Training the Immune System to Fight Cancer

The *ImmunoPhage*™ platform induces robust, focused immune responses

March 2021

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FINANCIAL SUMMARY

- **IPO on NASDAQ in February 2021, raising approximately $152 million in net proceeds**
  - Listed on Nasdaq Global Market under the ticker “SNSE”
  - Bookrunners: Citigroup, Piper Sandler, Berenberg
  - Manager: Oppenheimer
  - Use of proceeds:
    - Clinical development of SNS-301
    - Preclinical and clinical development of SNS-401
    - Preclinical and clinical development of SNS-VISTA
    - Development of ImmunoPhage platform and other pipeline programs
    - Working capital and other general corporate purposes

- **Financing history:**
  - Completed $30M Series B financing in January 2021; Co-led by Aperion Investment Group and Catalio Capital Management
    - Included new investors Pura Vida Investments and existing investors Cambrian Biopharma, Moore Strategic Ventures, Steve Jurvetson’s Future Ventures, and Presight Capital
  - Completed $28.5M Series AA financing in October 2020; Co-led by Cambrian Biopharma and H&S Ventures

PROVEN TEAM WITH DEEP EXPERIENCE

MANAGEMENT

John Celebi, MBA
President and CEO

- Over 23 years of experience in biotechnology sector
- Former Chief Operating Officer of X4 Pharmaceuticals and Chief Business Officer of Igenica Biotherapeutics

Marie-Louise Fjaellskog, MD, PhD
Chief Medical Officer

- Over 25 years of experience in clinical oncology, translational research, and drug development
- Previously served as VP of Clinical Development at Merus where she lead the development of several bispecific antibody therapeutics in oncology

Robert Pierce, MD
Chief Scientific Officer

- Over 20 years of experience in immuno-oncology
- While at Merck, he led a team focused on the development of tissue-based biomarkers for its anti-PD- 1 therapeutic antibody, KEYTRUDA
- Medical lead of the clinical trials of KEYTRUDA in MCC and CTCL

Anu Hoey, MS, MBA
Chief Business Officer

Michael Boychyn, PhD
SVP, CMC

Erin Colgan
SVP, Finance and Administration

Pauline Callinan, PhD
VP, Business Operations and Strategy

Jean Campbell, PhD
VP, Biologics Discovery

Alice Drumheller
VP, Clinical Operations

Edward van der Horst, PhD
VP, Preclinical Development

KEY ADVISORS

Samuel Broder, MD

- Co-developer of the first effective treatments for AIDS
- Oversaw the development of numerous cancer therapies, including Taxol
- Director of National Cancer Institute 1989-1995

James Peyer, PhD

- Chair, Board of Directors, Sensei Bio
- Founder and CEO, Cambrian Biopharma
- Previously, founder of Apollo Ventures

Robert Schreiber, PhD

- Cancer immunoediting hypothesis
- Multiple scientific awards and member of the National Academy of Sciences
- Alumni Endowed Professor of Pathology and Immunology; Professor of Molecular Microbiology

OUR IMMUNOPHAGE PLATFORM SOLVES THE TOUGHEST PROBLEMS IN IMMUNO-ONCOLOGY

- Proprietary ImmunoPhage platform technology
- Pipeline of clinical-stage immunotherapies for cancer with potential to expand into additional disease areas
- Ongoing Phase 1/2 clinical trial for SNS-301 in head and neck cancer that has shown promising anti-tumor activity and has been well-tolerated
- Collaborations with AstraZeneca, AdiMab and multiple academic institutions
- In-house GMP manufacturing capabilities

ROBUST PIPELINE UTILIZING PIONEERING IMMUNOPHAGE PLATFORM

Program	Approach/Target	Indication	Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestones
ONCOLOGY								
SNS-301	 Targeting ASPH; HPV-specific E6/E7	1st Line+ Head & Neck Cancer	**In combination with pembrolizumab**					**YE 2021:** • Addition of HPV-specific E6/E7 ImmunoPhage combination • Phase 1/2 data readout from large subset of patients
		Head & Neck Cancer – Neoadjuvant	**In combination with durvalumab**					**YE 2021:** Phase 2 trial initiation
SNS-401	 Cocktail with MCPyV	Merkel Cell Carcinoma						**1H 2022:** IND filing with FDA
SNS-VISTA	 Targeting VISTA	Solid Tumors						**YE 2021:** Initiate IND-enabling studies
Antibodies and Nanobodies		**Discovery and validation of multiple antibodies and nanobodies utilizing ImmunoPhage platform**						

 ImmunoPhage  Antibody

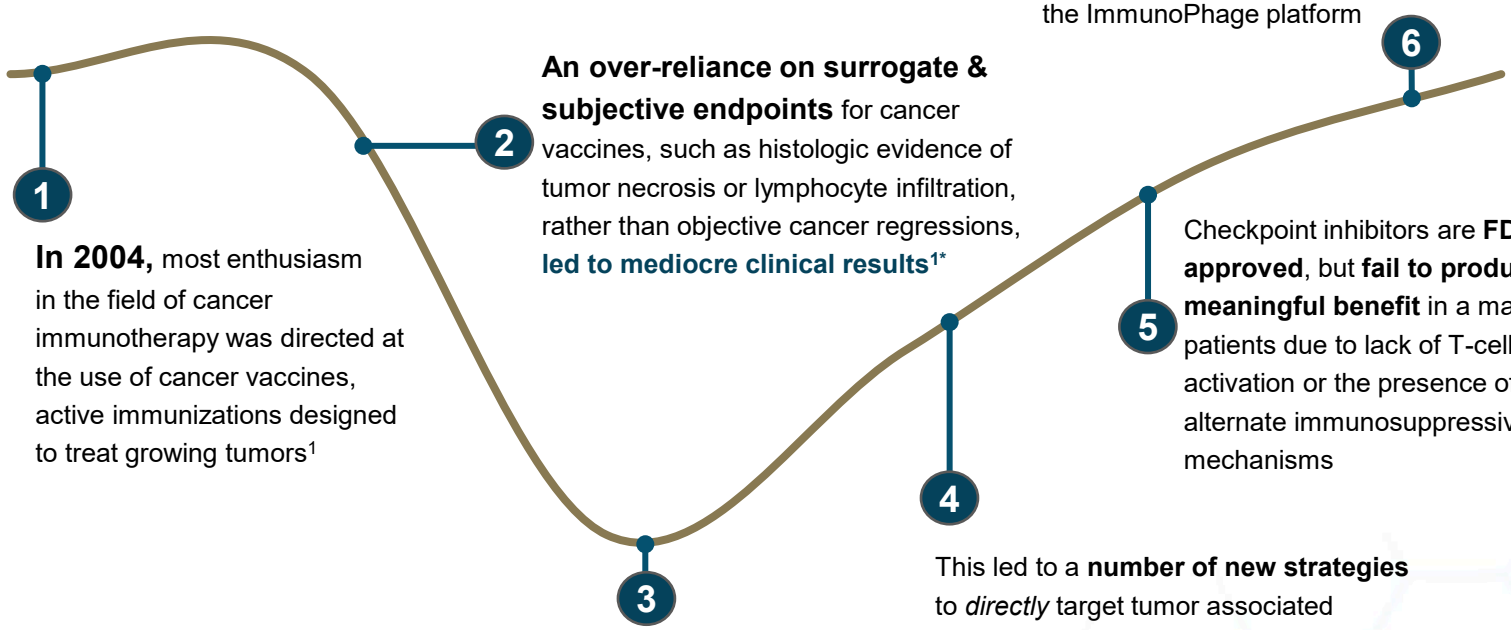
SENSEI BIOTHERAPEUTICS

ImmunoPhage Platform

IMMUNO-ONCOLOGY VACCINE HISTORY

The emergence of checkpoint inhibitors has rekindled interest in mechanisms that activate T-cells and block alternate immunosuppressive mechanisms

Immuno-oncology vaccine history



1 **In 2004,** most enthusiasm in the field of cancer immunotherapy was directed at the use of cancer vaccines, active immunizations designed to treat growing tumors[1]

2 **An over-reliance on surrogate & subjective endpoints** for cancer vaccines, such as histologic evidence of tumor necrosis or lymphocyte infiltration, rather than objective cancer regressions, **led to mediocre clinical results[1*]**

3 **The conclusion by many is that cancer vaccines generally fail** to immunologically destroy established tumors that lead to objective responses**

4 This led to a **number of new strategies** to *directly* target tumor associated antigens, including the advancement of TCR and CAR-T therapies, and checkpoint inhibitors

5 Checkpoint inhibitors are **FDA approved**, but **fail to produce meaningful benefit** in a majority of patients due to lack of T-cell activation or the presence of alternate immunosuppressive mechanisms

6 **In 2019,** Sensei Bio launches the ImmunoPhage platform

*At the NCI, among 440 patients treated with cancer vaccines, the objective response rate was 2.6%[1]

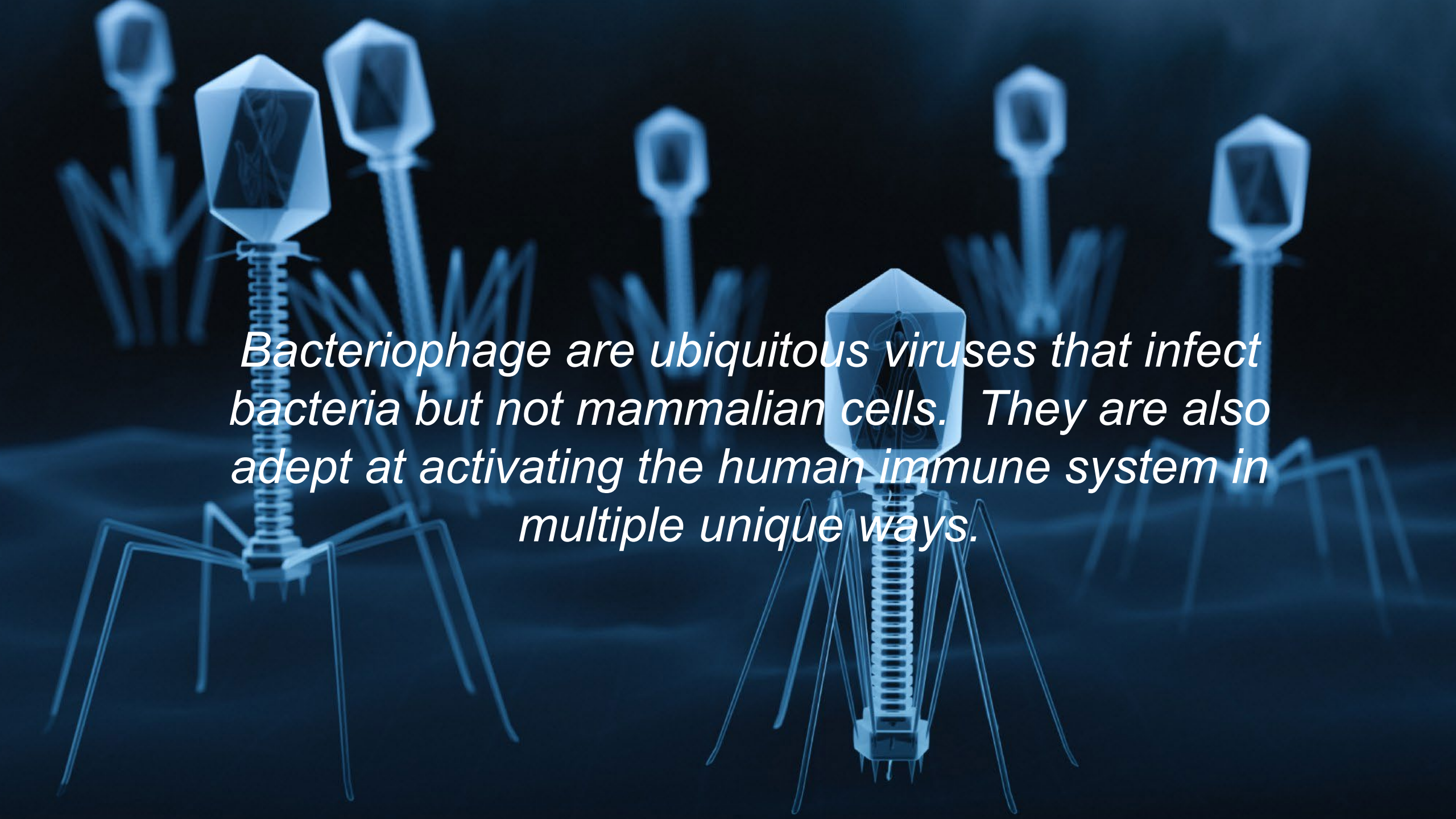
** Due to insufficient numbers of high avidity T-cells with recognition of tumor antigens, trafficking and infiltration of T-cells to the tumor and stroma, and activation of T-cells at the site of the tumor.

1 Rosenberg SA, Nature Medicine, 2004; 2 McKinsey report, "The next wave of innovation in oncology"

What if we could **engineer** a virus to **target** the key mechanisms of **checkpoint resistance?**

What if we could do this in a **personalized** way, with an **off-the-shelf** product concept?

At ***Sensei Biotherapeutics***, we have built the ***ImmunoPhage*** platform dedicated to **develop** therapeutics that induce a **robust, focused** and **coordinated immune response** to treat cancer


Bacteriophage are ubiquitous viruses that infect bacteria but not mammalian cells. They are also adept at activating the human immune system in multiple unique ways.

THREE AXES OF INNOVATION TO FIGHT CANCER

Cancer is a complex problem requiring a multi-pronged solution. Our *ImmunoPhage*s 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

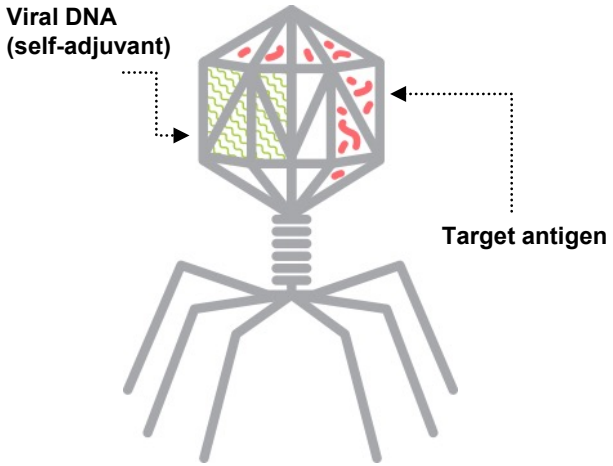
Localized gene therapy

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

GENERATING STRONG ANTIBODY AND T-CELL RESPONSES

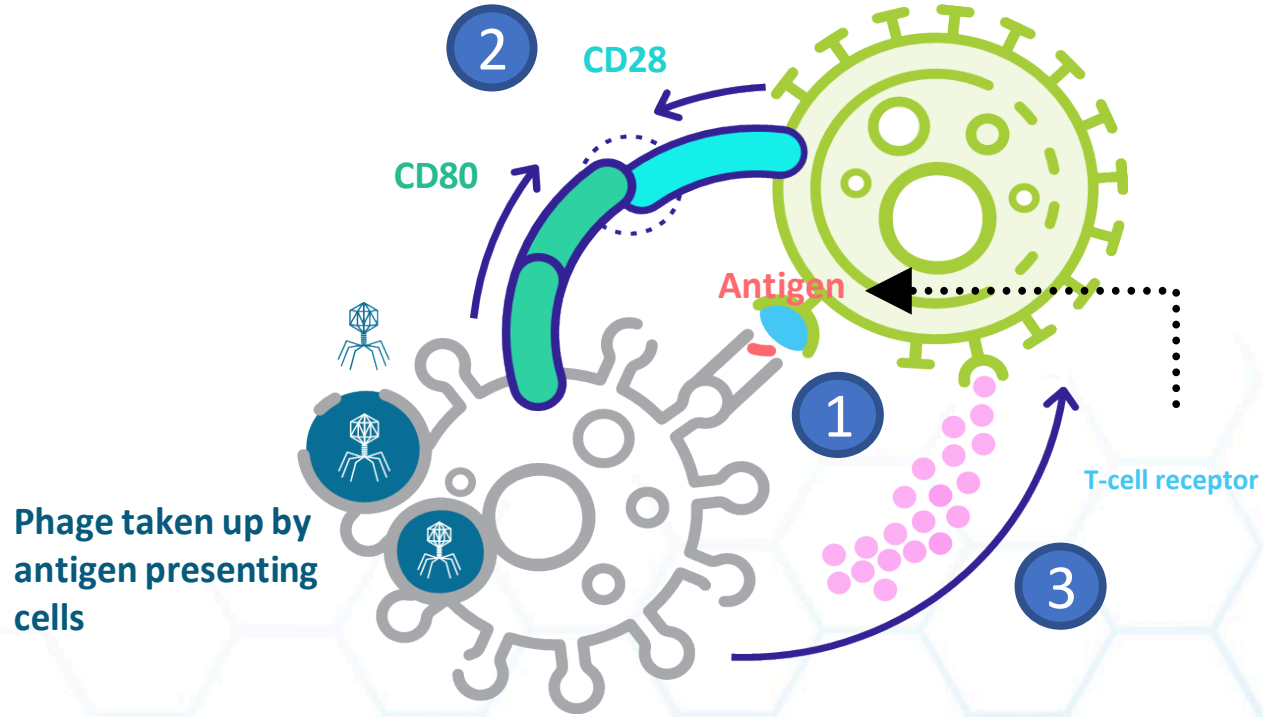
Our *ImmunoPhage*s contain an **engineered display of target antigens on the surface of a bacteriophage.** We use non-infectious lambda bacteriophage viruses to mimic a pathogenic virus, driving strong T cell and B cell mediated antibody responses.

1 **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.

2 **ImmunoPhages are taken-up by APCs and deliver the three critical signals required to drive activation of T cells. 1) Activation of CD8 T cells through cross presentation 2) Positive Co-stimulation of T cells 3) Generation of a Th1-biased immune response and cytotoxic T-lymphocytes**



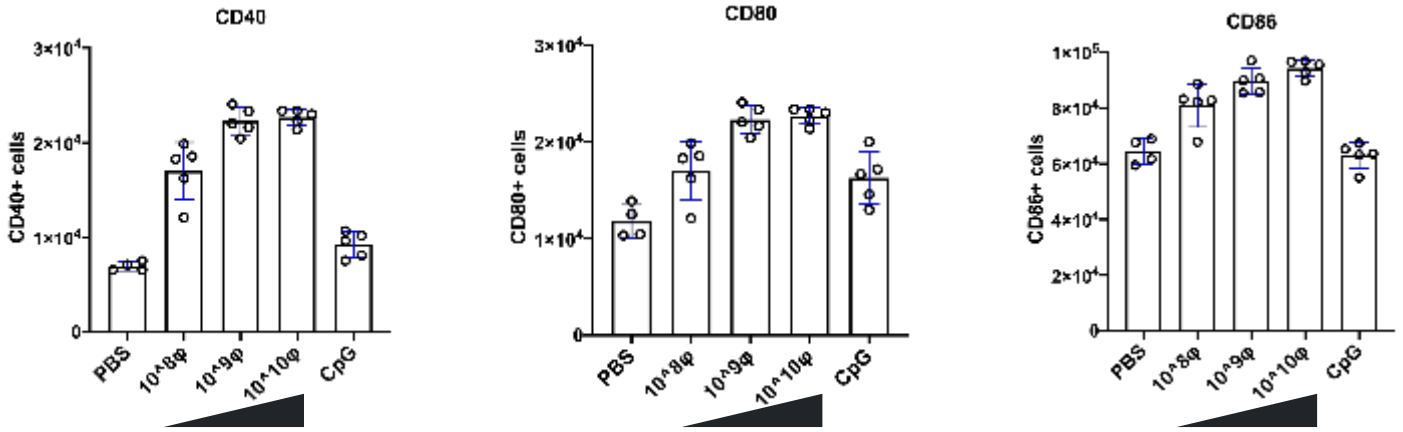
MECHANISM OF ACTION: ACTIVATION AND MATURATION OF DENDRITIC CELLS

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

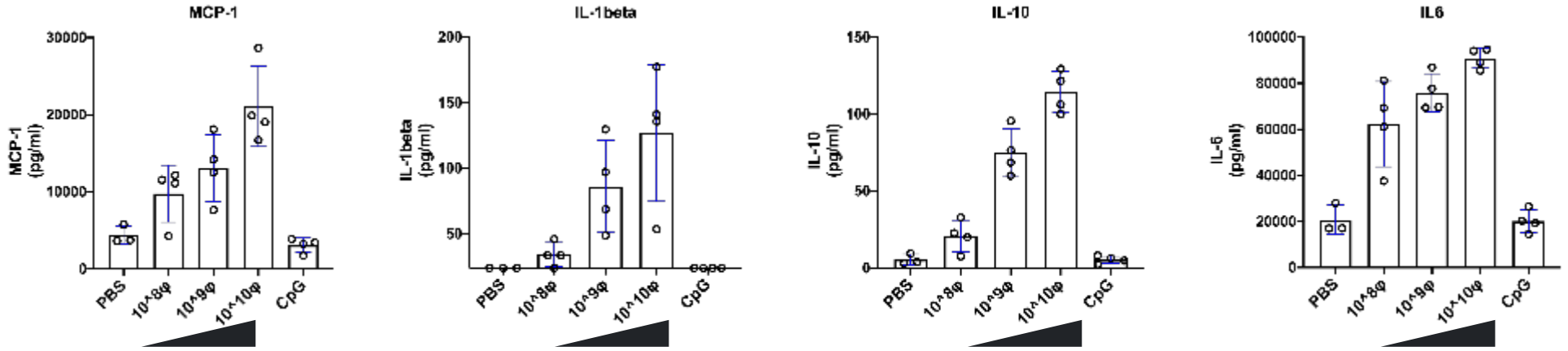
Bacteriophage-expressed antigens are then processed and presented efficiently by MHC class I and class 2 pathways, leading to **robust CD4 and CD8 T cell responses**

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

Signal 2
Dendritic cell co-stimulatory molecules



Signal 3
Cytokine secretion

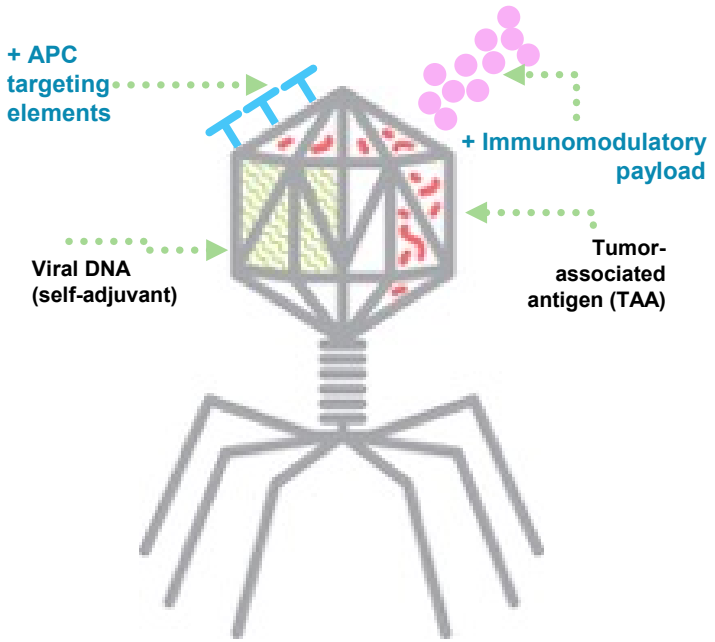


ALPACA-DERIVED NANOBODIES ENHANCE IMMUNOSTIMULATORY EFFECTS

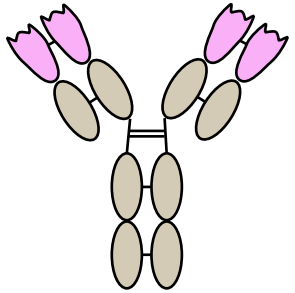
Direct targeting to the APC and modulation of the tumor microenvironment.

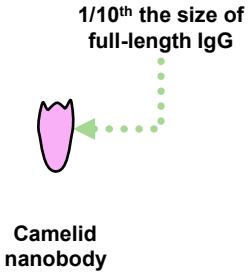
Payloads are comprised of key immunomodulatory nanobodies, which possess additional advantages to conventional antibodies.

1 Addition of APC-targeting elements and modification of phage genome to deliver immunomodulatory payload



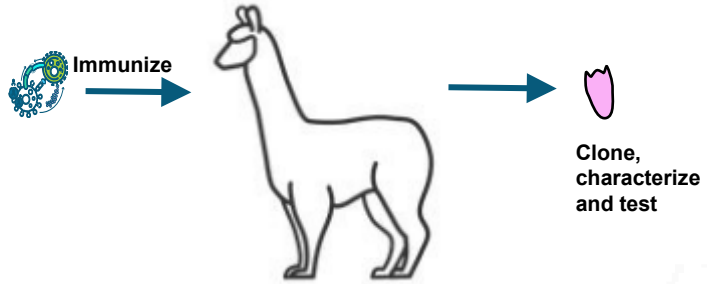
2 Nanobodies are used to deliver immunomodulatory payloads and have superior properties compared to human antibodies.





- Commercially validated
- Extremely high-affinity and exhibit other unique binding characteristics
- Can access nanoscale protein domains, conferring enhanced properties and uses (e.g. TCR mimicry)
- Robust “drug-like” protein characteristics: stable, easy to manufacture, amenable for use as modular protein binding domains in multi-specific scaffolds

3 *ImmunoPhage* are leveraged to immunize alpaca against key immunomodulatory targets. Several important nanobodies have already been generated.



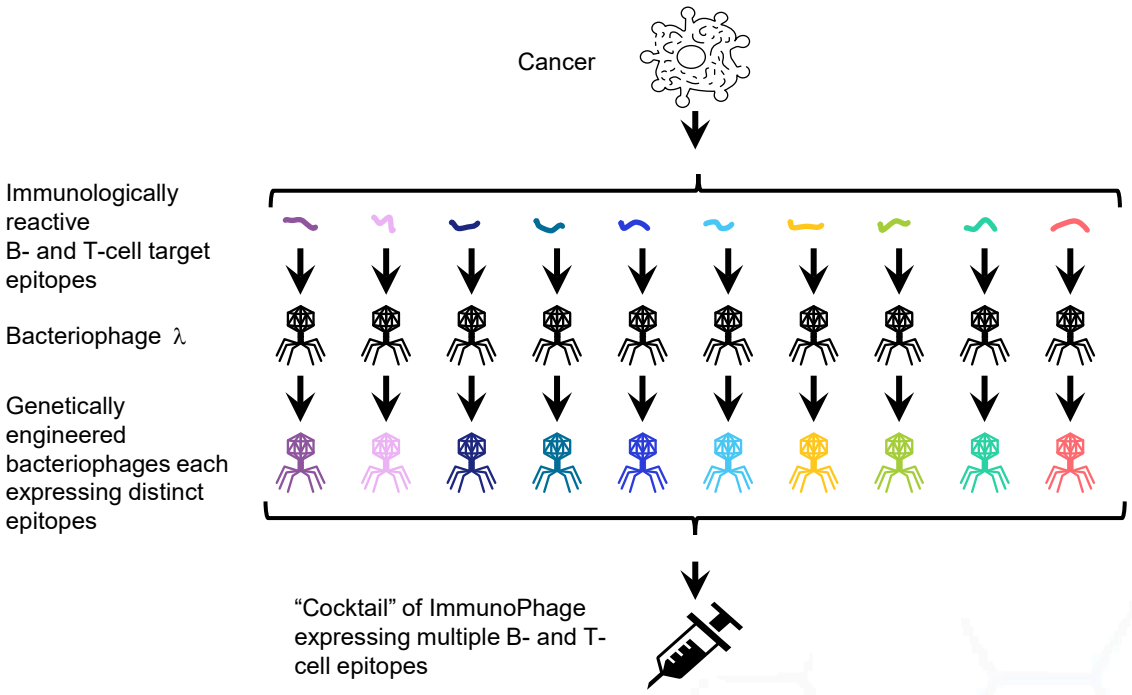
6 key immunomodulatory nanobodies have already been generated

- **TIGIT**
- **PD-1**
- **TGF-ß**
- **IL-10rα**
- **CD301**
- **B7-H3**

COMBINATIONS CAN BE CUSTOMIZED FOR PERSONALIZED IMMUNOTHERAPY

Our proprietary library of *ImmunoPhage* – *Phortress* – harnesses the intrinsic immunostimulatory characteristics and capabilities of bacteriophage to create a personalized, coordinated, and nuanced multi-modal immune response.

Phortress is our proprietary library of personalized vaccine cocktails with off-the-shelf *ImmunoPhage* "ingredients"



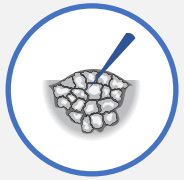
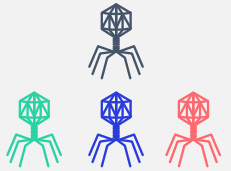
- These "cocktails" are defined by the disease or patient genetics
- Combinations are customized to cover multiple epitopes, protein domains or targets
- Each *ImmunoPhage* is pre-manufactured to target a discrete antigen

PERSONALIZED IMMUNOTHERAPY APPROACH

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

Personalized yet Off-the Shelf TAA Therapy

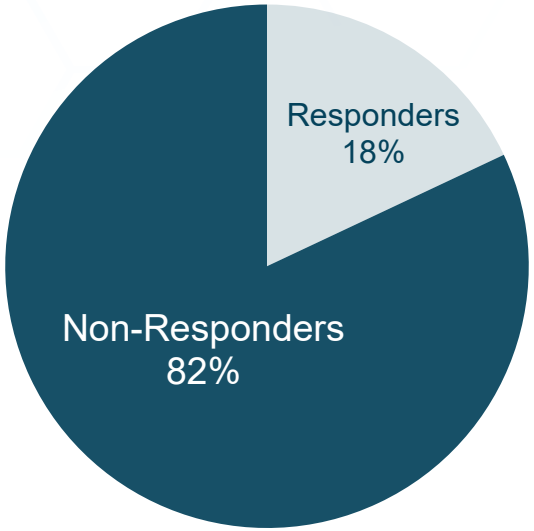
Off-the-Shelf + Patient-specific Neoantigen Therapy

1	2	3	4	5	6
Routine Biopsy	**Tumor Sequencing**	**Personalized yet Off-the-shelf ImmunoPhage Cocktail**	**Neoantigen Prediction**	**Neoantigen ImmunoPhage Manufacturing**	**ImmunoPhage Injection Including Neoantigens**
					
Clinical biopsy of tumor as input material	Tumor DNA Tumor RNA Normal DNA	Assemble a personalized cocktail from off-the-shelf TAA ImmunoPhage for administration	Identify additional tumor specific neoantigens	Engineer novel ImmunoPhages expressing distinct tumor specific epitopes	Deliver neoantigen ImmunoPhage cocktail for administration and add neoantigen phages to bank for future use

Pipeline Programs

SCCHN; A DIFFICULT TO TREAT CANCER WITH HIGH UNMET NEED

High Unmet Need



Despite their commercial success, PD-1 inhibitors only lead to objective responses in <20% of SCCHN cancers[1].

Unmet needs exist for PD-1 inhibitor resistant/refractory patients and PD-L1 low patients.

H&N Incidence

650k

Head and neck cancer cases diagnosed/year worldwide[2]

HPV+ SCCHN

70%

% of SCCHN patients with HPV+ in United States[3]

Immunotherapy

7 PD-1/ PD-L1 inhibitors

Only 2 of 7 (pembrolizumab and nivolumab) have been approved for use in SCCHN

Market Value

$36B

Estimated Global PD-1 / PD-1L inhibitor market value (USD) by 2024[4]

1 Pembrolizumab Average Response Rate Across All SCCHN Patients (KEYNOTE-012, KEYNOTE-48)
2 Up To Date. Epidemiology and risk factors for head and neck cancer. Jan 21, 2020. https://www.uptodate.com/contents/epidemiology-and-risk-factors-for-head-and-neck-cancer#H2
3 The prognostic value of HPV in head and neck cancer patients undergoing postoperative chemoradiotherapy, Randall J. Kimple, Paul M. Harari, Annals of Translational Medicine Journal, 2015.
4 EvaluatePharma, Evaluating the Immunotherapy Landscape: 2020-2024.

SNS-301 CLINICAL DEVELOPMENT PATH

Phase 1/2 data readout expected by YE 2021

PHASE 1/2
COMBINATION SAFETY AND EFFICACY

TRIAL POPULATION FOR SNS-301/ PEMBROLIZUMAB COMBO

INCLUSION CRITERIA

1. Locally advanced / metastatic SCCHN
2. PD-1 blockade > 12 weeks
3. SD or unconfirmed PD

STAGE 1

Cohort 1 (n=15)

★ ***If Cohort 1 achieves ≥ 1 partial or complete response, add additional patients***

STAGE 2

ADD Additional n=15

ADD Cohort 2
PD-1 blockade naïve patients (1st Line)

ADD Cohort 3
E6/E7 HPV+ cocktail approach

RANDOMIZED PHASE 2/3
COMBINATION THERAPY VS. PEMBROLIZUMAB

PATIENTS RANDOMIZED TO

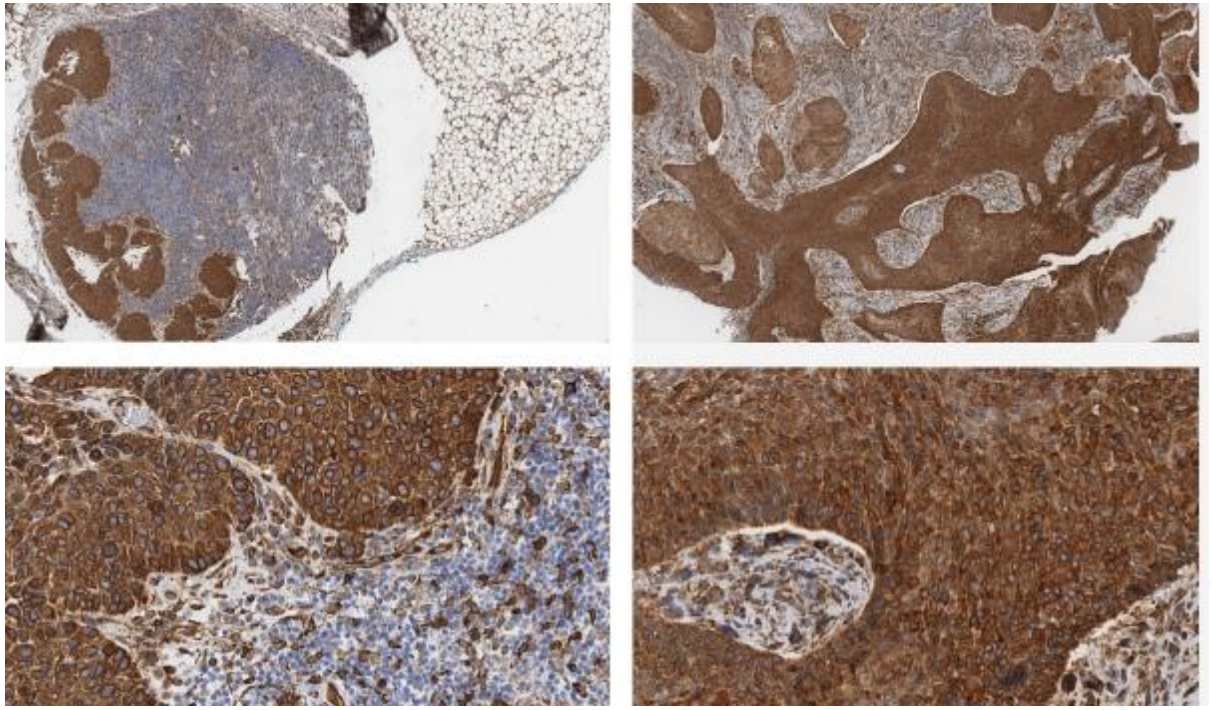
A **SNS-301/ pembrolizumab combo**

B **Pembrolizumab monotherapy**

SNS-301 ONGOING PHASE 1/2 TRIAL IN HEAD AND NECK CANCER

- Starting in Q4 2019, patients with H&N cancer on checkpoint inhibitors without observed tumor reductions started receiving SNS-301 in combination with pembrolizumab in a Phase 1/2 clinical trial
- SNS-301 induces patients' immune systems to generate a strong, specific response against the tumor associated antigen, ASPH
- Tumor samples collected from 30 patients screened for inclusion in the Phase 1/2 trial were stained for intratumoral ASPH expression, all of which demonstrated strong ASPH expression
- By combining checkpoint inhibitor therapy with SNS-301, we hope to strike at two ways that cancers evade the immune system at once

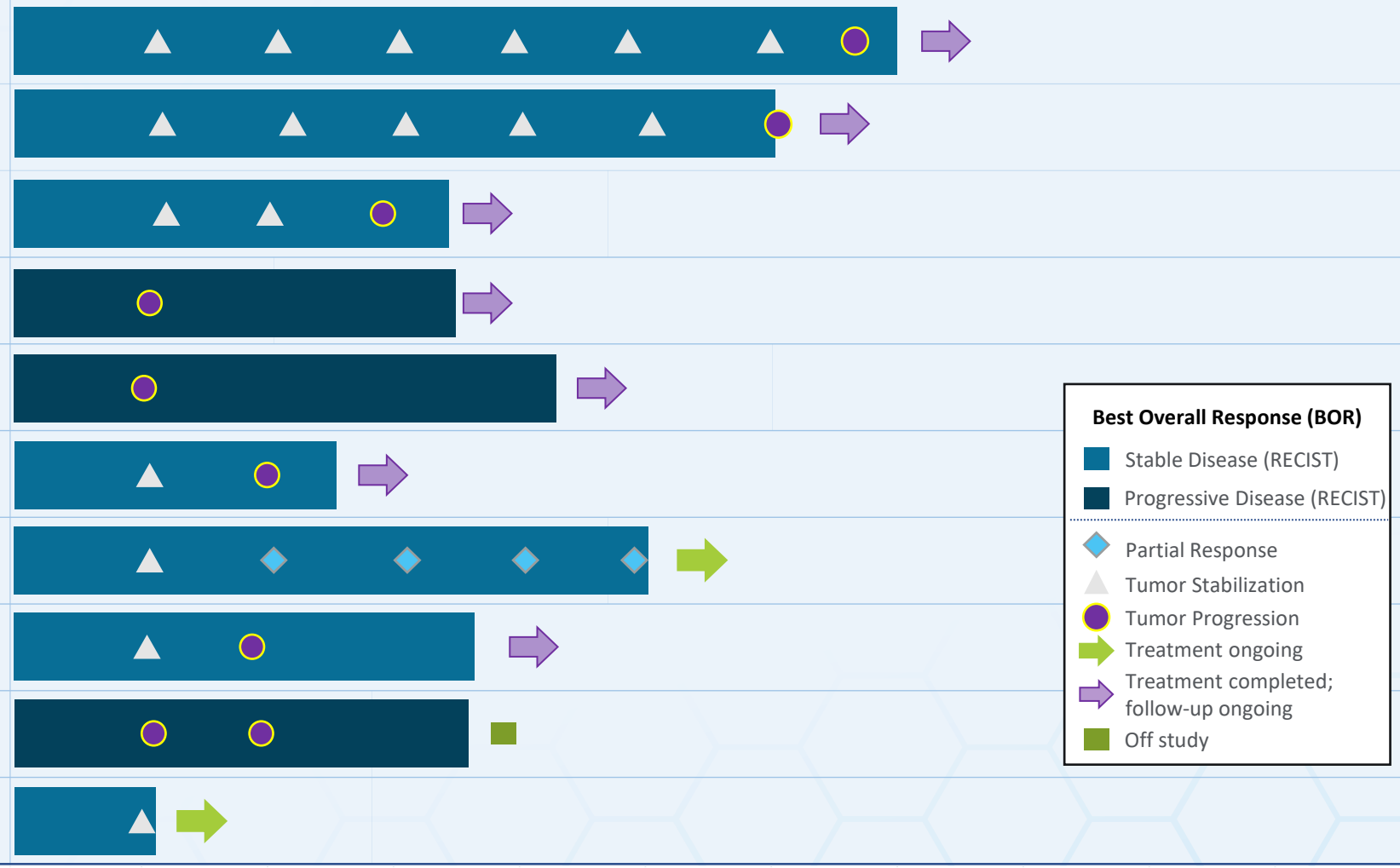
All head and neck cancer patients enrolled in the SNS-301 Phase 1/2 trial to date strongly express the tumor-associated antigen ASPH



ASPH = TUMOR-ASSOCIATED ANTIGEN

RESULTS FROM PHASE 1/2 TRIAL IN HEAD AND NECK CANCER (AS OF DECEMBER 10, 2020)

PD-L1 Status	Status Pre-Trial*	BOR On Trial	Patient
–	SD	SD	A
+	SD	SD	B
Unknown	uPD	SD	C
+	uPD	PD	D
Unknown	uPD	PD	E
+	SD	SD	F
–	SD	PR	G
Unknown	SD	SD	H
Unknown	SD	PD	I
Unknown	uPD	SD	J



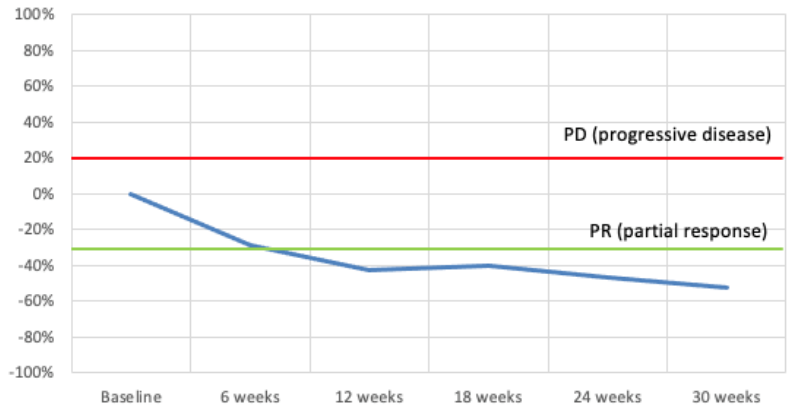
uPD = unconfirmed Progressive Disease
BOR = Best Overall Response
* Disease status at enrollment after ≥ 12 weeks of PD-1 blockade

PATIENT 006-001 DETAIL

The partial response is highly likely to be attributed to the addition of SNS-301 to pembrolizumab, given that the tumor was PD-L1 negative prior to study and no objective response was observed after >3 months of prior pembrolizumab.

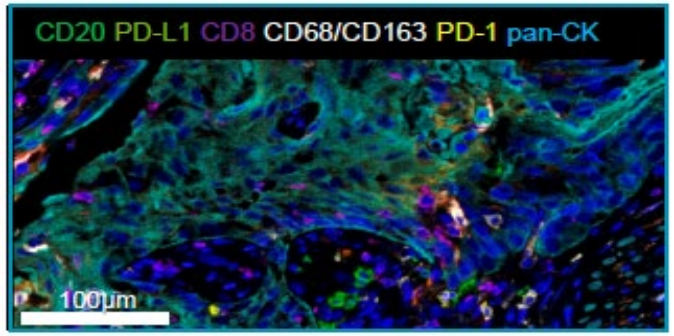
PATIENT
• 69-year-old woman / HPV and PD-L1 negative • Stage II T2N0M0 HNSCC (supraglottic larynx)
STATUS AT TRIAL ENTRY
• ECOG 1; PD-1 blockade (pembrolizumab) >3 months
PHASE 2 TRIAL OVERVIEW
• IMRT (2 - 54 Gy) 09JUL- 08AUG2018 • CARBO/PACLI/CETUX 08AUG-15AUG2019 (2 cycles) with best response PR • Pembrolizumab JAN2020 ongoing

Patient 006-001 Tumor Measurements

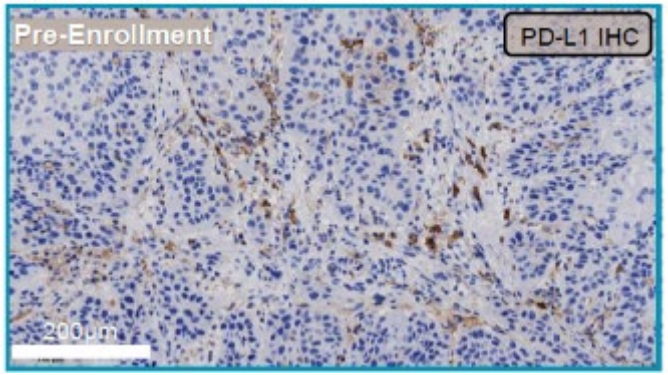


Pre-Treatment

Immune Markers by Multiplex IHC Pre-Treatment

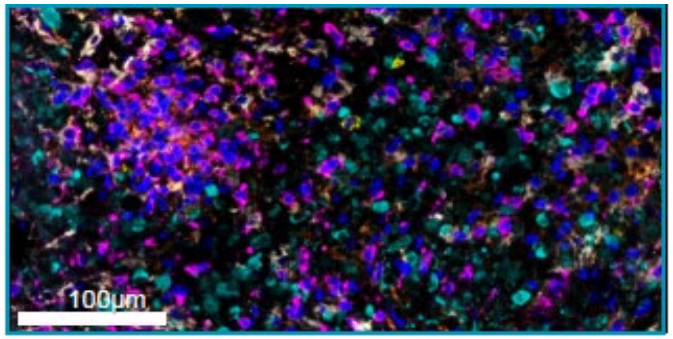


PD-L1 Negative Pre-Treatment

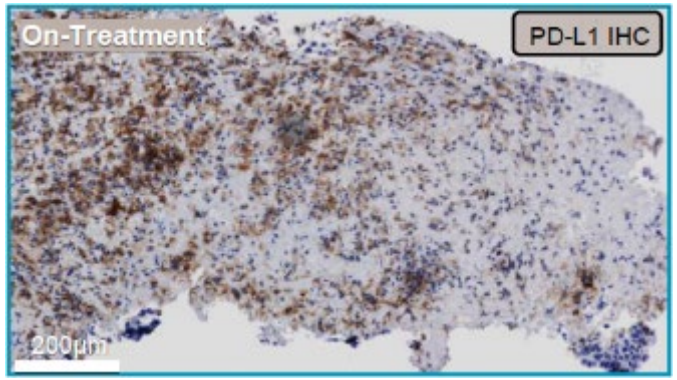


Post-Treatment

Increase in Immune Markers by Multiplex IHC



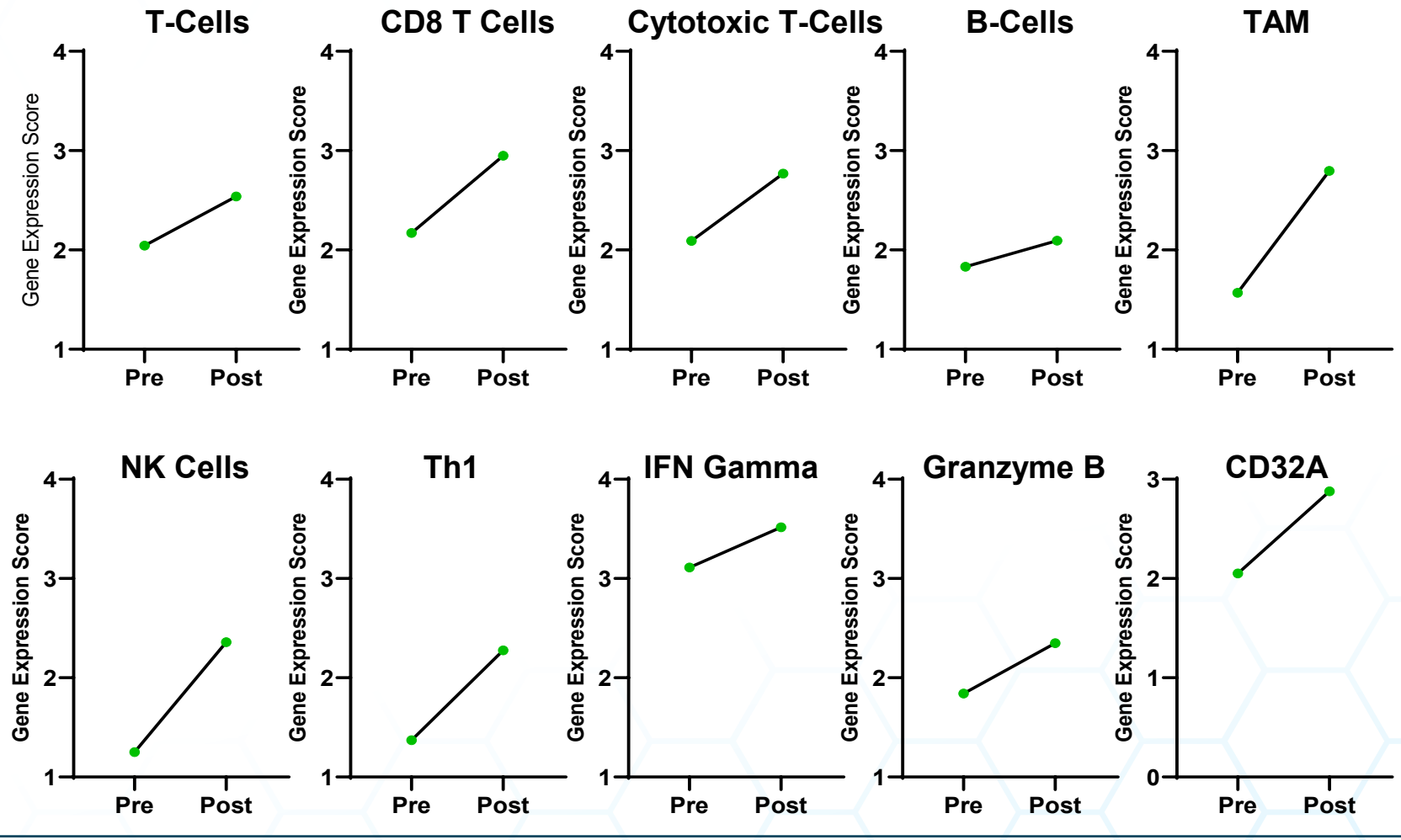
Strongly PD-L1 Positive Post-Treatment



PATIENT 006-001 DETAIL

This PD-L1 negative patient had a complete set of attributes consistent with a strong Th1 anti-tumor immune response.

Translational data suggest cellular and humoral responses to SNS-301, including conversion from poorly to highly inflamed tumor in this patient that did not have an objective response to checkpoint blockade monotherapy.



T cells: *CD3D, CD3E, CDeG, SH2D1A and TRAT1.* ***CD8***: *CD8A and CD8B.* ***Cytotoxic T cells***: *CTSW, GNLY, GZMA, GZMB, GZMH, KLRB1, KLRK1, NKG7 and PRF1.* ***B-Cells***: *CD19, FCRL2, MS4A1, PNOC, SPIB, TCL1A and TNFRSF17.* ***Tumor associated macrophages (TAM)***: *Arg1, BAMBI, MARCO.* ***NK Cells***: *IL21R, KIR2DL3, KIR3DL1, KIR3DL2, and NCR1. Th1: TBX2.* ***IFNg***: *CXCL10, CXCL9, HLA-DRA, IDO1, IFNG and STAT1.* ***Granzyme B***: *GZMB.* ***CD32A***: *FCGR1A.*

SNS-301 HAS BEEN WELL TOLERATED IN COMBINATION WITH PEMBROLIZUMAB

	(n=11)					
Related Events	**Grade 1 n (%)**	**Grade 2 n (%)**	**Grade 3 n (%)**	**Grade 4 n (%)**	**Grade 5 n (%)**	**All Grades n (%)**
Decreased appetite	1(9.1)	1(9.1)	0	0	0	2 (18.2)
Fatigue	2(18.2)	0	0	0	0	2 (18.2)
Pruritus	2(18.2)	0	0	0	0	2 (18.2)
Back pain	0	1(9.1)	0	0	0	1 (9.1)
Constipation	0	1(9.1)	0	0	0	1 (9.1)
Dehydration	0	0	1(9.1)	0	0	1 (9.1)
Diarrhea	1(9.1)		0	0	0	1 (9.1)
Dizziness	0	1(9.1)	0	0	0	1 (9.1)
Electrocardiogram QT prolonged	0	0	1(9.1)	0	0	1 (9.1)
Erythema	1(9.1)	0	0	0	0	1 (9.1)
Headache	1(9.1)	0	0	0	0	1 (9.1)
Injection site pain	1(9.1)	0	0	0	0	1 (9.1)
Nausea	0	1(9.1)	0	0	0	1 (9.1)
Non-cardiac chest pain	0	1(9.1)	0	0	0	1 (9.1)
Urine output decreased	1(9.1)	0	0	0	0	1 (9.1)
Weight decreased	1(9.1)	0	0	0	0	1 (9.1)

Data as of October 6, 2020.

- No DLTs and mostly Grade 1-2 unrelated adverse events.
- Two Grade 3 events were reported: hypertension (not related) and dehydration (related), reported as a serious adverse event (SAE). An additional SAE, Systemic Inflammatory Response Syndrome (G1), occurred during follow-up.

BUILDING THE FIRST CUSTOM MERKEL CELL POLYOMA VIRUS (MCPyV) IMMUNOPHAGE

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

1 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

2 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.

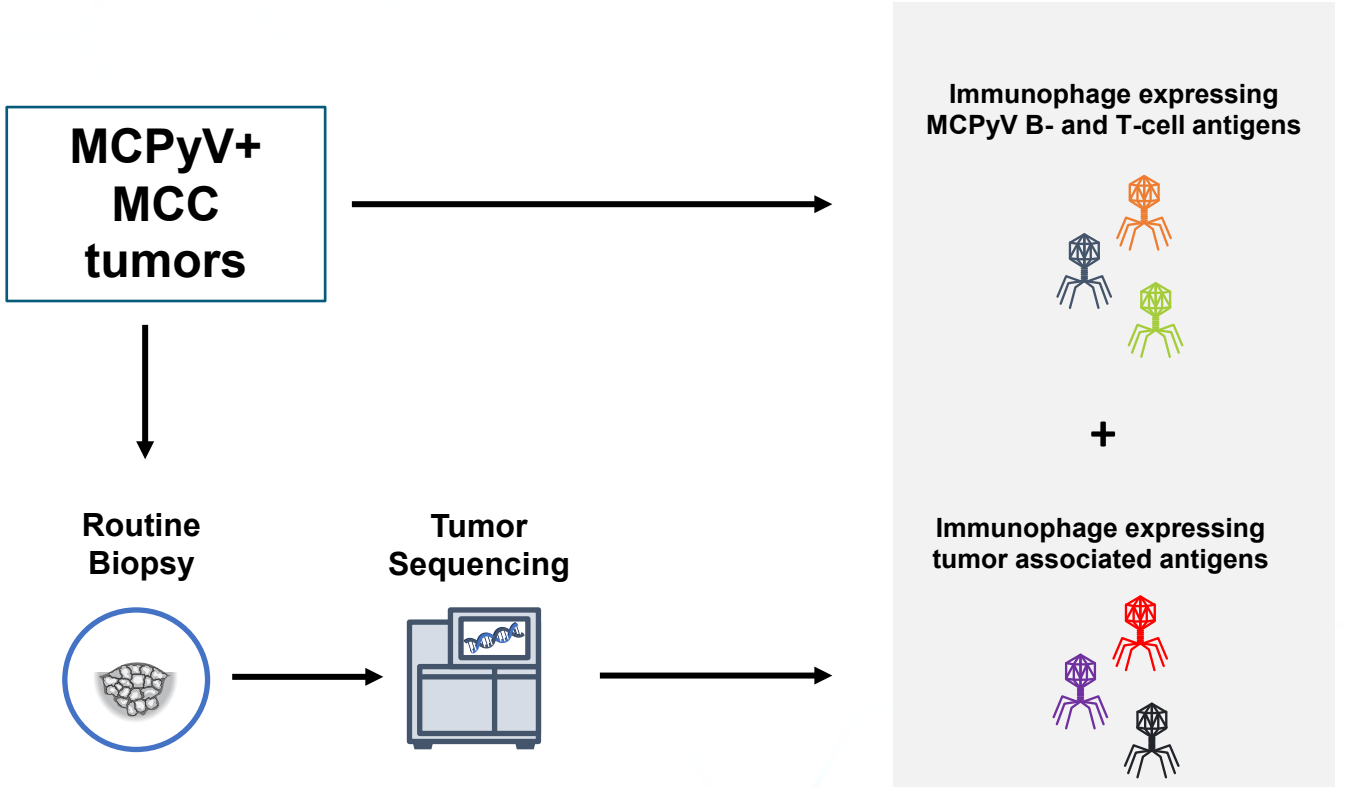
3 Discovery partnership with University of Washington

- UW will design MCPyV T-cell constructs and will determine the immunogenicity and mechanism of candidate *ImmunoPhages*
- Sensei will develop *ImmunoPhages* specifically targeting MCPyV T-cell constructs and other tumor associated antigens (TAAs) using a cocktail approach

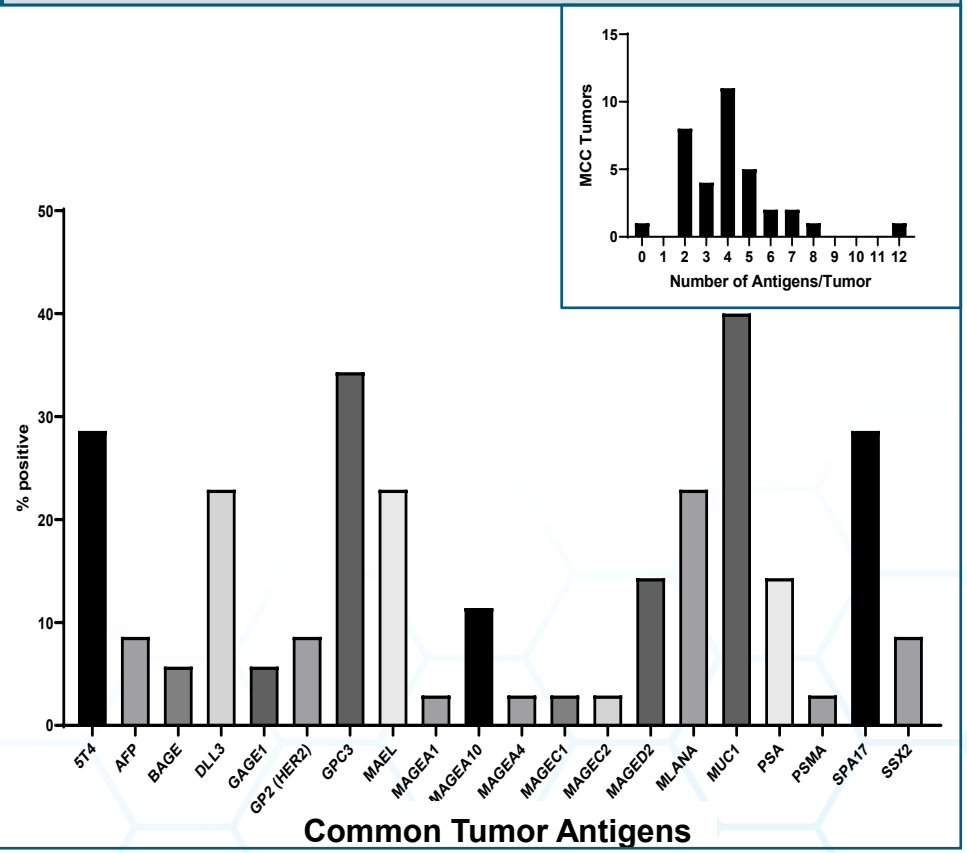
BUILDING THE FIRST CUSTOM MERKEL CELL POLYOMA VIRUS (MCPyV) IMMUNOPHAGE

SNS-401 has the potential to be the first **fully customized, yet off-the-shelf**, product.

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



2 Most MCC tumors contain five or fewer TAAs[1]



1 Based on internal data.

PARTNERSHIP WITH ADIMAB TO DEVELOP LEAD anti-VISTA ANTIBODY

A VISTA is recognized as an important immune checkpoint regulator

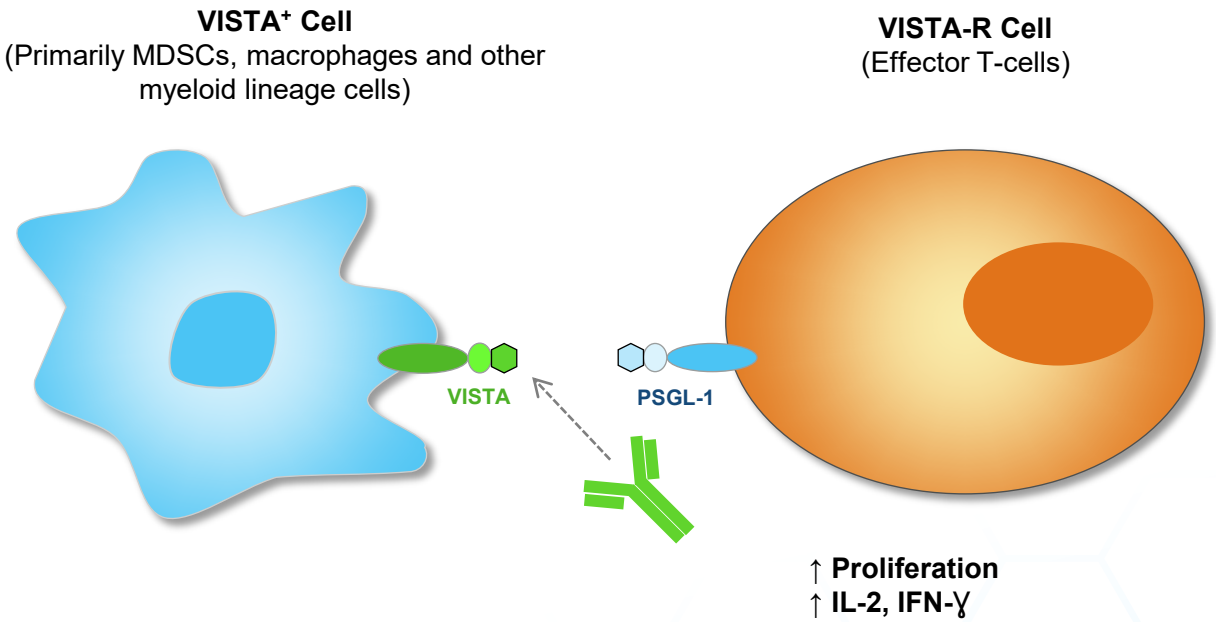
- Member of B7 family of proteins
- A negative regulator of T cell responses
- Blockade significantly enhances immune-mediated tumor rejection in vivo[1]
- Agonists exert protective effect in autoimmune models
- Plays a role in immune surveillance through phagocytic dead cell clearance[2]
- Mice lacking VISTA and PD-1 display enhanced ability to control tumor outgrowth[3]

[1] Le Mercier et al. VISTA Regulates the Development of Protective Antitumor Immunity. Cancer Res. 2014 Apr 1;74(7):1933-44.

[2] K. W. Yoon et al., Science 349, 1261669 (2015). DOI: 10.1126/science.1261669

[3] Liu J. et al. PNAS 2015

B Disruption of the extracellular VISTA-PSGL-1 interaction enhances T-cell proliferation and induces cytokine production

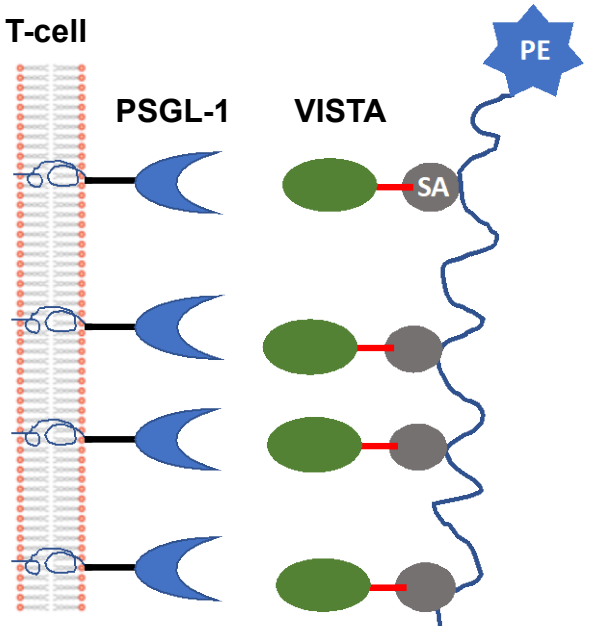


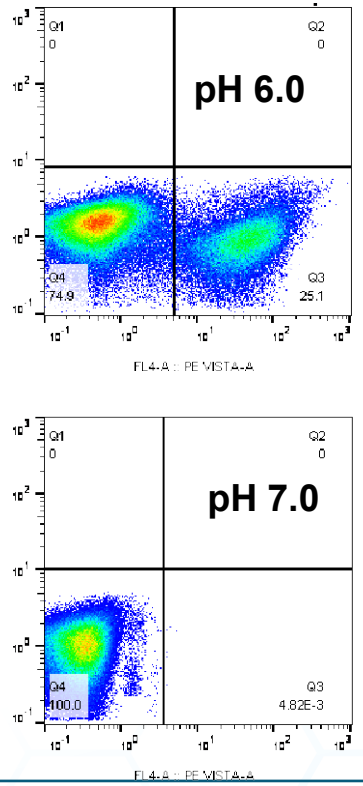
Knowledge of functional epitope between VISTA and PSGL-1 and potential patient population decreases development timeline and enhances early signal detection in Ph I trial

ASSESSMENT OF LEAD ANTIBODIES

A Assessment of anti-VISTA mAbs through interaction of VISTA and native PSGL-1 at varying pH

- Assay shows binding of VISTA protein to native PSGL-1 on activated CD4 T-cells

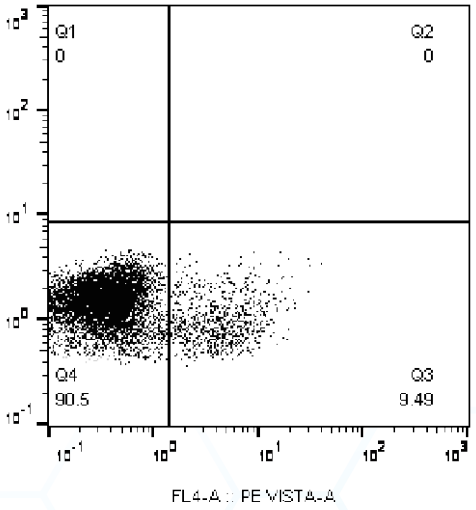




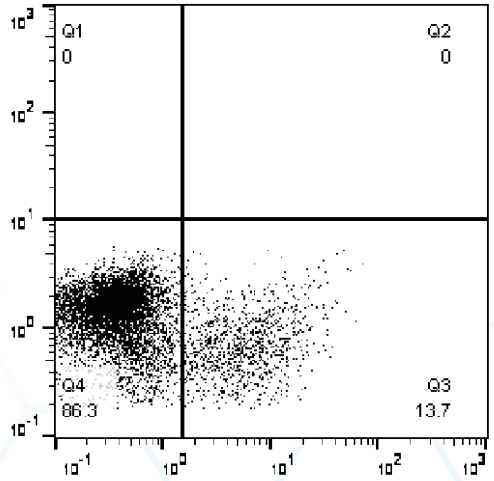
B Disruption of the VISTA-PSGL-1 interaction at pH 6 enhances T-cell proliferation and induces cytokine production

- Screened >80 mAb candidates
- Multiple candidates **inhibited VISTA binding** to cell surface of CD4+ T-cells at **pH 6.0,** including:

mAb 921



mAb 140



PH1/2 IN SCCHN TO READOUT BY YE 2021; 3 NEW PROGRAMS ENTERING THE CLINIC BY 2022

- Study initiation
- Study readout

R&D

R&D collaborations
Sign definitive agreement for R&D collaboration

SNS-301
Initiate Stage 2 of Phase 1/2 Trial

SNS-301
Incorporation of E6/E7 in HPV-positive SCCHN, expand to PD-1 naïve (1st line)

SNS-301 SCCHN – Neoadjuvant
Initiate Ph2 trial in combination with durvalumab

SNS-301
SCCHN Ph1/2 topline readout

SNS-VISTA
Initiate IND-enabling studies

SNS-401
Complete IND-enabling studies and submit IND

BUSINESS

Alvaxa acquisition
Asset integration into *ImmunoPhage* platform

Manufacturing
Complete first round of GMP manufacture of clinical *ImmunoPhage*

Platform patents
Multiple issues or to be issued

Cocktail approach
Receive regulatory guidance for cocktail approach in oncology

APPENDIX

SENSEI BIOTHERAPEUTICS

PATENT PORTFOLIO & IP STRATEGY

PATENT PORTFOLIO

US

2 Issued
5 Pending

2 Issued U.S. patents (SNS-301), approximately 4 pending U.S. (3 SNS-301 and 1 SNS-CoV2)

Foreign

>10 Patents
& patent apps

In the multiple foreign jurisdictions representing the major global pharmaceutical markets

SNS-301

2 U.S.
3 Foreign
10 Pending

KEY FEATURES

Vaccine editing “on the fly”

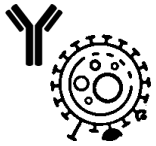
Antigen selection is optimized throughout clinical development through a dynamic cocktail approach to optimize patient therapy or limit specific liabilities (e.g., antibody-dependent enhancement (ADE)).

Large payload capacity

Multiple proteins and domains. Multiple phage per cocktail for virtually unlimited capacity.

Safety Profile

Well tolerated in Phase 1 and 2 settings.

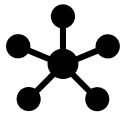


Complete immune responses

Self-adjuvanted through phage CpG motifs and icosahedral geometry. Intrinsic APC-targeting. B- and T-cell activation. Highly immunogenic and capable of breaking tolerance to self-antigens.

Cost-effective, fast and scalable manufacturing in-house

Manufacturing @ small 10L scale under cGMP conditions yields 5k-10k doses. Achieving cGMP manufacture of an ImmunoPhage in 4 weeks. Easy tech transfer.



Broadly applicable

An emerging approach for the treatment of multiple cancer indications and infectious diseases

ImmunoPhage COMBINES THE BEST FEATURES OF RNA AND ADENOVIRUS VACCINES

FEATURE	*ImmunoPhage*	Live Adenovirus vaccines	mRNA-based vaccines
DESCRIPTION	Bacteriophage λ nanoparticle ds DNA genome (48.5 kb)	Live attenuated adenovirus ds DNA genome (~26-45kb)	Lipid mRNA nanoparticle Minimal restrictions in construct length
"PAYLOAD" / MANUFACTURING CAPACITY	**Payload capacity ~30kb per *ImmunoPhage* x multiple**	Payload capacity ~8.5kb	N/A
ADJUVANT	**Self-adjuvanted** CpG motifs contained within vector + icosahedral capsid geometry	Exogeneous adjuvant Requires gp140 for optimal immune response	Exogenous adjuvant Ionizable cationic lipid in LNP
IMMUNE RESPONSE	**Humoral + cellular**	Primarily humoral	Humoral + cellular
EPITOPE SELECTION / VACCINE DESIGN	**Dynamic throughout clinical dev.** *ImmunoPhage* antigen cocktail can be optimized throughout clinical development	Static Single antigen expressing select antigen(s) chosen prior to clinical development	Static Single antigen locked in prior to clinical development
MANUFACTURING TIMELINE SEQUENCE SELECTION TO QA RELEASE	<12 weeks	6-12 months	<12 weeks
SCALABILITY	**High** ~10,000 doses @ 10L	High ~10,000 doses @ 10L	Med Est. 60,000 doses/run, non-commercial scale

POTENTIAL REGISTRATIONS PATHS IN H&N CANCER

SNS-301-2-2 is a signal seeking study, evaluating the immunogenicity of the phage, translational data and preliminary clinical efficacy.

A. Inclusion/exclusion criteria mostly mimic KEYNOTE-12, except SNS-301-2-2 only enrolls a subset of patients with SD/early PD
   - Expected ORR with continued PD1 inhibition alone is dependent on time on previous PD1 inhibition treatment; with current study population it is estimated to be **10%**

B. Several potential patient populations to pursue for registrations

(A) Phase II H&N cancer decision tree based on KEYNOTE-012

Go/No-go will be based on the following parameters:

A. Objective responses comparative or above those achieved by PD1 inhibition alone (ORR > 10%)

B. Duration of response

C. Evidence that that SNS-301 is immunogenic (ex: antigen specific T cell responses)

D. Demonstration of Immune activation in tumor microenvironment (ex: increased PD-L1 expression)

ORR, often used as a surrogate markers for overall survival (OS) and basis for accelerated approval, is a poor predictor of OS in checkpoint inhibitor trials (Kok et al. JAMA Sept 2020).

(B) Potential patient populations for Phase 2/3 registration study for H&N cancer

Encouraging efficacy data in patients with SD

1. Same population
   - Randomize against single agent PD1 inhibitor (PD1i)
2. PD1i naïve
   - Randomize against SA PD1i
   - Single arm and compare with KEYNOTE-48 (however, probably randomization needed anyway as next step)

Encouraging efficacy data in patients with PD

3. Same population
   - Randomize against chemotherapy (platin combination) in second line

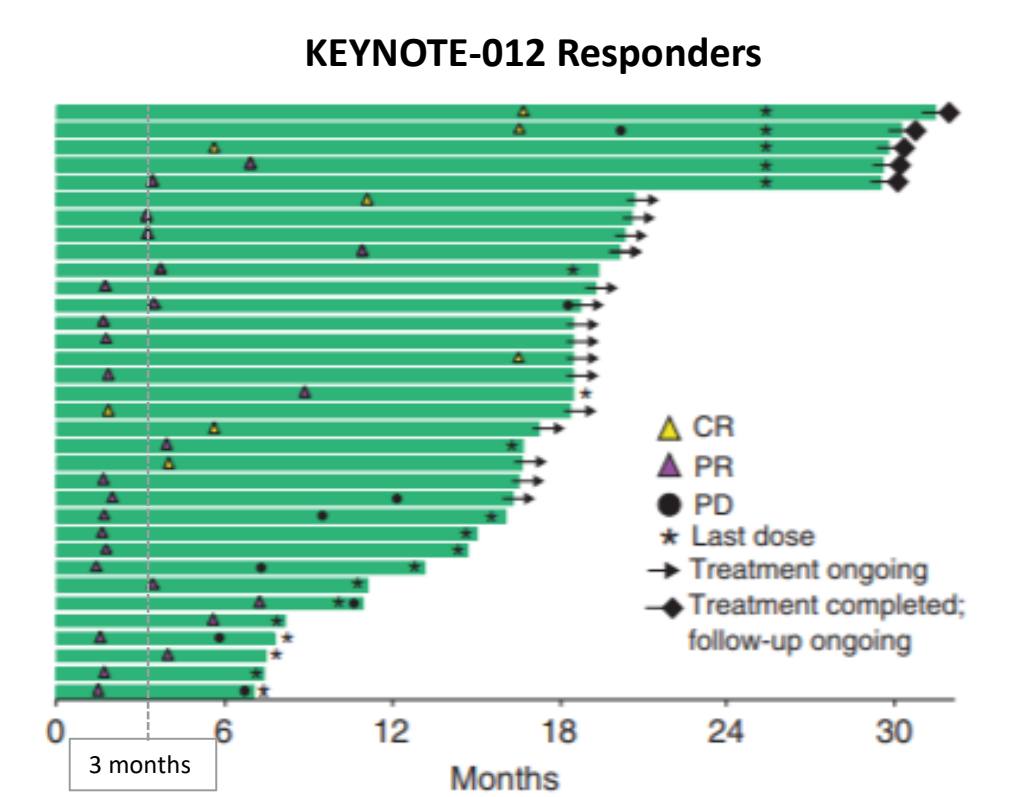
BENCHMARKING KEY H&N DATA (KEYNOTE-012)

- KEYNOTE-12 results available up to 30 months
- Demonstrated 18% ORR with pembrolizumab in 2nd line HNSCC
  - IO approvals mostly based upon duration of response and overall survival.
  - ORR does NOT predict OS in IO.
- Half of objective responses were observed within 3 months

Table 3. Tumour response to pembrolizumab as per RECIST v1.1 by central imaging vendor review (all-patients-as-treated population; $N = 192$)

	All $N = 192$		HPV associated $n = 45$		Non-HPV associated $n = 147$	
	No.	% (95% CI)	No.	% (95% CI)	No.	% (95% CI)
Overall response rate	34	18 (13–24)	11	24 (13–40)	23	16 (10–23)
Complete response	8	4 (2–8)	4	9 (3–21)	4	3 (1–7)
Partial response	26	14 (9–19)	7	16 (7–30)	19	13 (8–19)
Stable disease	33	17 (12–23)	7	16 (7–30)	26	18 (12–25)
Progressive disease	93	48 (41–56)	19	42 (28–58)	74	50 (42–59)
Non-CR/Non-PD	7	4 (2–7)	1	2 (0.1–12)	6	4 (2–9)
No assessment	21	11 (7–16)	6	13 (5–27)	15	10 (6–16)
Not evaluable	4	2 (0.6–5)	1	2 (0.1–12)	3	2 (0.4–6)

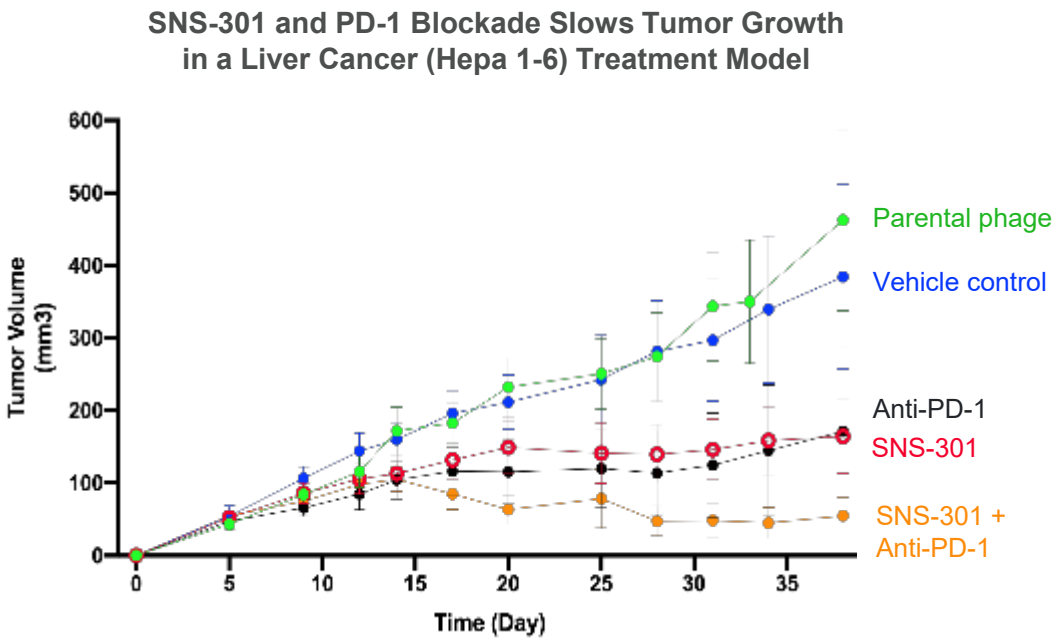
Only confirmed responses are included. *CR* complete response, *PD* progressive disease, *RECIST* Response Evaluation Criteria in Solid Tumours. No assessment: patient discontinued before the first imaging assessment (reasons: progressive disease [$n = 12$]; adverse event [$n = 3$]; withdrawal by patient [$n = 3$]; death [$n = 2$]; protocol violation [$n = 1$]). Not evaluable: patient had post baseline imaging, but images were not of sufficient quality to determine response



POSITIVE DATA AS MONO OR COMBINATION THERAPY

SNS-301 demonstrated strong preclinical and Phase 1 data – currently in Phase 2 trials

1 SNS-301 in combination with PD-1 blockade in preclinical models has shown a trend toward combinatorial efficacy



- SNS-301 works to drive antigen-specific immune activation. PD-1/PD-L1 works by 'releasing the brakes' on immune suppression.
- Combining these two modalities may have synergistic effects.

2 Well-tolerated in Phase 1 trial

0/12

Patients with Related Serious Adverse Events

- Well tolerated.
- No patients experienced Grade 4 adverse events.
- Most importantly, there were no signs of off-target auto-immune responses.

100%

Patients with Antigen-Specific Immune Responses

Multivariate immune responses involve both activated, antigen-specific T cells and B cells. SNS-301 induced both in the majority of patients treated.

58%

Patients Showing Signs of Clinical Biomarker Activity

- Phase I trial conducted in prostate cancer patients
- While the study was designed to measure safety and not anti-tumor responses, 7 of 12 patients saw the growth rate of their tumor markers slow.