Immuneering

Investor Presentation

Ticker: IMRX

October 2021

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An Emerging Pipeline Focused on Achieving Broad Activity

13 Years in the Making

- **2008 Goal:** understand what is happening in responders, expand to more people (achieve broad activity)
- **Approach:** translational bioinformatics (analyzing transcriptomic, genomic, and/or proteomic data)
- **Early Years:** partnered with pharma to perform analyses of ibrutinib, ipilimumab, daratumumab, others. Built platform.
- **2018 Turning Point:** decision to use our platform to create a wholly owned pipeline

Lead Product Candidate for RAS Mutant Solid Tumors

- **IMM-1-104:** a dual-MEK inhibitor designed to target RAS mutant tumors
- **Existing RAS Inhibitors:** are limited to specific mutations (e.g., $KRAS^{G12C}$); reports of acquired resistance
- **Our Approach:** aims to treat broad patient populations durably and safely by disrupting signaling dynamics of tumors
- **IMM-1-104 Preclinical Studies:**
  - ✓ *Broad activity in RAS and RAF mutated tumor models and wide range of "humanized" 3D tumor models*
  - ✓ *Synergy with covalent $KRAS^{G12C}$ inhibitor, sotorasib (formerly known as AMG-510)[1]*
  - ✓ *Greater anti-tumor activity and improved tolerability vs selumetinib, binimetinib[1]*

A Robust Pipeline Enabled By Signaling Dynamics

- **Our Focus:** the MAPK and mTOR pathways, which are inappropriately activated in more than half of all cancers
- **Our Pipeline Beyond IMM-1-104:**
  - ✓ **MEK-io:** designed for immunologically "cold" solid tumors. In an animal model that does not respond to a checkpoint inhibitor alone, adding MEK-io inhibited tumor growth.
  - ✓ **Trifecta-MEK:** designed for BRAF mutant tumors. A potential alternative to RAF and MEK inhibitor combinations. Displayed 10X less pERK than registered RAF or MEK inhibitors *in vitro.*
  - ✓ **4** additional oncology programs
  - ✓ **2** neuroscience programs

Experienced Leadership Team Driving Innovation

[1]As demonstrated in head-to-head studies versus IMM-1-104 in certain tumor models

Experienced Leadership Team

Ben Zeskind, Ph.D., M.B.A.

Chief Executive Officer

Brett Hall, Ph.D.

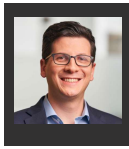
Chief Scientific Officer

Biren Amin, M.B.A.

Chief Financial Officer

Scott Barrett, M.D.

Chief Medical Officer



Michael Bookman, J.D.

General Counsel & Secretary

Becky Kusko, Ph.D.

VP, Business Dev & **Corporate Affairs**

Peter King, Ph.D.

VP, Head of Discovery **Oncology**

Kevin Fowler, Ph.D.

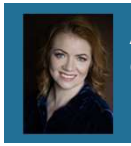
VP, Translational Res. **Oncology**

Sarah Kolitz, Ph.D.

VP, Translational Med. **Oncology**

J.L. Ross, Ph.D.

VP, Head of **Neuroscience**



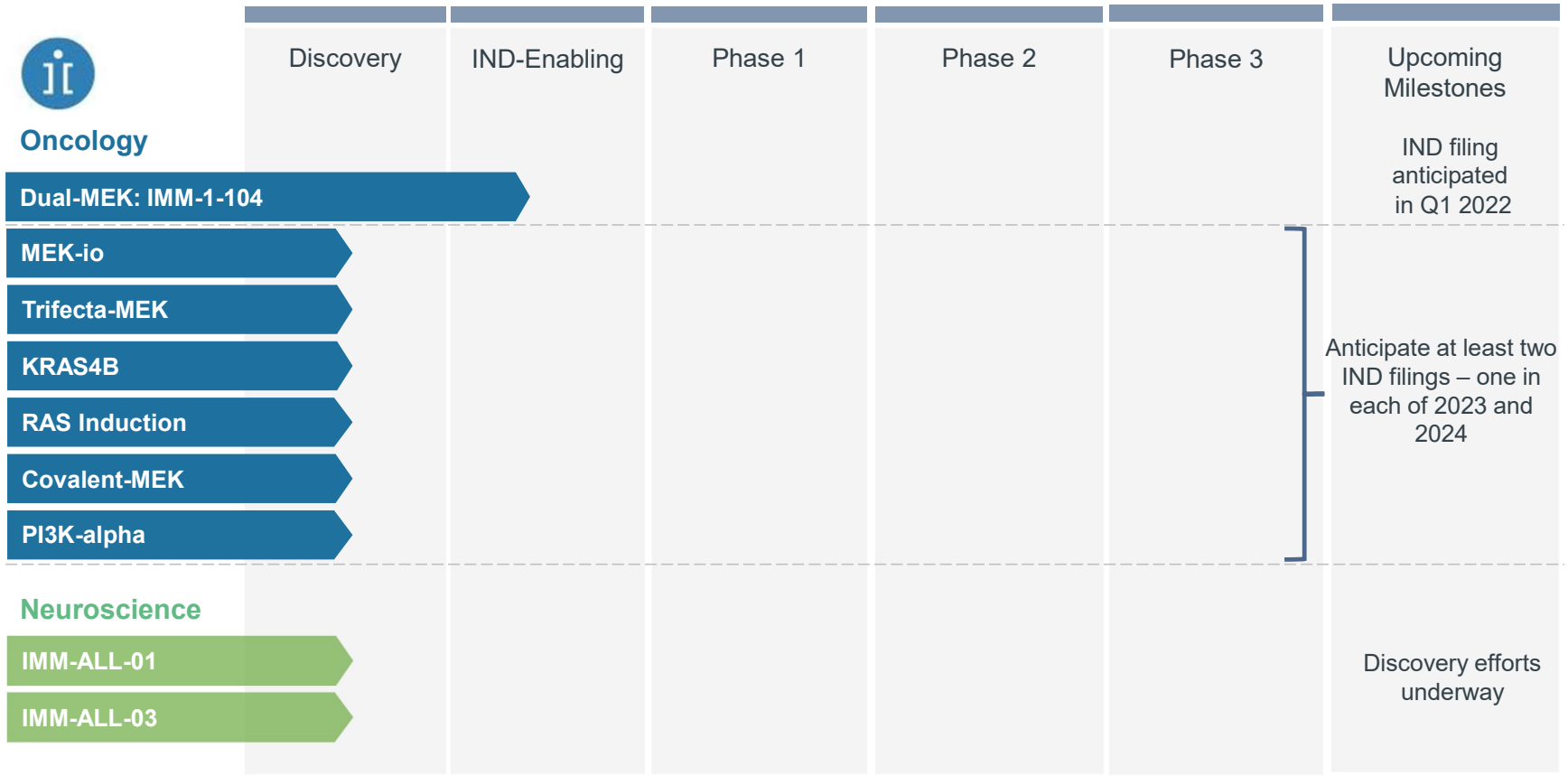
Anastasia Gutierrez, M.D.

VP, Clinical Operations **Oncology**

Mallory Morales, CPA

VP, **Finance**

7 Precision Oncology Programs Enabled by Signaling Dynamics



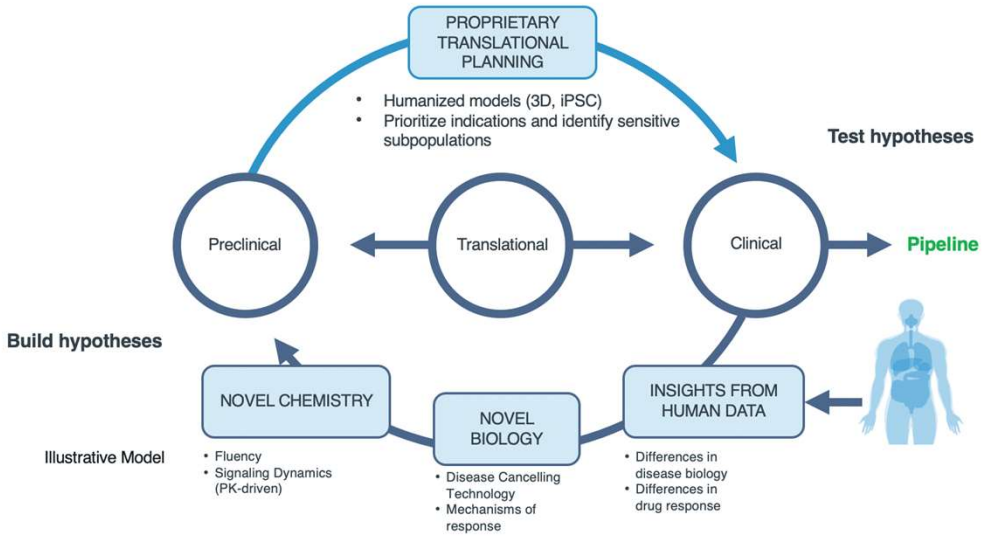
RAS Mutations are a Major Driver of Global Cases of Cancer

Indication	Annual Incidence (U.S.)	RAS^{mut}	Annual Incidence (EU)	RAS^{mut}	Annual Incidence (China)	RAS^{mut}
NSCLC Adenocarcinoma (Ras Mutations)	101,376	32%	205,340	24%	331,702	12%
Pancreatic Ductal Adenocarcinoma	54,387	86%	119,303	80%	104,661	82%
Colorectal Adenocarcinoma	149,500	41%	341,419	41%	303,853	49%
Cutaneous Melanoma	106,110	23%	150,627	24%	20,000	10%

Sources: 1) www.seer.cancer.gov; 2) Prior et al. Cancer Res 2020 Jul 15; 80(14):2969-2974 doi: 10.1158/0008-5472. CAN-19-3682; 3) Globocan registry; www.uicc.org/news/globocan-2020-new-global-cancer-data; 4) Turpin et al. Cancer Management and Research 2019 Sept 13; 11 8337-8344; doi: 10.2147/CMAR.S211119; 5) Scheffler et al. J Thorac Oncol 2019 Apr; 14(4): 606-616; doi; 10.1016/j.jtho.2018.12.013; 6) Heppt et al. BMC Cancer 2017 Aug 10; 17(1): 536. doi: 10.1186/s12885-017-3529-5; 7) Ho-Fung Loong et al. Transl Lung Cancer Res 2020 Oct; 9(5): 1759-1769. doi: 10.21037/tlcr-20-455.

IMM-1-104: Novel Approach to Targeting MEK

- We aim to achieve **broader activity and better tolerability** in the RAS mutant disease setting, versus existing FDA-approved agents:
  - AMG-510, a KRAS inhibitor, is indicated for the treatment of $KRAS^{G12C}$ mutated locally advanced NSCLC[1]
  - Current FDA-approved MEK inhibitors are indicated for BRAF mutant tumors and have shown little or no activity in RAS mutant disease.

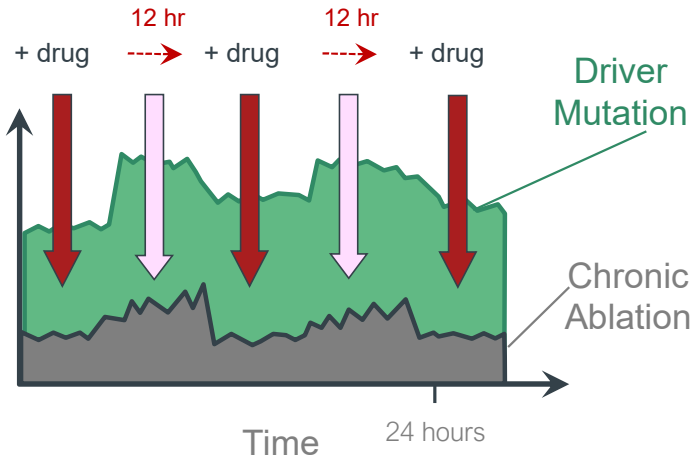


- Unlike existing FDA-approved agents, IMM-1-104 is designed to prevent RAF-mediated activation of MEK (CRAF-bypass) and is designed with a short plasma half-life to potentially achieve **deep cyclic inhibition** of the pathway.

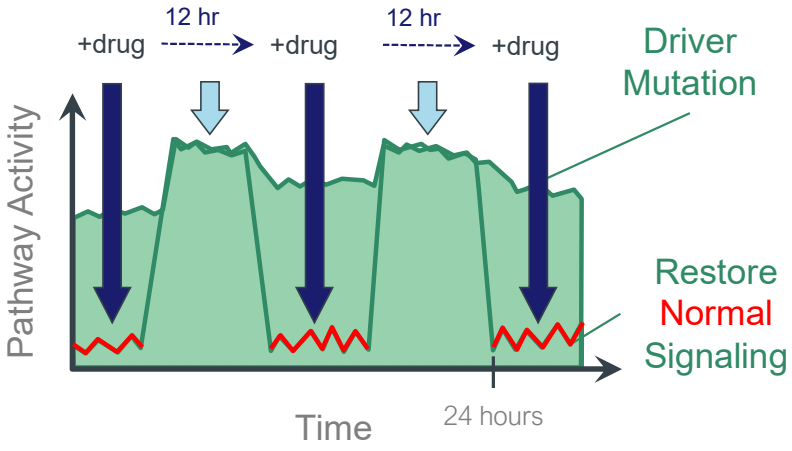
[1]In patients who have received at least one prior systemic therapy

Our Unique Approach: Deep Cyclic Inhibition

Traditional Approach



Signaling Dynamics



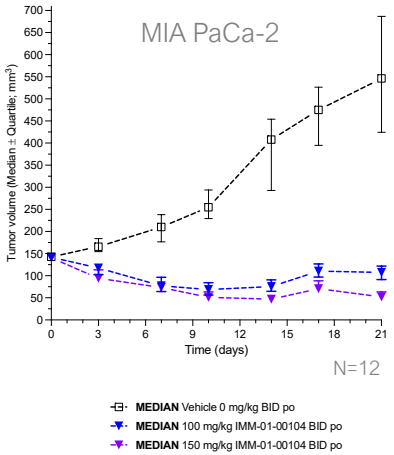
Chronic Suppression → TOXICITY

Cyclic Disruption → TOLERABILITY

Demonstrates Broad Activity in RAS and RAF Mutant Cancer

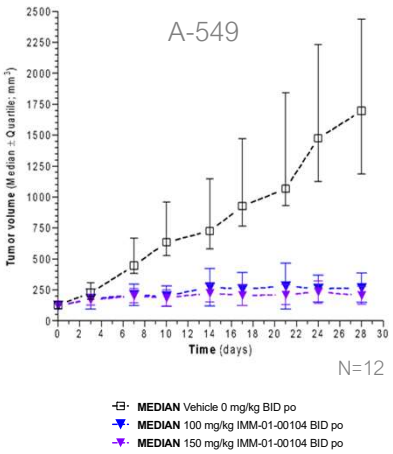
IMM-1-104

$KRAS^{G12C}$ **PANC**



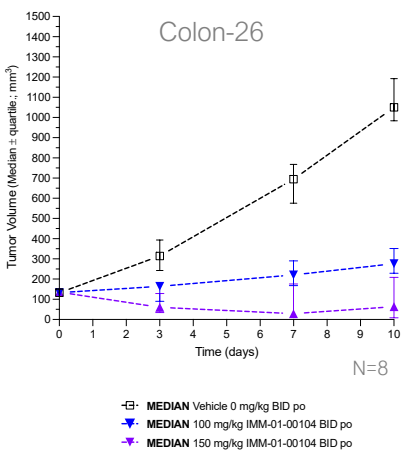
IMM-1-104

$KRAS^{G12S}$ **NSCLC**



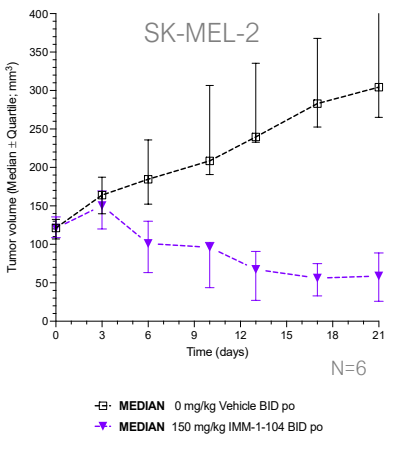
IMM-1-104

$KRAS^{G12D}$ **CRC**



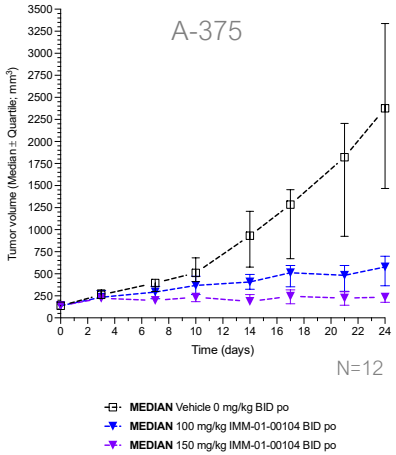
IMM-1-104

$NRAS^{Q61R}$ **MEL**



IMM-1-104

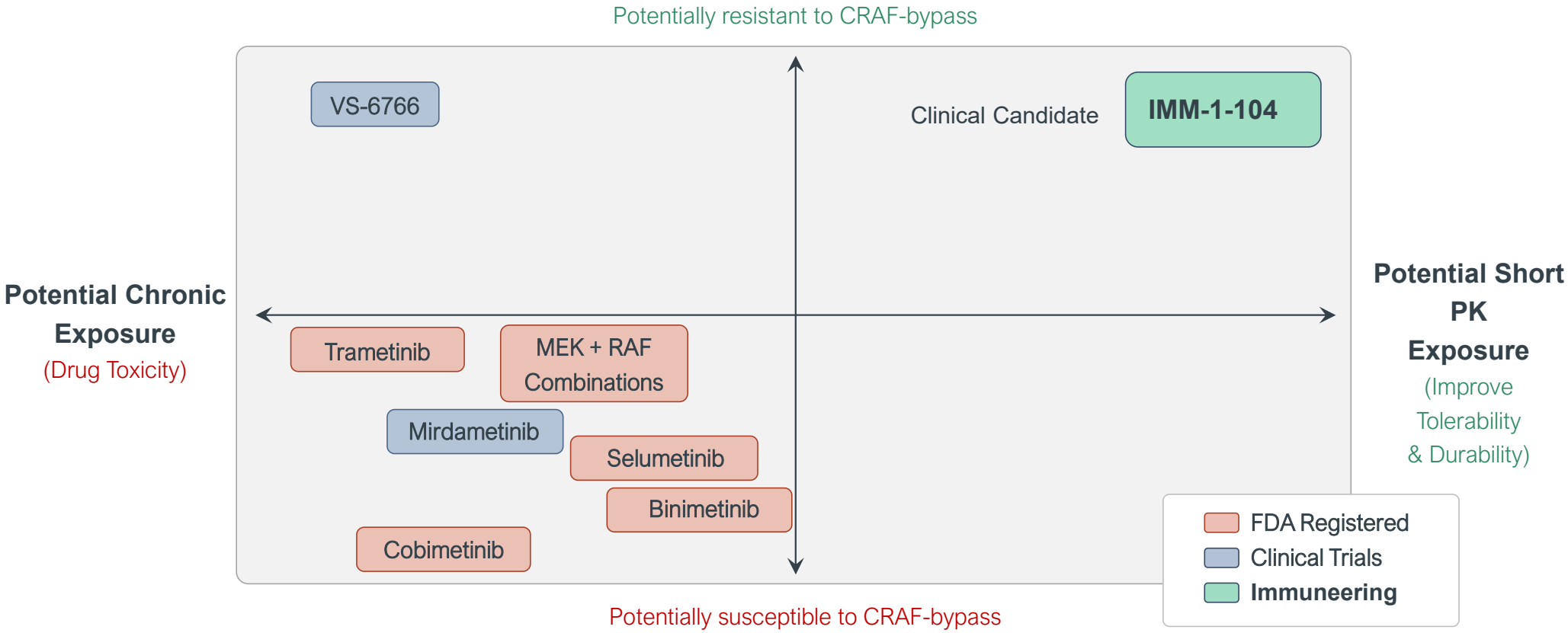
$BRAF^{V600E}$ **MEL**



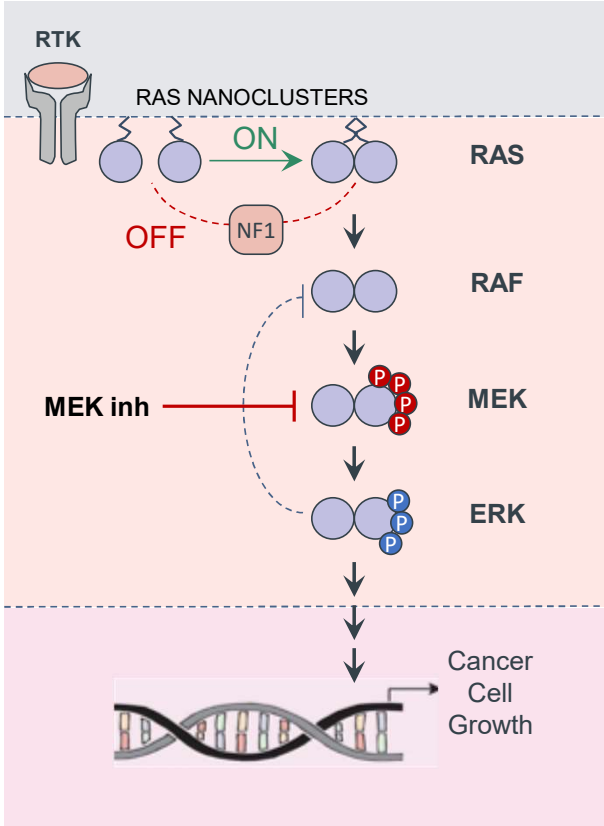
Goal: decouple therapeutic activity and tolerability; demonstrate **broader activity** in RAS mutant disease setting

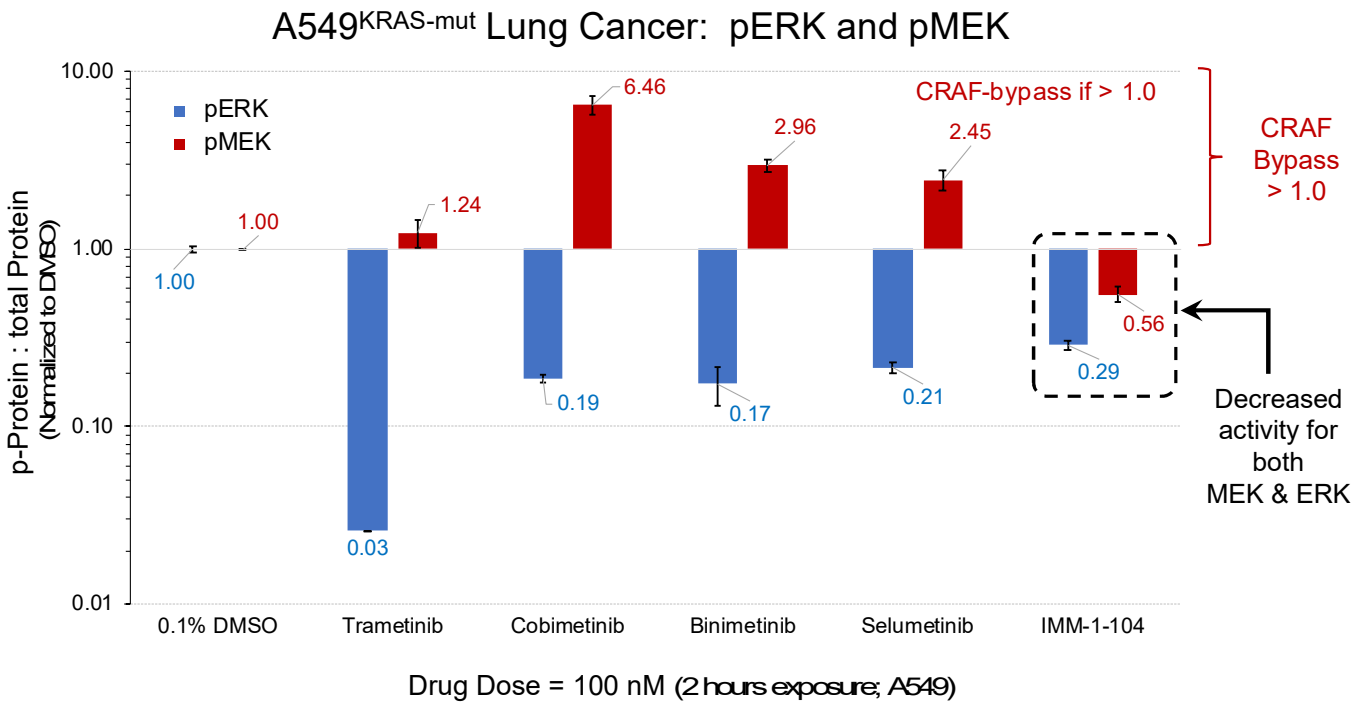
CRC = colorectal cancer; NSCLC = non-small cell lung cancer; MEL = melanoma; PANC = pancreatic cancer

IMM-1-104 Differentiation Versus MEK Inhibitors



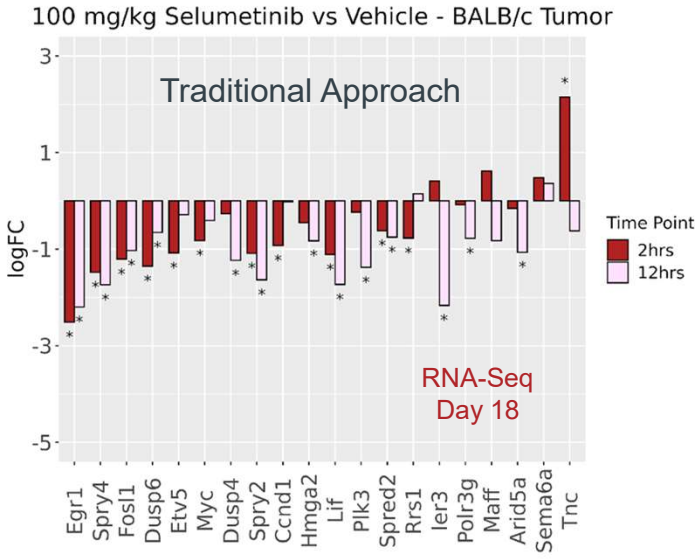
Head-to-Head Comparison of IMM-1-104 Against FDA-Approved MEK Inhibitors: CRAF-Bypass Resistance

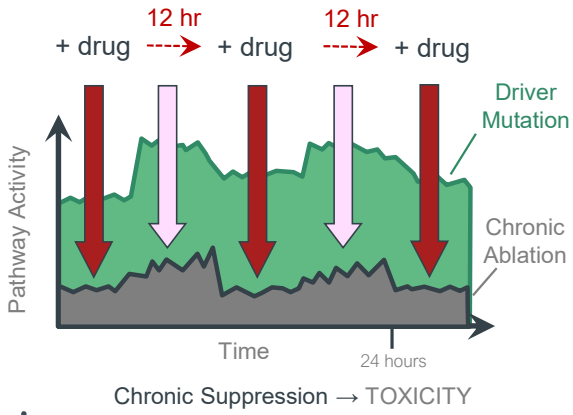




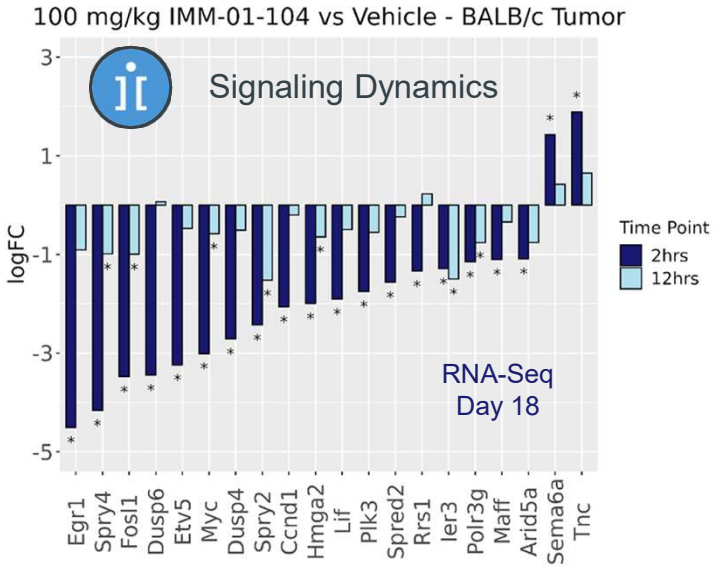
- **FDA-Approved MEK inhibitors:** Trametinib, Cobimetinib, Binimetinib, Selumetinib; Commercially purchased

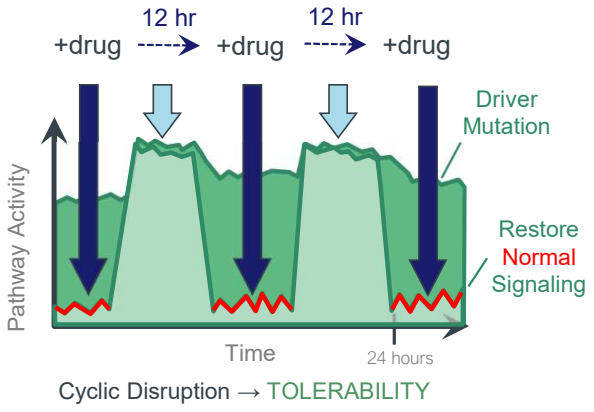
Deep Cyclic Inhibition Confirmed Using Transcriptomics





Chronic Suppression → TOXICITY





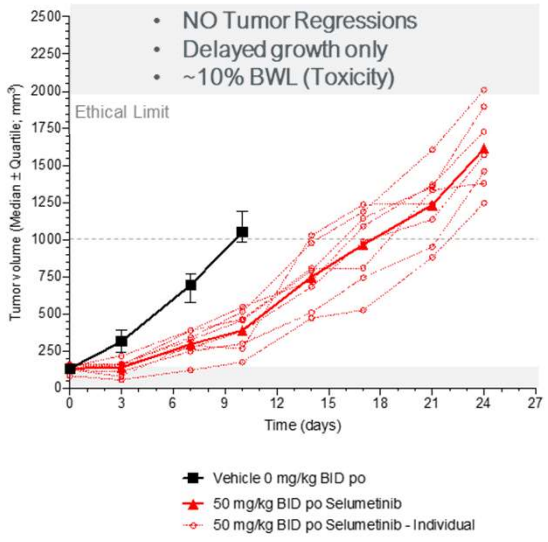
Cyclic Disruption → TOLERABILITY

Head-to-Head Comparison of IMM-1-104 Against Selumetinib and Binimetinib in KRAS^{G12D} CRC Tumor Model

IMM-1-104 as compared to selumetinib and binimetinib demonstrated greater tumor growth inhibition (TGI), lower body weight loss (BWL) and greater durability evidenced by sustained reductions in tumor volume (TV)

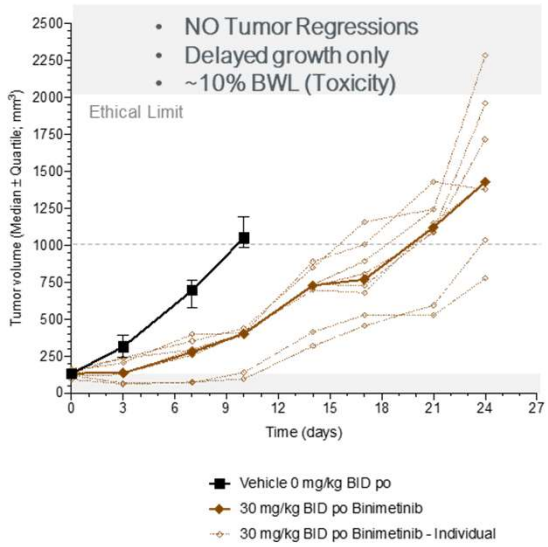
Selumetinib at MTD

(FDA-approved APR-2020)



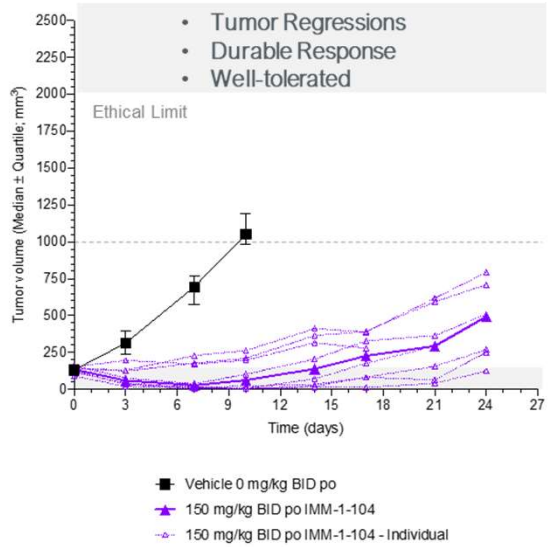
Binimetinib at MTD

(FDA-approved JUN-2018)



IMM-1-104 ≤ MTD

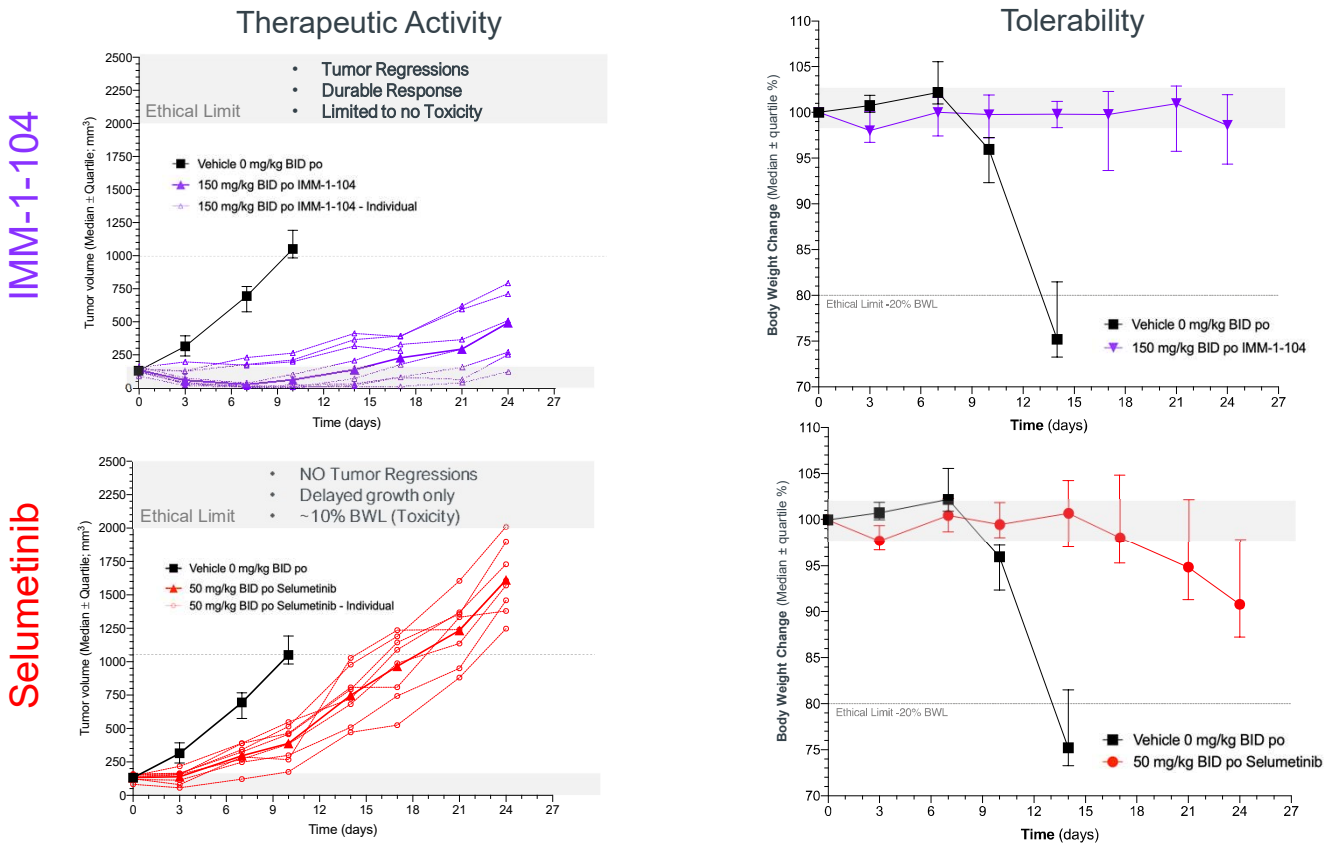
(IND-Enabling Stage)



Colon-26 (KRAS^{G12D}) BALB/c Syngeneic Colorectal Tumor Model

Colon-26 (KRAS^{G12D}) Syngeneic Colorectal Tumor Model in Immune Competent Mice
Selumetinib and Binimetinib were commercially purchased

Head-to-Head Comparison of IMM-1-104 Against Selumetinib in KRAS^{G12D} CRC Tumor Model

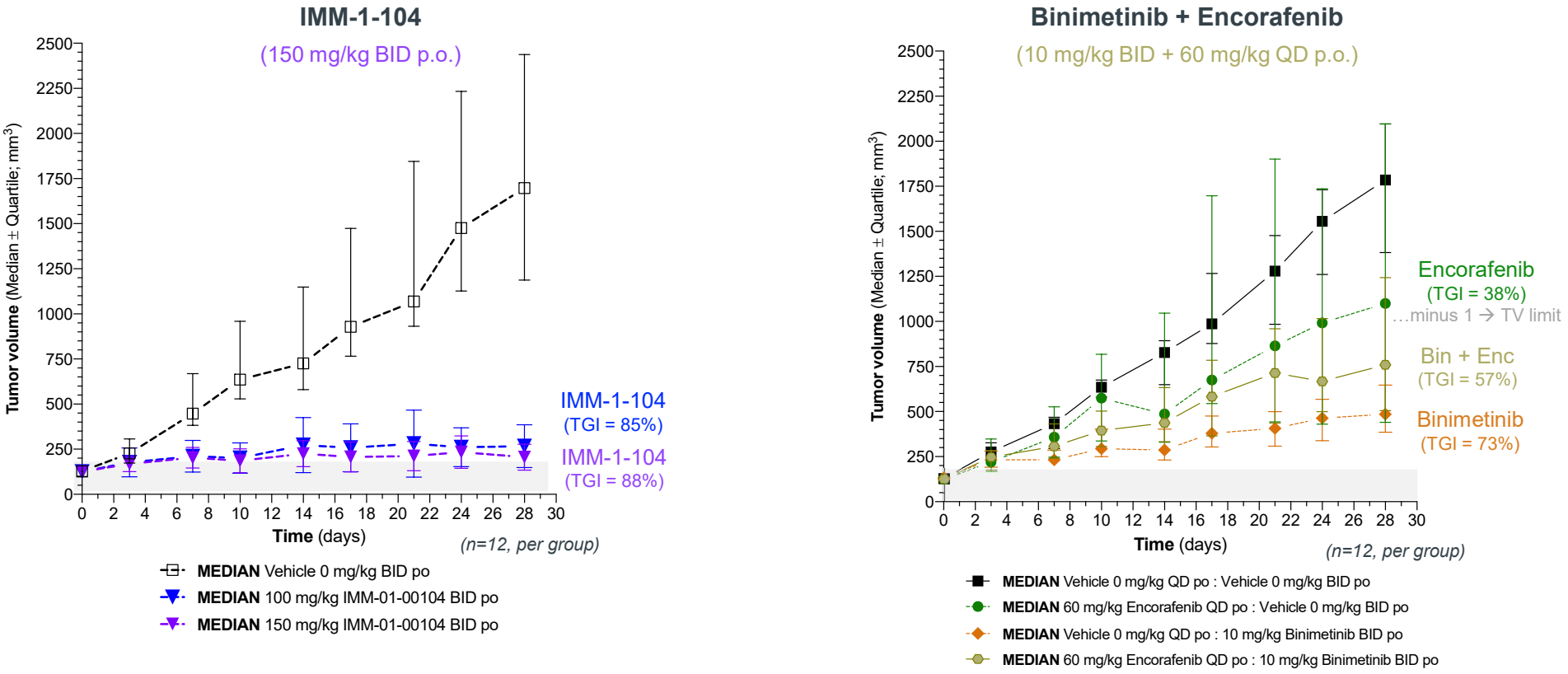


Colon-26 (KRAS^{G12D}) Syngeneic CRC Tumor Model

Selumetinib was commercially purchased

Head-to-Head Comparison of IMM-1-104 Against Binimetinib +/- Encorafenib in KRAS^{G12S} NSCLC Tumor Model

IMM-1-104 as compared to binimetinib monotherapy demonstrated greater tumor growth inhibition (TGI)

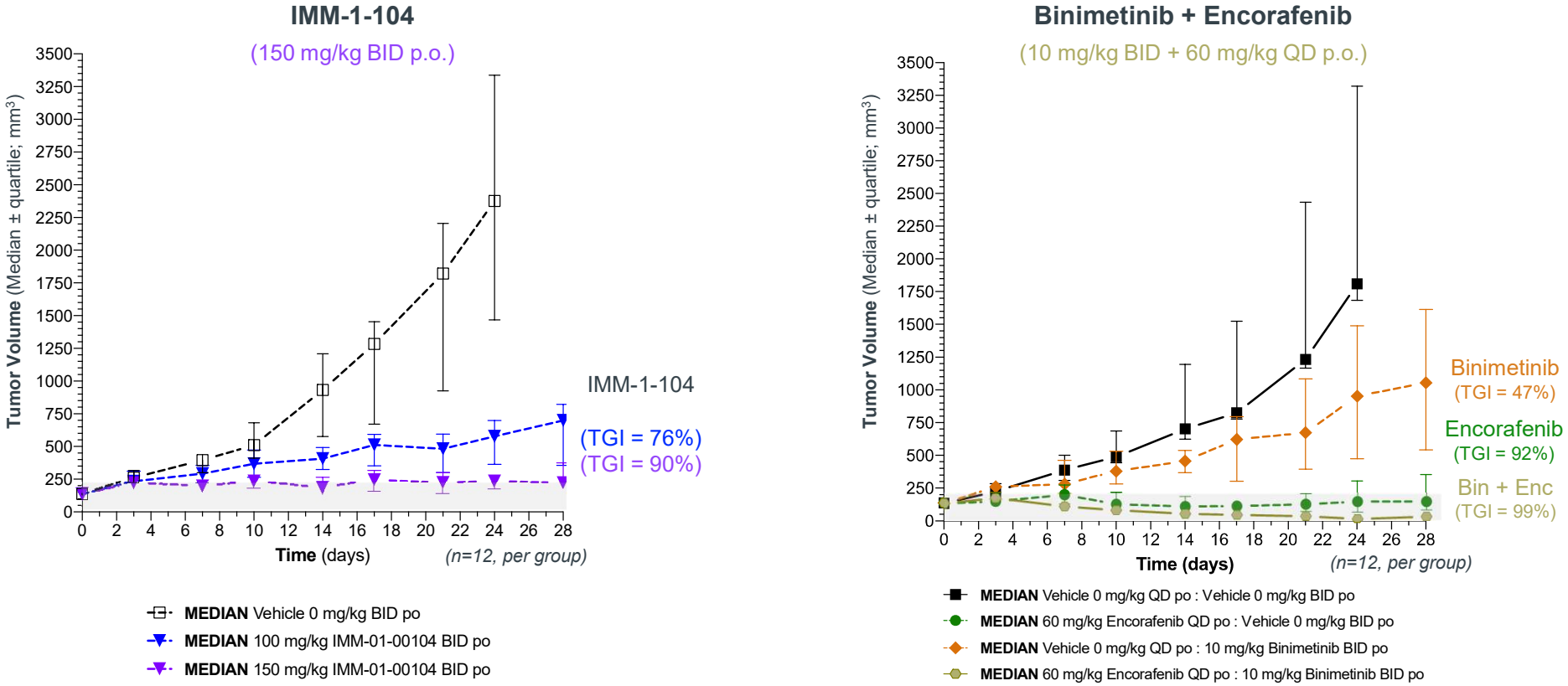


A549 (KRAS^{G12S}) NSCLC Xenograft Tumor Model in Athymic Nude Mice

Binimetinib and Encorafenib were commercially purchased

Head-to-Head Comparison of IMM-1-104 vs. Binimetinib +/- Encorafenib in BRAF^{V600E} Melanoma Tumor Model

IMM-1-104 as compared to binimetinib monotherapy demonstrated greater tumor growth inhibition (TGI)

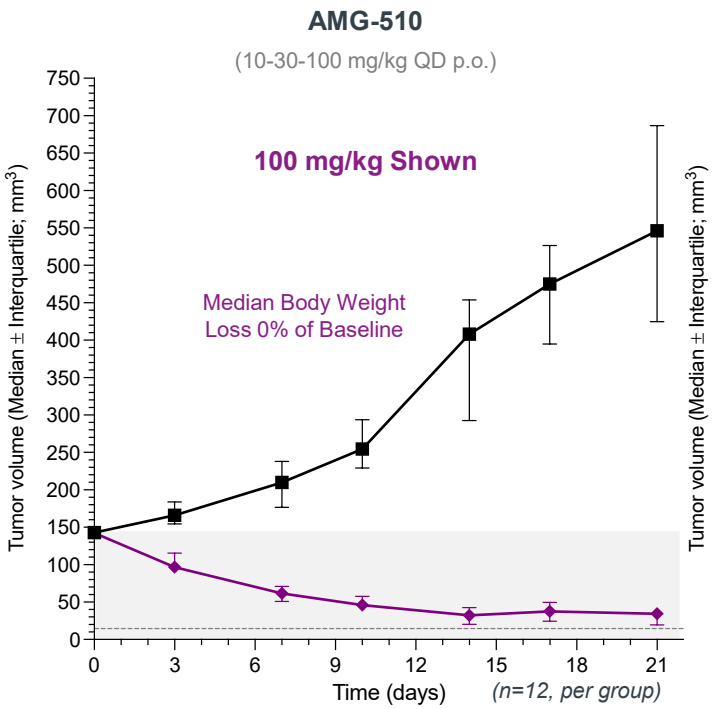


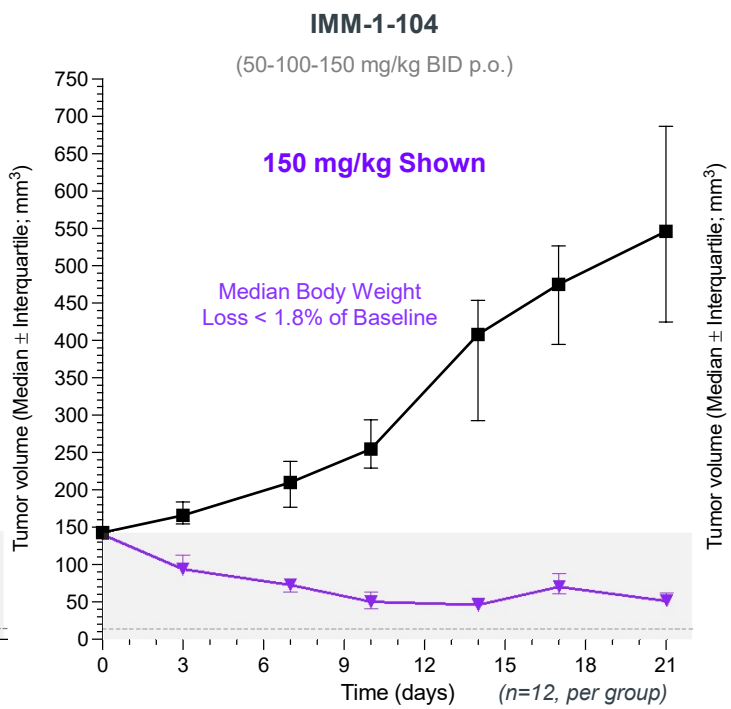
A375 (BRAF^{V600E}) Melanoma Xenograft Tumor Model in Athymic Nude Mice

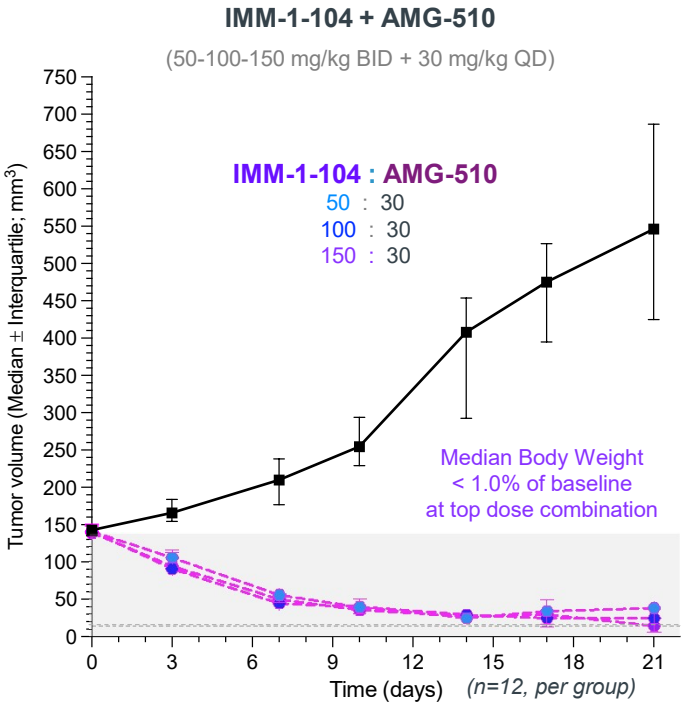
Binimetinib and Encorafenib were commercially purchased

Head-to-Head Comparison of IMM-1-104 +/- AMG-510 in a KRAS^{G12C} Pancreatic Tumor Model

IMM-1-104 as compared to AMG-510 demonstrated tumor regression with insignificant BWL





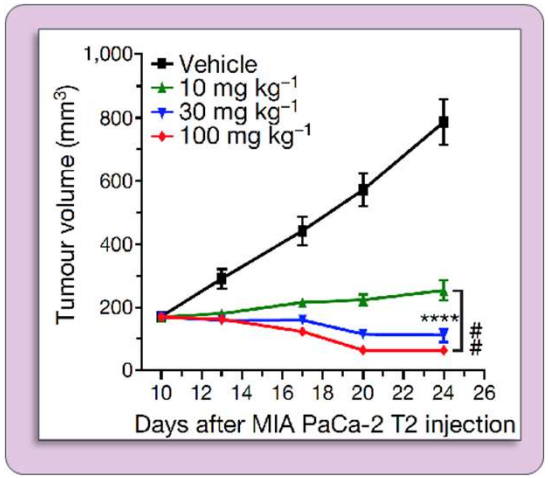


MIA PaCa-2 (KRAS^{G12C}) Pancreatic Xenograft Tumor Model in Athymic Nude Mice

Benchmark Comparisons in a KRAS^{G12C} Pancreatic Tumor Model

Amgen Benchmark - AMG-510

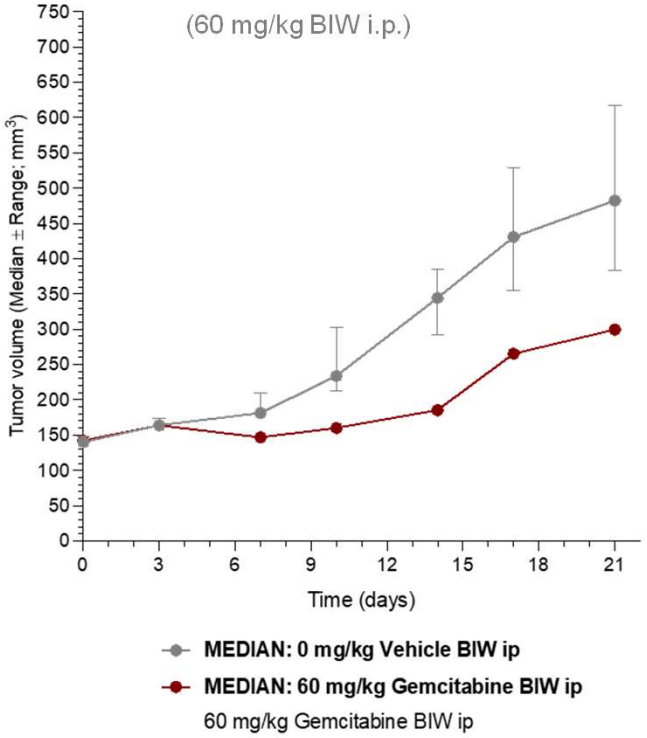
(10-30-100 mg/kg QD p.o.)



Canon, et al. 2019 Nature 575(7781):217

Gemcitabine

(60 mg/kg BIW i.p.)



MIA PaCa-2 (KRAS^{G12C}) Pancreatic Xenograft Tumor Model in Athymic Nude Mice

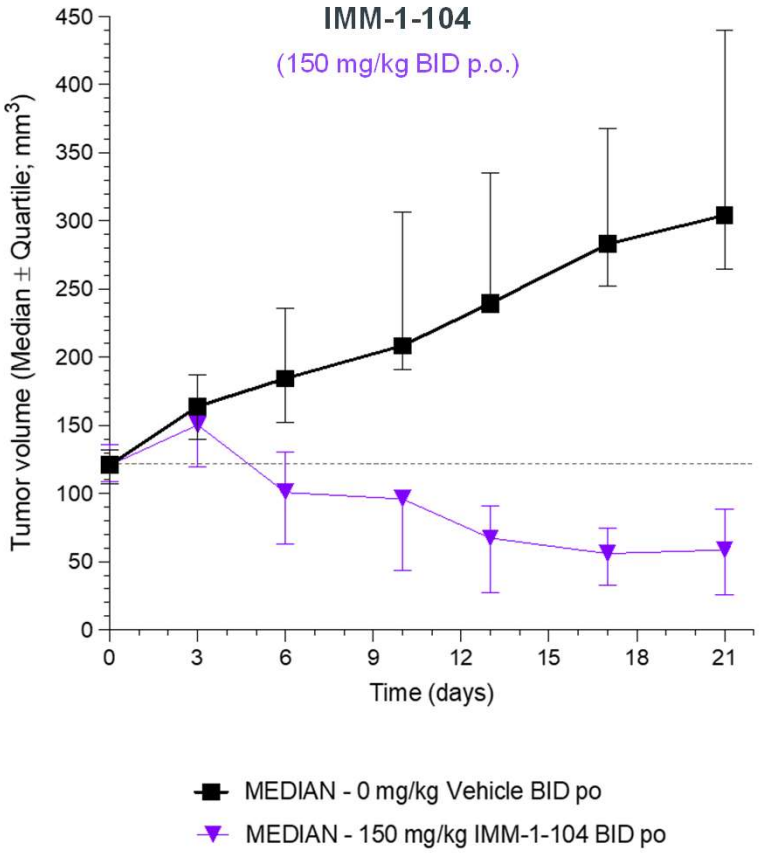
ELN: PJK03-0033PK – 21 Day Study *(Complete)*

IMM-1-104 Evaluated in NRAS^{Q61R} Melanoma Tumor Model

IMM-1-104 demonstrated tumor volume regression

IMM-1-104

SK-MEL-2 NRAS^{Q61R} Mutant Melanoma

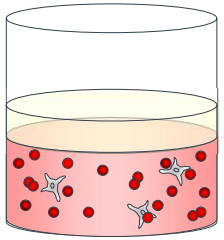


SK-MEL-2 (NRAS^{Q61R}) Melanoma Xenograft Tumor Model in Athymic Nude Mice

Leveraging Humanized 3D Tumor Models for Translational Profiling

- 3D tumor growth models mimic the tumor microenvironment more closely than standard 2D models
- 3D tumor growth models more accurately reflect human tumor biology and complexity when evaluating pharmacological data of MAPK pathway inhibition *in vivo*[1]
- Established several dozen humanized 3D tumor models that display mutations in the MAPK pathway to evaluate sensitivity to IMM-1-104
- Tumor models with KRAS or NRAS mutations were most sensitive to IMM-1-104 followed closely by tumor models with BRAF mutations
- Our 3D tumor growth model data suggests that KRAS mutant pancreatic cancer and NRAS mutant melanoma may be particularly sensitive to single agent IMM-1-104

Humanized 3D-TGA[2]



Tumor cells + Humanized Factors in 3D Extracellular Matrix

Complexity & TME-relevance

[1]Janes et al. Targeting KRAS Mutant Cancers with a Covalent G12C-Specific Inhibitor. Cell. 2018 Jan 25;172(3):578-589.e17. doi: 10.1016/j.cell.2018.01.006. PMID: 29373830.
[2] Sasser, et al. 2007 Cancer Letters (PMID:17467167); Casneuf, et al. 2016 BCTT (PMID: 26893580)

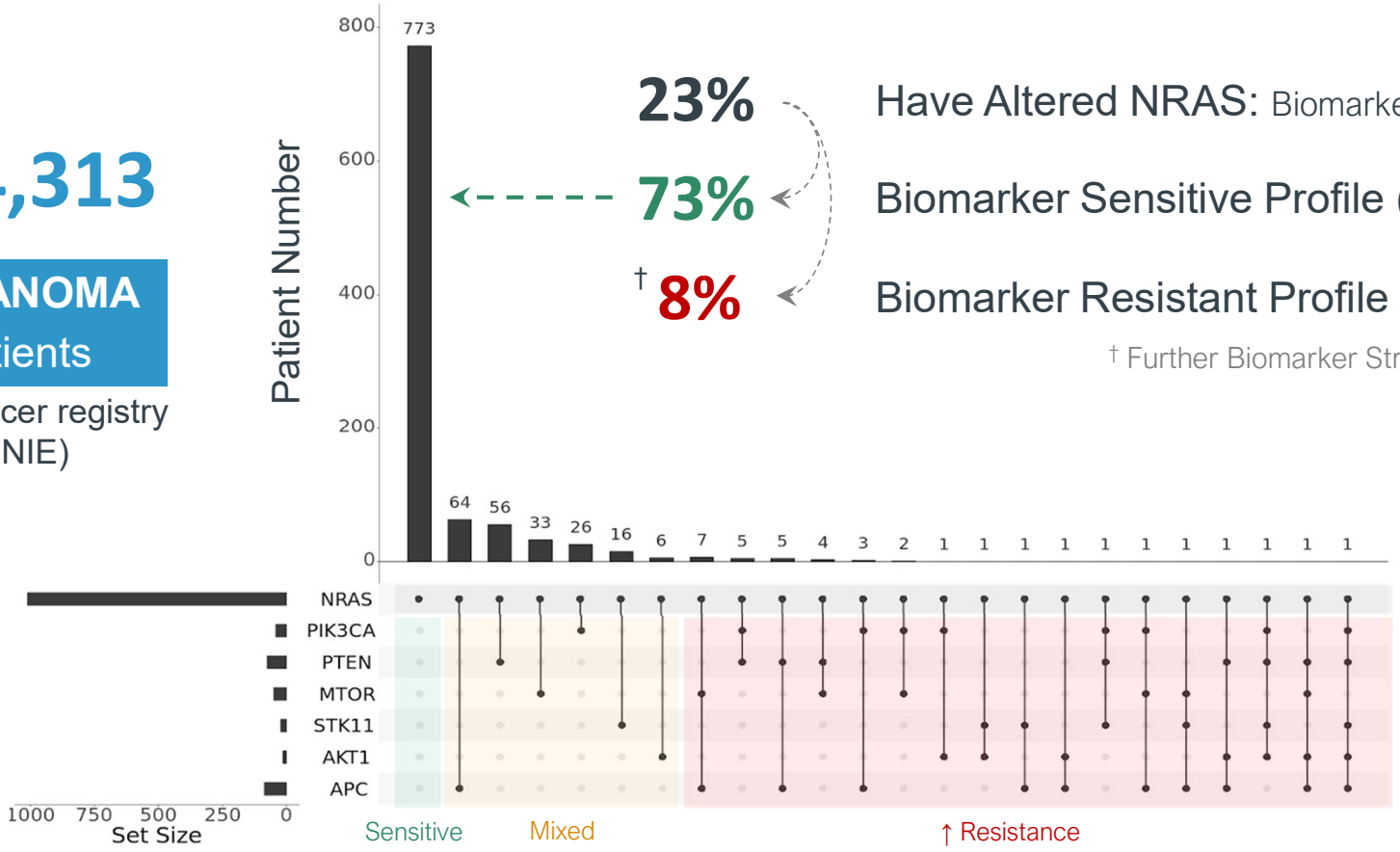
NRAS Mutant Melanoma: Translational Opportunity

(1.) High unmet clinical need population & (2.) Majority of patients display sensitivity profile to single agent IMM-1-104

of 4,313

MELANOMA Patients

in pan-cancer registry (GENIE)



23% Have Altered NRAS: Biomarker Positive

73% Biomarker Sensitive Profile (17.9% overall)

† **8%** Biomarker Resistant Profile (1.8% overall)

† Further Biomarker Stratification Not Required

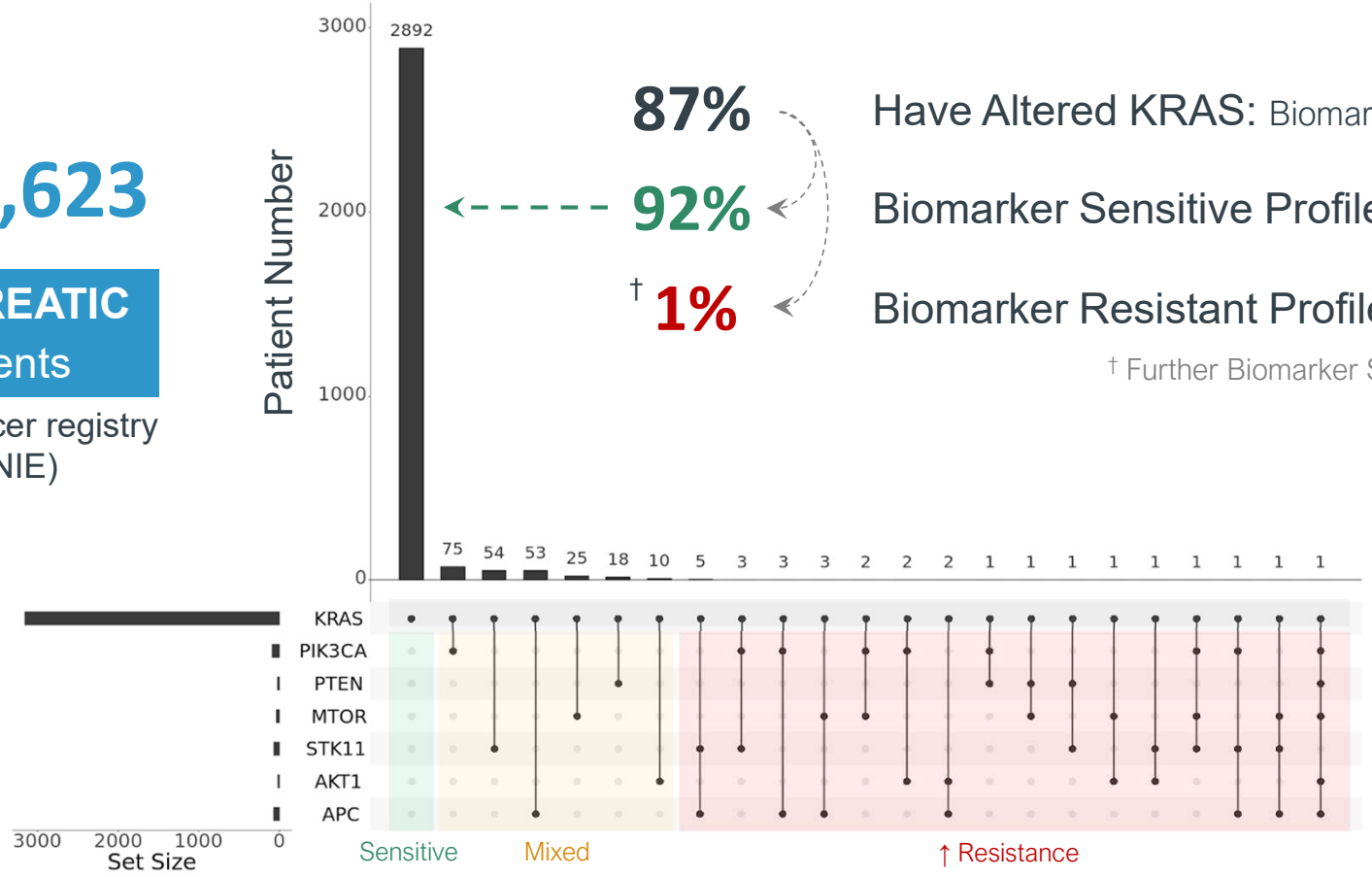
KRAS Mutant Pancreatic (PAAD): Translational Opportunity

(1.) High unmet clinical need population & (2.) Majority of patients display sensitivity profile to single agent IMM-1-104

of 3,623

PANCREATIC Patients

in pan-cancer registry (GENIE)



87% Have Altered KRAS: Biomarker Positive

92% Biomarker Sensitive Profile (79.8% overall)

† **1%** Biomarker Resistant Profile (0.8% overall)

† Further Biomarker Stratification Not Required

IMM-1-104: Phase 1/2 Trial Plan

Study 1
First Time in Humans

Dose Escalation Phase 1a[1]

IMM-1-104
(Dual MEK inhibitor)

→ Bayesian Design (mTPI-2) Escalation in select solid tumor types[1]

May expand tolerated dose cohorts for a max of 36 patients treated in Phase 1

→ **MTD / MAD**

Dose Expansion Phase 2a [2,3]

Expansion 1:

- **NRAS mutated Melanoma** (~40 pts)

Expansion 2:

- **KRAS Mutated Pancreatic (PAAD)** (~40 pts)

Expansion 3 & 4: *biomarker stratification*

- **NSCLC-Adeno** (40 pts) KRAS mutated
- **CRC** (40 pts) KRAS mutated, APC *wt*

IND filing expected in Q1 2022, Phase 1 clinical trial initiation in 1H 2022

[1] Solid tumor, all comer with evidence of increased MAPK pathway activation (especially: RAS^{mut})

[2] Proposed tumor types may change based upon ongoing preclinical PGx studies and clinical function review

[3] Simon 2-Stage Design; Stage 1, 10-14 patients; stage 2, up to 32 patients

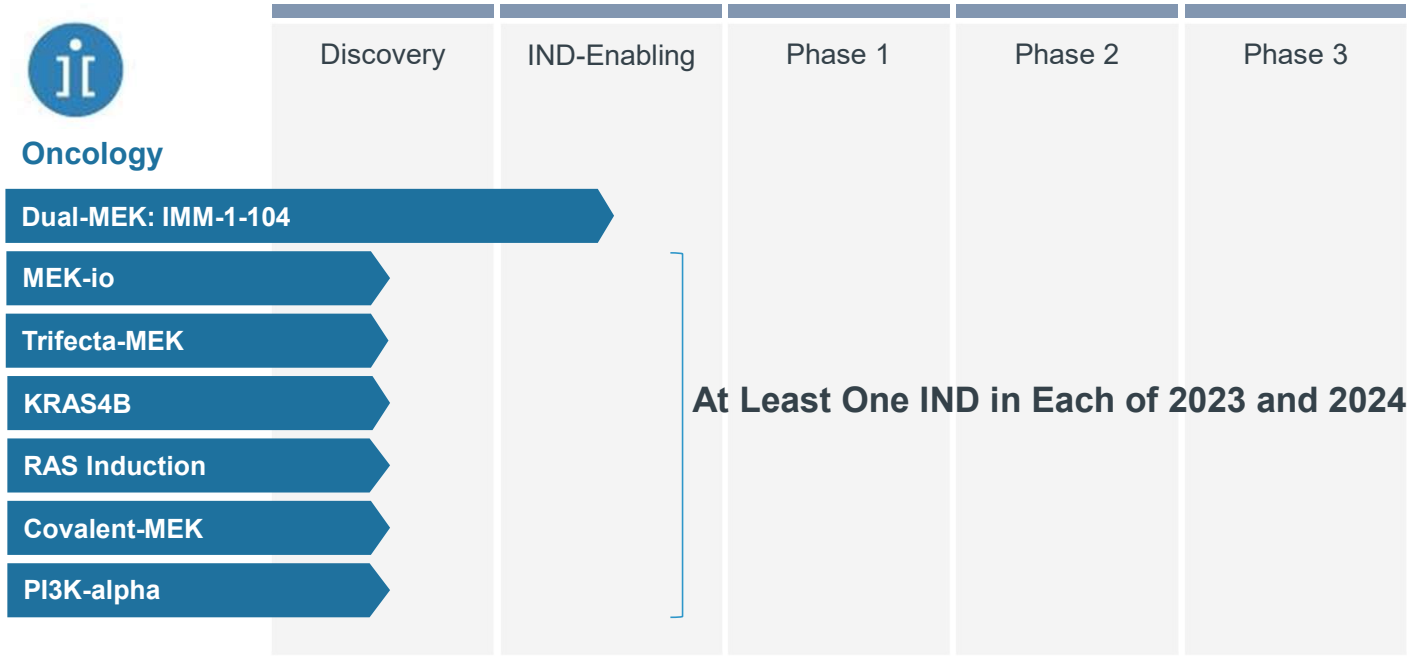
MTD = Maximum Tolerated Dose; MAD = Minimum Active Dose; RP2D = Recommended Phase 2 Dose; PGx = Pharmacogenomics; MAPK = Mitogen-Activated Protein Kinase

IMM-1-104: Competitive Landscape

IMM-1-104 Plans To Target RAS Mutant Tumors Without Requiring A RAF Inhibitor And Exhibits A Short Half-Life

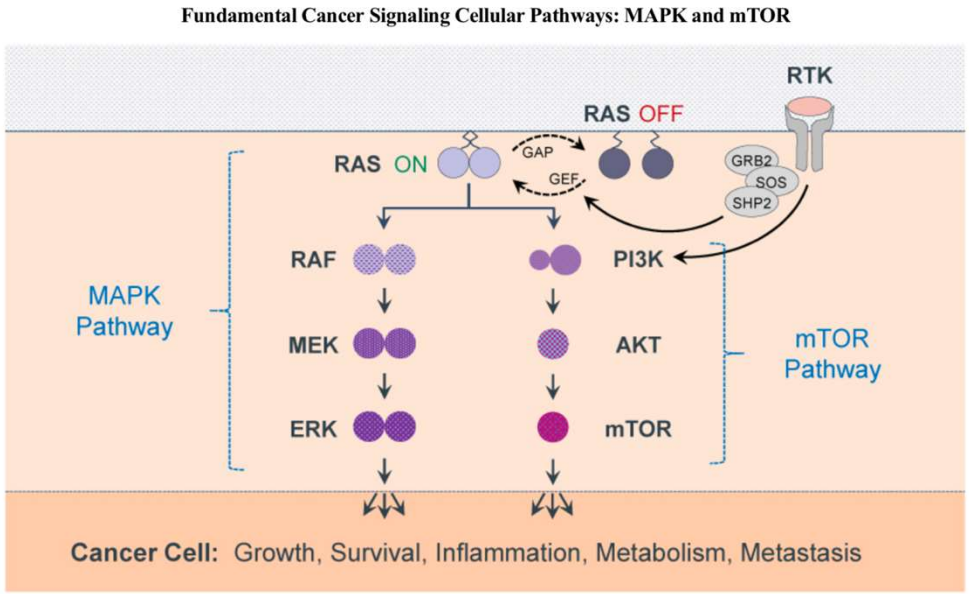
	Drug Name	Brand Name	Company	Indication	Target/MOA
FDA -approved	**Binimetinib**	Mektovi	Pfizer	Metastatic melanoma with BRAFV600 mutation with BRAF	MEK
	Cobimetinib	Cotellic	Roche	Metastatic melanoma with BRAFV600 mutation with BRAF	MEK
	Selumetinib	Koselugo	AstraZeneca	Neurofibromatosis Type 1	MEK
	Trametinib	Mekinist	Novartis	Metastatic melanoma with BRAFV600 mutation with BRAF Adjuvant in melanoma with BRAFV600 mutation with BRAF NSCLC with BRAFV600E with BRAF Anaplastic thyroid cancer with BRAFV600E with BRAF	MEK
Phase 1/2	**VS-6766**	-	Verastem	Low grade serous ovarian cancer (monotherapy and combo with FAKi) KRAS-mutant NSCLC (monotherapy and combo with FAKi) Metastatic uveal melanoma (combo with FAKi)	RAF/MEK
	Mirdametinib	-	SpringWorks Therapeutics	Neurofibromatosis Type 1 (monotherapy) Solid tumors (combination with Beigene's lifirfenib)	MEK
	Pimasertib	-	Day One Biopharma	MAPK-altered solid tumors (in combo with pan-RAFi)	MEK
	FCN-159	-	ShanghaiFosun	NRAS-aberrant and NRAS-mutant metastatic melanoma	MEK

A Focus on the Early-Stage Oncology Pipeline

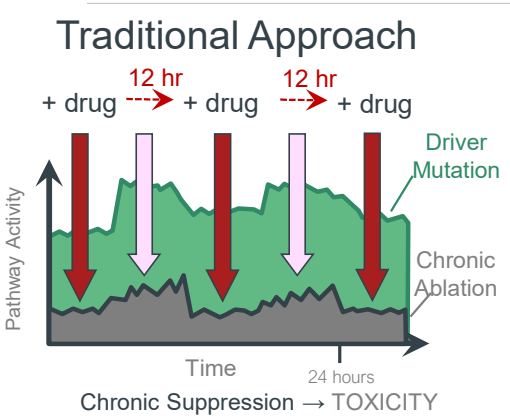


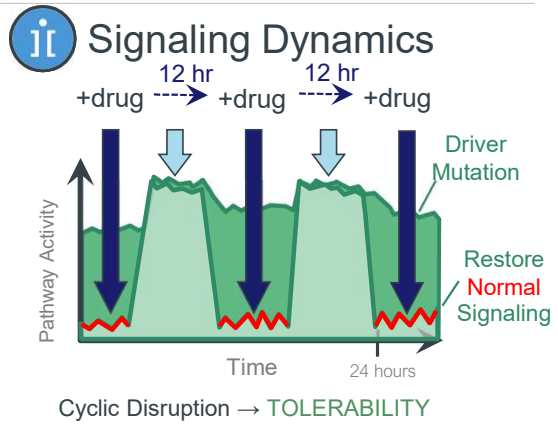
Building a Franchise Pipeline Against Core Oncology Addiction

Expand on novel approach of deep cyclic oncogenic pathway inhibition



Program	Target	Potential Attribute	Potential Differentiating Factors
Dual-MEK	MEK	Target + $T_{1/2}$	Unique Design
MEK-io	MEK	Target + $T_{1/2}$	IO Optimized
Trifecta-MEK	MEK	Engagement	Potency + Target
RAS-i	Pan-RAS	Nanoclusters	RAS Induction
KRAS4B-inh	KRAS4B	Nanoclusters	Novel Inhibition
Covalent-MEK	MEK	Engagement	Lock Down + $T_{1/2}$
PI3K-alpha	PI3K-α	Allosteric	Target + $T_{1/2}$

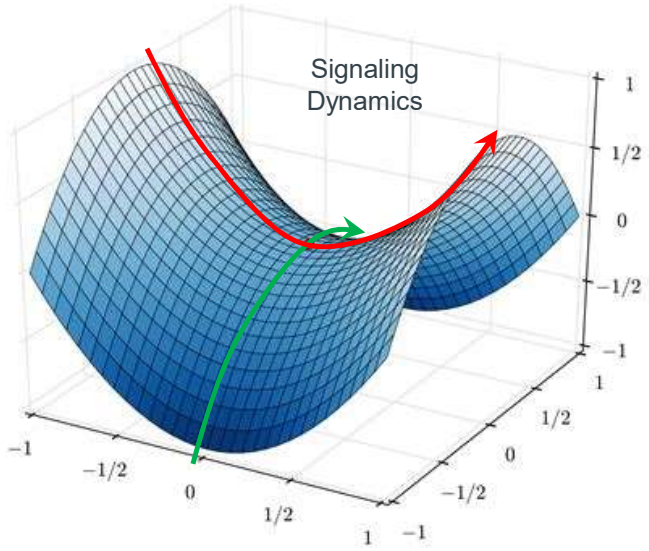




MEK-io: Optimize Immune Modulation of MAPK

Program in Lead Optimization: *In Vivo* PoC Achieved → Currently Screening for Potential Clinical Candidates

The MEK-io Saddle Point



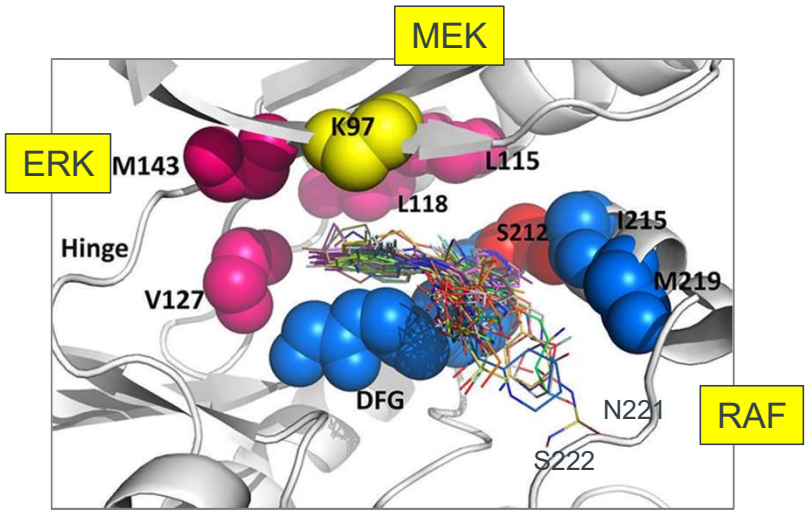
- **Maximize** Antitumor MEK Impact
- **Optimize** Antitumor Immunity

Int J Mol Sci 2019 Salaroglio, et al.

- Patients with immunologically cold tumors have suboptimal T-cell responses and represent unmet medical need
- MEK-io Goal: Develop a MEK inhibitor with unique PK and PD profiles that optimally boosts antitumor immunity via toggling ERK activity
- The investigational MEK-io program inhibitors are designed to optimize the patient's immune response and promote antitumor responses in combination with select immune modulators
- **Initial *in vivo* proof-of-concept observed** with synergistic antitumor activity in combination with a checkpoint inhibitor in a widely used syngeneic murine model

Trifecta-MEK: Unique MEK Engagement

Program is in Late Lead Identification: Goal is Superiority Versus BRAF/MEK inhibitor Combination Therapy



2017 Zhao PLoS One, MEK Type-III Inhibitors
2015 Wu Semin Oncol, MEK1/2 Inhibitors

A549 tumor model: $KRAS^{G12S}$ mutant NSCLC

Compound	pERK: tERK 100 nM A549 % of control, 4h	pMEK: tMEK 100 nM A549 % of control, 4h	Notes
0.1% DMSO	**1.000**	1.000	Vehicle Control
Encorafenib	**2.693**	3.431	Paradoxical MAPK Activation
Binimetinib	**0.173**	4.031	CRAF-bypass Evident
Trifecta-MEK*	**0.011**	**0.345**	**pERK and pMEK control**

A375 tumor model: $BRAF^{V600E}$ mutant Melanoma

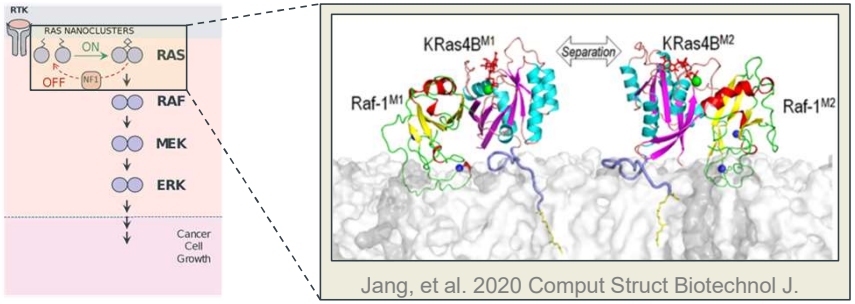
Compound	pERK: tERK 100 nM A375 % of control, 4h	pMEK: tMEK 100 nM A375 % of control, 4h	Notes
0.1% DMSO	**1.000**	1.000	Vehicle Control
Encorafenib	**0.023**	0.039	Prevents pMEK (BRAF inhibitor)
Binimetinib	**0.057**	1.094	BRAF activity stable (pMEK)
Trifecta-MEK*	**0.002**	**0.095**	**pERK and pMEK control**

*One of our investigational Trifecta-MEK program inhibitors

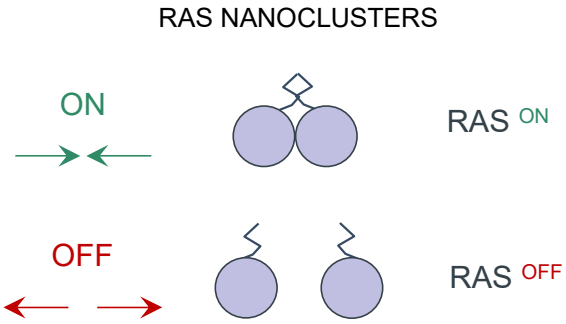
Binimetinib and Encorafenib were commercially purchased

RAS Forms Nanoclusters to Activate Signaling Cascade

Our KRAS4B and RASi programs are designed to modulate RAS-RAS interactions and are therefore potentially agnostic to common mutations



Jang, et al. 2020 Comput Struct Biotechnol J.



KRAS-4B
(inhibitor)

IC_{50} = 1 uM; 95% inhibition at 30 uM

Pan-Inhibitor Approach

- Inhibit formation of signalosome RAS clusters (covalent option)
- Disrupt KRAS4B activity by preventing RAS-RAS dimers

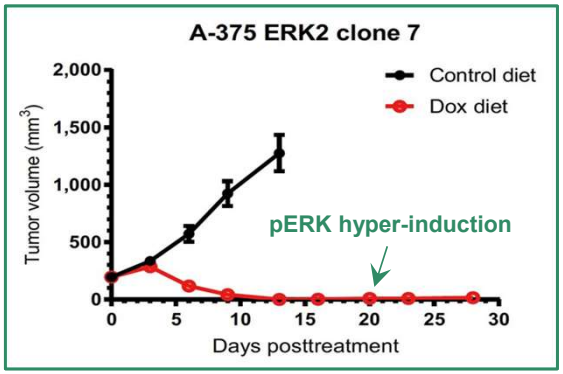
RASi
(induction)

At 10-30 uM ~ up to 844% induction

RAS Induction Approach

- Enhance RAS clusters and amplify MAPK signaling
- Leverage MAPK addiction against tumor cells

2019 Novartis



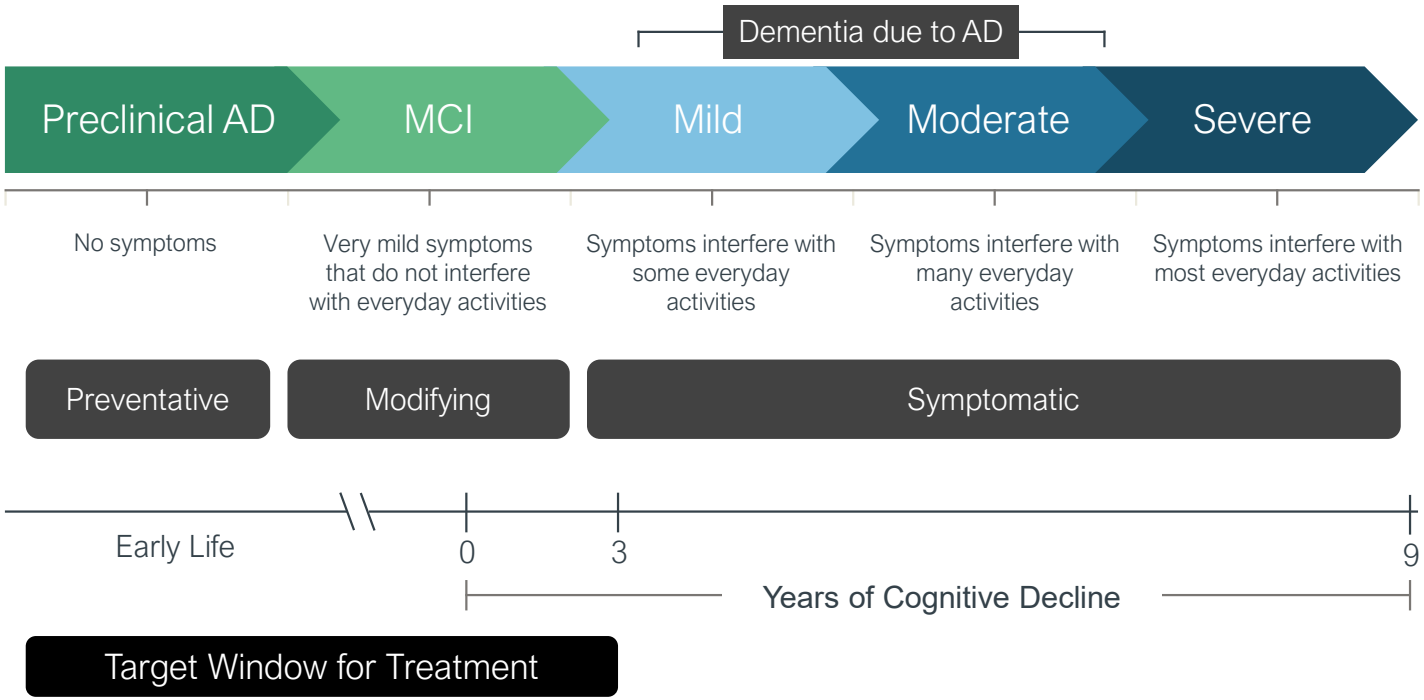
Leung, et al. 2019 Mol Can Res 17(1):199

Immuneering

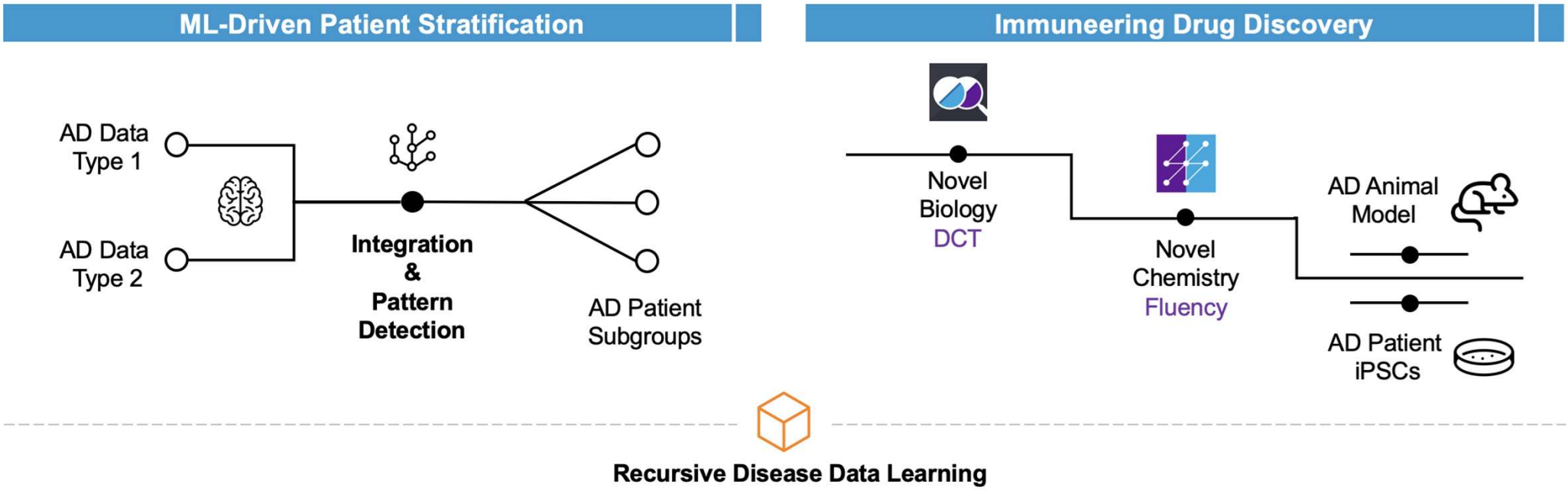
Neuroscience Pipeline

Alzheimer's Disease

Alzheimer's Disease Continuum

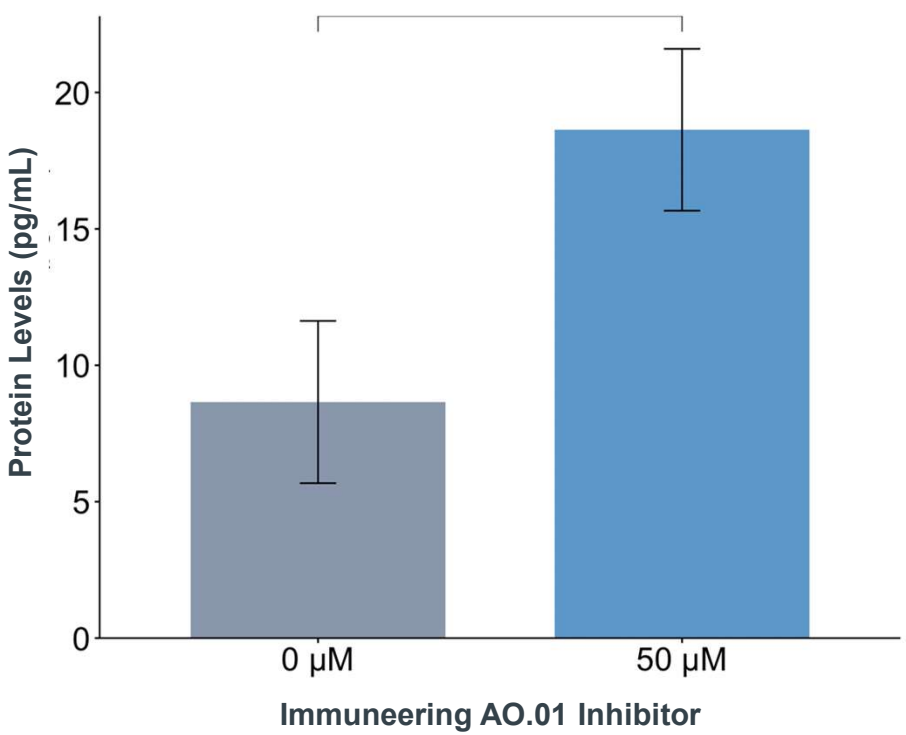


AD Patient Subgroup Stratification and Application of Our Drug Discovery Platform



In Vitro Observation of IMM-ALL-01 (AO.01 Inhibitors) Decreasing the Release of IL-6 and Promoting IL-10 Expression

A



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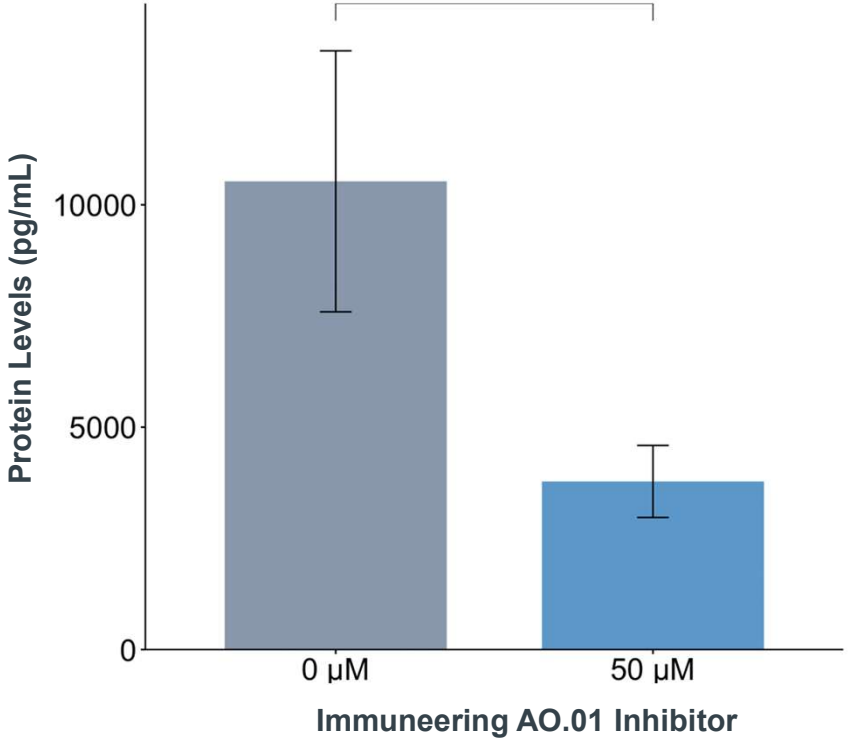
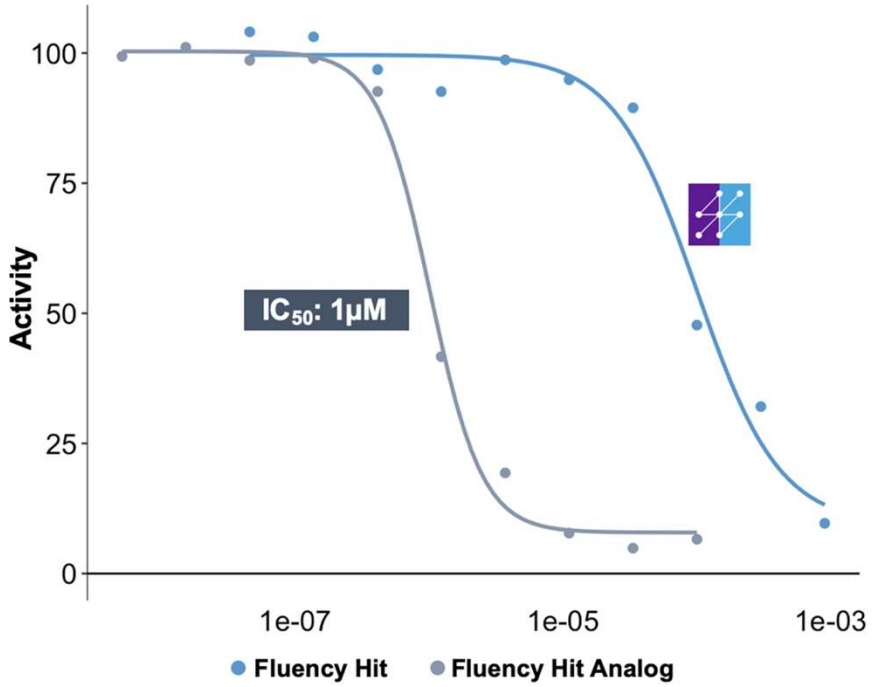
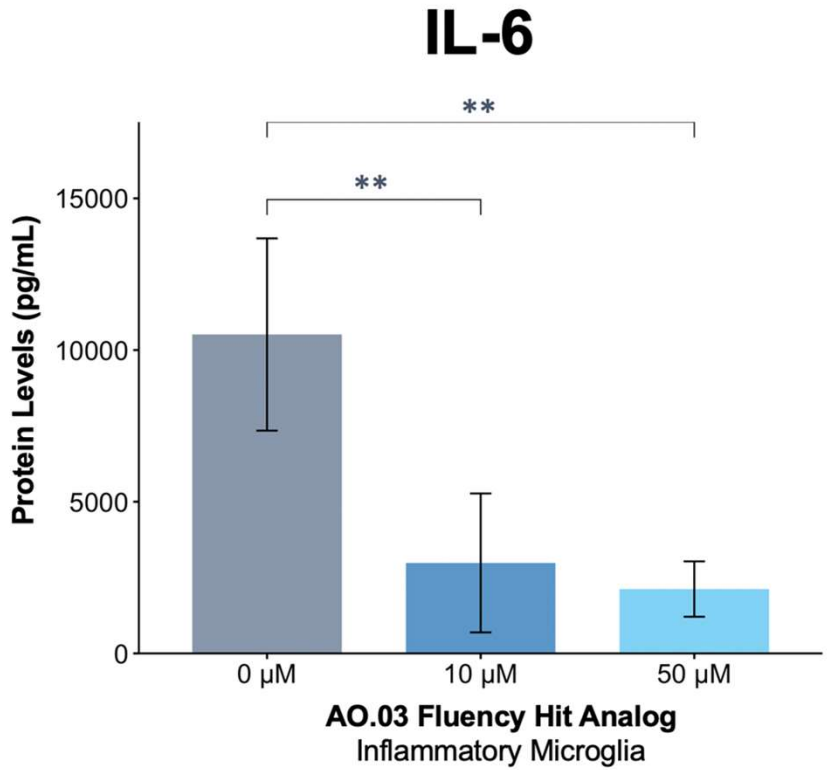


Fluency Platform Identifies Small Molecules of IMM-ALL-03 (AO.03 Inhibitors) Decreasing the Release of IL-6

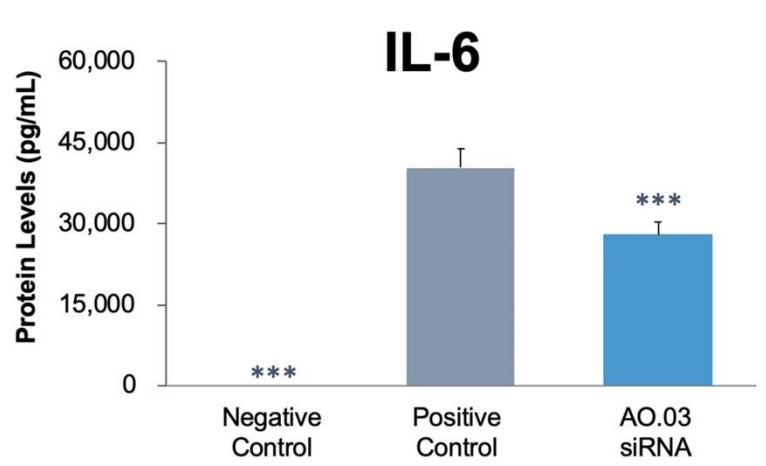


Improved Cell-Free IC_{50} for an AO.03 inhibitor, a Fluency-Identified Hit

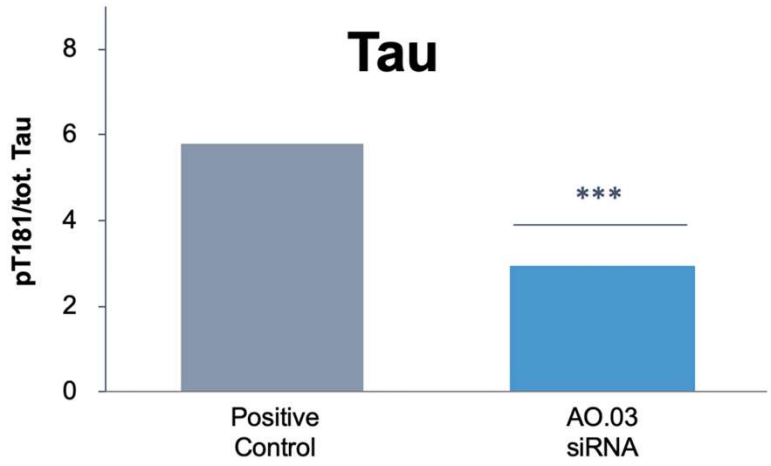


The Biological Effect of Reducing AO.03 Gene Expression on Inflammation and Tau Deposition, and Pathway Analysis of AO.01 versus AO.03

Microglia



Neuron: Human Tau



Microglia
Pathway Analysis

	AO.01 siRNA
↓	OXIDATIVE PHOSPHORYLATION
↓	PENTOSE PHOSPHATE PATHWAY

False Discovery Rate < 0.05

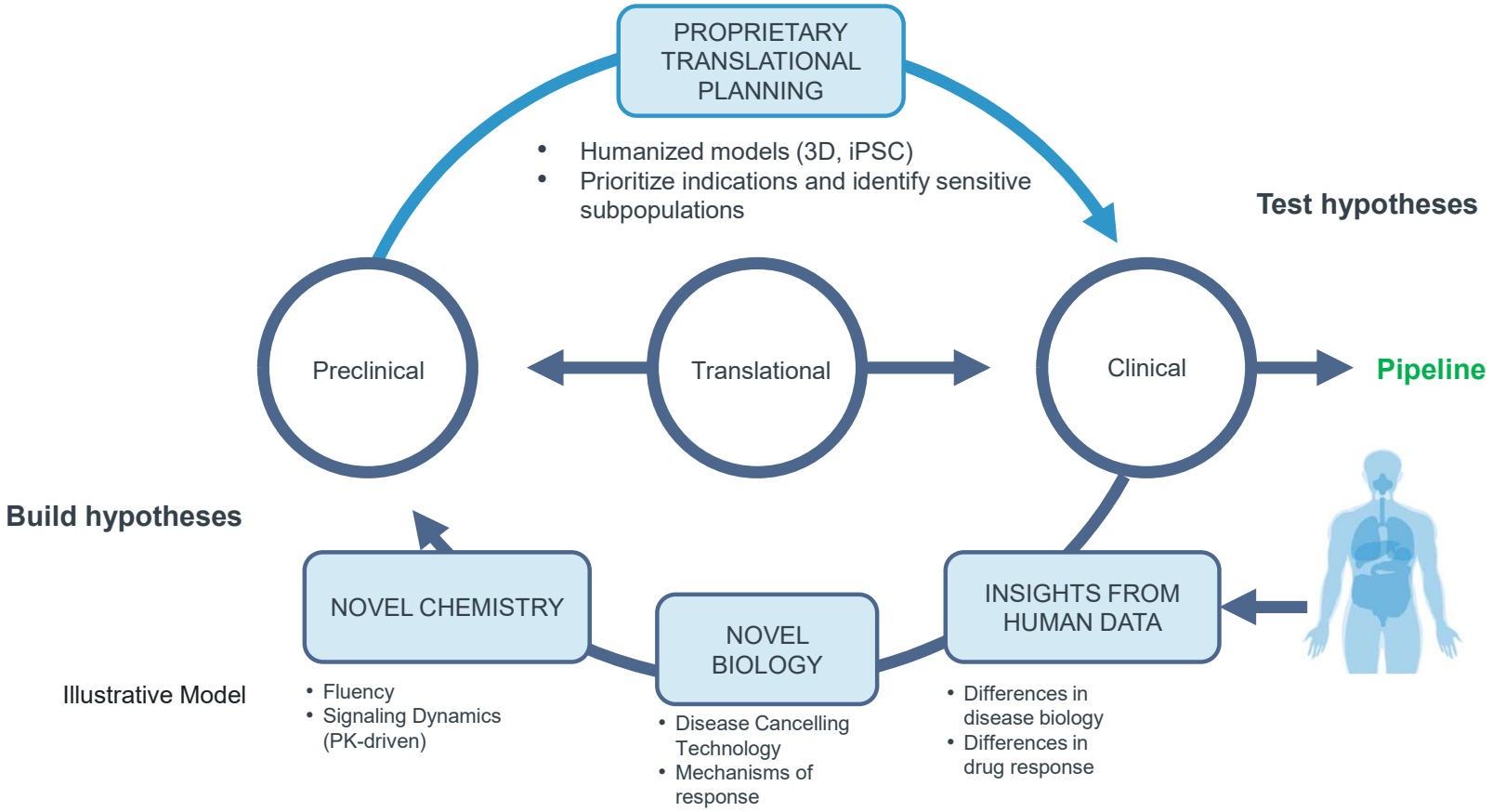
	AO.03 siRNA
↓	TOLL-LIKE RECEPTOR SIGNALING
↓	IL-6 PATHWAY

False Discovery Rate < 0.05

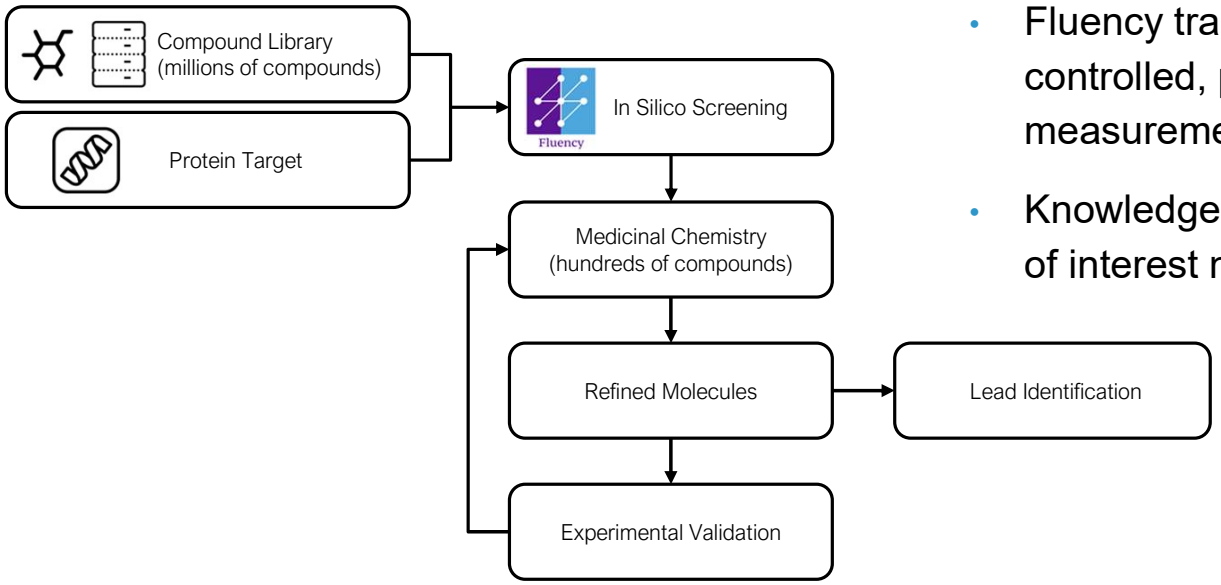
Immuneering

BioInformatics Platform

The Four Key Elements of Our Drug Development Process



Fluency's Role in Our Drug Discovery

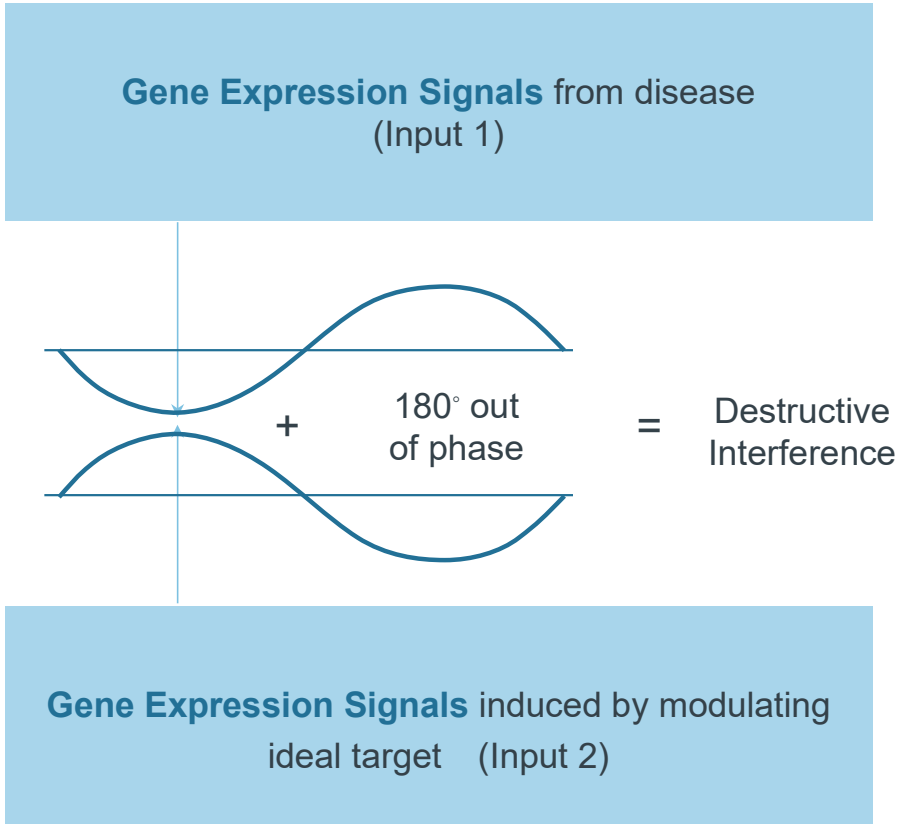


- Easy to use AI-based screening tool
- Fluency trained against millions of carefully quality controlled, publicly available binding affinity measurements
- Knowledge of the 3D protein structure for protein target of interest not required

Can Screen 10M+ Compounds in a Single Day

Workflow of Disease Cancelling Technology

- Carefully curated and QC human transcriptomic data representing a specific aspect of disease (**Input 1**) is input and vectorized for processing
- Gene expression signals associated with perturbing specific targets at specific time points and concentrations (**Input 2**) is input for processing
- Strength of disease cancellation is measured



Disease Cancelling Technology (DCT)

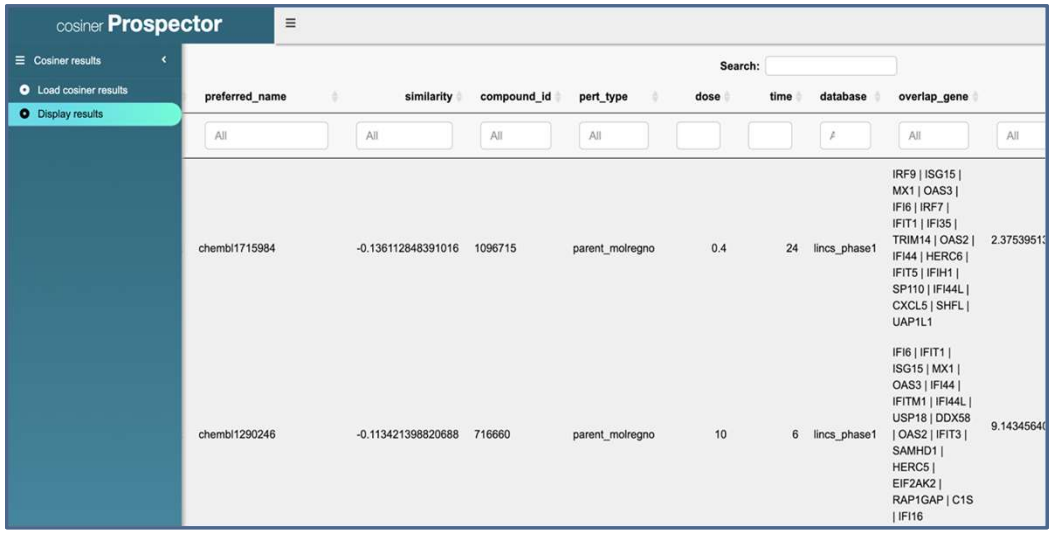
New Targets and Better Ways to Drug Existing Targets from Human Transcriptomic Data

DCT's key differentiators:

- ✓ Data Type: RNA
- ✓ Data Source: Patients (does not use literature)
- ✓ Drug Novelty: Produces new composition of matter
- ✓ Disease Areas: Positive track record in oncology, neuroscience, rare diseases

DCT's key capabilities:

- Identify novel targets
- Identify pathway contribution to disease cancelling
- MOA information (driver genes and pathways)
- Ideal target combinations
- Relevant cell types
- Orthogonal data to refine hypothesis



Financial Information

- Cash, cash equivalents and marketable securities as of June 30, 2021: $50.2 million
- July 2021 IPO with Net Proceeds of $118.3 million
- Cash Runway Into 2024 And Would Support:
  - IMM-1-104 IND Filing in Q1 2022
  - IMM-1-104 Phase 1 Trial – Plan to Initiate in 1H 2022
  - Two IND Filings in Oncology – One Each in 2023 and in 2024
  - Discovery Research in Four Additional Oncology Programs
  - Discovery Research in Neuroscience Targeting Neuroinflammation in Alzheimer's disease
- Shares outstanding (as of September 3, 2021): 25,398,064

THANK YOU!

13 Years in the Making

- **2008 Goal:** understand what is happening in responders, expand to more people (achieve broad activity)
- **Approach:** translational bioinformatics (analyzing transcriptomic, genomic, and/or proteomic data)
- **Early Years:** partnered with pharma to perform analyses of ibrutinib, ipilimumab, daratumumab, others. Built platform.
- **2018 Turning Point:** decision to use our platform to create a wholly owned pipeline

Lead Product Candidate for RAS Mutant Solid Tumors

- **IMM-1-104:** a dual-MEK inhibitor designed to target RAS mutant tumors
- **Existing RAS Inhibitors:** are limited to specific mutations (e.g., $KRAS^{G12C}$); reports of acquired resistance
- **Our Approach:** aims to treat broad patient populations durably and safely by disrupting signaling dynamics of tumors
- **IMM-1-104 Preclinical Studies:**
  - ✓ *Broad activity in RAS and RAF mutated tumor models and wide range of "humanized" 3D tumor models*
  - ✓ *Synergy with covalent $KRAS^{G12C}$ inhibitor, sotorasib (formerly known as AMG-510)[1]*
  - ✓ *Greater anti-tumor activity and improved tolerability vs selumetinib, binimetinib[1]*

..

A Robust Pipeline Enabled By Signaling Dynamics

- **Our Focus:** the MAPK and mTOR pathways, which are inappropriately activated in more than half of all cancers
- **Our Pipeline Beyond IMM-1-104:**
  - ✓ **MEK-io:** designed for immunologically "cold" solid tumors. In an animal model that does not respond to a checkpoint inhibitor alone, adding MEK-io inhibited tumor growth
  - ✓ **Trifecta-MEK:** designed for BRAF mutant tumors. A potential alternative to RAF and MEK inhibitor combinations. Displayed 10X less pERK than registered RAF or MEK inhibitors *in vitro*
  - ✓ **4** additional oncology programs
  - ✓ **2** neuroscience programs

[1]As demonstrated in head-to-head studies versus IMM-1-104 in certain tumor models