VISTA Science Symposium

November 16, 2021

Guest Speaker:

Prof. Robert Schreiber

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Sensei IOAB member

Sensei Presenters:

John Celebi
Chief Executive Officer

Dr. Robert Pierce
Chief Scientific Officer

Dr. Edward van der Horst
SVP, TMAb Antibody Development

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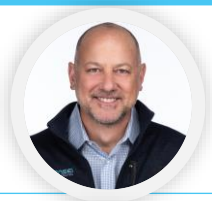
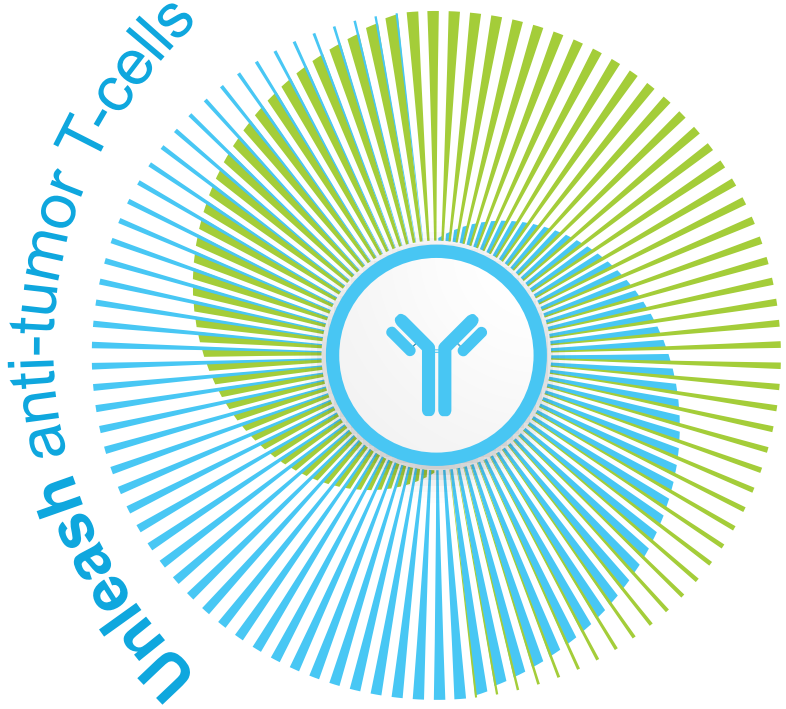
Agenda

Speaker	Topics
 **John Celebi** *President & CEO*	• Welcome/TMAb Mission
 **Professor, Robert Schreiber, Ph.D.** *Washington University School of Medicine* *Sensei IOAB member*	• VISTA biology
 **Robert Pierce, M.D.** *Chief Scientific Officer*	• SNS-101 preclinical data highlights from SITC
 **Edward van der Horst, Ph.D.** *SVP, TMAb Antibody Development*	• Join for Q&A

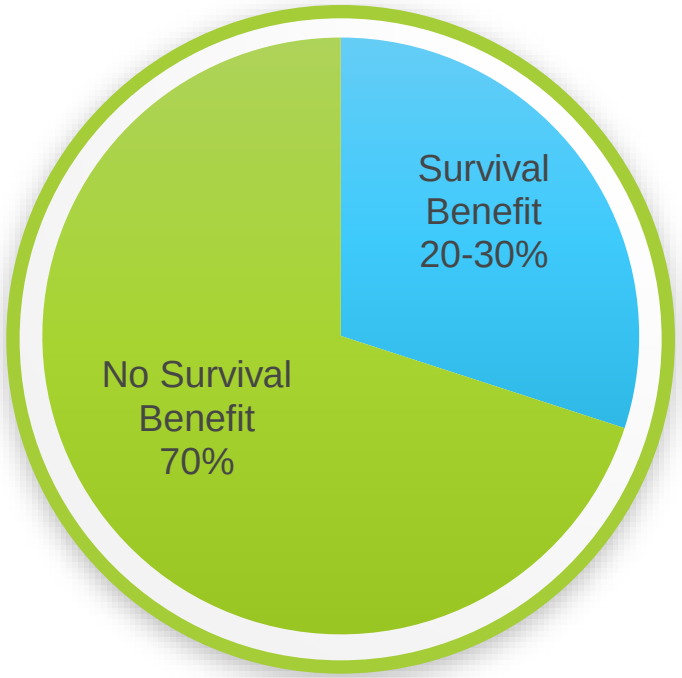
Our TMAb (Tumor Microenvironment Activated biologics) Platform Mission

Leverage unique features of the tumor microenvironment to selectively activate biologics that unleash clinically meaningful anti-cancer immune responses

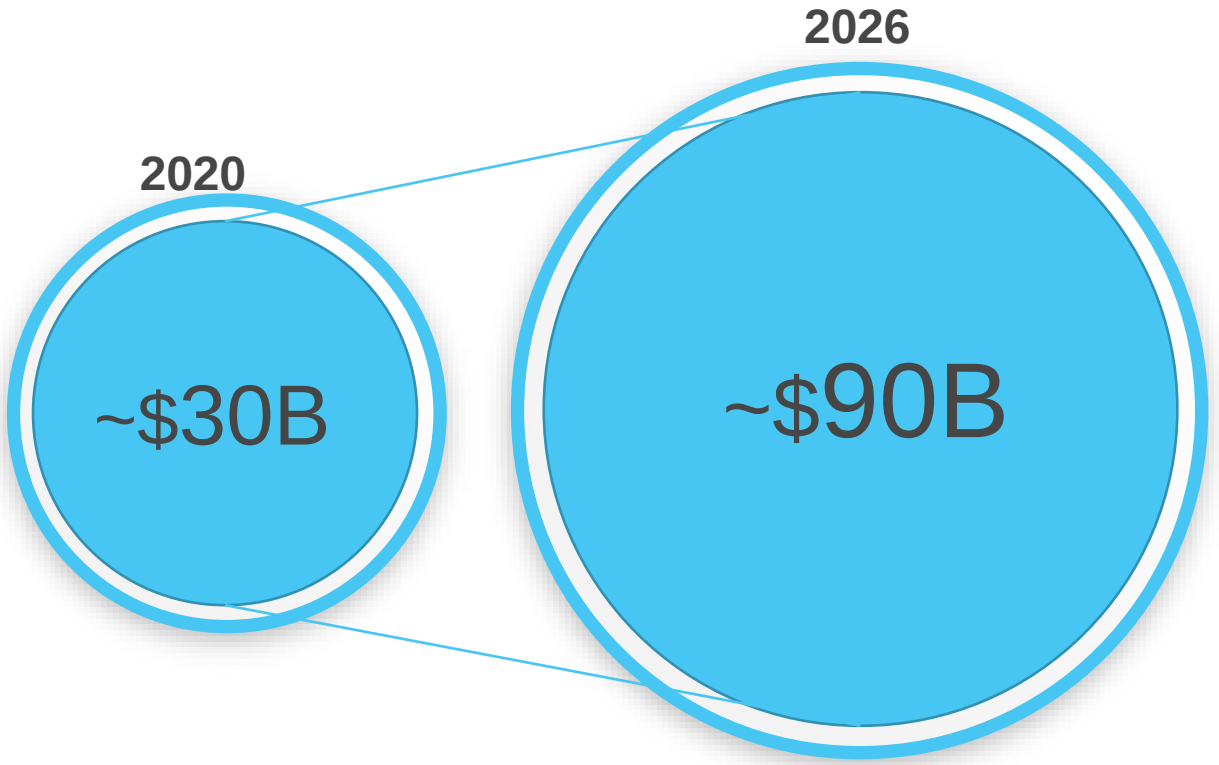


The Modern-Day Challenge in Immuno-Oncology

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



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



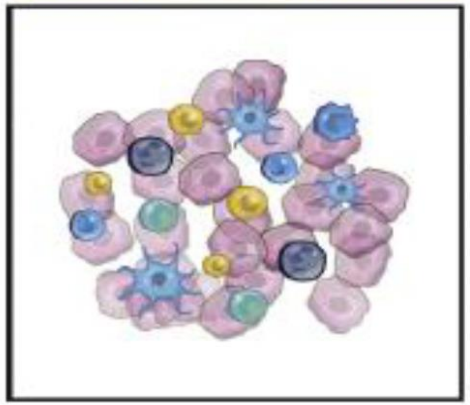
1. Gerber et al., Biochemical Pharmacology 2016
2. Market estimates from PD-1 and PDL-1 Inhibitors Market Size in 2021 – MarketWatch, 360 Research

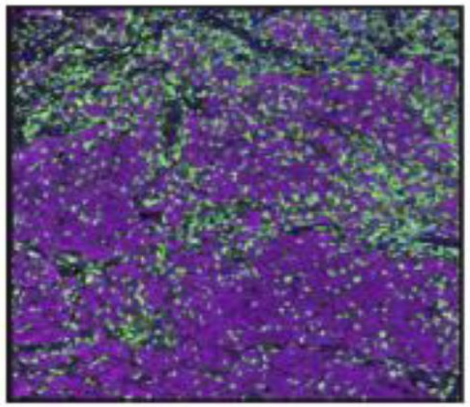
Two Major Types of Non-Responders to PD-1 Blockade

Responders

T-cells Inside Tumor

Hot (inflamed) tumor

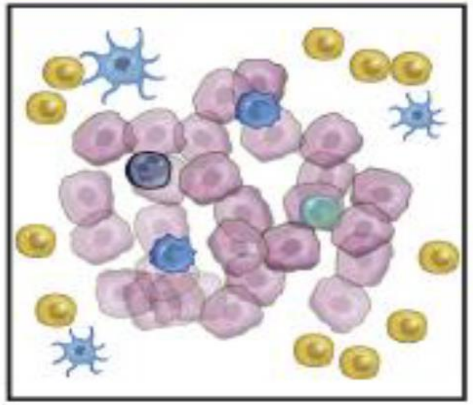


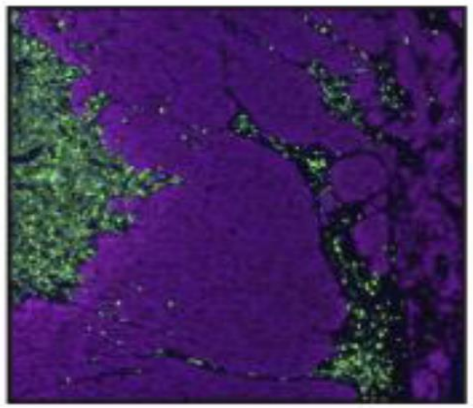


Non-Responders

T-cells Inactive or Outside Tumor

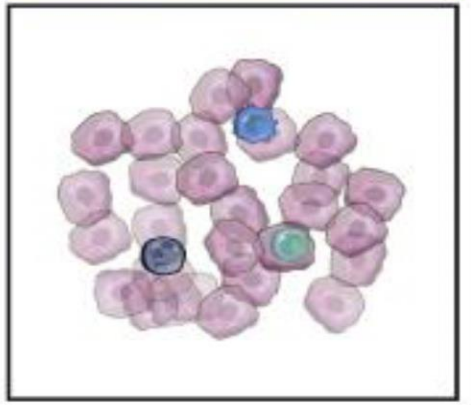
Cold (excluded) tumor

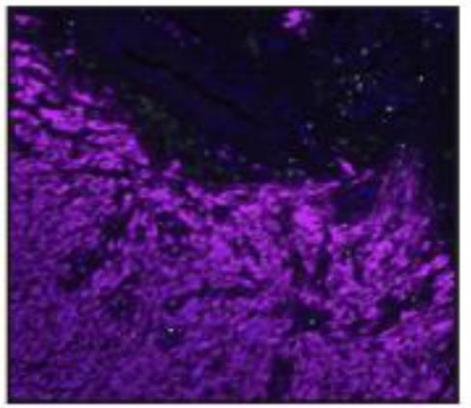




T-cells Absent

Cold (ignored) tumor



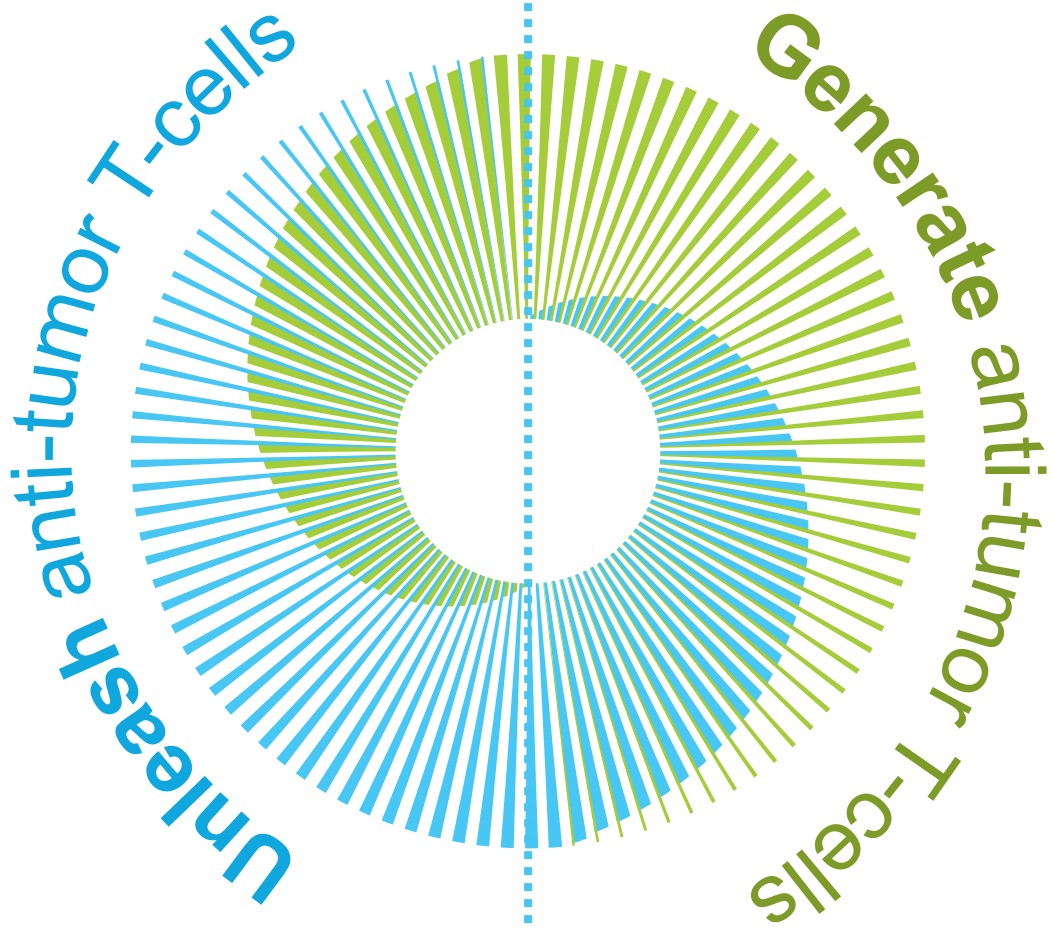


Green = T-cells
Purple = tumor

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

Two Platforms to Unleash Anti-Cancer T-cell Activity

TODAY'S DISCUSSION



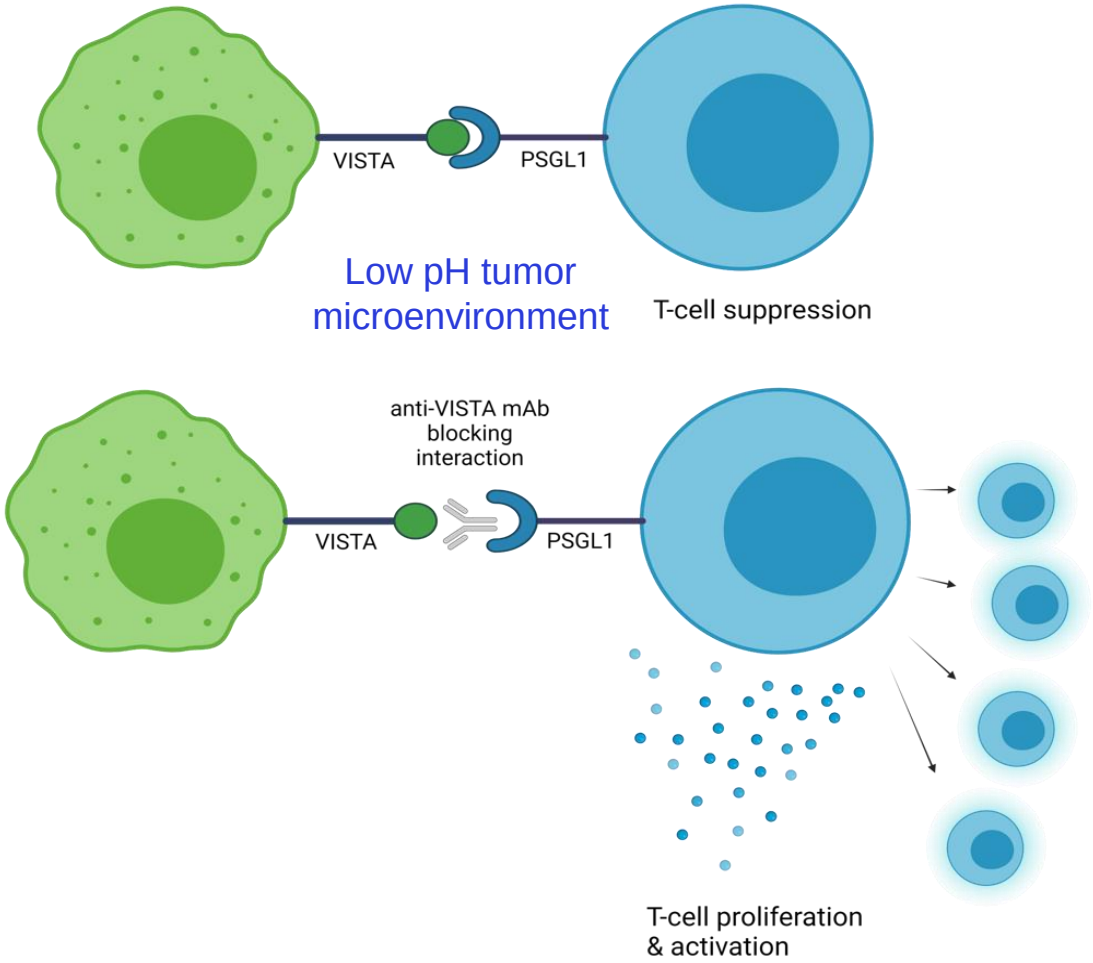
TMAb™ (Tumor Microenvironment Activated Biologics) Platform

- Next-generation tumor activated mAbs

ImmunoPhage™ Platform

- Powerfully self-adjuvanted nanoparticle vaccine that drive tumor-specific T cell responses

VISTA (V-domain Ig suppressor of T cell activation)



Target Overview:

- Established immune checkpoint target to overcome checkpoint resistance
- Large market opportunity
- Extensive expression on normal myeloid cells

Sensei's Competitive Advantage:

Leverage extensive understanding of VISTA biology to deliver a differentiated approach

SNS-101:

- A 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 suppressive myeloid cells in the tumor microenvironment

Leveraging a Team with Decades of Experience

Dr. Schreiber

VISTA Biology

VISTA (B7-H5) is recognized an important immune checkpoint and B7 family member that is expressed on myeloid cells, a hub of immunosuppressive activity, and is activated via binding to its receptor on T-cells (PSGL-1) at sub-physiologic pH

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 has Emerged as an Important Checkpoint Regulator 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**).

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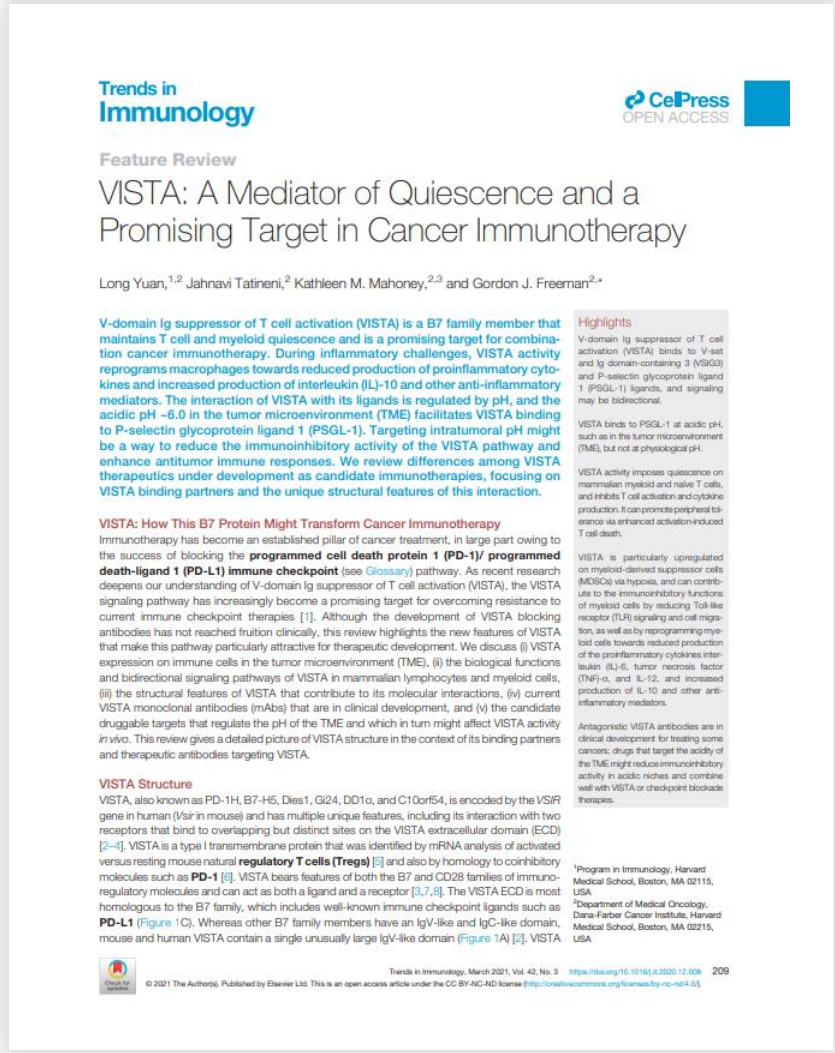
nature medicine

Gao. J., et al



Trends in Immunology

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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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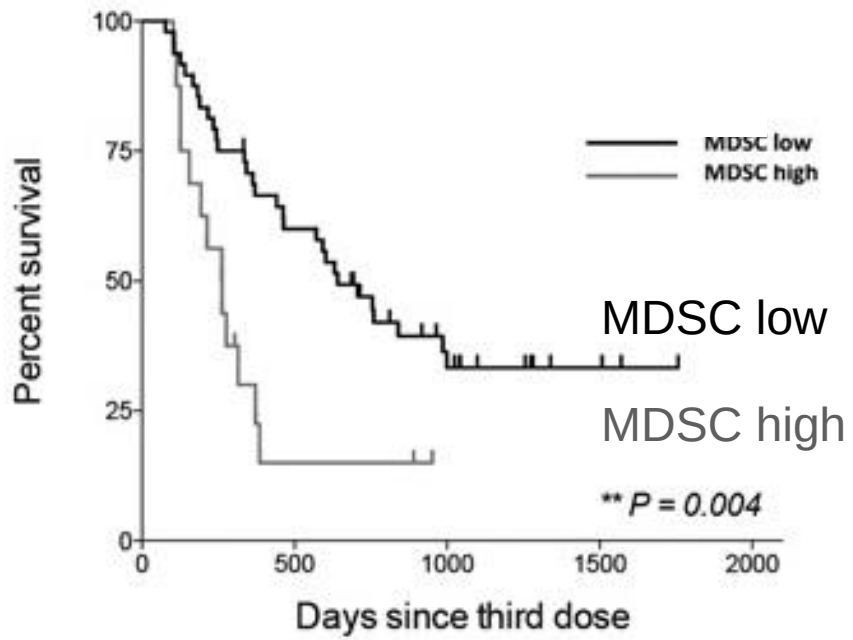
Science that inspires

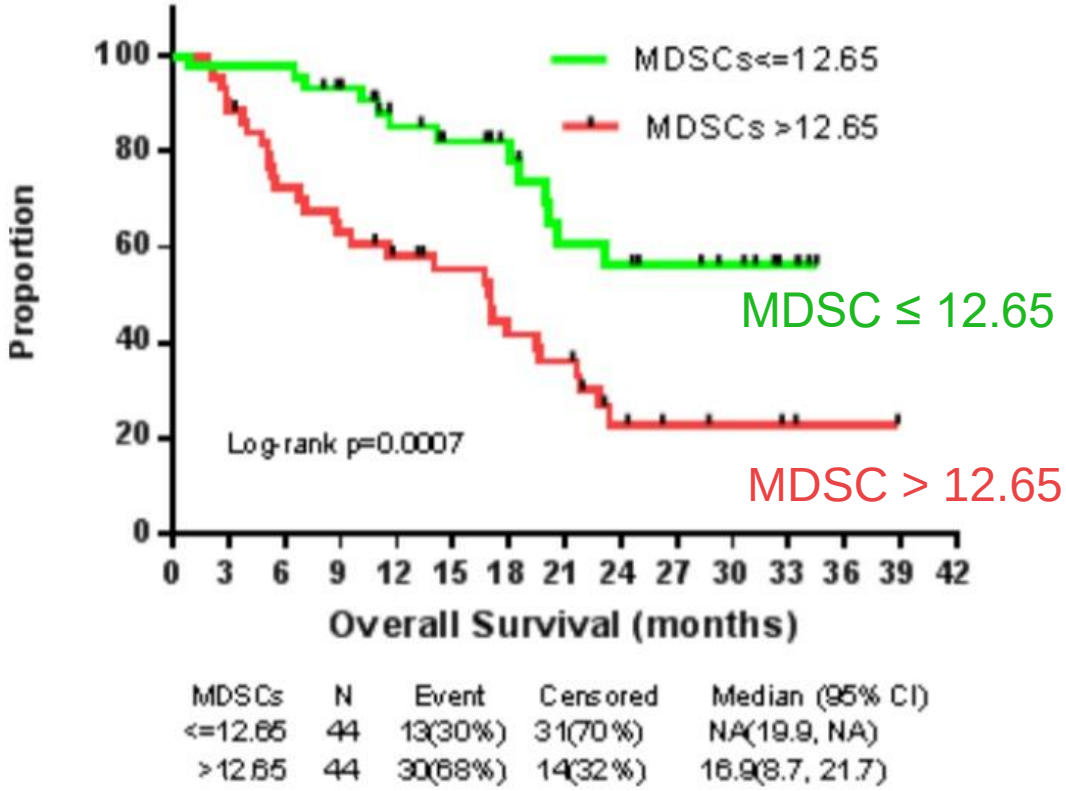
Yuan, L., et.al

Patients with High Circulating Myeloid Cells Have Shown Lower Overall Survival When Treated with Checkpoint Blockade

Ipilimumab-treated Melanoma Patients



Nivolumab-treated Melanoma Patients

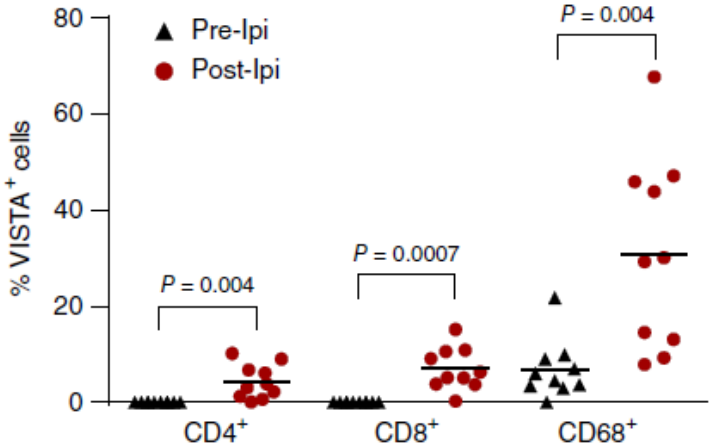


Kitano et al., *Cancer Immunol Res*. 2012
Weber et al., Cancer Immunol Res. 2016

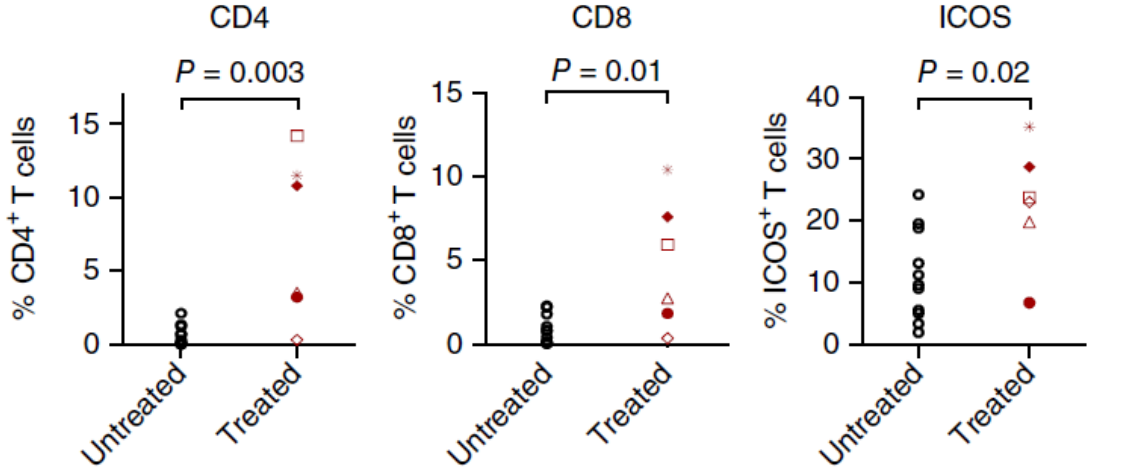
VISTA may be a Compensatory Pathway Following Checkpoint Therapy

Can targeting VISTA augment T-cell checkpoint blockade in refractory tumors?

VISTA Increases on Prostate Tumor Cell Infiltrates Following Ipilumimab Treatment

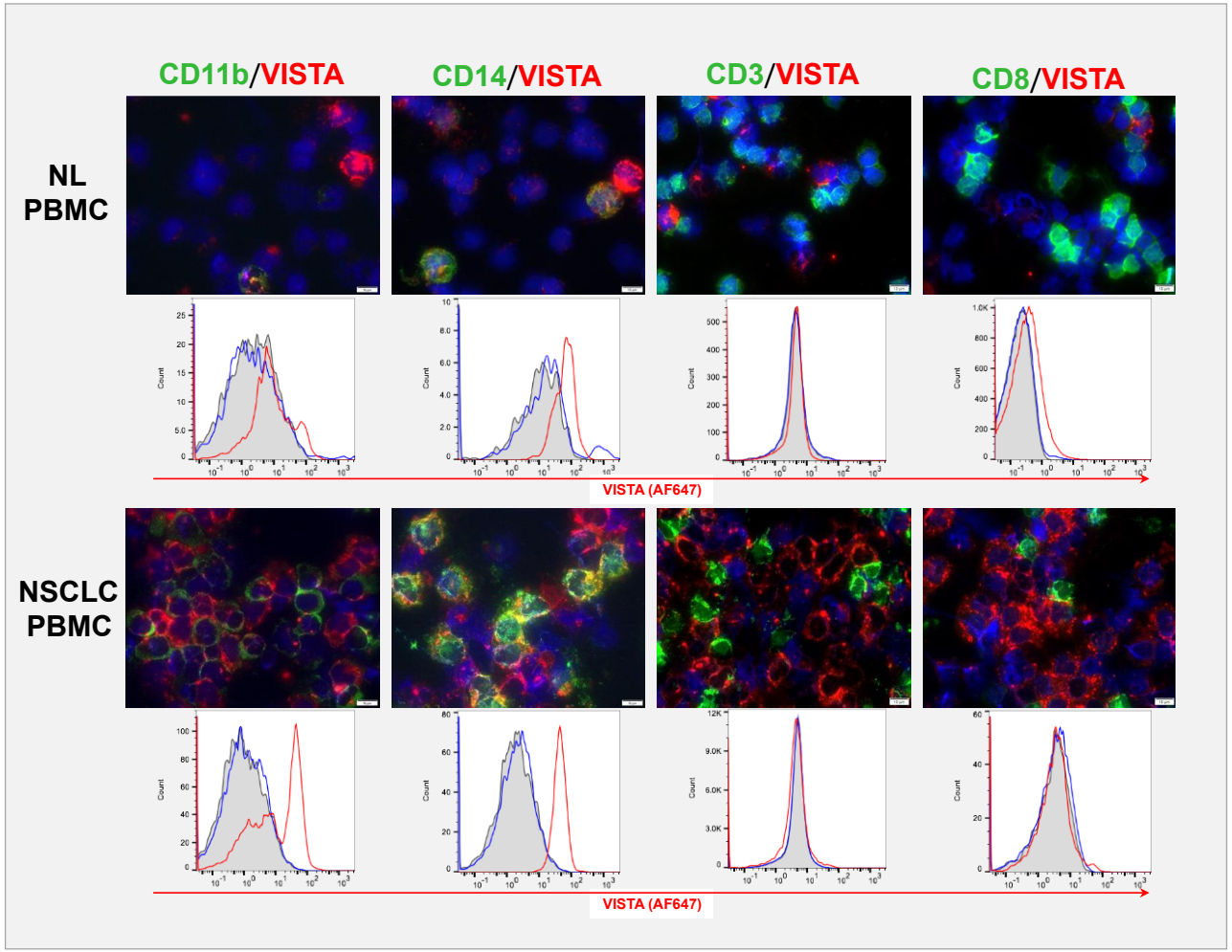


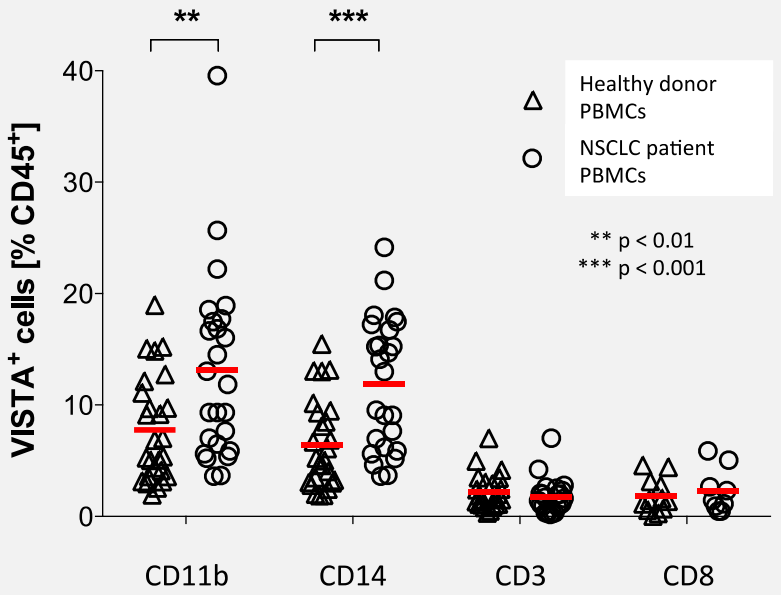
Prostate Tumor Cell Infiltrates Increase Following Ipilumimab Treatment



Gao et al., Nat Med. 2017

VISTA Expression Increases in PBMC Subsets of Patients with Non-Small Cell Lung Cancer (NSCLC)

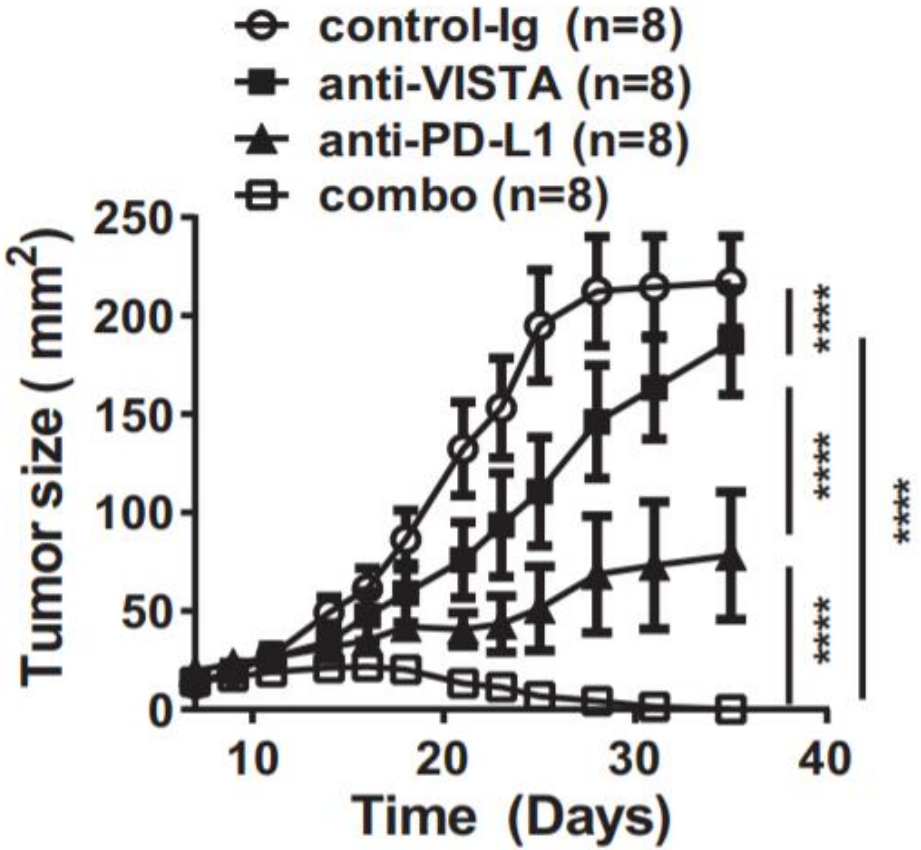


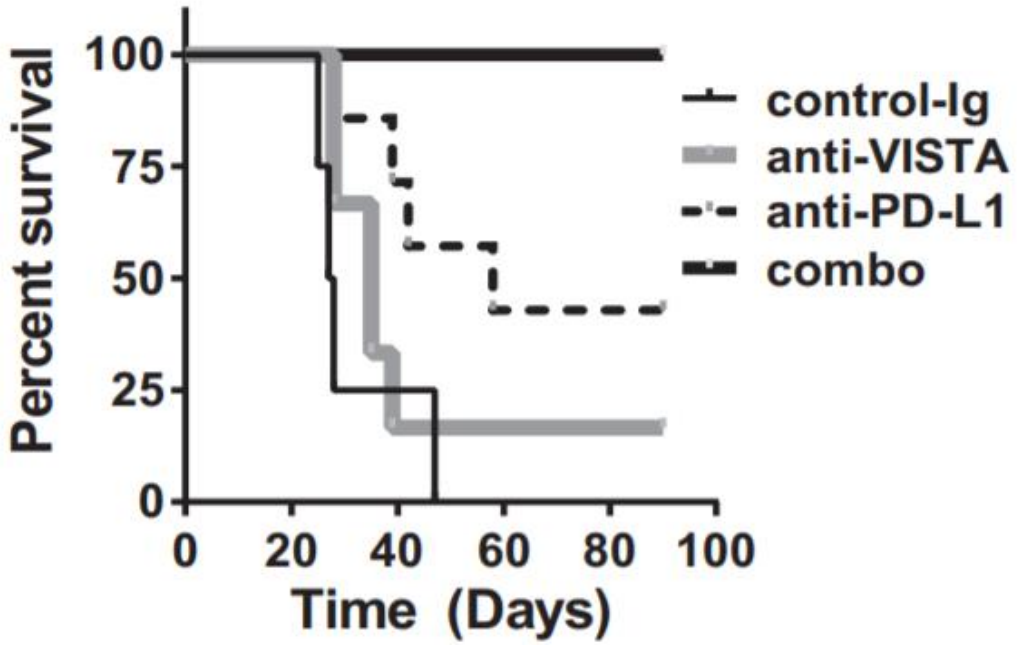


Cell Subset	VISTA [Copy Numbers]	
	Normal	NSCLC
CD11b+	102611	***141143***
CD14+	102937	***191727***
CD3+	Below Level of Quantitation	
CD8+	Below Level of Quantitation	

VISTA Blockade Synergizes With PD-1/L-1 Pathway Inhibition

CT26 Syngeneic Tumor Model



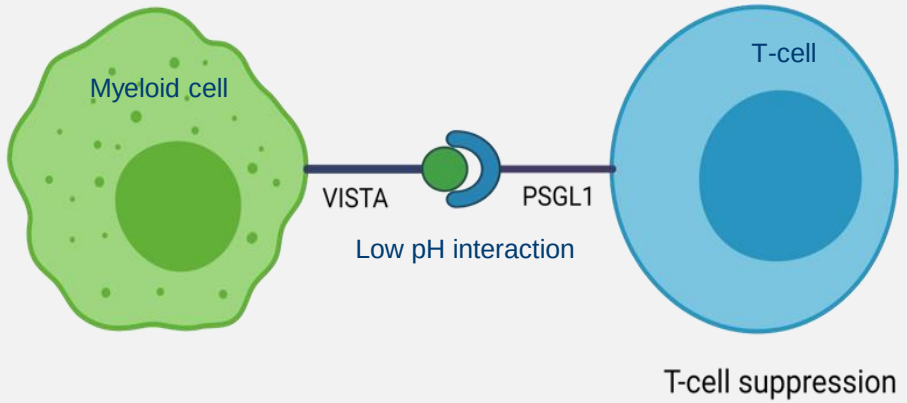


Liu J. et al. *PNAS* 2015

VISTA is an Emerging Target on Myeloid Cells and Key Resistance Mechanism for PD-1/PD-L1 Blockade

- VISTA is a B7 family (e.g., same protein family as PD-L1) ligand expressed on **myeloid cells**, a hub of immunosuppressive activity[1]
- VISTA is a key player in controlling checkpoint blockade
- VISTA has been implicated in **resistance to PD-1/PD-L1 inhibitors**[2]

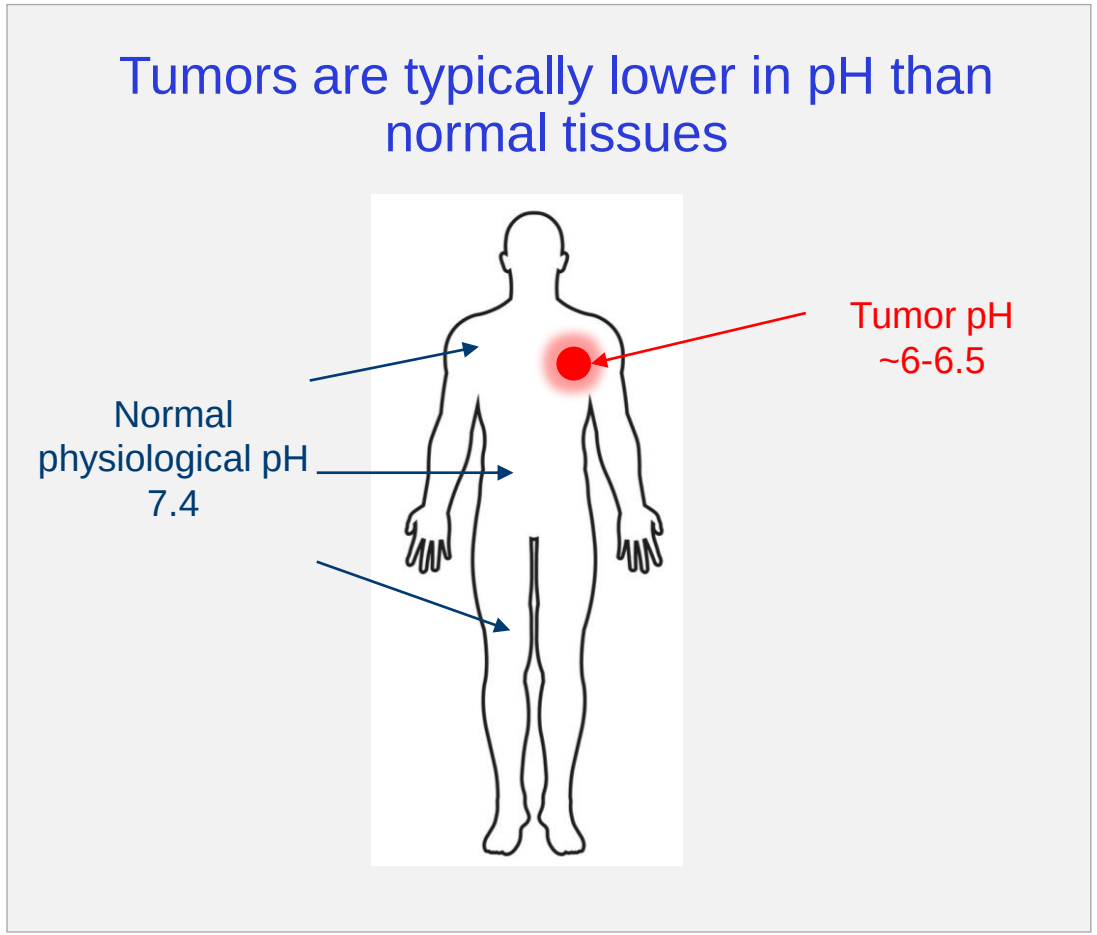
VISTA is a Negative Regulator of T cell Function at Low pH



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

VISTA is an Emerging Target on Myeloid Cells and Key Resistance Mechanism for PD-1/PD-L1 Blockade

- Tumors are typically lower in pH than normal tissues
- At low pH, key amino acids in VISTA become protonated, changing its charge, and likely, its shape
  - This change activates VISTA **enabling VISTA to** bind to PSGL-1 on T cells, engaging its checkpoint function

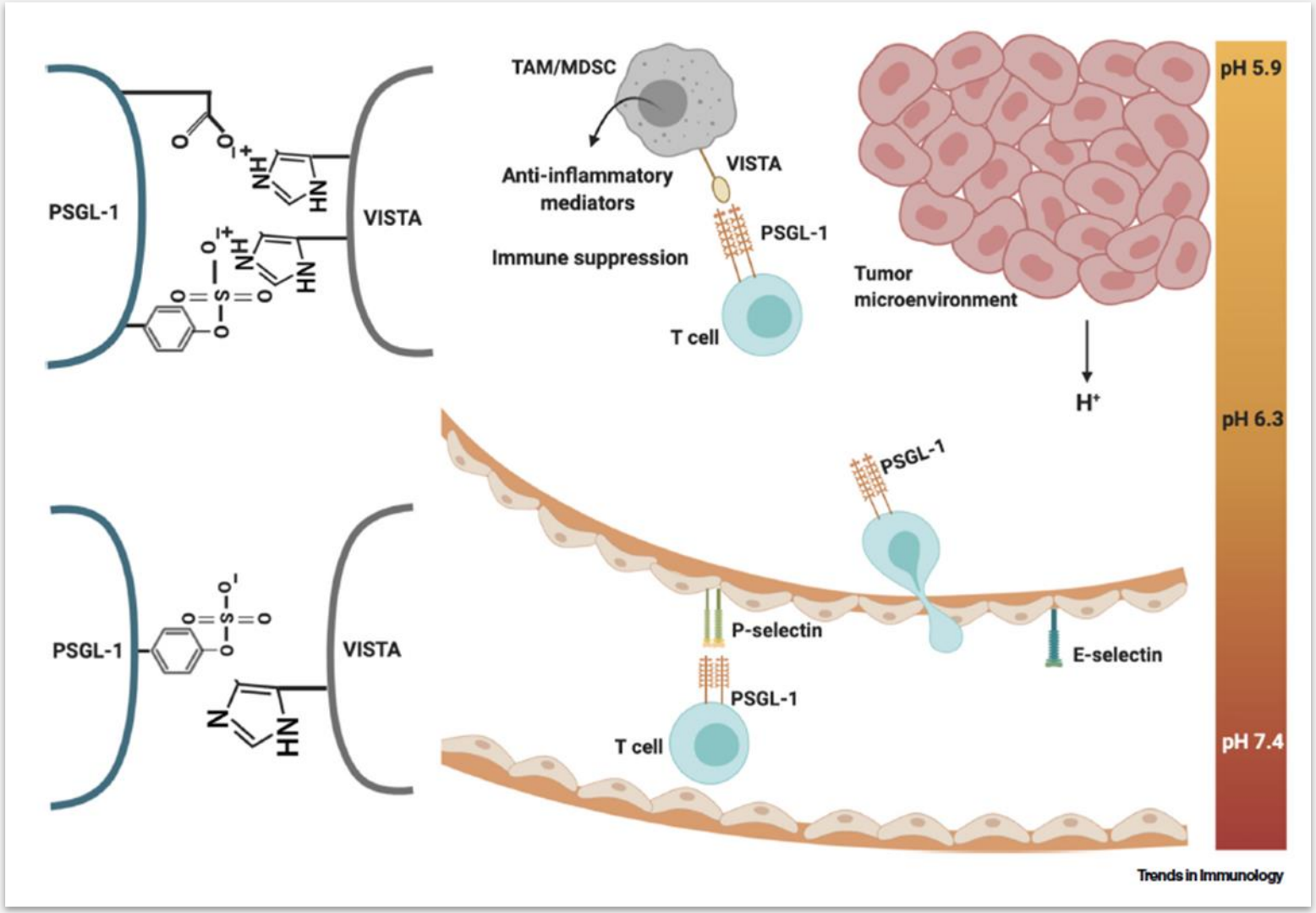


1 Lines et al. *Cancer research* vol. 74,7 (2014)
2 Gao et al. *Nature medicine* vol. 23,5 (2017)

The Binding of VISTA to PSGL-1 is pH Dependent

Active VISTA

Inactive VISTA



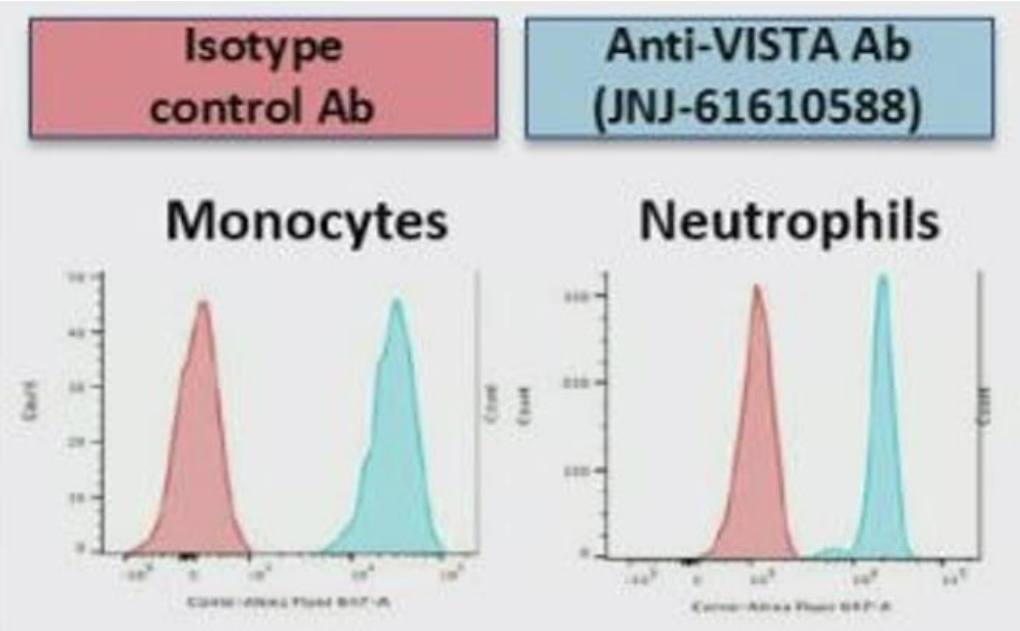
Adapted from Gao et al. *Nature medicine* vol. 23,5 (2017)

Dr. Schreiber

VISTA has been difficult to drug due to its unique biology

VISTA is Expressed at High Levels on Human Monocytes and Neutrophils

Flow Cytometry Analysis of VISTA Expression on Normal Human Peripheral Immune Cells



Snyder et al, 2016 AACR Annual Meeting, Oral Presentation

High VISTA Expression on Monocytes and Neutrophils Results in Sub-Optimal PK and may Decrease the Therapeutic Window

- Antibodies binding VISTA^{+} cells (e.g. monocytes) at physiological pH result in rapid elimination from circulation through **targeted-mediated drug disposition (TMDD)**
- Efficacious drug occupancy levels may be difficult to reach and potentially narrow the therapeutic window

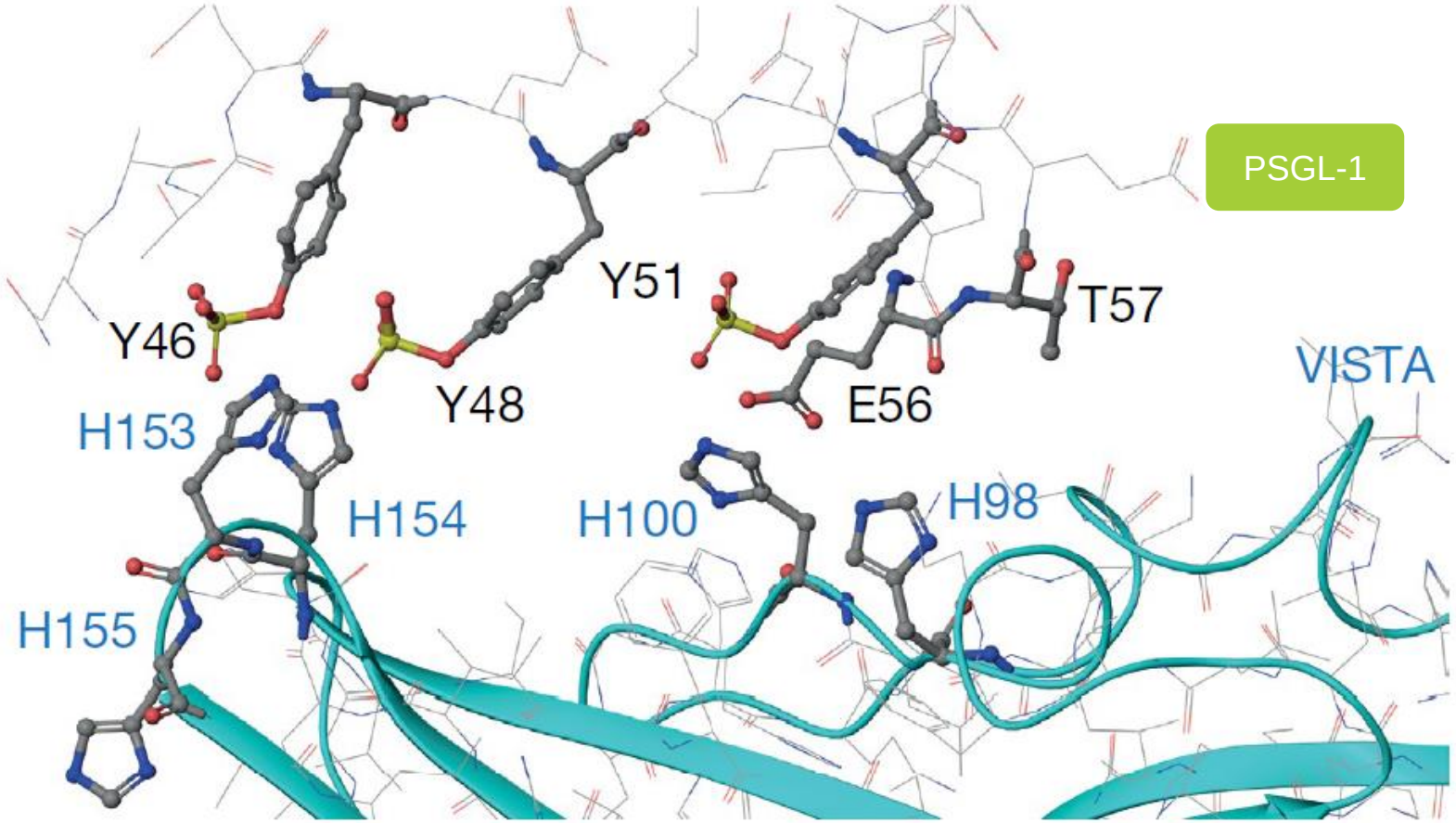
Case Study

CI-8993 Clinical Ongoing Clinical Study

- Phase 1 Dose Escalation Study
- 12 patients enrolled with advanced refractory solid tumors
- Initial dose of 0.005 mg/kg and above
- Low-grade transient Cytokine Release Syndrome (CRS) seen at 0.15 mg/kg and above
- Study halted after 1 DLT at sub-therapeutic dose level

The VISTA Checkpoint Itself is Only "ON" Under Low pH Conditions

Antibodies that block VISTA histidines H153, H154 and H155 interrupt PSGL-1 binding[1]

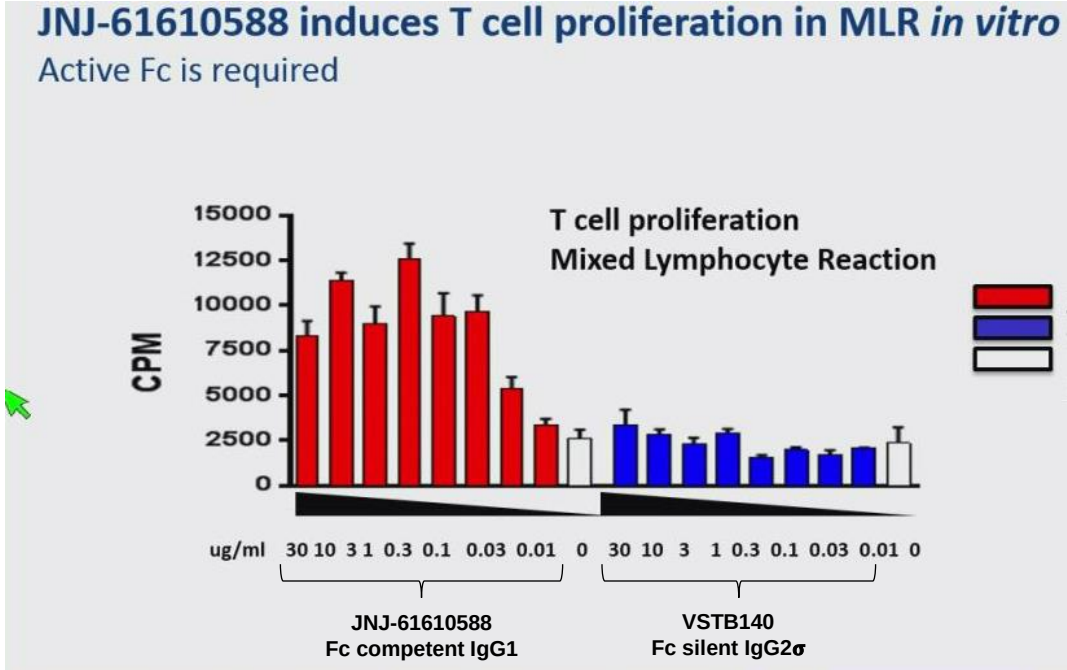


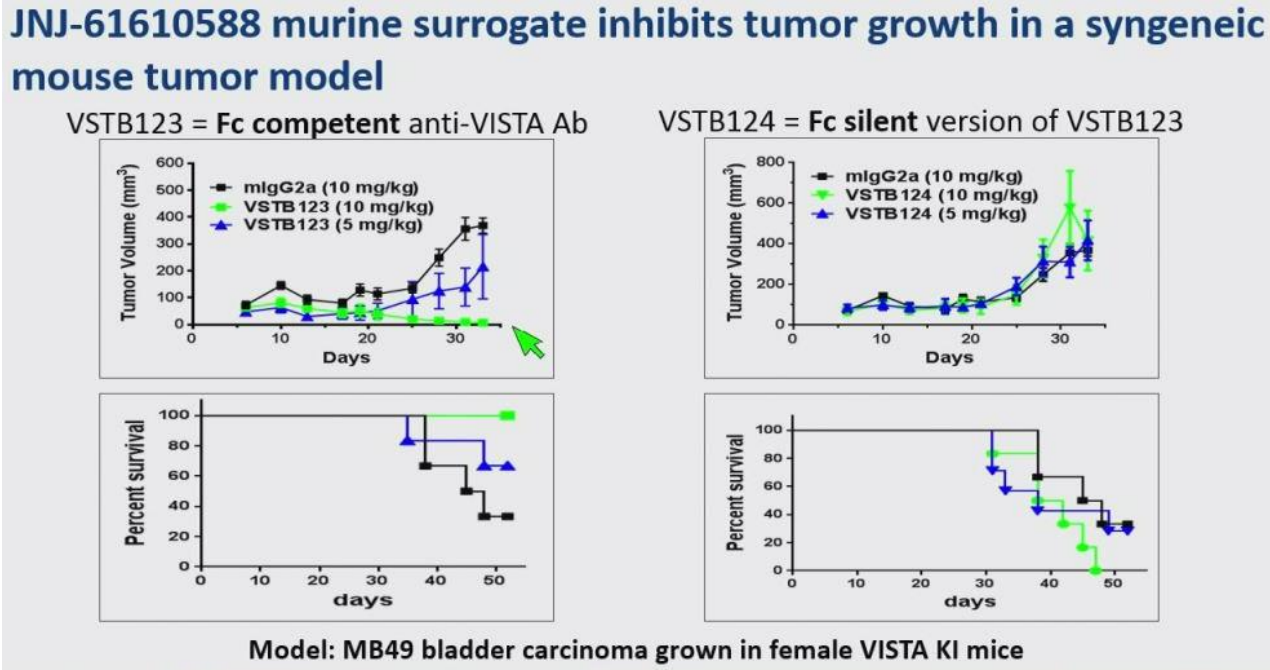
VISTA's extracellular domain is uniquely rich in histidines[1]

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

1. Johnston et al., Nature 2019

Engagement of FcγR may be Required for Optimal Activity of Anti-VISTA Monoclonal Antibodies





Summary

Reasons Why VISTA Has Been Difficult to Drug Historically

- VISTA is expressed at high levels on monocytes and neutrophils
- For non-pH-dependent blocking antibodies, high expression on monocytes and neutrophils results in a sub-optimal PK due to target-mediated clearance and may decrease the therapeutic window
- The VISTA checkpoint itself is only "ON" under low pH conditions
  - VISTA's immune checkpoint function is only active (i.e. capable of binding PSGL-1 at low pH)
  - Other receptors for VISTA are active at physiologic pH but do not appear to function as immune checkpoints
- Engagement of FcγR may be a prerequisite for optimal activity of anti-VISTA antibodies
  - Fc silent antibodies are not effective at T cell proliferation ex vivo or anti-tumor activity in vivo despite picomolar binding affinity to VISTA
  - Engagement in the blood may result in untoward "off tumor" activation (i.e. CRS)

Dr. Rob Pierce

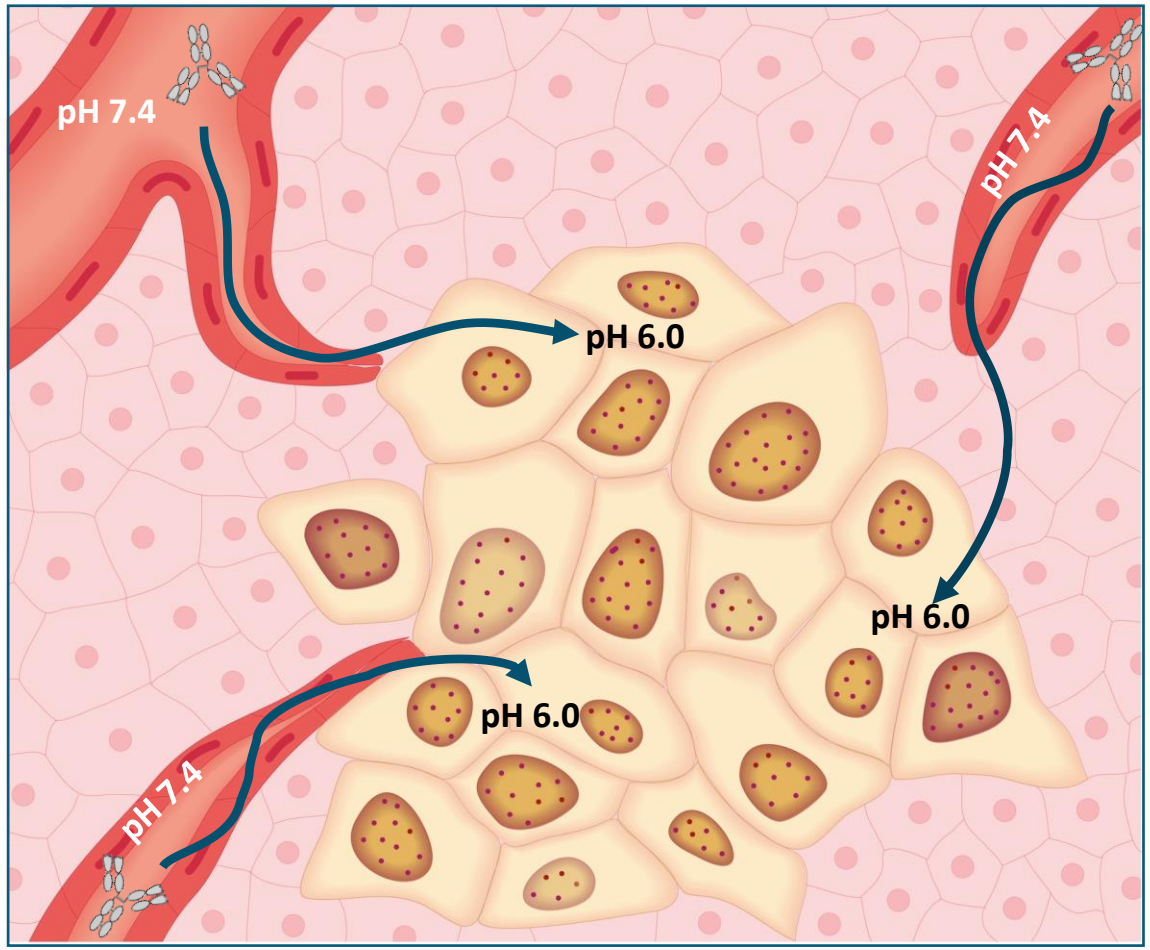
SITC 2021: SNS-101 Preclinical Data

Poster Presentation

SENSEI BIOTHERAPEUTICS

pH-sensitive Antibodies Primarily Bind Their Antibodies in the Low pH Tumor Microenvironment

The tumor microenvironment of pH~6.0 is lower than physiological pH of 7.4

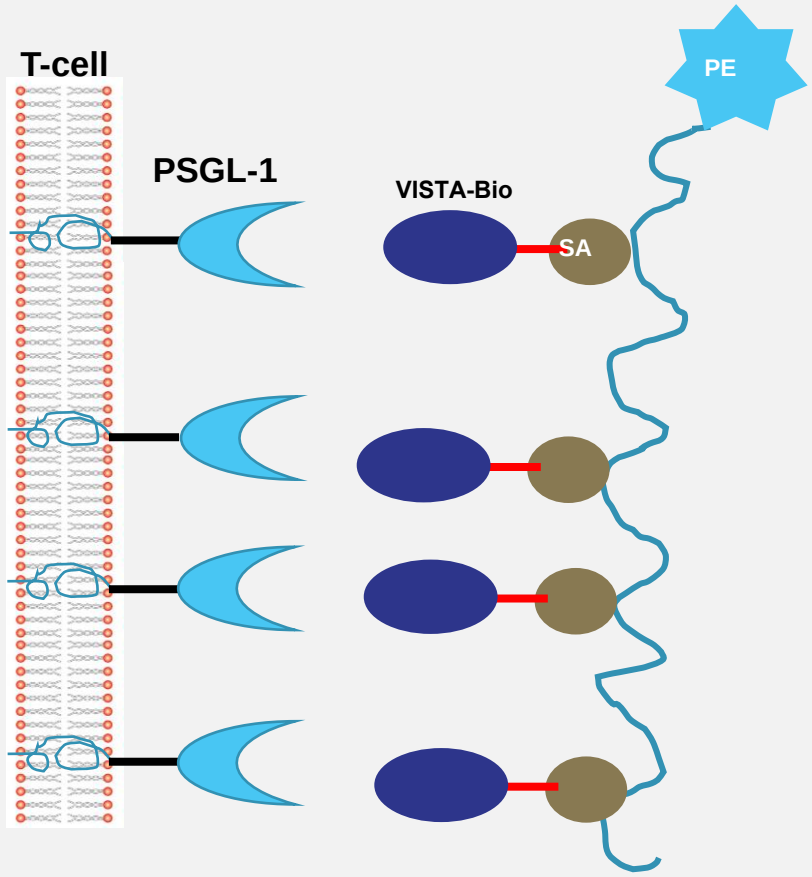


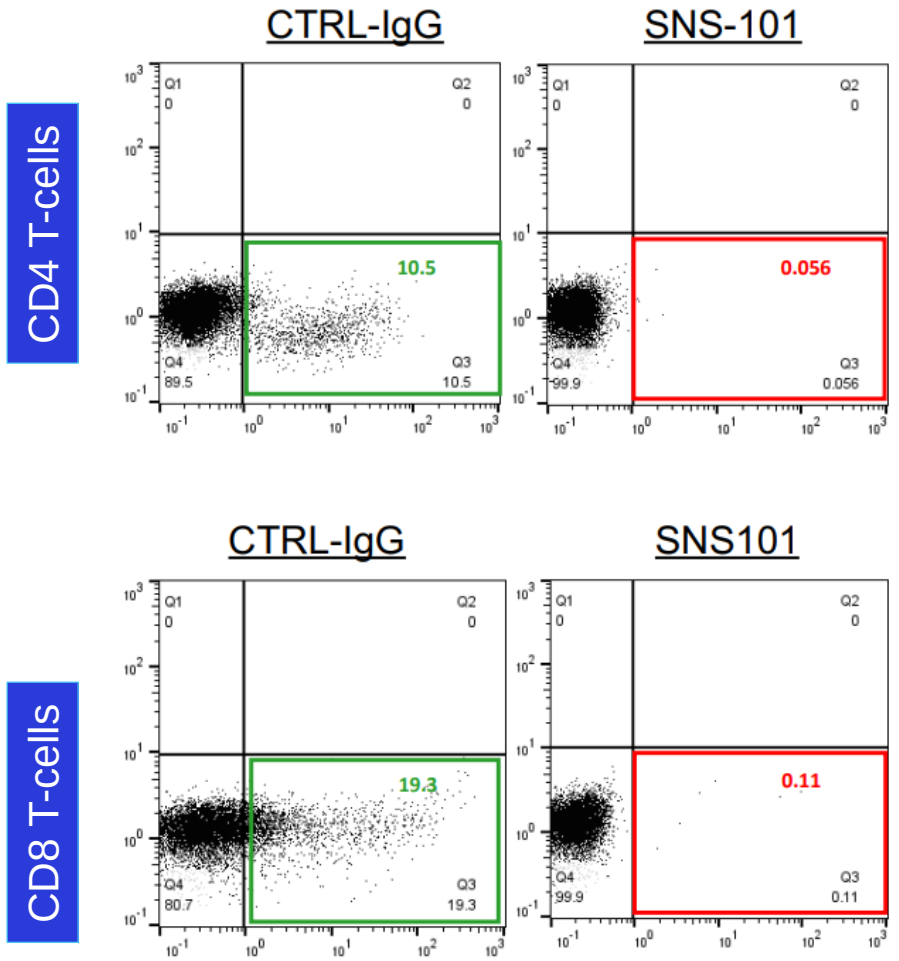
Sensei's technology identifies pH-sensitive antibodies that bind primarily at the tumor

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

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

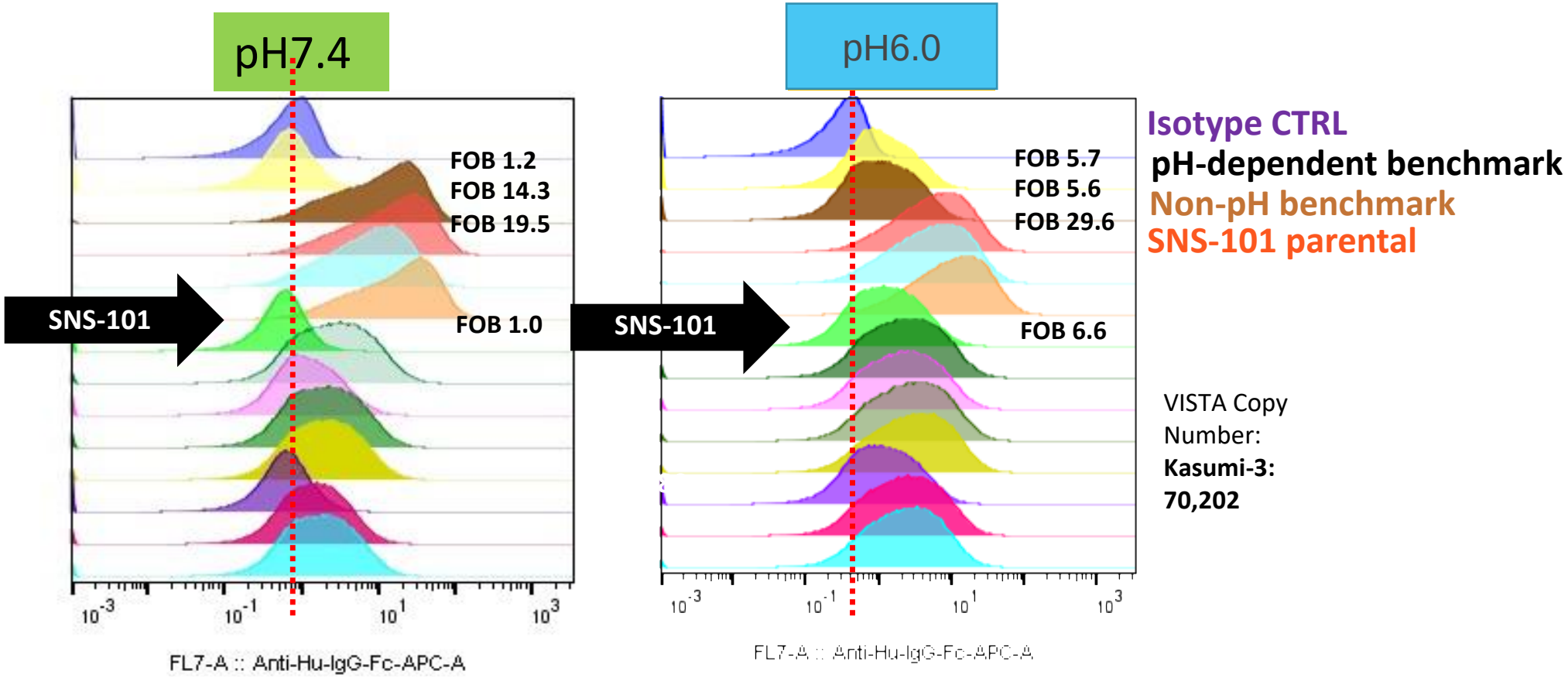
PSGL-1: VISTA Interaction on primary T-cells at pH 6.0





SNS-101 Identified Based on Stringent Cell-Based Assay

Candidate profile: no significant binding at pH 7.4



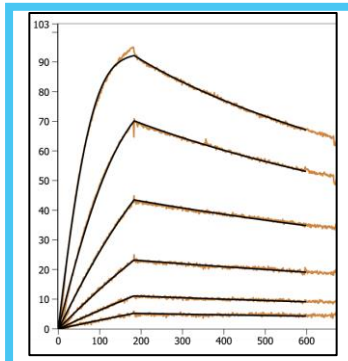
SNS-101 Has >600-Fold Selectivity for VISTA^{pH6}

- 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)

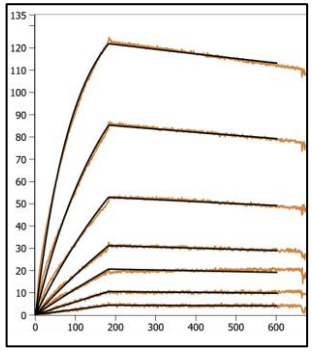
pH 6.0

SNS-101



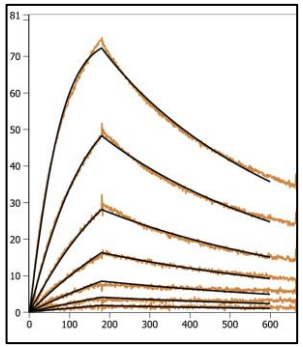
k_a = 4.59E+06 $M^{-1}s^{-1}$
K_d = 1.00E-03 s^{-1}
K_D = 2.18E-10 M
[Vista] – 7.5 nmol – 0.2 nmol

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

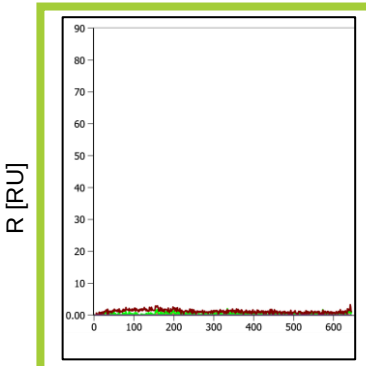
pH-independent "benchmark"



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

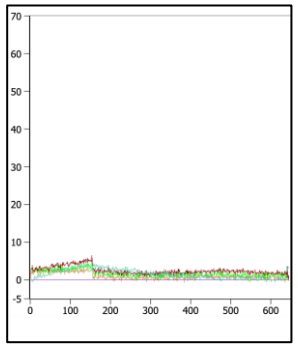
pH7.4

SNS-101



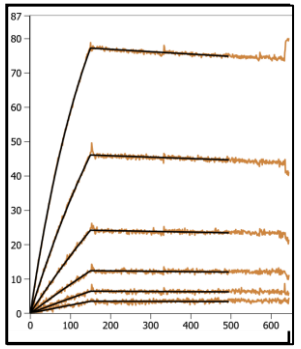
[Vista] – 7.5 nmol – 0.2 nmol

pH-dependent "benchmark"



[Vista] – 15 nmol – 0.2 nmol

pH-independent "benchmark"

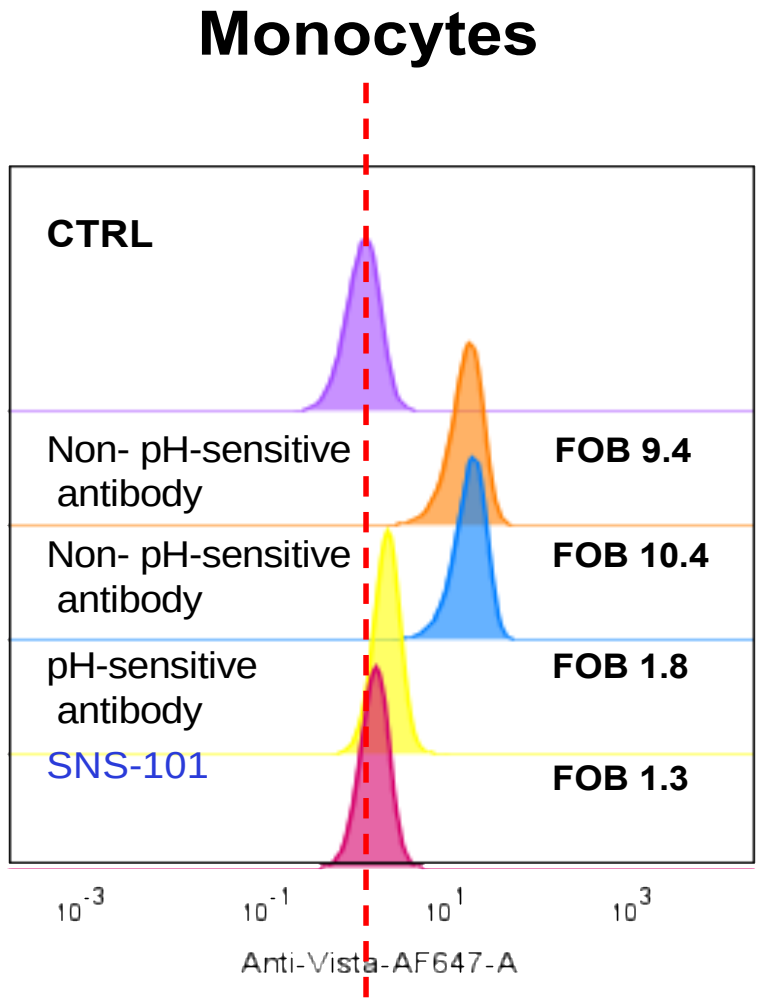


k_a = 4.162E+05 $M^{-1}S^{-1}$
K_d = 9.27E-05 S^{-1}
K_D = 2.23E-10 M
[Vista] – 15 nmol – 0.47 nmol

SNS-101 Does Not Significantly Bind to VISTA+ Monocytes at pH 7.4

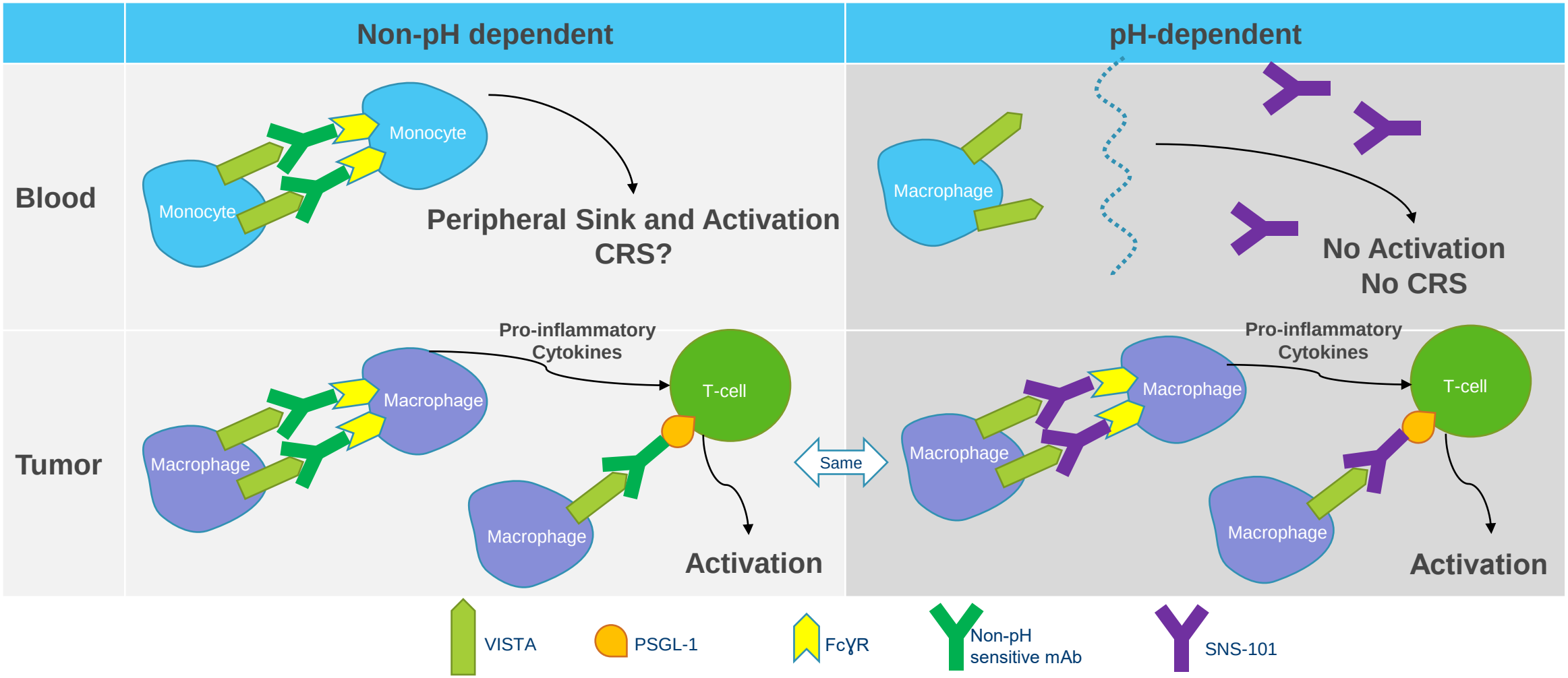
- VISTA+ monocytes are one of the main causes of TMDD
- Non-pH sensitive VISTA mAbs bind to monocytes at pH 7.4 thus allowing TMDD and have potential for on-target/off-tumor toxicity

VISTA Copy Number:	
Kasumi-3:	70,202
CD14+ Monocytes:	~103,000

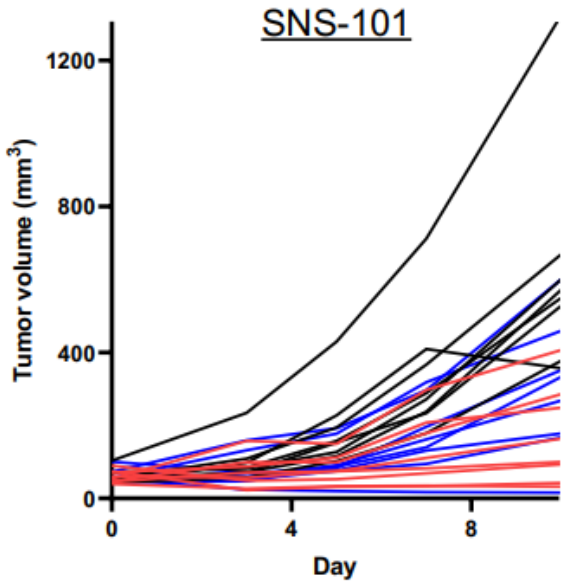


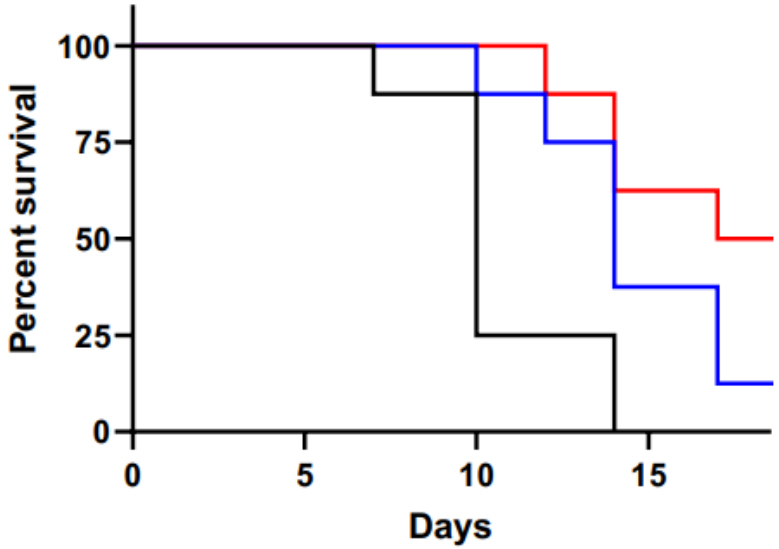
Proposed Mechanism of Action for SNS-101

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



‘High-bar’ *In Vivo* Screening Test of SNS-101 Activity
1-week Administration





Antibodies were administered I.P. 2/wk **for 1 week** at 40 mg/kg total (20 mg/kg each)

Black Line (IgG Control human & ratl)
Blue Line (IgG Control human & rat anti-mPD-1)
Red Line (rat anti-mPD-1 & anti-VISTA)

SNS-101 Is a Differentiated Anti-VISTA Antibody

TMAb Platform

	SNS-101 (SENSEI BIOTHERAPEUTICS)	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; no data reported	• First-patient to be dosed in 4Q'21

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

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

IND-Enabling Studies are Underway for SNS-101

Question & Answer Session

VISTA Science Symposium

November 16, 2021

Guest Speaker:

Prof. Robert Schreiber

Andrew M. Bursky and Jane M. Bursky Distinguished Professor of Pathology and Immunology, Professor of Molecular Microbiology and co-leader of the tumor immunology program at the Siteman Comprehensive Cancer Center, Founding Director of the Center for Human Immunology and Immunotherapy Programs at The Washington University School of Medicine
Sensei IOAB member

Sensei Presenters:

John Celebi
Chief Executive Officer

Dr. Robert Pierce
Chief Scientific Officer

Dr. Edward van der Horst
SVP, TMAb Antibody Development