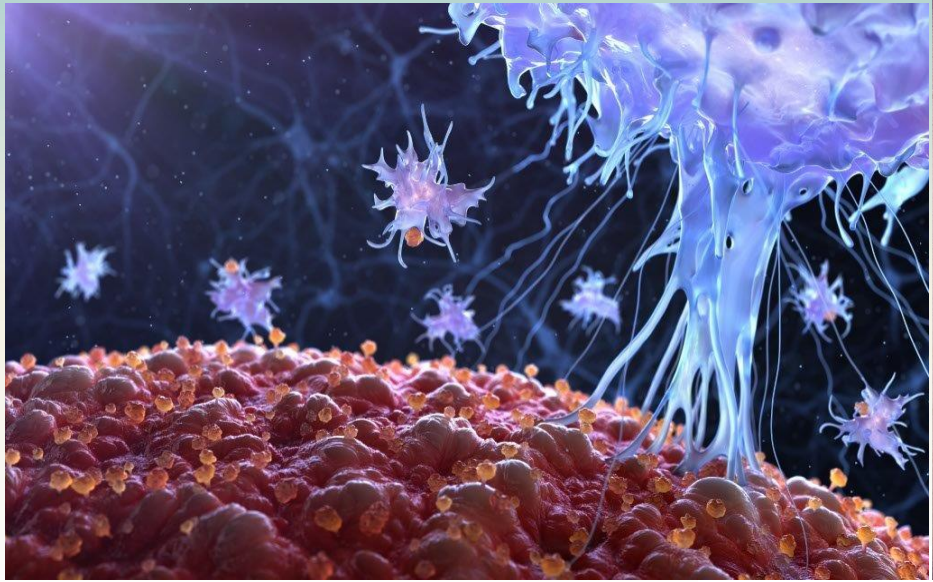
Improving Lives of Patients with Cancer



Innova therapeutics

Investment Highlights: IVT-8086 - An Exciting Novel Anticancer Therapy

Monoclonal Antibody (mAb) Platform Selectively Targeting Secreted Frizzled-Related Protein-2 (SFRP2)

Multi-faceted mechanism with DIRECT inhibition of Secreted Frizzled Related Protein 2 (SFRP2) in cancer including:

- Reduced tumor growth (primary and metastatic disease), including increased apoptosis of tumor cells
- Reduced angiogenesis, tumor cell migration and metastasis
- Rescues T Cell dysfunction including T Cell exhaustion, impacting expression of PD-1 and CD-38

➢ Lead humanized mAb selected, IVT-8086, has been shown to antagonize SFRP2 by **selectively blocking the non-canonical Wnt/Ca2+ pathway which significantly reduces tumor growth across multiple cancers**

Highly Experienced Management/Development Team with a Successful Track Record

➢ First biotech management team to obtain "Breakthrough Therapy" designation from the FDA for their therapeutic product

➢ **Same management team from previous company, Scioderm.**
  - ➢ **4th largest venture capital (VC)- backed exit in biotech/pharmaceutical space - $22M total spend with exit deal totaling appr $957M within 2.5 years of company initiation**

➢ Selected as one of the "Fierce Top 15" by FierceBiotech, considered as one of the most promising emerging companies in the biotech industry

Defined Regulatory Development Pathway and Robust IP Portfolio

➢ Progress development of IVT-8086 into Phase 1 clinical trial in patients with advanced cancer to establish safety, tolerability and optimal treatment dose as a monotherapy and in combination

➢ Investigate IVT-8086 as targeted **monotherapy** treatment for cancers with high unmet need **(sarcomas (including osteosarcoma (OS)), pancreatic and triple negative breast cancer**), and in **combination** with anti-PD-(L)1 checkpoint inhibitors

➢ **Osteosarcoma is a rare disease which was granted both orphan designation and rare pediatric disease designation from the FDA**
  - **Fast regulatory approval timeline, including opportunity to obtain a Rare Pediatric Disease priority review voucher (value range $80-350M)**

➢ Robust global patent portfolio, including composition of matter patents

Innova Therapeutics is Led by an Experienced Senior Management Team with Extensive Development Experience...

Leadership Team

Dr. Robert Ryan
President and Chief Executive Officer

Dr. Doug Testa
Head of CMC

Willistine Lenon
Executive Vice President of Clinical Operations

Steve Cole
Head of Business Development and Licensing including Japan

Dr. Ron Nardi
Executive Vice President Development

Heather Howard
Office Manager

Key Consultants

Nancy Klauber-DeMore, MD, FACS
Medical consulting Co-Founder

Cam Patterson, MD, MBA
Medical consulting Co-founder

Chris Christoffersen
Previous Co-founder and Chairman of Board, Scioderm

…and a Renowned Scientific Advisory Board (SAB)

Nancy Klauber-DeMore, MD, FACS
Co-founder, BMW Endowed Chair in Cancer Research at Medical University of South Carolina (MUSC)

Dr. DeMore is Professor of Surgery, Medical Director of the MUSC Breast Center, and Program Director of the MD/PhD Program at MUSC. Dr. DeMore completed her Surgical Oncology Fellowship at Memorial Sloan Kettering Hospital, and Cancer Research Fellowship at Harvard Medical School. She is a practicing surgical oncologist with research interest in tumor angiogenesis and immunology.

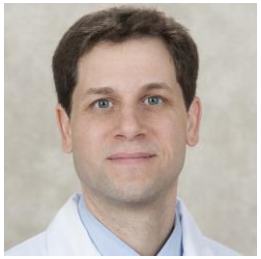
Cam Patterson, MD, MBA
Co-founder, Chancellor of the University of Arkansas for Medical Sciences (UAMS)

Prior to being named Chancellor at UAMS, Dr. Patterson was previously the Senior Vice President and Chief Operating Officer at New York Presbyterian Hospital/Weill-Cornell Medical Center in New York, from 2014-2018; and the Physician-in-Chief of the UNC Center for Heart and Vascular Care, the Chief of the Division of Cardiology, and the Director of the McAllister Heart Institute at the University of North Carolina at Chapel Hill from 2001-2014. Dr. Patterson research interests are in the areas of angiogenesis and vascular development, cardiac hypertrophy, protein quality control, and translational genomics and metabolomics.

Elizabeth Claire Dees, MD, MSc
Professor of Medicine, Division of Hematology and Oncology, UNC Hospital

Dr. Dees is a practicing medical oncologist, an active member of the UNC Breast Center, and the founding chair of the Developmental Therapeutics (Phase I trials) Working Group at UNC. She is the co-leader of the Clinical Research Program at UNC Lineberger. Dr. Dees completed her internship and residency in internal medicine at the Brigham and Women's Hospital in Boston and her medical oncology fellowship training at the Johns Hopkins Oncology Center where she worked with the Phase I trials group and the breast cancer program.

William D. Tap, MD
Chief, Sarcoma Medical Oncology Service, Memorial Sloan Kettering Cancer Center



Dr. Tap is the Chief of the Sarcoma Medical Oncology Service at Memorial Sloan Kettering Cancer Center in New York. He is a medical oncologist who specializes in the treatment of patients with soft tissue and bone sarcomas and the development of novel therapies in rare cancers and neoplasms. Bill's academic research interests are focused on understanding the genetic and molecular nuances of sarcoma with an emphasis on identifying and validating therapeutic targets, treatment biomarkers, and modeling drug resistance. Bill received his MD from Jefferson Medical College, was a resident in Internal Medicine at the Vanderbilt University Medical Center, and a fellow in Hematology and Medical Oncology at the UCLA Medical Center in Los Angeles, CA.

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Previous Team Success with Recent Company, Scioderm

COMPANY ACCOLADES	DEVELOPMENT MILESTONES	SIGNIFICANT EXIT	ROBERT RYAN
First Biotech to receive "Breakthrough Therapy Designation" from FDA	Progressed program from Pre-IND to Phase 3 in less than 2 years	$22M total spend prior to merger with Amicus for deal totaling appr $957M	Selected as N.C. CEO of the Year for Life Sciences
Selected as one of the "Fierce Top 15" by FierceBiotech, considered as one of the most promising emerging companies in the biotech industry	Agreed PIP in Europe resulting in 12 years total exclusivity	**4th largest venture capital (VC) backed exit of 2015 in biotech/pharmaceutical space, 9th largest exit across all sectors.**	Selected as CEO of the Year for Biotechs by Financial Times
	Identical development program through commercialization with FDA and EMEA, with waiving of all long-term toxicology	Multiple termsheets from Japan for in-licensing within 6 months of discussions	

>$1B Commercial Opportunity

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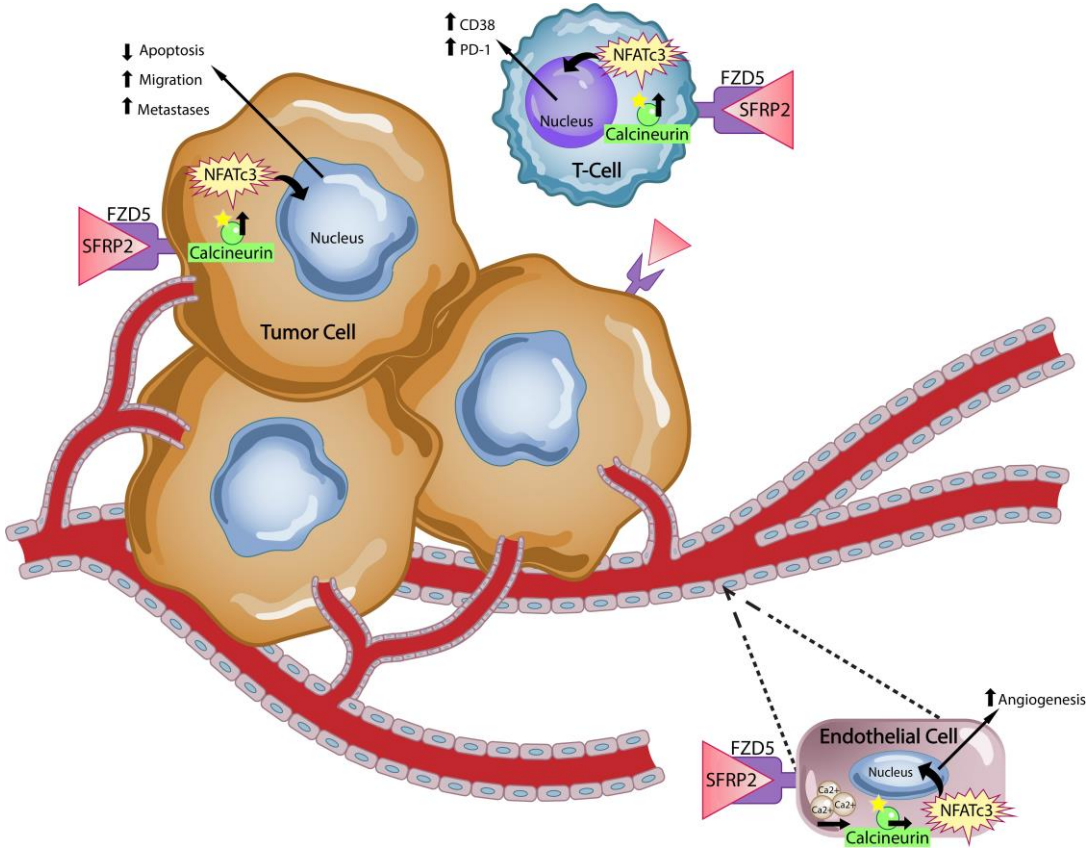
Multi-faceted Mechanism of Action

SFRP2 Regulates the *Non-Canonical* Wnt-Signaling Cascade In Tumor Cells, Endothelial Cells, and T-Cells, Effecting Tumor Growth and Metastases, Angiogenesis, and T-Cell Exhaustion



Klauber-DeMore Lab Pubs, Bhati, R. et. al., Am J Pathol, 2008

Courtwright, A. et al, Cancer Research , 2009

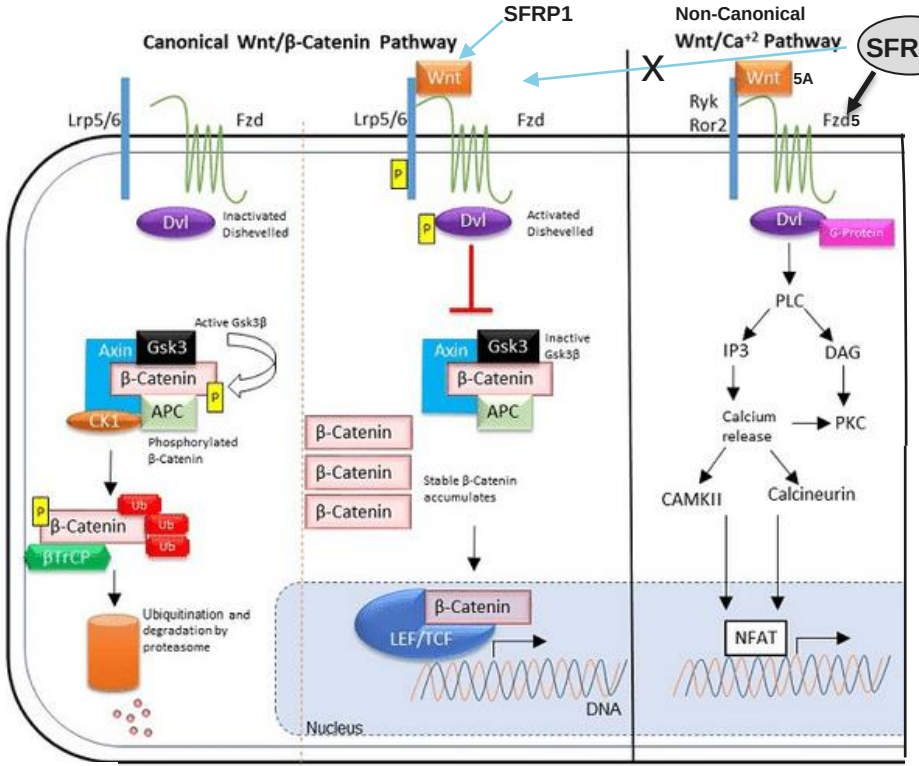
Siamakpour-Reihani, R. et al. Plos One, 2011

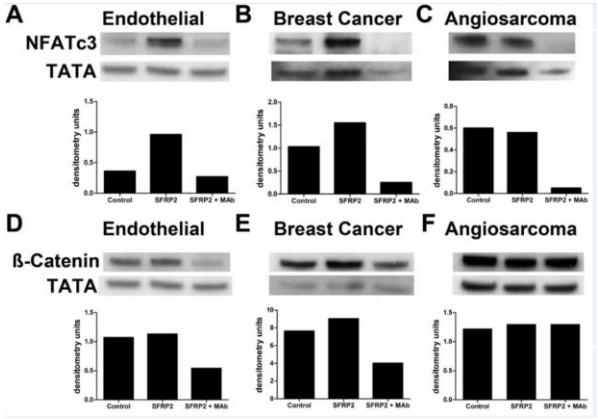
Fontenot, E. et. al., Mol Cancer Ther., 2013 Tsuruta, JK Plos One, 2014, Tsuruta, JK Plos One, 2017

Peterson, YK et. Al., Angiogenesis, 2017

SFRP2 Targeted Antagonism of Non-Canonical Wnt/Ca^{+2} Pathway

Key in Terms of Efficacy and Safety in Treating Cancer





* SFRP2 stimulates NFATc3, but does not stimulate β-catenin

* Inhibition of SFRP2 blocks NFATc3, and does not stimulate β-catenin

- SFRP2 selectively binds directly to the frizzled 5 (FZD5) receptor and activates the calcineurin/NFATc3 pathway resulting in stimulation of tumor growth
  - **Antagonism of SFRP2 causes a decrease in angiogenesis, tumor growth, migration, metastasis, T-cell exhaustion markers (PD-1, CD38), and an increase in tumor apoptosis.**
  - SFRP2 **<u>does not</u>** interact with the canonical β-catenin pathway

* Peterson YK, et al. Angiogenesis. 2017;20(4):615-28.

* Fontenot E, et al. Mol Cancer Ther. 2013 May;12(5):685-95.

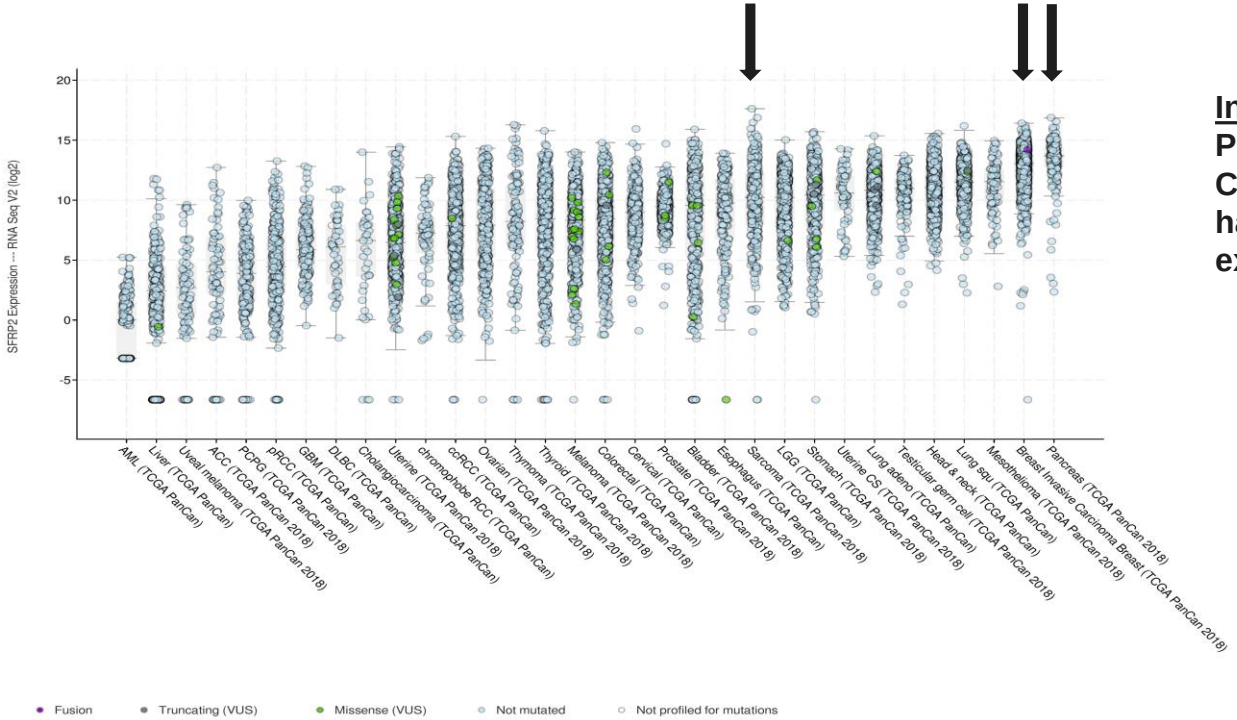
SFRP2 Validated As a Target Across Multiple Cancers

Tumor Type	Journal
Breast	**Mol. Cancer Thera, Annals of Surgical Oncology, Am J Path, PLoS ONE,** Nature Cell Biology, Dis Markers, Breast Cancer Res Treatment,
Angiosarcoma	**Mol. Cancer Thera, Annals of Surgical Oncology, Am J Path, PLoS ONE,** Nature Cell Biology, Dis Markers, Breast Cancer Res Treatment,
Osteosarcoma	PNAS, BMC Cancer, **Cancers**
Pancreatic	PLOS Compute Biol, International Journal of Obesity
Melanoma	Nature, Clin Cancer Res
Renal Cell	Mol. Cancer Thera
Malignant Glioma	Oncogene
Lung	Oncol. Rep.
Prostate	Oncogene
Colorectal	Developmental Cell
Multiple Myeloma	Blood

Bold – DeMore publications; Non-bold – other investigators

SFRP2 is Overexpressed Across Many Tumor Types

TCGA Data Evaluating SFRP2 mRNA Expression Across Human Tumors



<u>Initial cancers targeted:</u>
Pancreatic, Breast Cancer, and Sarcomas have high SFRP2 RNA expression

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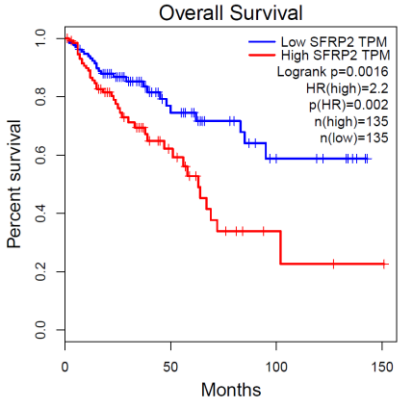
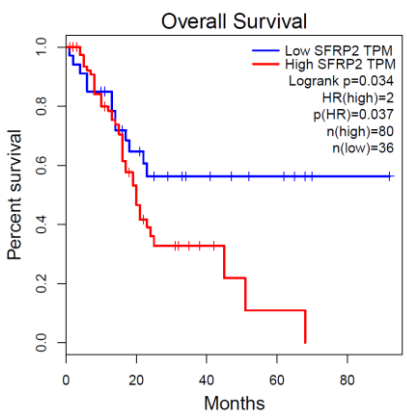
SFRP2 mRNA Expression Correlates with Survival in Cancer Patients

TCGA Data Evaluating SFRP2 mRNA Expression Across Human Tumors

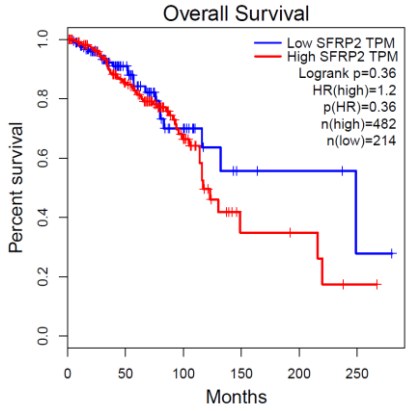
Colon Adenocarcinoma



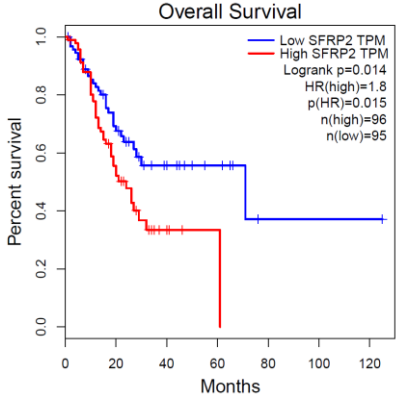
Pancreatic Adenocarcinoma



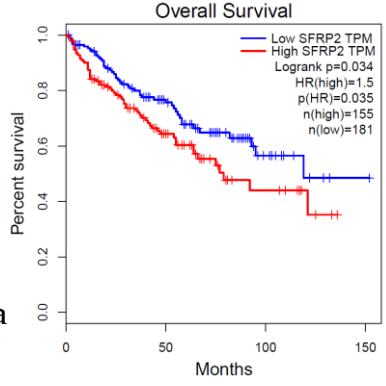
Breast Invasive Carcinoma



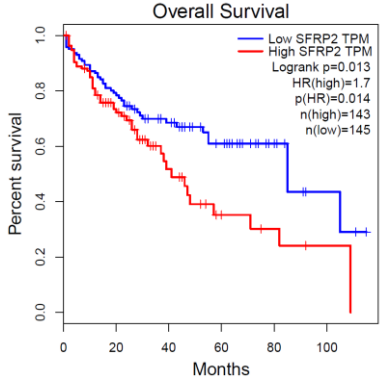
Stomach Adenocarcinoma



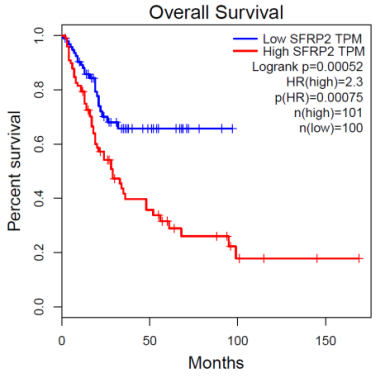
Renal Clear Cell Carcinoma



Hepatocellular Carcinoma



Bladder Urothelial Carcinoma



GEPIA is a interactive web server for analyzing the RNA sequencing expression data of 9,736 tumors and 8,587 normal samples from the TCGA and the GTEx projects.

Abbreviations
TPM – Transcripts per million
HR – Hazard Ratio

Tang, Z. et al. (2017) GEPIA: a web server for cancer and normal gene expression profiling and interactive analyses. Nucleic Acids Res, 10.1093/nar/gkx247.

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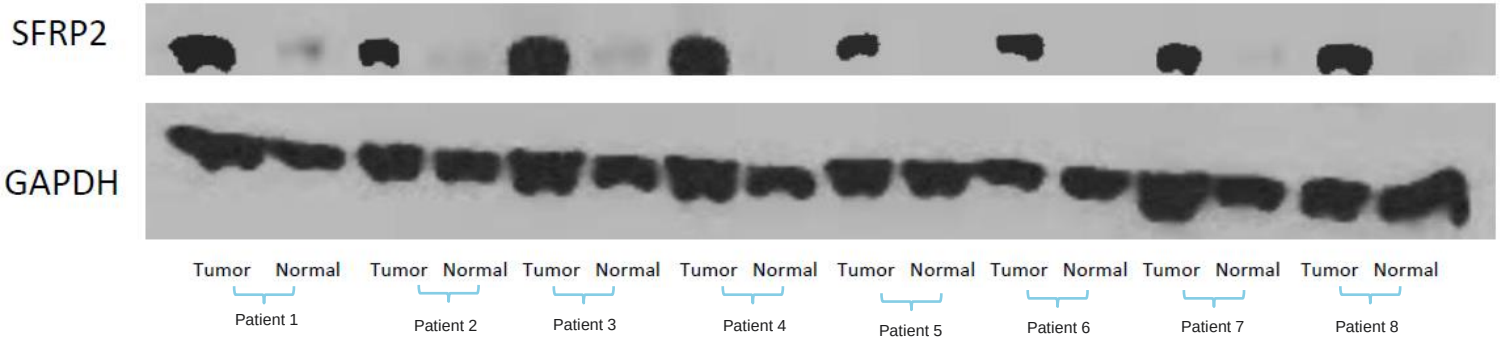
SFRP2 Protein Levels Correlate with Survival in Pancreatic Cancer Patients

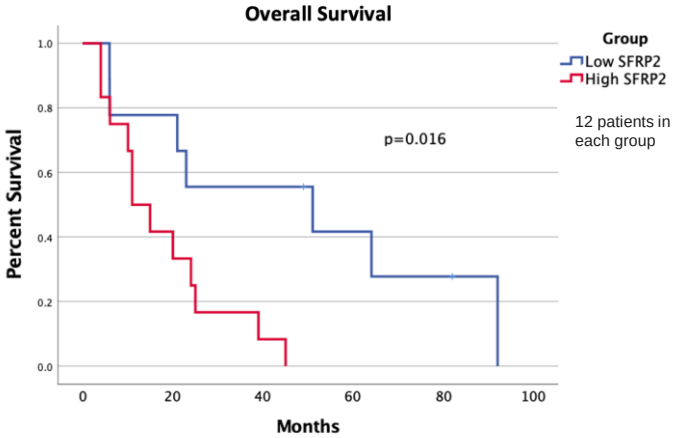
SFRP2 is an independent prognostic factor for poor prognosis

Patient Pancreatic Tumors with Matching Normal Adjacent Pancreatic Tissue



Medical University of South Carolina (MUSC) Tumor Bank samples

Stage I-III patients



Demore Unpublished data

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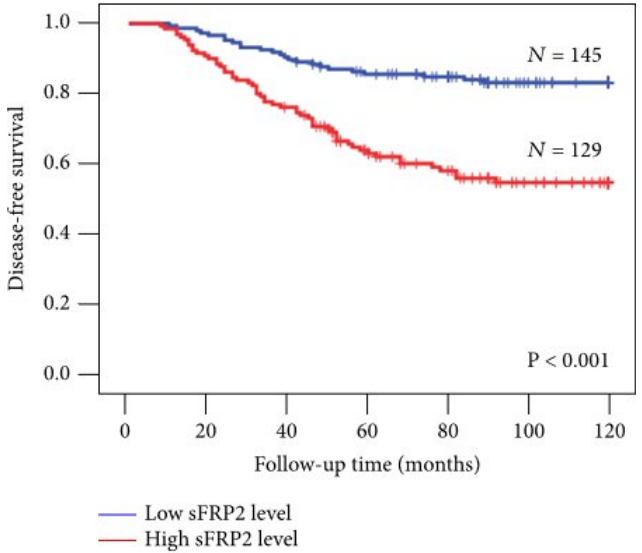
SFRP2 is Associated with Disease Progression and Aggressiveness in Breast Cancer Patients

- SFRP2 plasma concentrations compared between 274 breast cancer patients and 147 healthy controls
- SFRP2 elevated in breast cancer patients compared to normal
- SFRP2 levels are positively associated with tumor size, lymph node metastases, TNM stage, and Ki67 rate.
- SFRP2 serum levels associated with progression free survival
- Multivariate analyses shows SFRP2 independent prognostic factor for poor prognosis

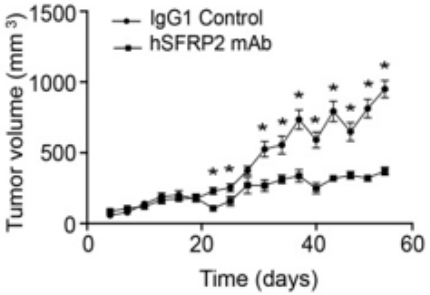


Kaplan-Meier survival curves of breast cancer patients. Progression-free survival rate of breast cancer patients with high (>58 ng/mL) and low (≤58 ng/mL) serum SFRP2 levels.

Chumei Huang, Zhuangjian Ye, Jianxin Wan, et al., "Secreted Frizzled-Related Protein 2 Is Associated with Disease Progression and Poor Prognosis in Breast Cancer," Disease Markers, vol. 2019, Article ID 6149381, 7 pages, 2019.

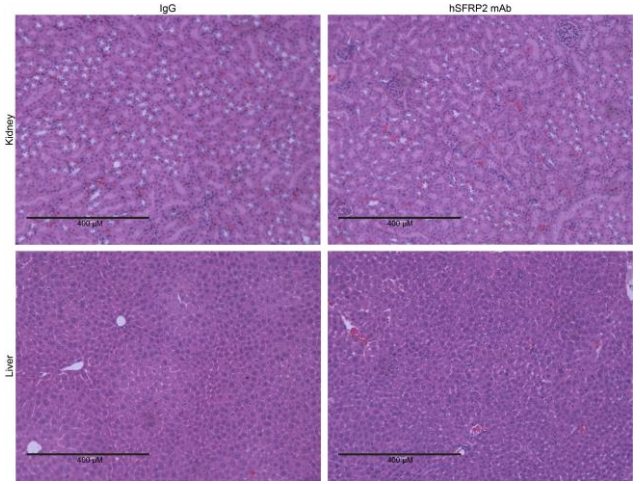
IVT-8086 Significantly Reduced Tumor Growth in A Highly Chemoresistant Hs578T Triple Negative Breast Tumor *in vivo* with No Signs of Toxicity

Metaplastic Triple Negative Breast (Hs578t)
CHEMORESISTANT



*Day 0 is counted from baseline date, which is 30 days from tumor inoculation.

- **61% reduction in tumor volume in the IVT-8086 treated mice (n=11, (*P<0.05)**
- **No adverse clinical signs or weight loss seen over 50 days of treatment.**



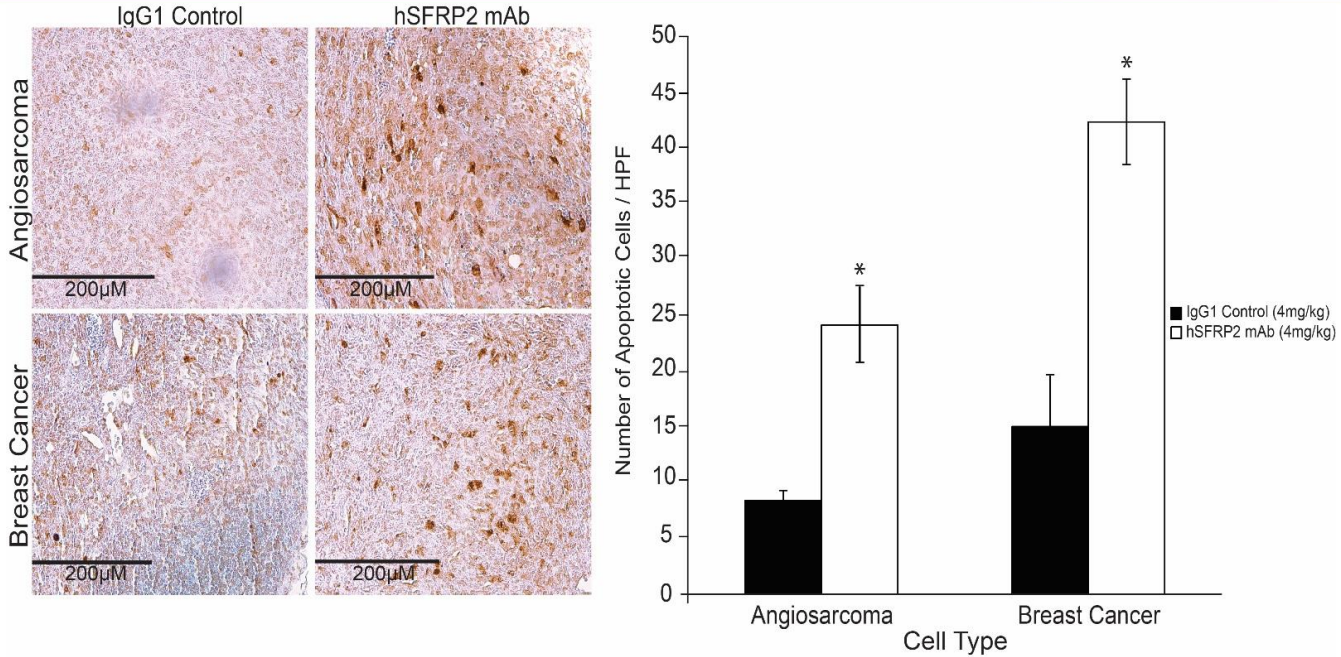
There were No Histological Changes in the Liver and Kidneys Following Multiple Dosing Administration at 4 times the Efficacious Dose (20 mg/kg) of IVT-8086 in Mice Injected with Angiosarcoma Cells

Nude mice with Hs587t breast cancer xenografts **that were established (appr 100 mm³)** were treated with hSFRP2 mAb or IgG1 control every three days beginning of Day 30.

Mice injected with SVR angiosarcoma cells were treated with hSFRP2 mAb at a dose of 20 mg/kg i.v.every three days; or IgG1 control, for 21 days. Histological evaluation of kidneys and livers from all mice at 20 mg/kg dose was conducted by a board-certified pathologist.

Garcia D, et al. Ann Surg Oncol. 2019 Dec;26(13):4782-4790.

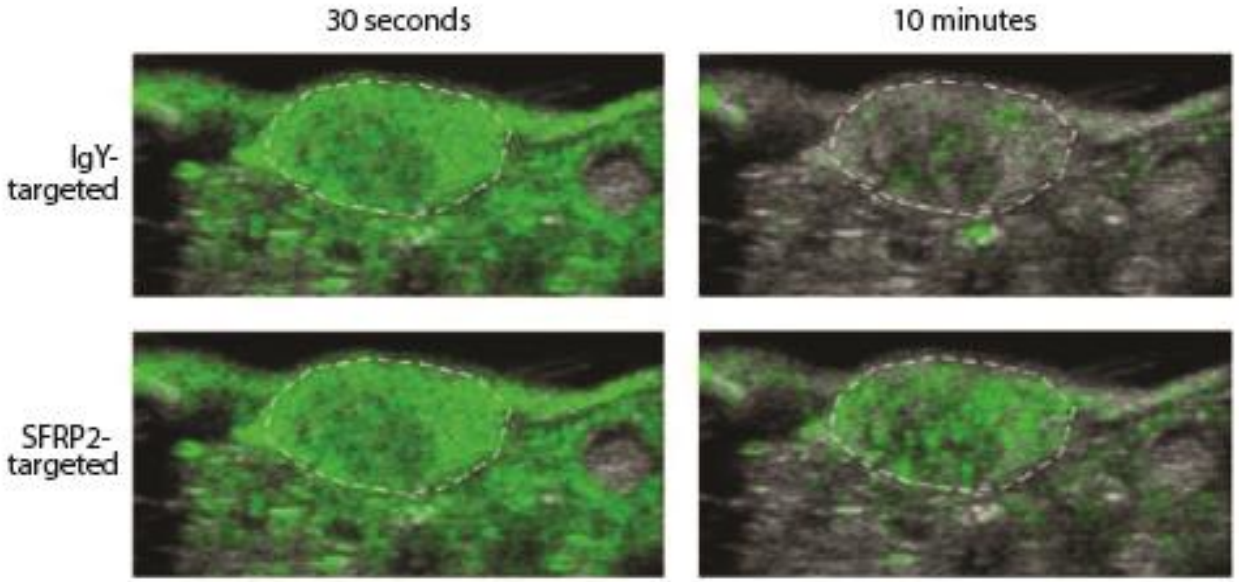
IVT-8086 Increases *in vivo* Tumor Apoptosis



IVT-8086 promotes apoptosis in tumors. (Left) Paraffin embedded SVR angiosarcoma (upper panels) and Hs578T metaplastic breast cancer (lower panels) were sectioned and processed for TUNEL staining. The number of apoptotic cells (brown) was counted in each field. A total of 10 tumors per treatment (n=10) were used for the analysis. (Right) Bar graph showing the increase in the number of apoptotic cells in tumors treated with IVT-8086 (white bars) compared to IgG1 control treated tumors (black bars). *:$p \leq 0.05$.

Garcia D, et al. Ann Surg Oncol. 2019 Dec;26(13):4782-4790.

SFRP2 Antibody Microbubble Contrast Agent Redistributes Rapidly to Tumor From Systemic Circulation



Ultrasound molecular imaging of angiosarcoma in animal receiving SFRP2-targeted and control IgY-targeted contrast was assessed after bolus injective via the tail vein.

A white dashed line outlines tumors. The contrast-specific signal (green) was superimposed over the b-mode image (grey). At 30 seconds, average video pixel intensity was similar between control and SFRP2-targeted contrast. The contrast-specific video intensity was retained in tumors at much higher levels when using the SFRP2-targeted contrast compared to the IgY-targeted contrast.

Tsuruta,JK, et al. PLoSONE 12(3):e0174281.

SFRP2 Overexpression Enhances Osteosarcoma Metastases and Correlates with Poor Survival Outcome in Patients – Clinical Validation of Target

- SFRP2 within localized human and mouse OS cells significantly increased cell migration and invasive ability *in vitro* and enhanced metastatic potential *in vivo*[1]
- Knockdown of SFRP2 within metastatic human and mouse OS cells demonstrated decreased cell migration and invasion ability *in vitro*[1]
- Strong SFRP2 expression in OS patient samples correlates with poor survival[2]
- SFRP2 overexpression suppresses normal osteoblast differentiation, promotes OS features, and facilitates angiogenesis via autocrine and paracrine mechanisms[2]

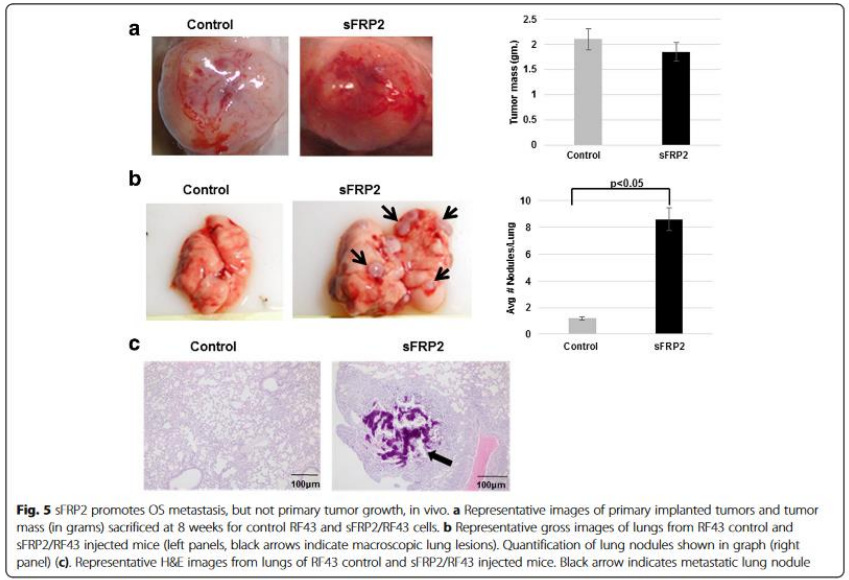
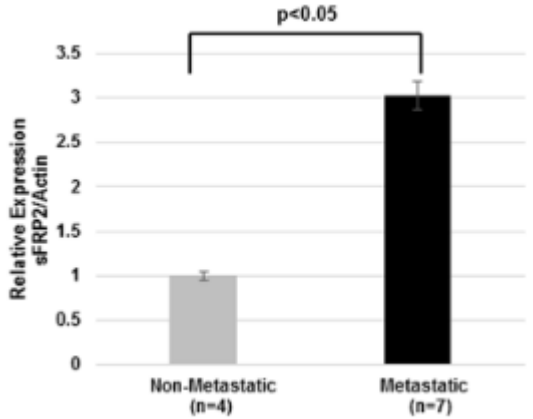


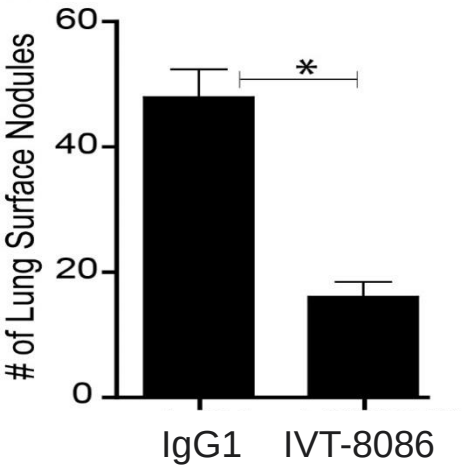
Fig. 5 sFRP2 promotes OS metastasis, but not primary tumor growth, in vivo. **a** Representative images of primary implanted tumors and tumor mass (in grams) sacrificed at 8 weeks for control RF43 and sFRP2/RF43 cells. **b** Representative gross images of lungs from RF43 control and sFRP2/RF43 injected mice (left panels, black arrows indicate macroscopic lung lesions). Quantification of lung nodules shown in graph (right panel) (**c**). Representative H&E images from lungs of RF43 control and sFRP2/RF43 injected mice. Black arrow indicates metastatic lung nodule



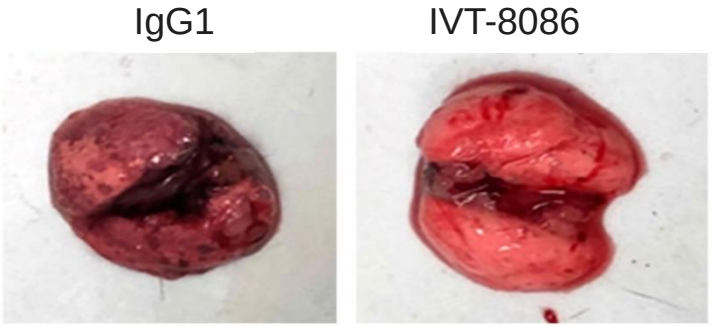
qPCR analysis comparing expression of SFRP2 in metastatic primary human osteosarcoma tumor tissue to non-metastatic tumor

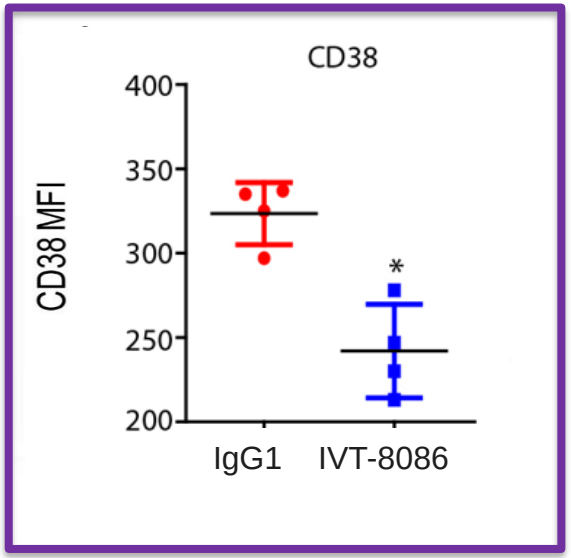
1. *Techavichit P, et al.BMC Cancer. 2016 Nov 8;16(1):869.*
2. *Kim H, et al. Proc Natl Acad Sci U S A. 2018;115(47)*

IVT-8086 Reduces Established Metastatic Osteosarcoma in Mouse GEMM Model as Monotherapy and Reduces CD38 in T-Cells



*p≤0.000, n=12

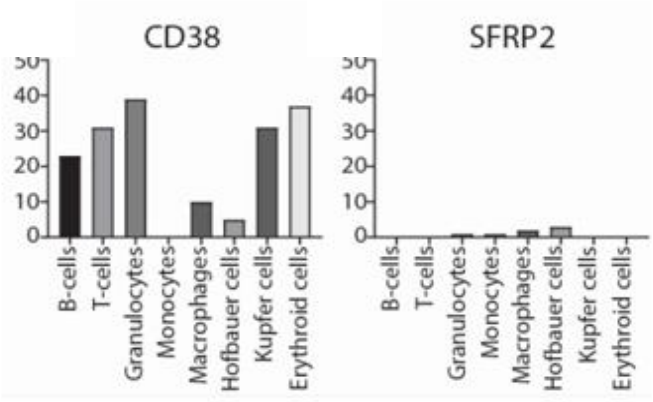




*p≤0.02

Nasarre, P. et al. Overcoming PD-1 Inhibitor Resistance with a Monoclonal Antibody to Secreted Frizzled- Related Protein 2 in Metastatic Osteosarcoma. Cancers 2021, 13, 2696.

Resistance to PD-1 Inhibitors Has Been Shown to be Associated with Increased CD38 Expression in T-Cells[1]



CD38 is ubiquitously expressed on most cells, including normal hematopoietic cells

- Darzalex is an approved CD38 mAb which binds directly to CD38 wherever its expressed
- Unacceptable toxicity in combination with PD-1 inhibitors in clinical trials, all trials terminated

SFRP2 is selectively expressed only in the tumor microenvironment and not in normal hematopoietic cells

Targeting SFRP2 should specifically inhibit CD38 only in cells that express SFRP2 restricted to the tumor and tumor microenvironment

- Would expect an inhibitor of SFRP2 to improve efficacy of PD-1 inhibitors without off target toxicity

IVT-8086 reduces CD38 and improves efficacy of PD-1 inhibitors, with no observed toxicity

1. Chatterjee S, et. al., Cell Metab 2018, Philip M, et al, Nature 2017

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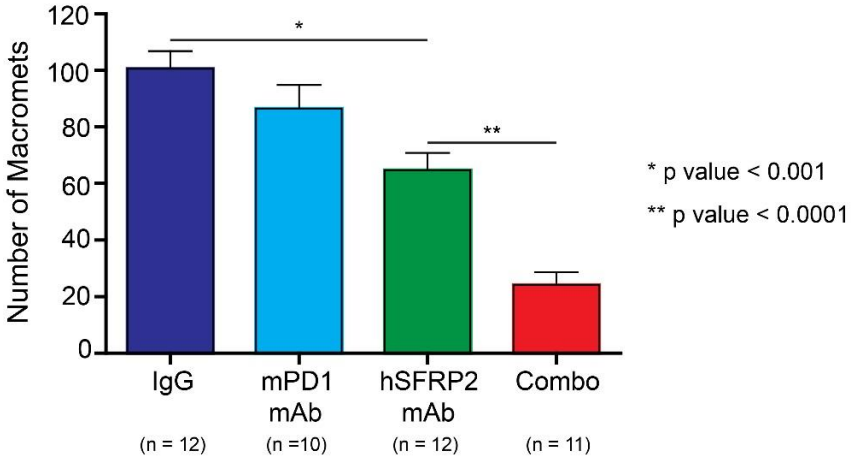
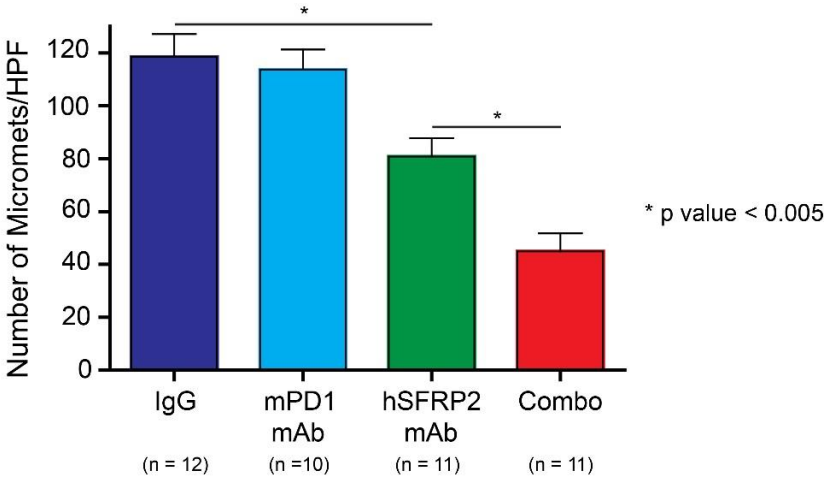
IVT-8086 in Combination with a PD-1 mAb is Synergistic at Inhibiting Metastatic Osteosarcoma in GEMM Osteosarcoma Cell Line Model (RF420)

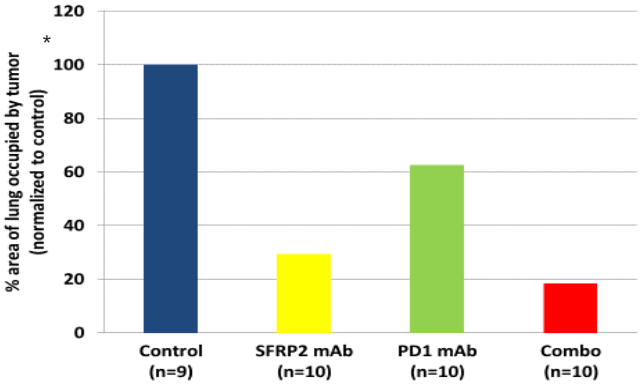


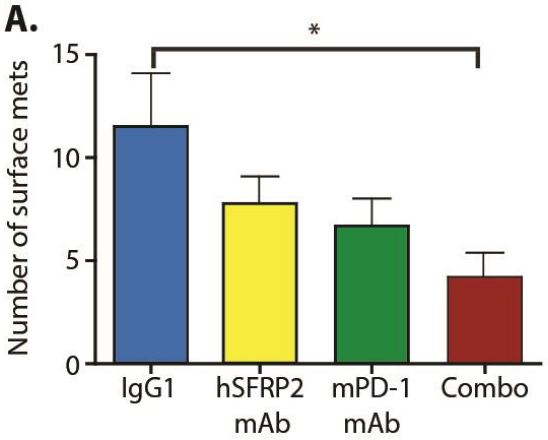


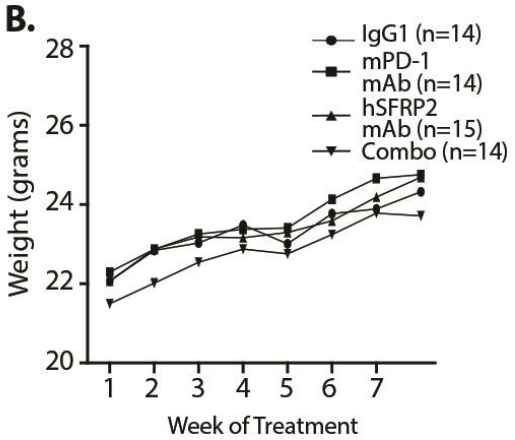
RF420 mouse osteosarcoma cells were injected in the tail vein of C57BL/6 mice. Starting on day 7 mice were treated with either IgG1 control (Xolair) 4 mg/kg iv weekly, or mouse PD-1 ab (200ug/mouse) every 3 days, or IVT-8086 4 mg/kg iv every 3 days, or the combination of both antibodies every 3 days for 21 days. .

Nasarre, P. et al. Overcoming PD-1 Inhibitor Resistance with a Monoclonal Antibody to Secreted Frizzled- Related Protein 2 in Metastatic Osteosarcoma Cancers 2021, 13, 2696.

Monotherapy and Combination Therapy Inhibits Osteosarcoma Lung Metastases in 2nd GEMM Model (RF577)







IVT-8086 treatment reduced lung metastatic tumor volume by 71%. The combination of antibodies reduced tumor volume compared to control by 82%.

IVT-8086 treatment reduced the number of lung surface mets alone and in combination with PD-1 mAb

No evidence of toxicity as noted by no change in body weights in any groups during the study

Nasarre, P. et al. Overcoming PD-1 Inhibitor Resistance with a Monoclonal Antibody to Secreted Frizzled- Related Protein 2 in Metastatic Osteosarcoma. Cancers 2021, 13, 2696.

Clinical Development Plan for IVT-8086 Initially Focused on Three Cancers, Sarcoma (including Osteosarcoma), Triple Negative Breast, and Pancreatic Cancer

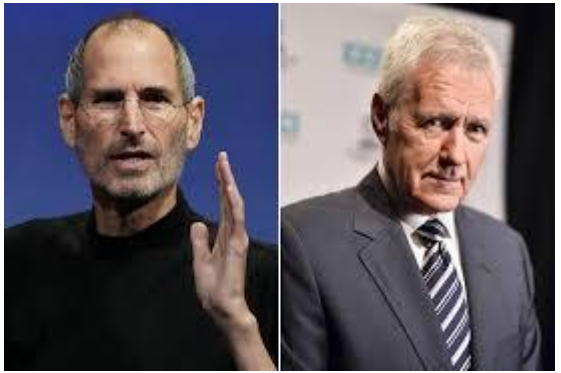
Osteosarcoma (OS) and Other Sarcomas

- Monotherapy treatment significantly reduced Lung Surface Nodules in OS ($p \leq 0.0001$)
- IVT-8086 in combination with a PD-1 mAb was synergistic at inhibiting lung metastases in multiple osteosarcoma GEMM Models ($p<0.005$)
- Significant reduction in tumor growth in angiosarcoma tumor model compared to control group.($p<0.05$)
- Strong SFRP2 expression in OS patients correlates with poor long-term survival

Triple Negative Breast Cancer (TNBC)

- SFRP2 highly expressed in all human breast cancer subtypes, including TNBC
- In vivo inhibition in tumor growth in chemo-resistant triple negative breast cancer in nude mice
- SFRP2 levels in serum levels in patients across all types of breast cancer was shown to be an independent prognostic factor for poor prognosis
- Kaplan-Meier curves showed a significant association of serum SFRP2 with progression-free survival

Pancreatic Cancer



- SFRP2 highly expressed in pancreatic cancer
- Adipocytes shown to induce epithelial-to-mesenchymal transition (EMT) and aggressiveness in models of pancreatic preneoplastic lesions by orchestrating a complex paracrine signaling of soluble modulators of the **non-canonical WNT signaling pathway,** which in turn, produces a more aggressive phenotype in models of pancreatic preneoplastic lesions.

Tremendous Commercial Opportunity as a Monotherapy and in Combination with Checkpoint Therapy (e.g. PD-1 Inhibitors)

Sarcomas (including OS)

Sarcoma is a term used to describe a family of cancers that arise in the body's connective tissues, which include fat, muscle, blood vessels, deep skin tissues, nerves, bones, and cartilage.

Sarcoma is broken down into two types: soft tissue tumors and bone tumors. There are approximately 7000 new cases of soft tissue sarcomas a year in the United States, and approximately 2500 new cases a year of bone cancers.

Osteosarcoma (OS) is a type of malignant bone cancer that mostly occurs in teenagers, young adults, and older adult population. Treatment options for this cancer are very limited, with **no new therapy approved since 1991.**

Osteosarcoma is an orphan disease and has an accelerated development pathway and would qualify for a rare pediatric voucher (value $100-300M). The market projection alone for OS is projected as $800M/yr by 2025.

Pancreatic Cancer

Pancreatic cancer is one of the most dangerous malignancies and is the fourth leading cause of cancer-related death in Europe and the United States. Pancreatic cancer is expected to be the second most common cause of death in the U.S., by 2030.

Among patients with metastatic disease, **the 5-year survival rate is only 2%, with median survival with treatment by existing therapies ranging from only approximately 5.5 to 8.5 months.**

The Global Pancreatic Cancer market accounted for $1,904.20 million in 2017 and **is expected to reach $4,728.19 million by 2026.**

Source: The "Pancreatic Cancer - Global Market Outlook (2017-2026)" report

Triple Negative Breast Cancer (TNBC)

Breast cancer is the second most common cancer among women in the United States. Research estimates that in 2018 there were 8.6 million five-year prevalent cases of breast cancer worldwide, which by 2027 are expected to increase to 9.3 million cases.

TNBC is one of the most aggressive breast cancers, and accounts for about 15-20 percent of all breast cancers.

The TNBC market will experience rapid growth over the next 10 years across the US, Japan, and five major European markets (France, Germany, Italy, Spain, and the UK).

Global Cancer Monoclonal Antibodies Market & Clinical Trial Insight 2024*

*Global Cancer Monoclonal Antibodies Market Opportunity: **US$140 Billion***

Immuno-Oncology Market, By Type [mAb (Naked, Conjugate), Cancer Vaccines, Immune Checkpoint Inhibitors (PD-1, PD-L1, CTLA-4)], By Application (Lung, Melanoma, Leukemia, Lymphoma) - Global Forecast to 2022*

*The Global Immuno-Oncology Market is **Anticipated to Cross US$100 Billion by 2022***

**Source: Research and Market Reports*

BCC Research report titled 'Checkpoint Inhibitors: Global Markets'

*It states that "according to an analysis by BCC Research, the global market for checkpoint inhibitors is currently worth $14.9 billion. It is forecast to expand at a compound annual growth rate (CAGR) of 14.4% to reach $29.3 billion in 2023.***

** Source: https://drug-dev.com/checkpoint-inhibitors-novel-targets-global-markets/*

Innova therapeutics

IVT-8086 - An Exciting Novel Anticancer Therapy Targeting Secreted Frizzled Related Protein 2 (SFRP2)

- SFRP2 is a novel therapeutic target for multiple cancers affecting both primary tumor and metastatic disease
  - Multiple tumor types secrete SFRP2 in tumor cells, endothelial cells, and activated T-cells.

- Lead mAb, IVT-8086, has been shown to **directly antagonize SFRP2 resulting in selectively blocking the non-canonical Wnt/Ca2+ pathway** which significantly reduces tumor growth across multiple cancer

- Multi-faceted mechanism with inhibition of SFRP2 in cancer including:
  - Reduced tumor growth (primary and metastatic disease), including increased apoptosis of tumor cells
  - Rescues T Cell dysfunction including T Cell exhaustion, impacting expression of PD-1 and CD-38
  - Reduced angiogenesis, reduced migration and metastasis

- IVT-8086 monotherapy and combination therapy (with PD-1 mAb) has demonstrated significant efficacy (with no adverse safety effects) in multiple animal models implanted with human xenografts:
  - Results comparable in Human xenograft (immunodeficient) and Mouse syngeneic (immunocompetent) models both in primary tumor and metastatic disease

- SFRP2 has been validated as an important molecular target in human cancers which has been shown to correlate with patient outcome including overall survival across multiple cancers:
  - SFRP2 levels in plasma across all types of breast cancer were positively associated as an independent prognostic factor for tumor size, lymph node metastases, and poor prognosis including progression-free survival.
  - High expression levels of SFRP2 in osteosarcoma patients correlated with poor survival
  - SFRP2 is a secreted protein that can be measured in the blood of cancer patients
    - Development of companion diagnostic as a potential prognostic and predictive biomarker

- Clearly defined regulatory path
  - Broad therapeutic opportunities across multiple solid and hematological tumors including **fast regulatory approval timeline for Osteosarcoma, which was granted both Orphan designation and Rare Pediatric Disease (RPD) designation by the FDA with opportunity to obtain a Priority Review voucher (value range $80-350M)**