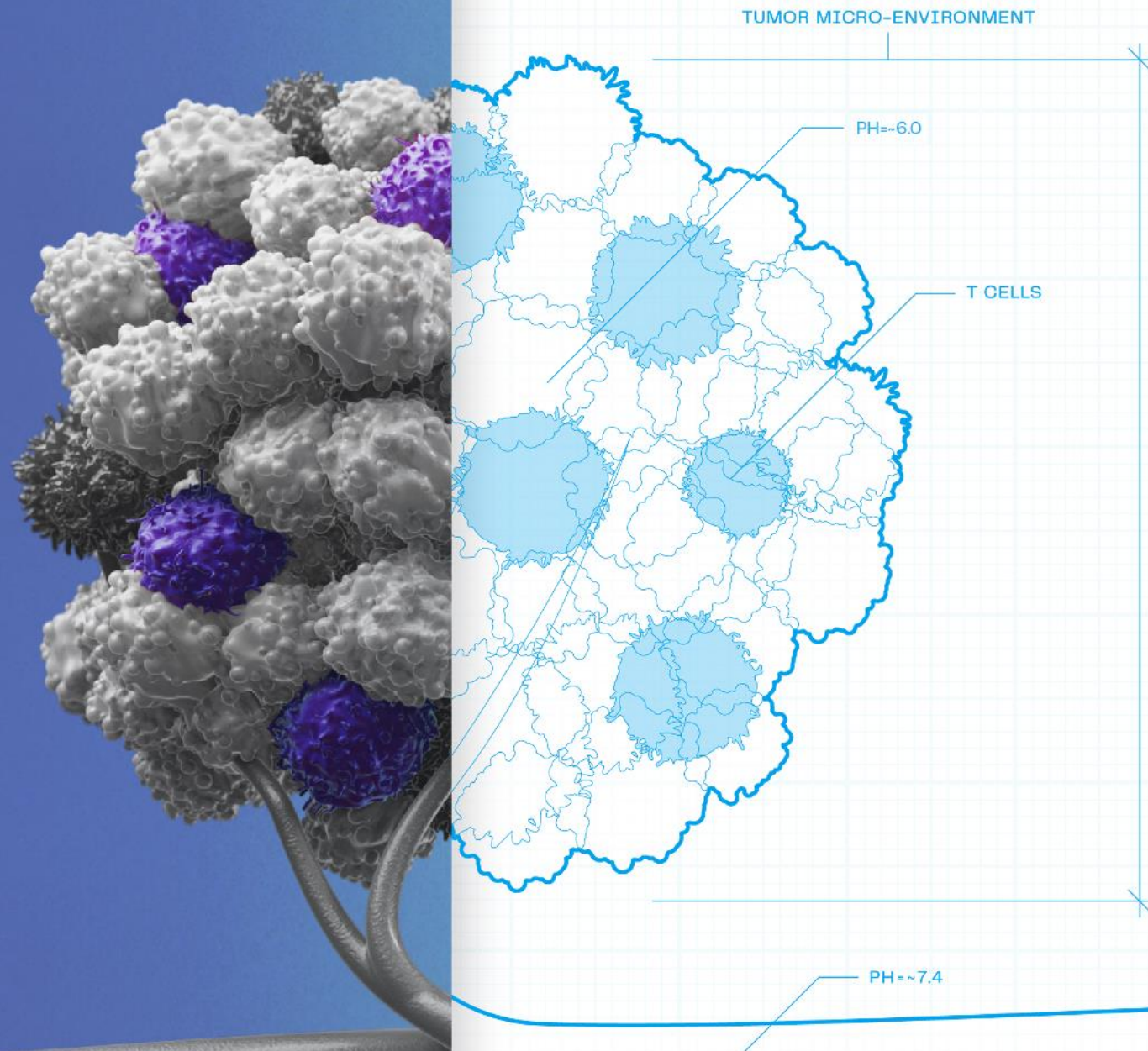




Conditionally Active Antibodies for Immuno-oncology

Corporate Deck | April 2024



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When used in this presentation, the words and phrases "designed to," "may," "believes," "intends," "seeks," "anticipates," "plans," "estimates," "expects," "should," "assumes," "continues," "could," "will," "future" and the negative of these or similar terms and phrases are intended to identify forward-looking statements. Forward-looking statements involve known and unknown risks, uncertainties and other factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Risks and uncertainties that may cause actual results to differ materially include uncertainties inherent in the development of therapeutic product candidates, such as preclinical discovery and development; conduct of clinical trials and related regulatory requirements, including the risk of delay or cessation of any clinical trials of Sensei's product candidates; the risk that prior results, such as signals of safety, activity or durability of effect, observed from preclinical trials and early results from the clinical trial of SNS-101, will not be replicated or will not continue in ongoing or future studies or clinical trials involving Sensei's product candidates, including SNS-101; our reliance on third parties over which we may not always have full control; risks regarding the accuracy of our estimates of expenses, capital requirements and needs for additional financing; and other risk and uncertainties that are described in our Annual Report on Form 10-K filed with the SEC on or about February 29, 2024 and our other Periodic Reports filed with the SEC. Forward-looking statements represent our management's beliefs and assumptions only as of the date of this presentation and include all matters that are not historical facts. Our actual future results may be materially different from what we expect. Except as required by law, we assume no obligation to update these forward-looking statements publicly, or to update the reasons actual results could differ materially from those anticipated in the forward-looking statements, even if new information becomes available in the future.

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Company Highlights



Sensei Bio's proprietary platform is designed to harness the unique acidic tumor microenvironment to widen the therapeutic window and enable druggability of promising oncology targets



SNS-101, the company's lead asset, targets VISTA, a critical negative regulator of T-cell function and promising immune checkpoint target



SNS-101 is currently in Phase 1 clinical testing with initial data displaying an attractive safety profile and potentially best-in-class pharmacokinetics



Anticipated near-term milestones include topline Phase 1 monotherapy & combination dose escalation data in Q2 2024



Three additional early-stage drug candidates



Cash runway into the fourth quarter of 2025, which is expected to fund operations midway into Phase 2 studies of SNS-101

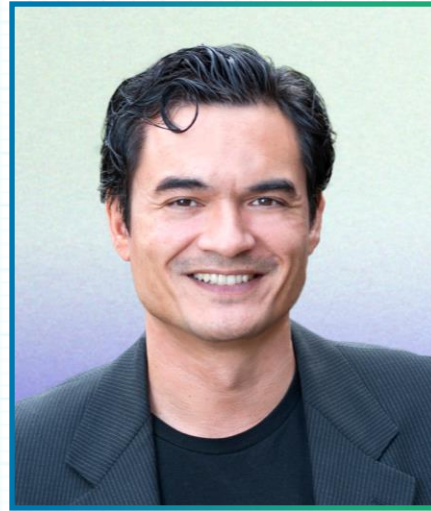
Seasoned Leadership Team



John Celebi, MBA
President and CEO



Christopher Gerry, J.D.
SVP, General Counsel



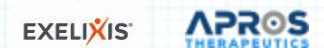
Edward van der Horst, Ph.D.
Chief Scientific Officer



Stephanie Krebs, M.S., MBA
Chief Business Officer

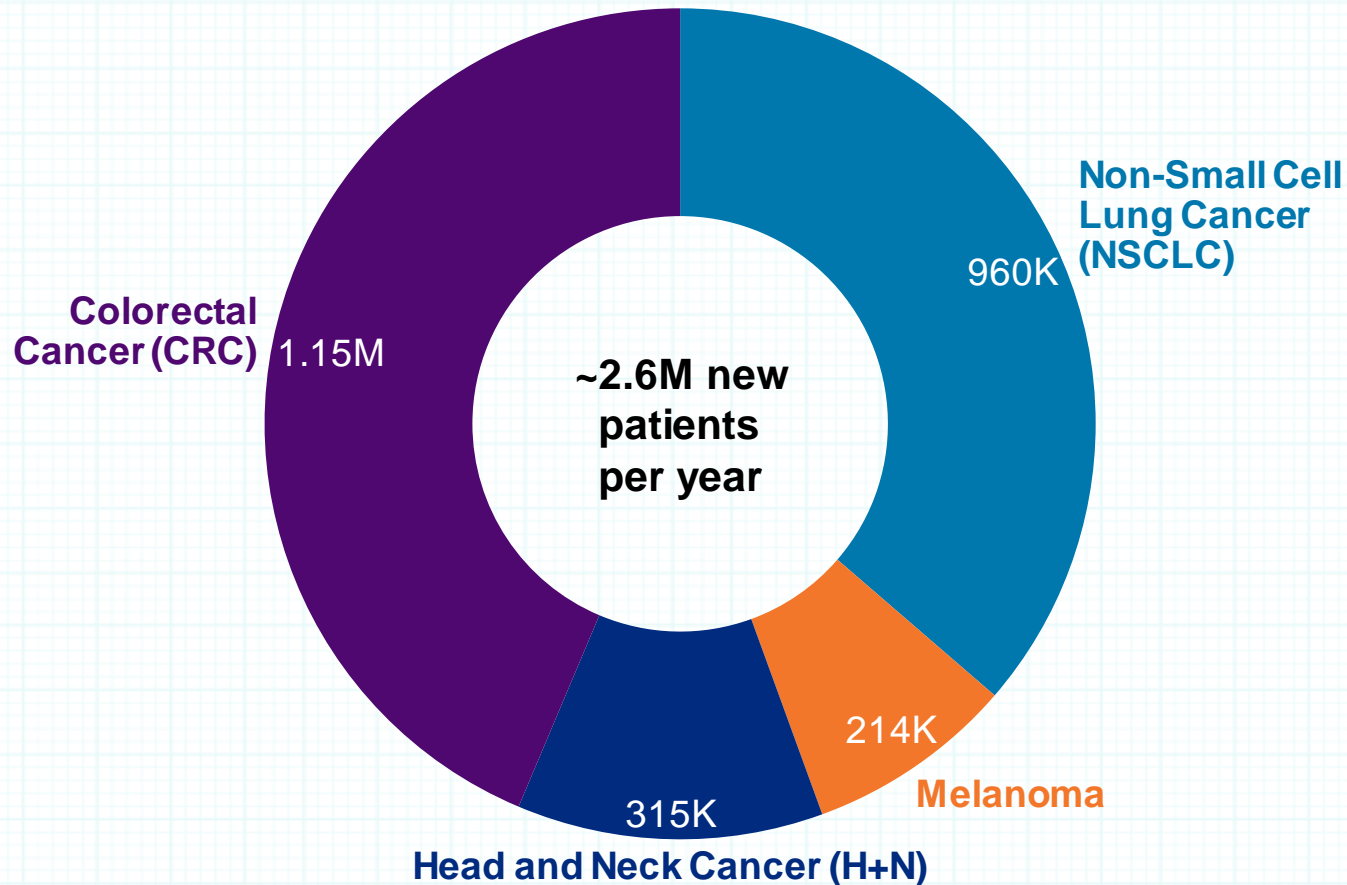


Ron Weitzman, M.D.
Chief Medical Officer (part-time)



Large Commercial Opportunity still exists in Immuno-Oncology (IO)

Newly Diagnosed Patients Annually in 2026²



VISTA's Potential Commercial Impact

- ❖ The checkpoint market is large and growing fast¹
- ❖ Despite the widespread use of checkpoint inhibitors, only 20% of patients experience an objective response
- ❖ Indications such as CRC see little to no benefit from current treatment options
- ❖ VISTA is implicated in numerous solid tumor types with large patient populations

Lack of Tumor Targeting is a Major Obstacle in IO Innovation

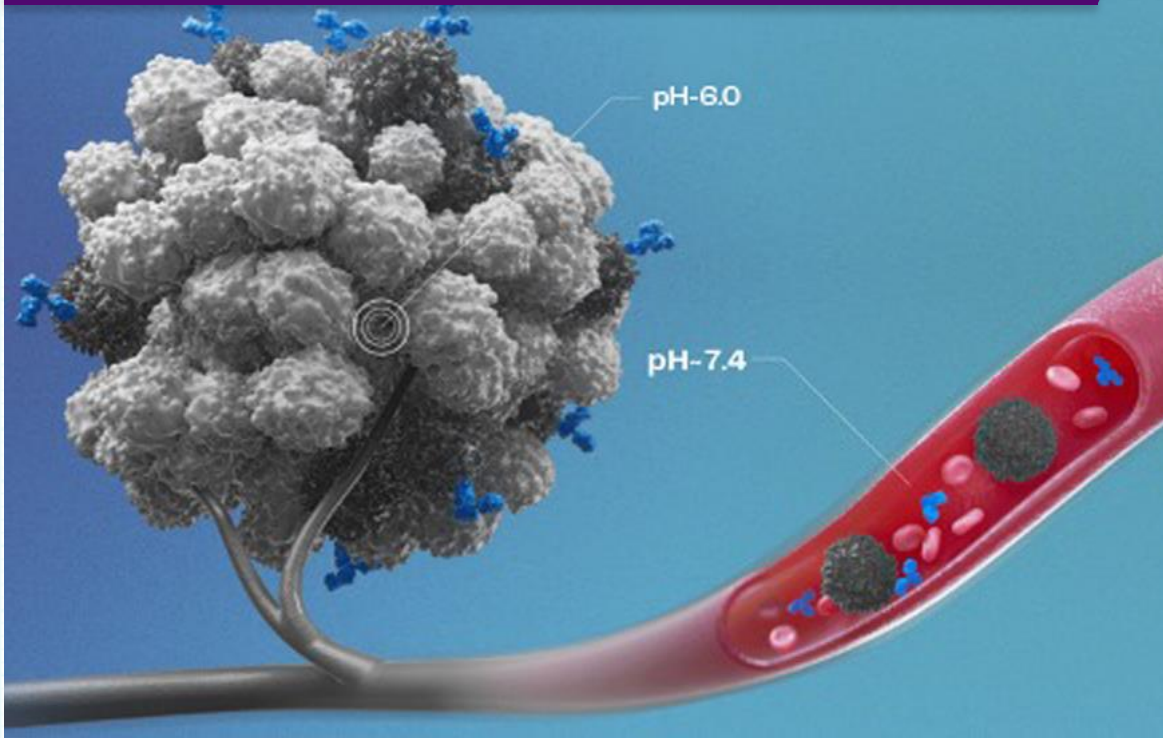
Industry Problem	Sensei's Solution
<p>Conventional antibodies target immune checkpoints that are highly expressed in normal tissues, resulting in:</p> <ul style="list-style-type: none">• Dose-limiting toxicities due to on-target/off-tumor action• Pharmacological sink effect requires higher & more frequent dosing• Suboptimal activity due to poor PK & dose-limiting toxicities	<p>Conditionally active antibodies are selectively targeted to the tumor microenvironment, potentially providing:</p> <ul style="list-style-type: none">• Little or no toxicity due to selective on-target/on-tumor action• Lower & less frequent doses with tumor-specific binding• Powerful activity selectively focused on the tumor microenvironment



One new IO checkpoint inhibitor approved after the CTLA-4 and PD-1/PD-L1 group

The TMAb Platform: pH-sensitive Antibodies Selectively Bind to Targets in the Low-pH Tumor Microenvironment

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



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

- Exploits the tumor microenvironment using pH-selective properties
- Intended to alleviate undesirable PK/PD properties:
 - Dose-limiting toxicities due to on-target/off-tumor binding
 - Higher and more frequent dosing due to poor pharmacokinetics
- Bolsters specific activities
- Unlocks previously undruggable immune targets

Innovative Pipeline of IO Drugs with Broad Commercial Potential

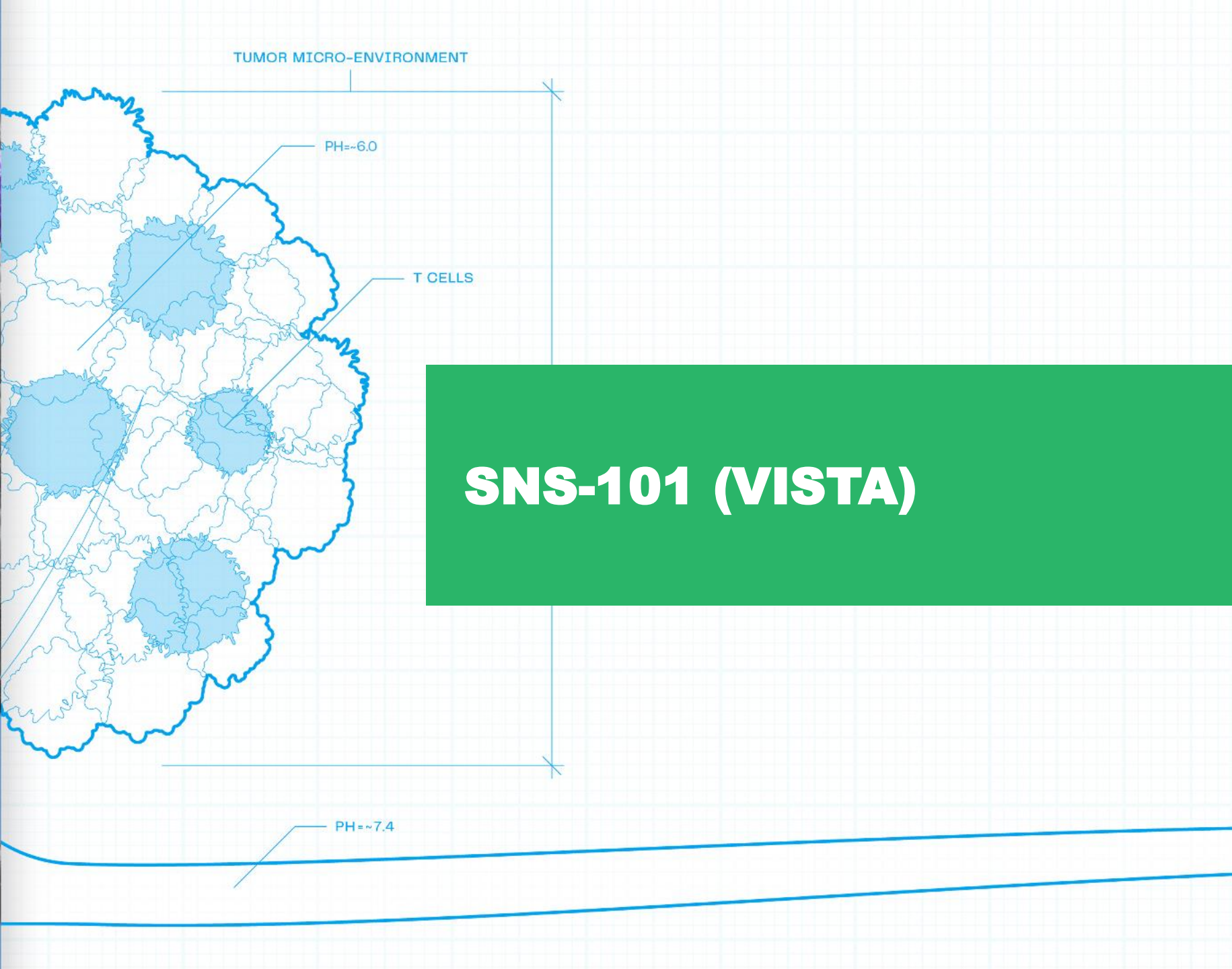
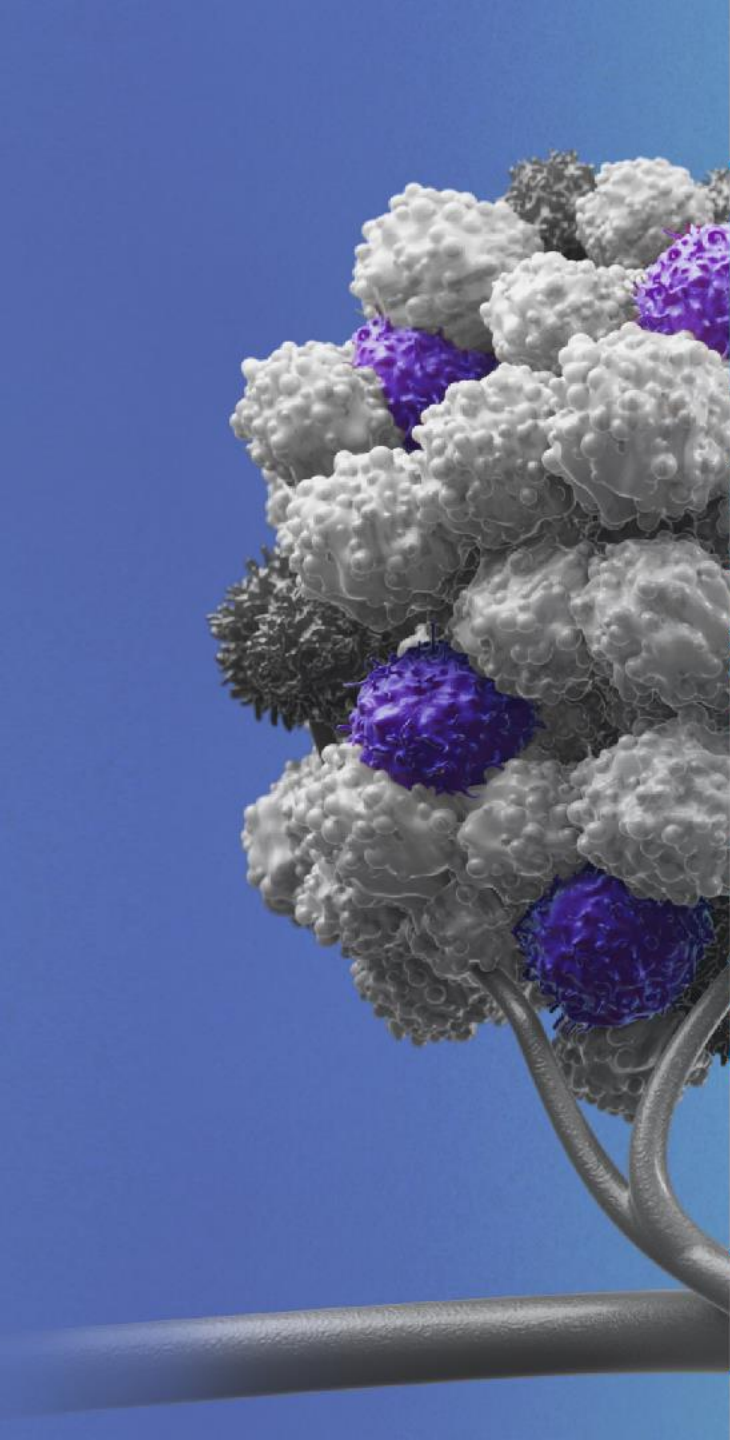
Program (Target)	Indication	Discovery	IND-enabling	Phase 1 / 2 Clinical
SNS-101* (VISTA)	Solid Tumors			
SNS-102 (VSIG4)	Solid Tumors			
SNS-103 (ENTPDase1/CD39)	Solid Tumors			
SNS-201 (VISTAxCD28)	Solid Tumors			

REGENERON

*Sensei has entered into a clinical supply agreement with Regeneron supporting the planned evaluation of SNS-101 in combination with Regeneron's anti-PD-1 therapy Libtayo® (cemiplimab) in a Phase 1/2 clinical trial in solid tumors.



*Sensei has entered into a Cooperative Research and Development Agreement (CRADA) with the National Cancer Institute. The goal of this collaborative effort is to further elucidate the role of VISTA in immune checkpoint resistance and expand the potential of SNS-101 as a combination therapy beyond anti-PD-1.



SNS-101 (VISTA)

VISTA is a Potent T cell Checkpoint Extensively Expressed on Myeloid Cells

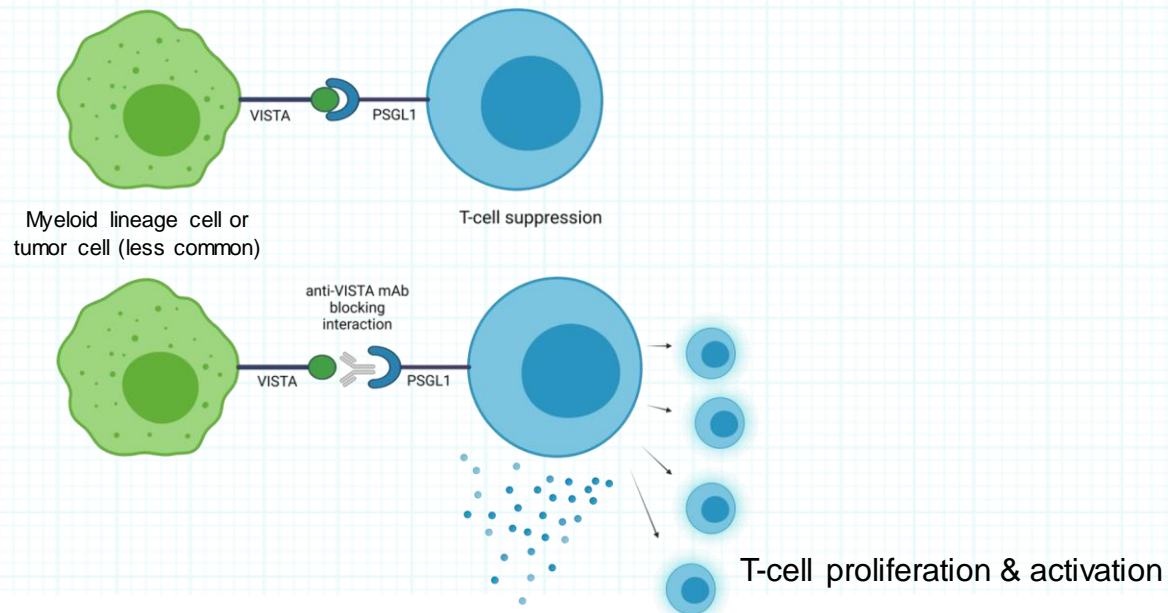
VISTA is a B7 family member that inhibits T cell activation¹

Immunosuppressive function believed to be mediated by PSGL-1 receptor

Upregulated on immune suppressive myeloid-derived suppressor cells (MDSCs) via hypoxia²

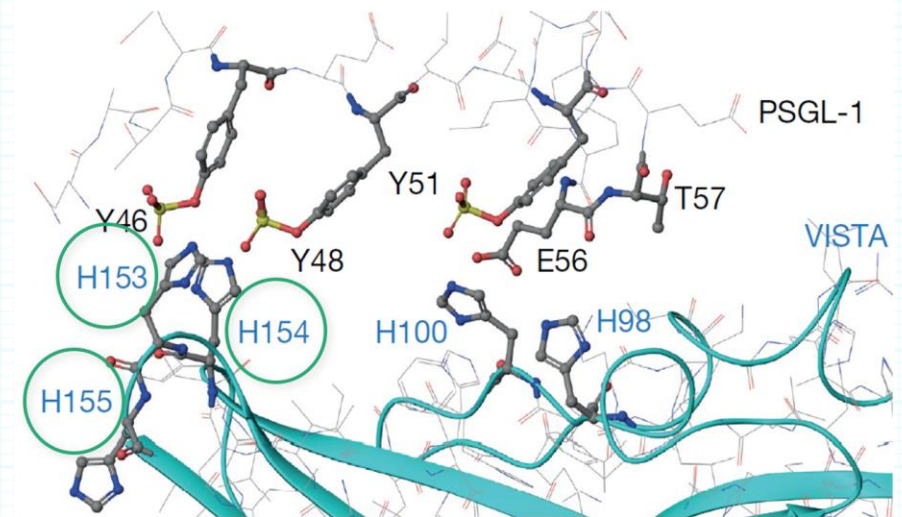
Increased expression on tissue infiltrating immune cells upon checkpoint therapy failure³

IS ACTIVATED IN A pH SENSITIVE MANNER



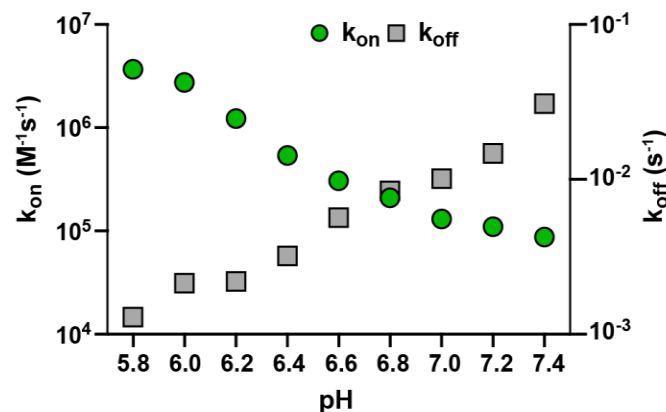
Extensive VISTA expression on off-tumor myeloid-lineage cells demands a conditionally active antibody approach

VISTA has inherent pH sensitivity: its extracellular domain is uniquely rich in histidines⁴



SNS-101 is a pH-sensitive Antibody Selective for VISTA

Selectivity for active VISTA^{pH6} over VISTA^{pH7.4}



pH 6.0

0.218 nM

pH 7.4

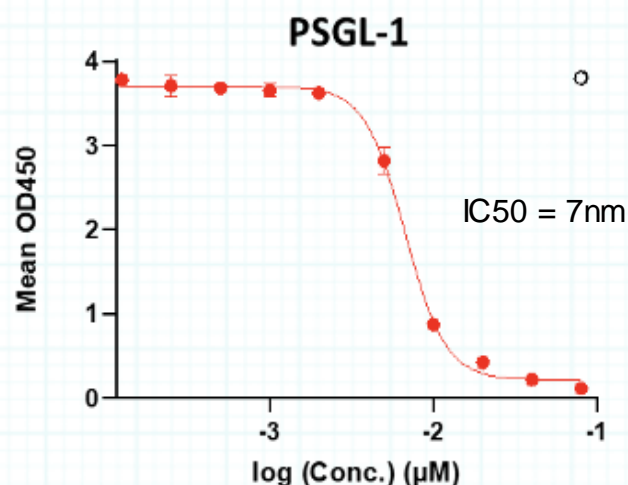
132 nM
(~No binding)

Monovalent Affinity (K_D)

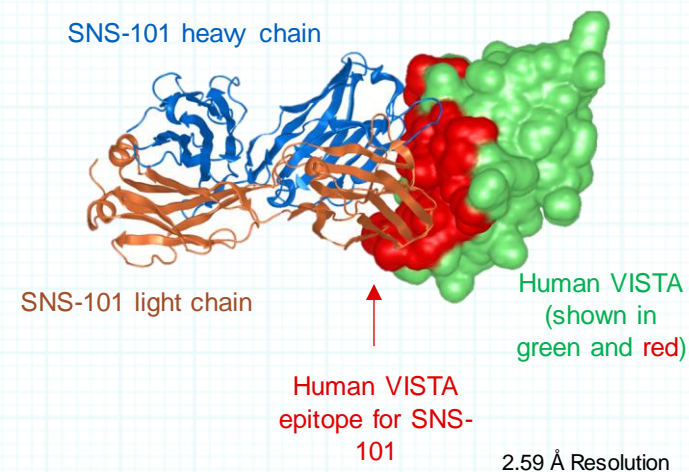
Additional SNS-101 features

- IgG1 format
- Active Fc

Blocks the key receptor regulating VISTA's immunosuppressive activity



SNS-101 potently inhibits the VISTA:PSGL-1 interaction and all other potential binding partners at pH 6.0 *in vitro*

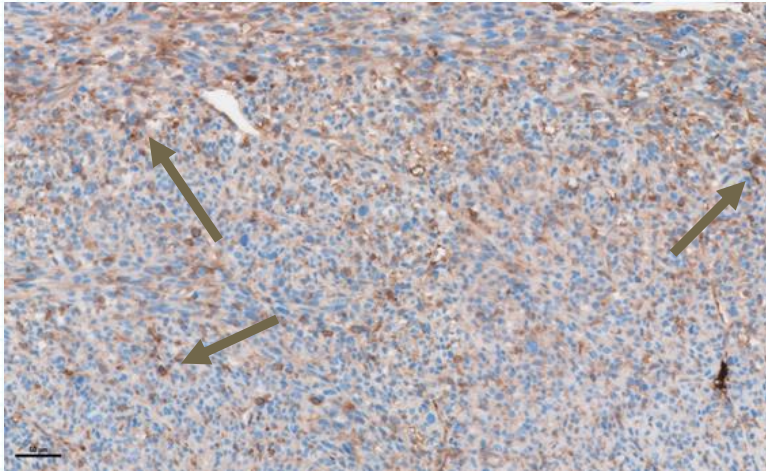


VISTA:SNS-101 co-crystal structure demonstrates SNS-101 encompasses VISTA's PSGL-1 epitope

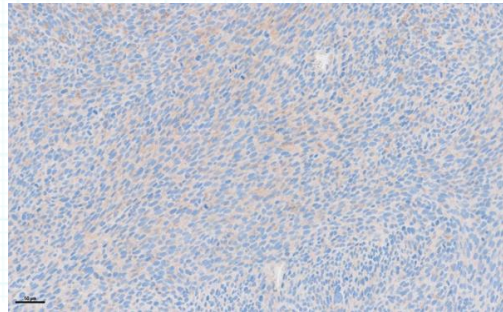
SNS-101 Rapidly Accumulates in Tumors and Binds to VISTA+ Myeloid Cells

Immunohistochemistry

SNS-101
6h post-dosing



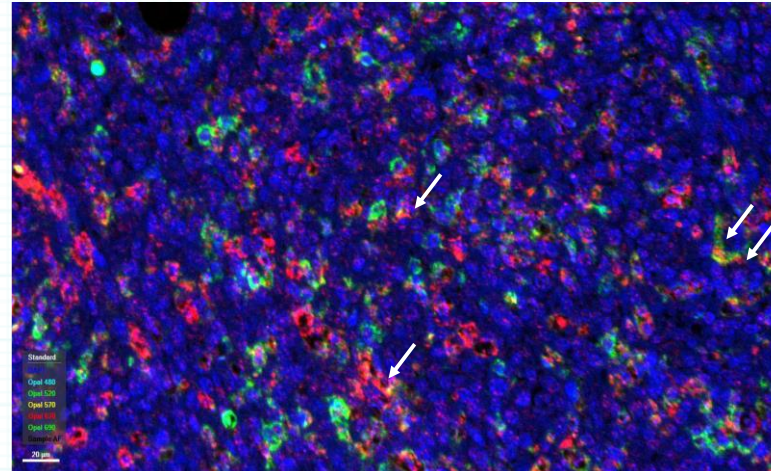
Isotype control
6h post-dosing



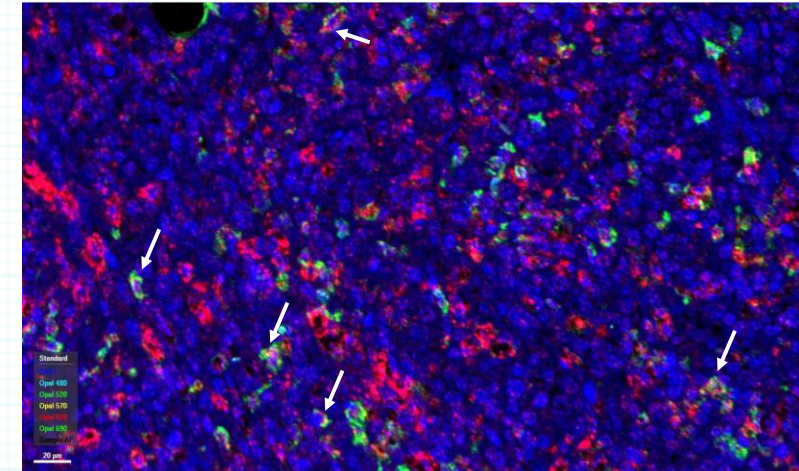
Blue = tumor
Brown = SNS-101

Immunofluorescence

SNS-101 + CD45
6h post-dosing



SNS-101 + CD11b
6h post-dosing



Competitors Halted Development of VISTA Antibodies as a Result of Severe Toxicities From Non-Tumor Activity & Poor PK

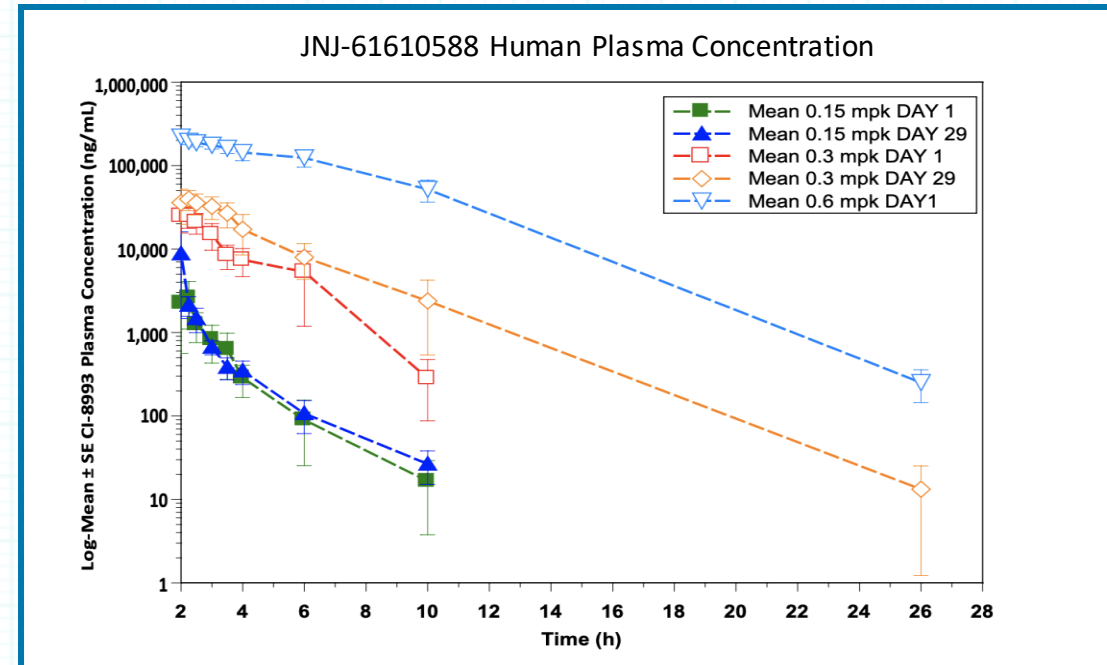
Dose-limiting toxicity

Grade 3 CRS-associated encephalopathy

- JNJ-61610588 (CI-8993) was the first anti-VISTA antibody to be studied in clinical trials in 2016 (NCT02671955) ¹
- Transient Cytokine Release Syndrome (CRS) observed in several patients at **0.15 mg/kg**
- Transient **Grade 3 CRS-associated encephalopathy** observed at **0.3 mg/kg**, after which Janssen halted the study

Challenging PK profile


Non-linear PK, short $t_{1/2}$



SNS-101 is Designed to Overcome VISTA's Unique Challenges

Differentiated Design and Mechanism	IgG1, Fc-active antibody designed to selectively block VISTA in the acidic tumor microenvironment
Enrolling Phase 1/2 Clinical Trial	Multi-center U.S. study as single agent and in combination with PD-1 inhibitor Libtayo®
Potential Best-in-Class Safety and PK Profile Supported by Initial Clinical Data	Well-tolerated with no observed DLTs and no evidence of target-mediated drug disposition*
Achieving "Firsts" for the VISTA Field	First VISTA-blocking antibody administered at a dose anticipated to be therapeutically relevant without eliciting dose-limiting toxicity**
Anticipated Near-Term Clinical Milestones	Topline monotherapy & combination dose escalation data in Q2 2024 Initial dose expansion data by end of 2024

SNS-101 is Unique and Differentiated From Its Peers

	SNS-101 	CI-8993; JNJ-61610588 (J&J/Curis)	K01401-020; W0180 (Pierre Fabre)	HMBD-002 (Hummingbird)	KVA12123 (Kineta)	VISTA.18 (BMS)	(PMC-309) Pharm Abcine
Inhibit PSGL-1 Binding	✓	✓	✓	✗	✓	✓	✓
pH Sensitive Binding	✓	✗	✗	✗	✗	✓	✗
Fc Active	✓ (IgG1)	✓ (IgG1)	✓ (IgG1)	✗	✓ (IgG1)	✗ (IgG4)	✓ (IgG1)
Stage	Phase 1	Phase 1	Phase 1	Phase 1	Phase 1	Preclinical	Phase 1

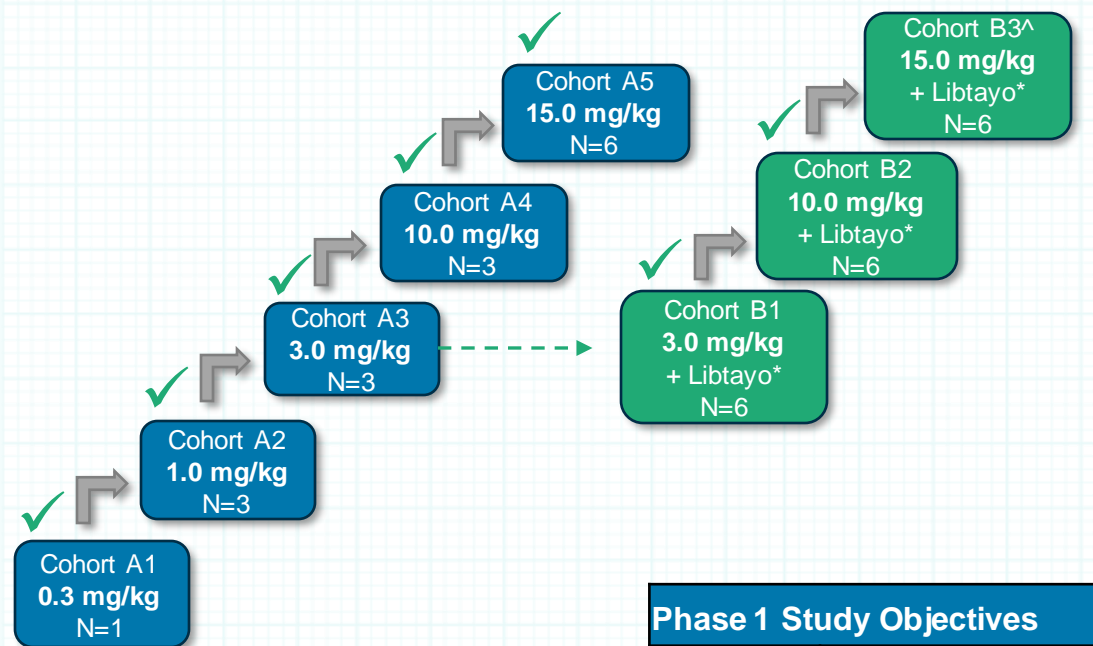
SNS-101 Phase 1/2 Study

Phase 1 Dose Escalation

BOIN design in patients with advanced solid tumors

Monotherapy Dose Escalation
SNS-101 (Q3W)

Combination Dose Escalation
SNS-101 + Libtayo* (Q3W)



Phase 1 Dose Expansion

Monotherapy
15 mg/kg
N=up to 10

CRC

Combination
Dose TBD
N=up to 30

Basket
(CRC,
NSCLC,
H&N,
Melanoma)

Additional tumor types and doses
may be considered for both the
monotherapy & combination dose
expansion

Phase 2

Single-arm, Simon two-stage minimax design

SNS-101 (RP2D) +/- Libtayo
(Q3W)

Indications
TBD

Phase 1 Study Objectives

Primary	Safety, tolerability, MTD/RP2D
Secondary	PK, immunogenicity & anti-tumor activity

Phase 2 Study Objectives

Primary	Anti-tumor activity
Secondary	Safety, tolerability, PK & immunogenicity

* Libtayo 350 mg
 ^ As of February 23, 2024, cleared Cohort B2 (10.0 mg/kg of SNS-101 + Libtayo)
 * As of February 23, 2024, Cohort B3 (15.0 mg/kg of SNS-101 + Libtayo) is enrolling
 As of February 23, 2024, patient enrollment has started for the monotherapy expansion cohort

RP2D = Recommended Phase 2 Dose
 MTD = Maximum Tolerated Dose
 CRC = colorectal cancer
 NSCLC = non small cell lung cancer
 H&N = head and neck cancer

SNS-101 Alone or in Combination with Libtayo Has Been Well-Tolerated With No DLTs Observed*

Monotherapy Dose Escalation: 16 patients enrolled over 5 monotherapy dose levels

- Cleared 5 monotherapy dose levels through 15 mg/kg
- Well-tolerated and no DLTs observed with 13/16 pts (81%) experiencing at least one TEAE
- Majority of AEs were Grade 1 or 2

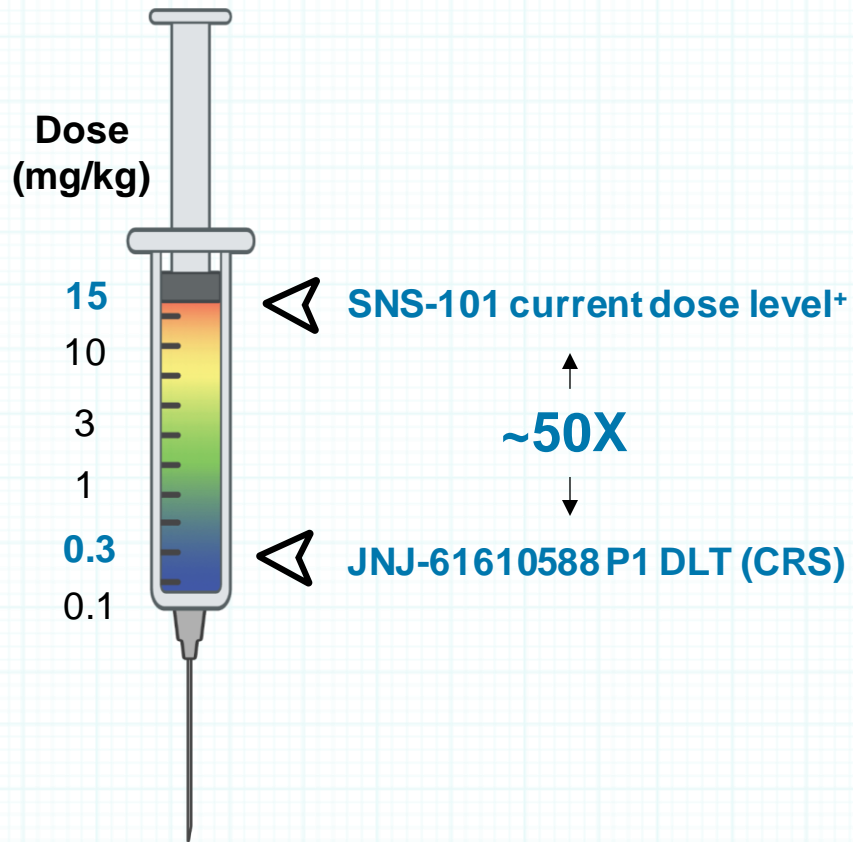
Combination Dose Escalation: 17 patients enrolled over 3 combination dose levels

- Cleared 2 combination dose levels through 10 mg/kg + Libtayo
- Well-tolerated and no DLTs observed with 10/17 pts (59%) experiencing at least one TEAE
- Majority of AEs were Grade 1 or 2

Monotherapy & Combination Pharmacokinetic (PK) Profile

- Demonstrated potentially best-in-class PK with linear elimination kinetics and dose-proportional increases in exposure across monotherapy and combination cohorts
- No notable difference in PK between monotherapy and combination dosing
- Supports once every 3-week dosing

Key SNS-101 Differentiators: Potential Best-in-Class Therapeutic



Potential Best-in-Class PK Profile

- Dosing every 3 weeks vs. every 1 or 2 weeks for competitors
- Linear elimination kinetics vs. non-linear for competitors



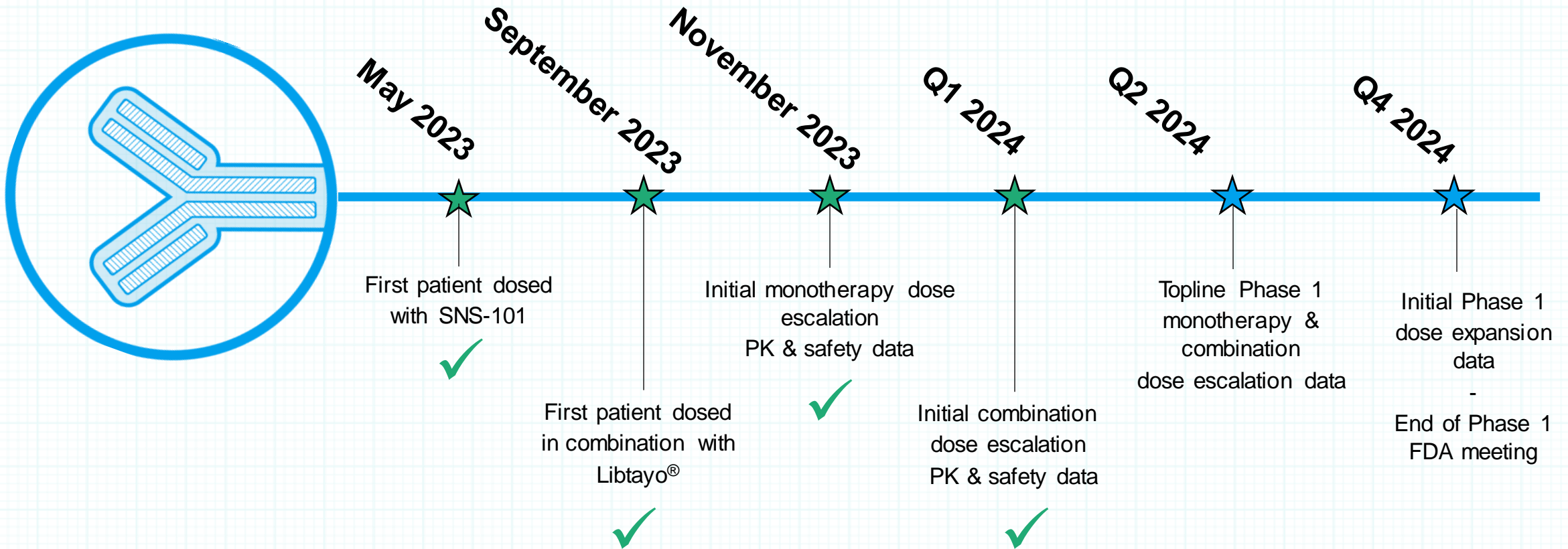
Safety Parameters On Track

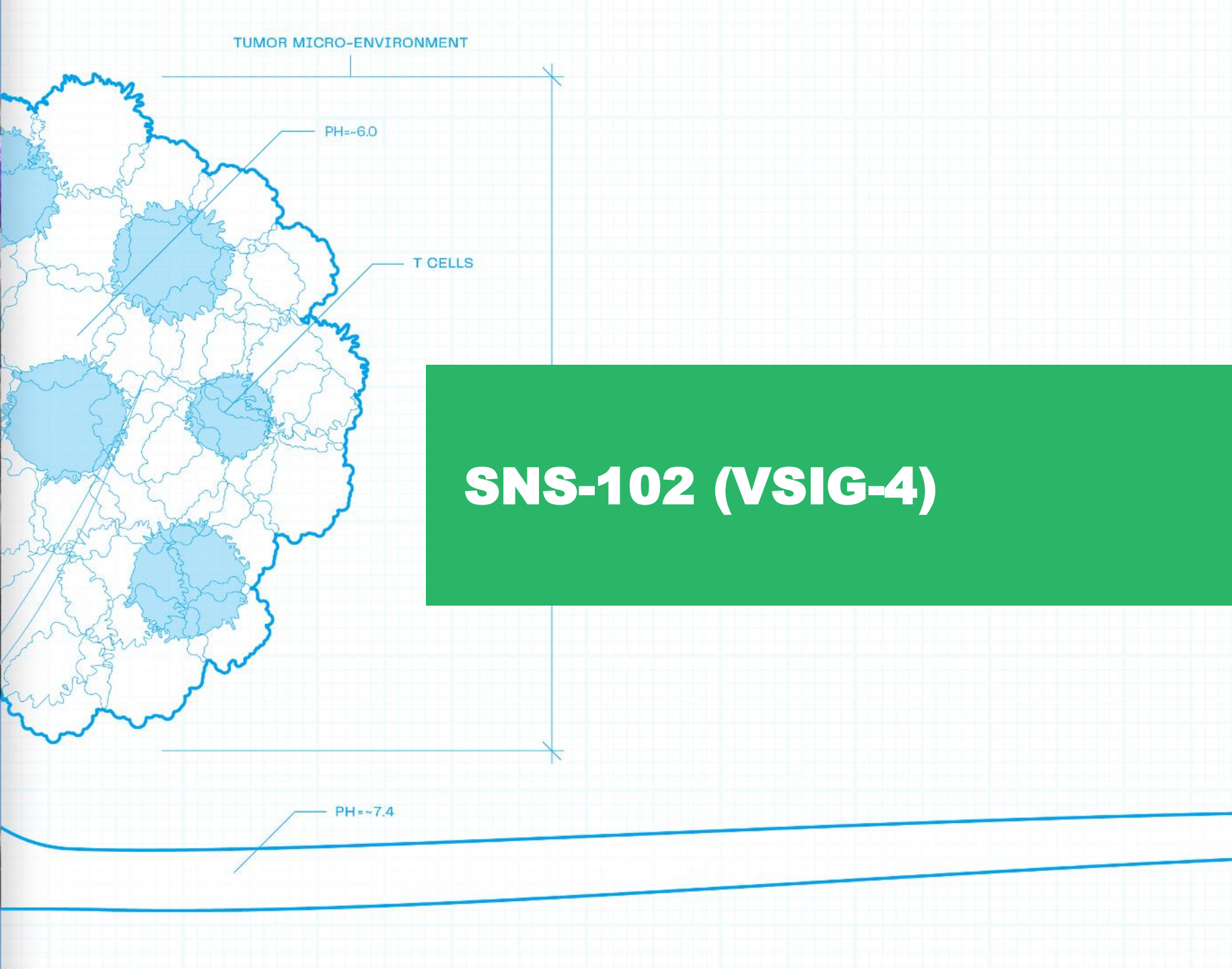
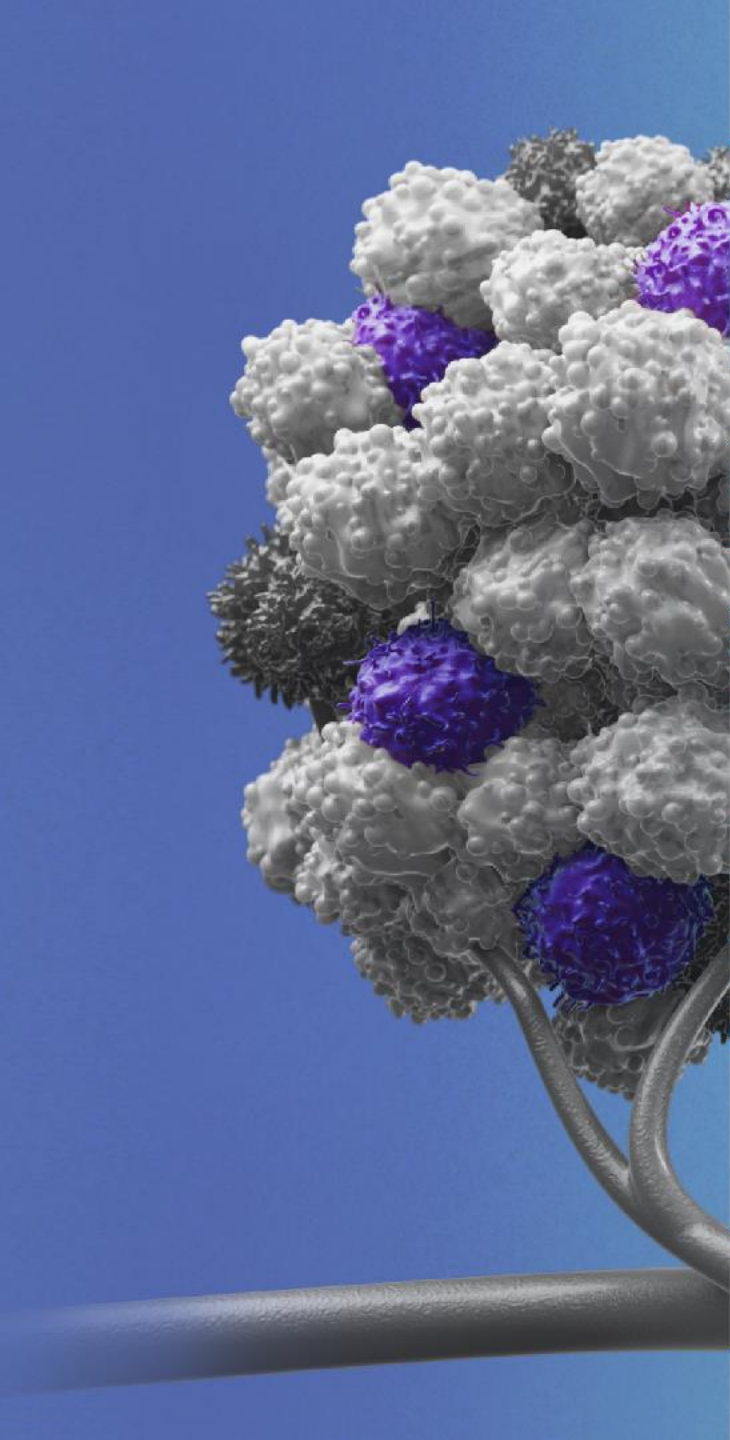
- Highest dose to date for any anti-VISTA antibody
- SNS-101 at a dose ~50x higher than the JNJ dose (0.3mg/kg) that caused DLT and termination of trial
- Well-tolerated with no observed DLTs[^]
- No routine prophylaxis per protocol

Anti-Tumor Activity

- Preclinical data demonstrate monotherapy activity in PD-1 resistant tumor model and deepened anti-tumor responses to PD-1 combo
- Topline monotherapy & combination data expected in Q2 2024

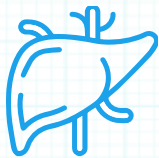
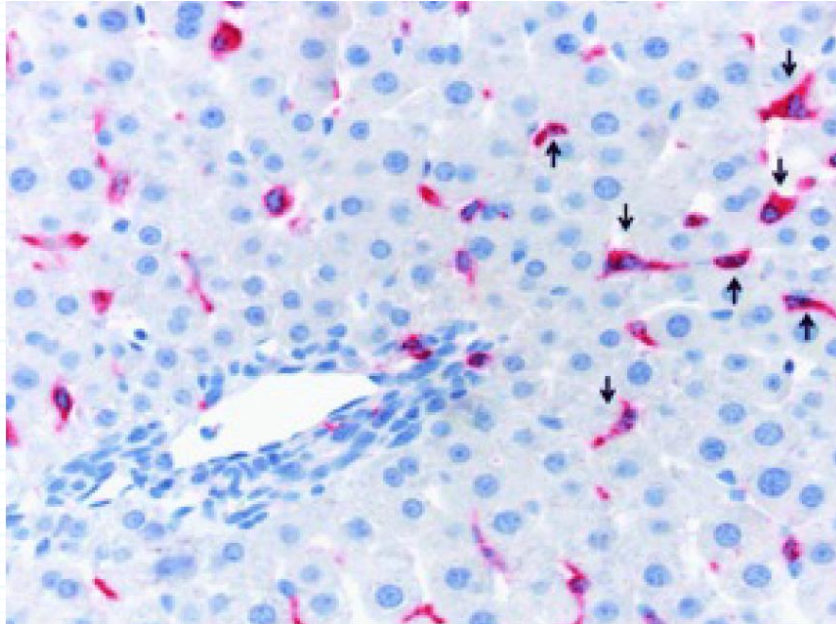
Completed and Anticipated SNS-101 Clinical Milestones





VSIG4 is an Immunosuppressive Receptor with On-Target, Off-Tumor Challenges

Tissue macrophages (Kupffer cells)
in the liver

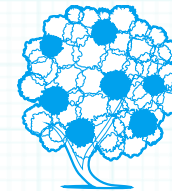
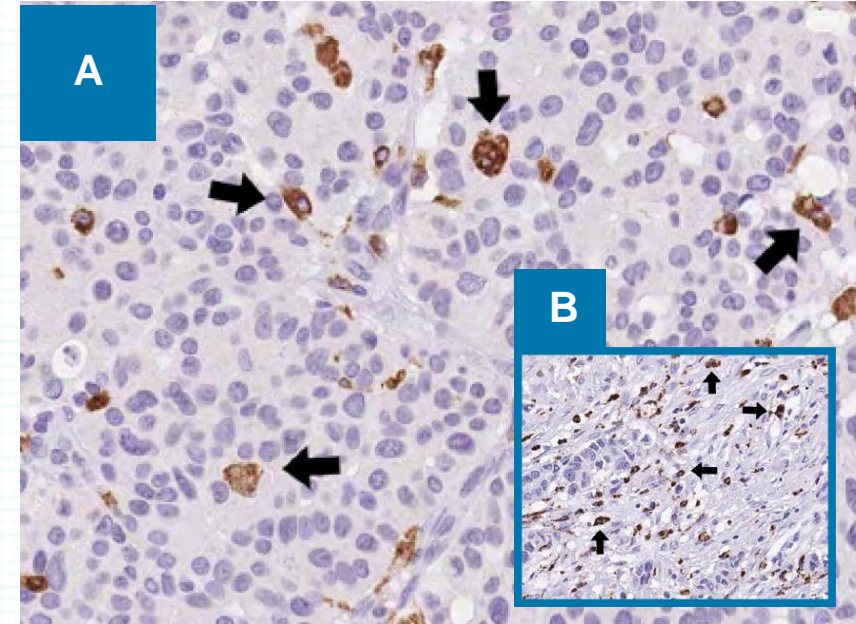


In the liver, VSIG-4 ...

Is expressed on Kupffer cells¹⁻²

Appears to drive significant target-mediated drug disposition (TMDD) and clearance

Tumor-associated macrophages in
tumors & stroma (inset)



In the tumor microenvironment, VSIG-4 ...

Correlates with immunosuppressive "M2" macrophage infiltration³

Inhibits T cell activation⁴

Promotes tumor growth based on data from a syngeneic Lewis lung carcinoma model in knockout mice⁵

SNS-102 is a pH-sensitive Antibody Designed With the Goal of Reversing T-cell Suppression within the Tumor Microenvironment

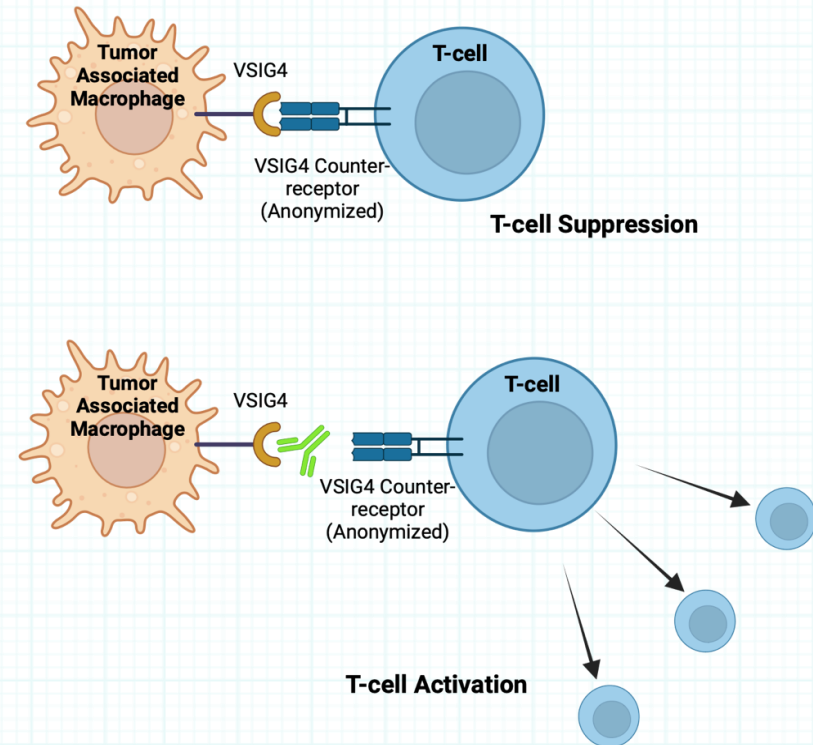
SNS-102 blocks the interaction of VSIG4 with its novel counter-receptor, which has been provisionally identified

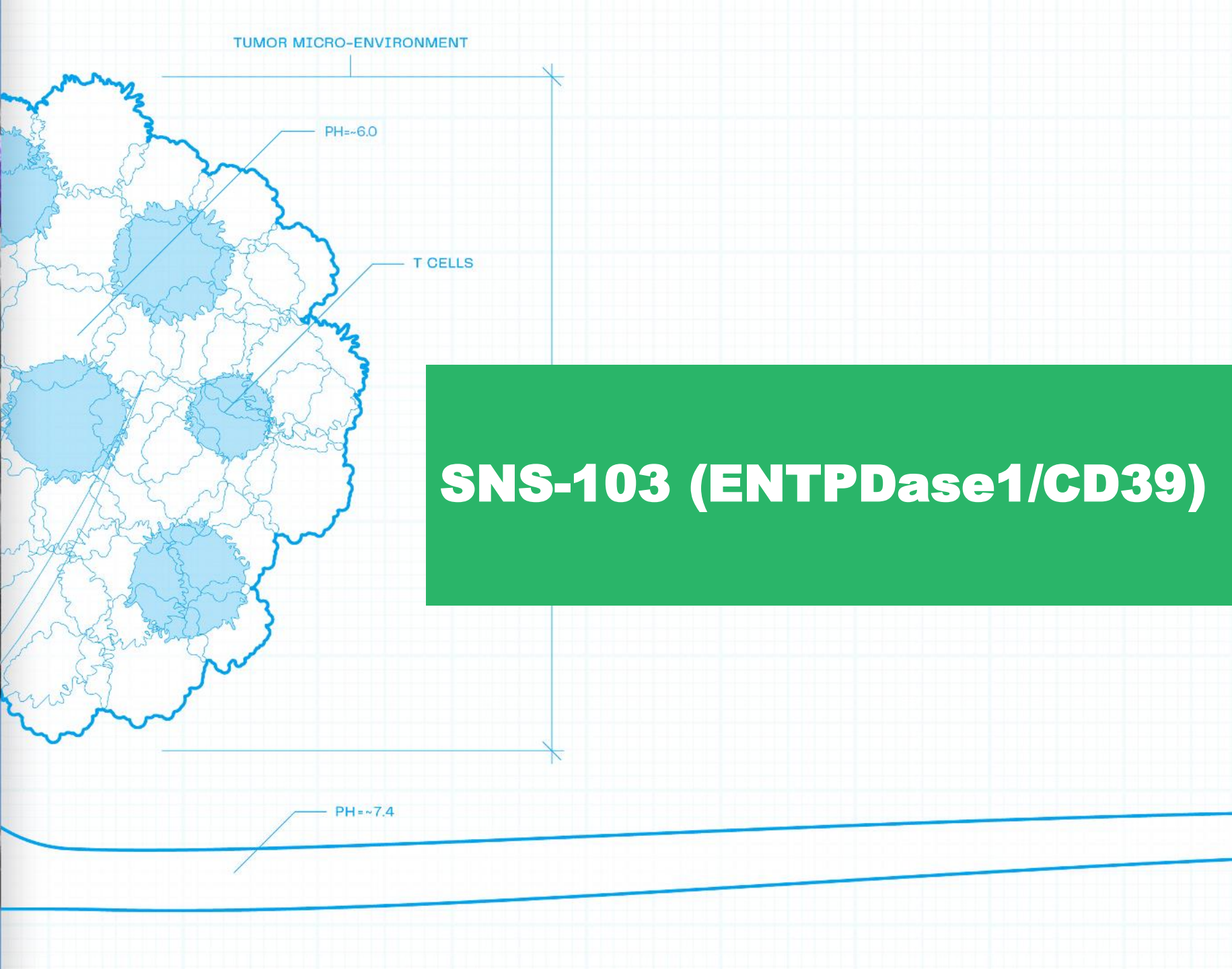
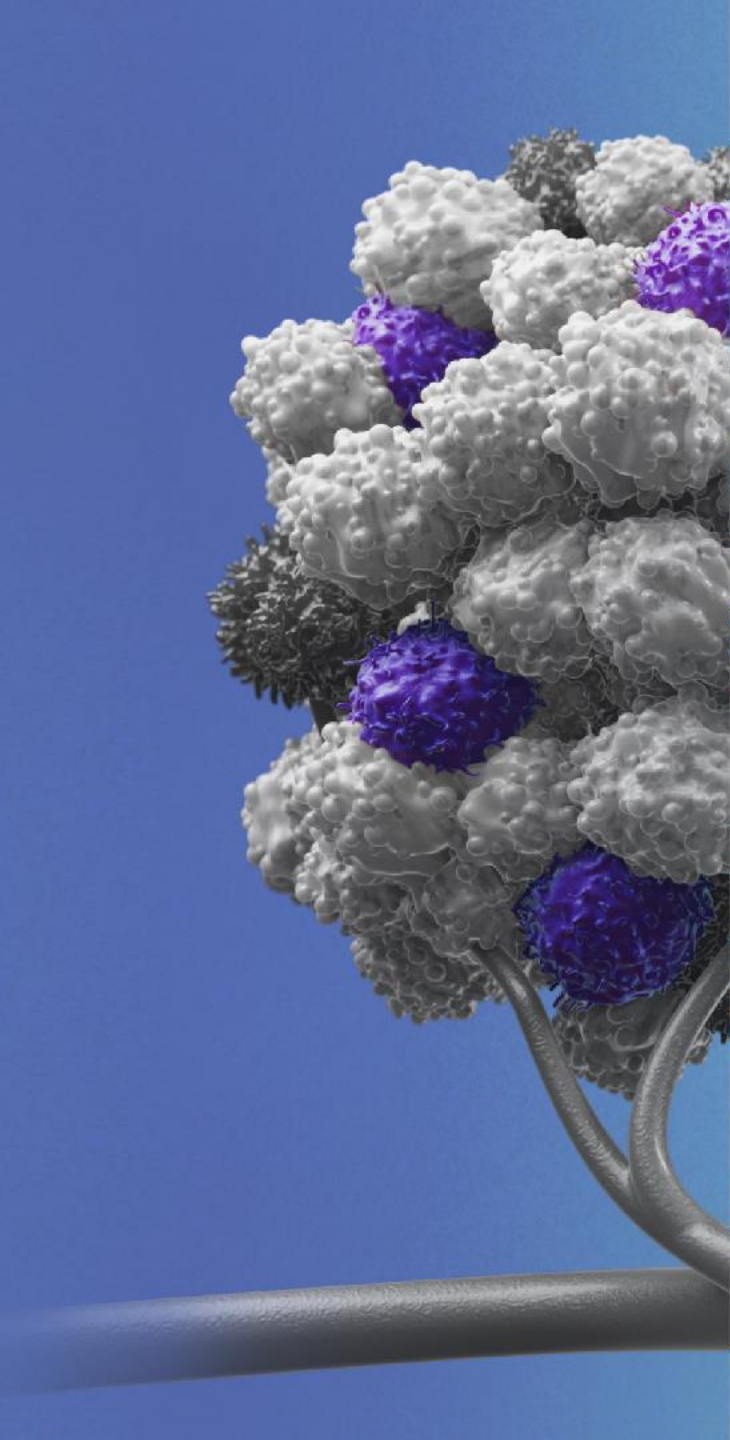
Monovalent Affinity (K_D)

pH 6.0	pH 7.4
0.7nm	410 nm (~No binding)

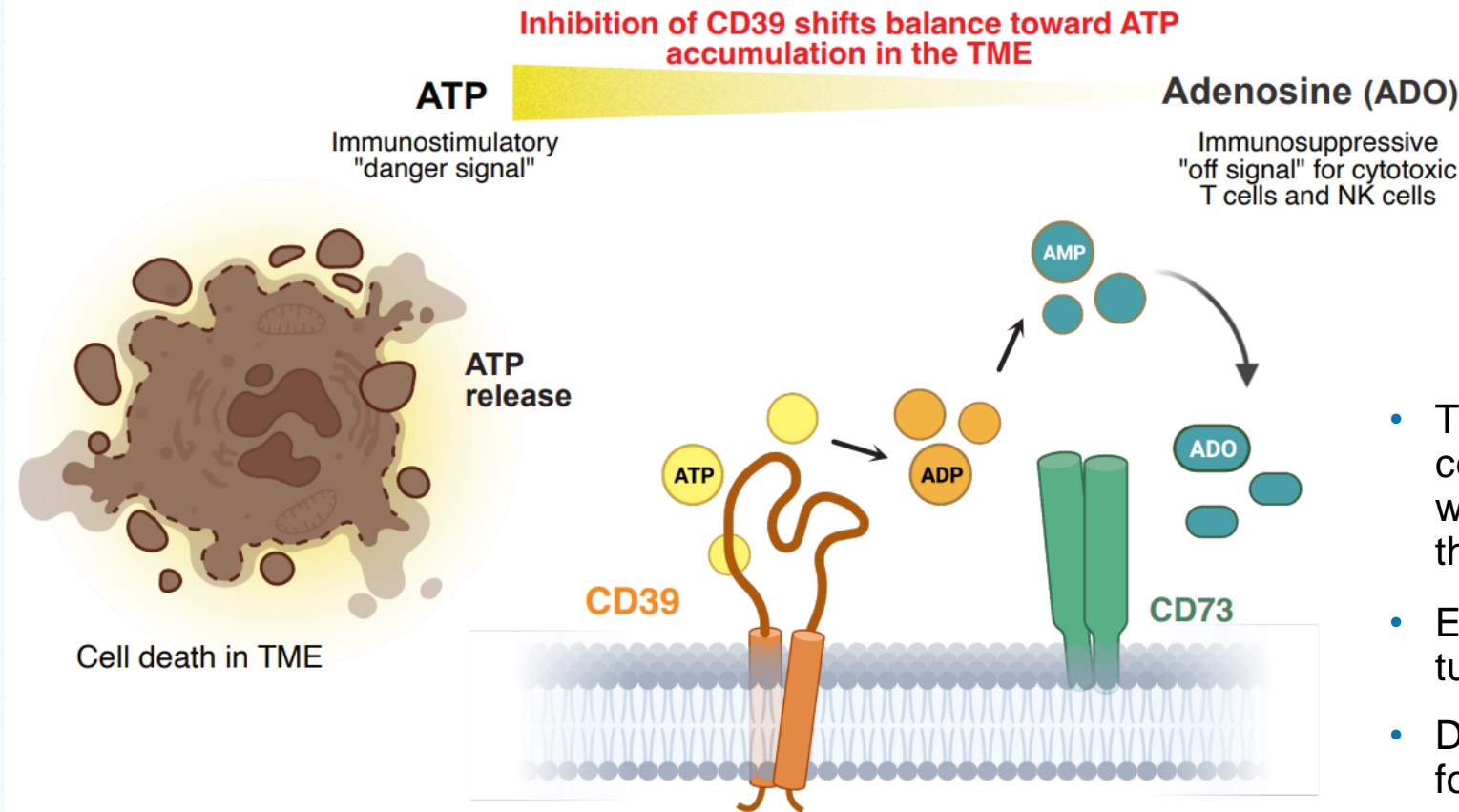
Ratio = 585

SNS-102 is **585-fold more selective** for VSIG4 at low pH conditions

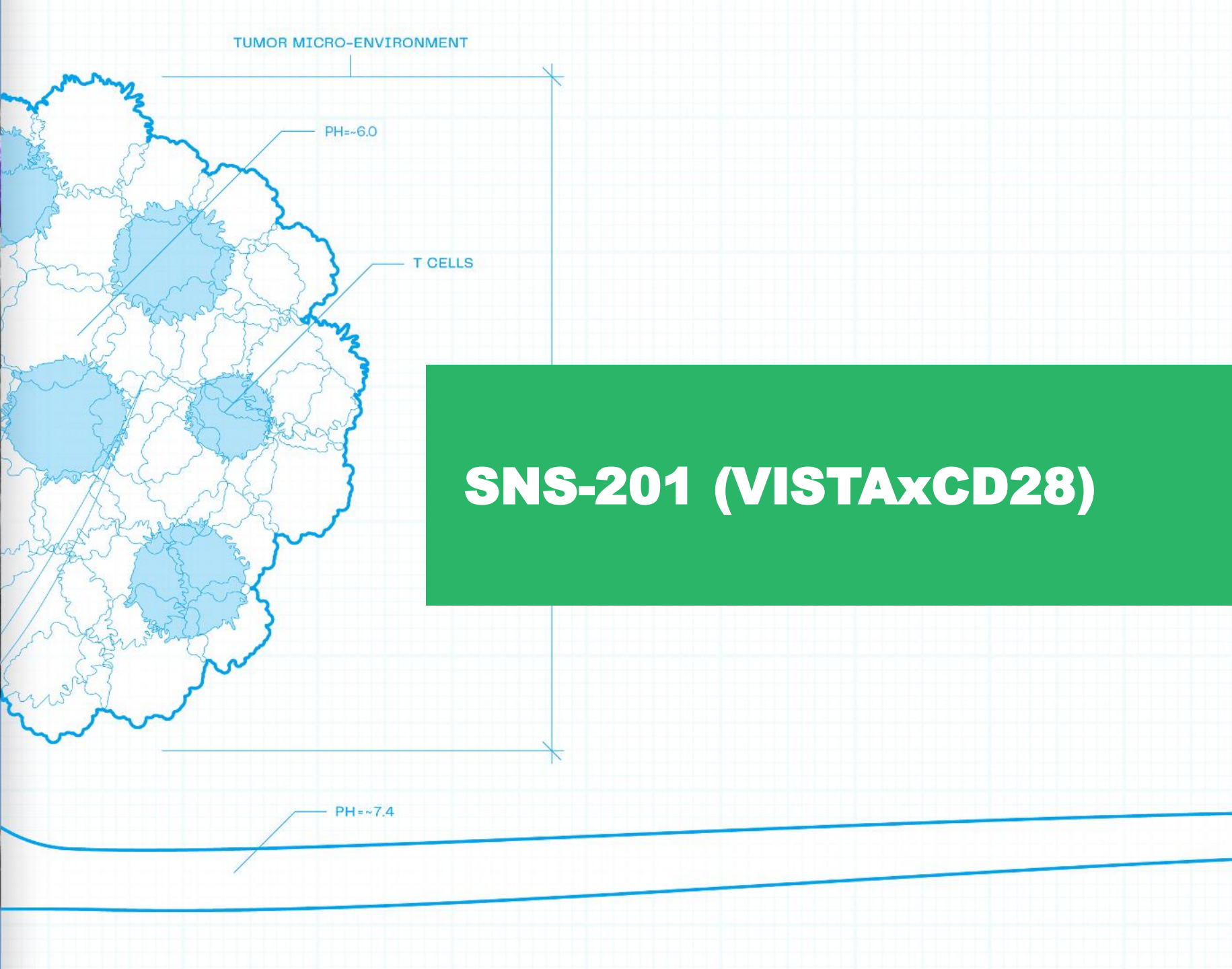
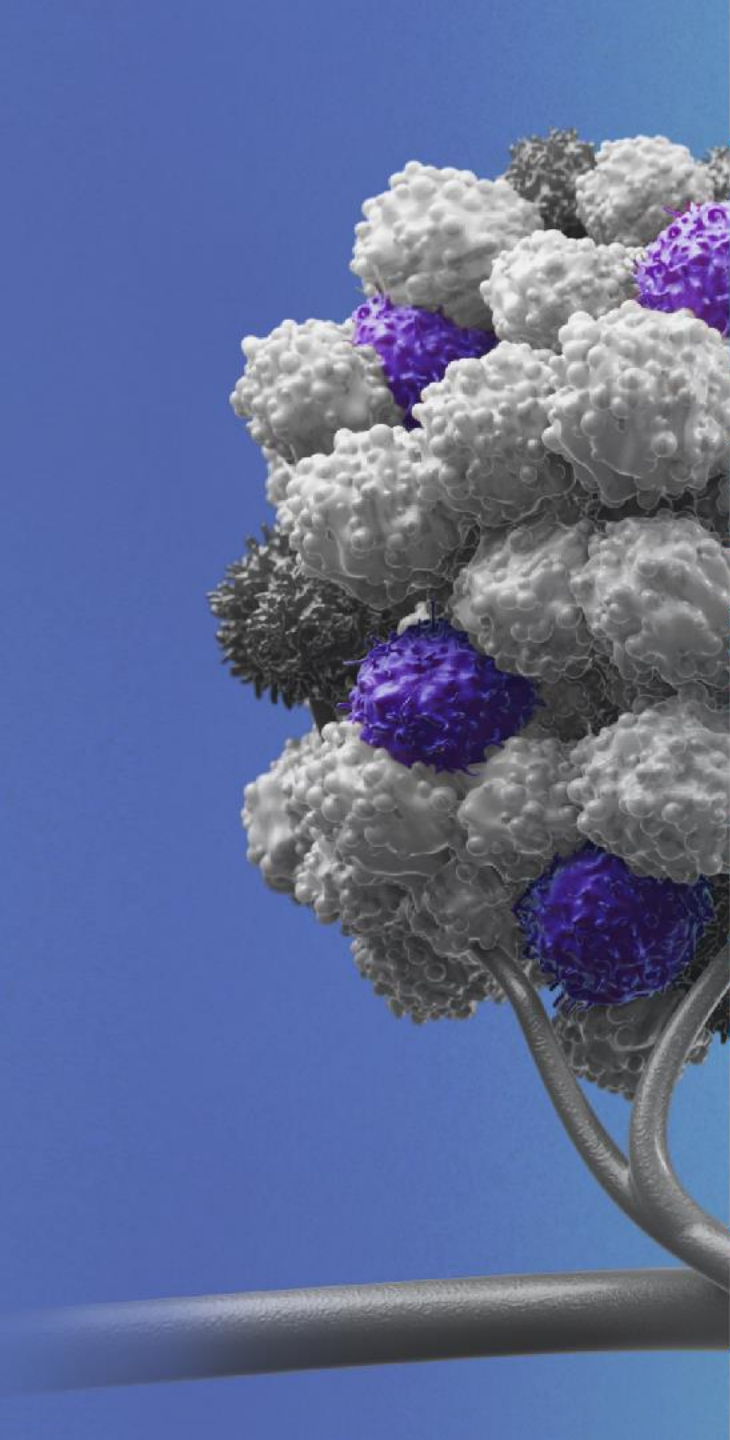




ENTPDase1 (CD39) is the Rate Limiting Enzyme in the Production of Immunosuppressive Adenosine



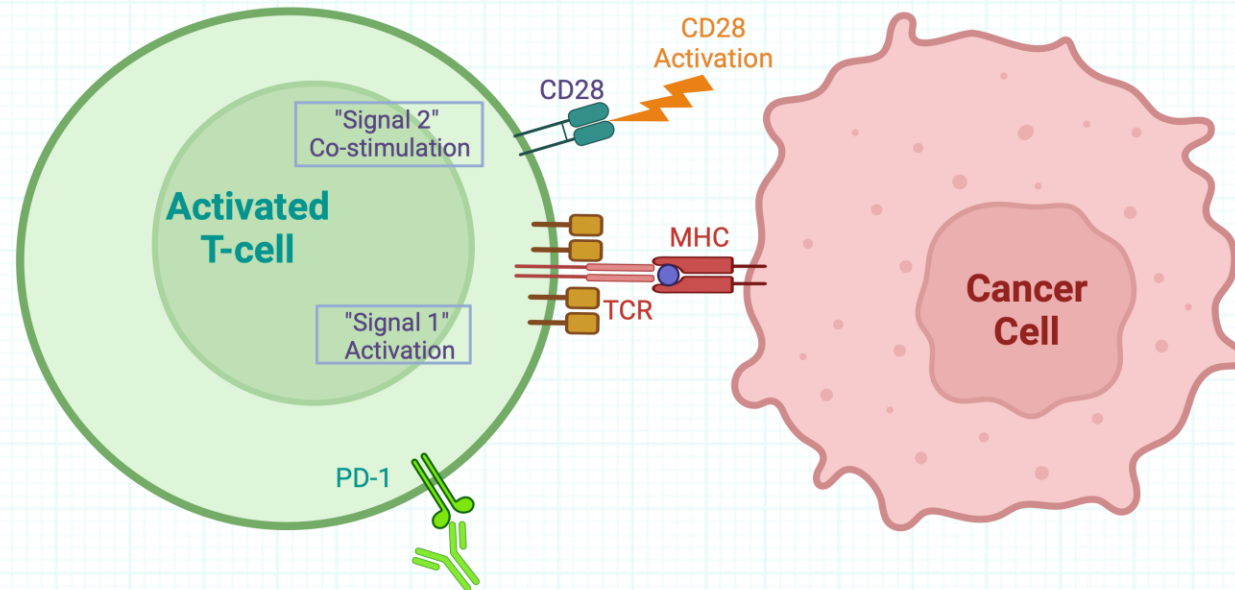
- The primary function of ENTPDase 1 is conversion of extracellular ATP to adenosine, which exerts immunosuppressive properties through binding to A2a/A2b receptors
- Expressed on various immune cells in both tumors and normal tissues
- Development of a TMAb antibody has potential for improved safety and PK profile compared to competitor CD39 mAbs



SNS-201 (VISTA \times CD28)

Overcoming Toxicity Challenges Associated with Targeting CD28

CD28 is a major co-stimulatory pathway for T cells and a clinically validated therapeutic target for activating T cells in the tumor microenvironment



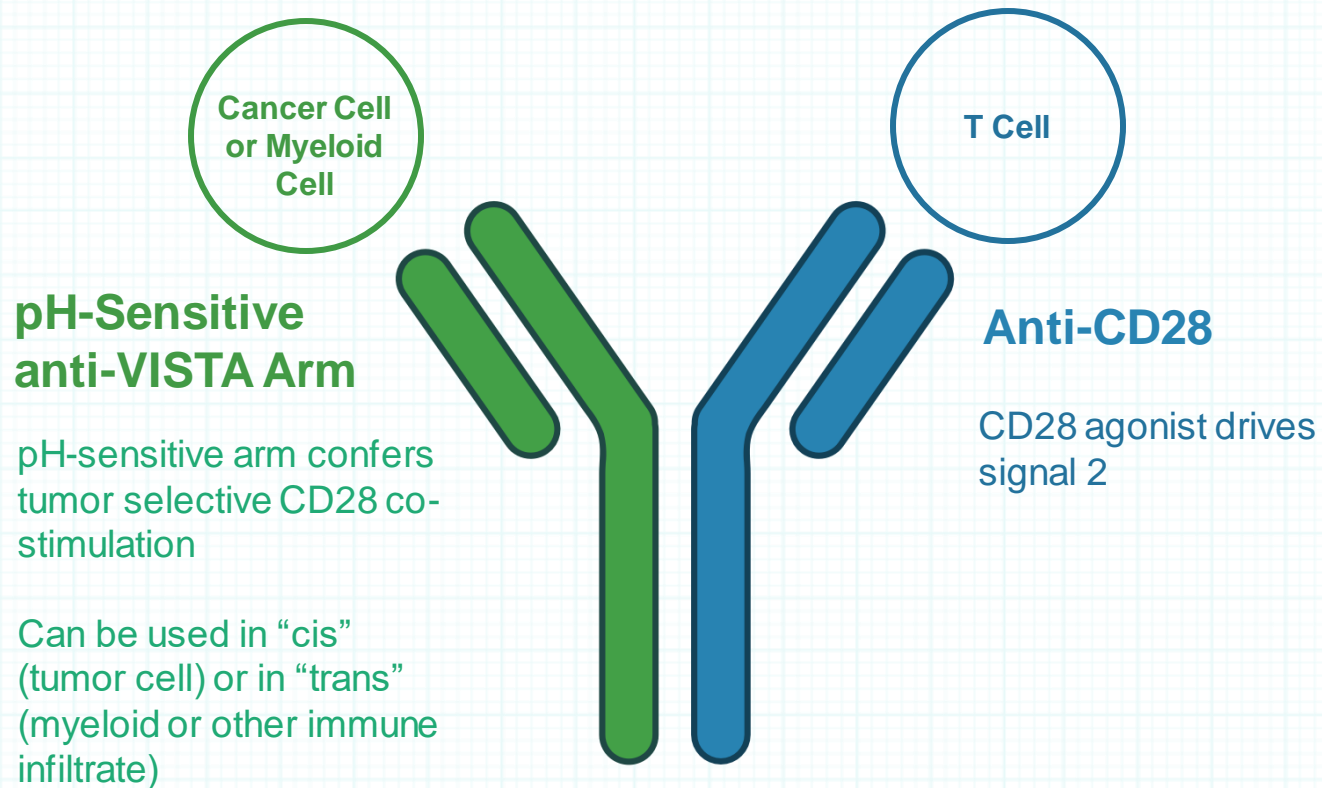
The Challenge

Targeting CD28 has shown clinical evidence of efficacy, but has been limited by dose-limiting toxicities resulting from systemic CD28 activation

Sensei's Solution

Leverage TMAb approach to potentially restrict CD28 activation to the tumor microenvironment, with no co-stimulation in the periphery

Bispecific TMAb Approach Can Generate T Cell Co-Stimulation Selectively Within the TME



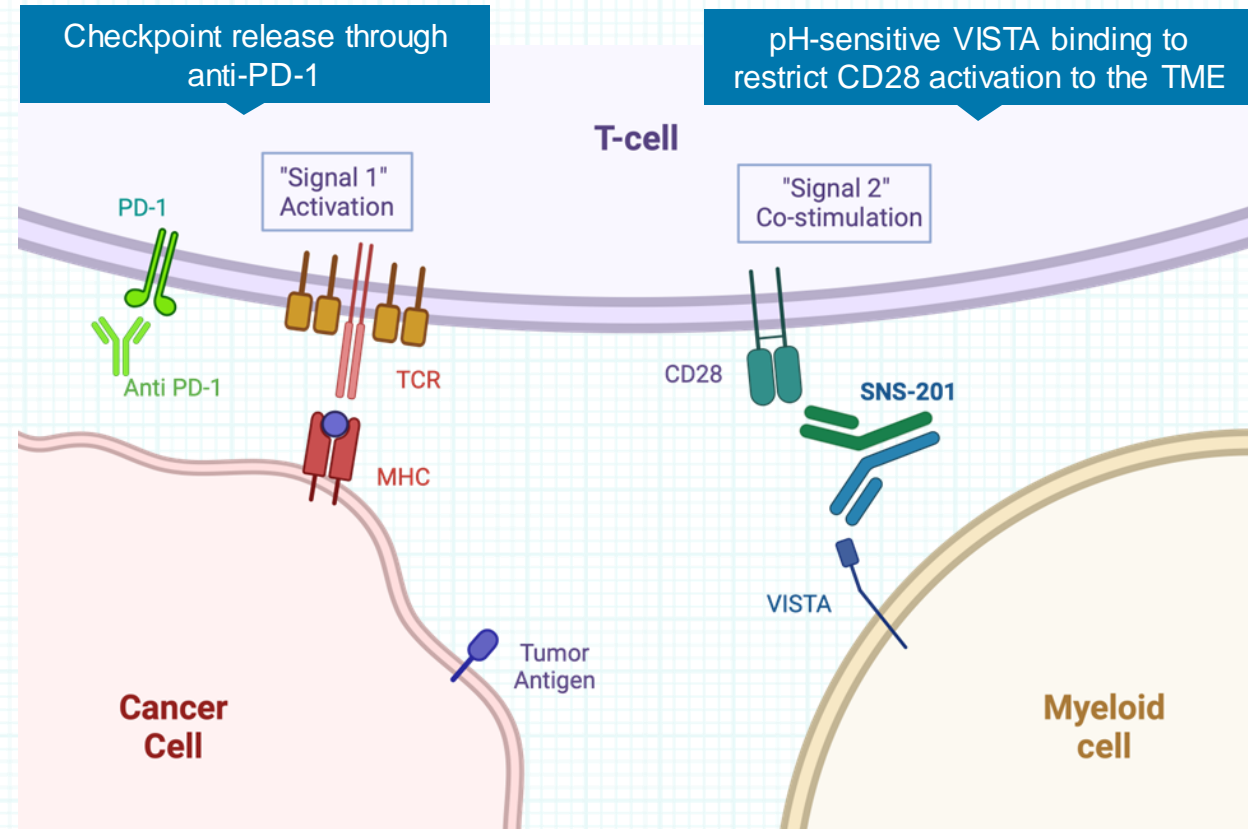
Co-Stimulatory VISTA \times CD28 Bispecific

- ✓ Powerful co-stimulatory "signal 2" selectively within the TME
- ✓ Potential for little or no toxicity due to selective targeting
- ✓ No linkers or masks
- ✓ A single, off-the-shelf bispecific approach
- ✓ Avoids use of "tumor associated" antigens

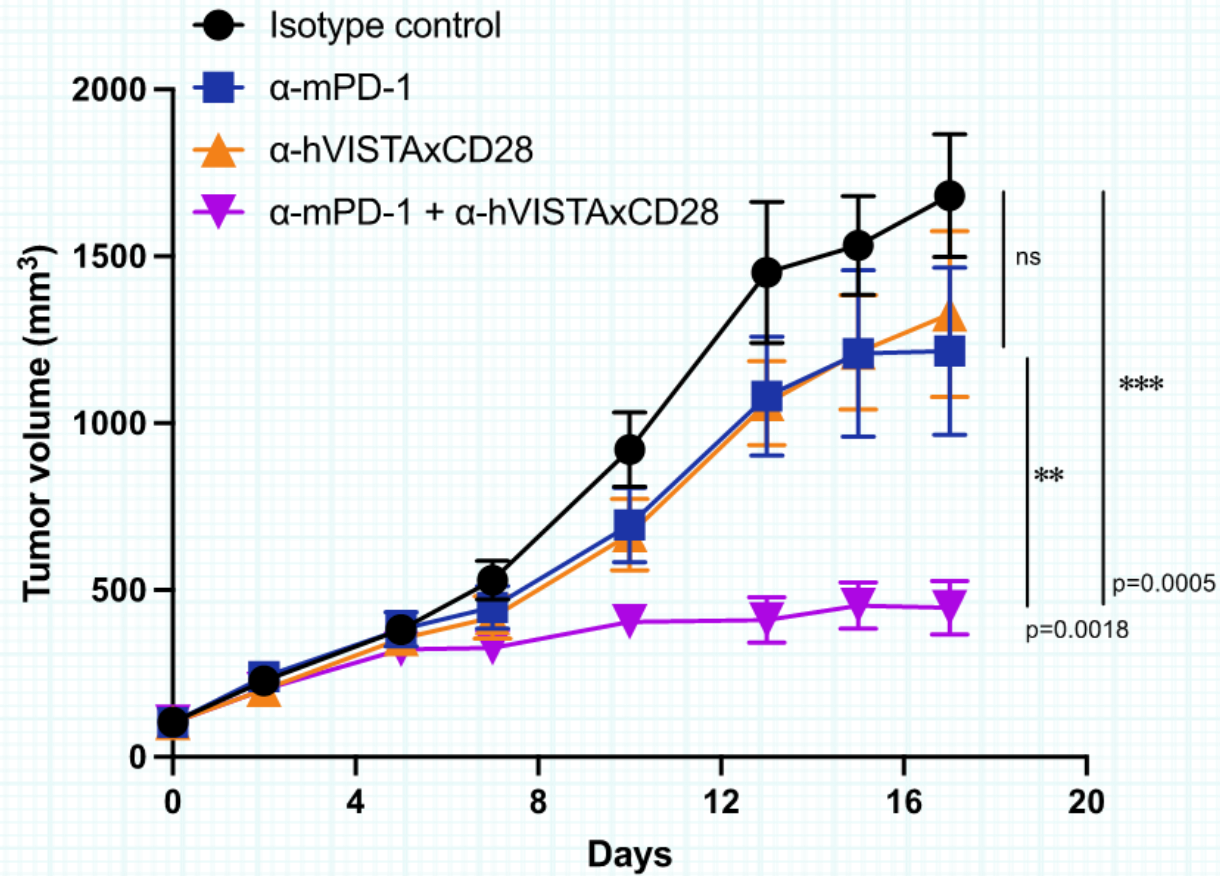
SNS-201 Provides Potential for Profound Anti-Tumor Activity By Selectively Co-Stimulating T Cells

SNS-201 (VISTAxCD28)

- Bispecific format with monovalent CD28 engagement
- Bivalent pH-selective VISTA binding for efficient engagement at low pH with cells displaying moderate VISTA copy numbers
- IgG1 backbone with silencing mutations abolishing Fc receptor interactions



***In Vivo* Study Shows Prototype Bispecific CD28xVISTA Induces Significant Tumor Growth Inhibition in Combo with anti-mPD-1**



NS = not significant
*** = P<0.001
** = P<0.01

Engineered Selectivity to Extend the Clinical Reach of IO Agents



LEAD PROGRAM

- SNS-101, a conditionally active antibody targeting VISTA
- Initial Phase 1 data demonstrate well tolerated safety profile & potentially best-in-class pharmacokinetics (PK)



TMAb PLATFORM

- Conditionally active antibodies designed to widen therapeutic window and enable druggability of promising oncology targets



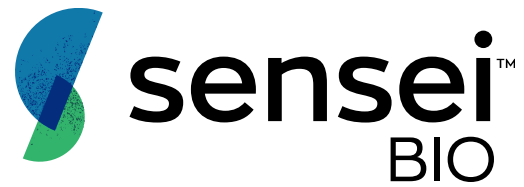
EXPECTED MILESTONES

- Topline monotherapy & combination dose escalation data in Q2 2024
- Initial dose expansion data by end of 2024



FINANCIALS

- Ended 2023: \$65.8M*
- Cash runway into Q4 2025
- Cash currently expected to reach midway into Phase 2 clinical studies for SNS-101



HQ: 1405 Research Blvd, Suite 125, Rockville, MD 20850 / **MA:** 22 Boston Wharf Rd, 7th floor, Boston, MA 02210

senseibio.com

SNS-101 Displayed Favorable Safety & Tolerability Profile Through 3 mg/kg Monotherapy

Well Tolerated with No Evidence of Cytokine Release Syndrome
and No Dose-Limiting Toxicities Observed

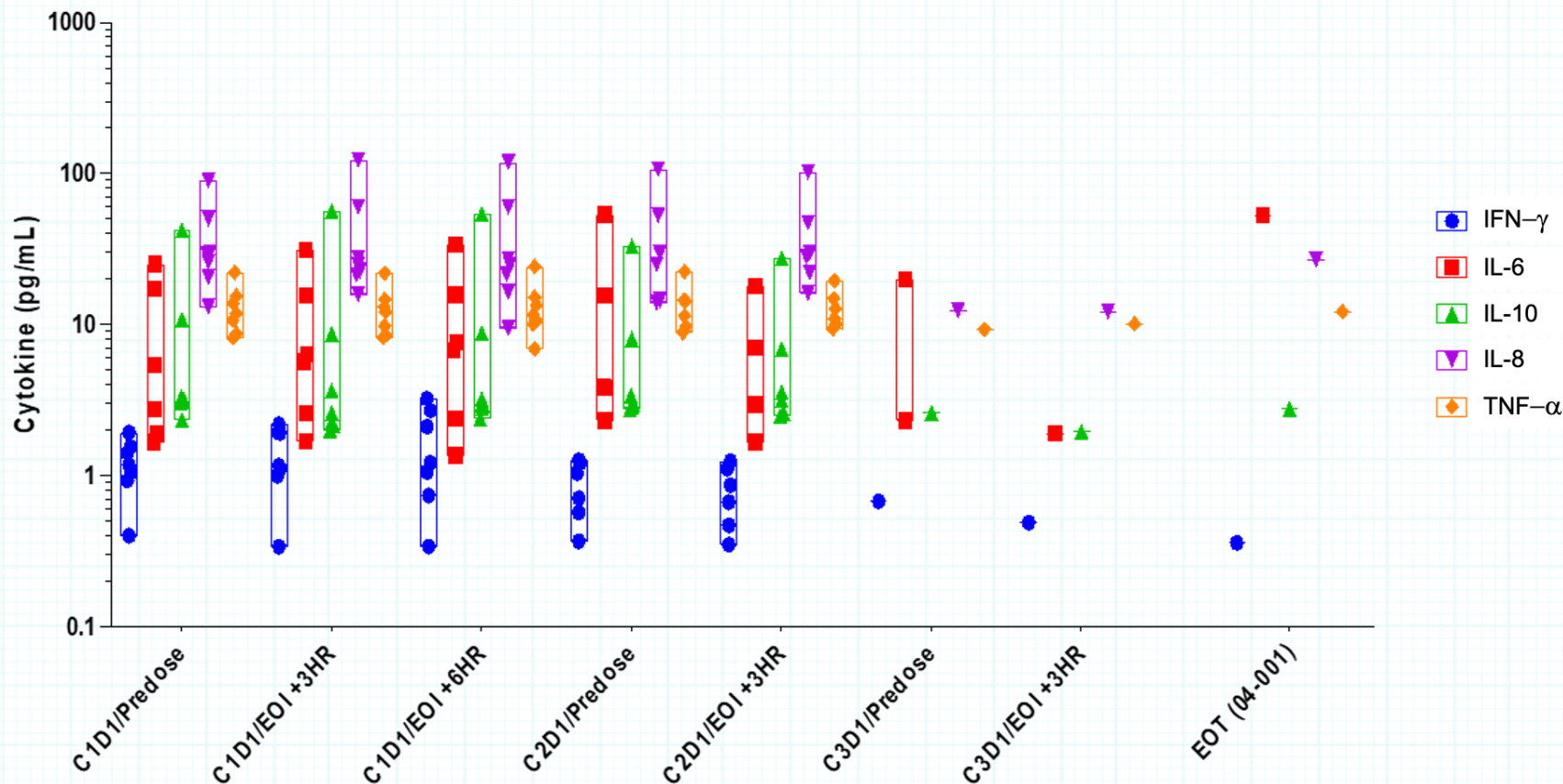
	0.3 mg/kg N=1 n (%)	1.0 mg/kg N=3 n (%)	3.0 mg/kg N=3 n (%)	Total N=7 n (%)
At least 1 TEAE	1	3	1	5 (71.4)
At least 1 SAE	0	0	1*	1* (14.3)
At least 1 TEAE leading to discontinuation	0	0	1*	1* (14.3)
DLTs	0	0	0	0
CRS events	0	0	0	0
≥Grade 3 TEAE	0	0	1*	1* (14.3)
Related TEAE	0	1 [#]	0	1 [#] (14.3)

*One patient experienced an SAE, Grade 5 bronchial obstruction, that resulted in death; Event was considered related to disease progression, not SNS-101.

[#] One patient experienced a Grade 2 dermatitis acneiform considered to be related to SNS-101. The event resolved following phototherapy treatment.

Monotherapy Data Consistent with Lack of Observed Cytokine Release Syndrome Through 3.0 mg/kg

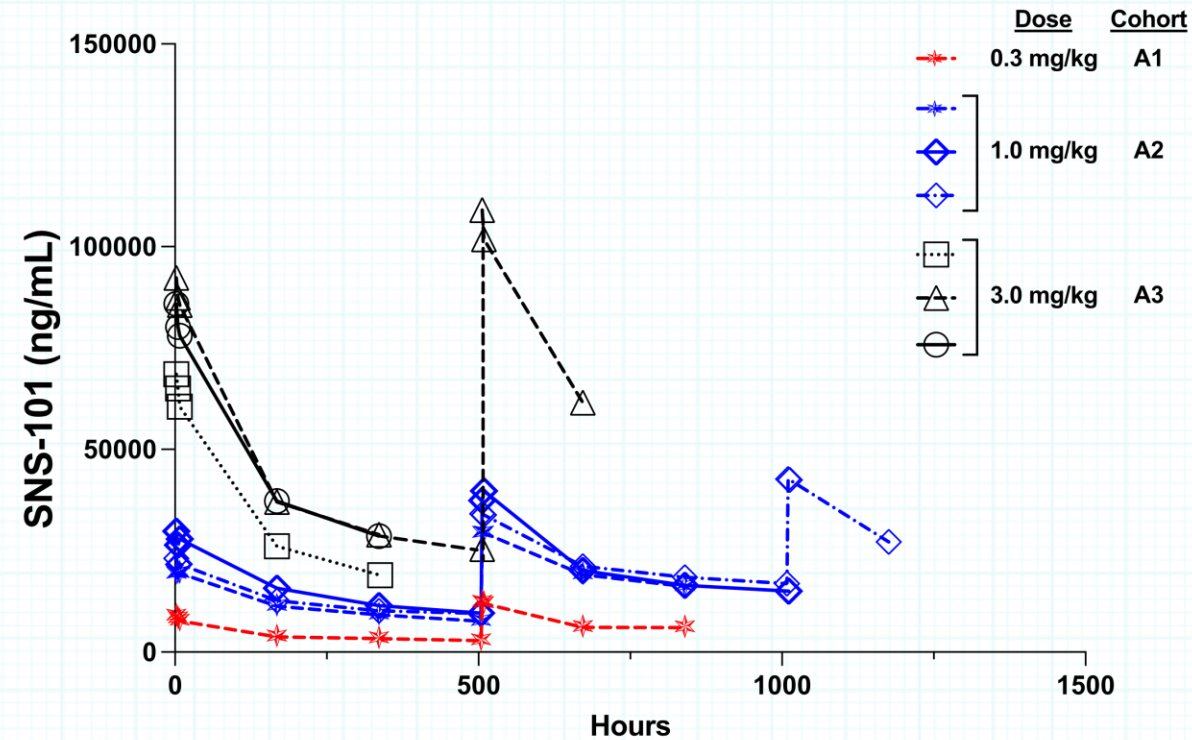
No Significant Changes in Key Inflammatory Cytokines



Cytokine analysis: Blood samples were taken pre-dose, 3 hours post-infusion and 6 hours post infusion at C1D1, and pre- and 3hr-post thereafter. Serum was assayed for indicated cytokines using a platform (MSD) that has been validated for clinical sample analysis.

Data from monotherapy dose escalation arm as of cut-off date of October 3, 2023




SNS-101 Monotherapy Data Show Linear Pharmacokinetics and Long Half-Life in Stark Contrast to Prior Anti-VISTA mAbs



Supports Every 3 Week Dosing

Key Partnerships Supporting SNS-101's Clinical Development

Potential opportunities for combination therapy and biomarker identification

Partner/ Collaborator	Goal	Description
 Clinical Supply Agreement	Support evaluation of SNS-101 in combination with Libtayo® (cemiplimab) in planned Phase 1/2 clinical trial	<ul style="list-style-type: none"> • Sensei to fund planned clinical trial • Regeneron to provide Libtayo® • Sensei maintains global development and commercial rights to SNS-101
 Cooperative Research & Development Agreement	Further elucidate role of VISTA in immune checkpoint resistance and expand potential of SNS-101 as a combination therapy beyond anti-PD-1	<ul style="list-style-type: none"> • Sensei collaborating with NCI Center for Immuno-Oncology Co-Directors, Jeffrey Schlom, Ph.D., and James Gulley, M.D., Ph.D. • Preclinical studies will assess SNS-101 mechanism of action in combination with therapies beyond anti-PD-1
 Research Collaboration	Further study the mechanism of SNS-101's anti-tumor activity	<ul style="list-style-type: none"> • Sensei collaborating with laboratory of immuno-oncology KOL, Robert Schreiber, Ph.D. • Preclinical studies will include identification of SNS-101 response biomarkers

Commercially Validated Precedent for pH-sensitive Approach

Soliris® (eculizumab), an anti-C5 monoclonal antibody (mAb), was considered standard of care for patients with paroxysmal nocturnal hemoglobinuria (PNH) and atypical hemolytic uremic syndrome, but had a short half-life as a result of extensive TMDD.

Engineering eculizumab using histidine substitutions resulted in a pH-sensitive mAb with markedly improved half-life.

Antibody Engineering of Solaris Led to pH Sensitivity and Half-Life Improvements

	Soliris (Eculizumab)	→	Ultomiris (Ravulizumab, ALXN1210)
K _D pH 7.4 (nM)	0.03		0.49
K _D pH 6.0 (nM)	0.6		22
t _{1/2} (d)	3.9		13.4

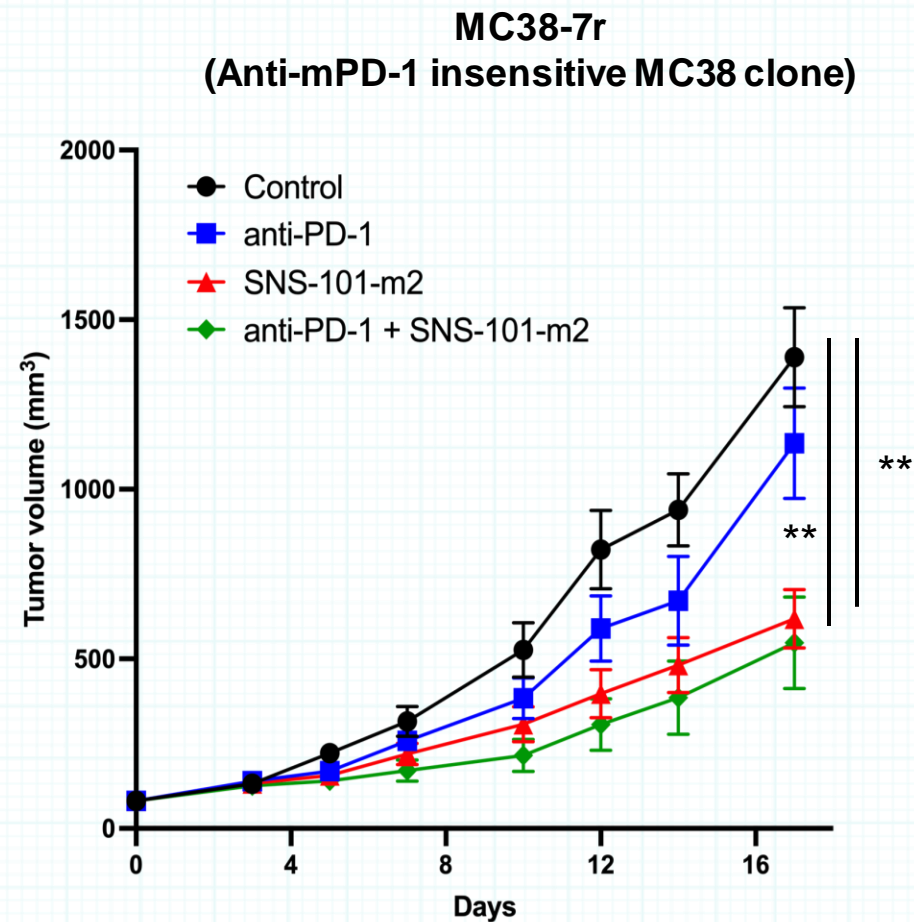
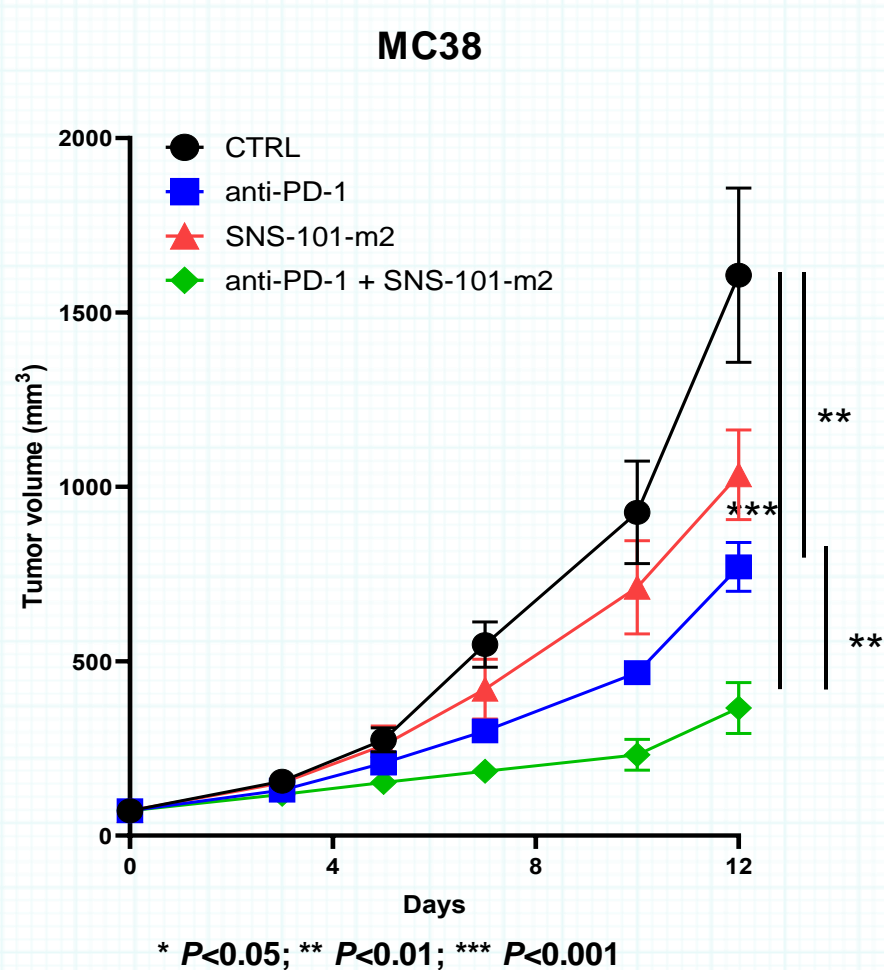
Ravulizumab utilized histidine insertions into the CDR regions (VH_Y27H, VH_S57H) and Fc substitutions (M428L, N434S) of eculizumab

Due to its longer half-life (13.4d vs 3.9d), ravulizumab given every 8w achieved noninferiority compared with eculizumab given every 2w for all efficacy endpoints, while maintaining a similar safety profile.

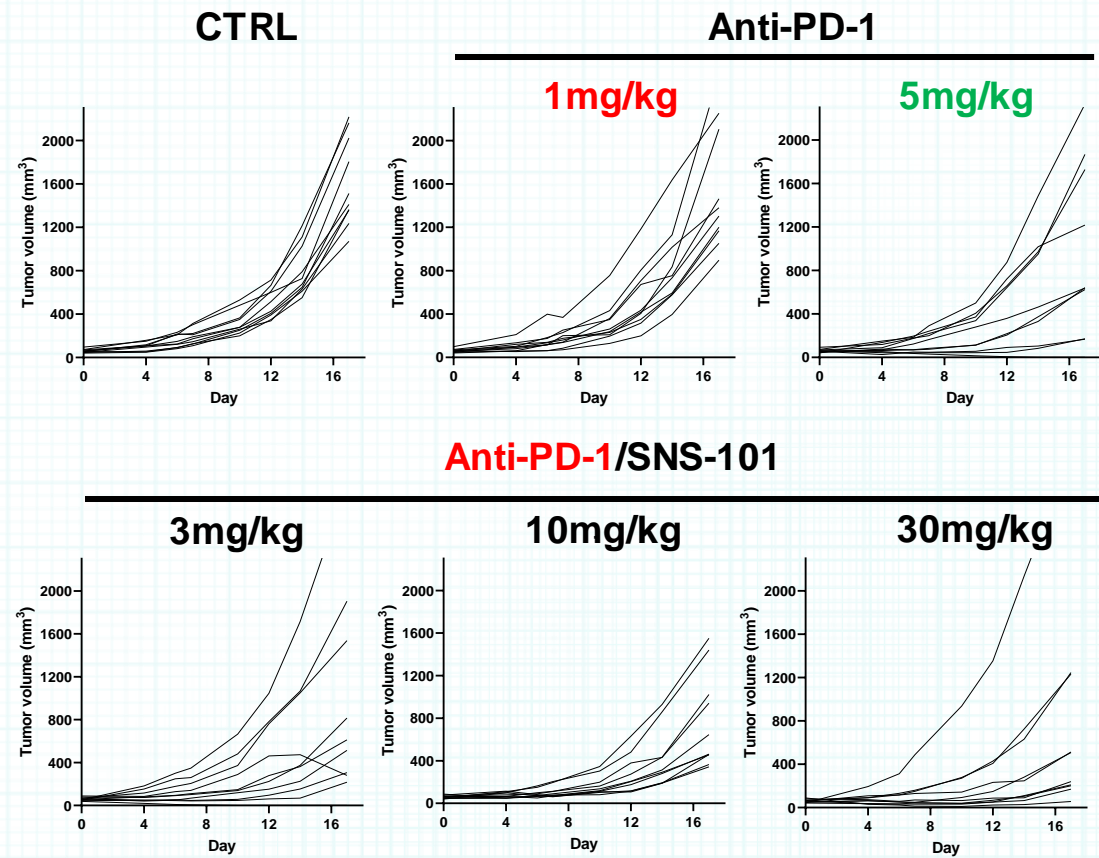
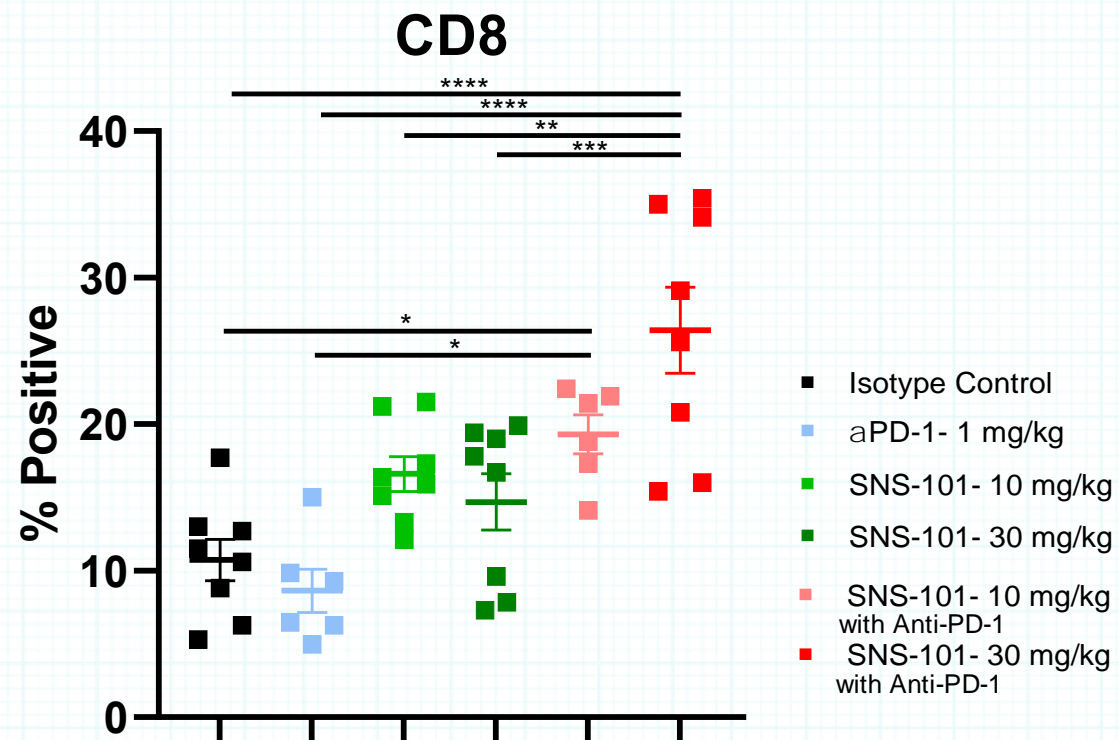
2018: FDA approves Ultomiris® (ravulizumab, ALXN1210)

2020: Ultomiris sales = \$1.08 billion

Single-agent Activity and Deepened Anti-tumor Responses to PD-1 Combo in Human VISTA KI Mice *In vivo*



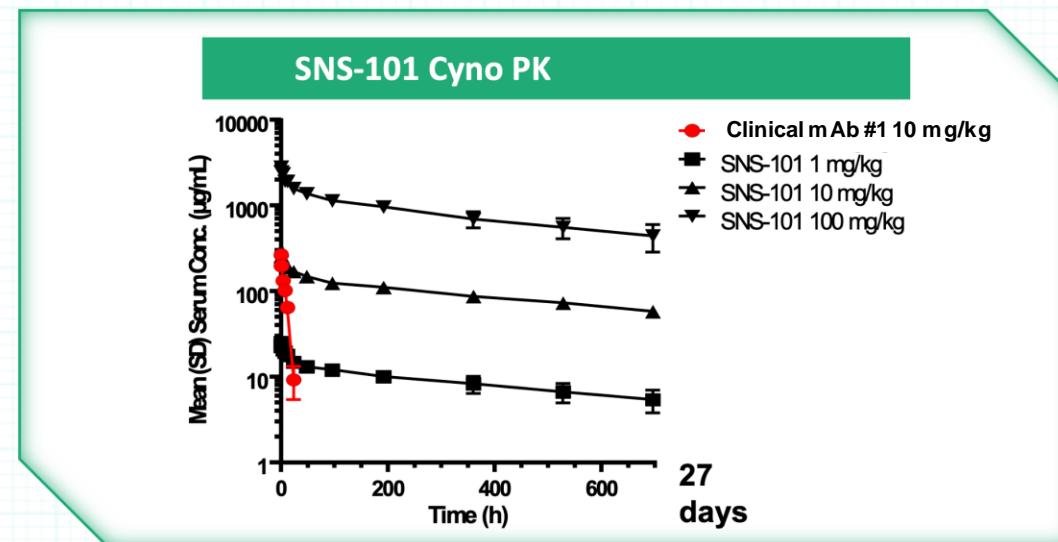
SNS-101 Increased CD8 T-cells in Combination With Anti-PD-1 in MC38 Tumors *In Vivo*



PK Sink Continues to be an Issue with Non-pH-Sensitive VISTA programs*

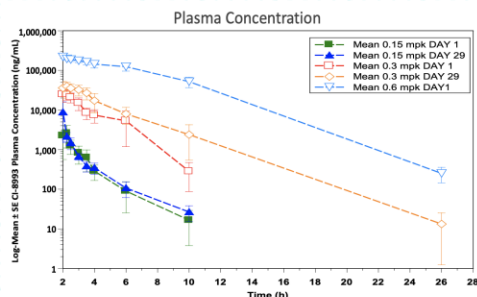
SNS-101 is designed to overcome elimination kinetics and half-life related to PK sink observed in non-pH-sensitive VISTA programs

Linear

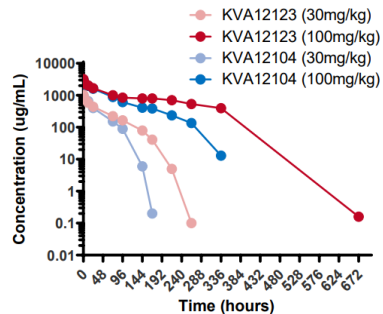


Non-linear

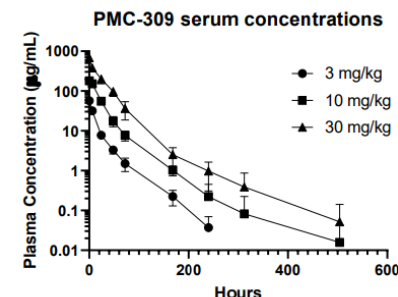
Curis CI-8993 Plasma Concentration



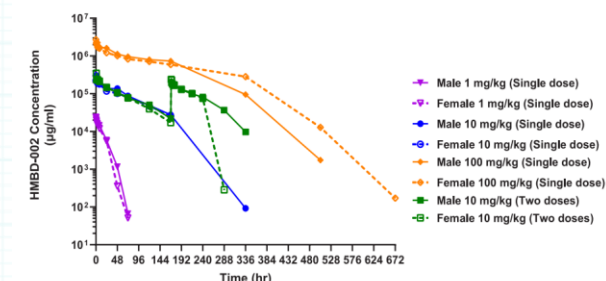
Kineta KVA12123 Cyno PK



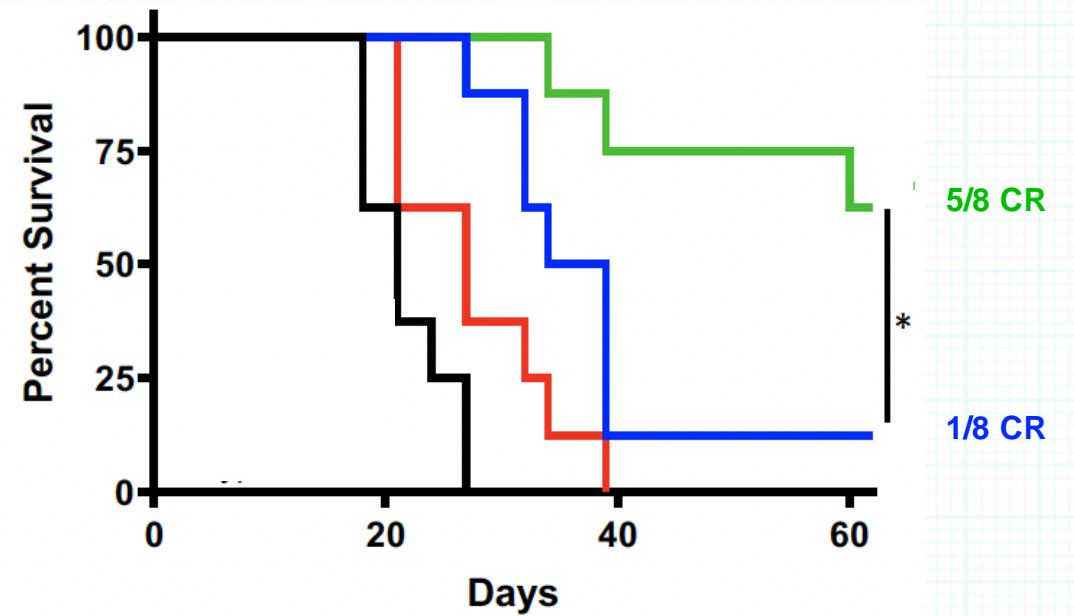
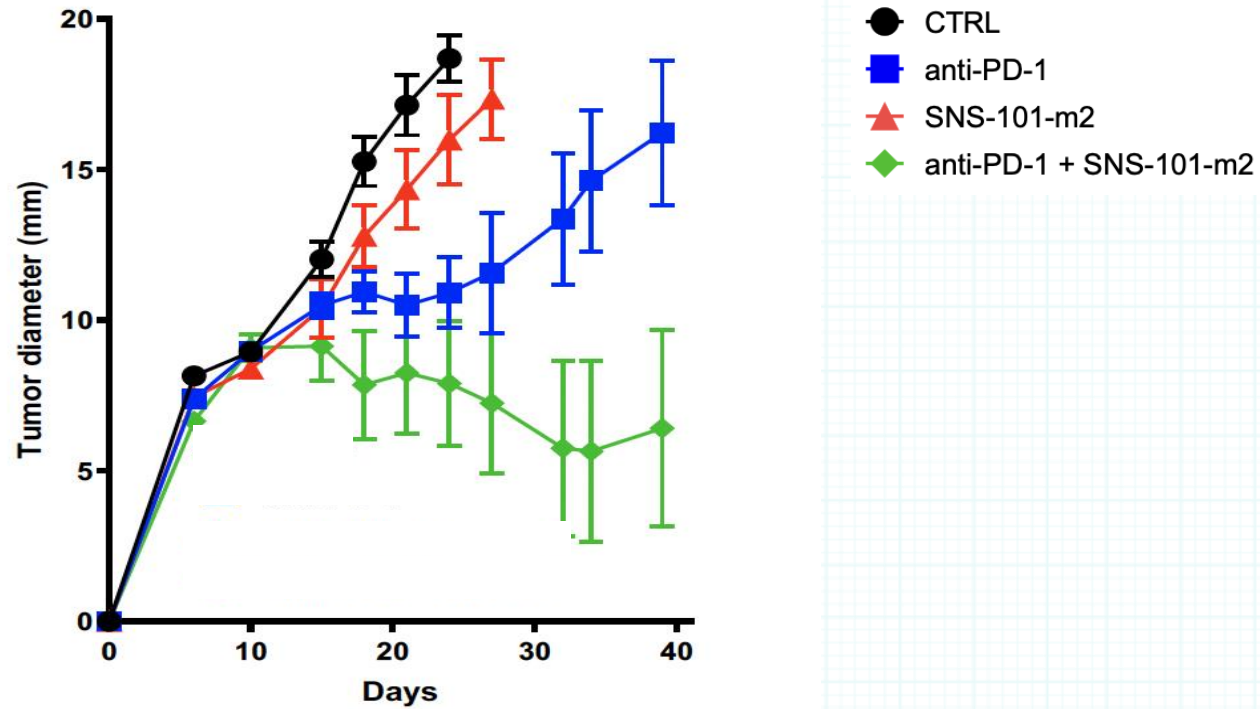
Abcine PMC-309 Serum Conc Cyno



Hummingbird HMBD-002 Preclinical PK

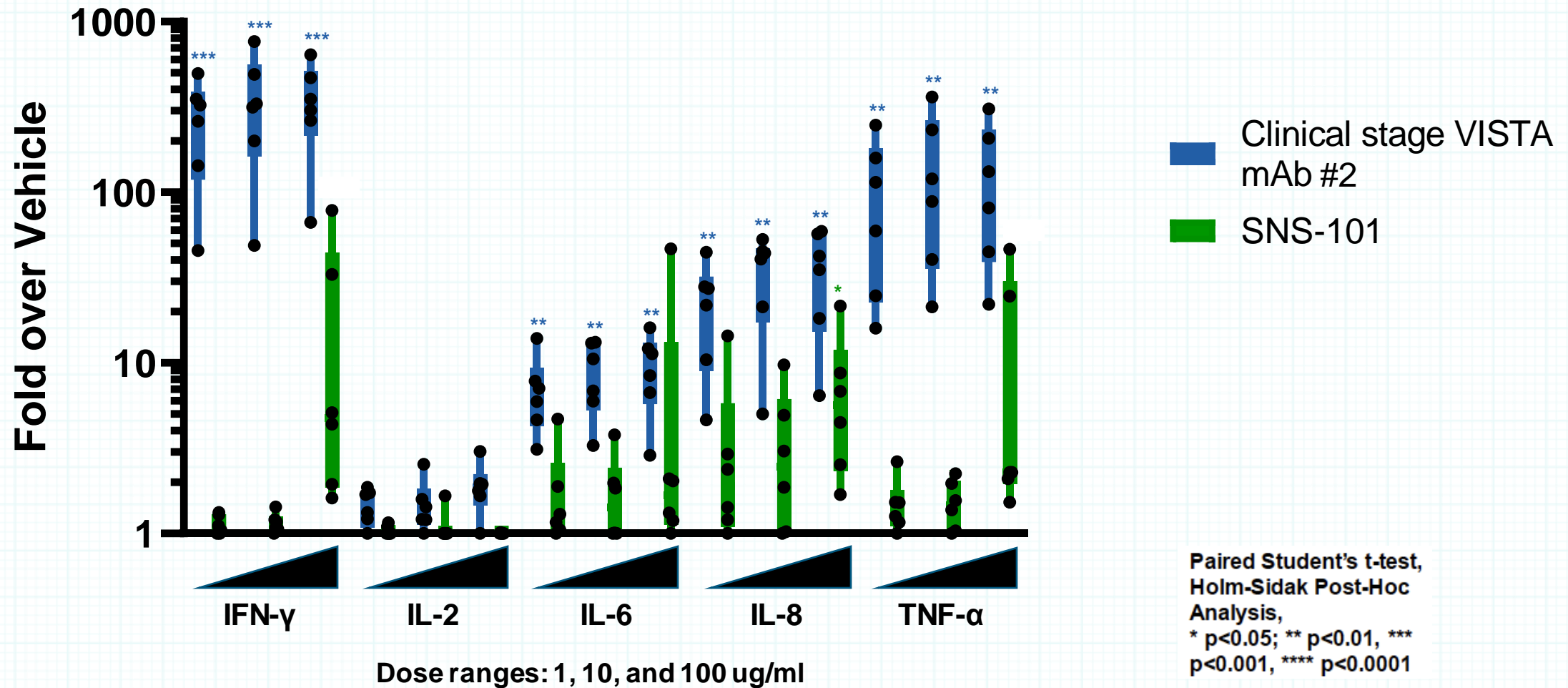


SNS-101 Re-sensitized Anti-PD-1 Insensitive Sarcoma Tumors in Human VISTA Knock-in Mice



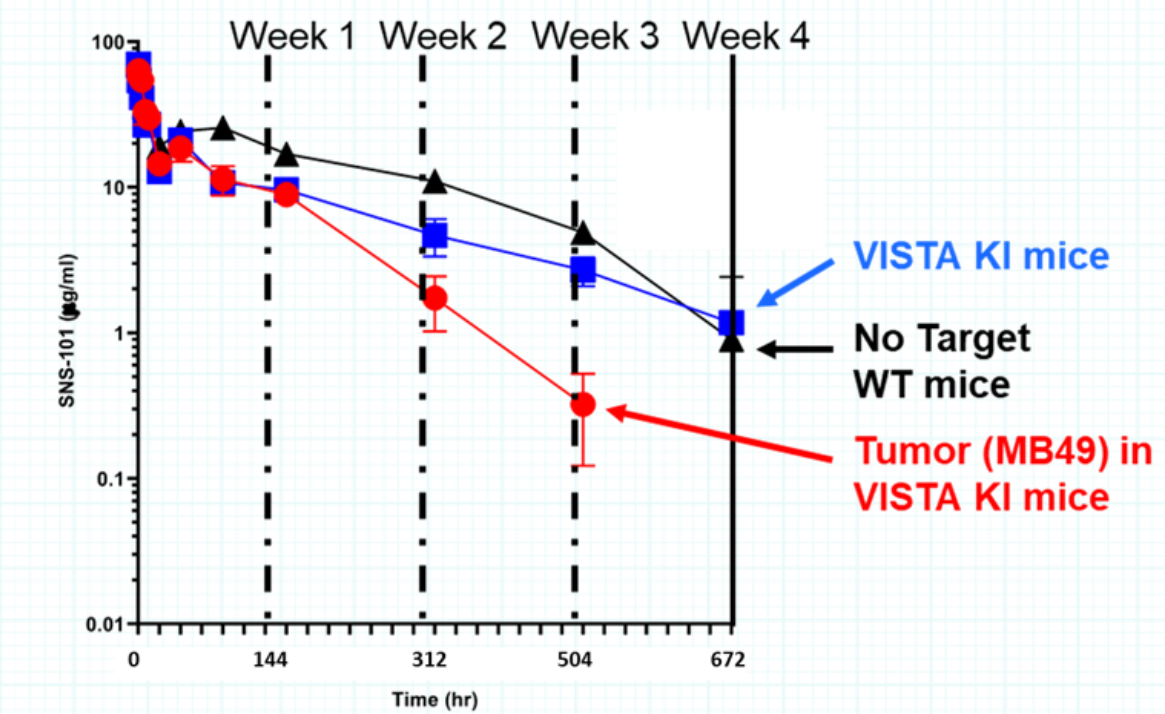
* p<0.05

SNS-101 Induced Substantially Lower Cytokine Release in Whole-blood Assay at Neutral pH Compared to pH-independent VISTA Antibody



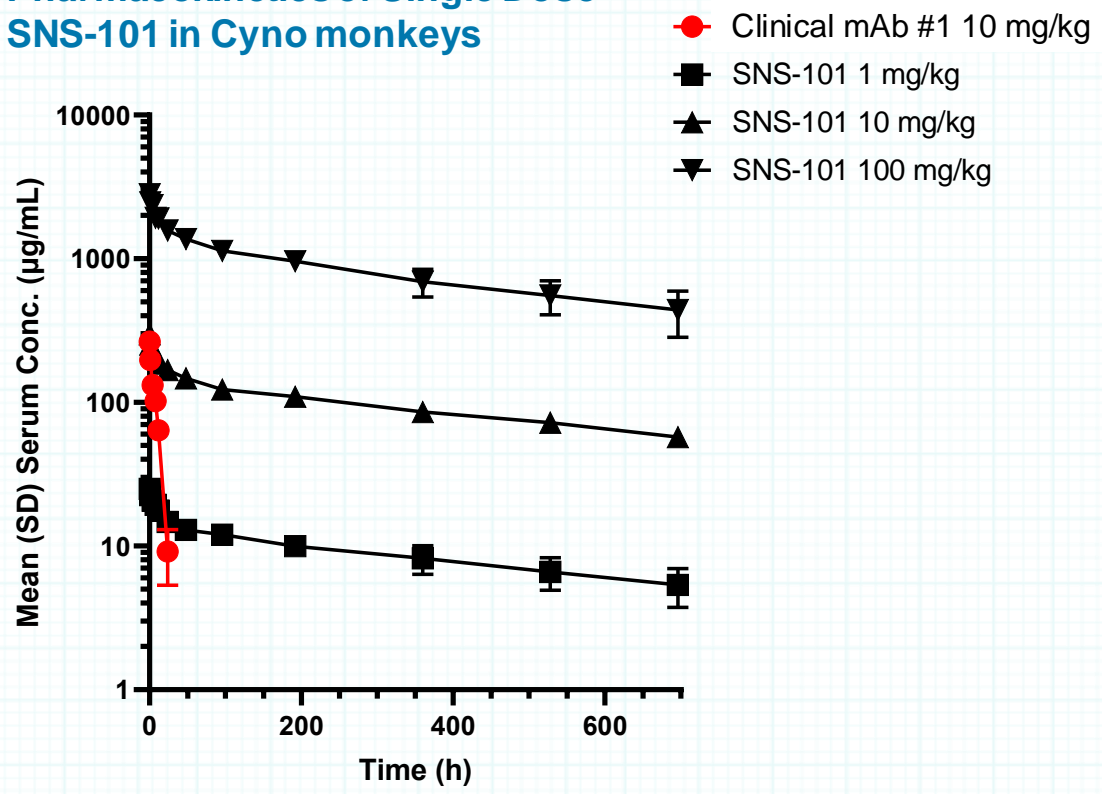
SNS-101 Has Displayed a Favorable Single-dose PK Profile in Preclinical Studies - *No Significant TMDD in Human VISTA KI Mice or Non-human Primates*

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



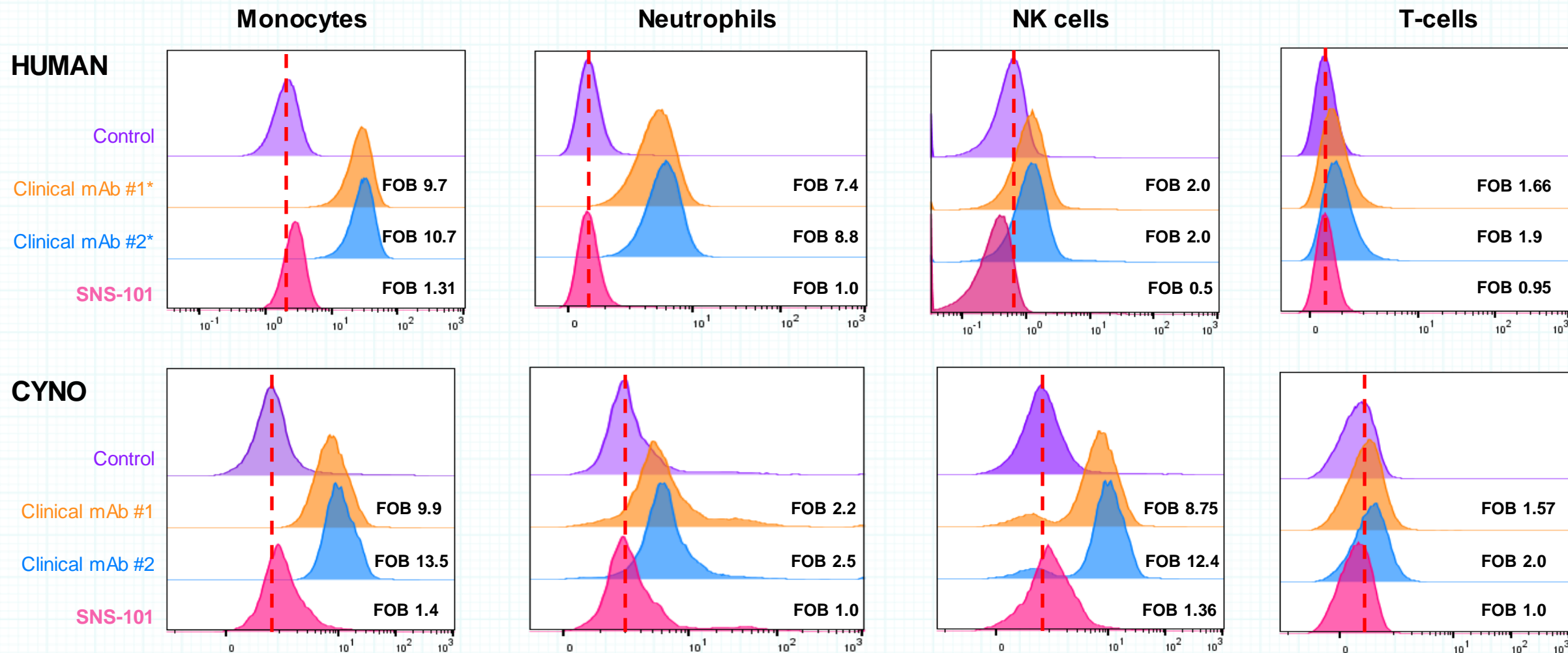
Demonstrated a long mean residence time in the blood, indicating a lack of significant target-mediated drug disposition (TMDD) and clearance in non-malignant tissues

Pharmacokinetics of Single Dose SNS-101 in Cyno monkeys



SNS-101 displayed linear elimination kinetics unlike a pH-independent anti-VISTA mAb, which demonstrated TMDD and rapid clearance

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

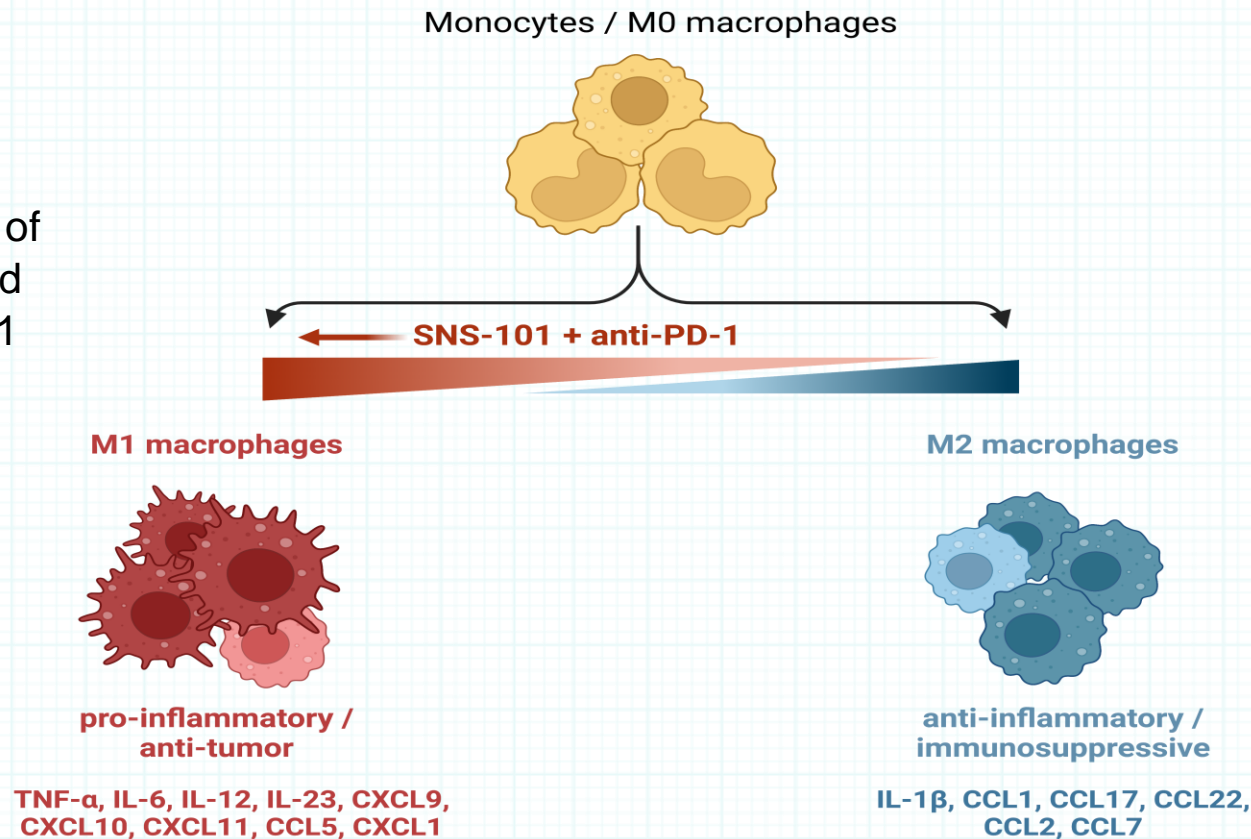


*Clinical mAb #1 & #2 are pH-independent VISTA antibodies

Targeting VISTA and PD-1 Checkpoints in the TME Promotes Anti-tumoral M1 Macrophage Polarization

SNS-101 targets suppressive signaling in the myeloid compartment, and in combination with anti-PD-1 may help shift the balance of macrophage polarization toward an anti-tumor M1 phenotype

M1 macrophages are anti-tumorigenic through secretion of pro-inflammatory cytokines and chemokines, and driving of Th1 CD4+ T cell responses



M2 macrophages are immunosuppressive; pro-tumor TAMs are a subset of M2-type cells