



Corporate Presentation

March 2021



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Experienced Executive Team

John Valliant, PhD
CEO



Eric Burak, PhD
CSO



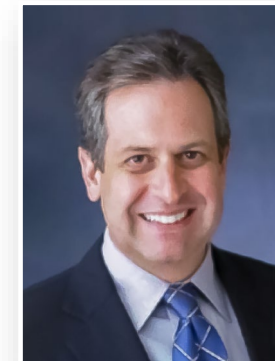
John Crowley, CPA
CFO



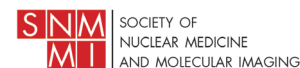
Cara Ferreira, PhD
Chief of Staff



James O'Leary, MD
CMO



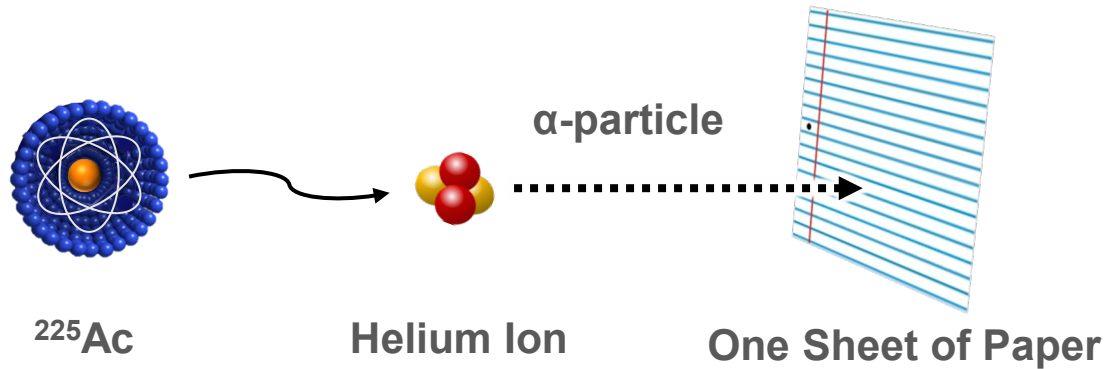
Maria Stahl
CLO



- **Precision medicine** approach to radiation therapy, a validated tumor-killing technology
- Lead clinical program targeting IGF-1R, showing uptake in **multiple tumor types**
 - **Recently advanced from single- to multi-dosing phase 1 study**
- **Proprietary *Fast-Clear*[™]** linker technology facilitates faster clearance of non-tumor localized drug, **improving the therapeutic window**
- **Platform technology** can be used with a range of different antibodies and other targeting molecules to enable **pipeline expansion**
 - **Recently signed long-term collaboration with AstraZeneca to develop novel TATs and combination therapies**
- Strong internal **R&D and manufacturing expertise and capabilities**, a barrier to entry into the radiopharmaceutical space that has recently experienced significant M&A and financing activity

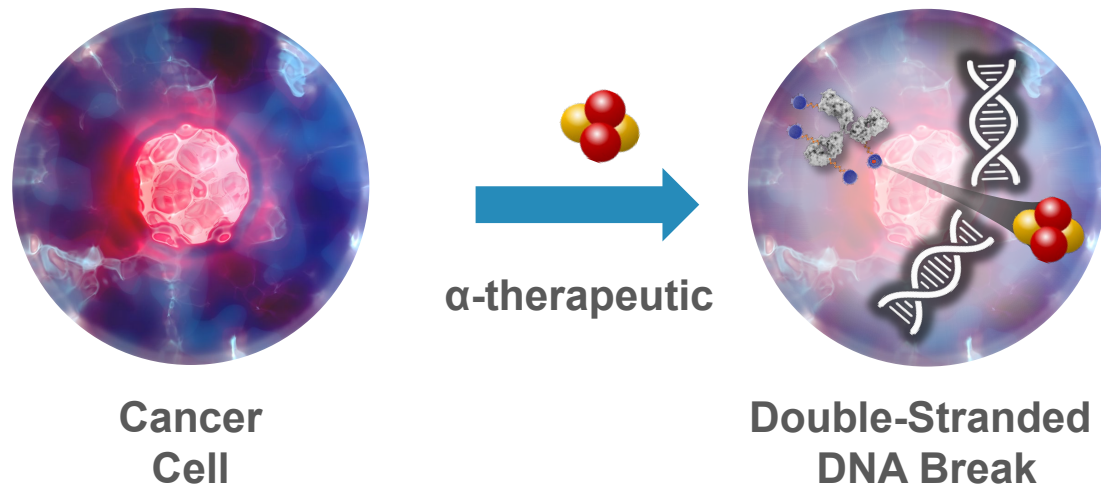


What Are Alpha Emitters?



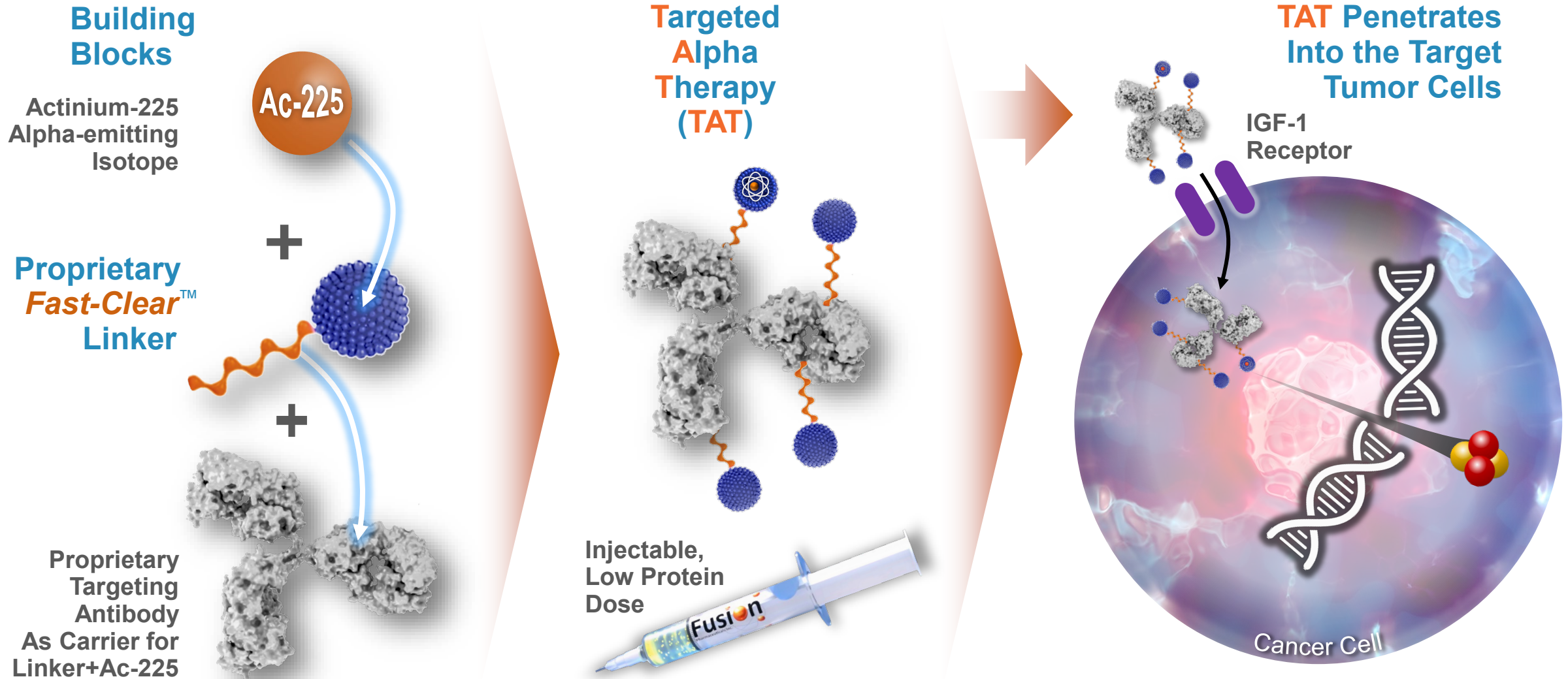
Properties:

- "Large" and energetic
- Travels a short distance (50-100 μm)
- Easy to shield (paper)

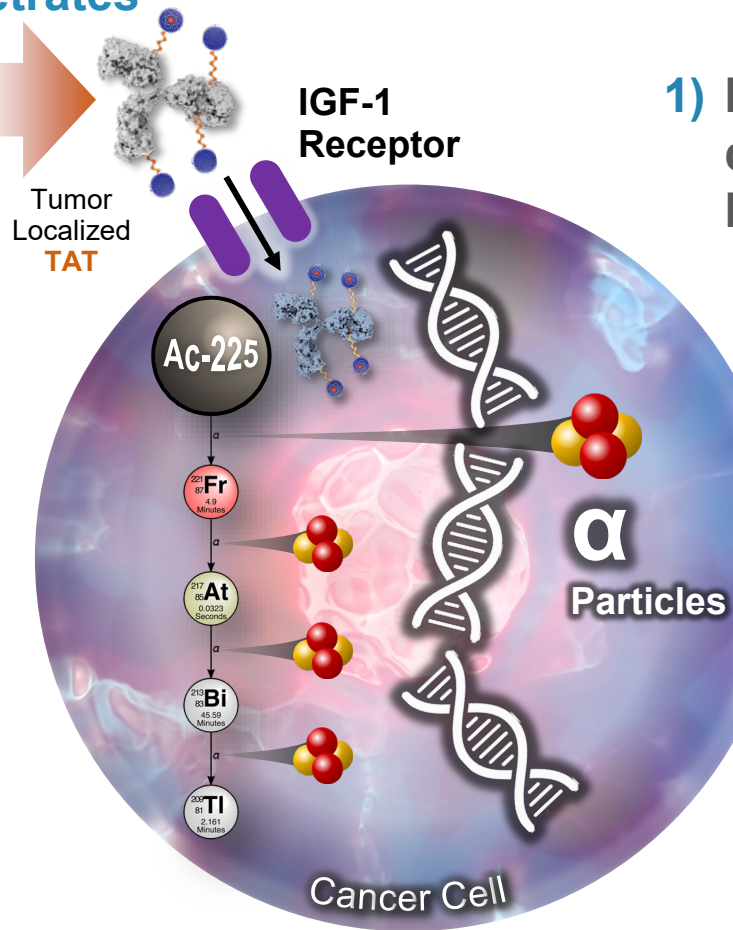


Advantages for Cancer Treatment:

- Highly localized massive cell damage
 - No resistance mechanism known to multiple double-stranded DNA breaks
- Comparatively low doses required for cell kill
- Administered intravenously (out-patient)



**TAT Penetrates
Into the
Target
Tumor
Cells**



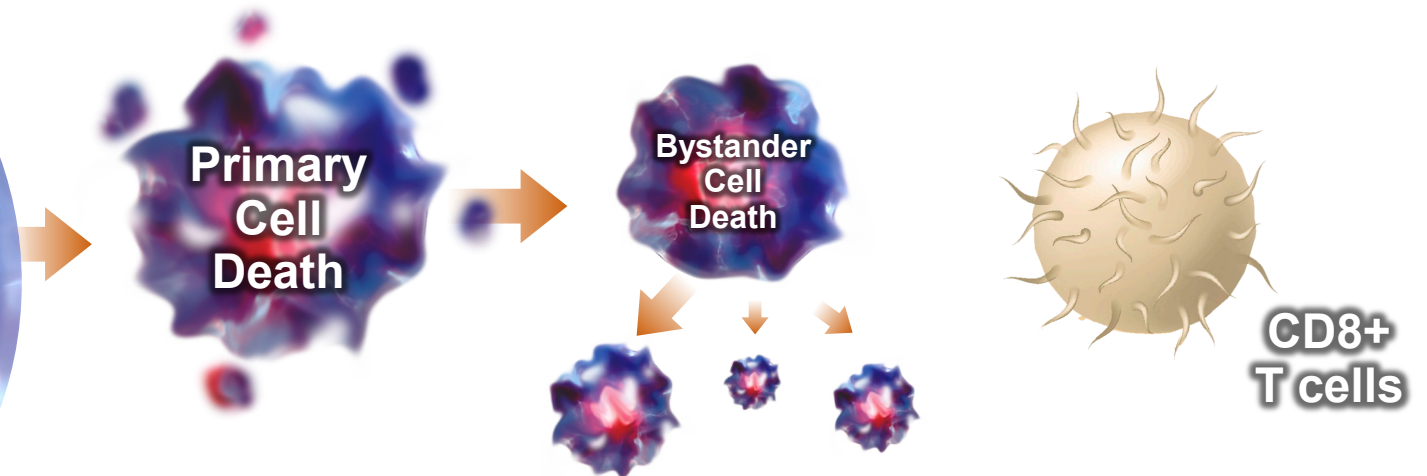
Ac-225
decay
yields
4 alpha
particles

Multiple Mechanisms of Action of a TAT

1) Multiple lethal
double-stranded
DNA breaks

2) Bystander
effect

3) Potential
vaccine effect



Fusion's research into the underlying biology of alpha emitting radiopharmaceuticals led to the understanding of our TATs' multiple mechanisms of action

TAT platform's advantage over ADCs

TAT has higher potential efficacy and better tolerability

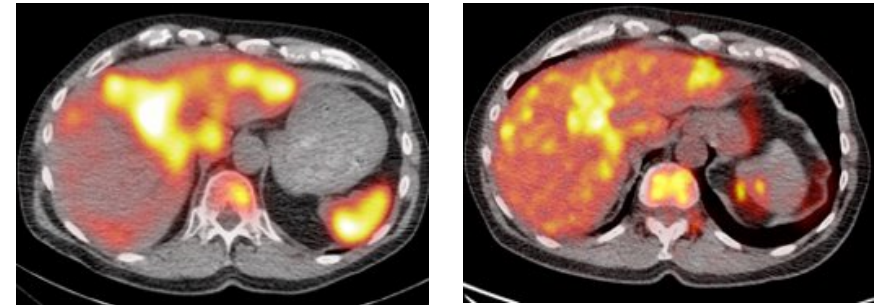
Key Property	TAT	ADC
Bystander effect providing efficacy in heterogeneous tumors	✓	✓*
Ability to incite an immune response against tumor cells	✓	✓*
Effective in tumors with low target receptor expression	✓	✗
Low toxin concentrations and recycling resulting in better tolerability	✓	✗
Effective against both dividing and non-dividing cells	✓	✓*
Built in biomarker for patient selection	✓	✗
Easy administration	✓	✓

* May have property (e.g., dependent on cytotoxin, linker)

- An underperforming ADC may convert to a **highly effective TAT but not vice versa**

TAT development de-risked by use of theranostic imaging for earlier PoC

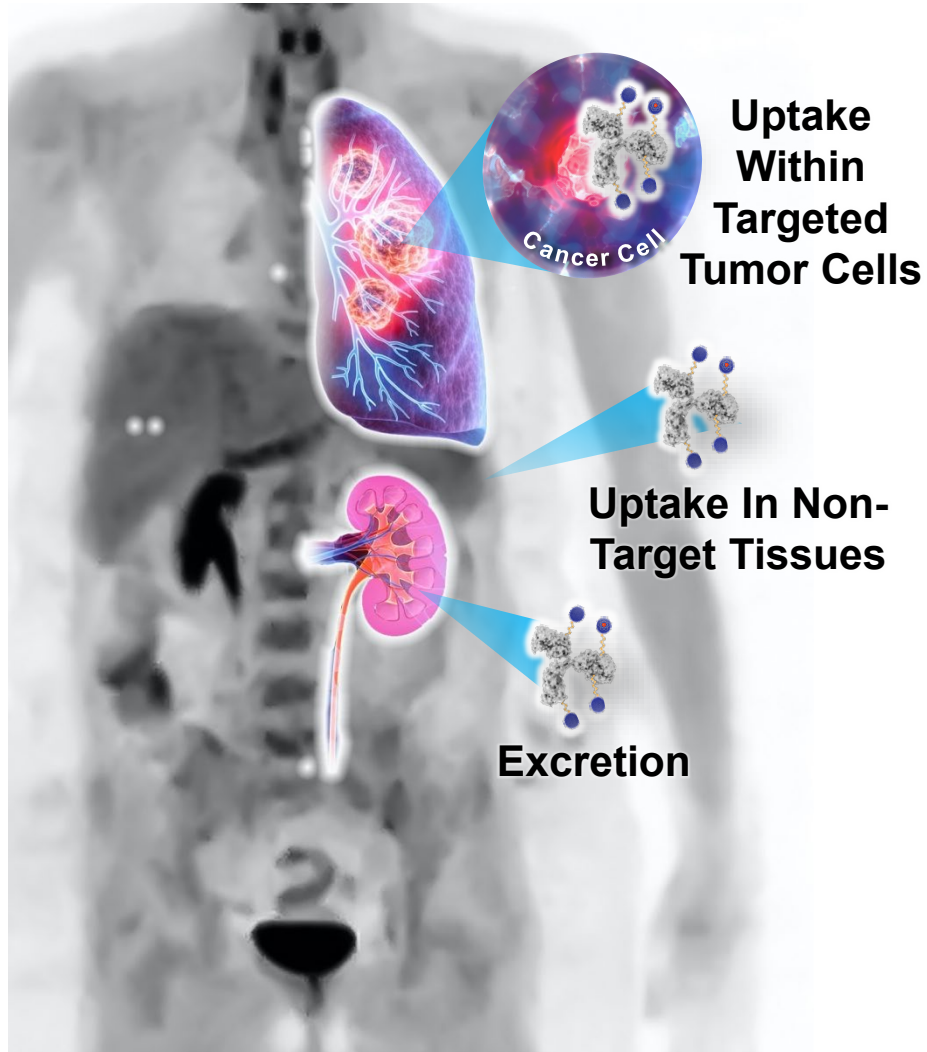
TAT uptake (left) correlates to lesion uptake in FDG PET (right) - confirms target uptake and identifies off-target tolerability issues



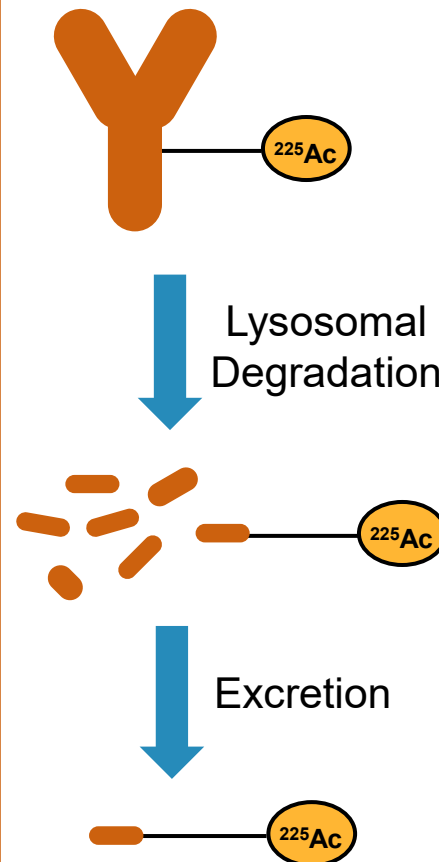
Proven applicability with a wider array of targeting modalities

- Delivery of medical isotopes has been **clinically demonstrated with almost all potential vector types** (e.g., antibodies, peptides, small molecules)
- Delivery of cytotoxic payloads has **limited clinical validation outside of antibodies**

*Fast-Clear*TM Enhances TAT Distribution Ratio

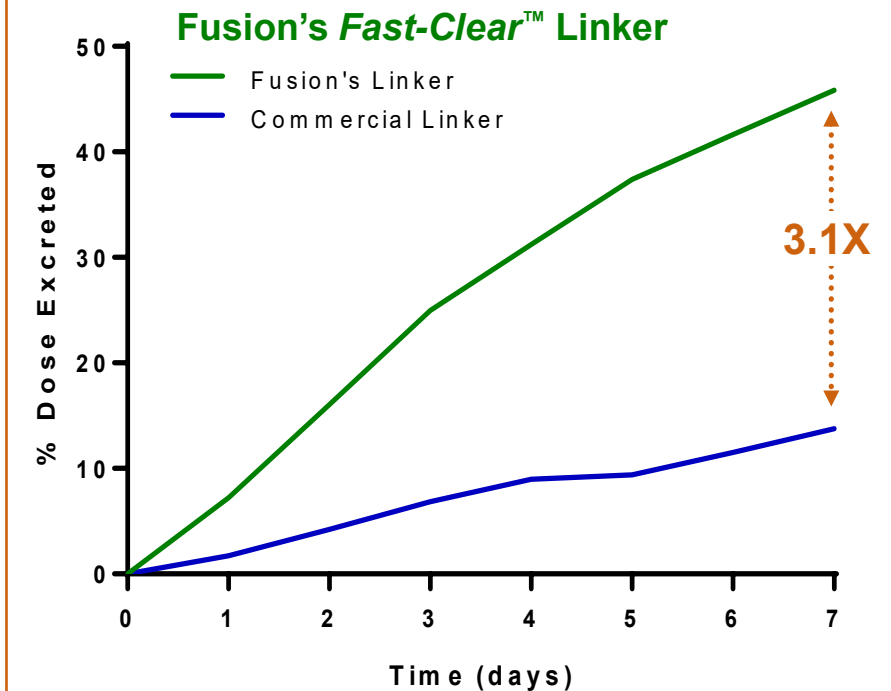


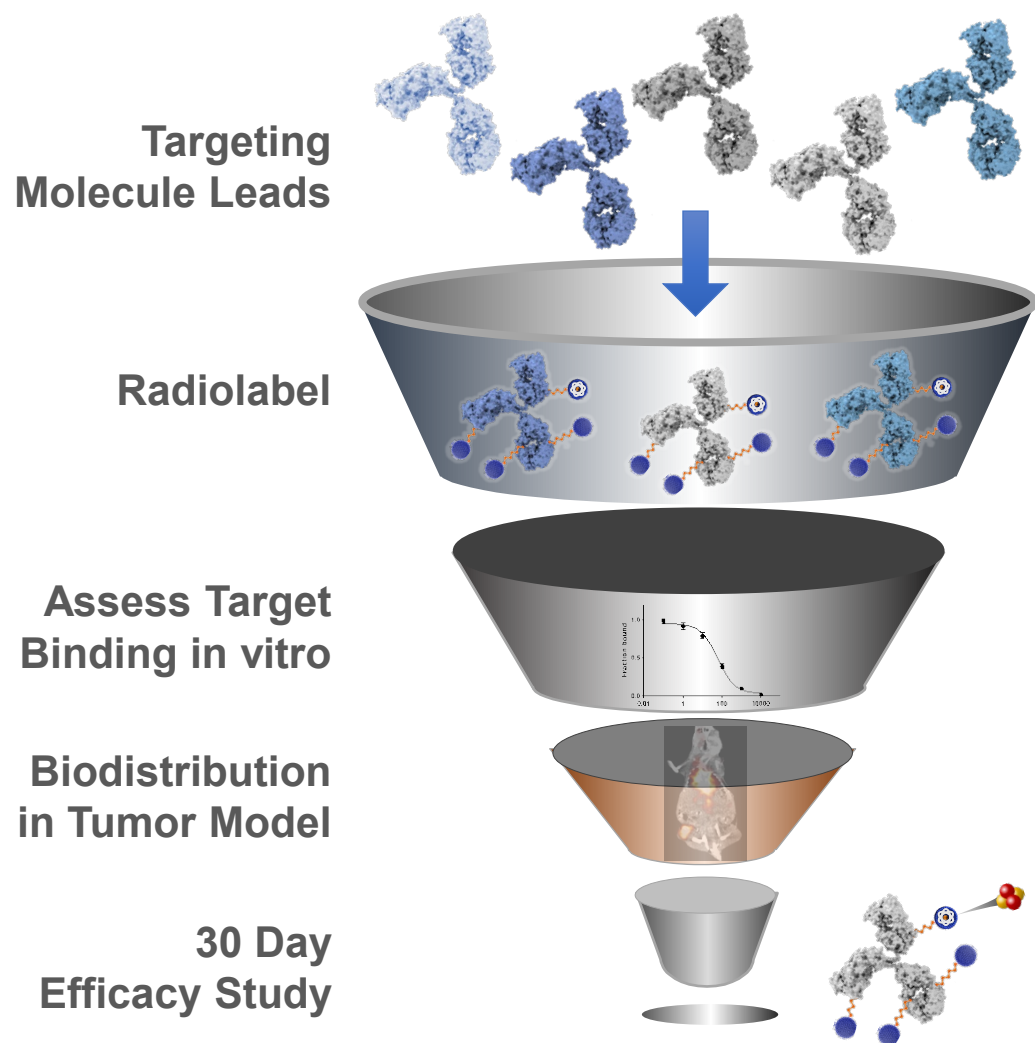
Excretion Process



*Fast-Clear*TM Linker Enhances Clearance of Non-Tumor Localized TAT in Mice

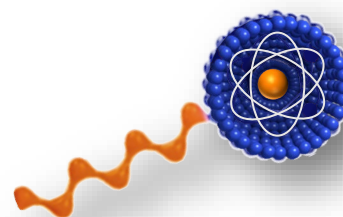
- ✓ Same tumor uptake and efficacy
- ✓ Faster and improved excretion



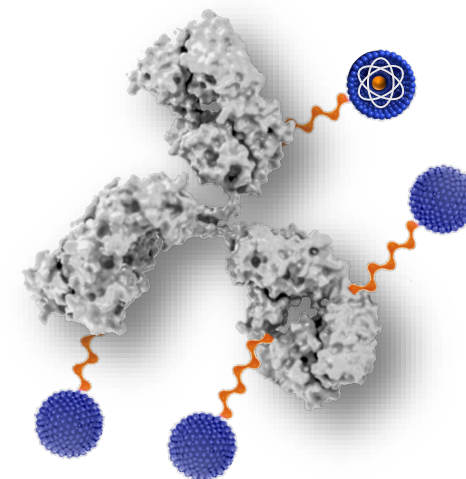


Adding Fusion's Linker Technology

promotes rapid excretion and improved therapeutic window

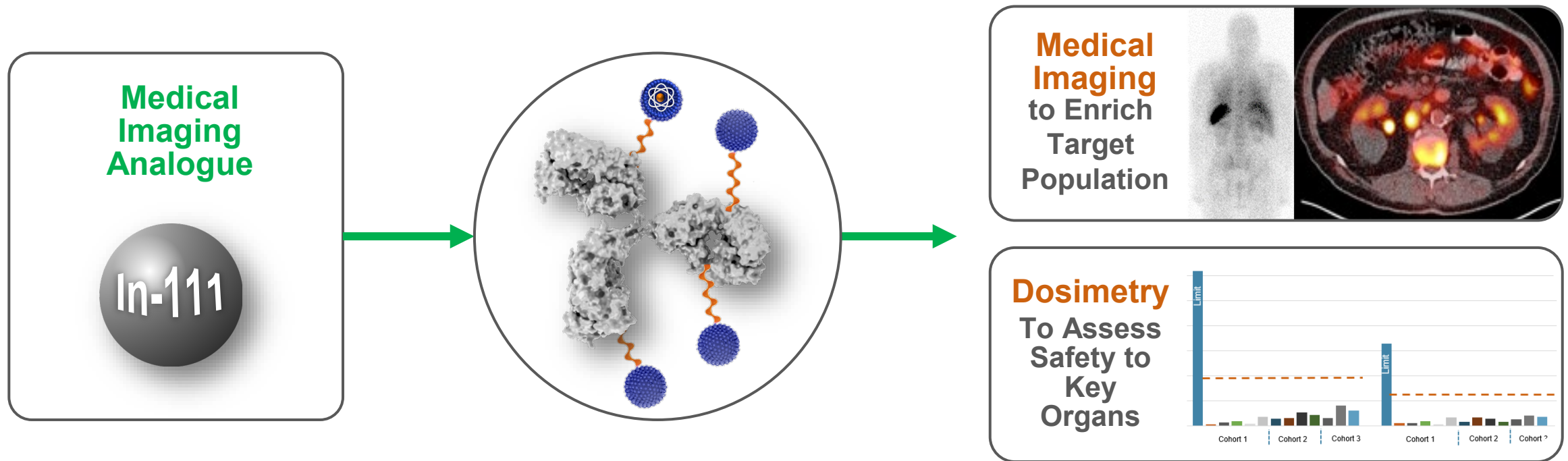


Creates Potent TAT



Use of Imaging Diagnostics to Enrich Target Populations

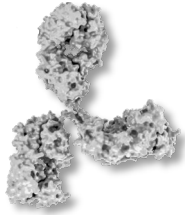
- Imaging analogues of TATs utilize the same targeting molecule and linker
- Replace Ac-225 with imaging isotope, In-111



Established Manufacturing Process and Supply Chain

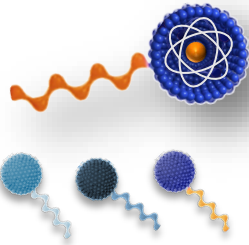
Core Competitive Advantage

Antibody Production



Multiple
Sources

Library of Fast-Clear™ Linkers



Isotope Production



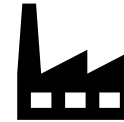
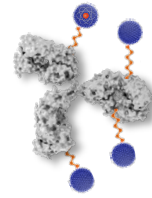
U.S. DEPARTMENT OF
ENERGY



TRIUMF

Radiopharmaceutical Manufacturing

World-class GMP manufacturing



CardinalHealth™

Multiple
Sources

Hospitals and Clinics



Standing goals as lead program
advances and pipeline expands:

- Continue strengthening supply chain advantage
- Scale manufacturing

Fusion's Platform and Capabilities Lead to Multiple Development Opportunities

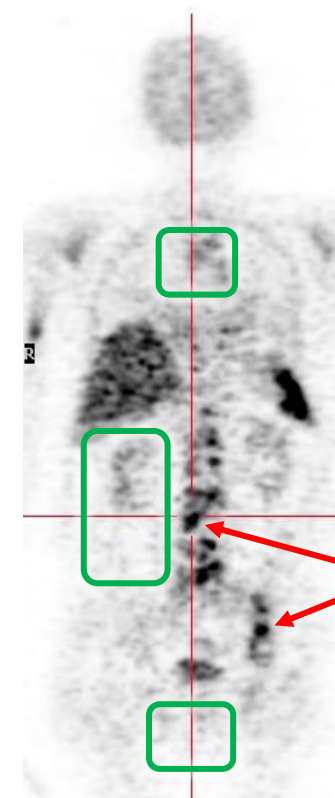


Fusion Programs		Early Discovery	Radiopharma Optimization	Preclinical Development	Phase 1	Phase 2	Phase 3
FPI-1434		Solid Tumors Expressing IGF-1R					
FPI-1966		Head & Neck and Bladder Cancers Expressing FGFR3					
FPI-2059 (IPN-1087)		Solid Tumors Expressing NTSR1					
FPI-1434 Combination		Solid Tumors Expressing IGF-1R					
Early Pipeline Targets not disclosed		Solid Tumor					
		Solid Tumor					
		Solid Tumor					
Partnered Programs		Early Discovery	Radiopharma Optimization	Preclinical Development	Phase 1	Phase 2	Phase 3
AZ Novel TATs (Up to 3) Targets not disclosed							
AZ Combinations (Up to 5) Targets not disclosed							

FPI-1434 – Fusion’s Lead Program: IGF-1R Targeted Alpha Therapeutic Monotherapy

- **IGF-1R: Ideal alpha therapeutic delivery mechanism**
 - Over-expressed on the surface of cancer cells
 - Low expression on surface of normal tissue
 - Rapidly internalizing receptor to concentrate alpha-particles inside tumor cells
- **MOA: Alpha particle-based cell kill – NOT based on blocking the IGF-1R pathway**
 - IGF-1R is used only to identify and deliver the alpha emitting payload to the tumor
- **Strategy leverages prior investments – toxicology package and antibody manufacturing**
- **Imaging demonstrates uptake in tumors**
- **Fusion converted an IGF-1R antibody with poor clinical efficacy into a therapeutic candidate in less than 1 year**
- **Currently in a dose escalation Phase 1 clinical trial**

Fusion previously showed antibodies can selectively target tumors expressing IGF-1R



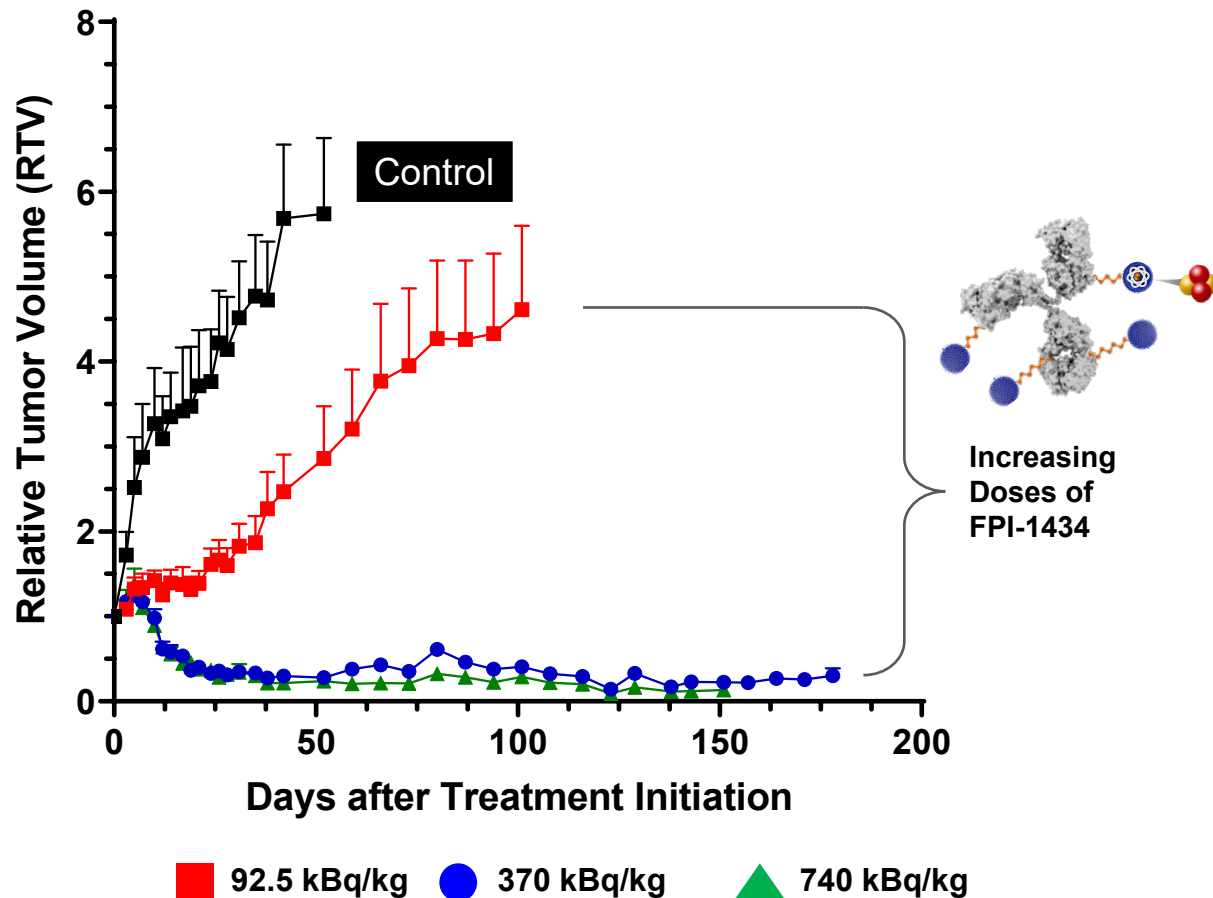
Imaging shows targeted uptake in known metastases with lack of uptake in normal tissue with IGF-1R expression: heart, GI tract, lung, testes

Bone Metastases in Spine & Hip

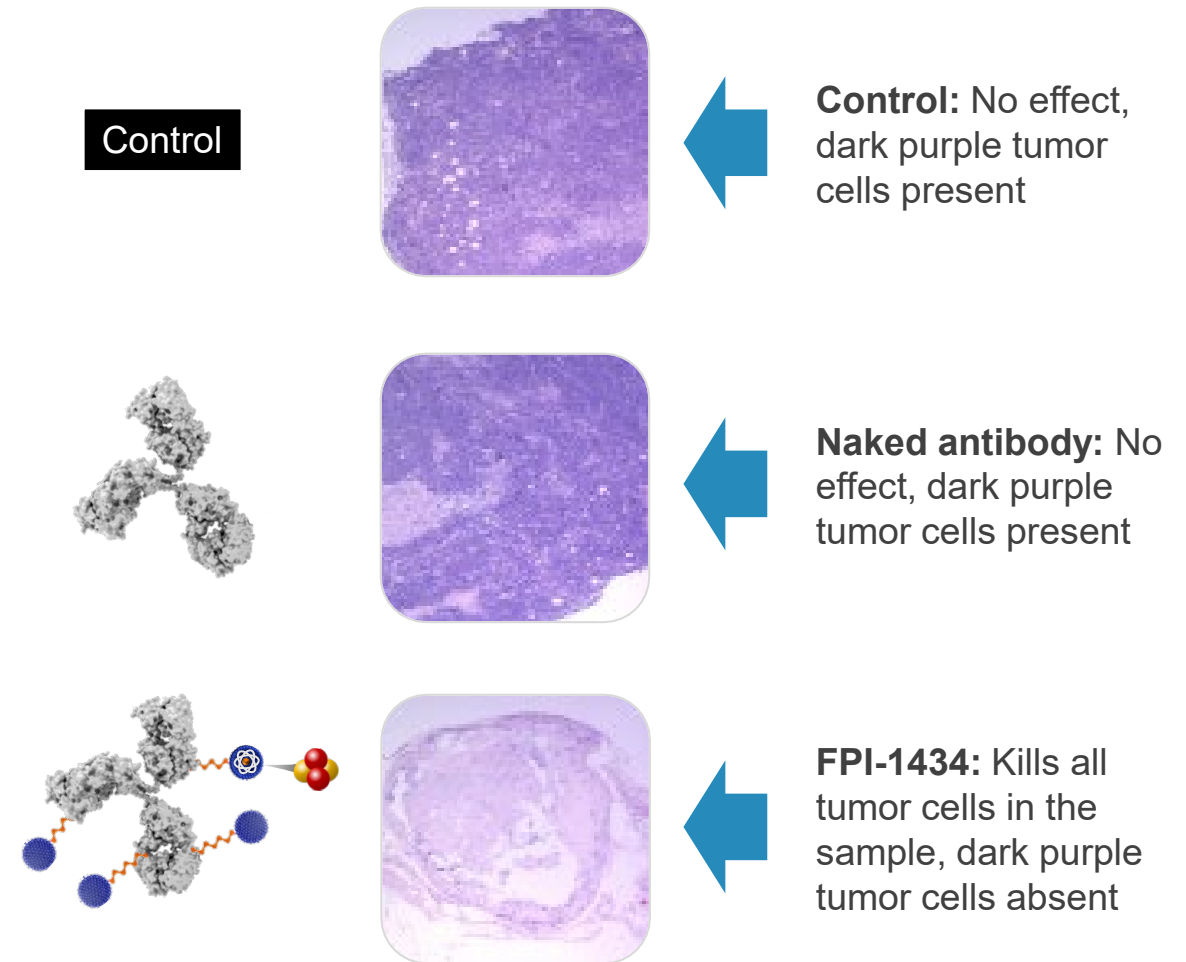
Normal Tissues Expressing IGF-1R

PET

Single Dose Eradicated Tumors in Preclinical Model (CRC xenograft mouse model)

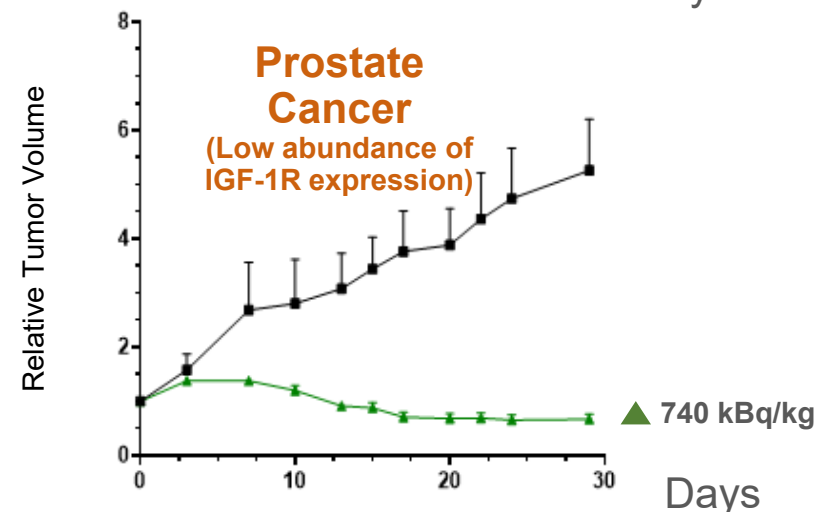
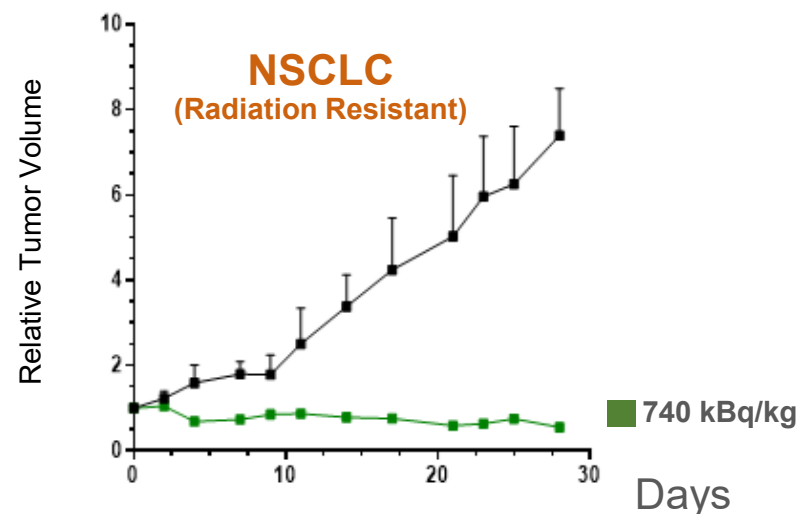


Histological Eradication of Tumors by Pathology (H&E tumor cell staining following treatment)



Compelling Anti-Tumor Activity Across Multiple Tumor Models and Tumor Sizes

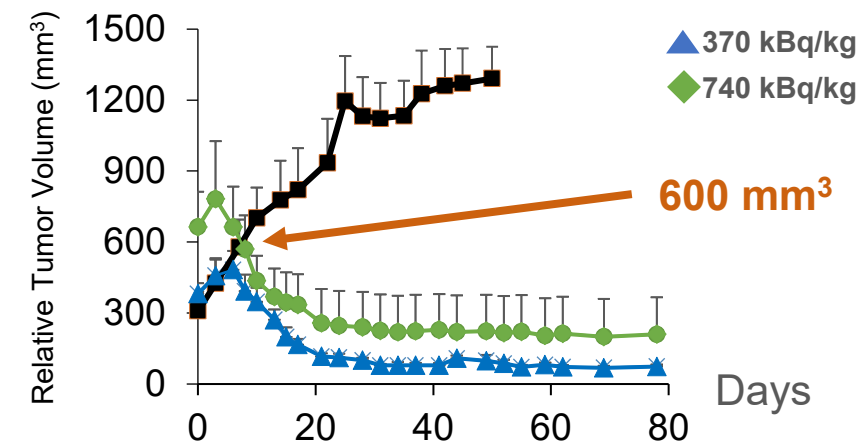
Different Tumor Types



Data Show:

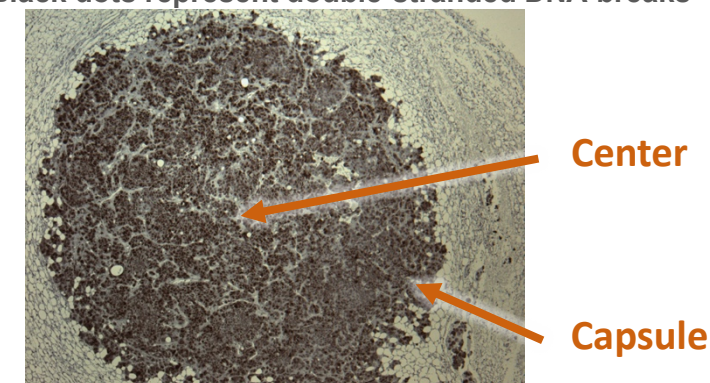
- Ability to **kill tumors of various types with a single dose**
- Ability to **kill both large and small tumors**
- Ability to **penetrate tumor with alphas using the right targeting agent**

Large Tumors



Depth of Tumor Penetration

Black dots represent double-stranded DNA breaks



96 h

Overview

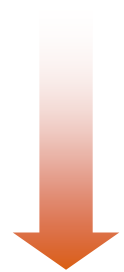
- FPI-1175 (naked IGF-1R antibody) – single and multiple dose studies
- Dosimetry study with imaging form of FPI-1434
 - Assesses radiation organ exposure to normal tissue
- Dose range-finding study with FPI-1434
- GLP late radiation toxicity study with FPI-1434 (IND-enabling study)

Findings

- The dose limiting toxicity is myelosuppression, which is reversible
- No evidence of toxicity to kidney, bladder, intestines, or lung
 - FDA approved FPI-1434 IND without the need to give Spironolactone to protect against potential kidney toxicity

FPI-1434 – Overview of Clinical Trial and Timelines

Phase 1 Single-Dose



- ✓ Single-dose cohorts complete
- ✓ FPI-1434 well tolerated; no DLTs or SAEs related to study treatment
- ✓ Safety Review Committee supports moving to multi-dose portion

Phase 1 Multi-Dose

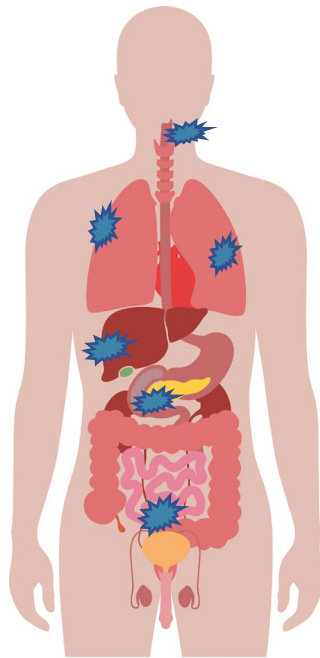
Data for Multi-Dose Treatments:
Imaging, dosimetry, safety and response data
Recommended Phase 2 dose and regimen
Anticipated in first half 2022

Phase 2a Study Initiation
Simon 2-stage design in 2-3 cohorts

-
- Variability in clinical trial duration attributable to timing of potential DLT observations
 - Timeline assumes no enrollment interruptions related to COVID-19 pandemic

IGF-1R Is Over-Expressed On Multiple Tumor Types

Target Broadly
expressed in tumor cells

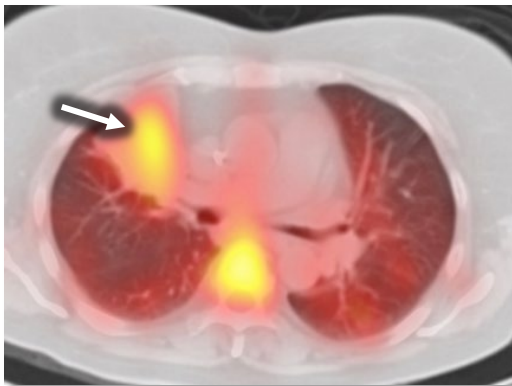
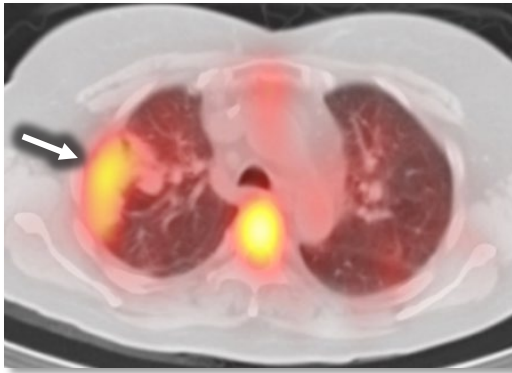


	% Patients with IGF-1R Expression
Ovarian	100%
Bladder	100%
Sarcomas	90%
Head and Neck	62%
Prostate	62%
NSCLC	59%
Pancreatic	57%
Colorectal	50%
Liver	50%
Breast	47%
Small Cell Lung	43%
Esophagus	40%
Renal	36%
ACC	36%

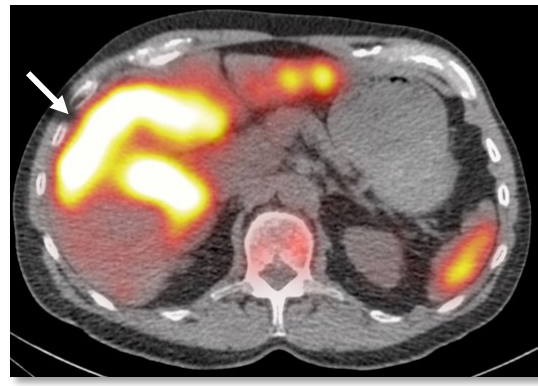
Tumor Uptake Has Been Observed In Different Types of Solid Tumors

Phase 1 Trial SPECT Imaging of Four Patients with Different Cancer Types (Transaxial Views)

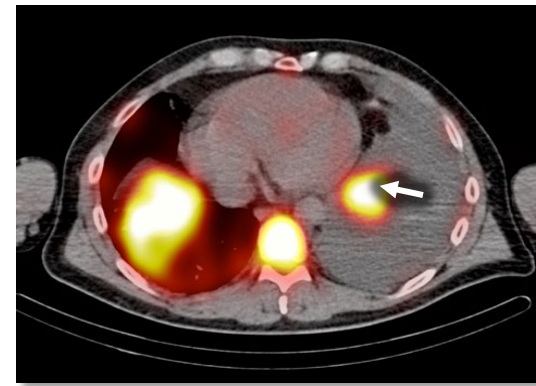
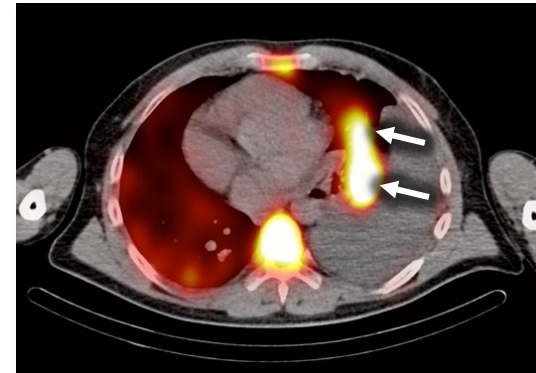
Ovarian
Pt # 204-007



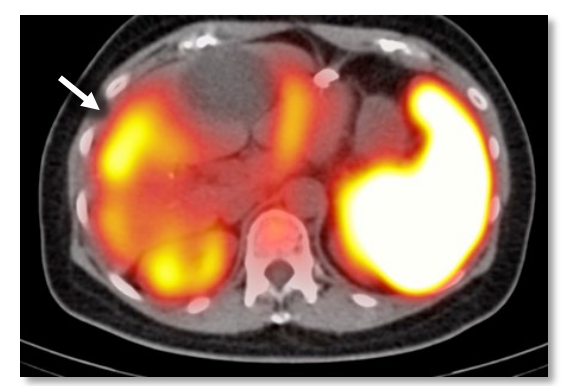
Prostate
Pt # 202-008



Sarcoma
Pt # 204-002

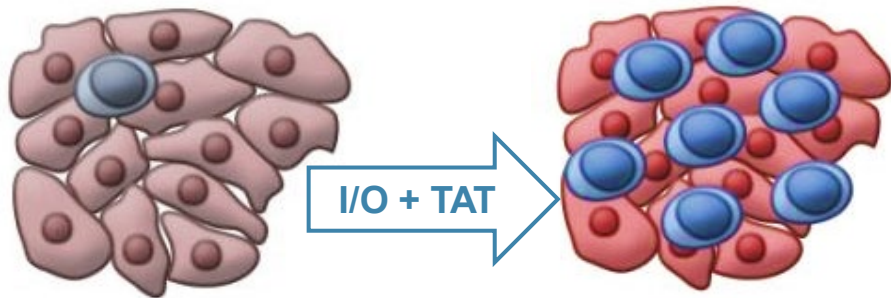


CRC
Pt # 204-008



Immuno-Oncology

Turning I/O “resistant” tumors into I/O “sensitive”



Immune desert
“cold tumor”

Immune
responsive
“hot tumor”

Enhancing antigen presentation and
stimulating T-cell recruitment: “Radiation
Activation and Vaccination”

DNA Damage Response Inhibitors (e.g., PARPi)

FPI-1434 = DNA Damage : DDRis Prevent DNA Damage Repair

Current Market:
DDRi Monotherapy

Breast



Ovarian

DDRi + TAT

DDRi Future Market:
Expanded with FPI-1434

NSCLC

Breast

HCC

Colorectal

Prostate

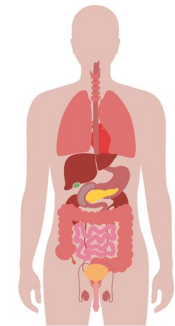
H&N

Recurrent
Thyroid

Pancreatic /
NETs

Adrenocortical
carcinoma

Sarcomas



Combination expands accessible indications
and reduces required doses

Utilize synergies with leading therapies to potentially move FPI-1434 up in the treatment paradigm

Fusion is Pursuing a Range of Opportunities to Build the Pipeline



TAT Clinical Combination

I/O

*DDRi
Market*

*DDRi
Novel*

PD-1/
CTLA4/etc.
(abscopal
effect)

PARP

ATM, ATR,
DNA-PK,
etc.

New Targeting Strategies

*Protein
Platform*

*Payload
Carrier*

Small molecules,
camelids,
nanobodies, others

Chelates/linkers
and
enhancement of PK
and other
properties

New Programs

Sourcing Targeting Molecules

Novel, existing (discontinued or LCM), single
or multi-asset in-licensing/partnership

Rationale

- 1) **FGFR3 is a validated cancer target** that is overexpressed on bladder and H&N cancers
 - FDA approved a pan-FGFR inhibitor for the treatment of bladder cancer with genetic alterations (i.e., translocation mutation)
- 2) **Potential clinical advantage:** An FGFR3-TAT may be more efficacious given the potency/MOA of a TAT
- 3) **Larger patient population / new indications:** Kinase inhibitor can only pursue mutations that cause cancer while a TAT can pursue the causative and/or correlative mutations of a cancer

Fusion's Approach

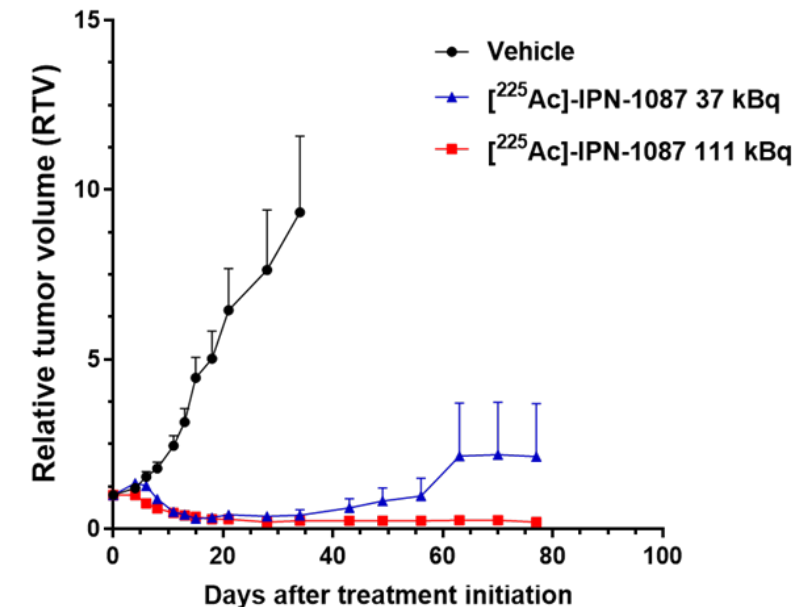
- An FGFR3-targeted TAT can address both driver and passenger mutations to deliver lethal radiation to the tumor
- Fusion acquired Vofatamab (naked anti-FGFR3 mAb) for conversion into a TAT
 - Vofatamab previously demonstrated good safety and tolerability in clinical trials in approximately 140 patients, most with advanced bladder cancer

Next Steps:

- Fusion will apply its refined process used with FPI-1434 to Vofatamab development
- IND planned for 1H 2021

- IPN-1087 **proven ability to deliver radiometals to tumors** in multiple cancer types makes it a promising candidate for targeted alpha therapy with ^{225}Ac
- Preclinical data with ^{225}Ac labeled IPN-1087 **show single dose tumor kill**
- Fusion will leverage its expertise to **quickly move** the ^{225}Ac labeled IPN-1087 (FPI-2059) **into clinical development**

^{225}Ac IPN 1087 Shows Single Dose Tumor Kill In Preclinical Models





1) Novel Targeted Alpha Therapies

- Jointly select up to three new TATs
- Co-fund
- Co-develop
- Option to co-commercialize in U.S.

2) Combination Therapies with TATs

- DNA Damage Response Inhibitors (DDRIs)
- Immuno-Oncology Agents
- AZ solely funds unless Fusion opts-in



\$299.5M

Cash, Cash Equivalents &
Investments

Balance Sheet as of 12/31/20

Cash to Fund Operations Through
End of 2023

Expected Cash Runway

41.7M

Basic Shares

Outstanding as of 12/31/20

Future Key Milestones by Program

Milestone	Timing*
FPI-1434 Mono	
● Phase 1 Multi-Dose Data	1H 2022
FPI-1434 Combo Studies	6 – 9 months following RP2D in monotherapy
FPI-1966	
● IND Submission	Q2 2021
FPI-2059	
● IND Submission	1H 2022

*Timelines assume no additional disruptions of pre-clinical or clinical activities resulting from the COVID-19 pandemic



Thank You

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