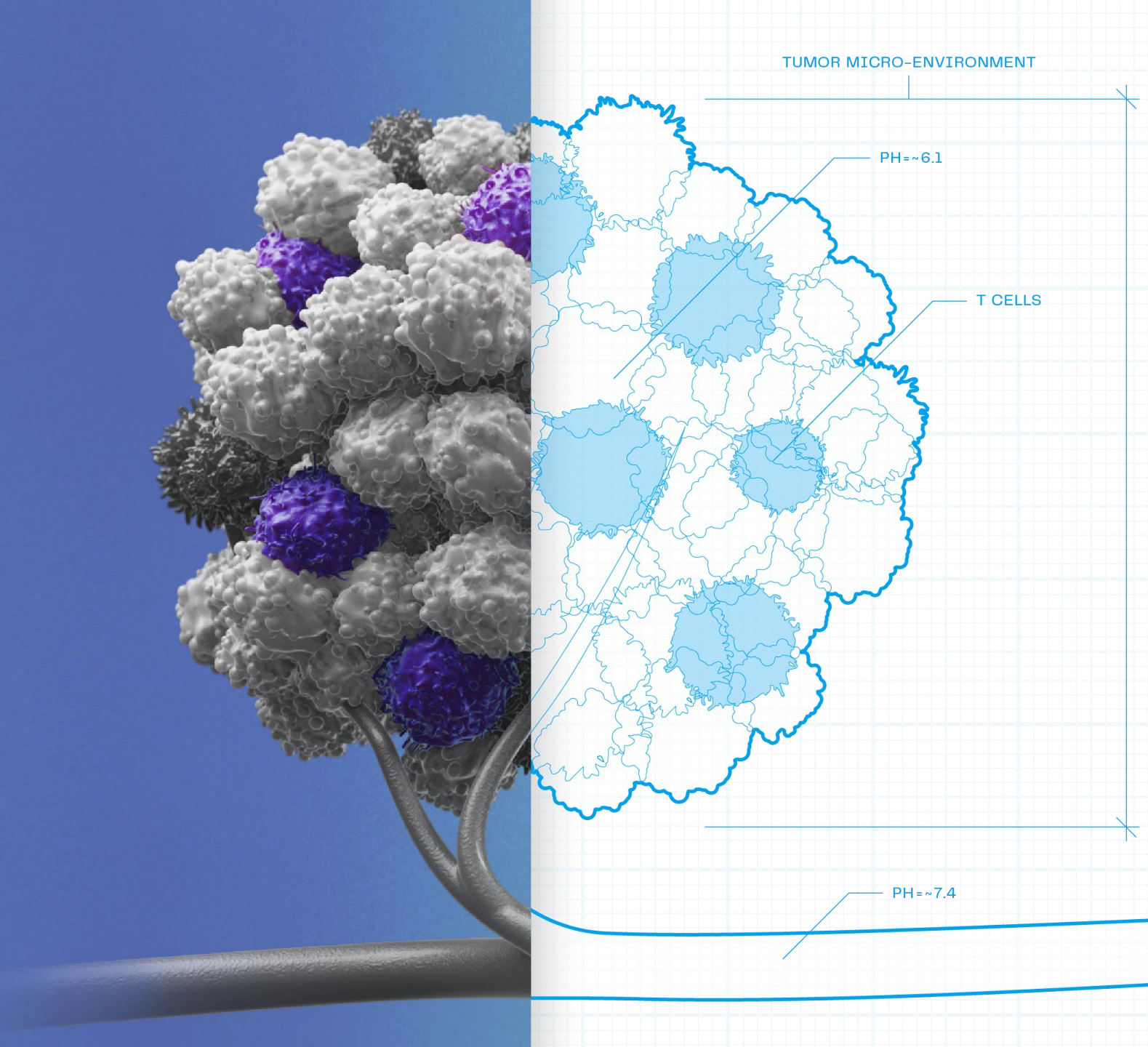

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Next Generation Immuno-Oncology Medicines

Jefferies Healthcare Conference

John K. Celebi, MBA

President & Chief Executive Officer

JUNE 2022 | NASDAQ: SNSE

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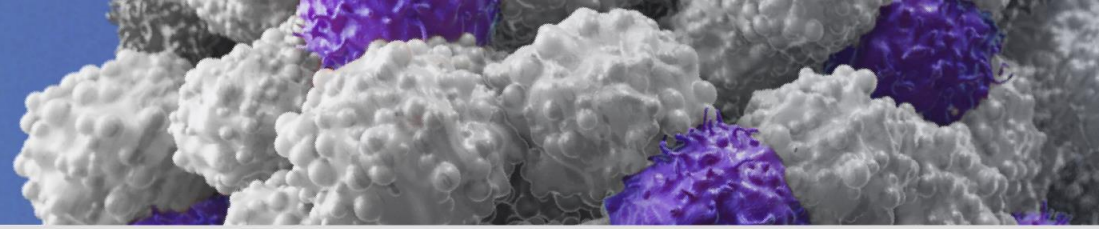

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Positioned to Drive Value with Next Generation Product & Platform Development

Two Platforms Generate Broad Pipeline Opportunities

TMAb™

ImmunoPhage™

Innovative Pipeline of Differentiated IO mAbs

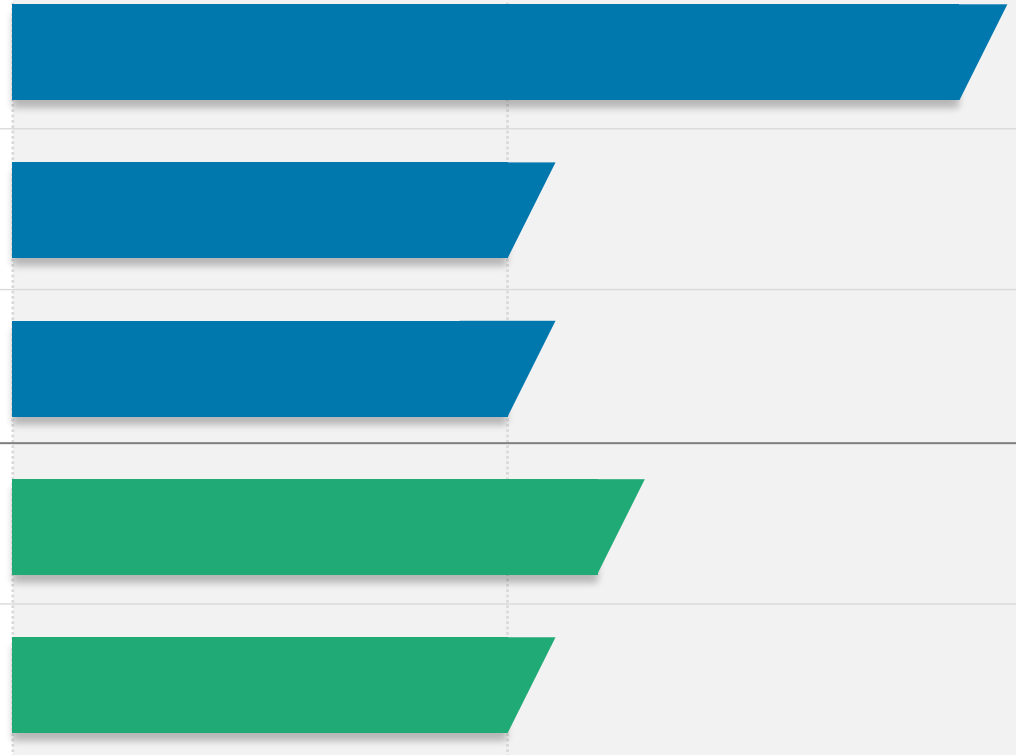
Strong Cash Position Ended 1Q 2022: $136M ** Cash runway into Q1 2025

Business Development

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

Innovative Pipeline of IO Drugs with Broad Commercial Potential

	Program (Target)	Indication	Discovery	IND-enabling	Phase 1 / 2 Clinical
TMAb	SNS-101 (VISTA)	Solid Tumors			
	SNS-102 (VSIG4)	Solid Tumors			
	SNS-103 (ENTPDase1/CD39)	Solid Tumors			
ImmunoPhage	SNS-401-NG (Multiple Tumor Antigens)	Merkel Cell Carcinoma			
		Multiple Indications			



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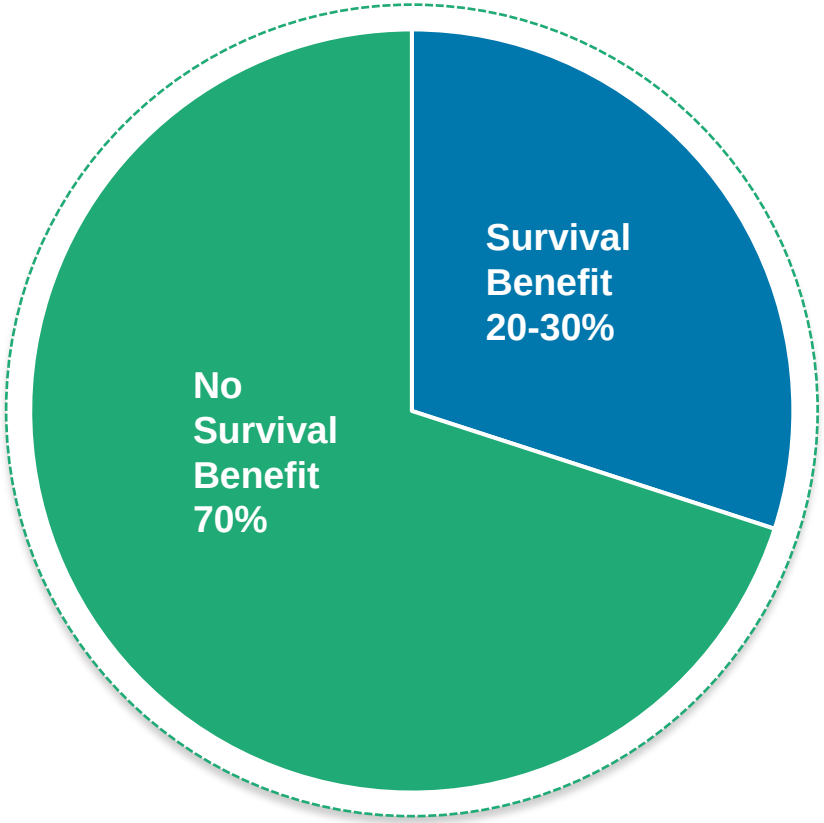
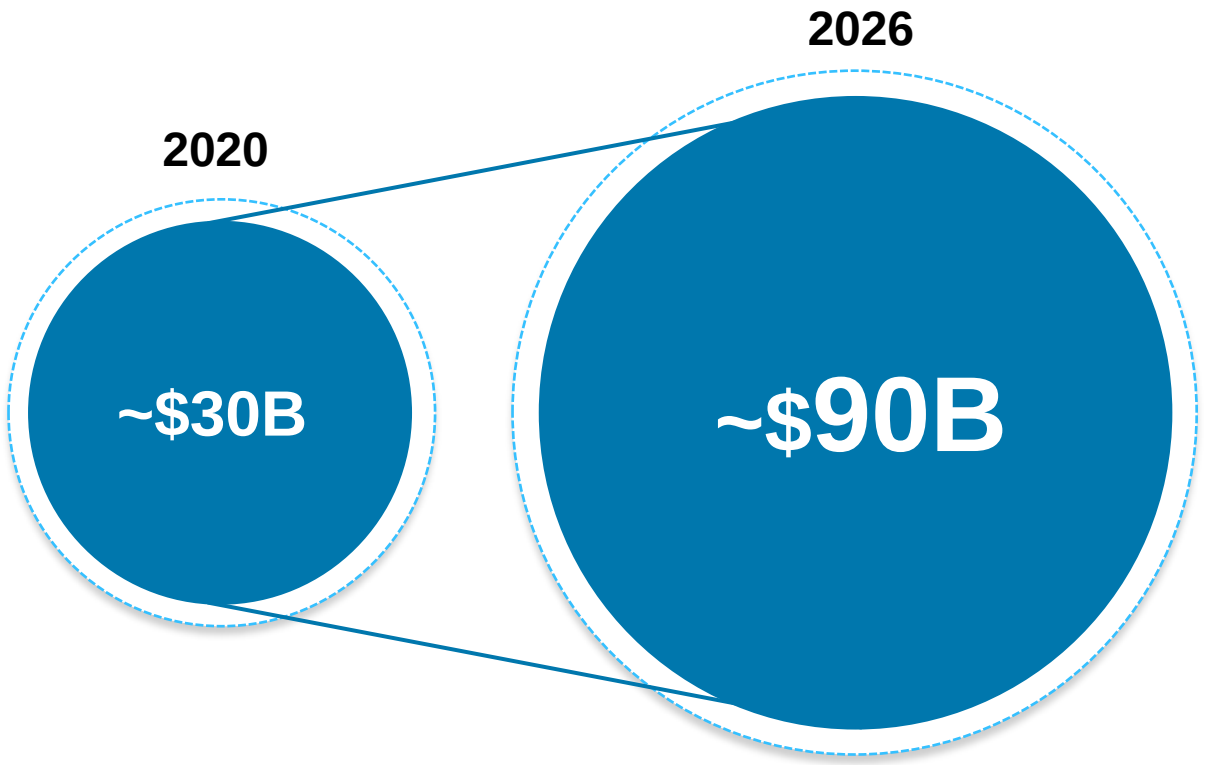
The Checkpoint Blockade Paradox

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



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

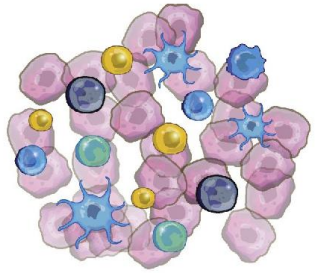
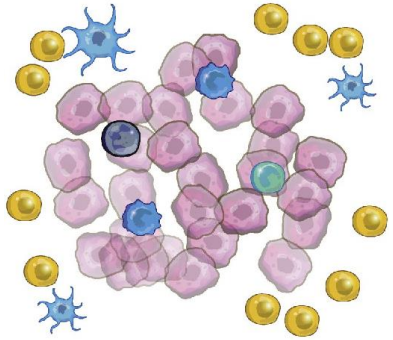
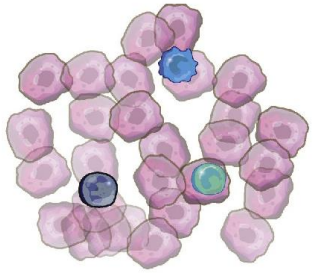
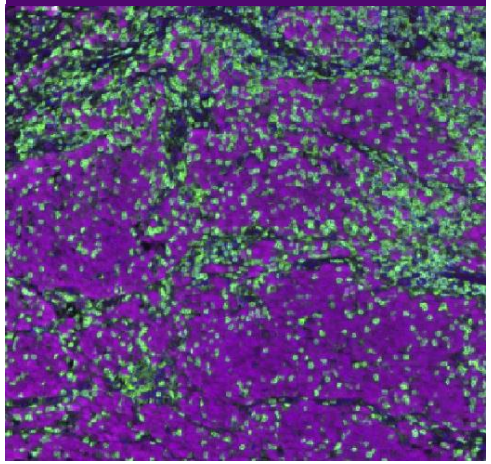
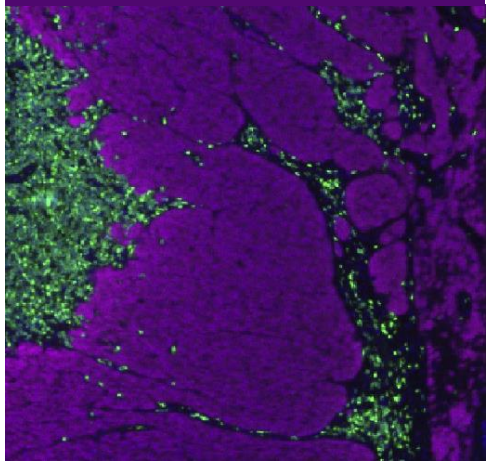
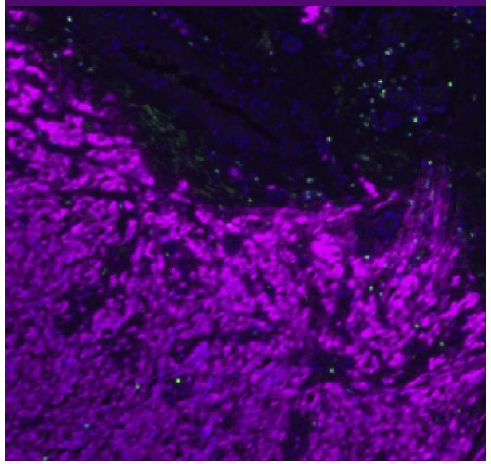


1. Gerber et al., Biochemical Pharmacology 2016
2. Market estimates from PD-1 and PDL-1 Inhibitors Market Size in 2021 – MarketWatch, 360 Research

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

Anti-PD-1 or PD-L1 Treatment

Green = T-cells
Purple = tumor

Responders	Non-Responders	
T-cells Inside Tumor	T-cells Inactive or Outside Tumor	T-cells Absent
		
Hot (inflamed) tumor	Cold (excluded) tumor	Cold (ignored) tumor
		

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

The Challenge of Next Generation Checkpoint Blockade

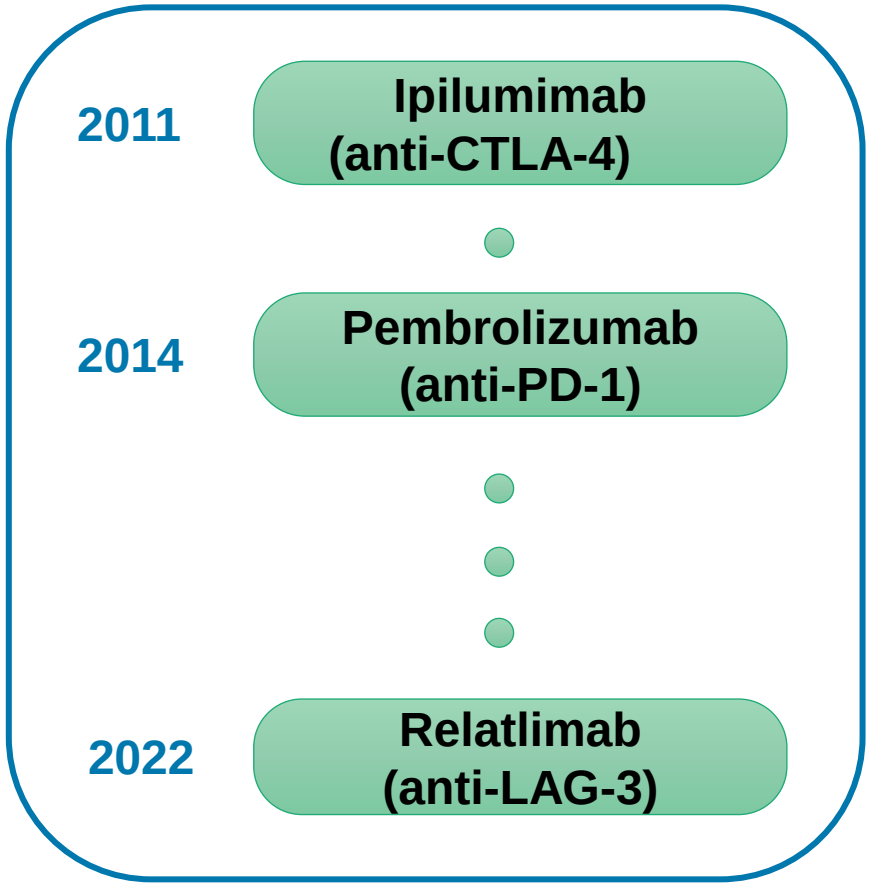
- **Few new classes of checkpoint blocking antibodies approved since landmark approvals of CTLA-4 and PD-1**

- **Antibodies blocking immune checkpoints are often limited by dose limiting toxicities that prevent maximal therapeutic outcomes**
  - Immune checkpoints are frequently expressed in normal tissues, including monocytes, neutrophils, NK cells, and T cells
  - Antibodies may encounter a pharmacological “sink” in those tissues and drive on-target/off-tumor toxicity, preventing therapeutic concentrations at the tumor

- **Next generation antibodies that have preferential activity and selectivity for tumors are needed to unleash the potential of immune targets**

- **pH-selective antibodies have demonstrated preferential biodistribution in tumors in mice, reduced toxicity in NHPs, and improved efficacy** [1]

Landmark Checkpoint mAb FDA Approvals



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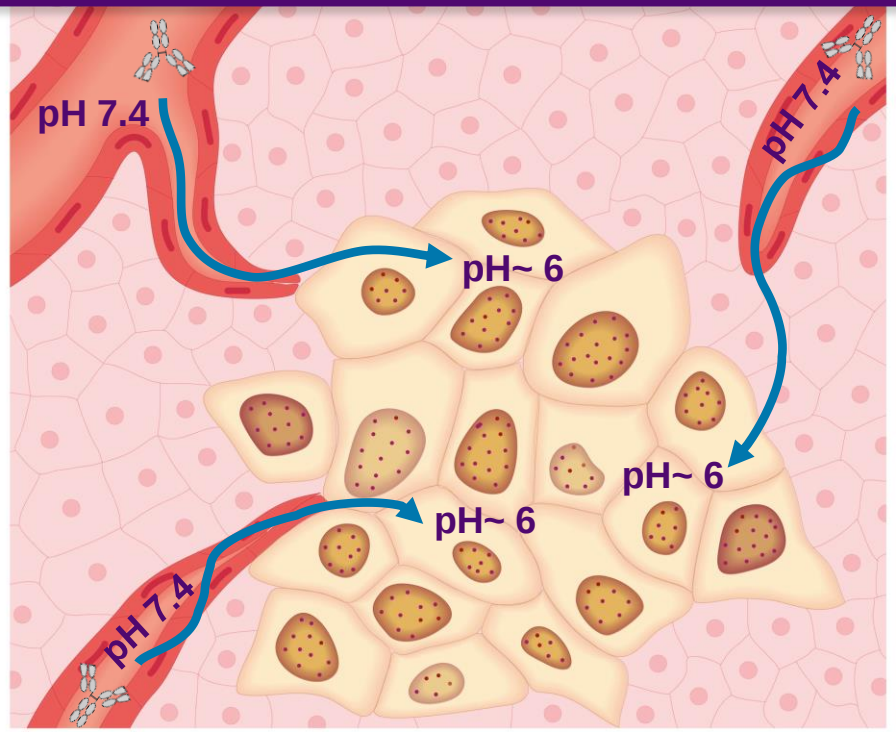
1 Lee et al, Mabs, 2022

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

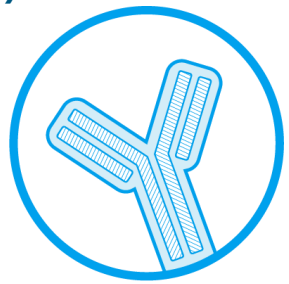
TMAb Platform

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

TMAb™
(Tumor Microenvironment Activated Biologics)



- Next-generation tumor activated mAbs
- Binding only in the low-pH tumor microenvironment; bypass other tissues
- Enable improved PK/PD and toxicity profiles
- Unlock previously undruggable immune checkpoint targets

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Increased Understanding of VISTA as a Promising Immune Checkpoint Target

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

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.

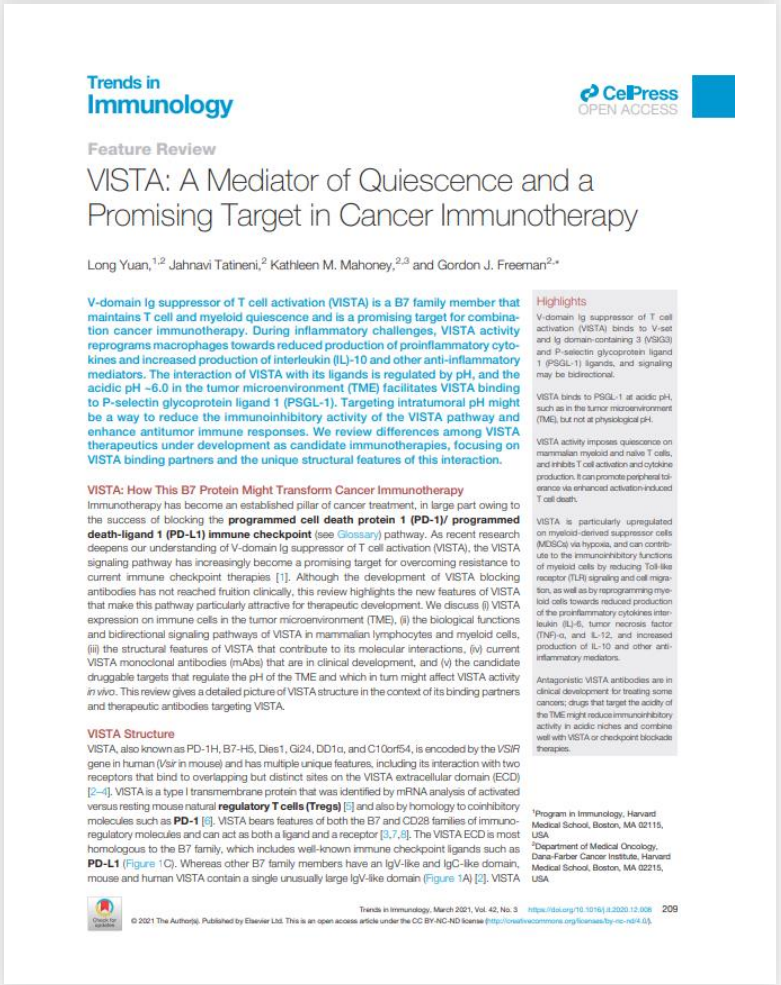
We did not observe clinical responses consisting of pathologic complete response, as we did previously for patients with bladder cancer[8]. To identify potential mechanisms that might explain this lack of response, we performed an unbiased gene expression study and found that ipilimumab therapy resulted in significant changes in the expression of a total of 690 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. (padsharma@mdanderson.org).

Received 16 December 2016; accepted 17 February 2017; published online 27 March 2017; doi:10.1038/nm.4308



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.

Highlights

V-domain Ig suppressor of T cell activation (VISTA) binds to V-set and Ig domain-containing 3 (VSIG3) and P-selectin glycoprotein ligand 1 (PSGL-1) ligands, and signaling may be bidirectional.

VISTA binds to PSGL-1 at acidic pH, such as in the tumor microenvironment (TME), but not at physiological pH.

VISTA activity imposes quiescence on mammalian myeloid and naïve T cells, and inhibits T cell activation and cytokine production. It can promote peripheral tolerance via enhanced activation-induced T cell death.

VISTA is particularly upregulated on myeloid-derived suppressor cells (MDSCs) via hypoxia, and can contribute to the immunoinhibitory functions of myeloid cells by reducing Toll-like receptor (TLR) signaling and cell migration, as well as by reprogramming myeloid cells towards reduced production of the proinflammatory cytokines interleukin (IL)-6, tumor necrosis factor (TNF)-α, and IL-12, and increased production of IL-10 and other anti-inflammatory mediators.

Antagonistic VISTA antibodies are in clinical development for treating some cancers; drugs that target the acidity of the TME might reduce immunoinhibitory activity in acidic niches and combine well with VISTA or checkpoint blockade therapies.

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

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[2]Department of Medical Oncology, Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA 02215, USA

VISTA: An Emerging Checkpoint Target on Myeloid Cells

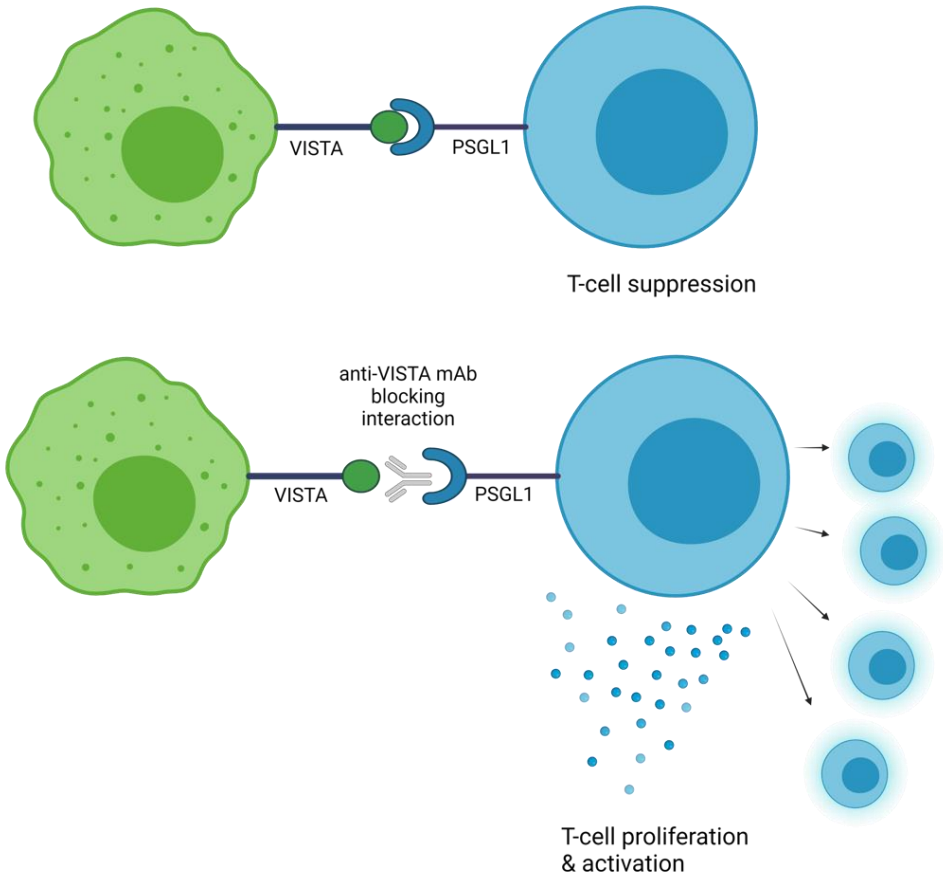
Target Overview:

- B7 family ligand
- Extensive expression on myeloid cells[1] correlating with poor survival rates across multiple cancers
- Novel development program with no approved therapies
- Large market opportunity

Sensei's Competitive Advantage:

- Extensive understanding of VISTA biology
- Unique tumor selective antibody

VISTA is a Negative Regulator of T cell Function



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

Key to Unlocking the Power of VISTA

1. Block the pH-dependent binding of VISTA to PSGL-1 on T cells at low pH

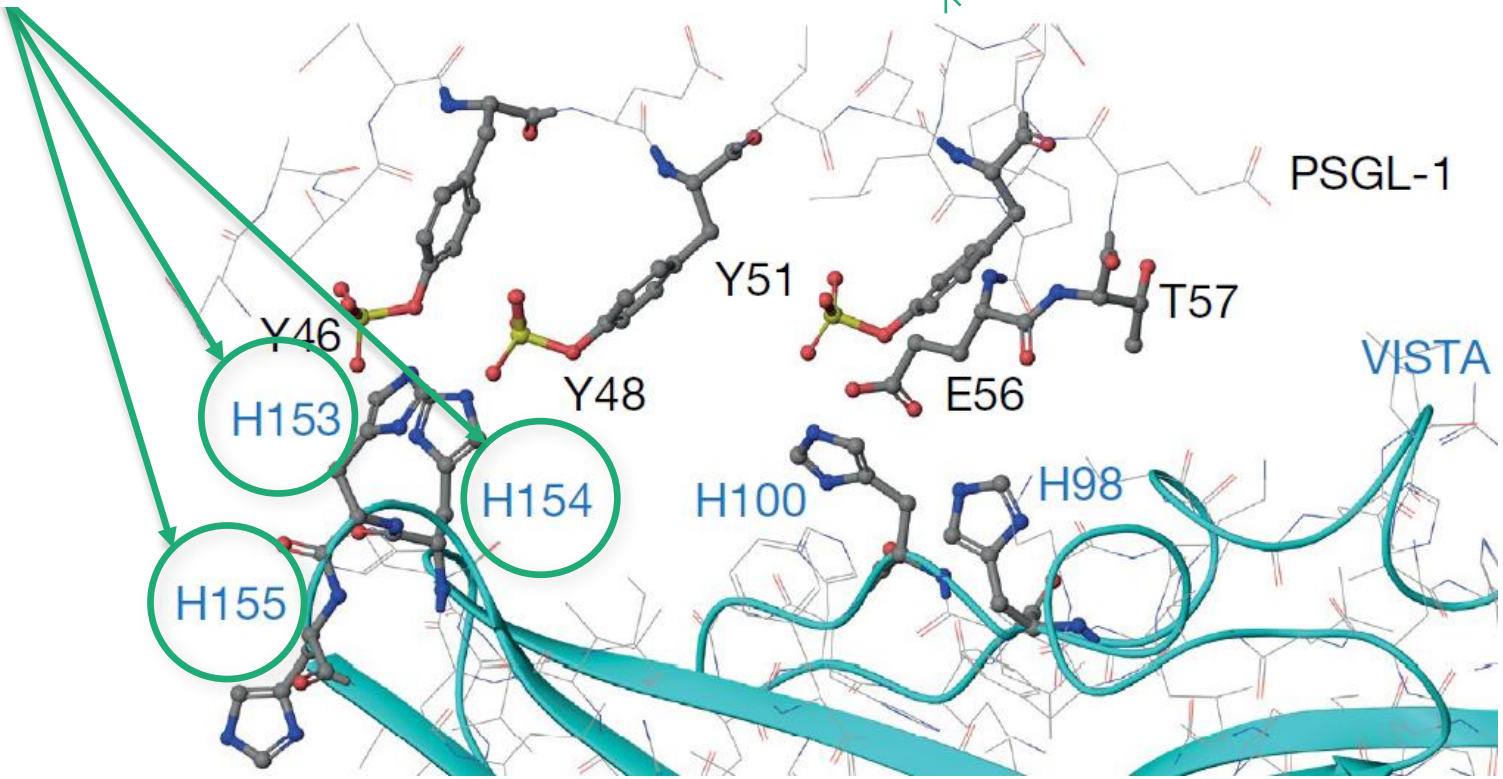
2. Selectively bind VISTA at low pH to avoid:
   - target mediated drug disposition (TMDD)
   - on-target/off-tumor side effects

3. Utilize an Fc-competent IgG backbone to engage and activate FcɣR on tumor-infiltrating myeloid cells

SNS-101

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

Antibodies that block protonated VISTA histidines interrupt PSGL-1 binding[1]



- 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

1. Johnston et al., Nature 2019

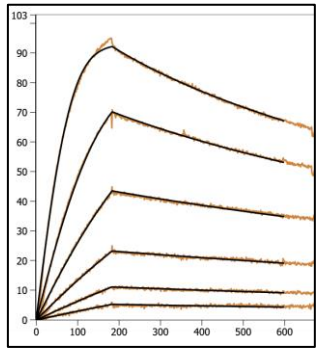
SNS-101 Has >600-Fold Selectivity for VISTA^{pH6}

- >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)

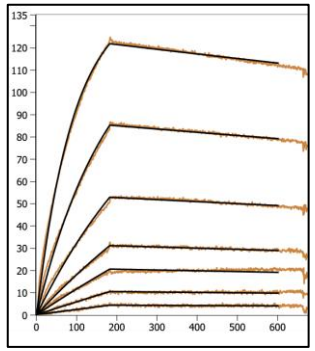
pH 6.0

SNS-101



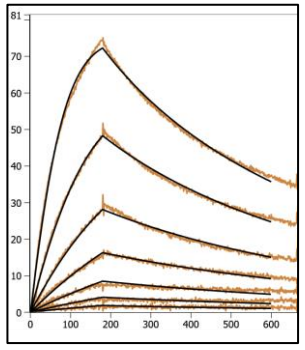
k_a = 4.59E+06 $M^{-1}s^{-1}$
K_d = 1.00E-03 s^{-1}
K_D = **2.18E-10 M**
[Vista] – 7.5 nmol – 0.2 nmol

pH-dependent "benchmark"



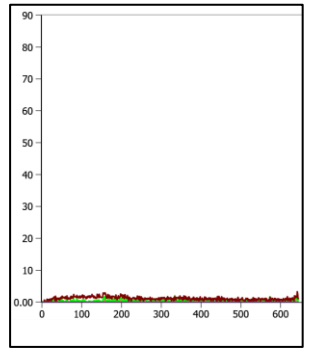
k_a = 7.11E+05 $M^{-1}s^{-1}$
K_d = 1.78E-04 s^{-1}
K_D = **2.5E-10 M**
[Vista] – 15 nmol – 0.2 nmol

pH-independent "benchmark"



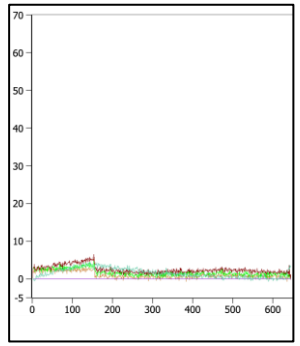
k_a = 1.45E+06 $M^{-1}S^{-1}$
K_d = 2.25E-03 S^{-1}
K_D = 1.55E-09 M
[Vista] – 15 nmol – 0.2 nmol

pH7.4



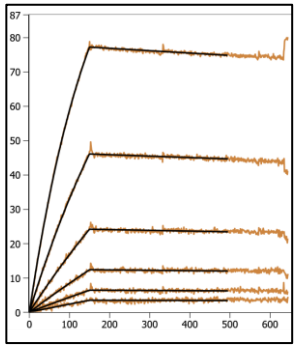
[Vista] – 7.5 nmol – 0.2 nmol

pH-dependent "benchmark"



[Vista] – 15 nmol – 0.2 nmol

pH-independent "benchmark"



k_a = 4.162E+05 $M^{-1}S^{-1}$
K_d = 9.27E-05 S^{-1}
K_D = 2.23E-10 M
[Vista] – 15 nmol – 0.47 nmol

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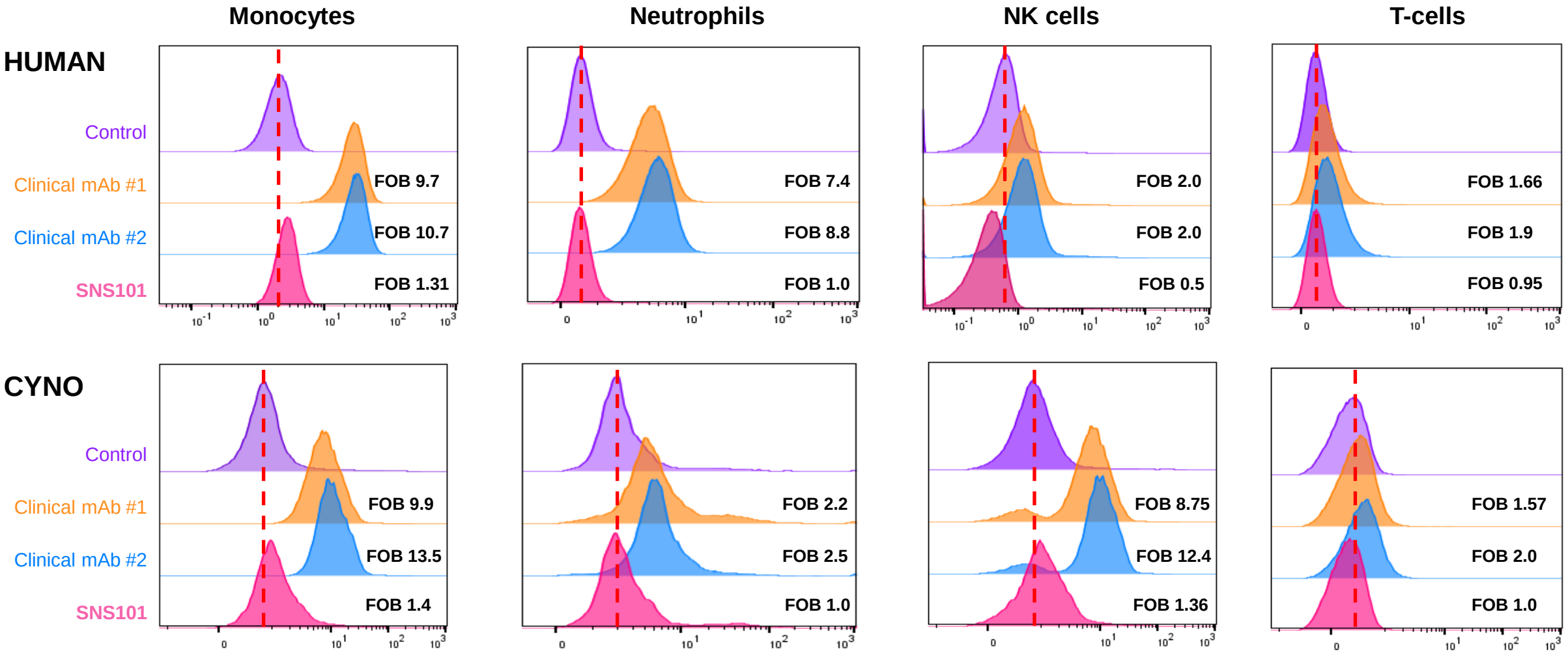
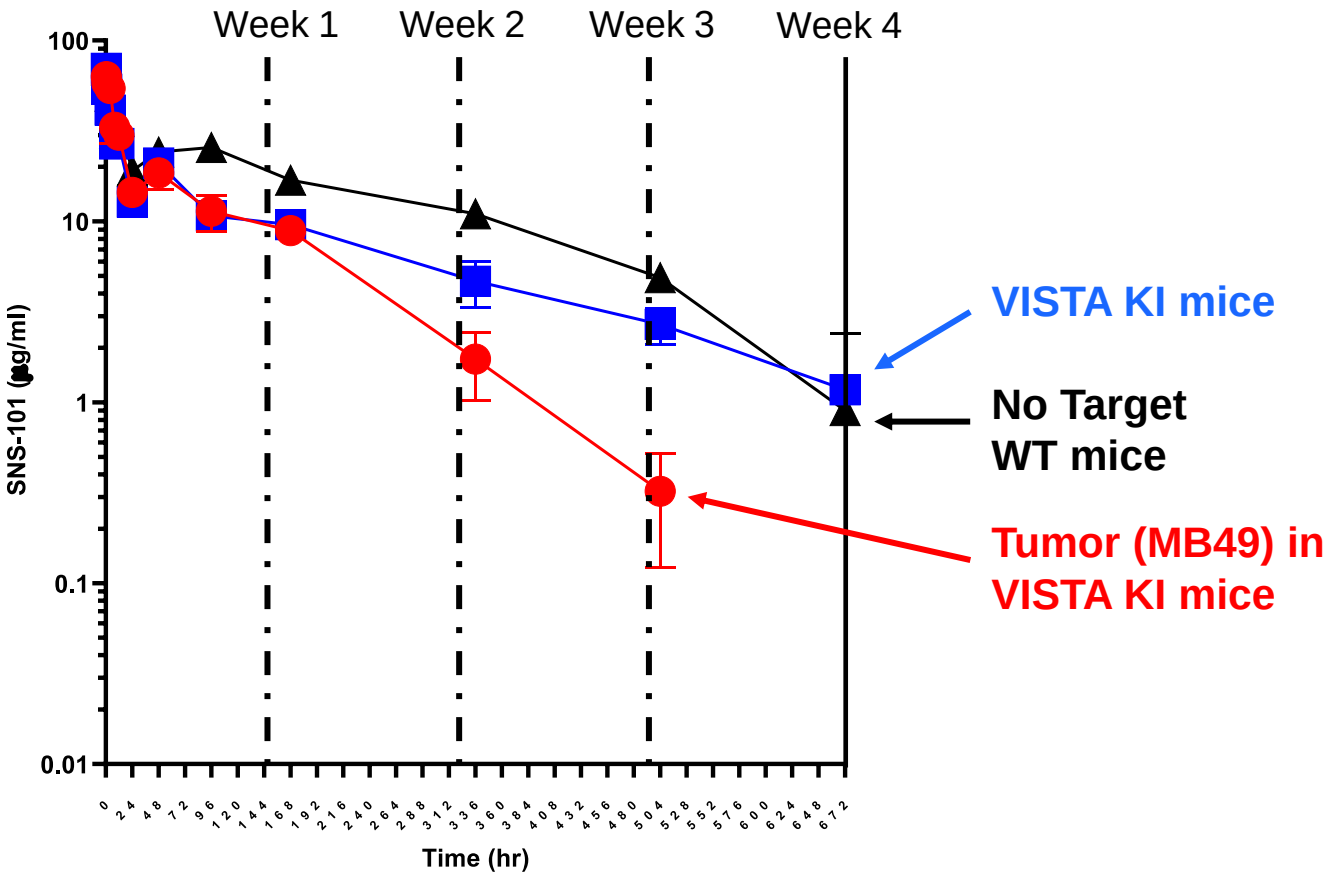
SITC 2021: Poster titled: Antagonistic pH-selective VISTA antibody SNS-101 potentiates anti-PD-1/PD-L1-induced anti-tumor immunity.

No Significant Binding of SNS-101 to Monocytes, Neutrophils, NK Cells and T-cells in Whole blood at Physiological pH



SNS-101 Displays a Favorable PK Profile
No significant TMDD in human VISTA KI mice

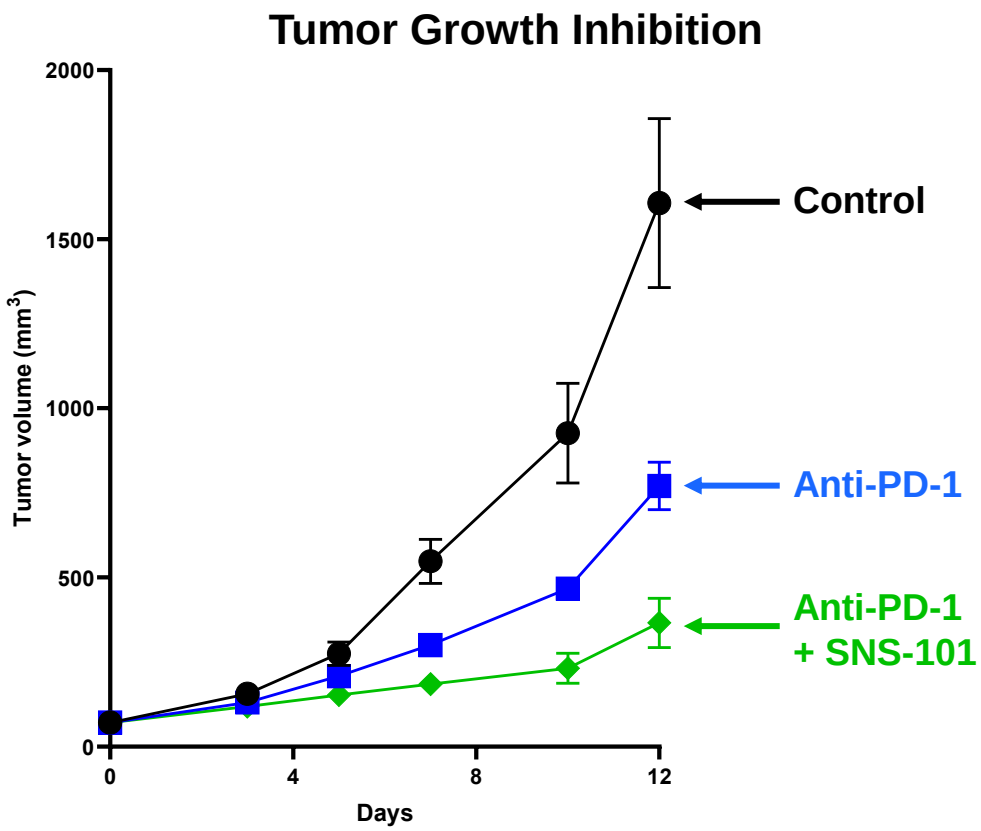
Pharmacokinetics of Single Dose 5 mg/kg SNS-101 in VISTA Knock-in Mice

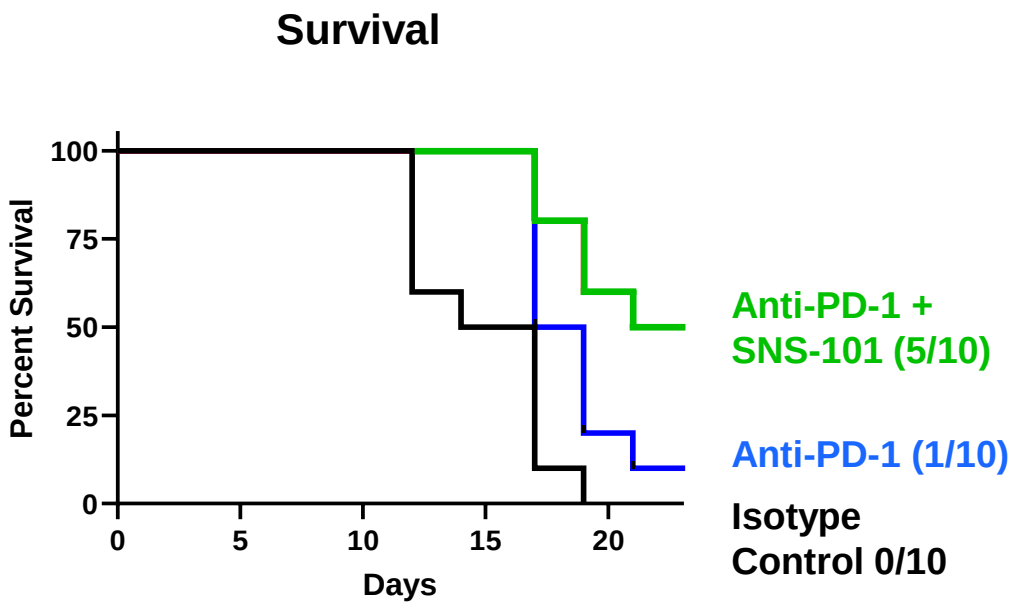


Demonstrated a long mean residence time in the blood, indicating a lack of significant target-mediated drug disposition (TMDD) and clearance in non-malignant tissues

SNS-101 Demonstrates Activity in a PD-1 Resistant Syngeneic Tumor Model

SNS-101* in Combination with Anti-mouse PD-1





*SNS-101 was grafted on to a mouse IgG2a framework to decrease anti-drug antibody production

SNS-101 Is a Differentiated Anti-VISTA Antibody

TMAb Platform

	SNS-101 sensei BIO	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	No
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	Phase I
Clinical Data / Notes	• Demonstrated activity in preclinical models • Demonstrated potential for best-in-class safety profile and PK in mouse model • 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 • Phase I ongoing	• Not published	• Not published

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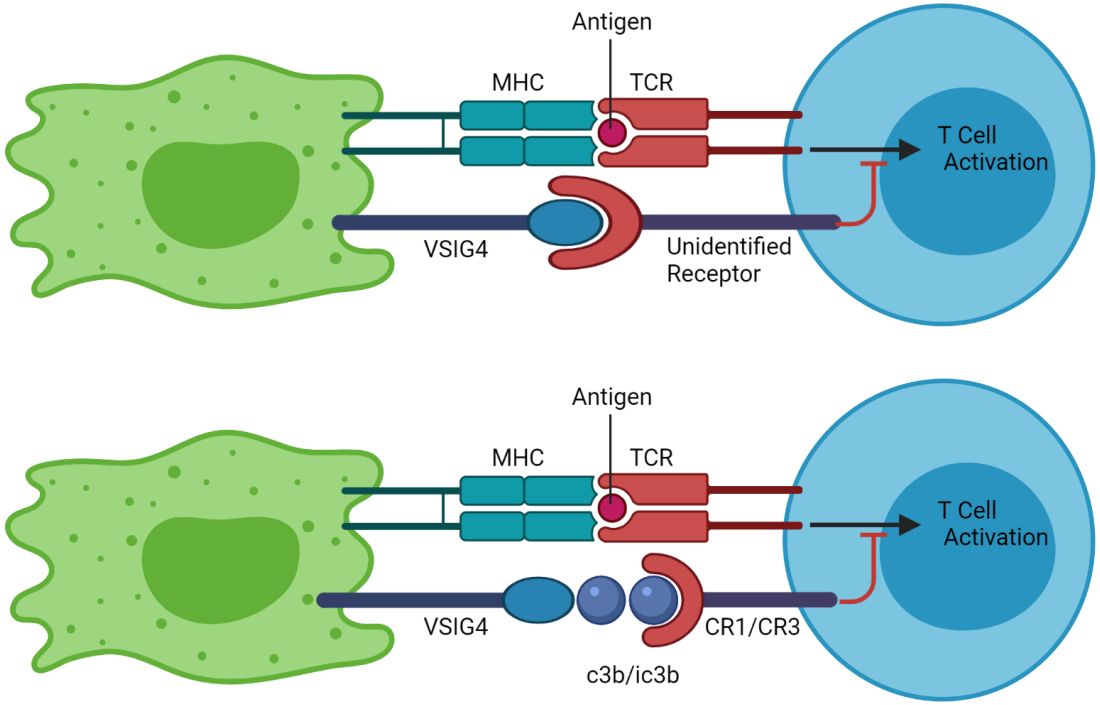
Johnston et al, Nature, 2019; Kineta website; Snyder et al, AACR Annual Meeting 2016; Pierre Fabre website; Hummingbird website; Thakkar et al, J of Immunother Cancer, 2022

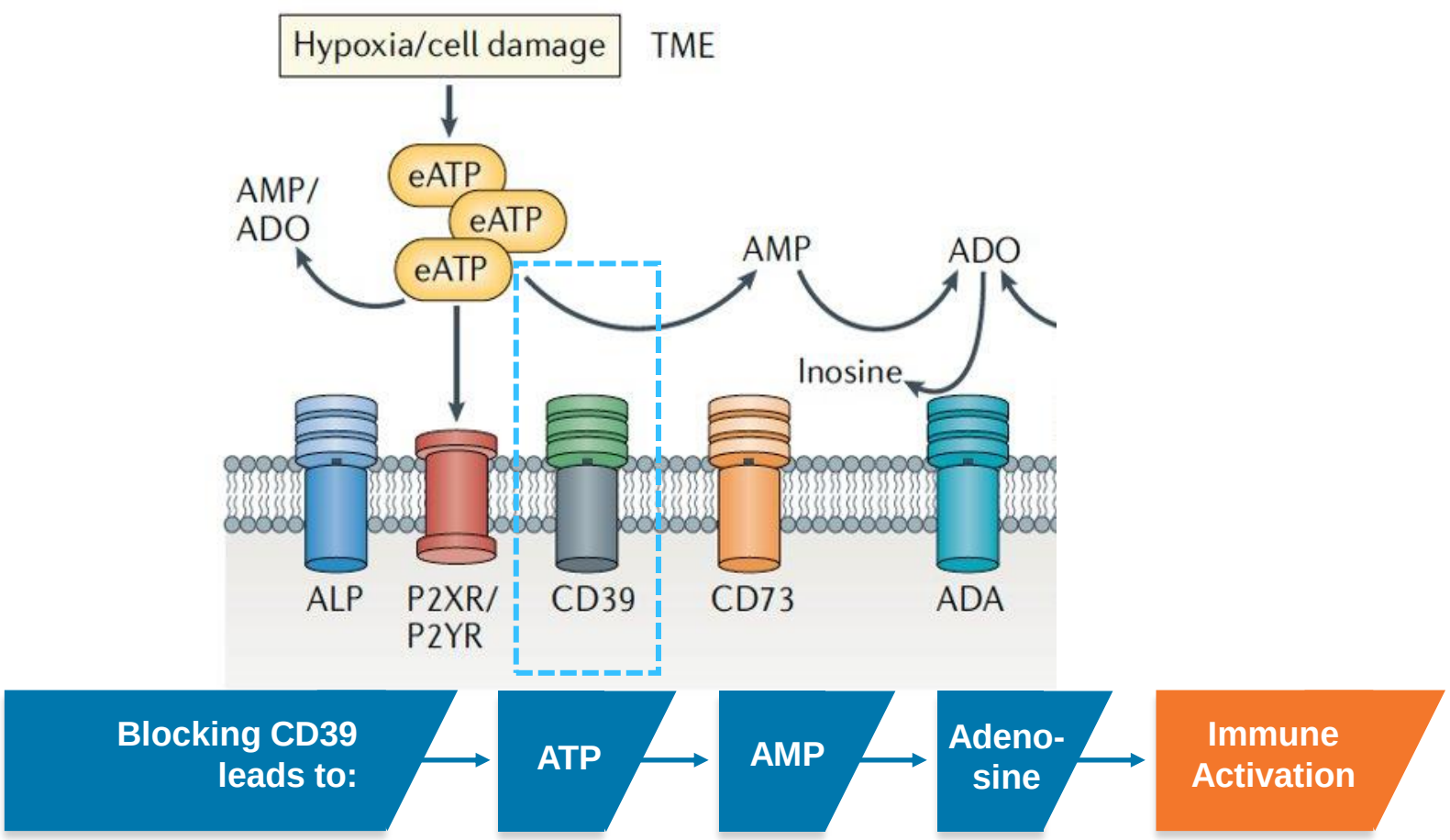
VSIG4 Plays a Critical Suppressive Role in T-cell Activation



- B7 family related protein
- Expressed primarily on macrophages and inhibits T-cell activation
- Generated first set of antibodies; currently screening

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

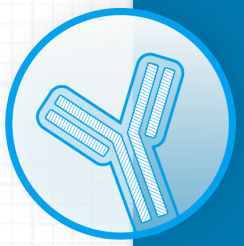
ENTPDase1 (CD39) is the Rate Limiting Enzyme in the Production of Immunosuppressive Adenosine



- Primary function is conversion of extracellular ATP / ADP to adenosine, which exerts immunosuppressive properties through binding to A2a/A2b receptors
- Expressed on various immune cells in both tumors and normal tissues
- Development of a TMAb antibody has potential for improved safety and PK profile compared to competitor CD39 mAbs

Figure adapted from: Moesta et al, *Nat Rev Immunol.* 2020; 20 (12)

Expected Program Milestones



SNS-101 (anti-VISTA)

- **1H 2023:** IND filing
- **Mid-2022:** Toxicology and PK data

SNS-102 (anti-VSIG4)

- **2023:** Select product candidate / initiate IND-enabling studies

SNS-103 (anti-ENTPDase1/CD39)

- **2023:** Select product candidate

sensei™ BIO

Positioned to Drive Value with Next Generation Product & Platform Development

Two Platforms Generate Broad Pipeline Opportunities

TMAb™

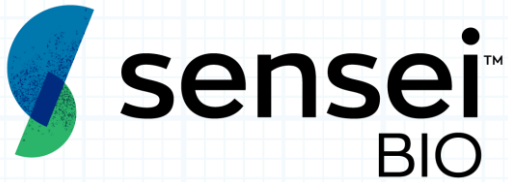
ImmunoPhage™

Innovative Pipeline of Differentiated IO mAbs

Strong Cash Position Ended 1Q 2022: $136M ** Cash runway into Q1 2025

Business Development

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



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