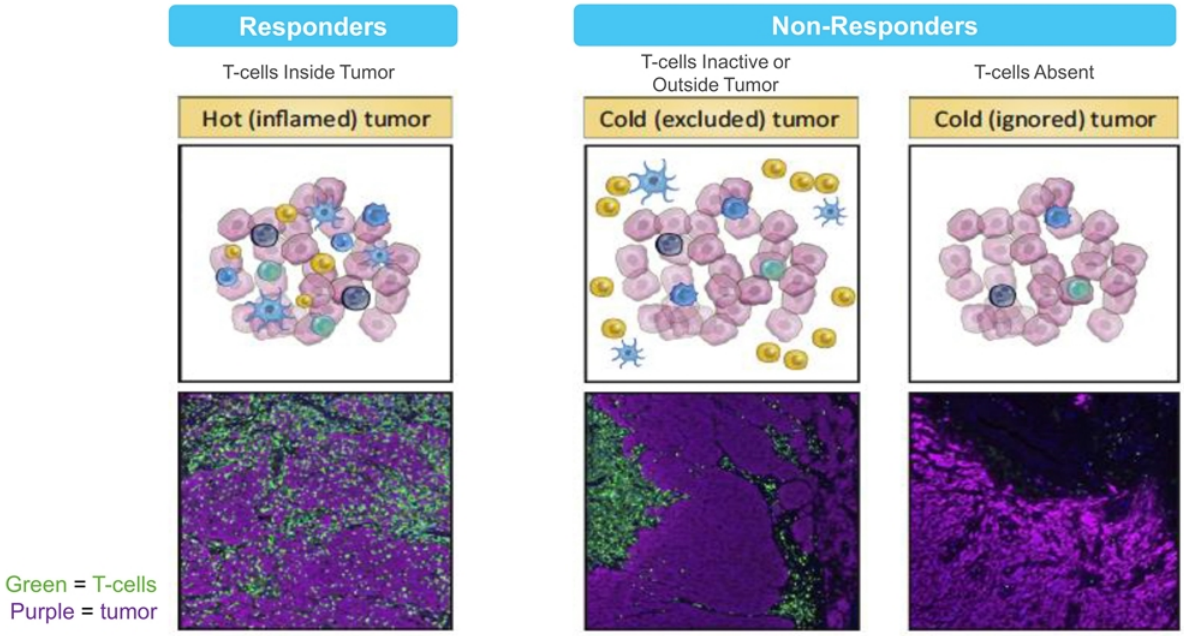


Global PD-1/PD-L1 Market[2]



1. Gerber et al., Biochemical Pharmacology 2016
2. Market estimates from PD-1 and PDL-1 Inhibitors Market Size in 2021 – MarketWatch, 360 Research

Two Major Types of Non-Responders to PD-1 Blockade

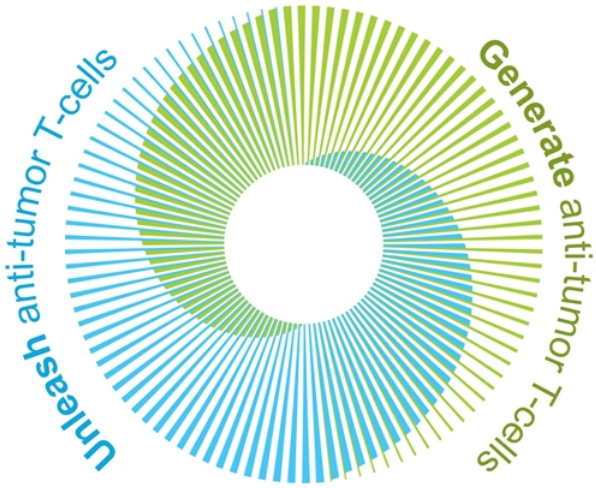


Adapted from Van der Woulde-LL, et al, Trends in Cancer, 2017

Two Platforms to Unleash Anti-Cancer T-cell Activity

TMAb™ (Tumor Microenvironment Activated Biologics) Platform

- Next-generation tumor activated mAbs
- Binding only in the low-pH tumor microenvironment
- Target checkpoints and/or other immune pathways
- Enable improved PK/PD and toxicity profiles

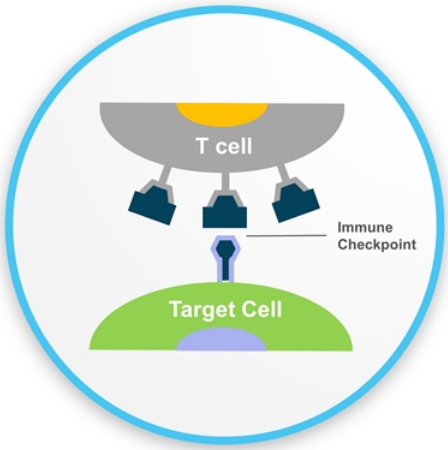


ImmunoPhage™ Platform

- Powerfully self-adjuvanted nanoparticle vaccine can drive B cell and T cell responses
- Multi-antigen vaccine enables personalized approach from “off-the-shelf” components
- Targets APCs
- Enhanced through addition of immunostimulatory nanobodies & cytokines

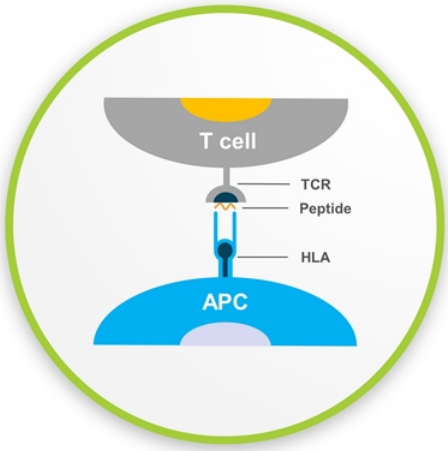
T-Cells Are Central to Our Approach and the Key to Unlocking Groundbreaking Clinical Activity

TMAb



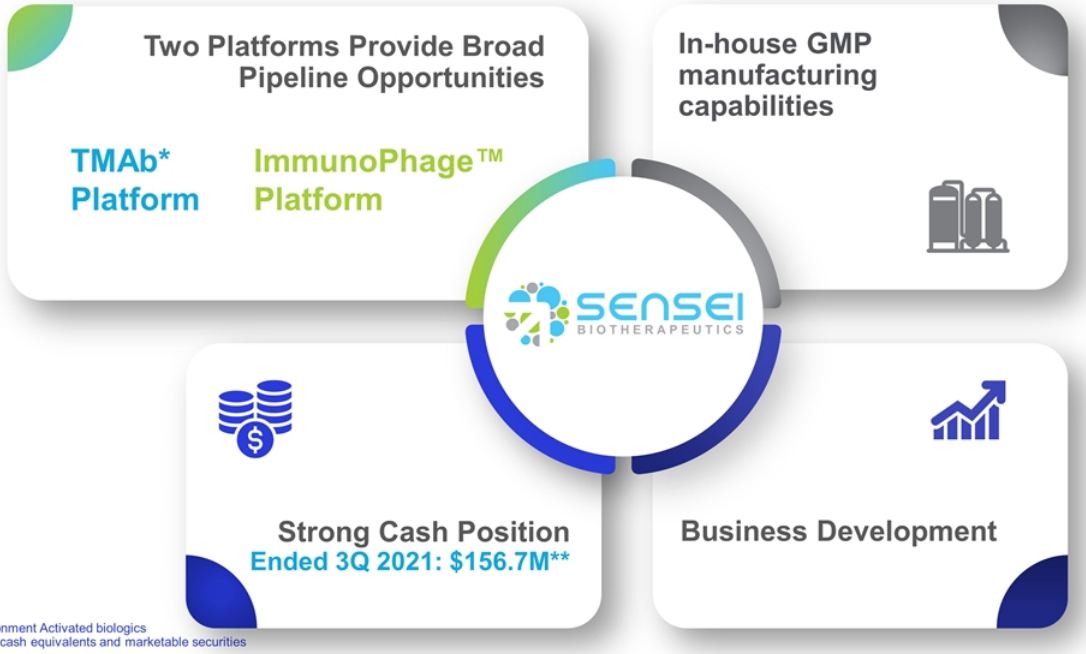
Focus on novel immune checkpoints to **UNLEASH** anti-tumor T-cells

ImmunoPhage™



Focus on multi-antigen approach for HLA-mediated immunotherapy to **GENERATE** anti-tumor T-cells

Positioned to Drive Value with Next Generation Product & Platform Development



*Tumor Microenvironment Activated biologics
**Consists of cash, cash equivalents and marketable securities

Pipeline Utilizing Pioneering ImmunoPhage Platform, TMAb Platform

	Program (Target)	Indication	Discovery	IND-enabling	Phase 1 / 2 Clinical
TMAb	SNS-101 (VISTA)	Solid Tumors			
TMAb	SNS-VSIG4	Solid Tumors			
ImmunoPhage	SNS-401-NG (Multiple Tumor Antigens)	Merkel Cell Carcinoma			
ImmunoPhage		Head and Neck Cancer			
ImmunoPhage		Lung Cancer			
ImmunoPhage		Melanoma			
ImmunoPhage		Breast Cancer			

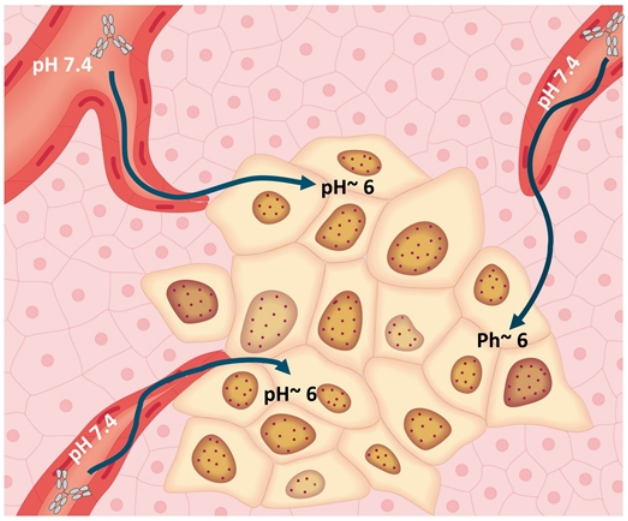
TMAb (Tumor Microenvironment Activated biologics) Platform

SENSEI BIOTHERAPEUTICS

pH-sensitive Antibodies Only Bind Their Targets in the Low-pH Tumor Microenvironment

TMAb PLATFORM

The tumor microenvironment of pH ~6 is lower than physiological pH of 7.4



Sensei's technology identifies pH-sensitive antibodies that bind only at the tumor

- Antibodies that bind at physiological pH may encounter a "sink"
  - Prevents effective binding at the tumor and may lead to toxicity
- TMAb antibodies bypass tissue compartments other than the low-pH tumor microenvironment
- Potential for improved safety and clinical activity profile

The Promise and Challenge of Immunotherapy

Targeting Immunosuppressive myeloid cells is a promising strategy to overcome resistance to checkpoint Inhibitor therapy

THE PROMISE

- Using the body's own immune system to attack cancer
- Capitalizing on immunological specificity and long-term memory
- Achieving durable cures with minimal toxicity

THE CHALLENGE

- 70-80% of patients do not achieve increased survival with CPI monotherapy[1]
- The immunosuppressive tumor microenvironment (TME) influences response to immune checkpoint blockade
- Innate immune cells such as myeloid cells are a key driver of immunosuppressive TME

1 Gerber, et al Biochemical Pharmacology 2016

VISTA: An Emerging Checkpoint Target on Myeloid Cells

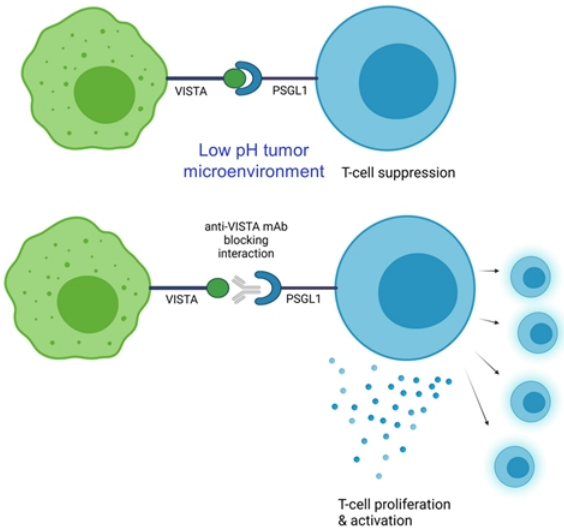
Target Overview:

- Large market opportunity
- B7 family ligand
- Extensive expression on myeloid cells[1]
- Inhibition of VISTA may lead to activation of myeloid cells
- Excellent therapeutic combinability with CTLA-4 or PD-1/PD-L1 ICIs, especially in cold tumors[2]
- VISTA expression correlates with poor survival rates across multiple cancers
- Novel development program with no approved therapies

Sensei's Competitive Advantage:

- Extensive understanding of VISTA biology and differentiated candidate antibody

VISTA is a Negative Regulator of T cell Function



1 Lines et al. Cancer research vol. 74,7 (2014)
2 Gao et al. Nature medicine vol. 23,5 (2017)

Increased Understanding of VISTA as a Promising Target to Address the Needs of Patients with Cancer

BRIEF COMMUNICATIONS

nature medicine

VISTA is an inhibitory immune checkpoint that is increased after ipilimumab therapy in patients with prostate cancer

Jianjun Gao[1], John F Ward[2], Curtis A Pettaway[3], Lewis Z Shi[4], Sumit K Subudhi[5], Luis M Vence[6], Hao Zhao[5], Jianfeng Chen[4], Hong Chen[5], Ileni Efstathiou[5], Patricia Troncoso[7], James P Allison[5,8], Christopher J Logothetis[3], Ignacio I Wistuba[6], Manuel A Sepulveda[2], Jingjing Sun[9], Jennifer Wargo[6], Jorge Blando[5] & Padmanee Sharma[3,5,8]

To date, anti-CTLA-4 (ipilimumab) or anti-PD-1 (nivolumab) monotherapy has not been demonstrated to be of substantial clinical benefit in patients with prostate cancer. To identify additional immune-inhibitory pathways in the prostate-tumor microenvironment, we evaluated untreated and ipilimumab-treated tumors from patients in a presurgical clinical trial. Levels of the PD-L1 and VISTA inhibitory molecules increased on independent subsets of macrophages in treated tumors. Our data suggest that VISTA represents another compensatory inhibitory pathway in prostate tumors after ipilimumab therapy.

Immune checkpoint therapies, including anti-CTLA-4 and anti-PD-1 therapies, that block T cell inhibitory pathways have led to durable antitumor responses and clinical benefit in a substantial number of patients with cancer[1,2]. However, prostate cancer has proven to be poorly responsive to immune checkpoint monotherapy[3–5]. To better understand the immune profile within prostate tumors and potential compensatory immune inhibitory pathways that may arise in the setting of immune checkpoint monotherapy, we conducted a clinical trial (NCT01194271) with ipilimumab plus androgen deprivation therapy (ADT) before surgery in patients with localized prostate cancer (**Supplementary Fig. 1a–c** and **Supplementary Tables 1** and **2**).

We compared post-treatment and baseline blood samples (**Supplementary Fig. 1a**), evaluating the levels of $CD4^+$ and $CD8^+$ T cells (**Supplementary Fig. 2a**), as well as those of T cell subsets expressing inducible costimulator (ICOS), OX40, 4-1BB, PD-1, CTLA-4, and FoxP3 (**Supplementary Fig. 2a,b**). We observed an increase in $CD4^+$ and $CD8^+$ T cells, including $PD\text{-}1^+$ and $ICOS^+$ subsets, after ipilimumab therapy, which is similar to our previous findings with ipilimumab monotherapy in patients with melanoma and bladder cancer[6–8]. We also compared post-treatment tumor tissues (**Supplementary Fig. 1a**) to those of stage-matched untreated tumors from another cohort of patients (**Supplementary Fig. 1b**). Flow cytometric studies revealed a significantly higher frequency of $CD4^+$, $CD8^+$, and $ICOS^+$ T cells in the post-treatment tumors (**Fig. 1a**). Immunohistochemical (IHC) studies also demonstrated significant increases in tumor-infiltrating immune cells, including $CD4^+$, $CD8^+$, $ICOS^+$, $CD45RO^+$, granzyme-B (GrB^+), and $CD68^+$ cells (**Supplementary Fig. 3**). We found significantly greater immune cell infiltration in prostate tumors after ipilimumab therapy but not after ADT alone, although ADT monotherapy was associated with significantly higher levels of $ICOS^+$ and GrB^+ cells, which may represent an activated T cell subset (**Fig. 1b**). Taken together, our data suggest that the immunologic changes in post-treatment tumors were mostly due to ipilimumab therapy, as opposed to ADT. However, we cannot discount a possible synergistic effect between ipilimumab and ADT.

We did not observe clinical responses consisting of pathologic complete response, as we did previously for patients with bladder cancer[6]. To identify potential mechanisms that might explain this lack of response, we performed an unbiased gene expression study and found that ipilimumab therapy resulted in significant changes in the expression of a total of 698 genes (false discovery rate (FDR) < 0.2, $P < 0.028$, $\log_2$ (fold change) > 0.5)(**Supplementary Table 3**), most of which are related to immune responses (**Supplementary Fig. 4a**). We focused our analyses on a subset of genes that represent inhibitory immune checkpoints and identified increased PD-L1 and VISTA expression in post-treatment tumors (**Supplementary Fig. 4b**). Both PD-L1 and VISTA were previously reported as inhibitory molecules that can suppress murine and human T cell responses[9,10]. Here we found significantly greater protein expression of PD-1, PD-L1, and VISTA in prostate tumors after ipilimumab therapy (**Fig. 1c** and **Supplementary Fig. 5a**).

We also evaluated metastatic tumors and blood samples from patients with metastatic prostate cancer who took part in a separate clinical trial (NCT02113657) and received treatment with ipilimumab, finding an increase in PD-L1 and VISTA expression in tumor tissues (**Supplementary Fig. 5b**) as well as on monocytes in blood (**Supplementary Fig. 6a**), which was similar to data from a mouse model of prostate cancer (**Supplementary Fig. 6b**). We suggest that PD-L1 and VISTA are likely to be relevant inhibitory immune checkpoints in both localized and metastatic prostate cancer.

We evaluated PD-L1 and VISTA expression in different cell subtypes from matched pre- and post-treatment prostate tumors and observed significantly higher PD-L1 expression on $CD4^+$ T cells, $CD8^+$ T cells, and $CD68^+$ macrophages after treatment (**Supplementary Fig. 7a**).

[1]Department of Genitourinary Medical Oncology, University of Texas MD Anderson Cancer Center, Houston, Texas, USA. [2]Department of Urology, University of Texas MD Anderson Cancer Center, Houston, Texas, USA. [3]The Immunotherapy Platform, University of Texas MD Anderson Cancer Center, Houston, Texas, USA. [4]Department of Pathology, University of Texas MD Anderson Cancer Center, Houston, Texas, USA. [5]Department of Immunology, University of Texas MD Anderson Cancer Center, Houston, Texas, USA. [6]Department of Translational Molecular Pathology, University of Texas MD Anderson Cancer Center, Houston, Texas, USA. [7]Janssen Oncology Therapeutic Area, Janssen Research and Development, LLC, Pharmaceutical Companies of Johnson & Johnson, Spring House, Pennsylvania, USA. [8]Department of Surgical Oncology, University of Texas MD Anderson Cancer Center, Houston, Texas, USA. Correspondence should be addressed to P.S. (psharma@mdanderson.org).

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Trends in Immunology

CellPress OPEN ACCESS

Feature Review

VISTA: A Mediator of Quiescence and a Promising Target in Cancer Immunotherapy

Long Yuan,[1,2] Jahnavi Tatineni,[2] Kathleen M. Mahoney,[2,3] and Gordon J. Freeman[2,*]

V-domain Ig suppressor of T cell activation (VISTA) is a B7 family member that maintains T cell and myeloid quiescence and is a promising target for combination cancer immunotherapy. During inflammatory challenges, VISTA activity reprograms macrophages towards reduced production of proinflammatory cytokines and increased production of interleukin (IL)-10 and other anti-inflammatory mediators. The interaction of VISTA with its ligands is regulated by pH, and the acidic pH ~6.0 in the tumor microenvironment (TME) facilitates VISTA binding to P-selectin glycoprotein ligand 1 (PSGL-1). Targeting intratumoral pH might be a way to reduce the immunoinhibitory activity of the VISTA pathway and enhance antitumor immune responses. We review differences among VISTA therapeutics under development as candidate immunotherapies, focusing on VISTA binding partners and the unique structural features of this interaction.

VISTA: How This B7 Protein Might Transform Cancer Immunotherapy

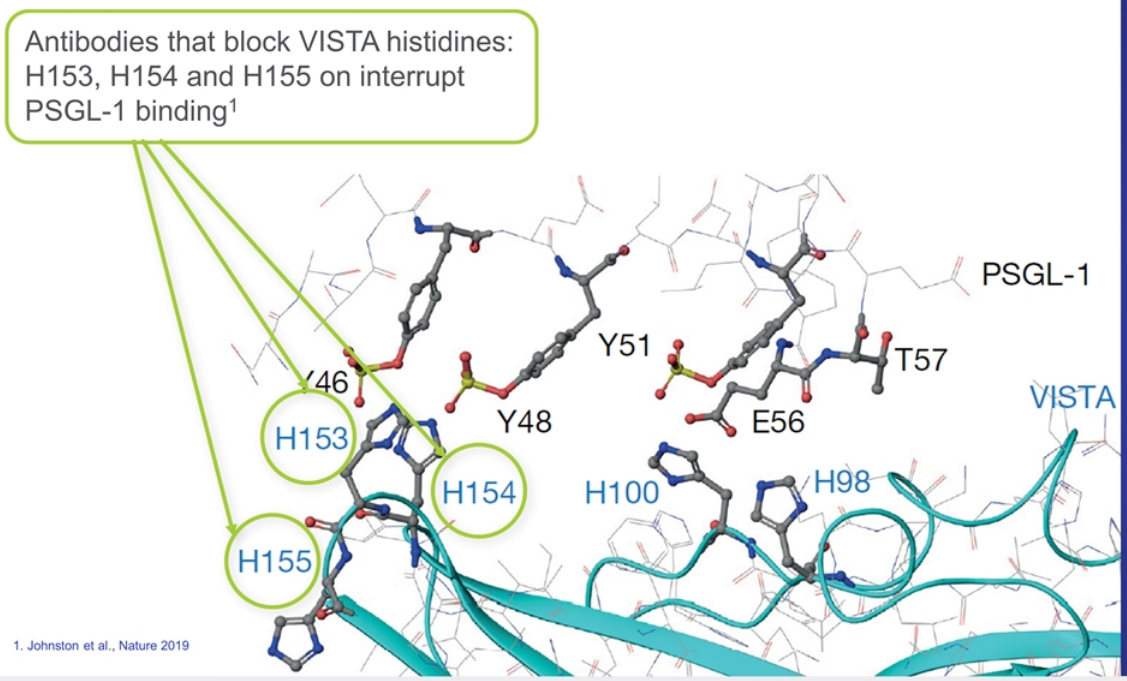
Immunotherapy has become an established pillar of cancer treatment, in large part owing to the success of blocking the **programmed cell death protein 1 (PD-1)/ programmed death-ligand 1 (PD-L1) immune checkpoint** (see Glossary) pathway. As recent research deepens our understanding of V-domain Ig suppressor of T cell activation (VISTA), the VISTA signaling pathway has increasingly become a promising target for overcoming resistance to current immune checkpoint therapies [1]. Although the development of VISTA blocking antibodies has not reached fruition clinically, this review highlights the new features of VISTA that make this pathway particularly attractive for therapeutic development. We discuss (i) VISTA expression on immune cells in the tumor microenvironment (TME), (ii) the biological functions and bidirectional signaling pathways of VISTA in mammalian lymphocytes and myeloid cells, (iii) the structural features of VISTA that contribute to its molecular interactions, (iv) current VISTA monoclonal antibodies (mAbs) that are in clinical development, and (v) the candidate druggable targets that regulate the pH of the TME and which in turn might affect VISTA activity *in vivo*. This review gives a detailed picture of VISTA structure in the context of its binding partners and therapeutic antibodies targeting VISTA.

VISTA Structure

VISTA, also known as PD-1H, B7-H5, Dies1, Gi24, DD1α, and C10orf54, is encoded by the *VSIR* gene in human (*Vsir* in mouse) and has multiple unique features, including its interaction with two receptors that bind to overlapping but distinct sites on the VISTA extracellular domain (ECD) [2–4]. VISTA is a type I transmembrane protein that was identified by mRNA analysis of activated versus resting mouse natural **regulatory T cells (Tregs)** [5] and also by homology to coinhibitory molecules such as **PD-1** [6]. VISTA bears features of both the B7 and CD28 families of immunoregulatory molecules and can act as both a ligand and a receptor [3,7,8]. The VISTA ECD is most homologous to the B7 family, which includes well-known immune checkpoint ligands such as **PD-L1** (Figure 1C). Whereas other B7 family members have an IgV-like and IgC-like domain, mouse and human VISTA contain a single unusually large IgV-like domain (Figure 1A) [2]. VISTA

[1]Program in Immunology, Harvard Medical School, Boston, MA 02115, USA
[2]Department of Medical Oncology, Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA 02215, USA

VISTA Checkpoint is Activated at the Low pH of the Tumor Microenvironment



1. Johnston et al., Nature 2019

VISTA's extracellular domain is uniquely rich in histidines[1]

Histidines are protonated at low pH enabling VISTA to distinguish the active (acidic pH) and inactive (neutral pH) PSGL-1 binding interface

Key to Unlocking the Power of VISTA

1. Block VISTA's interaction with PSGL-1 at pH 6 within the tumor microenvironment

2. Selectively bind VISTA at low pH to avoid:
   - target mediated drug disposition
   - on-target/off-tumor side effects

3. Design an Fc-competent IgG engaging with FcɣR on tumor-infiltrating myeloid cells

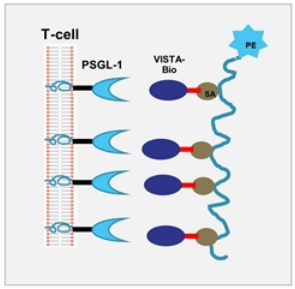
SNS-101 Inhibited Interaction of VISTA to its Receptor, PSGL-1, in CD4/CD8 T-Cells at Low pH 6.0

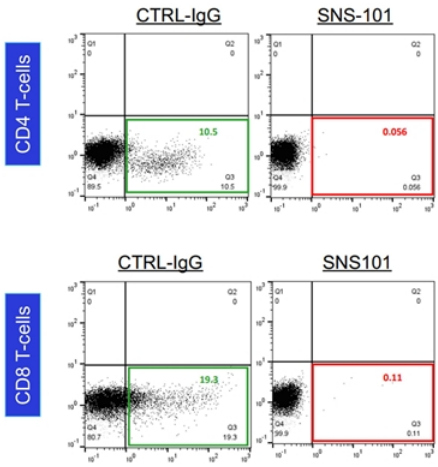
SENSEI BIOTHERAPEUTICS

SNS-101:

- Fully human monoclonal antibody that selectively binds active (low pH) VISTA, but not inactive VISTA in the blood
- Potent inhibitor of PSGL-1 binding to VISTA
- Fc-competent framework to deliver positive “kick” to suppress myeloid cells in the tumor microenvironment

PSGL-1: VISTA Interaction on primary T-cells at pH 6.0



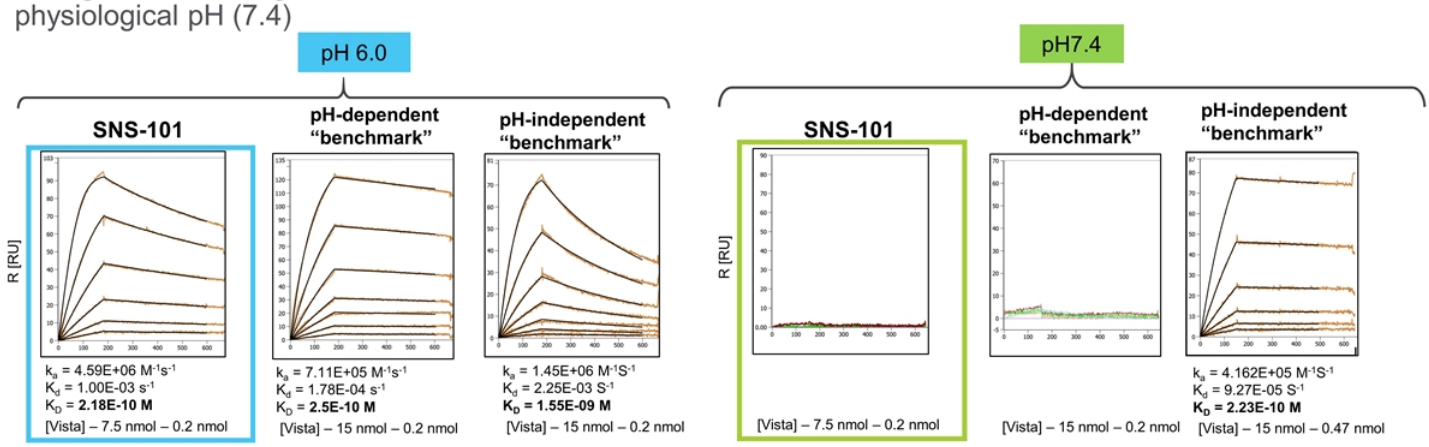


IND-Enabling Studies are Underway for SNS-101

SNS-101 Has >600-Fold Selectivity for VISTA^{pH6}

- Biophysical characterization demonstrates >600-fold selectivity for VISTA at pH 6.0
- Picomolar binding at low pH
- No significant binding observed at physiological pH (7.4)

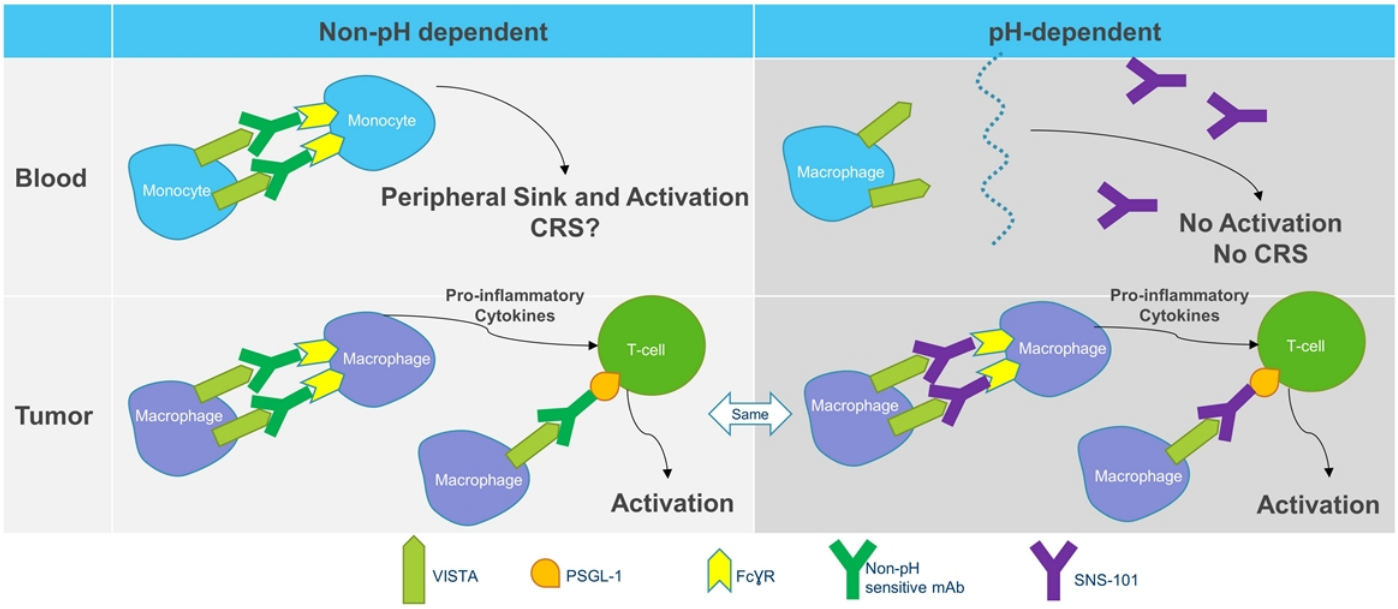
	pH 6.0	pH 7.4
Monovalent Affinity (K_D) [nM]	0.218	132 (~No binding)



SITC 2021: Poster titled: Antagonistic pH-selective VISTA antibody SNS-101 potentiates anti-PD-1/PD-L1-induced anti-tumor immunity

Proposed Mechanism of Action for SNS-101

Fc-competent framework is required for optimal activity, but FcγR engagement in the blood may result in untoward "off tumor" activation (i.e. CRS)



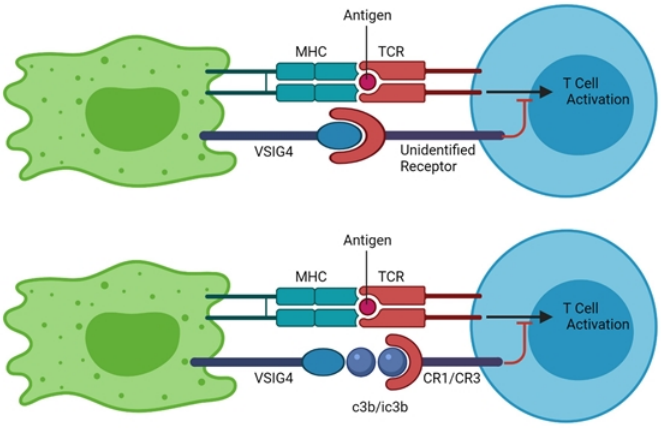
SNS-101 Is a Differentiated Anti-VISTA Antibody

TMAb Platform

	SNS-101	VISTA.18 (BMS)	KVA12.1 (Kineta)	CI-8993; JNJ-61610588 (J&J/Curis)	K01401-020; W0180 (Pierre Fabre)	HMBD-002 (Hummingbird)
Inhibit PSGL-1 Binding	Yes	Yes	unknown	Yes	unknown	unknown
pH Sensitive Binding	Yes	Yes	No	No	No	No
Fc Active	Yes (IgG1)	No (IgG4)	Yes (IgG1)	Yes (IgG1)	N/A	No (IgG4)
Stage	Preclinical	Preclinical	Preclinical	Phase I	Phase I	IND submission
Clinical Data / Notes	• Preclinical data presented at STIC • IND-enabling studies underway	• N/A	• N/A	• JNJ initiated Phase I study in 2016 • 12 pts enrolled; initial dose 0.005 mg/kg • Only patient treated at 0.3 mg/kg experienced grade 3 CRS-associated encephalopathy; trial was halted	• Ongoing	

Johnston et al, Nature, 2019; Kineta website; Snyder et al, AACR Annual Meeting 2016; Pierre Fabre website; Hummingbird website

VSIG4: A Novel Next Generation Checkpoint Modulating the Tumor Microenvironment



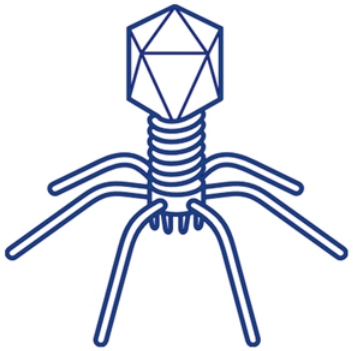
No approved therapies against VSIG4

Adapted from Zang et al., J Clin Invest. 2006

- Second TMAb program
- B7 family related protein
- Expressed on macrophages
- Inhibits T-cell activation
- Novel therapeutic combinability with existing IO drugs

ImmunoPhage™ Platform

Bacteriophage

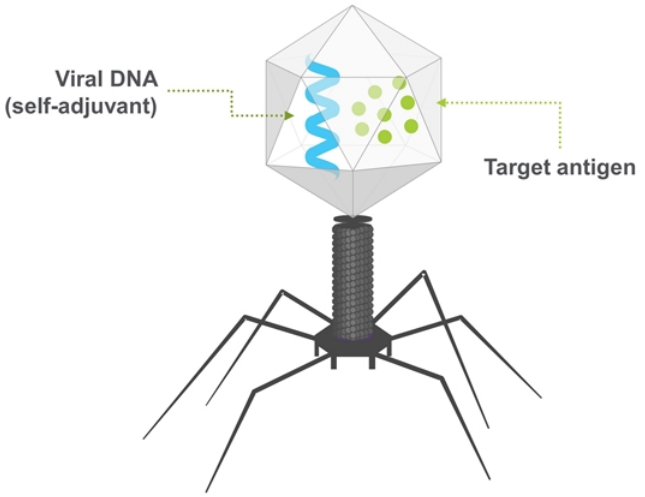


Ubiquitous viruses that infect bacteria but not mammalian cells. Adept at activating the human immune system in multiple unique ways

Generating Strong Antibody and T-cell Responses

ImmunoPhage Platform

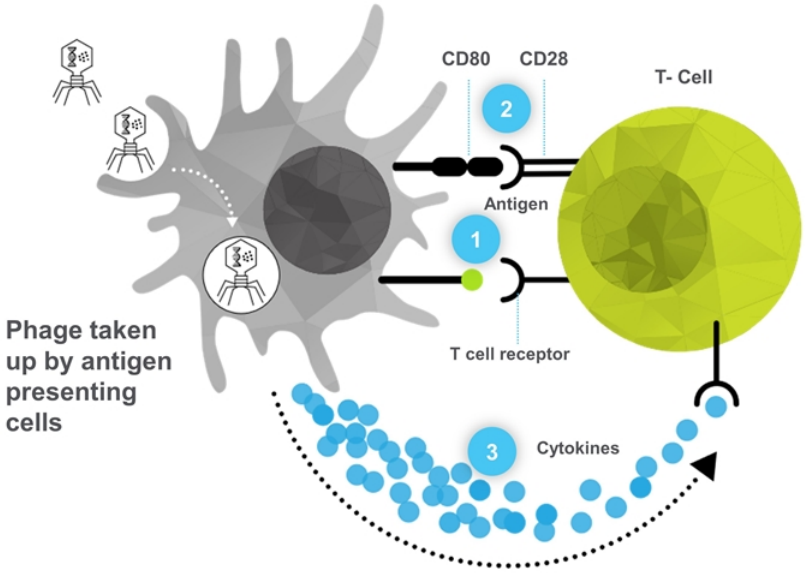
Bacteriophage virus is engineered and manufactured with both antigen and immune stimulatory viral DNA



The **ImmunoPhage™** bacteriophage is an icosahedron with a tail. This configuration can be viewed as an activating signal to the immune system

Generating Strong Antibody and T-cell Responses

***ImmunoPhages* are taken-up by APCs and deliver three critical signals required to drive activation of T cells.**



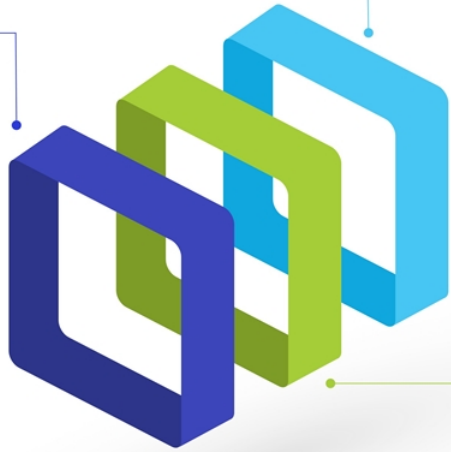
1. Antigen cross presentation
2. Positive co-stimulation
3. Generation of Th1-biased immune response & cytokines

ImmunoPhage™ A Multi-Pronged Approach to Address the Complexities of Cancer

Our **ImmunoPhages** can mount a multi-modal attack on cancer, combining the benefits of a traditional vaccine with localized gene therapy

Targeted therapeutic vaccine

- MHC-mediated immunity
- Bacteriophage have natural tropism for APCs
- Can be further targeted to APCs with non-antigen capsid modifications



Phortress™ library

- Personalized - yet off the shelf - medicines
- Pre-manufactured cost effectively - then combined based on genetic profile

Gene therapy vehicle

- Phage containing self-replicating RNA
- Used to deliver payloads consisting of immunomodulatory proteins or nanobodies

SNS-401-NG: Building the First Custom Merkel Cell Polyoma Virus (MCPyV) ImmunoPhage

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SNS-401-NG Development

Collaboration with University of Washington to build **first custom Merkel Cell Carcinoma (MCC) vaccine consisting of Merkel Cell Polyoma Virus epitopes** and other patient specific antigens

MCC is a rare, aggressive neuroendocrine skin cancer

- 33-46% disease-specific mortality
- 2,500 cases/yr with disease-specific mortality approaching 50%
- Vaccine combination therapy in adjuvant or neoadjuvant is attractive and feasible
  - PD-1/PD-L1 refractory MCC remains unmet medical need with aggressive clinical course
  - ~40% MCC patients recur <24 months following definitive local treatment

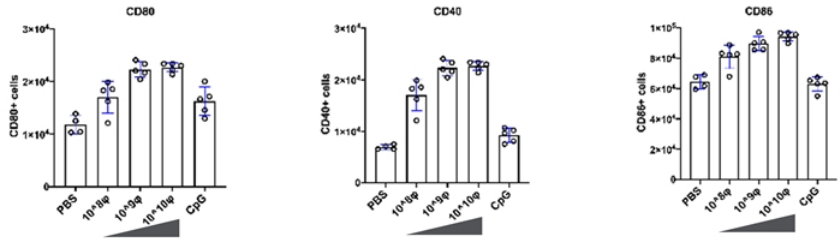
Integration of MCPyV is present in ~80% of U.S. cases

- In these cases, expression of a viral antigen (oncogenic T-antigen) **appears to be a strictly required tumor driver**
- Researchers at UW have mapped MCPyV epitopes and **determined CD8 T-cell, CD4 T-cell, and B-cell epitopes that are antigenic** in the context of MCPyV+ MCC tumors.

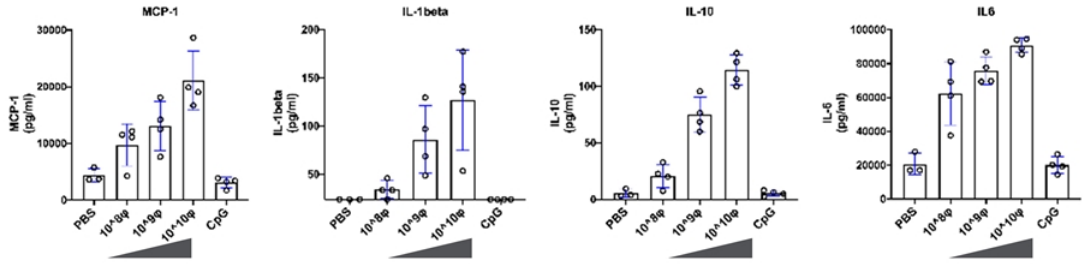
Mechanism of Action: Activation and Maturation of Dendritic Cells

Dose-response of engineered lambda phage on human skin-derived DC cultures

Signal: Dendritic cell co-stimulatory molecules



Signal: Cytokine secretion

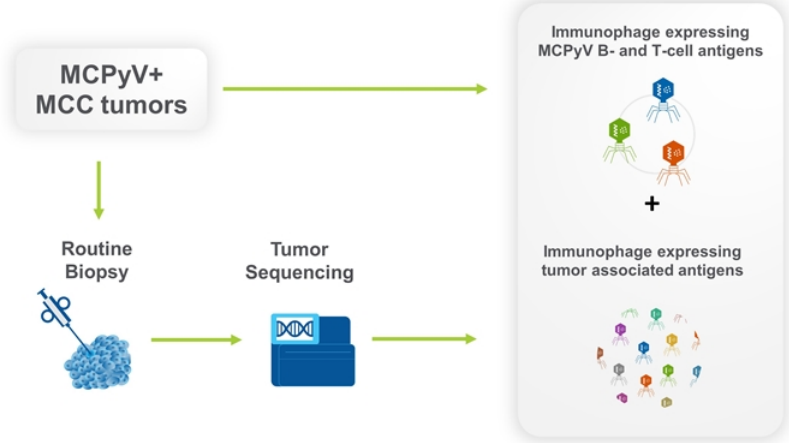


Critical signals of dendritic cell activation show dose-dependent increases when cells are exposed to increasing amounts of ImmunoPhages

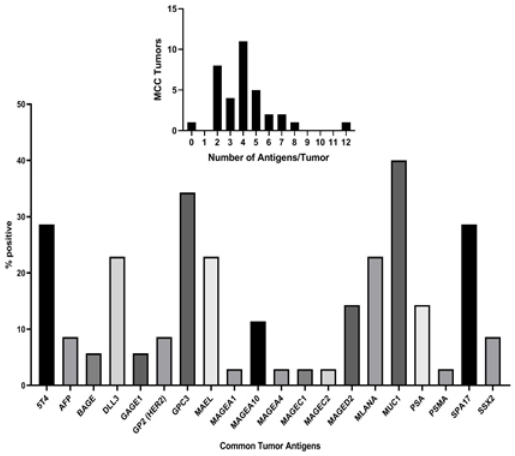
SNS-401-NG has Potential to be First Fully Customized, Yet Off-the-Shelf, Therapy

SNS-401-NG Development in Merkle Cell

Patients would receive a bespoke mixture of ImmunoPhage that included antigens from the MCPyV and a subset of TAA-expressing ImmunoPhage



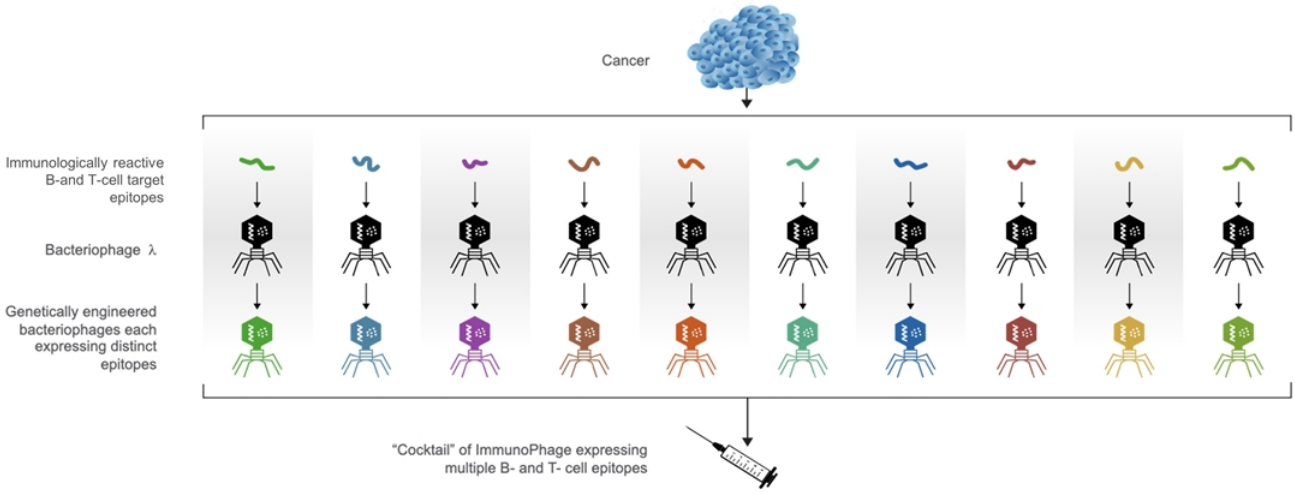
Most MCC tumors contain multipleTAAs[1]



Common Tumor Antigens

1. Based on internal data

Phortress: Proprietary Library of Personalized Vaccine Cocktails with Off-the-Shelf ImmunoPhage “ingredients”



- These “cocktails” are defined by the disease or patient genetics
- Combinations are customized to cover multiple epitopes, protein domains or targets
- Each *ImmunoPhage* is pre-manufactured to target a discrete antigen

Personalized Immunotherapy Approach Could Accelerate Speed to Treatment

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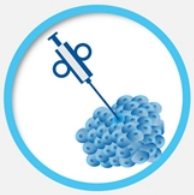
High speed and low cost-of-goods of ImmunoPhage allows a broader array of antigens

Personalized yet Off-the Shelf TAA Therapy

Off-the-Shelf + Patient-specific Neoantigen Therapy

Routine Biopsy



Clinical biopsy of tumor as input material

Tumor Sequencing

Tumor DNA
Tumor RNA
Normal DNA

Personalized yet Off-the-shelf ImmunoPhage Cocktail

Assemble a personalized cocktail from off-the-shelf TAA ImmunoPhage for administration

Neoantigen Prediction

Identify additional tumor specific neoantigens

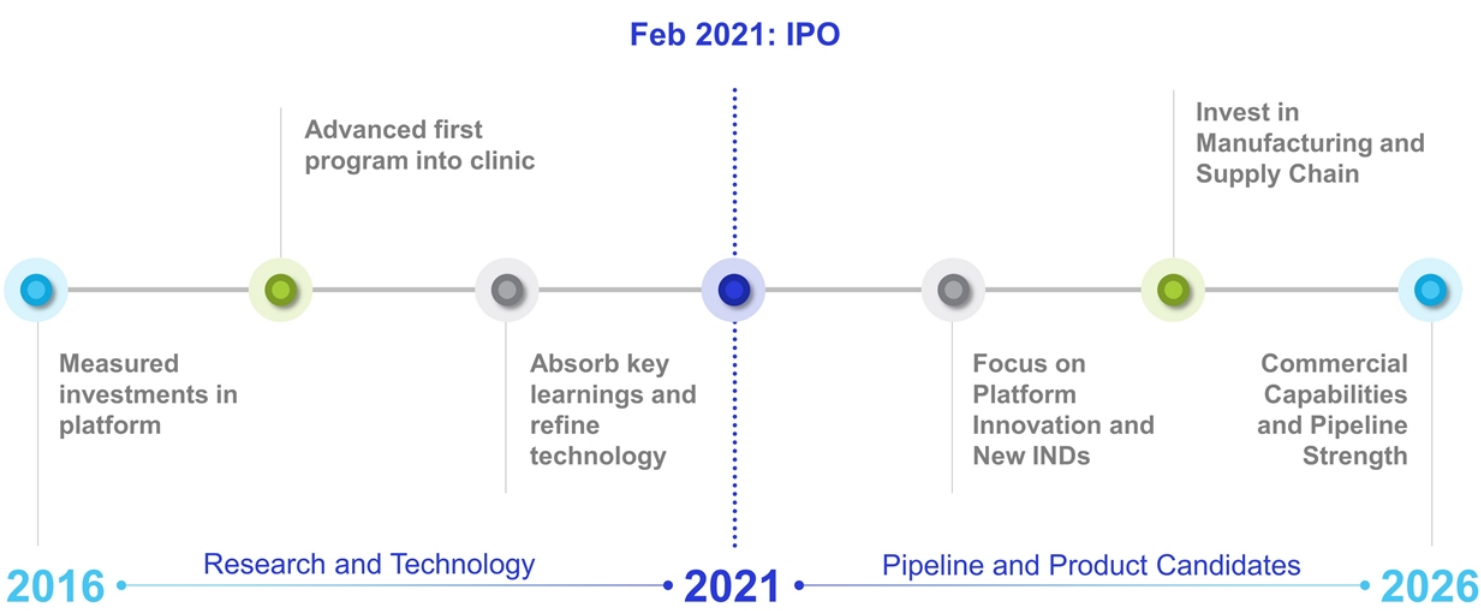
Neoantigen ImmunoPhage Manufacturing

Engineer novel ImmunoPhages expressing distinct tumor specific epitopes

ImmunoPhage Injection Including Neoantigens

Deliver neoantigen ImmunoPhage cocktail for administration and add neoantigen phages to bank for future use

Sensei's Vision to Capture Platform and Pipeline Value



Proven Team With Deep Experience

John Celebi, MBA
President and CEO

Robert Pierce, MD
Chief Scientific Officer

Erin Colgan
SVP, Finance and Administration

Michael Boychyn, PhD
SVP, CMC

Elisabeth Colunio
VP, Human Resources

Lora Pike
VP, Investor Relations Communications

Pauline Callinan, PhD
VP, Business Operations and Strategy

Alice Drumheller
VP, Clinical Operations

Edward van der Horst, PhD
VP, Preclinical Development

Jean Campbell, PhD
VP, Biologics Discovery

Bao Le
VP, Regulatory

Upcoming Expected Program Milestones

SNS-101 (anti-VISTA)

YE 2021:

- ✓ Present preclinical data at 36th Annual SITC Conference
- ✓ Select lead candidate
- ✓ Initiate IND-enabling studies underway

SNS-401-NG

2H 2022:

- Initiate IND-enabling studies

SNS-VSIG4

2023:

- Select product candidate

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Training the Immune System to Fight Cancer

December 6, 2021

NASDAQ: SNSE