

**UNITED STATES
SECURITIES AND EXCHANGE COMMISSION**
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of
the Securities Exchange Act of 1934

Date of report (Date of earliest event reported): April 18, 2023

Immuneering Corporation
(Exact name of Registrant as Specified in its Charter)

Delaware
(State or other jurisdiction
of incorporation or organization)

001-40675
(Commission
File Number)

26-1976972
(I.R.S. Employer
Identification No.)

245 Main St.
Second Floor
Cambridge, MA 02142
(Address of principal executive offices) (Zip Code)

(617) 500-8080
(Registrant's telephone number, include area code)

N/A
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

- Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Title of each class
Class A Common Stock, par value \$0.001 per share

Securities registered pursuant to Section 12(b) of the Act:
Trading Symbol(s)
IMRX

Name of each exchange on which registered
The Nasdaq Global Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act.

Item 2.02 Results of Operations and Financial Condition.

The information under the heading “Preliminary Financial Information” set forth under Item 8.01 of this Current Report on Form 8-K (this “Current Report”) is incorporated by reference into this Item 2.02.

Item 7.01 Regulation FD Disclosure.

On April 18, 2023, Immuneering Corporation (“we”, “our” and “us”) posted an updated corporate slide presentation in the “For Investors” portion of its website at www.immuneering.com. A copy of the slide presentation is furnished as Exhibit 99.1 to this Current Report.

The information in this Item 7.01 of this Current Report is intended to be furnished and shall not be deemed “filed” for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the “Exchange Act”), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended (the “Securities Act”), or the Exchange Act, except as expressly provided by specific reference in such a filing.

Item 8.01 Other Events.**Recent Developments**

On April 18, 2023, we announced initial pharmacokinetic (“PK”), pharmacodynamic (“PD”) and safety data from the Phase 1 portion of our Phase 1/2a clinical trial of IMM-1-104 in patients with advanced solid tumors harboring RAS mutations.

As of April 10, 2023, we had PK, PD and safety data from four patients with pancreatic or colorectal cancer available for evaluation. Of these patients, we dosed one patient at 40 mg once daily oral dose, or the first dose level, one patient at 80 mg once daily oral dose, or the second dose level, and two patients at 160 mg once daily oral dose, or the third dose level. At the third dose level, we observed significant PK C_{max} levels, which is the plasma concentration of therapy in a specific area of the body, with IMM-1-104 of over 2,000 ng/mL or approximately 1 uM drug free-fraction. In addition, we observed greater than 90% PD inhibition of phosphorylated extracellular signal-regulated kinase (pERK) with IMM-1-104 compared to pretreatment baseline for patients at the third dose level. A median plasma half-life of 1.94 hours was observed with IMM-1-104 across the first three dose levels evaluated in patients with pancreatic and colorectal cancer with different RAS mutations, including KRAS-G12D, the most common mutation present in pancreatic cancer. IMM-1-104 was well tolerated in these four patients, as well as one patient dosed at 320 mg once daily oral dose, or the fourth dose level, with no dose limiting toxicities or serious adverse events observed and no drug-related adverse events beyond Grade 1 observed.

Based on this initial data, we plan to announce the recommended Phase 2 dose in early 2024 instead of our prior guidance of mid-2024.

The full text of the press release issued in connection with the announcement is furnished as Exhibit 99.2 to this Current Report.

Preliminary Financial Information

Based upon preliminary estimates and information available to us as of the date of this Current Report, we expect to report that we had approximately \$91.5 million of cash and cash equivalents and marketable securities as of March 31, 2023. Based on our current plans, we expect that our existing cash and cash equivalents and marketable securities will enable us to fund our operating expenses and capital expenditure requirements into the fourth quarter of 2024.

We have not yet completed our financial close process for the three months ended March 31, 2023. This estimate of our cash and cash equivalents and marketable securities as of March 31, 2023 is preliminary and is subject to change upon completion of our financial statement closing procedures. There can be no assurance that our final cash position as of March 31, 2023 will not differ from this estimate, including as a result of adjustments as a result of our review, and any such change could be material. Our independent registered public accountants have not audited, reviewed or performed any procedures with respect to such preliminary financial data and accordingly do not express an opinion or any other form of assurance with respect to such data. Complete results will be included in our Quarterly Report on Form 10-Q for the three months ended March 31, 2023.

Forward-Looking Statements

This Current Report contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements contained in this Current Report that do not relate to matters of historical fact should be considered forward-looking statements, including, without limitation, statements regarding our cash and cash equivalents and marketable securities as of March 31, 2023, the sufficiency of our existing cash and cash equivalents and marketable securities, the treatment potential of IMM-1-104, the design, timing, enrollment criteria and conduct of the Phase 1/2a clinical trial, and the ability of initial clinical data to de-risk IMM-1-104 and be confirmed as the study progresses, including the safety, tolerability, pharmacokinetics, pharmacodynamics and potential efficacy of IMM-1-104.

These forward-looking statements are based on our current expectations and are subject to inherent uncertainties, risks and assumptions that are difficult to predict. Factors that could cause actual results to differ include, but are not limited to, the risks inherent in oncology drug development, including clinical trials, and risks related to financial reporting.

These and other important factors discussed under the caption "Risk Factors" in our Annual Report on Form 10-K for the year ended December 31, 2022 and filed with the U.S. Securities and Exchange Commission (the "SEC") on March 6, 2023 and our other reports filed with the SEC could cause actual results to differ materially from those indicated by the forward-looking statements made in this Current Report. Any such forward-looking statements represent management's estimates as of the date of this Current Report. While we may elect to update such forward-looking statements at some point in the future, we disclaim any obligation to do so, even if subsequent events cause our views to change. These forward-looking statements should not be relied upon as representing our views as of any date subsequent to the date of this Current Report.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Immuneering Corporation Corporate Slide Presentation as of April 18, 2023
99.2	Press release issued on April 18, 2023
104	Cover Page Interactive Data File (embedded within the inline XBRL document)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

IMMUNEERING CORPORATION

Date: April 18, 2023

By: /s/ Benjamin J. Zeskind
Name: Benjamin J. Zeskind, Ph.D.
Title: Co-Founder, President, Chief Executive Officer

Investor Presentation



Nasdaq: IMRX

APRIL 2023



Building a Universal-F Franchise

FORWARD-LOOKING STATEMENTS AND OTHER DISCLAIM

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. All statements contained that do not relate to matters of historical fact should be considered forward-looking statements, including, without limitation, statements regarding the Co equivalents and marketable securities as of March 31, 2023. In addition to the Company's plans to develop, manufacture and commercialize its product cand potential of IMM-1-104, the design, enrollment criteria and conduct of the Phase 1/2a clinical trial, the translation of preclinical data into human clinical data, clinical data to de-risk IMM-1-104 and be confirmed as the study progresses, including the safety, tolerability, pharmacokinetics, pharmacodynamics and po 1-104; the potential advantages and effectiveness of the company's clinical and preclinical candidates, the timing of additional trial updates, recommended additional safety data, the indications to be pursued by the Company in the Phase 2a portion of the study, the timing of submission of the IND for IMM-6-41 approval by, regulatory authorities of our product candidates, the sufficiency of funds to operate the business of the Company, statements regarding the Cc advance its pipeline and further diversify its portfolio and make progress towards its longstanding goal of creating better medicines for cancer patients, the needs and availability, including our revenue streams, projected cash runway and current operating plans, and the plans and objectives of management for

These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including, without limitation: our limited operating hist operating losses; our ability to raise the substantial additional capital that will be required to finance our operations; the difficulty of obtaining regulatory ap current or future product candidates; our ability to submit an Investigational New Drug application ("IND"), or IND amendments or comparable documents in order to commence clinical trials on the timelines we expect; our limited experience in designing and conducting clinical trials; the timing of the initiatio potential results of our ongoing and planned preclinical studies and clinical trials and our research programs, including our Phase 1/2a clinical trial; our abili complete our Phase 1/2a clinical trial, or any planned clinical trials and for those trials to produce positive results; the risk of substantial delays in completing development and commercialization of our current or future product candidates; risks related to adverse events, toxicities or other undesirable side effects or future product candidates; the risk of delays or difficulties in the enrollment and/or maintenance of patients in clinical trials; our substantial reliance on th development of our current and future product candidates, as well as our platform, including our proprietary technologies such as DCT and Fluency; risks re in our industry; the market opportunity for our product candidates, if approved; risks related to manufacturing; risks related to our reliance on third parties; intellectual property; and risks related to the pandemic related to COVID-19, its variants and future pandemics.

These and other important factors discussed under the caption "Risk Factors" in the Company's Annual Report on Form 10-K for the year ended December SEC and its other reports filed with the SEC could cause actual results to differ materially from those indicated by the forward-looking statements made in t such forward-looking statements represent management's estimates as of the date of this presentation. While the Company may elect to update such forw statements at some point in the future, it disclaims any obligation to do so, even if subsequent events cause its views to change. These forward-looking stat relied upon as representing the Company's views as of any date subsequent to the date of this presentation.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and other data about ou involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data and estimates. In addition, projections, ass estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertaint nor our affiliates, advisors or representatives makes any representation as to the accuracy or completeness of that data or undertake to update such data af presentation.

Data of trametinib, cobimetinib, binimetinib, selumetinib, encorafenib and AMG-510 (now known as sotorasib) as compared to IMM-1-104 presented in this j on head-to-head studies where these therapies have been purchased from commercial sources rather than the pharmaceutical company commercializing applicable, the compound.

Differentiated Approach

- Targeting **Universal-RAS** patient population versus limited single mutation targeted approaches
- **Once-Daily Oral Dosing**
- **Deep cyclic inhibition** targeted, based on:
 - **Manyfold higher C_{MAX}**
 - **and short half-life**
- Approach designed to **spare healthy cells** and potential to **limit adaptive resistance**
- **Monotherapy-Focused** initially, with combination potential

IMM-1-104 Demonstrated Universal-RAS Potential

- **Robust preclinical activity** observed in:
 - Pancreatic Cancer (KRAS^{G12C} & G12V)
 - NSCLC (KRAS^{G12S})
 - CRC (KRAS^{G12D})
 - Melanoma (NRAS^{Q61R})
 - And others
- Hypothesis for IMM-1-104 from **proprietary model** that identified counterintuitive and novel deep cyclic inhibition approach
- **Validated using** proprietary **bioinformatics & 3D tumor growth assays**

Key Inflection Expected in Ne

- **Initial Phase 1 PK, data support profile** 104 believed to be **Deep Cyclic Inhibi**
- **Investigator enth** broad inclusion crit
- **Additional trial up** **expected on a per** **RP2D** expected in e
- **IMM-6-415** IND exp 2023
- **Cash runway proje** **Q4 2024**

Development Pipeline

Wholly Owned Product Portfolio Differentiated by Indication and Half-life

PROGRAM	INDICATION	DISCOVERY	IND-ENABLING	PHASE 1	PHASE 2	PHASE 3	ANTI-MILI
IMM-1-104	Universal-RAS	Once Daily (QD)					Phase Enrol expected
IMM-6-415	Universal-MAPK	Twice Daily (BID)					IND filin in c
Multiple additional programs in discovery phase							

Preliminary estimated Cash, cash equivalents and marketable securities as of March 31, 2023 of \$91.5M* projected to fund operations into Q4 2024

*Our actual consolidated financial results as of March 31, 2023 are not yet available. Our financial closing procedures for the first quarter ended March 31, 2023 are not yet complete and, as a result, our final results upon completion of those procedures may differ materially from our preliminary estimates. The preliminary consolidated financial data presented above as of March 31, 2023 is not a comprehensive statement of our financial position or operating results and reflects our preliminary estimates based on information available as of the date of this presentation; and is subject to change, and those changes may be material. Accordingly, you should not place undue reliance upon these preliminary estimates.

IMM-1-104

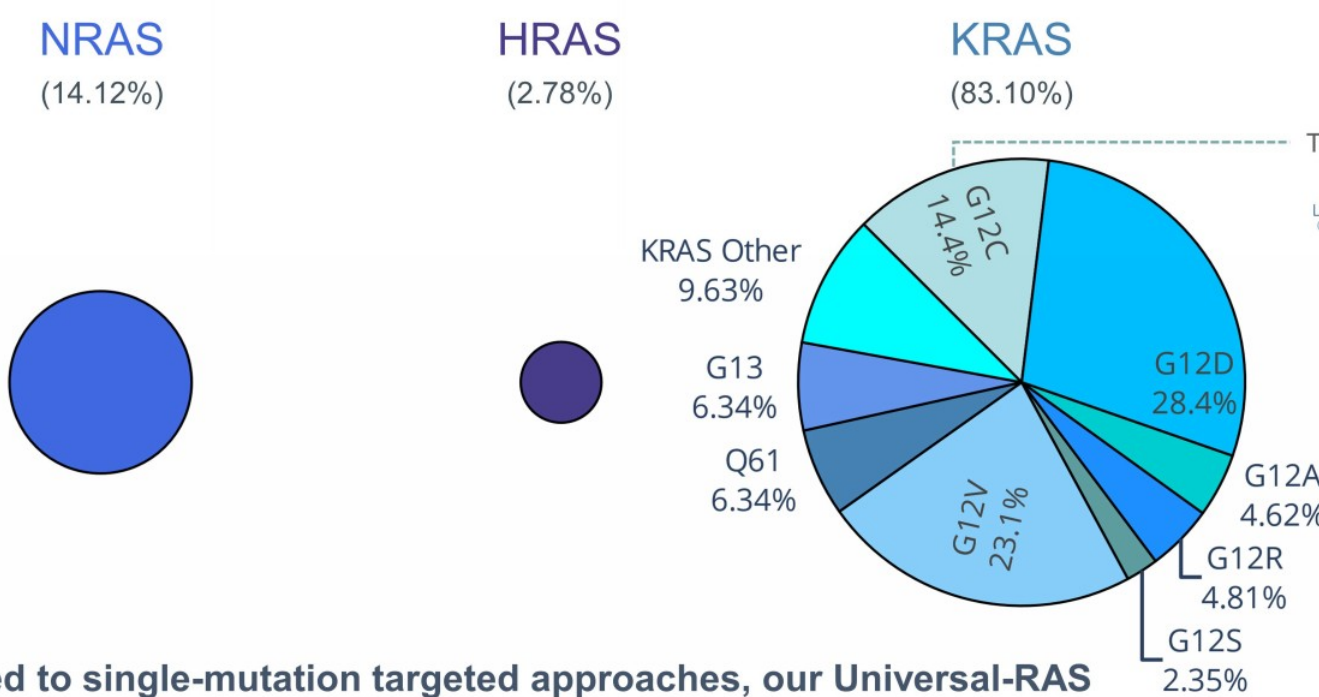
 Immuneering



IMM-1-104 Target Profile

- **Potential for Universal-RAS Activity**
 - **Deep Cyclic Inhibition; Once-Daily Oral Dosing**
 - **Short plasma half-life to minimize drug trough**
 - **Initially being developed as Monotherapy**
 - **Designed to Resist CRAF-bypass in RAS mutant tumors**
- Phase 1 recruiting all-comer RAS solid tumors: [NCT05585320](https://clinicaltrials.gov/ct2/show/study/NCT05585320)
 - First patient dosed November 2022
 - Initial Phase 1 PK, PD and safety data reported at AACR 2023

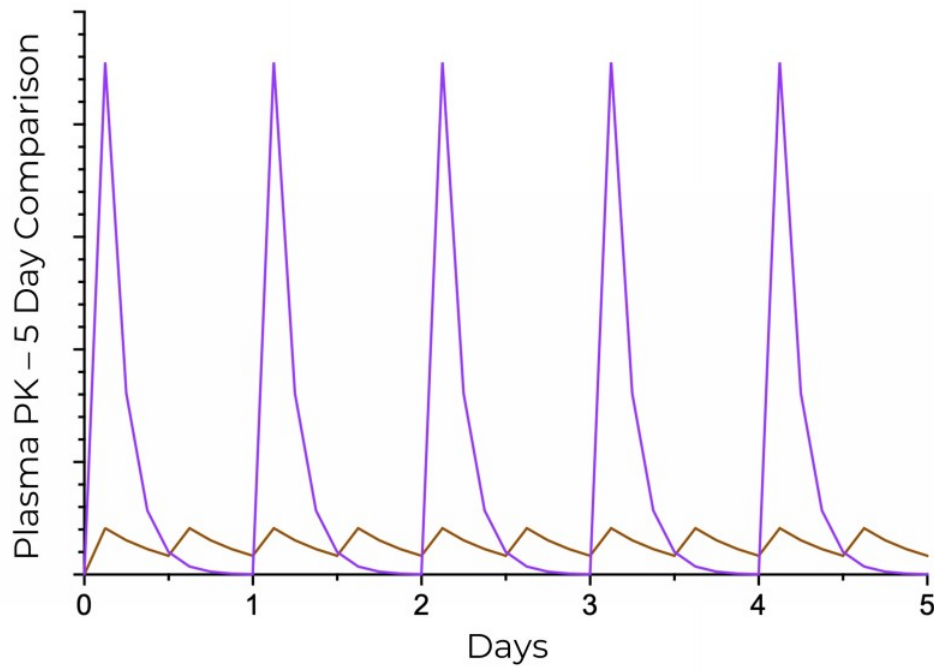
IMM-1-104's Potential: Universal-RAS



Compared to single-mutation targeted approaches, our Universal-RAS approach potentially addresses a much larger patient population

- Based on 27,461 out of 148,268 (18.5%) patients with RAS-mutated tumors in the AACR GENIE database, v13.0
- Percentage of overall mutation shown under RAS paralogue. Select mutation percentages within KRAS are shown

Deep, Cyclic Inhibition



Conceptual illustration of deep cyclic inhibition (purple) vs. chronic pathway ablation (brown)

Dramatic PK C_{MAX}

GOAL: Achieve many fold higher fraction C_{MAX} to **break tumor**

Near-Zero Drug T

GOAL: Short plasma half-life, tolerability and limit adaptive **every day is a drug holiday**

MoA Target Engag

GOAL: Prevent MAPK-pathway events, for **expanded activity mutant setting**

Initial Phase 1 PK, PD and Safety Data for IMM-1-104

First demonstration of novel deep cyclic inhibition mechanism in humans

Deep Cyclic Inhibition (DCI)

Initial pharmacokinetic (PK), pharmacodynamic (PD) and safety data support profile for IMM-1-104 believed to be necessary for Deep Cyclic Inhibition (DCI)

Significant PK C_{MAX}

Significant levels of PK C_{MAX} observed (~1 μ M free fraction C_{MAX} at 160 mg QD p.o. doses)

Short Half-life

Observed IMM-1-104 approximately 2 hours, Immuneering's preclin

Safety Data

IMM-1-104 well tolerated with no dose-limiting toxicities (DLTs) or serious adverse events (SAEs) observed

Trial Timeline Acceleration

Potentially therapeutic doses of IMM-1-104 now likely to be reached earlier than previously planned due to acceleration of trial timeline

RP2D

Recommended Phase 2 expected in ea

Clinical data timeline reported through April 10th, 2023 (i.e., ~20 weeks since first patient dosed)

Patient Status Summary for IMM-1-104 Phase 1 Dose Escalation

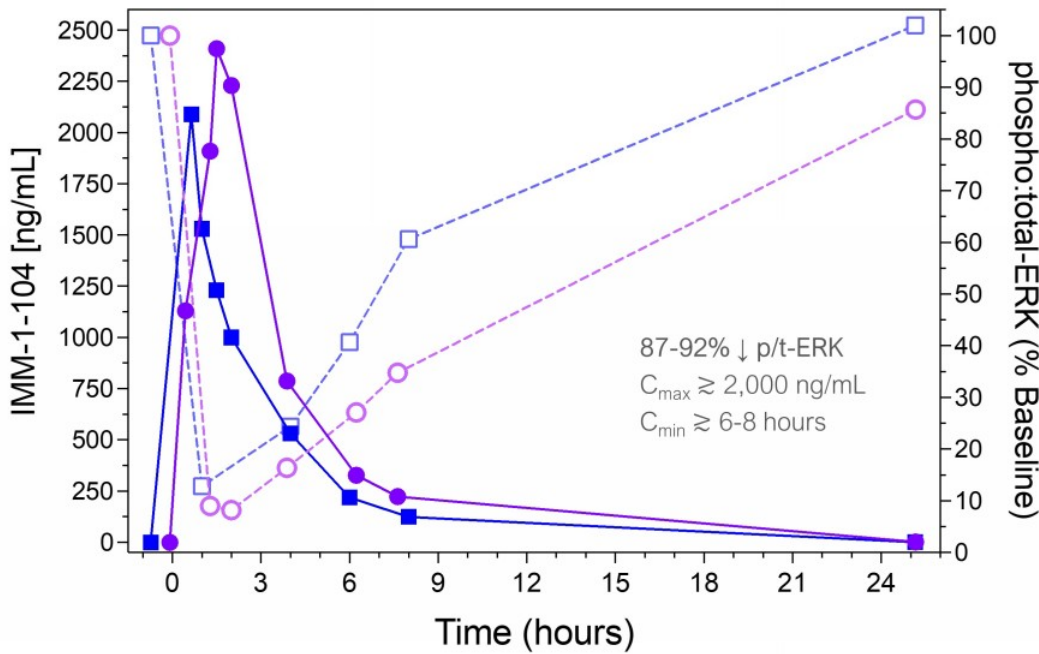
#	Patient	RAS Mutation	Dose Level	Dose	C1D1 (t _{1/2})	C1D15 (t _{1/2})	Mean (t _{1/2})	II
1.	PANCREATIC	KRAS-G12D	I	40 mg QD p.o.	1.82 hours	2.10 hours	1.96 hours	
2.	COLORECTAL	KRAS-G12V	II	80 mg QD p.o.	1.41 hours	1.43 hours	1.42 hours	
3.	COLORECTAL	NRAS-Q61L	III	160 mg QD p.o.	2.04 hours	1.83 hours	1.94 hours	
4.	COLORECTAL	NRAS-Q61K	III	160 mg QD p.o.	1.91 hours	1.97 hours	1.94 hours	
5.	PANCREATIC	KRAS-G12D	IV	320 mg QD p.o.	t.b.d.	t.b.d.	t.b.d.	

Clinical data timeline reported through April 10th, 2023 (i.e., ~20 weeks since first patient dosed)

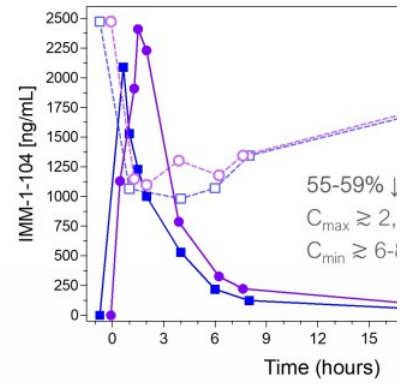
- **No DLTs or SAEs observed;** No drug-related AEs beyond grade 1 have been reported in dose escalation
- Early PK data were approximately dose linear with no drug accumulation
- **Actively enrolling patients at 320 mg QD p.o. with 2 additional patients already consented** (Pancreatic and KRAS-G12S Colorectal)

~ 90% Pharmacodynamic Inhibition of pERK

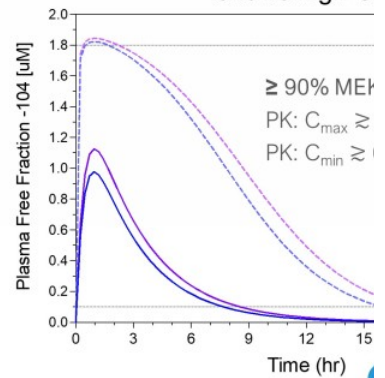
IMM-1-104 PK/PD (pERK) shows MAPK pathway suppression



IMM-1-104 PK/PD (pME suppression of path)



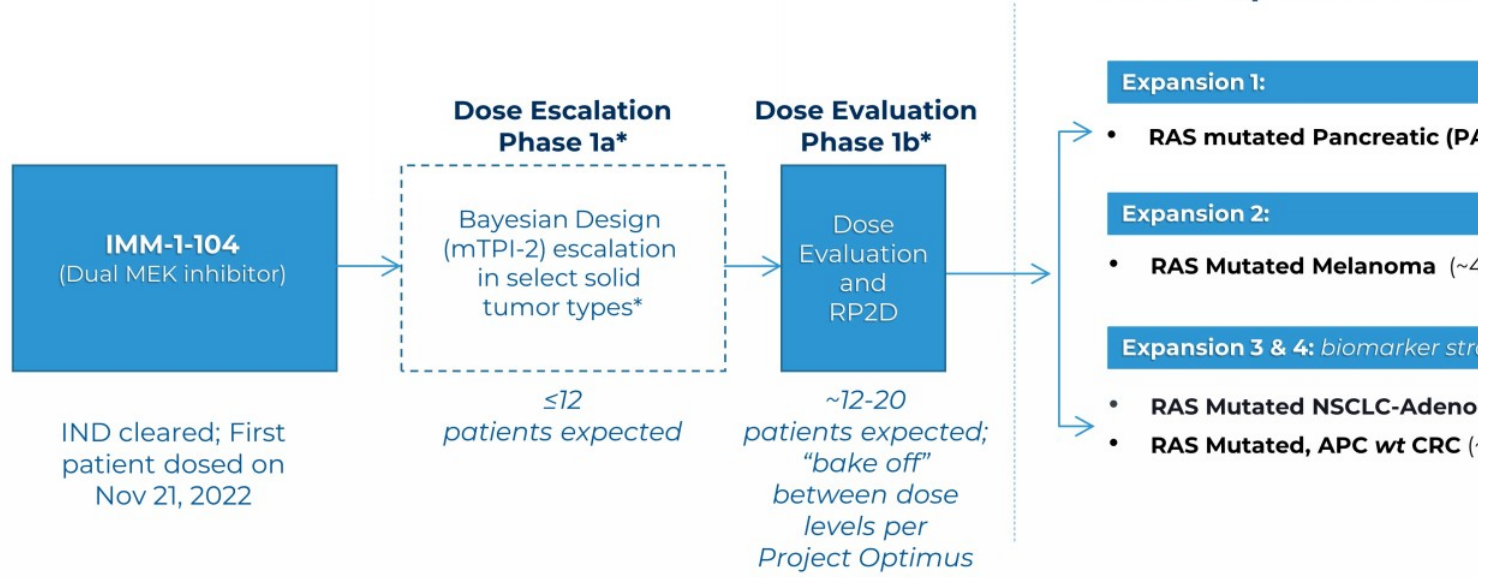
Modeled PK/BK, Drug Free shows high C



- Dose Level III: Cycle 1 Day 1 (CID1) PK (solid), BK (dotted), and PD (dotted) for patient 3 (purple) and 4 (blue), both at 160 mg QD p.o.
- PK plasma: pre-dose (0), 0.5, 1, 1.5, 2*, 4, 6, 8, 24 hours; PD plasma: PK-matched without 0.5 or 1.5 hours (*poor sample quality for Pt.4 at 2 hour)
- PD method: A549 (KRAS^{G12S}) cells were exposed to patient plasma for 2-hours before quantifying phosphorylated and total ERK and MEK

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

First Time in Humans



* Solid tumor, all come with evidence of RAS mutation

** Simon 2-Stage Design. Proposed tumor types may change based upon preclinical PGx studies and clinical function review

• RP2D = Recommended Phase 2 Dose
• PGx = Pharmacogenomics

Phase 1 Sites

ClinicalTrials.gov Identifier: NCT05585320 -----

 United States, California	City of Hope › Duarte, California, United States, 91010 › Principal Investigator: Vincent Chung, MD	
 United States, New York	MD Weill Cornell Medicine › New York, New York, United States, 10021 › Principal Investigator: Anna Pavlick, DO	
 United States, Texas	MD Anderson Cancer Center › Houston, Texas, United States, 77030 › Principal Investigator: Shubham Pant, MD	NEXT Oncology › San Antonio, Texas, United States. 78229 › Principal Investigator: David Sommerhalder,
 United States, Virginia	NEXT Oncology › Fairfax, Virginia, United States, 22031 › Principal Investigator: Alex Spira, MD, PhD	

- Investigator enthusiasm remains high
- Broad RAS inclusion criteria (Solid Tumors, All Histologies)

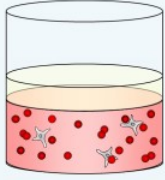
IMM-1-104
Preclinical Data



IMM-1-104 Demonstrated Universal-RAS Potential

132 Tumor Models

75 = RAS Mutant



Humanized
3D-TGA

Kolitz, et al. 2023
AACR: Targeting RAS
Philadelphia, PA

Tissue	Response #	Non-Response #
Pancreatic †	17	2
Melanoma †	22	0
Lung †	19	6
CRC	20	5
Thyroid	6	1
Soft Tissue	2	1
Breast	2	6
Gastric	4	2
Ovary	3	2
Prostate	1	2
Fibrosarcoma	1	0
Liver	4	2
Neuroblastoma	1	1
Total	102 (77.3%)	30 (22.7%)

RAS, RAF mutation	Response #
NRAS G12	2
NRAS G13	1
NRAS Q61	17
KRAS A146	1
KRAS G12	36
KRAS G13 ^	3
KRAS Q61	3
HRAS G13 *	1
BRAF (Class I or II)	21
Total	85 (85.0%)

RAS, RAF mutation	Response #
Not Present	17
Total	17 (53.1%)

^ 1 model also bearing KRAS Q61 /// * 1 model als

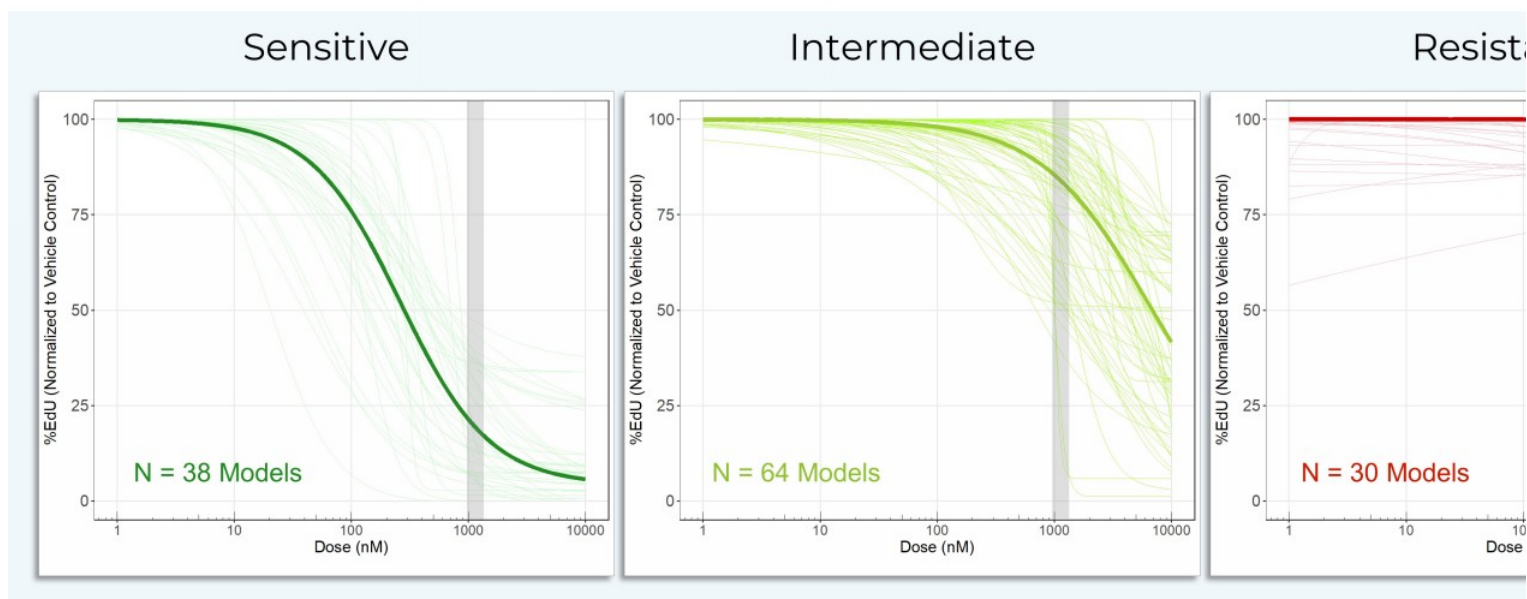
Response to IMM-1-104 based on 3D-TGA and other preclinical modeling. Parallel translational efforts are focused on projecting patient-aligned molecular profiles or 'Targetability'.

Models tested in 3D-TGA were assigned responsive if dose response IC50 < 1uM (sensitive) or IC50 ≥ 1 with >25% reduction at 10uM (intermediate), and non-responsive otherwise (resistant)

† Select 3D-TGA models: (1.) Pancreatic MIA PaCa-2 (sensitive/responsive), (2.) Pancreatic Capan-2 (intermediate/responsive), (3.) Melanoma SK-MEL-2 (sensitive/responsive), (4.) Lung A549 (inte

Humanized 3D-TGA Pharmacogenomics (PGx) Model

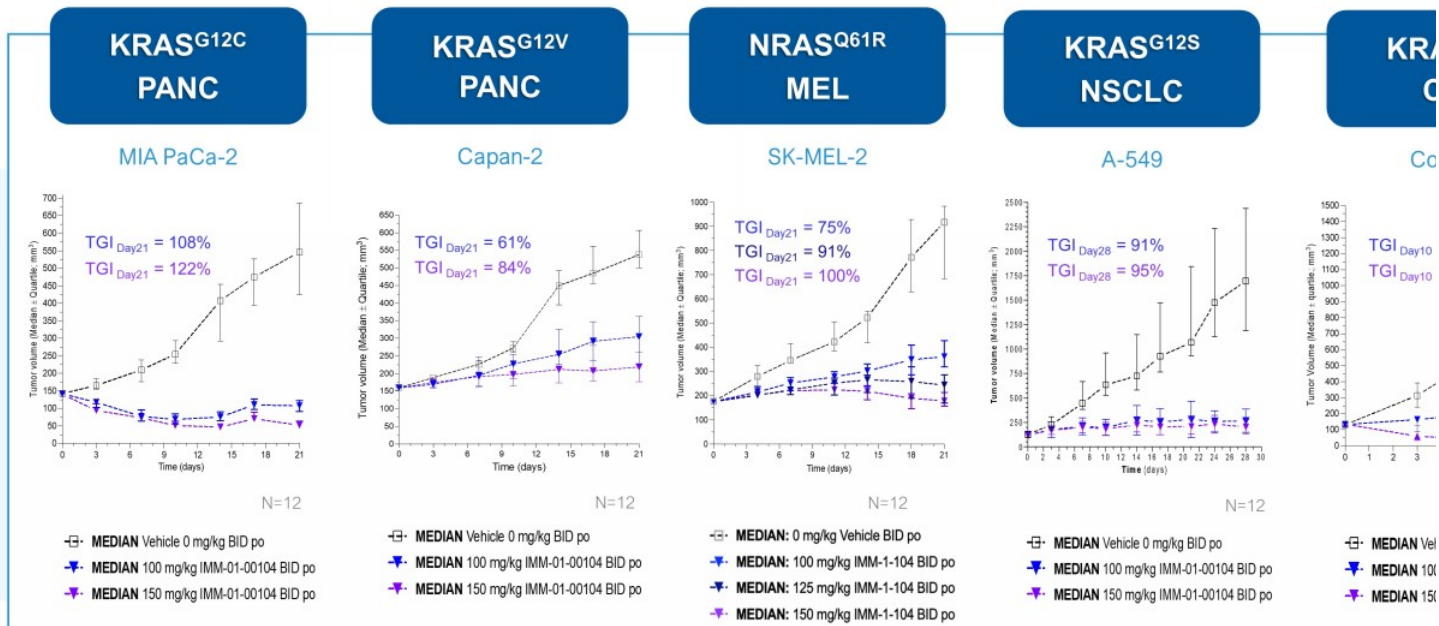
Attained Clinical Free Fraction C_{\max} of ~ 1 μM Aligns with 3D-TGA Response Categorization



- Cell lines tested in 3D-TGA (N=132) were assigned response of sensitive ($IC_{50} < 1 \mu\text{M}$), intermediate ($IC_{50} \geq 1$ and $>25\%$ reduction at $10 \mu\text{M}$), and resistant otherwise
- The dark line on each plot represents the median of the individual curves
- Vertical gray boxes match C_{\max} IMM-1-104 drug free-fractions attained in patients 3 and at 160 mg QD p.o. (both at Dose Level 3 in Phase 1 trial)

IMM-1-104 Demonstrated Universal-RAS Potential

IMM-1-104 demonstrated significant and consistent Tumor Growth Inhibition (TGI)



Well Tolerated: Median Body Weight Loss ≤ 3-6%* of baseline at top doses

Maximum Effective Dose Range in Mice (plasma $t_{1/2}$ = 1.3 hours) is 100 mg/kg to 150 mg/kg BID po

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

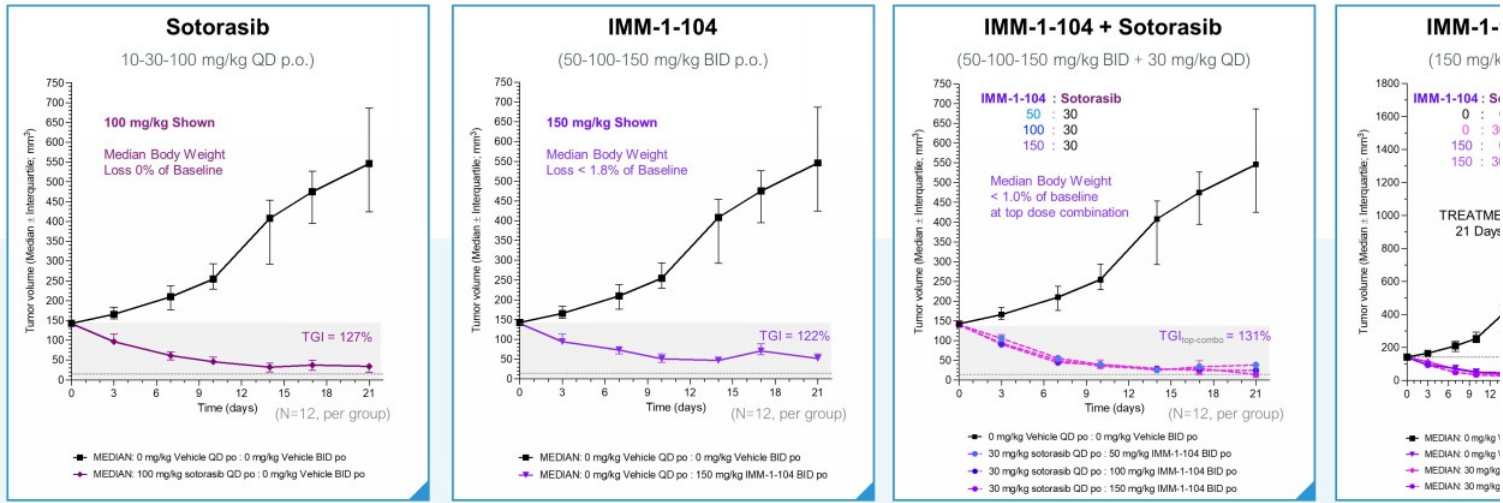
Tumor Growth Inhibition (TGI) % = $[1 - (T_i - T_0)/(C_i - C_0)] \times 100$; T = treatment groups; C = control g

*Well-tolerated at top doses with no more than 3-6% median body weight loss (BWL)

**Capan-2 PANC model, as reported at ASCO 2022 (0% TGI for sotorasib and adagrasib at top doses)

Pancreatic: Head-to-Head Comparison of IMM-1-104 +/- Sotorasib in a KRAS-G12C Pancreatic Tumor Model

IMM-1-104 as compared to sotorasib demonstrated tumor regression, both with insignificant BWL

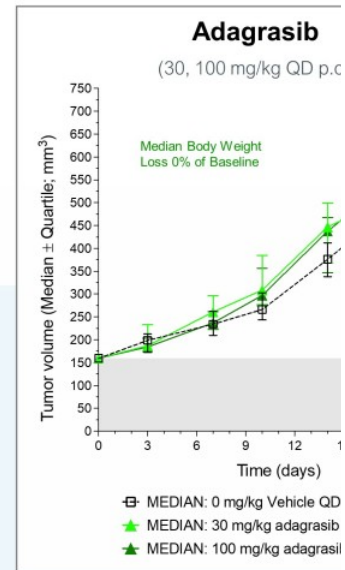
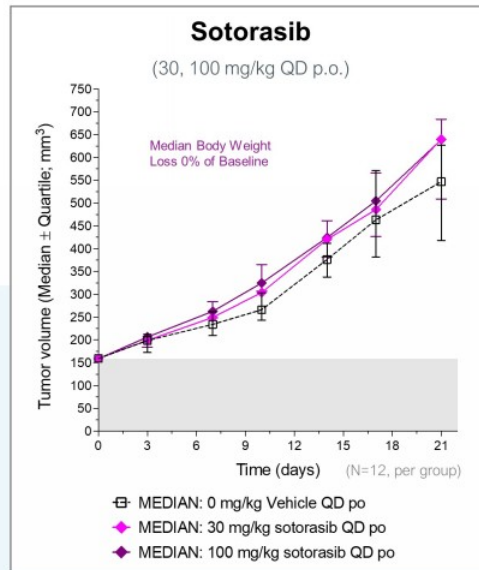
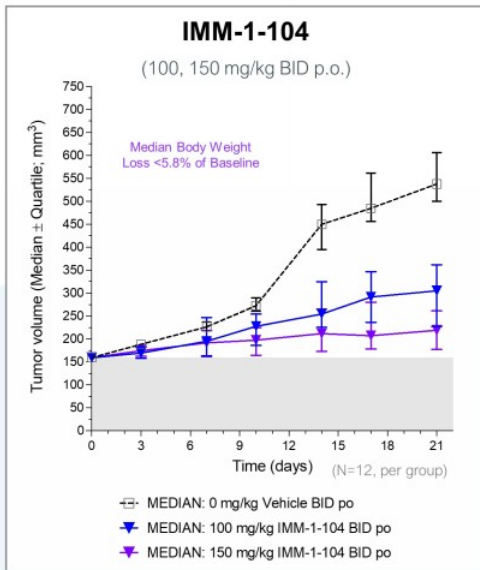


> MIA PaCa-2 (KRAS^{G12C}) Pancreatic Xenograft Tumor Model in Athymic Nude Mice

> Sotorasib was commercially purchased
Tumor Growth Inhibition (TGI) % = [1 - Expanded TGI formula vs. previous 1-

Pancreatic: Head-to-Head Comparison of IMM-1-104 vs. Sotorasib and Adagrasib in a KRAS-G12V Pancreatic Tumor Model

IMM-1-104 demonstrated tumor regression as compared to no reduction with sotorasib or adagrasib, with insignificant BWL

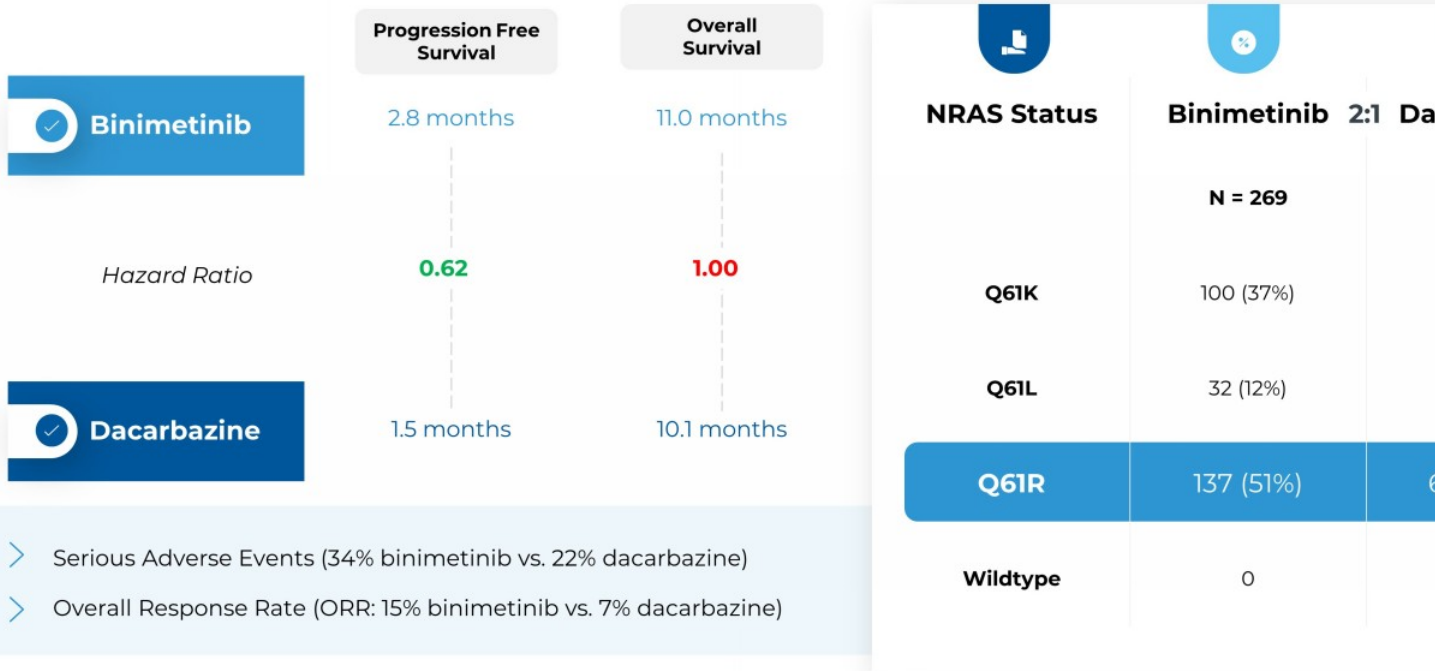


> Capan-2 (KRAS^{G12V}) Pancreatic Xenograft Tumor Model in Athymic Nude Mice

> Sotorasib and adagrasib were compared to vehicle. Tumor Growth Inhibition (TGI) % = [1 - (Tumor Volume / Vehicle Tumor Volume)] × 100. Expanded TGI formula vs. previous 1-

Melanoma: Phase 3 NEMO Study: Binimetinib vs. Dacarbazine (NRAS^{mut})

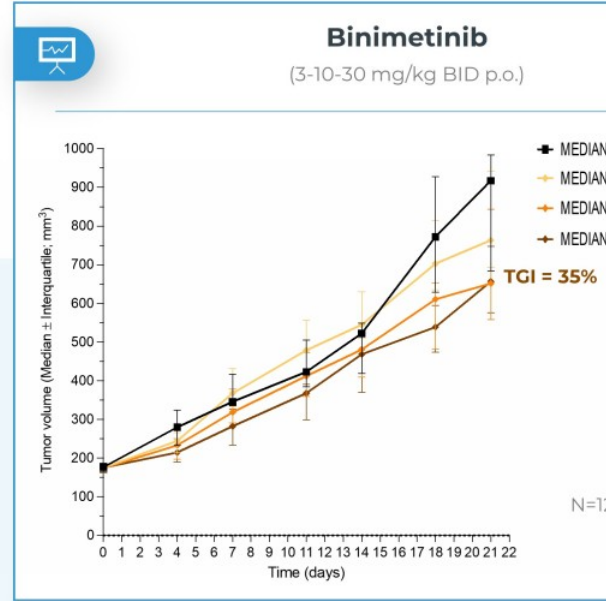
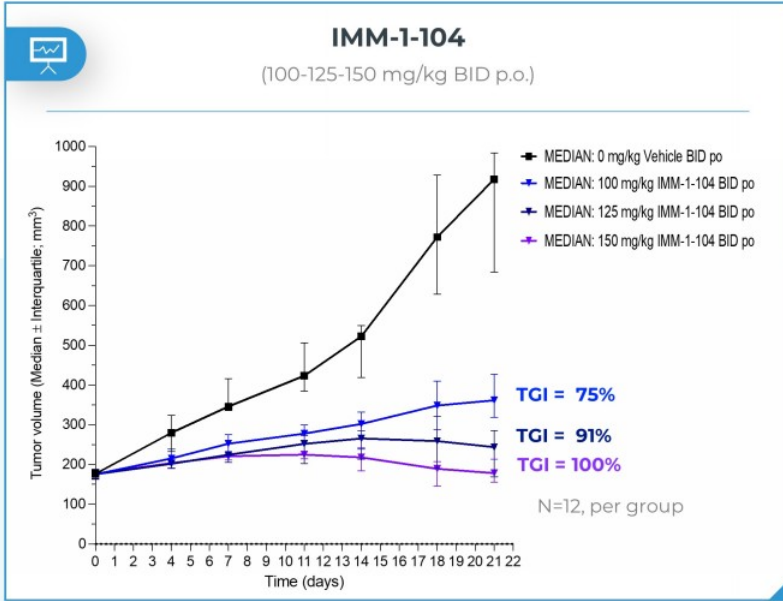
Summary of Phase 3 NEMO study of Binimetinib as reported in Lancet (c.2017) - [a potential opportunity for I](#)



Melanoma: Head-to-Head NRAS-Q61R Melanoma Xenograft Study

Binimetinib vs. IMM-1-104 in SK-MEL-2

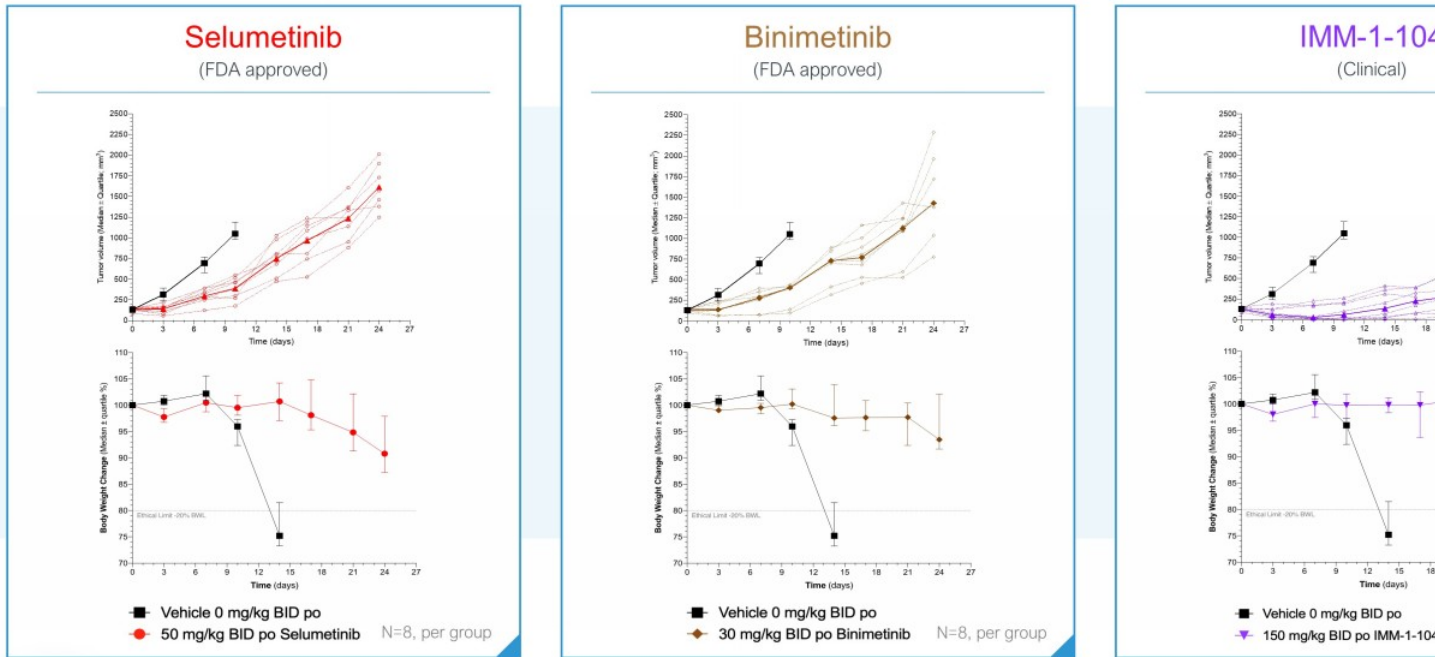
IMM-1-104 as compared to binimetinib monotherapy demonstrated greater tumor growth inhibition (TGI)



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

CRC: Head-to-Head Comparison of IMM-1-104 Against Selumetinib and Binimetinib in a KRAS-G12D CRC Syngeneic Mouse Tumor Model

IMM-1-104 demonstrated greater tumor growth inhibition (TGI), lower body weight loss (BWL) and greater durability via reductions in tu



Colon-26 (KRAS-G12D) Syngeneic Colorectal Tumor Model in Balb/c Mice

> Selumetinib and binimetinib were commercially purchased
 Tumor Growth Inhibition (TGI) % = $1 - \frac{(T - T_0)}{(C - C_0)} \times 100\%$; Expanded
 $1 - [T/C] \times 100\%$ method

IMM-6-415

 Immuneering



IMM-6-415: Monotherapy Activity in RAF and RAS Mutant T

**NRAS^{Q61R}
MEL**

SK-MEL-2

**BRAF^{V600E}
MEL**

A-375

**BRAF^{V600E}
MEL**

SK-MEL-28

**KRAS^{G12S}
NSCLC**

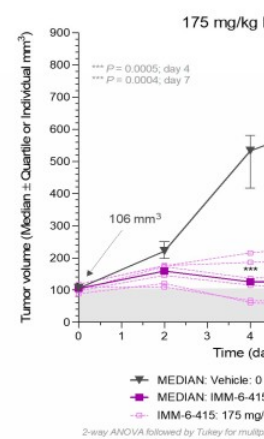
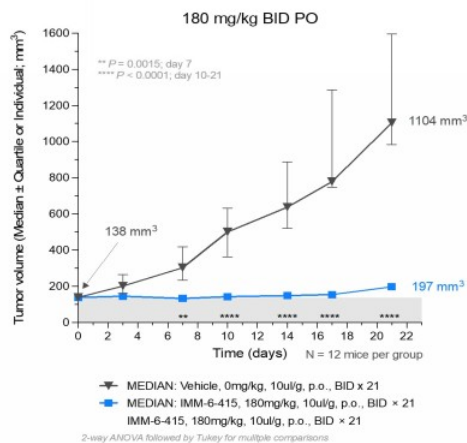
A-549

**KRAS^{G12C}
CRC**

Colon-2

Model	HRAS	NRAS	BRAF	NF1	3D-TGA
SK-MEL-2		p.Q61R			
MM127		p.G13R	p.G464E		n.t.
MM415		p.Q61L			n.t.
MEL-JUSO	p.G13D	p.Q61L			n.t.
SK-MEL-30		p.Q61K			n.t.
Hs852T		p.G12V			n.t.
MeV6				LoF	n.t.
A375			p.V600E		
SK-MEL-28			p.V600E		

3D-Tumor Growth Assay (3D-TGA) sensitivity (green)



Well Tolerated up to Maximum Monotherapy Effective Dose Range of 150 to 180 mg/kg

SITC 2022 Presentation: Maximum Effective Dose Range in Mice (plasma $t_{1/2}$ = 0.3 to 0.4 hours): 150-180 mg/kg BID CRC = colorectal cancer; NSCLC = non-small cell lung cancer;

Accelerated Cadence of IMM-6-415 Enhanced Activity of Checkpoint

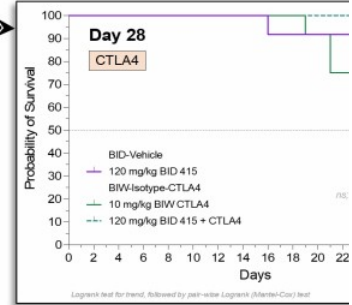
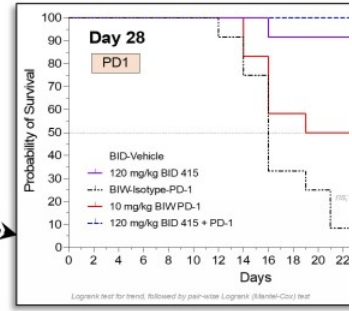
**KRAS^{G12D}
CRC**

Syngeneic CT-26 Model

Schedule	Dose (mg/kg)	Veh	αPD-1 (BIW)	αCTLA-4 (BIW)	IMM-6-415	IMM-6-415 + PD-1	IMM-6-415 + CTLA-4
BID	120	0/12			7/12	11/12	10/12
BID	60	0/12			0/12	8/12	8/12
BID	30	0/12			0/12	5/12	5/12
QD	120	0/12			0/12	7/12	7/12
QD	60	0/12			1/12	4/12	8/12
QD	30	0/12			0/12	6/12	7/12
BIW	10	0/12	4/12				
BIW	10	0/12		4/12			

• Number of BALB/c mice (out of 12) with tumors through Day 28 with volumes lower than 2,000 mm³

 Monotherapy Treated Alive at Day 28
 Combination ≥ 3 Advantage



Cyclic disruption of MEK improved overall survival with check point inhibitors (SITC 2022)

Corporate

 Immuneering



Finance & Intellectual Property

Finance

- Preliminary estimated Cash, cash equivalents and marketable securities as of March 31, 2023: **\$91.5M***
- **Cash runway projected into Q4 2024** supports:
 - IMM-1-104:
 - Multiple data readouts from Phase 1/2a trial
 - IMM-6-415:
 - Anticipate IND filing in Q4 2023
 - Research in additional oncology programs

Intellectual Property

Patents issued/pending:

- Pending U.S. and ex-U.S. application relating to IMM-1-104
- Pending U.S. provisional and PCT applications relating to IMM-6-415
- Issued U.S. patent and pending application relating to DCT
- Pending U.S. applications to Fluency

Expected patent expiration:

- (excluding patent term adjustments, extensions, and renewals)
- IMM-1-104 = 2041
 - IMM-6-415 = 2043
 - DCT = 2039
 - Fluency = 2039

*Our actual consolidated financial results as of March 31, 2023 are not yet available. Our financial closing procedures for the first quarter ended March 31, 2023 are not yet completed, and, as a result, our final results upon completion of those procedures may differ materially from our preliminary estimates. The preliminary consolidated financial information above as of March 31, 2023 is not a comprehensive statement of our financial position or operating results; reflects our preliminary estimates based on information available as of the date of this presentation; and is subject to change, and those changes may be material. Accordingly, you should not place undue reliance upon these preliminary estimates.

**As reported in the Company's most recent Annual Report on Form 10-K filed with the SEC

Milestones

Program	Milestone	Expected T
IMM-1-104	Initial Phase 1 pharmacokinetic (PK) and safety data	COMPL
IMM-1-104	Initial Phase 1 pharmacodynamic (PD) modeling data and additional PK and safety data	COMPL
IMM-1-104	Additional trial updates	On a period
IMM-1-104	RP2D and Additional Safety data	Early 20
IMM-6-415	IND filing	Q4 20

Differentiated Approach

- Targeting **Universal-RAS** patient population versus limited single mutation targeted approaches
- **Once-Daily Oral Dosing**
- **Deep cyclic inhibition** targeted, based on:
 - **Manyfold higher C_{MAX}**
 - **and short half-life**
- Approach designed to **spare healthy cells** and potential to **limit adaptive resistance**
- **Monotherapy-Focused** initially, with combination potential

IMM-1-104 Demonstrated Universal-RAS Potential

- **Robust preclinical activity** observed in:
 - Pancreatic Cancer (KRAS^{G12C} & G12V)
 - NSCLC (KRAS^{G12S})
 - CRC (KRAS^{G12D})
 - Melanoma (NRAS^{Q61R})
 - And others
- Hypothesis for IMM-1-104 from **proprietary model** that identified counterintuitive and novel deep cyclic inhibition approach
- **Validated using** proprietary **bioinformatics & 3D tumor growth assays**

Key Inflection Expected in Ne

- **Initial Phase 1 PK, data support profile** 104 believed to be **Deep Cyclic Inhibi**
- **Investigator enth** broad inclusion crit
- **Additional trial up** **expected on a per** **RP2D** expected in e
- **IMM-6-415** IND exp 2023
- **Cash runway proje** **Q4 2024**

Appendix



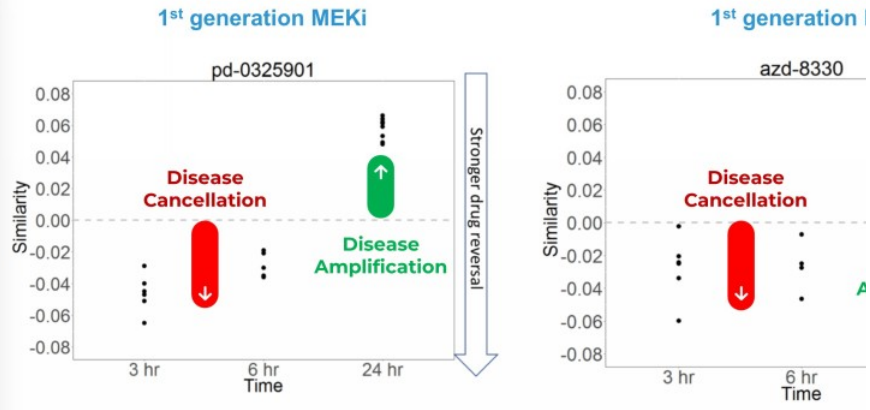
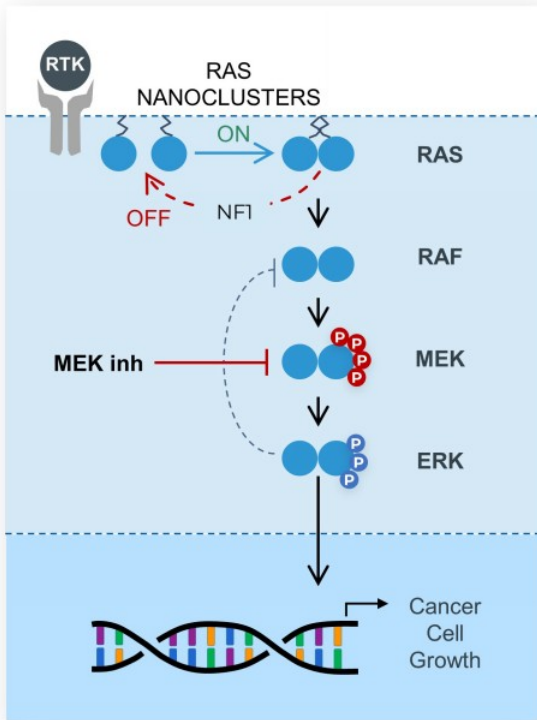
Nasdaq: IMRX

April 2023



Our Platform Converts Gene Expression to Counterintuitive

Goal: achieve broader activity and better tolerability in RAS and beyond mutant disease



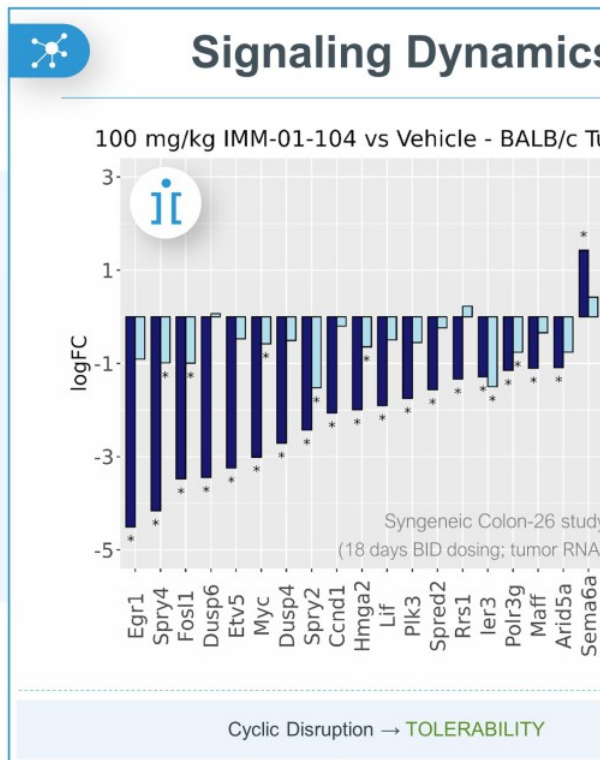
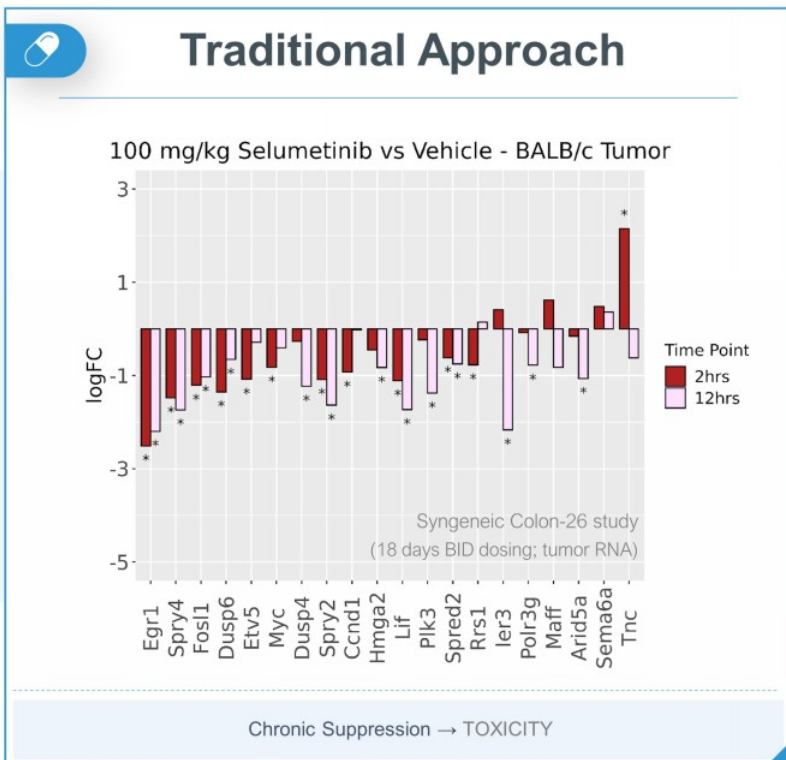
Note: dots are representative of vari

> IMRX Disease Cancelling Technology - US Patent 11,043,305

Unlike first generation MEK inhibitors, IMM-1-104 is designed to prevent RAF-mediated activation of MEK (i.e., CRAF-bypass) and displays a short plasma half-life, which potentially drive deep cyclic inhibition of the pathway.

Data-driven Identification and Optimization of New Medicines to Cancel Cancer Cachexia Presented by Ben Zeskind at the 12th International Conference of Cachexia, Sarcopenia & Muscle Wasting (SCWD) |

Deep Cyclic Inhibition Demonstrated Using Transcriptomic



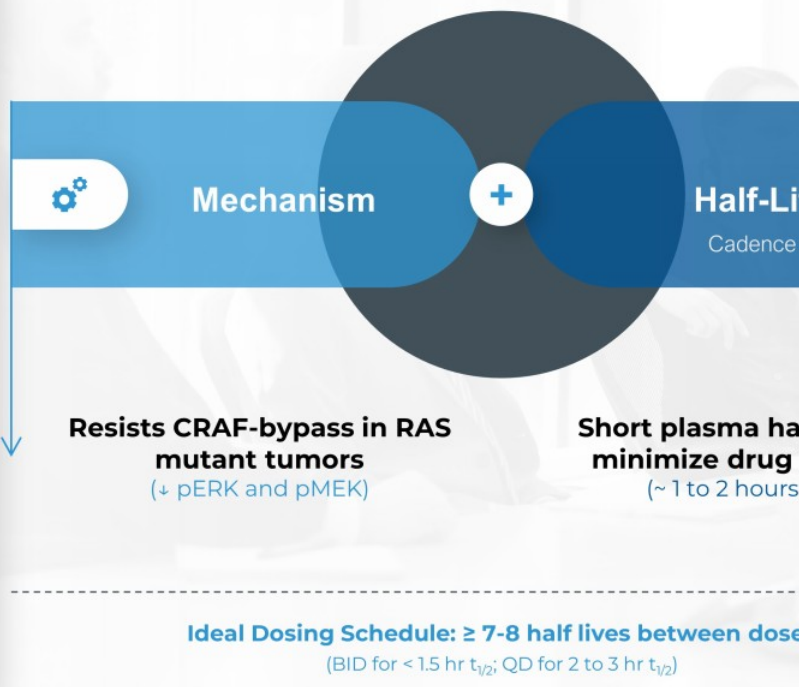
IMM-1-104 is a Dual-MEK Inhibitor

Goal

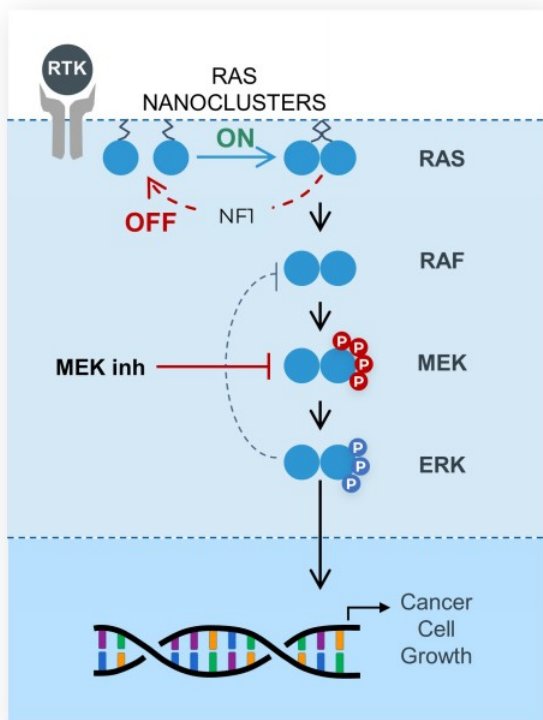
deep, cyclic inhibition with ability to prevent MEK-reactivation (improve tolerability and activity)



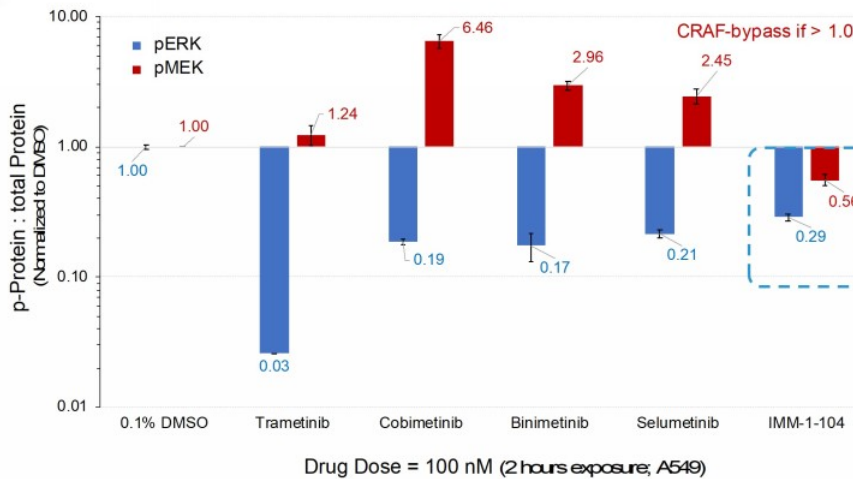
IMM-1-104



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



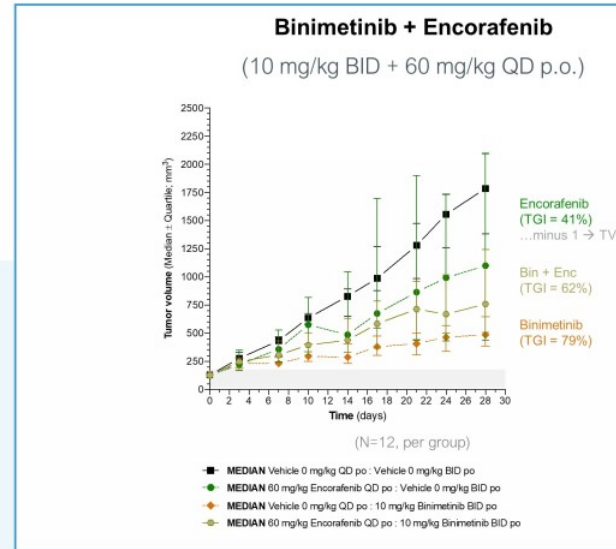
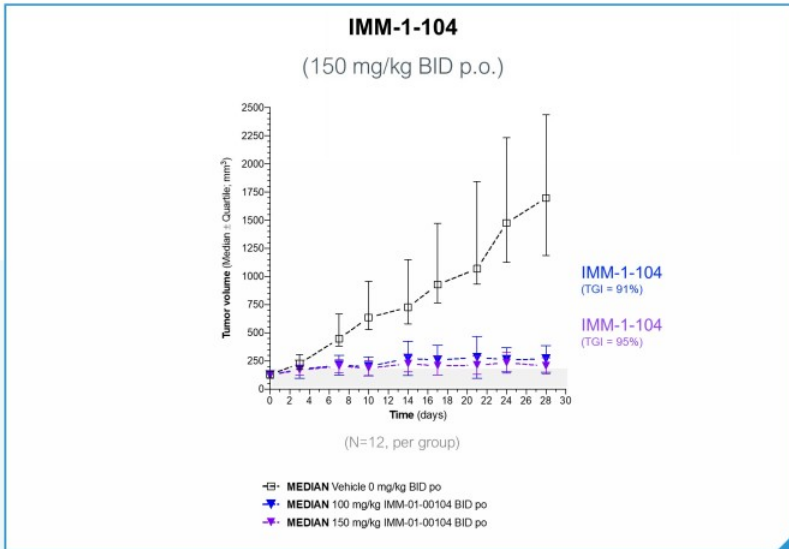
A549^{KRAS-mut} Lung Cancer: pERK and pMEK



> FDA-Approved MEK inhibitors: Trametinib, Cobimetinib, Binimetinib, Selumetinib com

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)

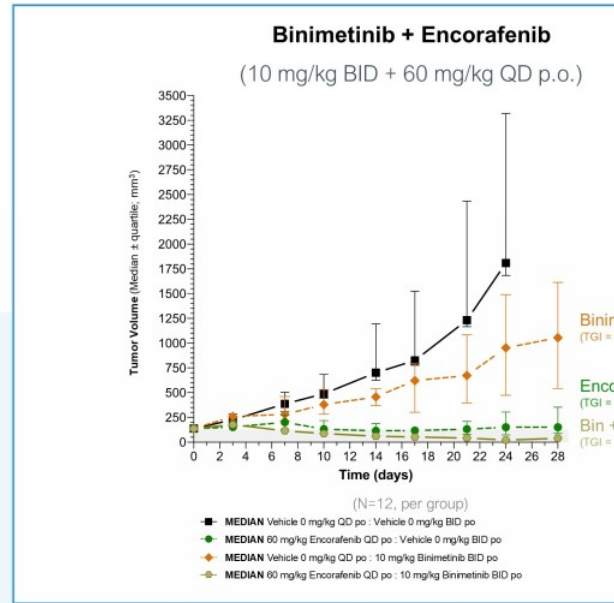
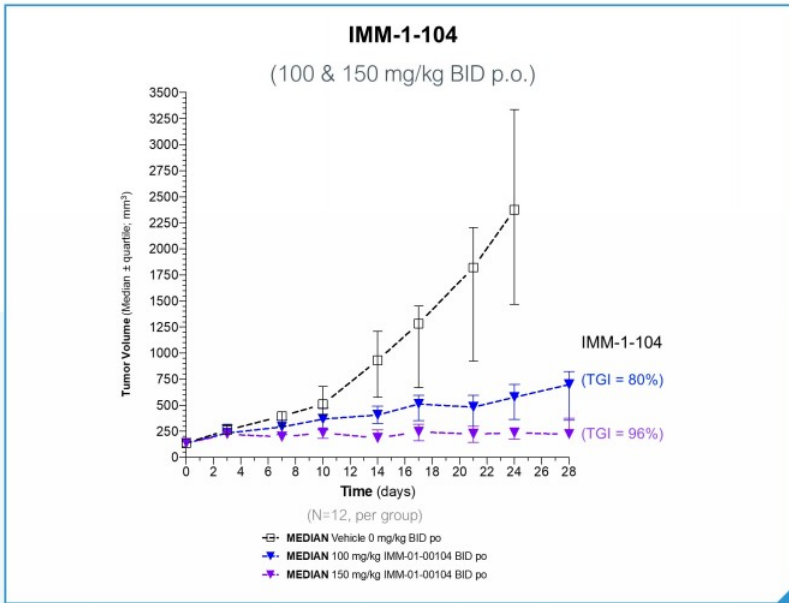


- > A549 (KRAS^{G12S}) NSCLC Xenograft Tumor Model in Athymic Nude Mice
- > Binimetinib and encorafenib were commercially purchased

- > Tumor Growth Inhibition (TGI) % = $[1 - (T_t - T_0) / (T_c - T_0)] \times 100$
Expanded TGI formula vs. previous $1 - [T/C] \times 100$
Human Dose Equivalent (HDE) binimetinib

Head-to-Head Comparison of IMM-1-104 versus 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

> Tumor Growth Inhibition (TGI) % = [1 - Expanded TGI formula vs. previous 1-] Human Dose Equivalent (HDE) binim

Conclusions (NRAS mutant Melanoma)



In the phase 3 NEMO study published in Lancet (c. 2017), binimetinib failed to substantially improve overall survival vs. dacarbazine (11.0 vs. 10.1 months) in NRAS mutant melanoma patients and led to a ~50% increase in serious adverse events (34% vs. 22%).



The most common NRAS mutation in the NEMO study was Q61R. We further compared binimetinib vs. IMM-1-104 in vivo using SK-MEL-2 (NRAS-Q61R).

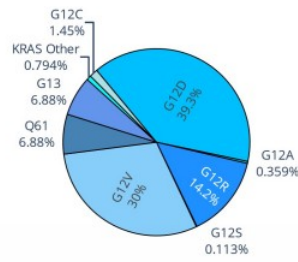


Collectively, our data suggest that binimetinib may not effectively inhibit the MAPK pathway reactivation in RAS mutant tumors. In contrast, deep cyclic inhibition combined with a different mechanism of action. IMM-1-104 may offer a unique therapeutic advantage over first-generation MEK inhibitors in RAS mutant tumors.

RAS Mutation Profiles Within Select Tumor Indications

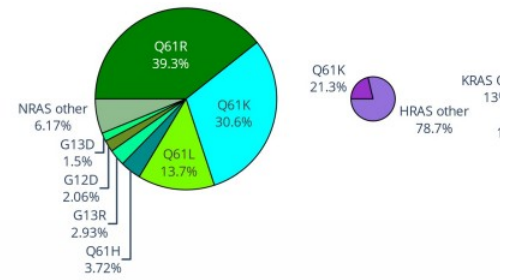
Pancreatic Cancer

NRAS = 0.34% HRAS = 0.06% KRAS = 99.60%



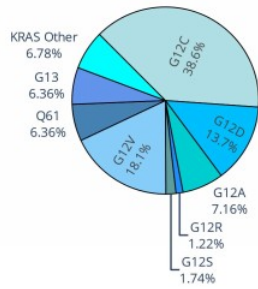
Melanoma

NRAS = 89.14% HRAS = 5.29% KRAS = 5.57%



Lung Cancer

NRAS = 2.67% HRAS = 0.31% KRAS = 97.02%



Colorectal Cancer

NRAS = 8.25% HRAS = 0.24% KRAS = 91.51%



- Based on given tumor type of patients with RAS-mutated tumors in the AACR GENIE database, v13.0
- Each RAS paralogue shown as percent overall RAS mutant tumors within each indication
- Presented at 2023 AACR: Targeting RAS. Koltz, et al. (Philadelphia, PA)

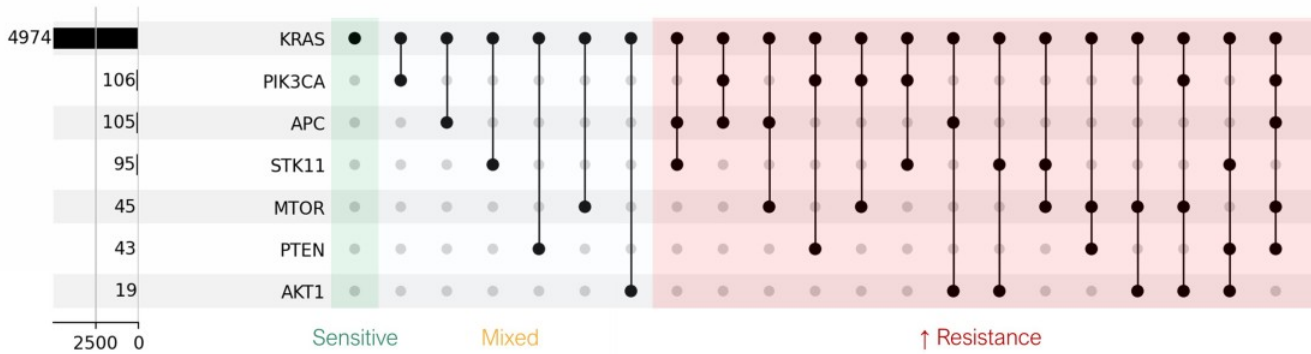
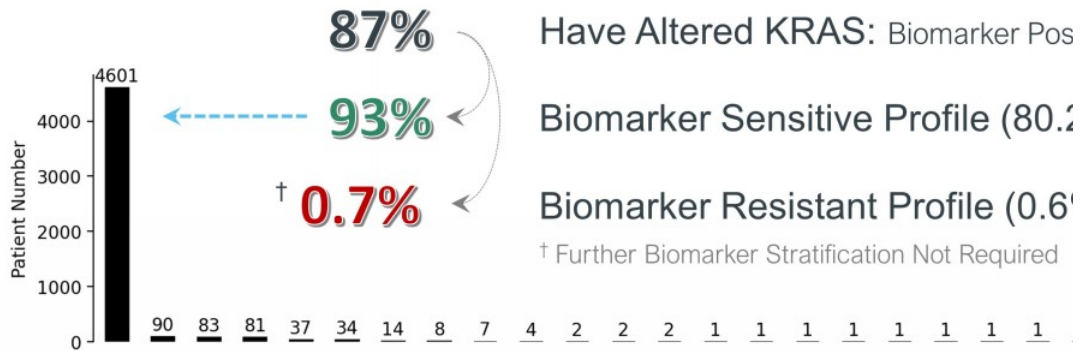
KRAS Mutant Pancreatic (PAAD): Translational Opportunity

Translational Step: Assess sensitivity of a panel of cell lines in the 3D-TGA; determine key mutations present in each

Bioinformatics Step: How many patients does each cell line represent, based on key mutations present in each

of 5,736

PANCREATIC Patients



GENIE cohort v13.0-public

The AACR Project GENIE Consortium. AACR Project GENIE: Powering Precision Medicine Through An International Consortium, Cancer Discov. 2017 Aug;7(8):818-831

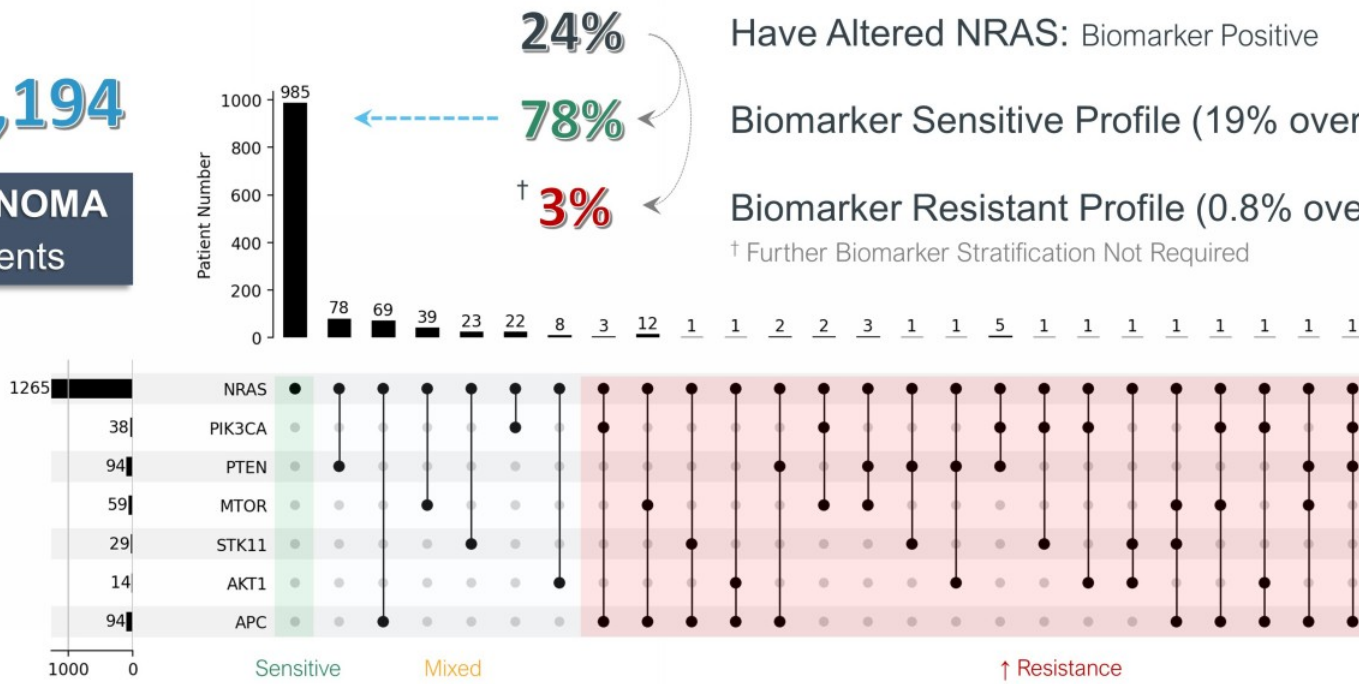
NRAS Mutant Melanoma: Translational Opportunity

Translational Step: Assess sensitivity of a panel of cell lines in the 3D-TGA; determine key mutations present in each

Bioinformatics Step: How many patients does each cell line represent, based on key mutations present in each

of 5,194

MELANOMA Patients



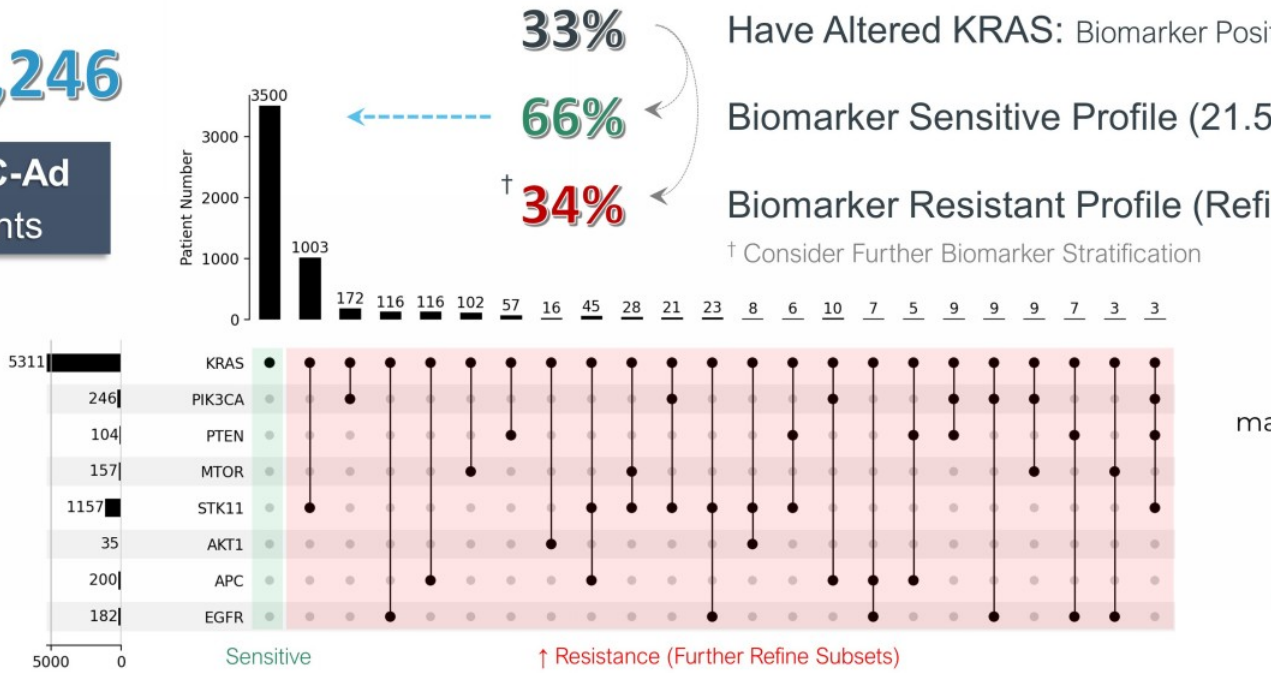
KRAS Mutant NSCLC (Adeno): Translational Opportunity

Translational Step: Assess sensitivity of a panel of cell lines in the 3D-TGA; determine key mutations present in each

Bioinformatics Step: How many patients does each cell line represent, based on key mutations present in each

of 16,246

NSCLC-Ad Patients



GENIE cohort v13.0-public

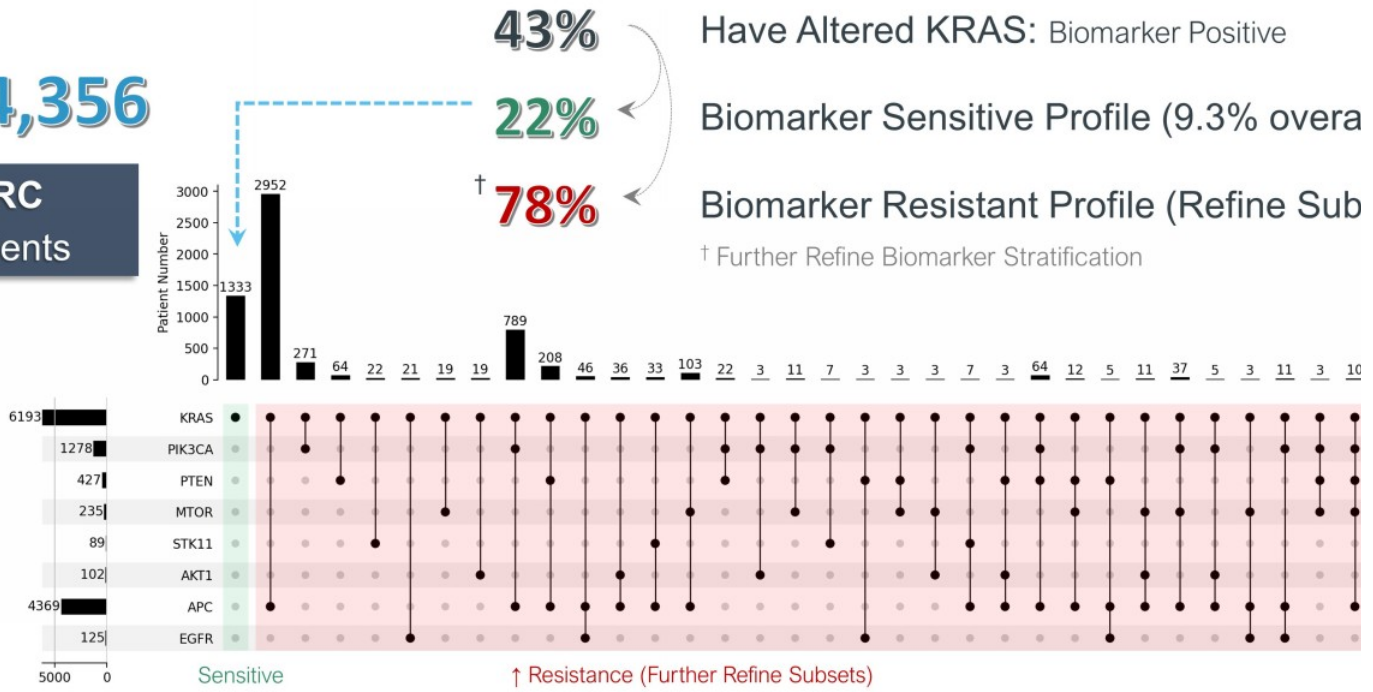
The AACR Project GENIE Consortium. AACR Project GENIE: Powering Precision Medicine Through An International Consortium, Cancer Discov. 2017 Aug;7(8):818-831

KRAS Mutant CRC: Translational Opportunity

Translational Step: Assess sensitivity of a panel of cell lines in the 3D-TGA; determine key mutations present in each
 Bioinformatics Step: How many patients does each cell line represent, based on key mutations present in each

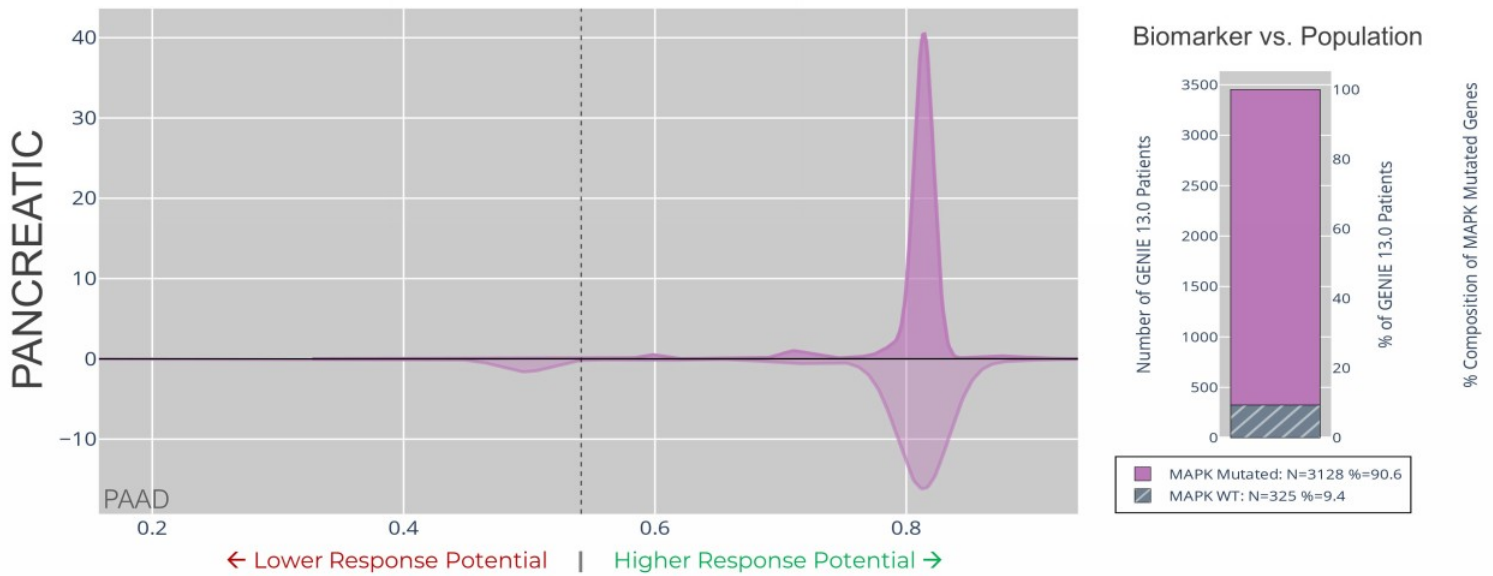
of 14,356

CRC Patients



Translating IMM-1-104's Universal-RAS Potential (AA

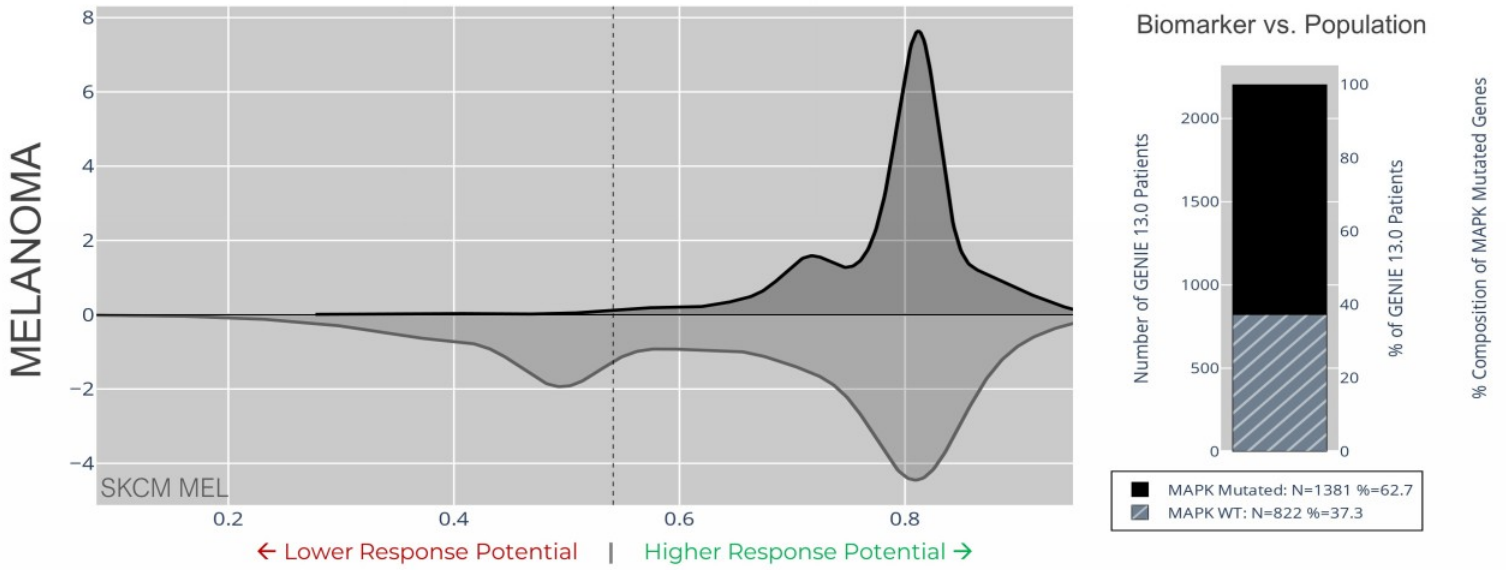
MAPK + = GENIE patients per indication with activation mutation in KRAS, NRAS, HRAS, BRAF (class I or II), based on 3D-TGA PGx Moc



For 122 3D-TGA cell lines where in-house WES data were also collected, the upper plateau-to-1 uM difference was used to delineate responder from non-responder. A logistic regression model with 10-fold cross validation. There were 28 features used, one comprised of a consolidated MAPK mutations status (mutations in BRAF class I/II), and 27 other genes representing key pathways. With an AUC of 0.84 on the training cohort and 0.72 on test cohort, the model was then a (above) where all features were included on the testing panel. For each indication of interest, a kernel density plot of the probability of predicted response (projected onto the negative y-axis), and the MAPK(+) subset (positive y-axis) shows enrichment for higher probability of response with restriction to MAPK alt

Translating IMM-1-104's Universal-RAS Potential (AA

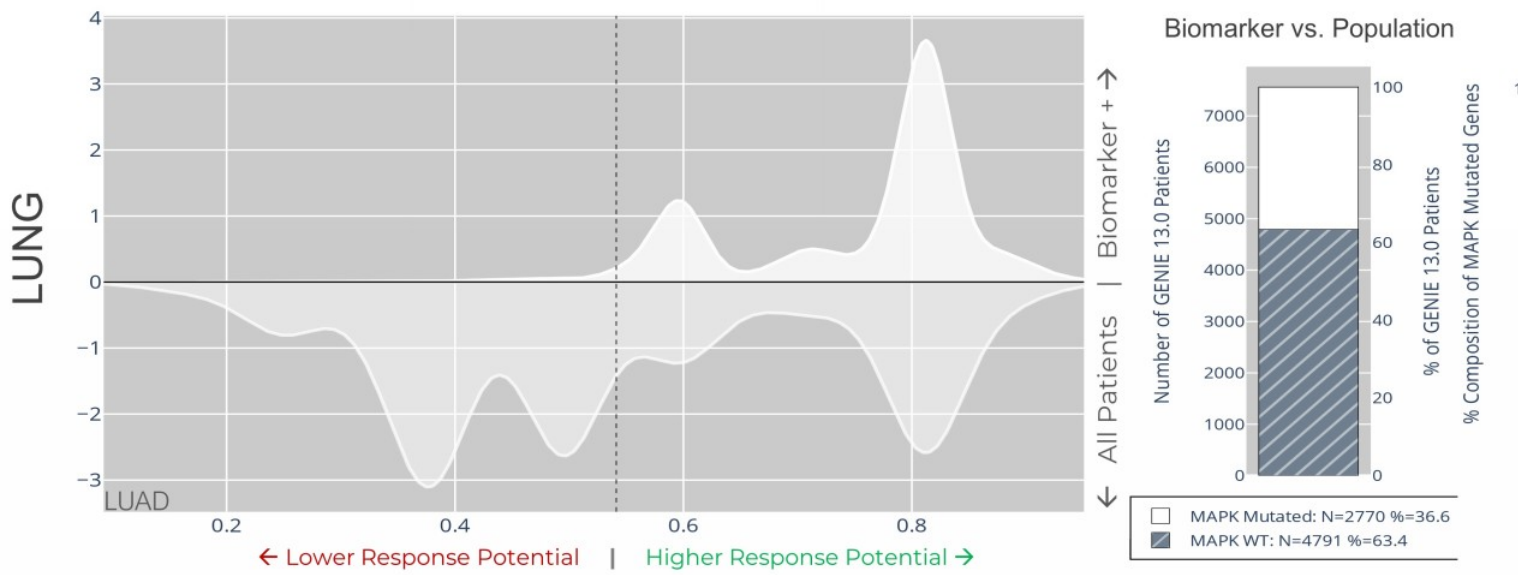
MAPK + = GENIE patients per indication with activation mutation in KRAS, NRAS, HRAS, BRAF (class I or II), based on 3D-TGA PGx Moc



For 122 3D-TGA cell lines where in-house WES data were also collected, the upper plateau-to-1 uM difference was used to delineate responder from non-responder. A logistic regression model with 10-fold cross validation was used. There were 28 features used, one comprised of a consolidated MAPK mutations status (mutations in BRAF class I/II), and 27 other genes representing key pathways. With an AUC of 0.84 on the training cohort and 0.72 on test cohort, the model was then applied to the testing panel. For each indication of interest, a kernel density plot of the probability of predicted response (projected onto the negative y-axis), and the MAPK(+) subset (positive y-axis) shows enrichment for higher probability of response with restriction to MAPK alterations.

Translating IMM-1-104's Universal-RAS Potential (AA

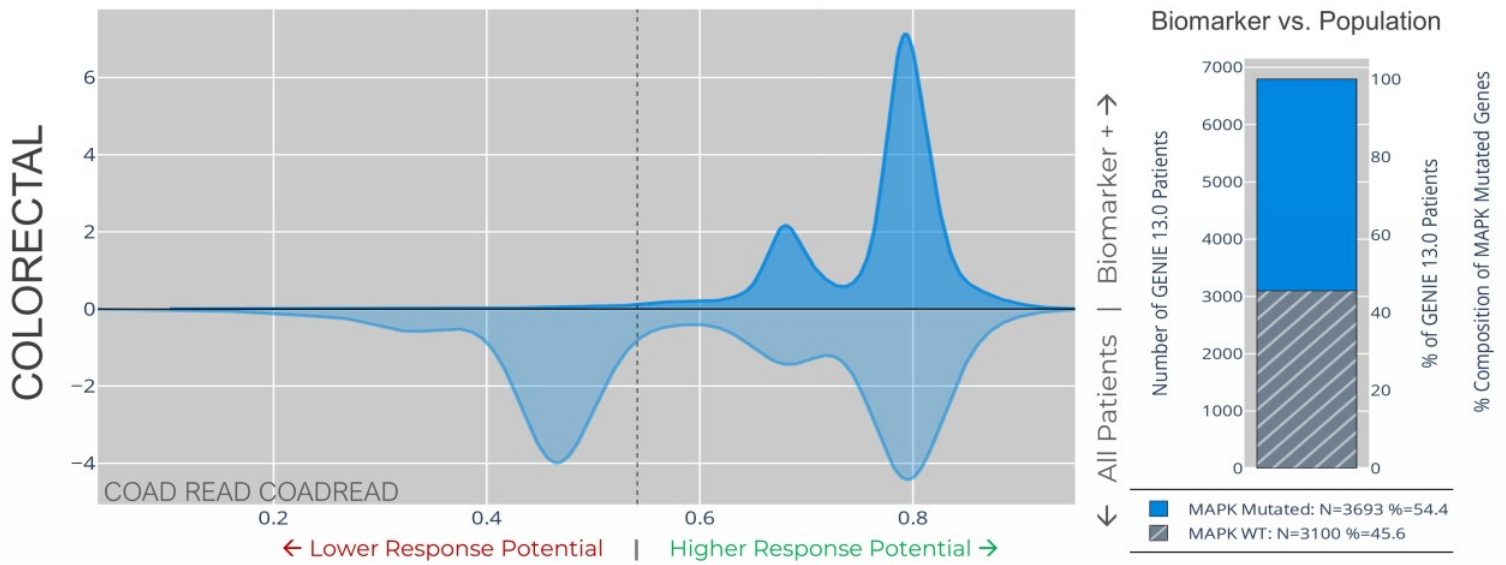
MAPK + = GENIE patients per indication with activation mutation in KRAS, NRAS, HRAS, BRAF (class I or II), based on 3D-TGA PGx Moc



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Translating IMM-1-104's Universal-RAS Potential (AA

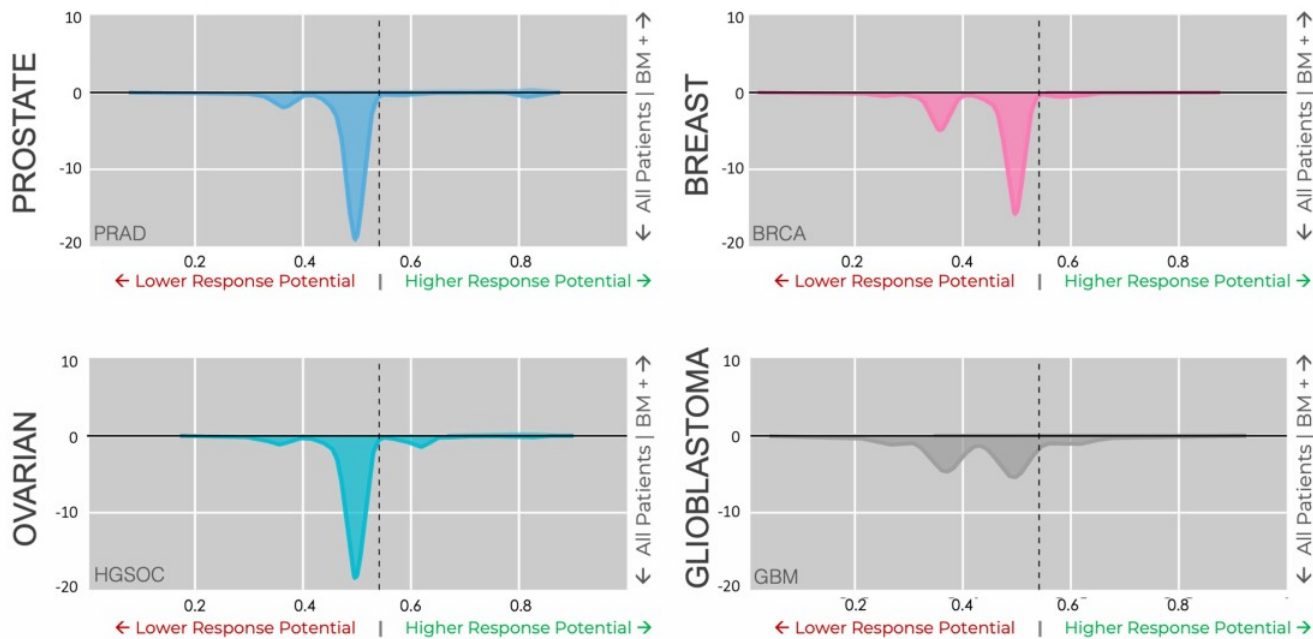
MAPK + = GENIE patients per indication with activation mutation in KRAS, NRAS, HRAS, BRAF (class I or II), based on 3D-TGA PGx Moc



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Translating IMM-1-104's Universal-RAS Potential (AA

MAPK + = GENIE patients per indication with activation mutation in KRAS, NRAS, HRAS, BRAF (class I or II), based on 3D-TGA PGx



For 122 3D-TGA cell lines where in-house WES data were also collected, the upper plateau-to-1 μ M difference was used to delineate responder from non-responder. A logistic regression model with 10-fold cross validation was used. There were 28 features used, one comprised of a consolidated MAPK mutations status (mutations in BRAF class I/II), and 27 other genes representing key pathways. With an AUC of 0.84 on the training cohort and 0.72 on test cohort, the model was then applied to the testing panel. For each indication of interest, a kernel density plot of the probability of predicted response (projected onto the negative y-axis), and the MAPK(+) subset (positive y-axis) shows enrichment for higher probability of response with restriction to MAPK activation.

**Immuneering Announces Positive Initial Phase 1 Pharmacokinetic, Pharmacodynamic and Safety Data for IMM-1-104 Universal-RAS Program; Accelerates Study Timeline**

- *Data presented at AACR Annual Meeting support IMM-1-104's potential to address a broad population of patients with RAS mutant tumors*
- *IMM-1-104 well tolerated with no dose limiting toxicities (DLTs) or serious adverse events (SAEs) observed*
- *First demonstration of novel deep cyclic inhibition mechanism in humans, with IMM-1-104 achieving significant levels of PK C_{max} and a half-life of approximately two hours as predicted*
- *Pharmacodynamic data support potential to evaluate preliminary efficacy sooner than expected*
- *Study timeline accelerated: recommended Phase 2 dose (RP2D) now expected in early 2024*
- *Investor call to be held today at 9.00 a.m. ET*

CAMBRIDGE, Mass., April 18, 2023 — Immuneering Corporation (Nasdaq: IMRX), a clinical-stage oncology company developing medicines for broad populations of cancer patients with an initial aim to develop a universal-RAS therapy, announced positive initial pharmacokinetic (PK), pharmacodynamic (PD) and safety data from the Phase 1 trial of IMM-1-104 (NCT05585320), which are being shared today in a poster presentation titled “Humanized 3D tumor models that are mutationally aligned with AACR GENIE patients predict IMM-1-104 activity in RAS-addicted tumors” (abstract #4265) at the American Association for Cancer Research (AACR) annual meeting.

“We are very pleased to share initial PK, PD and safety data from our Phase 1 trial of IMM-1-104 in patients with advanced RAS mutant solid tumors, ahead of schedule,” said Ben Zeskind, Ph.D., MBA, Co-founder, and Chief Executive Officer of Immuneering. “We believe these data from the first patients dosed in our study demonstrate the PK, PD and safety profile necessary for deep cyclic inhibition – the proprietary and novel mechanism through which our therapies are designed to selectively impact cancer cells to a greater extent than healthy cells, regardless of the specific RAS mutation driving the tumor. The data show that we were able to reach significant levels of PK C_{max} with the aim of breaking tumor addiction to the MAPK pathway, then rapidly clearing out drug with IMM-1-104’s short half-life. These results position us to accelerate the dose escalation portion of our study, reaching potentially therapeutic levels of IMM-1-104 earlier than previously planned.”

“These initial PK and PD Phase 1 data with IMM-1-104 mark a major milestone for Immuneering, and for patients affected by RAS mutant tumors. It is the first time IMM-1-104 has shown the profile we believe is necessary for deep cyclic inhibition in humans. Prior therapies have often suffered from steep increases in drug half-life in humans when compared to preclinical models. In contrast, initial clinical results for IMM-1-104 are in line with our preclinical modeling, which we believe helps to de-risk an important element of our universal-RAS program,” said Brett Hall, Ph.D., Chief Scientific Officer of Immuneering. “With today’s results showing an approximate two-hour half-life coupled with reaching target C_{max} values faster than expected plus encouraging pharmacodynamic, safety and tolerability results observed, we are accelerating the remaining dose-escalation portion of our trial. We now have an opportunity to assess potential preliminary efficacy earlier than anticipated.”



“We are highly encouraged by the initial safety and tolerability data generated to date. IMM-1-104 has been well tolerated with no DLTs or SAEs observed,” said Scott Barrett, M.D., Chief Medical Officer of Immuneering. “We are grateful to the patients participating in our trial, and to the investigators. Investigator enthusiasm remains high, which combined with our study’s broad inclusion criteria, gives us confidence in our ability to keep enrolling patients in an expeditious manner.”

The Phase 1/2a clinical trial is an open-label study designed to evaluate the safety, tolerability, PK and preliminary efficacy of IMM-1-104 in patients with advanced RAS mutant solid tumors. The Phase 1 portion of the study, which is being conducted at five clinical sites in the United States, is evaluating IMM-1-104 following a Bayesian mTPI-2 escalation design, which includes a dose escalation phase and dose evaluation phase to establish an optimized RP2D candidate. Following selection of the RP2D candidate, the Company expects to conduct a Phase 2a dose expansion phase to assess the safety and efficacy of IMM-1-104 at the RP2D in RAS mutated pancreatic, melanoma, lung and colorectal cancers.

Highlights of the initial IMM-1-104 Phase 1 PK, PD and safety data presented at AACR include (as of data cut-off date of April 10, 2023, including patients with pancreatic and colon cancer):

- Significant PK C_{max} levels (plasma concentration of therapy in a specific area of the body) observed with IMM-1-104 of over 2,000 ng/mL (or approximately 1 μ M drug free-fraction at 160 mg once daily oral dose)
- Greater than 90 percent PD inhibition of phosphorylated extracellular signal-regulated kinase (pERK) with IMM-1-104 compared to pretreatment baseline for patients at the third dose level (160 mg once daily oral)
- A median plasma half-life ($t_{1/2}$) of 1.94 hours observed with IMM-1-104 across the first three dose levels evaluable (40 mg, 80 mg and 160 mg once daily oral), in patients with pancreatic and colorectal cancer with different RAS mutations, including KRAS-G12D, the most common mutation present in pancreatic cancer
- IMM-1-104 was well tolerated with no DLTs or SAEs observed and no drug-related adverse events beyond Grade 1 observed

Based on the encouraging initial data presented today, Immuneering has updated guidance for the anticipated timing of announcing a RP2D for IMM-1-104 for its Phase 1/2a study. Management now expects to announce the RP2D in early 2024, versus prior guidance of mid-2024.

Other data presented at AACR:

In addition, IMM-1-104 was evaluated in humanized 3D preclinical tumor models displaying diverse mitogen-activated protein kinase (MAPK) pathway activation events. The MAPK pathway consists of a series of protein kinases such as RAS, RAF, MEK and ERK that are involved in many important cellular processes including cell proliferation, differentiation and survival. The antitumor activity of IMM-1-104 was evaluated in 132 tumor models spanning 12 distinct tumor types in a proprietary humanized 3D tumor growth assay (3D-TGA) conducted in Immuneering’s labs in San Diego. Based on drug-response sensitivity and resistance profiles, a biomarker signature for IMM-1-104 was developed to project potential therapeutic response in more than 100,000 cancer patients found in the AACR Project GENIE® database. Mutational landscapes of patients within GENIE helped identify preclinical models that represent patient profiles likely to be encountered in the clinic. These results were utilized in prioritizing indications for the planned Phase 2a clinical trial.



Updated Near-Term Milestone Expectations

IMM-1-104

- Additional trial updates expected on a periodic basis.
- RP2D and additional safety data expected in early 2024.

IMM-6-415

- IND filing expected in the fourth quarter of 2023.

Conference Call

Immuneering will host a conference call and live webcast at 9:00 a.m. ET / 6:00 a.m. PT on April 18, 2023, to discuss the results and provide a business update. Individuals interested in listening to the live conference call may do so by using the webcast link in the "Investors" section of the company's website at www.immuneering.com. A webcast replay will be available in the investor relations section on the company's website for 90 days following the completion of the call.

About IMM-1-104

IMM-1-104 aims to achieve universal-RAS activity that selectively impacts cancer cells to a greater extent than healthy cells, through deep cyclic inhibition of the MAPK pathway with once-daily dosing. IMM-1-104 is currently being evaluated in a Phase 1/2a study in patients with advanced solid tumors harboring RAS mutations (NCT05585320).

About Immuneering Corporation

Immuneering is a clinical-stage oncology company developing medicines for broad populations of cancer patients with an initial aim to develop a universal-RAS therapy. The company aims to achieve universal activity through deep cyclic inhibition of the MAPK pathway, impacting cancer cells while sparing healthy cells. Immuneering's lead product candidate, IMM-1-104, is in a Phase 1/2a study in patients with advanced solid tumors harboring RAS mutations. The company's development pipeline also includes IMM-6-415, a universal-MAPK program, as well as several early-stage programs. For more information, please visit www.immuneering.com.

Forward-Looking Statements

This press release includes certain disclosures that contain "forward-looking statements," including, without limitation, statements regarding Immuneering's expectations regarding the treatment potential of IMM-1-104, the design, enrollment criteria and conduct of the Phase 1/2a clinical trial, the translation of preclinical data into human clinical data, the ability of initial clinical data to de-risk IMM-1-104 and be confirmed as the study progresses, including the safety, tolerability, pharmacokinetics, pharmacodynamics and potential efficacy of IMM-1-104; the potential advantages and effectiveness of the company's clinical and preclinical candidates, the timing of additional trial updates, recommended phase 2 dose and additional safety data, the indications to be pursued by Immuneering in the Phase 2a portion of the study, the timing of submission of the IND for IMM-6-415, and Immuneering's ability to advance its pipeline and further diversify its portfolio and make progress towards its longstanding goal of creating better medicines for cancer patients. Forward-looking statements are based on Immuneering's current expectations and are subject to inherent uncertainties, risks and assumptions that are difficult to predict. Factors that could cause actual results to differ include, but are not limited to, the risks inherent in oncology drug research and development, including target discovery, target validation, lead compound identification, lead compound optimization, preclinical studies, and clinical trials. These and other risks and uncertainties are described more fully in the section titled "Risk Factors" in Immuneering's most recent Form 10-K filed with the U.S. Securities and Exchange Commission. Forward-looking statements contained in this announcement are made as of this date, and Immuneering undertakes no duty to update such information except as required under applicable law.



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