KINNATE BIOPHARMA

Investor Presentation

Disclaimer

This presentation (including the accompanying oral presentation) contains forward-looking statements that involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation, including statements regarding the future financial condition, results of operations, business strategy and plans, and objectives of management for future operations of Kinnate Biopharma Inc. ("we," "us" or "our"), as well as statements regarding industry trends, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "plan," "potentially" "predict," "should," "will" or the negative of these terms or other similar expressions. We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy and financial needs.

These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including, among other things: our ability to successfully complete our ongoing clinical trial and for that trial to produce positive results, the timing of the initiation, progress and potential results of our preclinical studies, additional clinical trials and our research programs; our ability to advance additional product candidates into, and successfully complete, preclinical studies and clinical trials with those additional product candidates; the timing or likelihood of regulatory filings and approvals; the negative impacts of the COVID-19 pandemic; our estimates of the number of patients who suffer from the diseases we are targeting and the number of patients that may enroll in our clinical trials; the commercializing of our product candidates, if approved; our ability and the potential to successfully manufacture and supply our product candidates for clinical trials and for commercial use, if approved; future strategic arrangements and/or collaborations and the potential benefits of such arrangements; our estimates regarding expenses, future revenue, capital requirements and needs for financing and our ability to obtain capital; the sufficiency of our existing cash and cash equivalents to fund our future operating expenses and capital expenditure requirements; our ability to retain the continued service of our key personnel and to identify, hire and retain additional qualified professionals; the implementation of our business model, strategic plans for our business and product candidates; the scope of protection we are able to establish and maintain for intellectual property rights, product candidates and our pipeline; our ability to contract with third-party suppliers and manufacturers and their ability to perform adequately; the pricing, coverage and reimbursement of our product candidates, if approved; and developments relating to our competitors and our industry, including competing product candidates and therapies.

These and other risks, uncertainties, assumptions and other factors are described in greater detail in our filings we have made and will make with the Securities and Exchange Commission, including, without limitation, under the heading "Risk Factors" in our Quarterly Report on Form 10-Q for the fiscal quarter ended September 30, 2021. New risk factors emerge from time to time and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance or achievements. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation. In addition, statements that "we believe" and similar statements reflect our beliefs and opinions on the relevant subject. These statements are based upon information available to us as of the date of this presentation, and while we believe such information forms a reasonable basis for such statements, such information may be limited or incomplete, and our statements should not be read to indicate that we have conducted an exhaustive inquiry into, or review of, all potentially available relevant information.

Certain information contained in this presentation relates to or is based upon our internal estimates and research and from academic and industry research, publications, surveys and studies conducted by third parties, including governmental agencies. Information that is based on estimates, forecasts, projections, market research or similar methodologies is inherently subject to uncertainties and actual events or circumstances may differ materially from events and circumstances that are assumed in this information. While we believe that the data we use from third parties are reliable, we have not separately verified this data. Further, while we believe our internal research is reliable, such research has not been verified by any third party. Any projections, assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

Kinnate: Kinase Inhibitors for Genomically Defined Cancers

Striving to Expand the Promise of Precision Medicine in Oncology

Programs

Multiple clinical and pre-clinical assets targeting validated oncogenic drivers

Lead RAF program targets large population not served by current approved RAF inhibitors

- IND clearance by FDA, Phase I initiated

FGFR program targets significant unmet need of resistance to current FGFR inhibitors

- GLP tox studies completed, IND filing expected in H1 2022

Multiple other compounds in pipeline, including CDK12 inhibitor

- All programs developed in house with IP & commercial rights fully retained

Platform

Productive Kinnate Drug Discovery Engine powered by structure-based drug discovery, translational research and patient-driven precision medicine

- 3 Years from inception to initial IND clearance

People

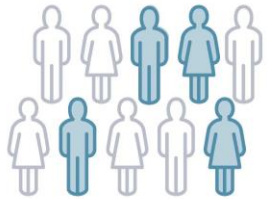
- Experienced management team responsible for multiple approved precision oncology drugs
- Strong scientific collaborations and KOL relationships with leading academic and medical centers
- Diverse board with broad experience across biopharma

Well-Funded with $348M on Hand*

*Cash and cash equivalents & investments as of September 30, 2021, excludes $34M cash in the China JV

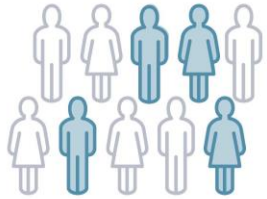
Limitations of Current Targeted Therapies Drive Clinical Need

Our Research and Development Programs Will Focus on Three Patient Populations



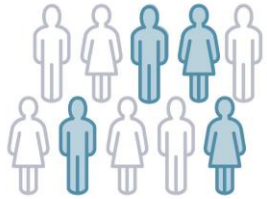
Target known oncogenic drivers in cancers that are not addressed by approved therapies

- Example: KIN-2787, our Class II and Class III BRAF-targeting small molecule kinase inhibitor



Overcome acquired resistance mutations to existing targeted therapies, potentially improving the durability of response

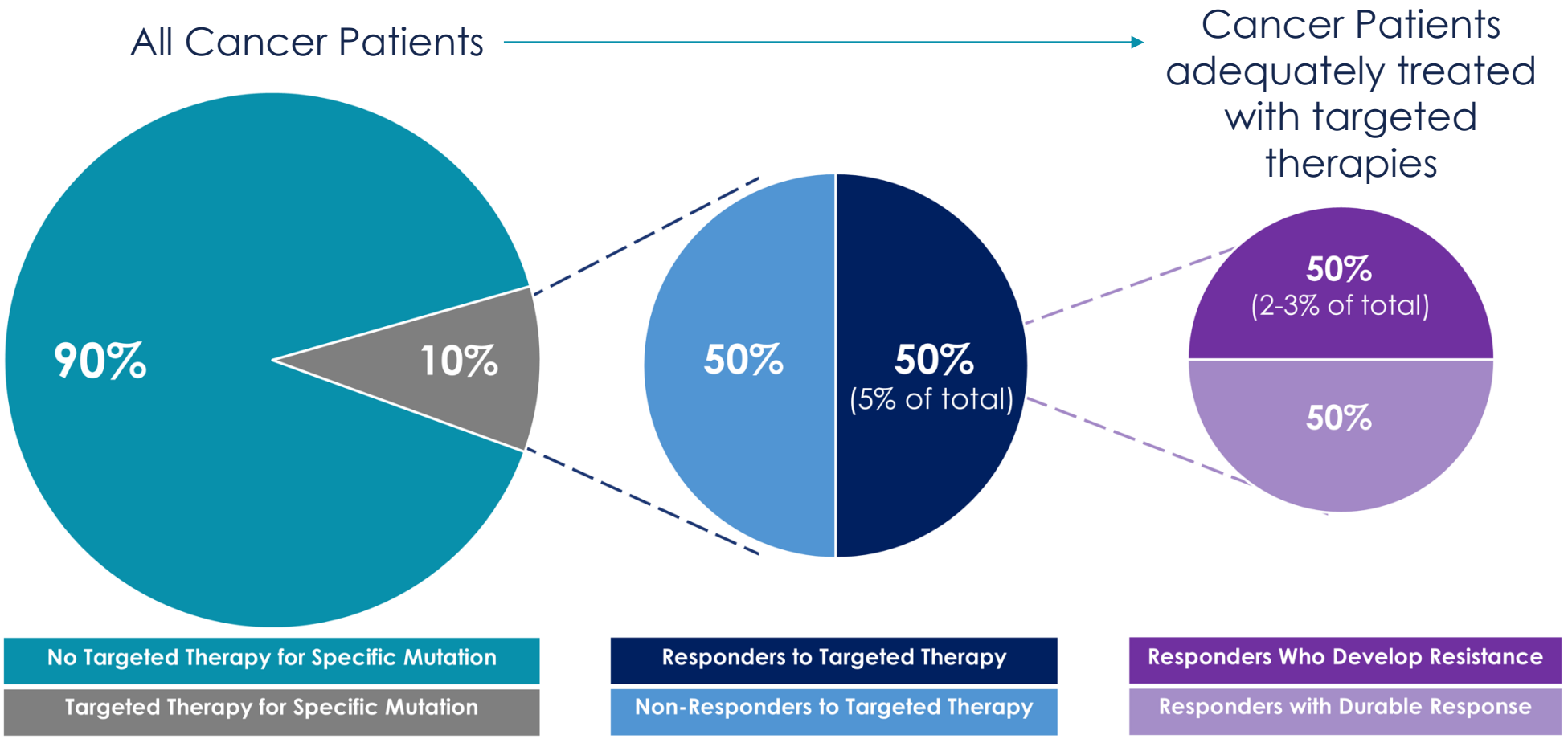
- Example: KIN-3248, our FGFR2/3-targeting small molecule kinase inhibitor



Treat non-responders to approved therapies by identifying genomic drivers of intrinsic resistance through advanced technologies

- Example: KIN004, our selective CDK12 kinase inhibitor

Substantial Opportunity in Targeted Therapies for Oncology

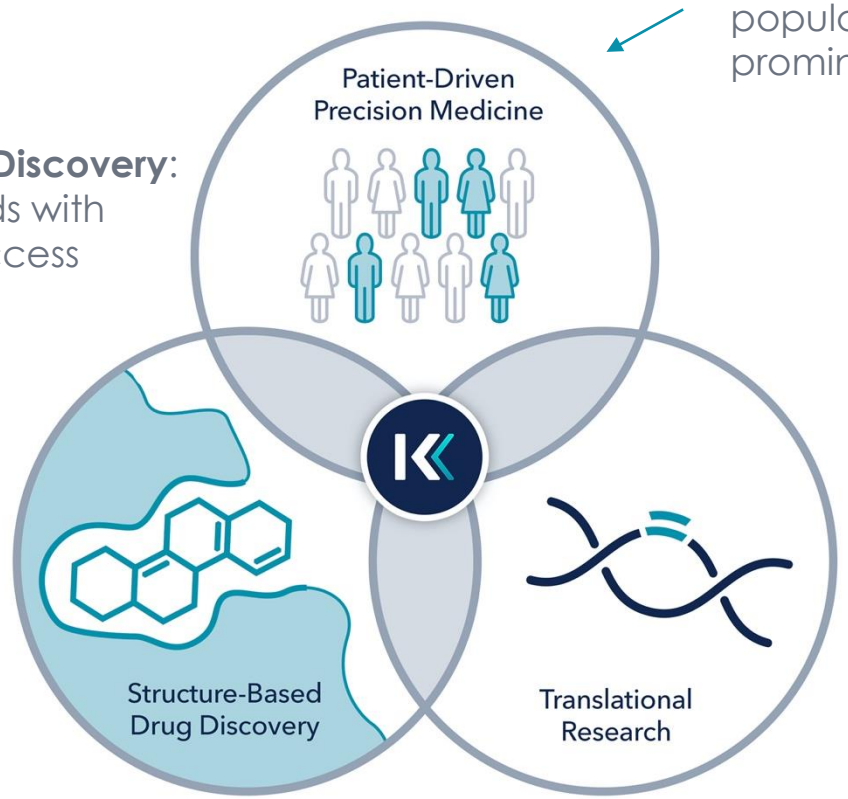


Source: Marquart JAMA ONOCLOGY 2018 https://pubmed.ncbi.nlm.nih.gov/29710180/; data available in USPIs for targeted cancer therapies approved in the US

Kinnate Drug Discovery Engine

Patient Driven Precision Medicine: Defining emerging patient populations. Guided by premier cancer centers such as MGH and prominent KOLs

Structure Based Drug Discovery: Identifying compounds with high probability of success



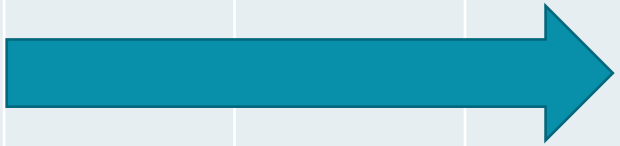
Since company funded in March 2018

- >5200 NCEs generated
- >220 unique in vitro assays developed
- 17+ xenograft models
- 17 provisional patent applications filed across 14 families
- **1 IND cleared in 2021 & 1 expected in H1 2022**

Translational Research: Biomarker-driven approach to predict and increase the likelihood of therapeutic response

Kinnate Pipeline

Multiple programs advancing into clinical stage

Target, Program	Discovery	Lead Optimization	IND-Enabling	Phase 1	Phase 2	Phase 3	Next Anticipated Milestones
RAF KIN-2787							Initial Clinical Data in mid 2022
FGFR2/3 KIN-3248							IND H1 2022 Initiate Phase 1 in H1 2022
CDK12 KIN004							

Multiple undisclosed targets in Research Stage

Kinnate Team

Creators of Multiple Marketed Drugs and High Value Exits

Nima Farzan

CEO, Board Member

- CEO PaxVax (BLA approval, M&A exit)
- Novartis – global & US commercial and development roles

Richard Williams, MD

CMO

- Wuxi NextCode, GRAIL, Puma & Amgen
- Led Amgen's early oncology program

Neha Krishnamohan

CFO

- Goldman Sachs Health Care Investment Banking
- Advised on over $100B in transactions

Mark Meltz

COO & GC

- Led $3.5B Corporate Development Transactions
- Public company GC (Audentes)
- Novartis, Biogen, PaxVax, Audentes

Rob Kania, PhD

SVP, Drug Discovery

- Led Pfizer Cancer Chemistry group
- Co-inventor of 13 DCs
- Co- Inventor of Inlyta (axitinib), Xalkori (crizotinib), Lorbrena (lorlatinib)

Ken Kobayashi, MD

SVP, Clinical Development

- FDA, NCI/CTEP, Novartis, J&J, AZ, DSI & Pfizer
- 28 investigational agents into clinic
- Lead reviewer on 3 NDAs at FDA

Kinnate Board of Directors & Scientific Collaborators

Leaders in the Field of Precision Oncology

Board of Directors*

Jim Tananbaum
Board Member
Foresite

Michael Rome
Board Member
Foresite

Carl Gordon
Board Member
Orbimed

Dean Mitchell
Board Chairman
Independent

Keith Flaherty
Board Member
Independent - MGH

Laurie Smaldone
Board Member
Independent

Melissa Epperly
Board Member
Independent

Helen Sabzevari
Board Member
Independent

Scientific Advisory Board

Keith Flaherty
- Professor, Harvard Medical School
- Director of Clinical Research, MGH Cancer Center
- Director, MGH Termeer Center for Targeted Therapy
- Loxo co-founder; RAF expert

MASSACHUSETTS GENERAL HOSPITAL CANCER CENTER · HARVARD MEDICAL SCHOOL

Ryan Corcoran
- Associate Professor, Harvard Medical School
- Scientific Director, MGH Termeer Center for Targeted Therapy
- RAF & FGFR expert

MASSACHUSETTS GENERAL HOSPITAL CANCER CENTER · HARVARD MEDICAL SCHOOL

Luis Diaz
- Head of the Division of Solid Tumor Oncology Memorial Sloan Kettering Cancer Center

Memorial Sloan Kettering Cancer Center

Andy Lowy
- Chief, Surgical Oncology
- PDAC expert

UC San Diego MOORES CANCER CENTER

Ezra Cohen
- Co-Director San Diego Center for Precision Immunotherapy
- Assoc. Dir, Translational Science

UC San Diego MOORES CANCER CENTER

John Iafrate
- Director of Center for Integrated Diagnostics at MGH
- Professor Pathology at Harvard Medical School

MASSACHUSETTS GENERAL HOSPITAL CANCER CENTER · HARVARD MEDICAL SCHOOL

Eric Murphy, PhD
- Co-Founder, Kinnate
- Novartis, CrownBio, Samumed, Moores UCSD Cancer Center
- Contributions: Braftovi, Mektovi, LXH254, Zykadia, Tabrecta, EGF816

*List excludes Nima Farzan, Kinnate CEO

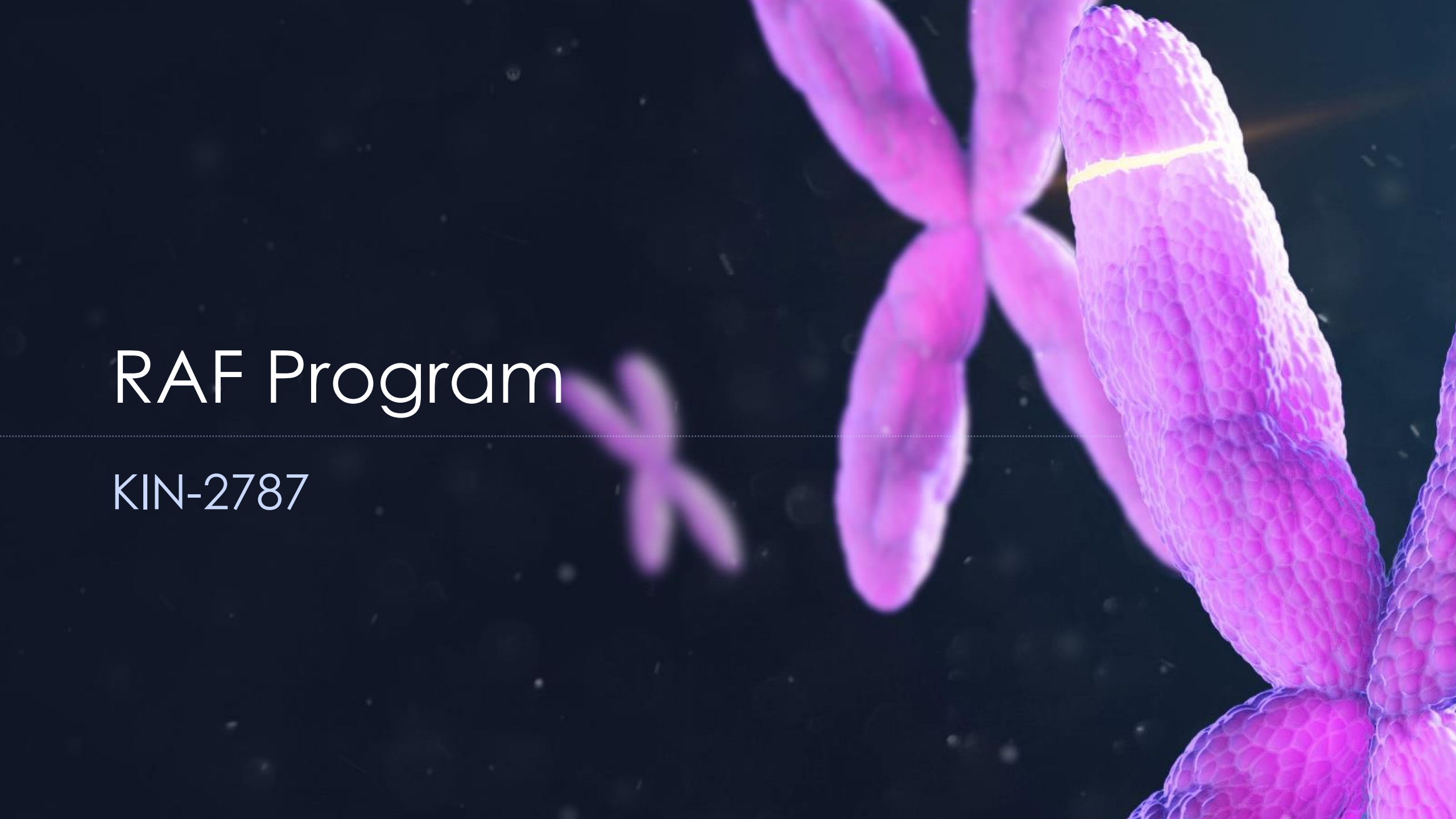
Kinnate Expansion into Greater China

Joint Venture Established with Experienced China Investor OrbiMed Asia Partners

- $35M Series A Financing of a new China JV based in Shanghai
- Investor OrbiMed Asia Partners brings tremendous expertise and connections in China to the JV
  - OrbiMed Private Investments and Foresite Capital also participated in round
- Kinnate is the majority shareholder of the China JV
  - JV has exclusive license to develop, manufacture and commercialize Kinnate's RAF, FGFR and CDK12 product candidates in Greater China (mainland China, Hong Kong, Taiwan, and Macau)
  - JV may obtain rights to other Kinnate pipeline candidates in Greater China, as well as pursue other candidates
  - Kinnate retains customary termination rights on license of IP
- Potential to accelerate enrollment of programs through global trial recruitment
- Veteran biopharmaceutical industry executive Wenn Sun, Ph.D. is Executive Chair of the China JV
  - Founder/President of Precision Medicine Asia (PREMIA), an oncology clinical genomic data company
  - Founder and Managing Partner of OxOnc Development, a venture company that, along with Pfizer Oncology, co-developed XALKORI in patients with ROS1 genetic alterations in Asia, including China
  - Head of Strategic Alliances for GSK Oncology

RAF Program

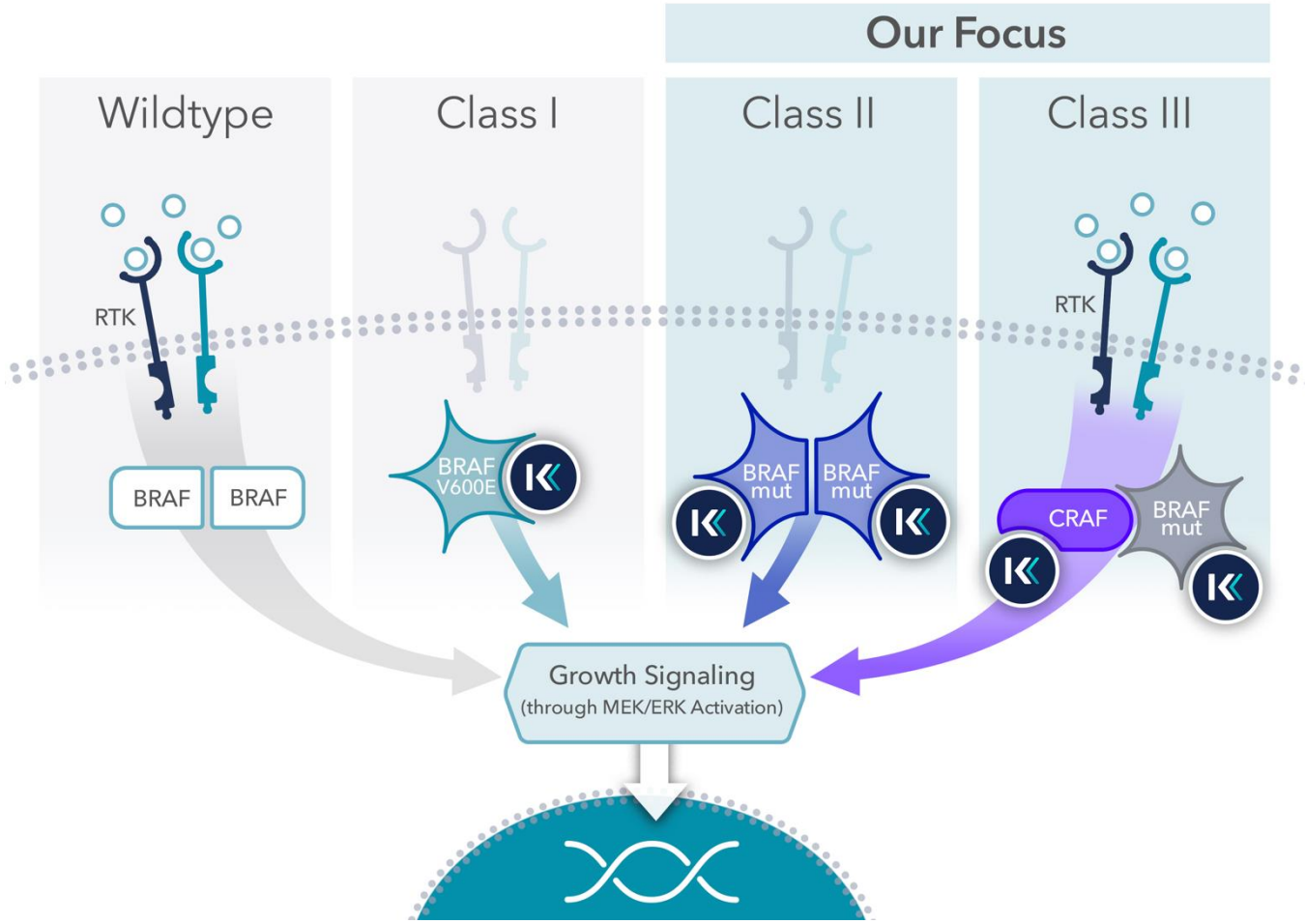
KIN-2787



The RAF Opportunity

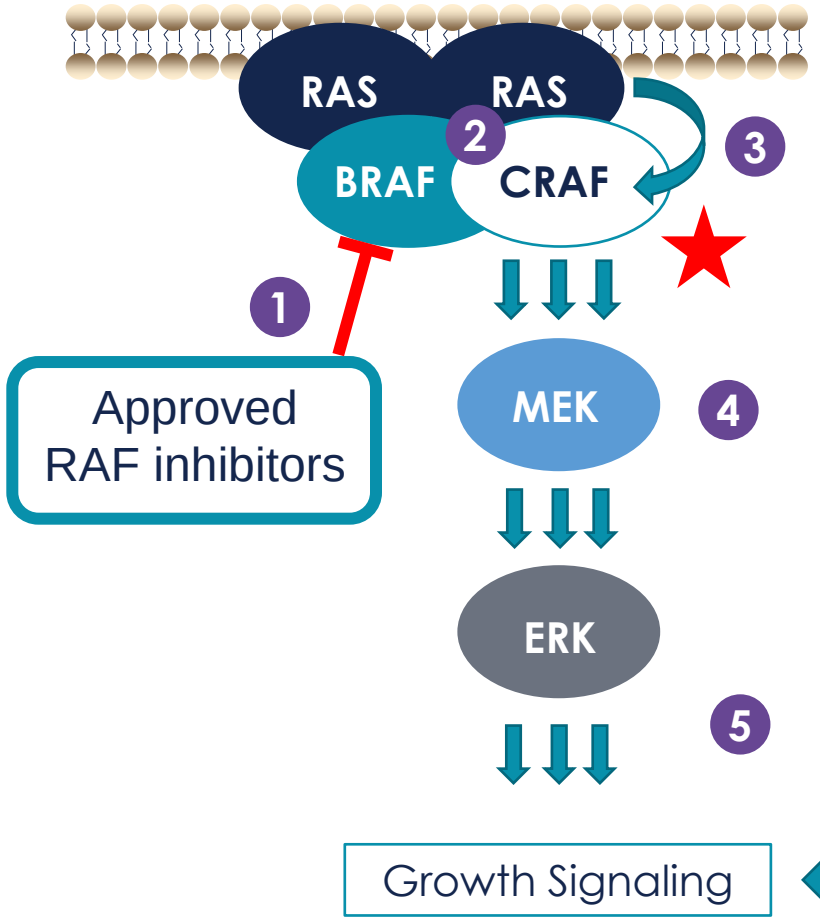
Targeting BRAF Mutant Populations Without Approved Precision Therapies

- Approved Class I BRAF inhibitors include Vemurafenib, Dabrafenib, Encorafenib
- The Class II and Class III BRAF mutants represent a patient population with unmet need
- Kinnate's approach targets dimer signaling in these patient populations while minimizing MAPK pathway rebound in normal wild type signaling



Inhibition of Both RAF Kinases in Dimer is Required

Otherwise Transactivation drives MAPK signaling and Pathway Activation



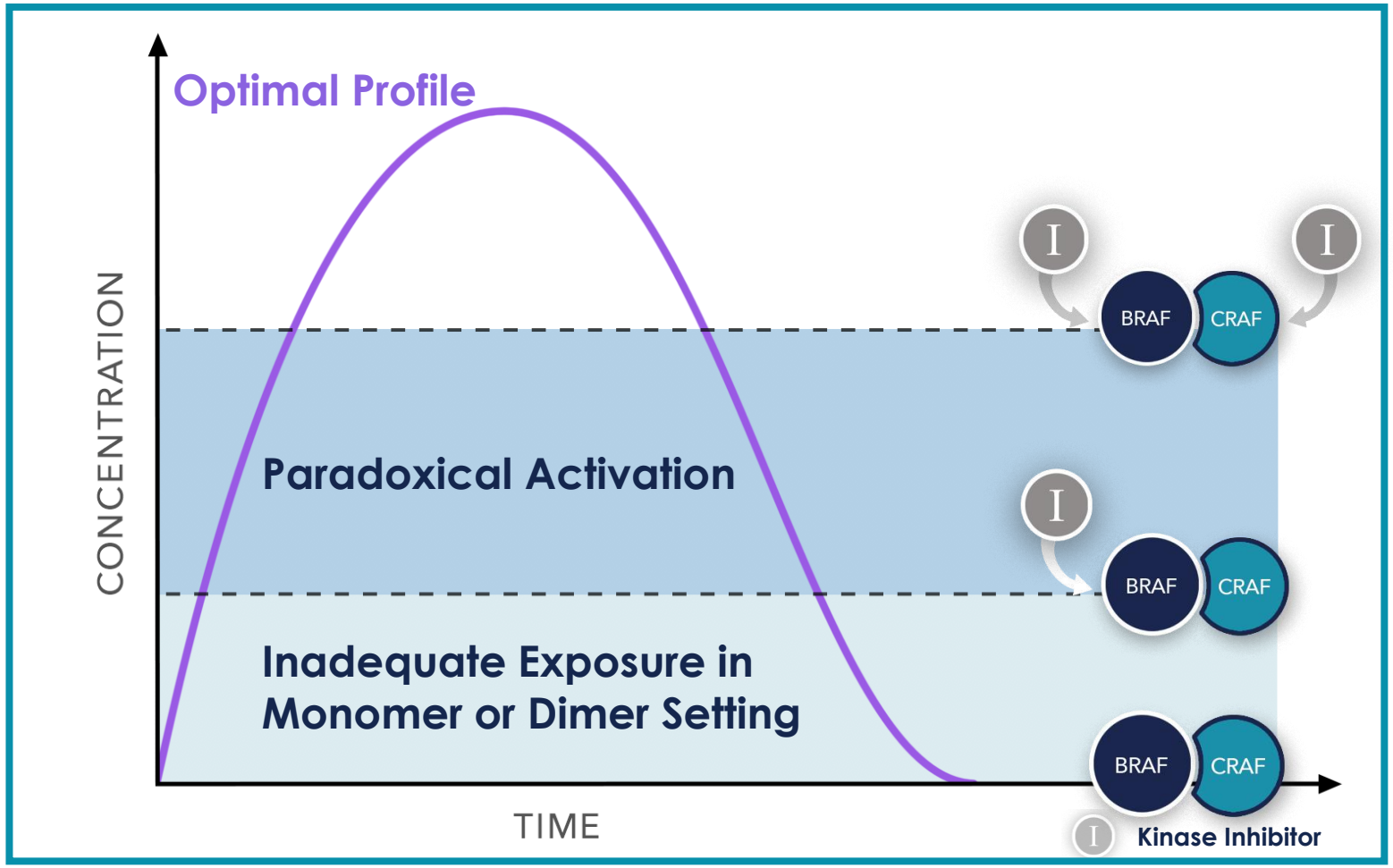
1. BRAF Inhibitor binds to BRAF Target
2. BRAF + CRAF heterodimerize, recruited to RAS
3. Transactivation of CRAF via RAS binding
4. MEK and ERK activation
5. Elevated growth promoting signaling

★
- Paradoxical activation from BRAF with altered drug binding site due to asymmetric dimerization or CRAF in heterodimer
- Need molecule that can inhibit second kinase active site

- Why approved BRAF inhibitors can cause squamous cell carcinoma (SCC) in skin cells
- Why Class I BRAF inhibitors are often combined with a MEK inhibitor

Adapted from: S. Heidorn SJ et. al., **Kinase-Dead BRAF and Oncogenic RAS Cooperate to Drive Tumor Progression through CRAF**. Cell 140: 209-221, 2010

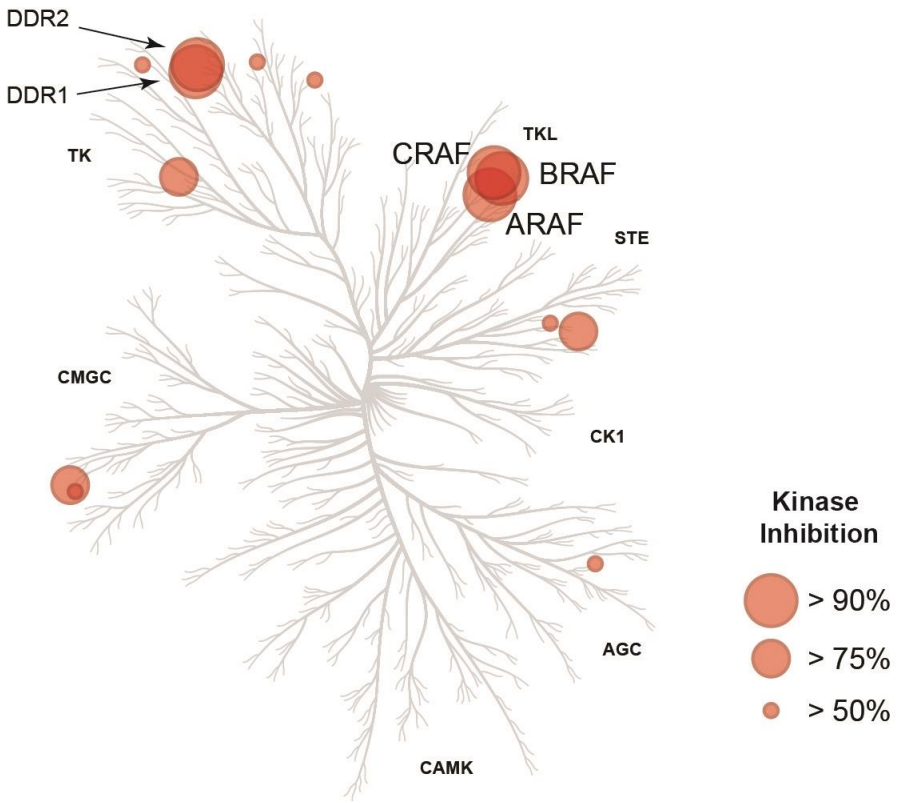
Optimal Target Coverage & Equivalent / Persistent Inhibition of Both RAF Kinases in Dimer is Required to Avoid Paradoxical Activation



- Paradoxical activation occurs when the non-inhibited RAF molecule in the RAF dimer is activated
- This can occur when the RAF molecule is in a homodimer (BRAF-BRAF) or a heterodimer conformation (BRAF-CRAF), depicted in the figure on the left
- Can occur:
  - As drug concentrations approach & dip below effective levels
  - If the inhibitor does not bind to 2nd molecule in the dimer in an equipotent manner
  - Adequate target exposure is not achieved

KIN-2787 Displays a Highly Selective RAF Kinase Profile

Kinome Profiling



10-point Dose Response

Kinase	KIN-2787 IC_{50} (nM)
CRAF	**0.573**
BRAF ^{V600E}	**1.53**
ARAF	**2.41**
BRAF	**3.46**
DDR1	108
PDGFRB	445
p38alpha	1230
EPHA2	>3000
KDR	>3000
LCK	>3000
SRC	>3000

- Kinome profiling @ 1µM across >600 enzymatic assays at Reaction Biology Corp *(372 WT, 23 atypical, 258 MT)*
- Follow-up 10 pt dose response enzymatic assays (right table) for known BRAF inhibitor off-targets

Dimer Inhibition Demonstrated Across Several Cell Lines

While Maintaining Selectivity Against Non BRAF Mutated Cells

BRAF Status	Tumor Cell Line	Lineage	MAPK Pathway Alteration(s)	pERK Inhibition EC50 (nM)		
				Pfizer Binimetinib	Novartis LXH-254	**Kinnate KIN-2787**
Class I	A-375	Melanoma	$BRAF^{V600E}$	7	171	**67**
	Colo800	Melanoma	$BRAF^{V600E}$	6	242	**112**
Class II	BxPC-3	Pancreatic	$BRAF^{indel(VTAPTP)}$	3	32	**51**
	OV-90	Ovarian	$BRAF^{indel(NVTAP)}$	4	24	**26**
	NCI-H2405	NSCLC	$BRAF^{indel(LNVTAP)}$	6	5	**10**
Class III	WM3629	Melanoma	$BRAF^{D594G}$, $NRAS^{G12D}$	5	6	**9**
	CAL-12T	NSCLC	$BRAF^{G466V}$	3	19	**18**
Wild Type	MIA PaCa-2	Pancreatic	$BRAF^{WT}$, $KRAS^{G12C}$	3	340	685
	NCI-H358	NSCLC	$BRAF^{WT}$, $KRAS^{G12C}$	1	153	351
	CHL-1	Melanoma	$BRAF^{WT}$, $NRAS^{WT}$	5	291	580

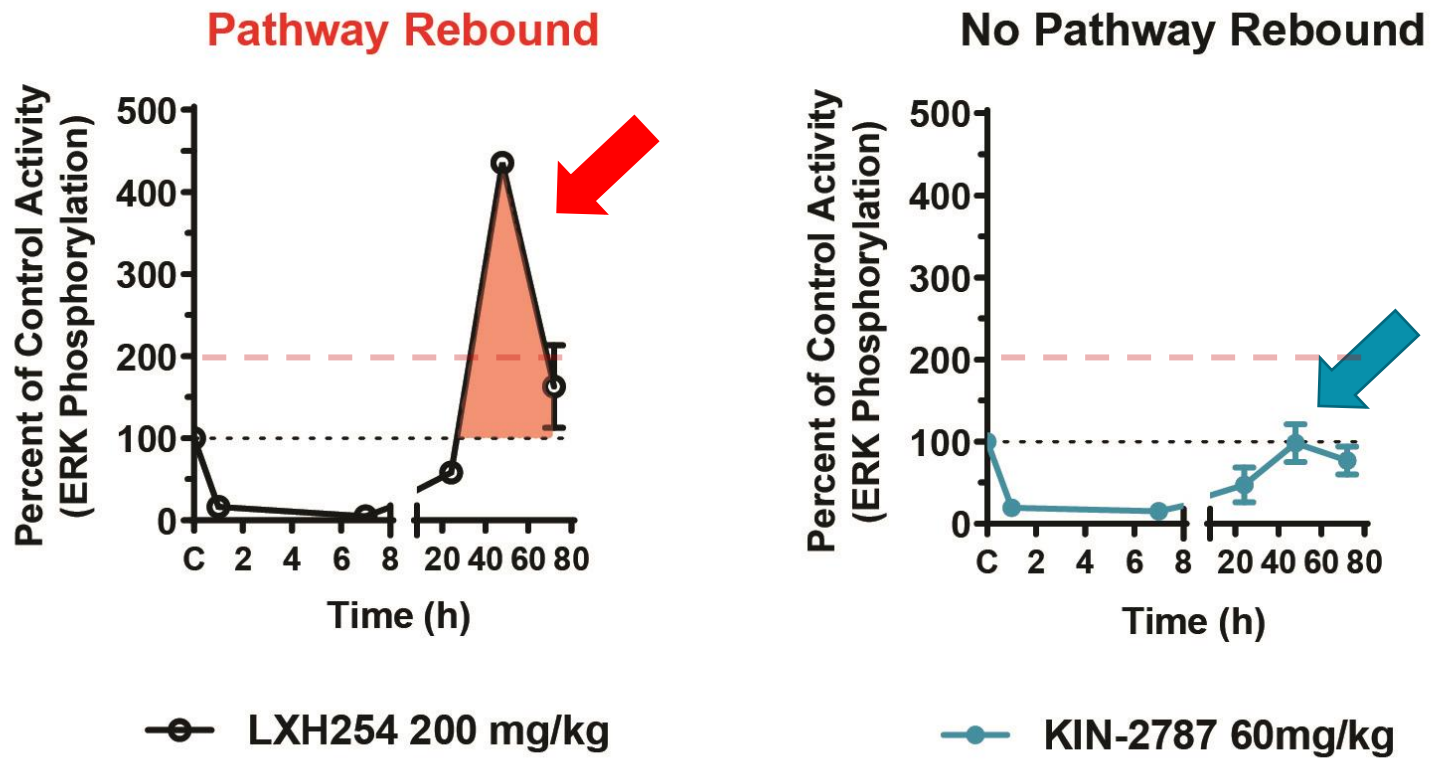
Note: More potent inhibition is reflected by a lower EC_{50} number presented in nM concentration

- Clear differentiation from MEK inhibitors that do not differentiate against WT (wild type)
- Novartis' LXH-254 has similar profile in cells, but suffers from sub-optimal exposure *in vivo*

Unlike LXH254, KIN-2787 Did Not Show Pathway Rebound

Due to Potent Dimer Inhibition & Improved Target Exposure

> 200% pERK characterized as Pathway Rebound



- No pathway rebound was observed with KIN-2787 in WM3629 (Class III, *BRAF*^{D594G}/*NRAS*^{G12D}) xenografts compared to >400% pERK levels observed with LXH254 at 48 hours post-dose

Improved Solubility Increases *In Vivo* Target Exposure

Increased Target Exposure Prevents Pathway Rebound & Lowers Necessary Dose

Feature	Parameter	Novartis LXH254	Kinnate KIN-2787
***In vitro* drug solubility**	**Aqueous Solubility (µM)** pH = 7.4 pH = 4.5 pH = 2.0	 **8** **7** **50**	 **29** **196** **312**
***In vivo* mouse pharmacology**	**100 mg/kg per oral dose** Clearance (mL/min/kg) AUC / dose (ng*h/mL)	 **10** **1123**	 **8** **3335**

Relevant physiological pH

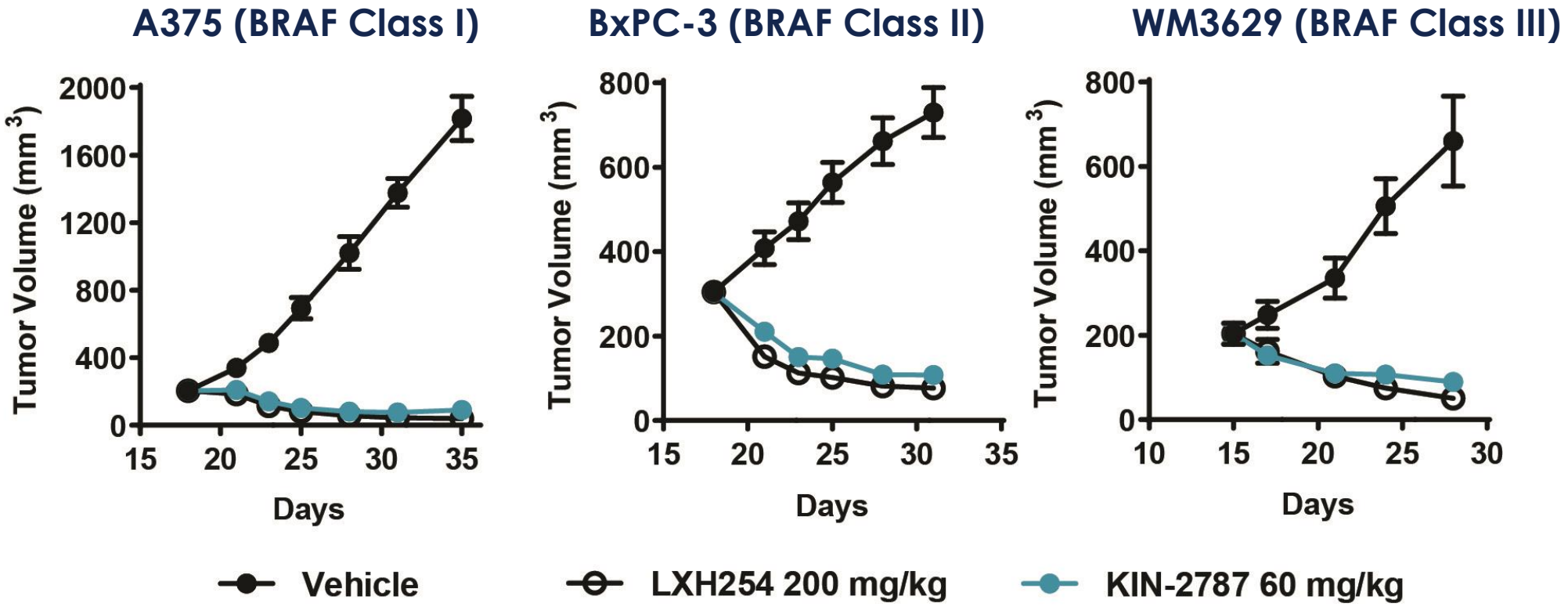
KIN-2787 has shown **~8x more** free fraction in human

Improved aqueous solubility, lower clearance in vivo, higher free fraction, and increased drug exposure all enhance the likelihood that KIN-2787 may achieve **greater target coverage** in the clinical setting

KIN-2787

Tumor Regressions Achieved Across All Classes of Mutation at Lower Doses Than LXH254

Head to Head Data Shows Benefit from Target Exposure & Limited Pathway Activation & Rebound



- KIN-2787 (60 mg/kg QD) and LXH254 (200 mg/kg QD) both demonstrated tumor regressions
- 200 mg/kg LXH254 is **>4-fold** increased free drug exposure relative to the highest clinical dose (600 mg BID)

Note: Reflects freebase formulation of KIN-2787; Both KIN-2787 and LXH254 were administered daily

Sensitivity to BRAF Inhibition in BRAF Mutation-Driven Cancers

Spectrum of Sensitivity Will be Directly Evaluated in our FIH Trial

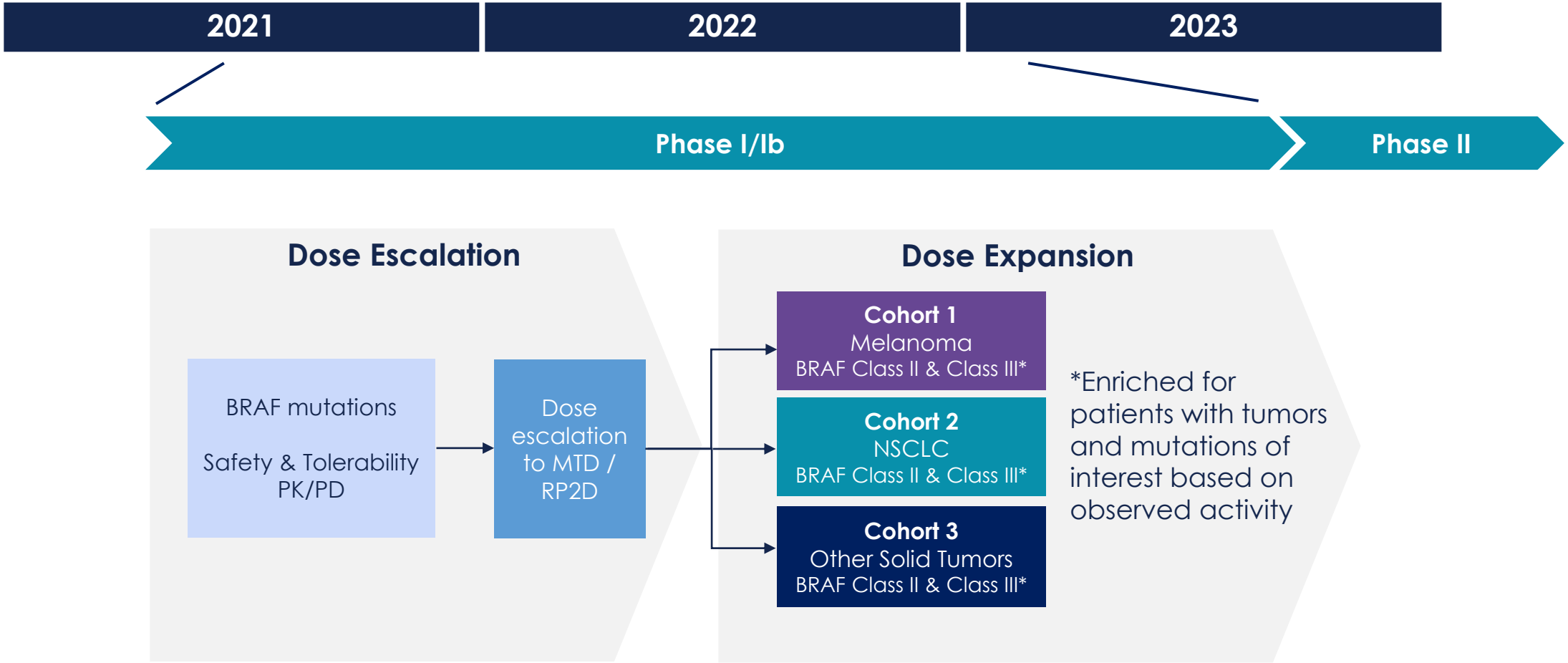
BRAF Class I: Refractory to prior RAFi therapy	**BRAF Class I:** BRAFi-Naive patients	**BRAF Class III:** SNVs	**BRAF Class II:** Indels + SNVs	**BRAF Class II:** Gene Fusions
▪ Diverse mechanisms drive resistance ▪ Dimer-dependent resistance presents KIN-2787 monotherapy opportunity ▪ Applicable in NSCLC and Melanoma	▪ KIN-2787 shows *in vitro* activity & *in vivo* tumor regressions ▪ Applicable in PTC and others	▪ KIN-2787 sensitivity likely predicated on identity of co-occurring upstream RAS activation* ▪ More sensitive in Melanoma than other cancer types	▪ KIN-2787 shows substantial *in vitro* activity & *in vivo* tumor regressions ▪ More sensitive in NSCLC / Melanoma	▪ KIN-2787 shows substantial *in vitro* activity & *in vivo* tumor regressions ▪ CR** reported in BRAF gene fusion-driven case treated with PLX8394 ▪ More sensitive in NSCLC

- FIH study design enables flexibility to enrich for cancer types and mutation classes with early activity

* e.g. EGFR amplification, KRAS mutation, NF1 loss
** CR: Complete regression

KIN-2787 Development Plan: Phase 1 Trial

Well Positioned for Expedited POC



- Dose Escalation phase with 6 US sites; Phase 1 initiated at multiple sites; First Patient Dosed

FIH Study: Patient Selection Strategy

Phase 1 Trial Patient Inclusion Criteria for Dose Escalation and Dose Expansion

Study Part	Population (Advanced or Metastatic cancers)
A. Dose Escalation (n=25)	Patients with any of the following: • Class I ($BRAF^{V600}$) mutant positive cancer, or • Class II BRAF mutant positive cancer, or • Class III BRAF mutant positive cancer
B. Dose Expansion (3 cohorts, n=25 each)	#1 Melanoma (*BRAF* Class II or Class III mutations)
	#2 NSCLC (*BRAF* Class II or Class III mutations)
	#3 Other solid tumors (BRAF Class II or Class III mutations) ex. Pancreatic, PTC, ovarian

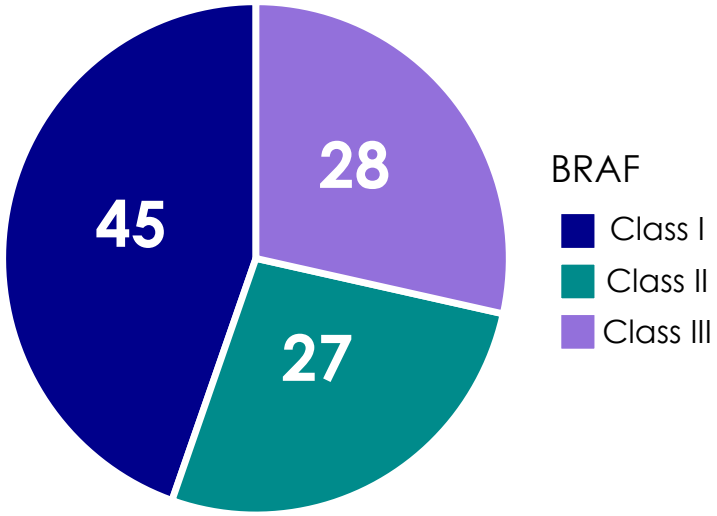
- Initiate Dose Escalation at 50 mg/day (25 mg BID) in Dose Level 1
- Single patient cohorts for first two Dose Levels

Pan-Cancer Prevalence of Patients Bearing BRAF Alterations

Majority of oncogenic BRAF alterations (~55%) are Class II or III

Guardant360® analysis of **~143,000** ctDNA positive samples from cancer patients with advanced or metastatic disease

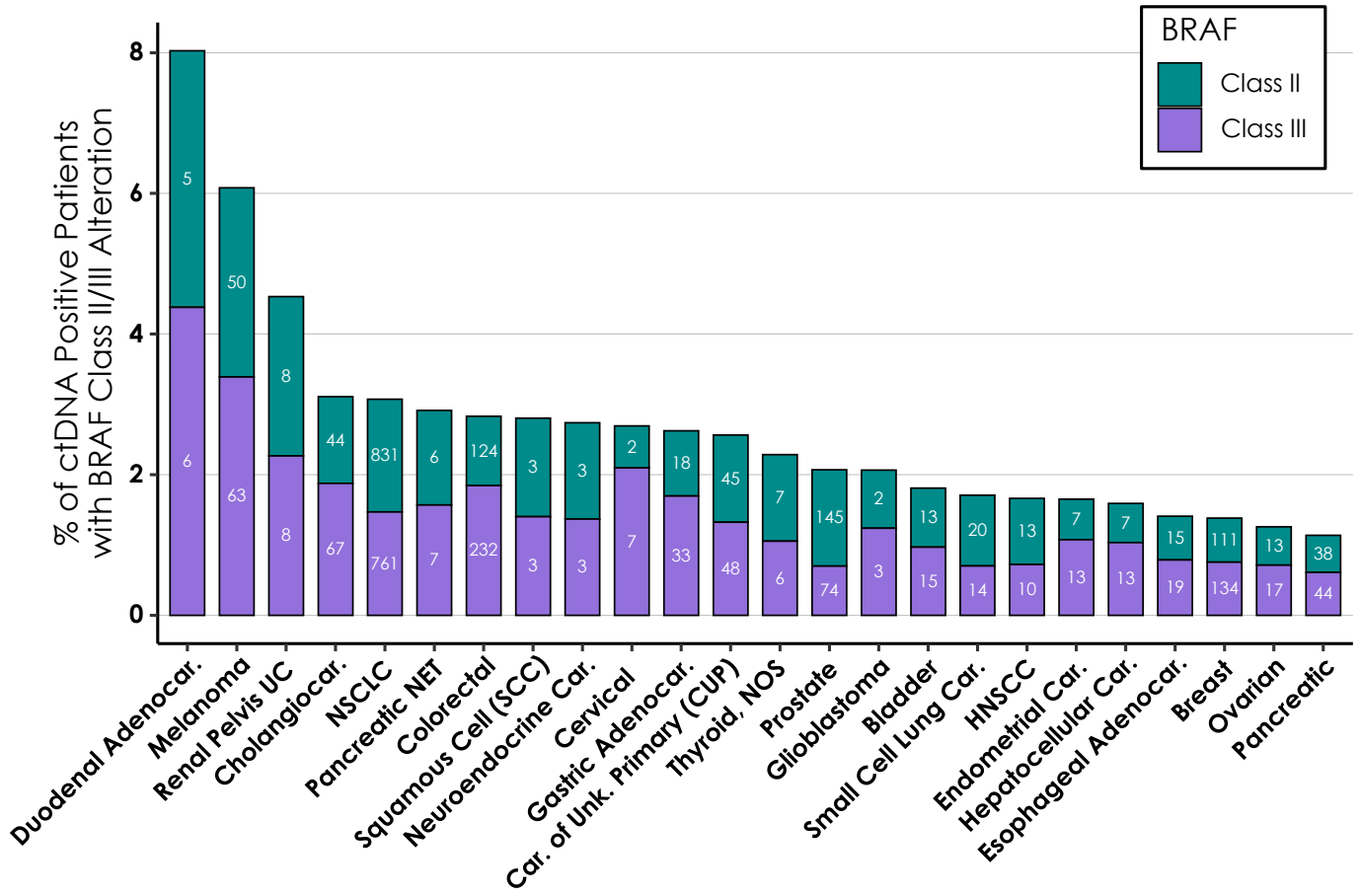
- **2.2%** of ctDNA positive patients had BRAF Class II or III



% of Oncogenic BRAF Alterations

Across all tumor types, liquid biopsy analysis in GuardantINFORM™ identified that the majority of patients with BRAF alterations have Class II & III alterations versus previous public sources based on smaller sample set showed a minority

BRAF Class II & III Alterations are Common Across Tumor Types



white labels indicate the # of patients
Figure includes tumor types with:
- ≥ 130 tested patients & ≥ 2% BRAF Class II/III or
- ≥ 1,000 tested patients & ≥ 1% BRAF Class II/III

A broad survey identified many tumor types with BRAF Class II & III occurrence rates > 1%

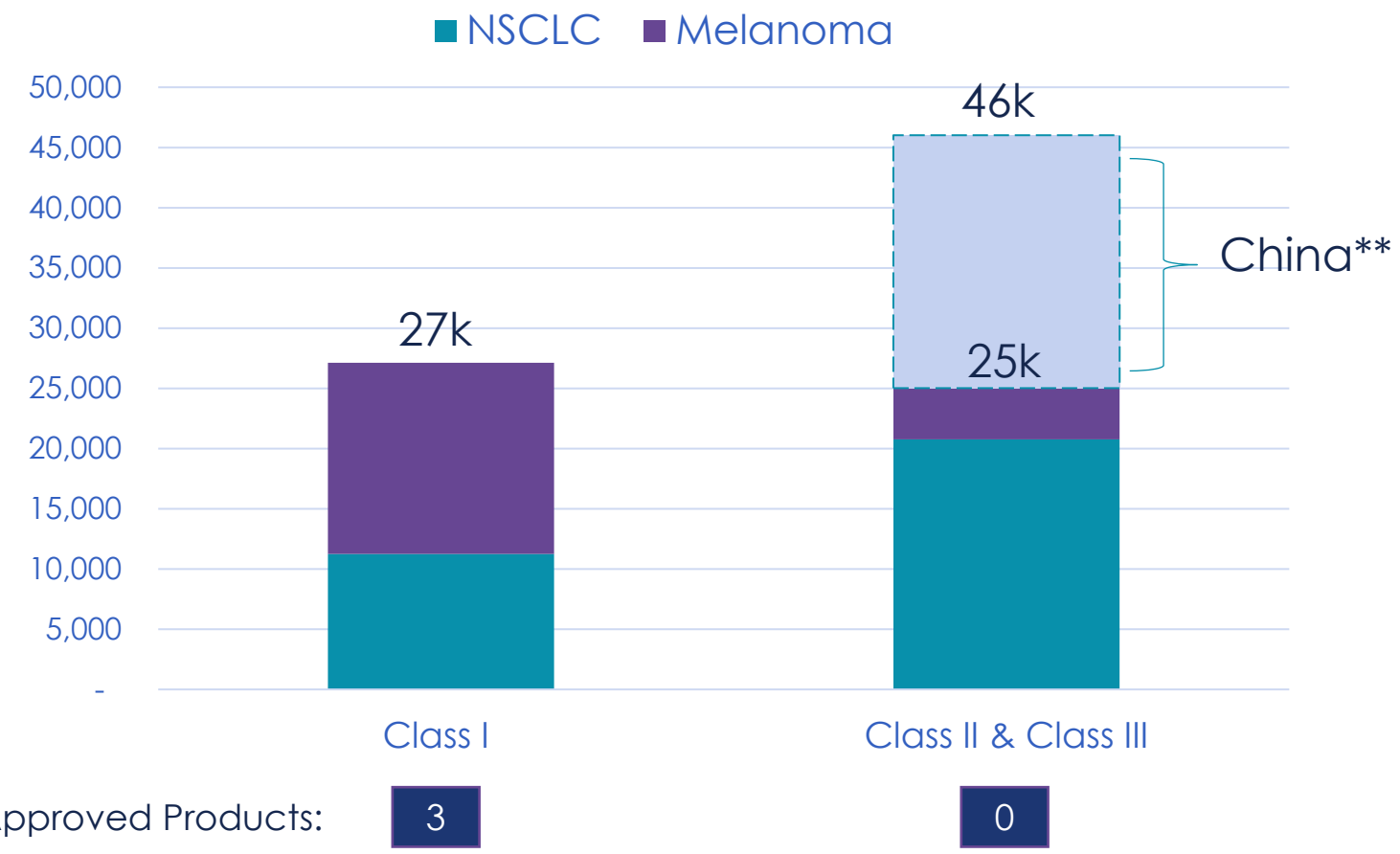
Tumor types with >100 patients each with BRAF Class II & III alterations in GuardantINFORM™:

- NSCLC
- Colorectal
- Breast
- Prostate
- Melanoma
- Cholangiocarcinoma

BRAF Class II & III alterations represent a sizable unmet need across a variety of tumor types

Class II & III Population **Across Tumor Types** is **Greater than** Class I But Without Any Approved Drugs

Patients with BRAF alterations for NSCLC & Melanoma*



Approved Products: 3 | 0

- 2020 sales of the 3 approved products for Class I BRAF alterations were $1.8B
  - 20% growth from 2019 sales
- Substantial opportunities for growth
  - Class II & III may have higher pricing, in-line with newer drug launches
  - Class II & III drug may not require combination therapy and/or have better profile
  - Broader use of NGS identifying more Class II & III patients
  - Additional tumor types with significant prevalence

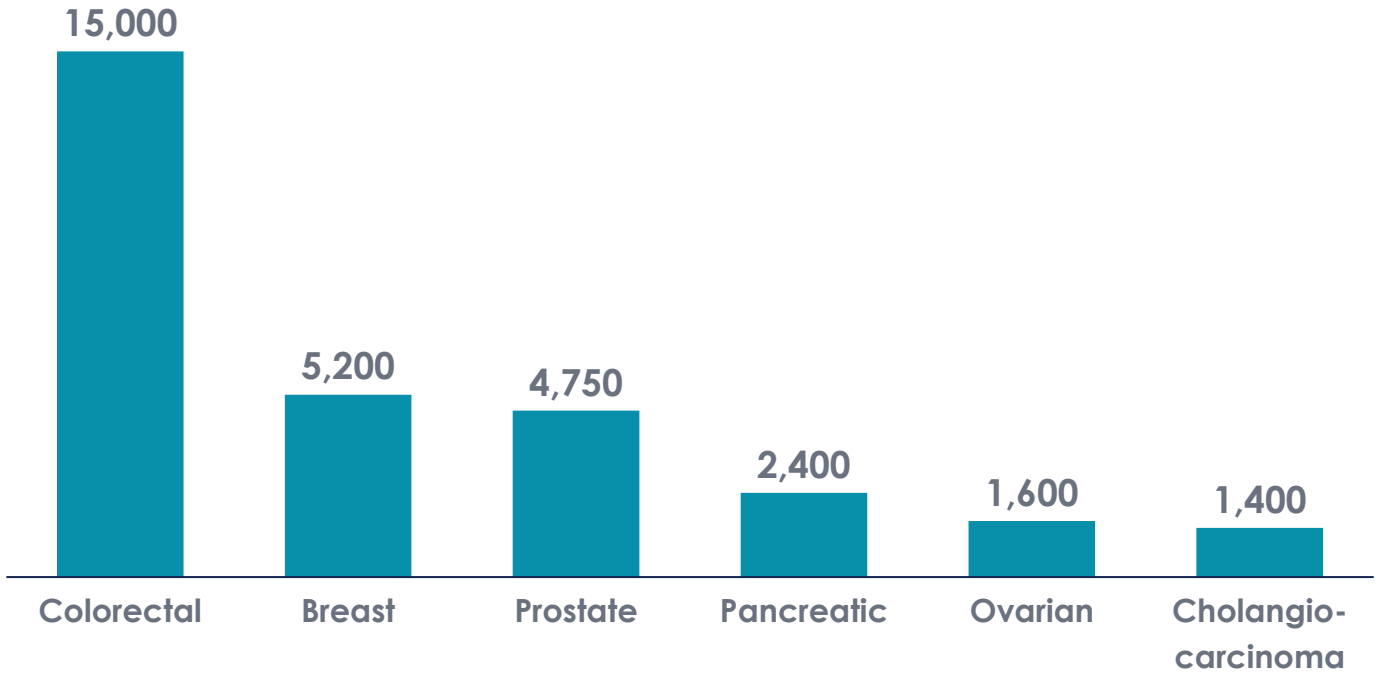
*US, EU5 and Japan; Stages IIIb and IV for NSCLC and Stage IV for Melanoma
** Stage IIIb and IV NSCLC in Urban Markets only

Kinnate calculations based on 2021 Kantar data and data generated from 2021 genomic landscape study with Guardant Health utilizing GuardantINFORM™

BRAF Program Opportunities for Expansion

Opportunities to Expand Beyond Current Target of ~46k patients

Additional Tumor Types with significant BRAF Class II or Class III alteration prevalence:



- Additional opportunities in various cancer types beyond NSCLC & Melanoma with Class II / Class III alterations
- Earlier treatment lines and less advanced disease settings
  - 2,700 patients have **Stage IIIa NSCLC** with Class II & Class III alterations
  - 3,200 patients have **Stage III** Melanoma with Class II & Class III alterations
- Class I BRAF alterations, including both first line and second line for intrinsic and acquired resistance
  - 27,000 patients have advanced NSCLC and Melanoma with Class I alterations + China
  - ~25% of acquired resistance may be dimer based
- Expanding into other geographies with high disease burden (e.g. South Korea, Australia, Canada)

Assumes unresectable or advanced metastatic disease. Kinnate calculations based on Kantar data, 2021 DRG data and data generated from 2021 genomic landscape study with Guardant Health utilizing GuardantINFORM™

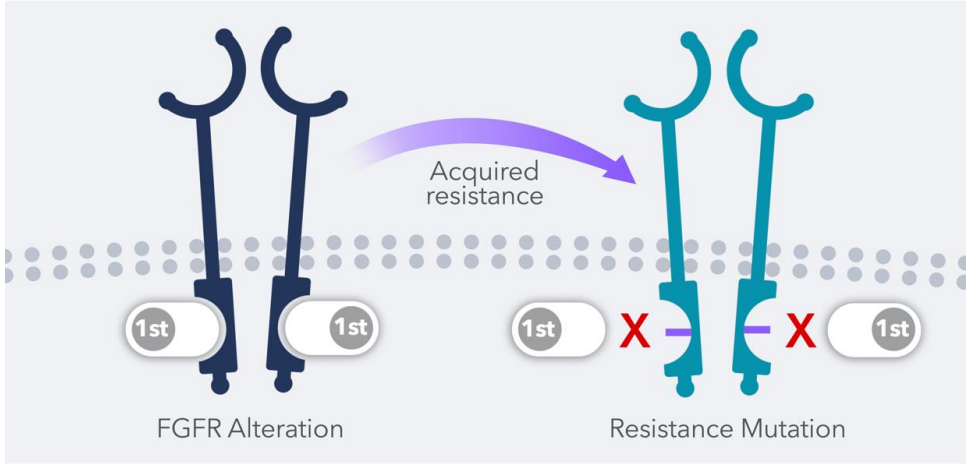
FGFR2/3 Program

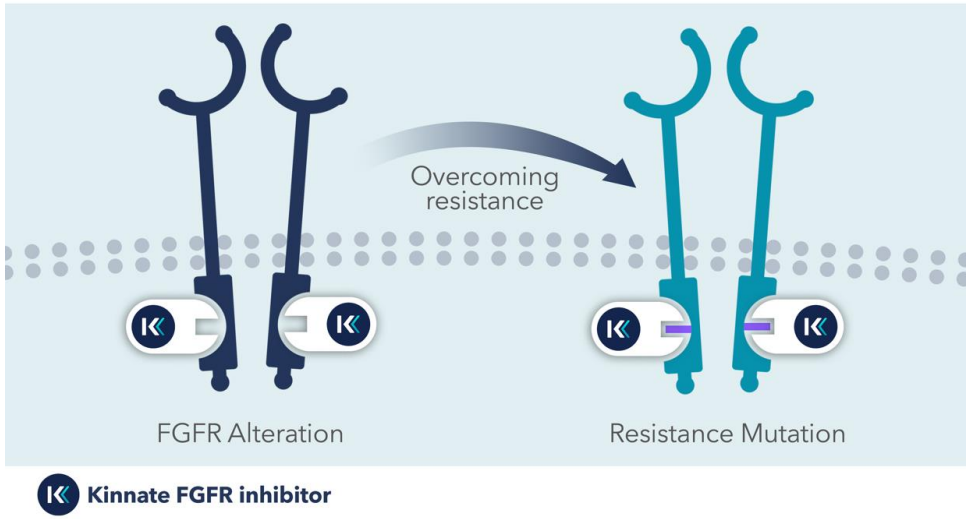
KIN-3248

Kinnate FGFR2/3 Inhibitor Program

KIN-3248 Directly Targets FGFR2 & FGFR3 Driver Alterations and Acquired Resistance Mechanisms

- Acquired resistance limits clinical benefit of approved & In-development FGFR inhibitors
- KIN-3248 is a potent & highly-selective, covalent FGFR inhibitor that targets:
  - FGFR2 & FGFR3 driver alterations in ICC & UC, and other tumor types
  - Known & predicted 'on target' FGFR2 & FGFR3 kinase domain mutations that confer clinical resistance (e.g. gatekeeper & molecular brake)
  - FGFR1, R2 & R3 isoforms, thereby reducing opportunities for bypass resistance



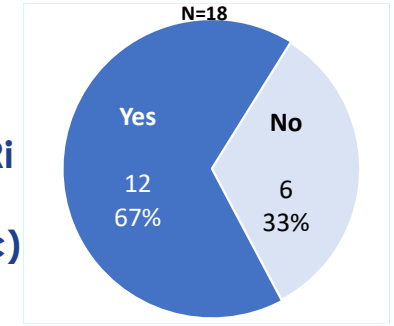


Meaningful On-Target Acquired Resistance to FGFR Inhibitors

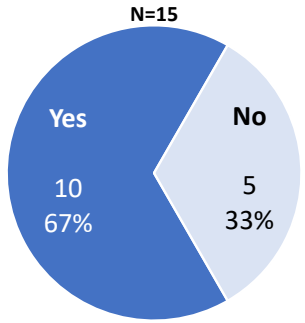
67% of FGFRi-treated ICC Patients Developed FGFR2 Kinase Domain (KD) Mutations at Progression

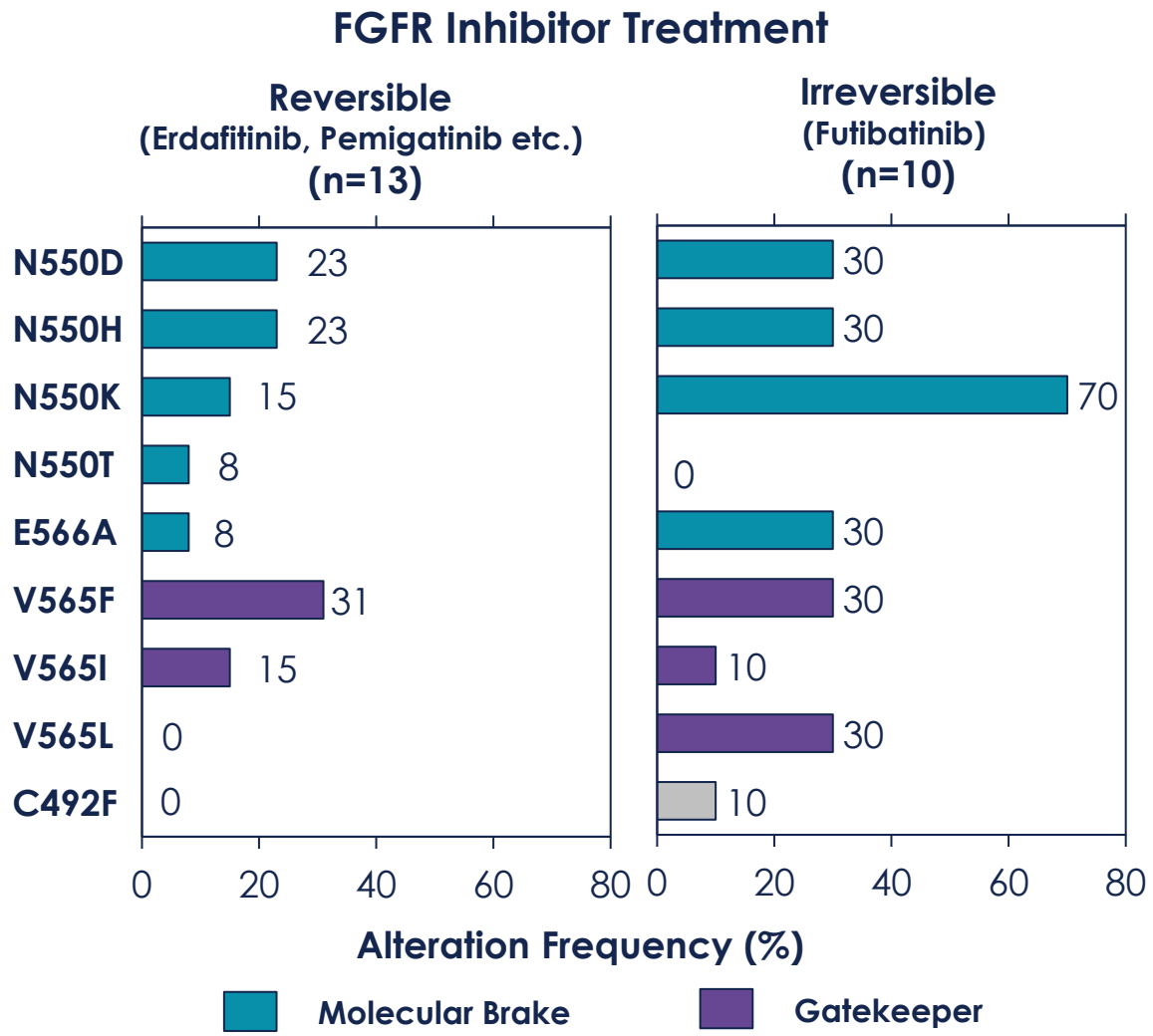
FGFR2 Kinase Domain Mutation

Reversible FGFRi (Erdafitinib, Pemigatinib, etc)



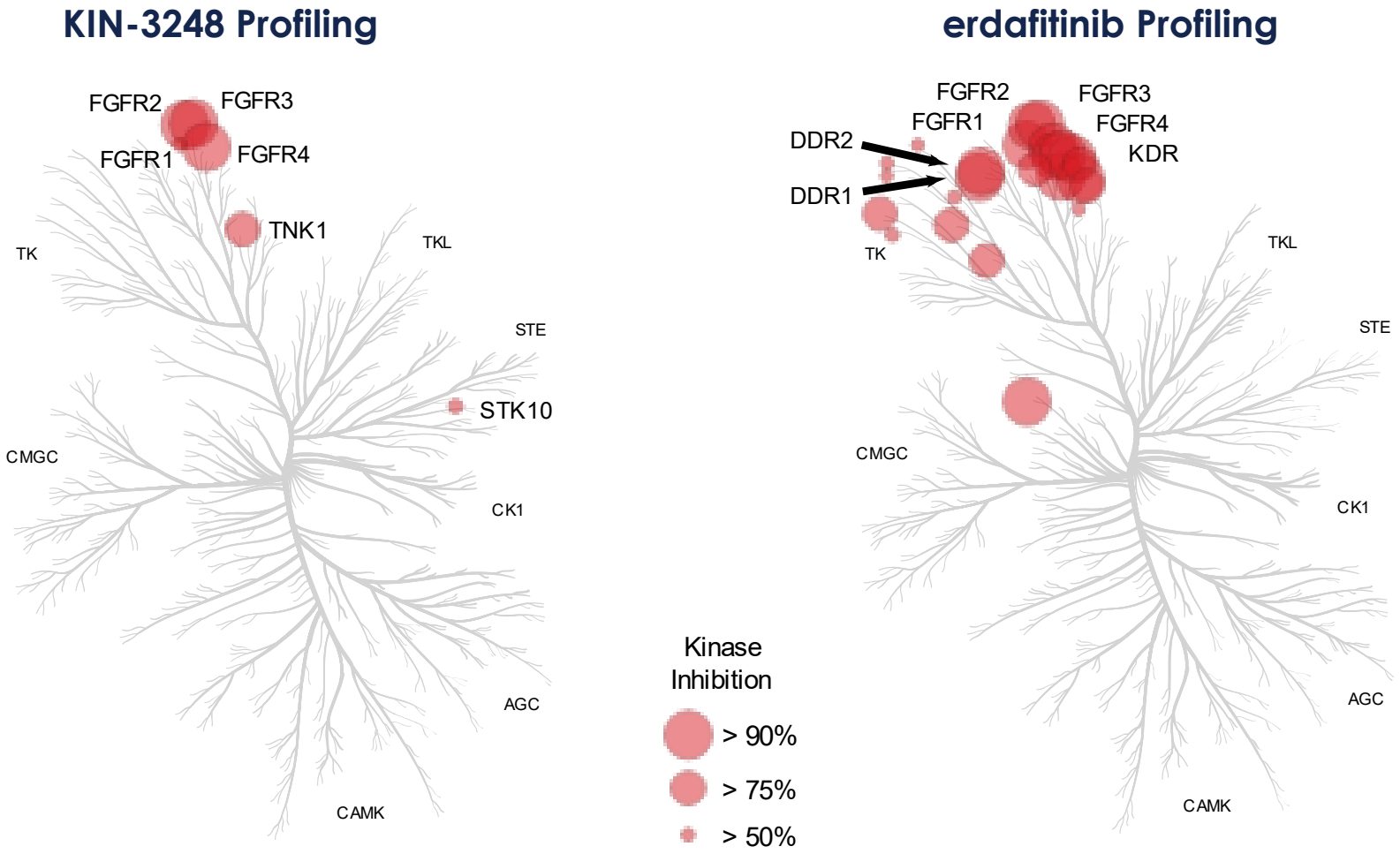
Irreversible FGFRi (Futibatinib)





Adapted from: Goyal et al., Landscape of Acquired Resistance to Selective FGFR Inhibitors in FGFR2 Fusion or Rearrangement+ Cholangiocarcinoma. **EORTC-NCI-AACR Symposium (October 2020).** Analysis includes Reversible FGFR inhibitor treated patients (n=13) and Irreversible FGFR inhibitor treated patients (n=10; all patients received futibatinib)

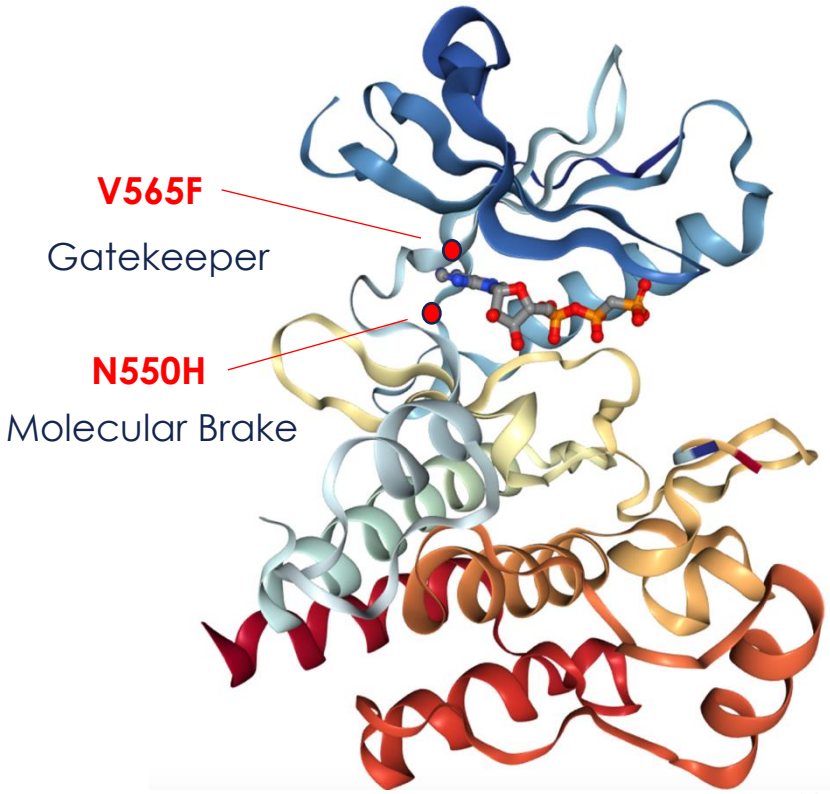
KIN-3248 Displays a Selective & Differentiated Kinase Profile



- Kinome profiling @ 1µM across 322 kinases at Carna Biosciences Corp
- Erdafitinib is approved for treatment of FGFR2 and FGFR3 alteration-driven urothelial cancer

KIN-3248 is Differentiated in Enzymatic Assays

Overcomes FGFR2 & FGFR3 Gatekeeper and Molecular Brake Resistance Mutations



Kinase Domain	Kinase Domain Alteration	Janssen erdafitinib IC_{50} (nM)	Incyte pemigatinib IC_{50} (nM)	Taiho futibatinib IC_{50} (nM)	Kinnate KIN-3248 IC_{50} (nM)
FGFR1 WT	-	0.2	0.4	2.1	3.9
FGFR2 WT FGFR2 V565F FGFR2 N550H	- Gatekeeper Mol. Brake	0.15 330 4.1	0.4 >500 19.8	1.4 >500 36.4	5.3 20.8 22.8
FGFR3 WT FGFR3 V555M FGFR3 K650M	- Gatekeeper Activ. Mut.	0.7 137 3.5	1.5 >500 20	5.3 324 8.3	9.7 24.3 4.6
Ratios of Resistance Mutations Compared to Wild Type (WT) (Fold Difference in IC_{50})					
R2 V565F / WT R2 N550H / WT	Gatekeeper Mol. Brake	2200X 27X	1250X 50X	385X 31X	**4X** **4X**
R3 V555M / WT R3 K650M / WT	Gatekeeper Activ. Mut.	188X 5X	>333X 13X	61X 1.6X	**3X** **0.5X**

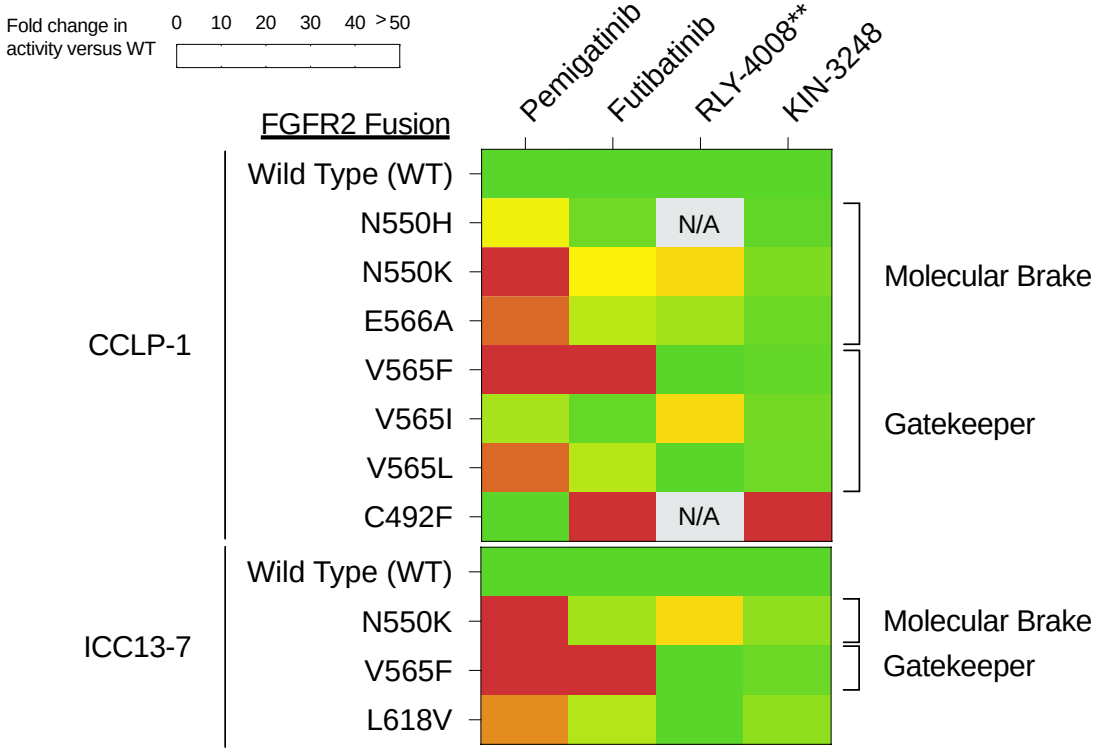
Note: Ratios <10x = equivalent kinase inhibition of either the resistance mutations or corresponding WT kinase. Ratios > 10x = substantial loss of activity against the indicated resistance mutations compared to the corresponding WT kinase

- KIN-3248 showed inhibition of the gatekeeper and molecular brake mutations when compared to the FDA approved and clinical candidate FGFR inhibitors

KIN-3248 is Active Against FGFR2 Resistance Mutations in ICC

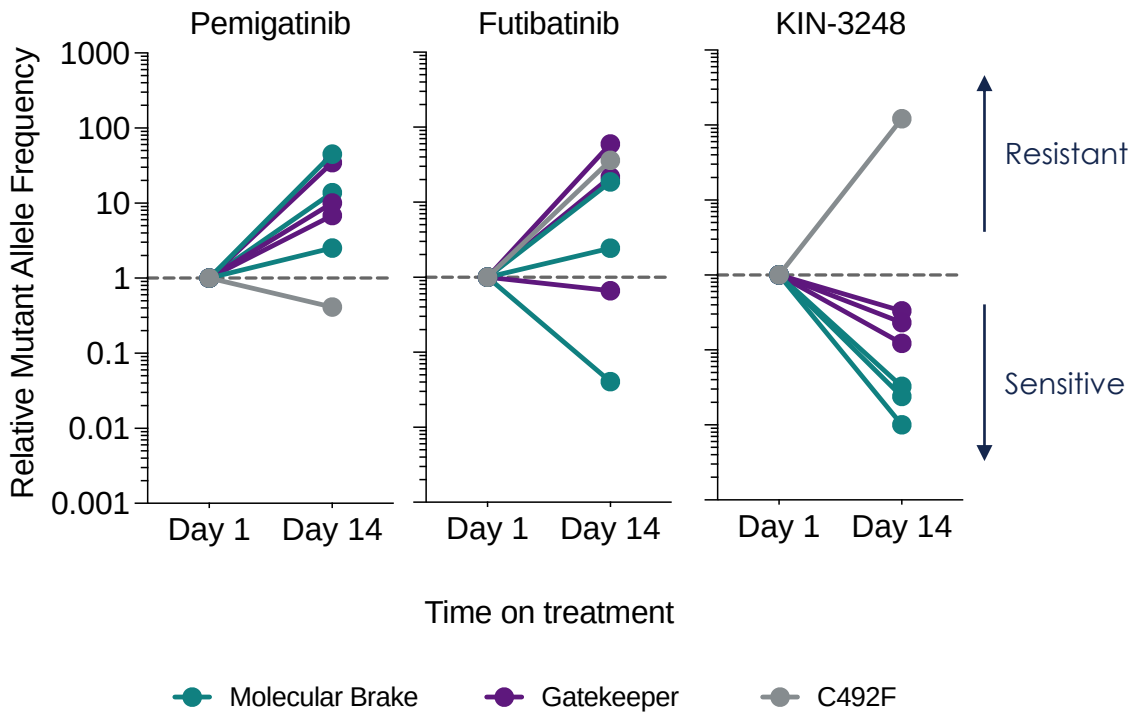
Inhibition of key mutations that drive resistance to first generation FGFR inhibitors

KIN-3248 inhibits the growth of FGFR2 fusion-positive ICC cells harboring secondary resistance mutations



- Additional preclinical studies conducted in ICC FGFR2 fusion models show that infigratinib is resistant to N550K and V565F mutations (data not shown)

KIN-3248 prevents the outgrowth of clinically-relevant FGFR2 resistance clones

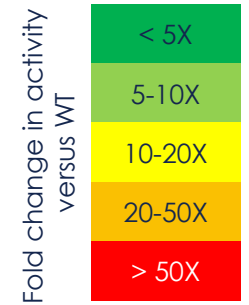


**Note: Analysis includes Kinnate-generated data for clonal competition & cellular sensitivity experiments, apart from data for RLY-4008's profile that was abstracted from Relay's S1 public SEC filing (https://www.sec.gov/Archives/edgar/data/0001812364/000119312520192936/d904779ds1a.htm#rom904779_12)

KIN-3248 is also Active Against FGFR3 Resistance Mutations in UC

KIN-3248 inhibits the growth of FGFR3 fusion-positive UC cells harboring secondary resistance mutations

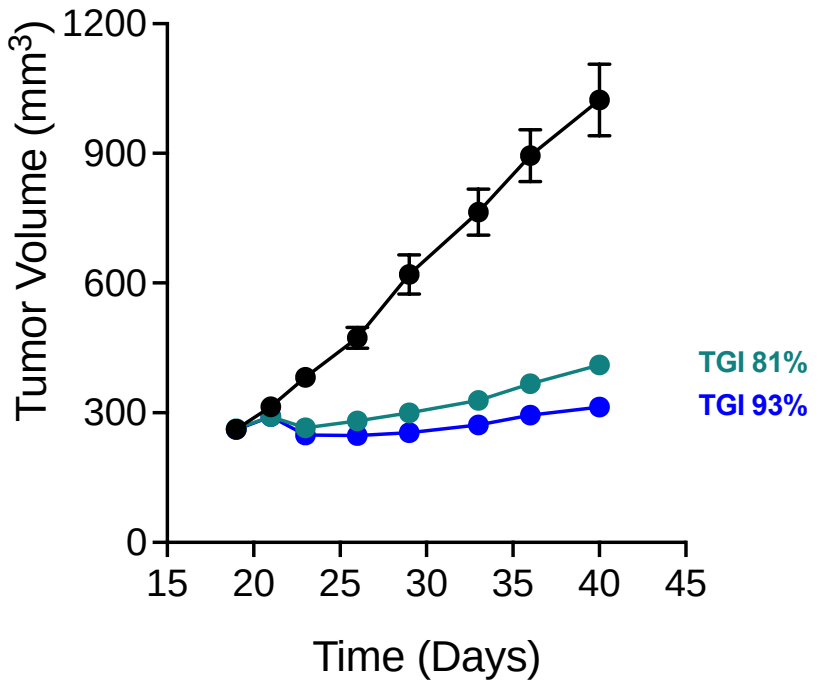
		Janssen Erdafitinib	Incyte Pemigatinib	BridgeBio / QED Infigratinib	Taiho Futibatinib	KIN-3248
FGFR3 Kinase Domain Alteration	N540K / R3 WT Molecular Brake					
	V555M / R3 WT Gatekeeper					
	K650M / R3 WT Activation Loop					



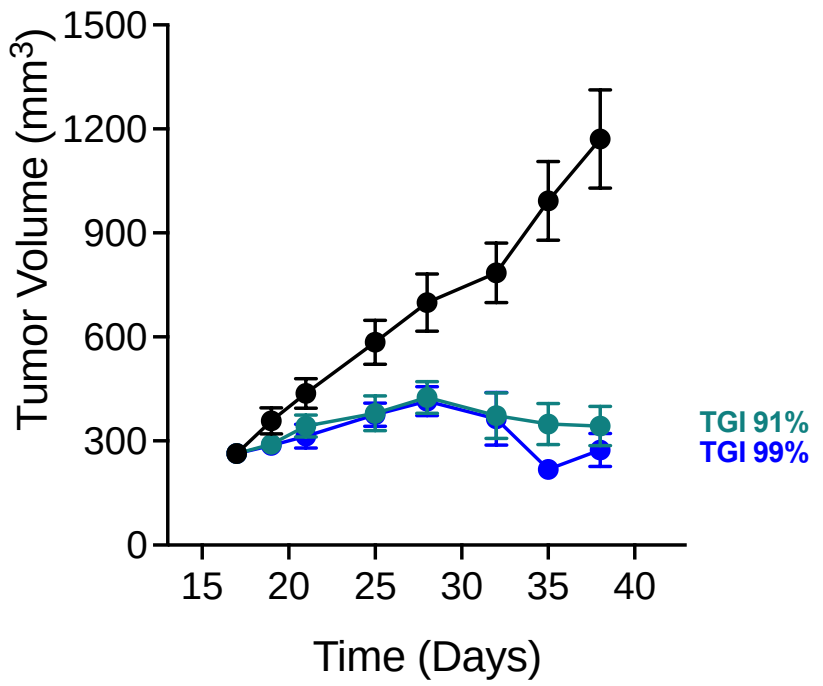
- KIN-3248 showed inhibition of both FGFR3 gatekeeper, molecular brake and activation loop resistant mutations when compared to the FDA approved and clinical candidate FGFR inhibitors

KIN-3248 is Efficacious Against Primary FGFR2 & FGFR3 Oncogenic Driver Alterations *In Vivo*

FGFR2 Amplified / Fusion+ Gastric Cancer



FGFR3 Fusion+ Urothelial Cancer

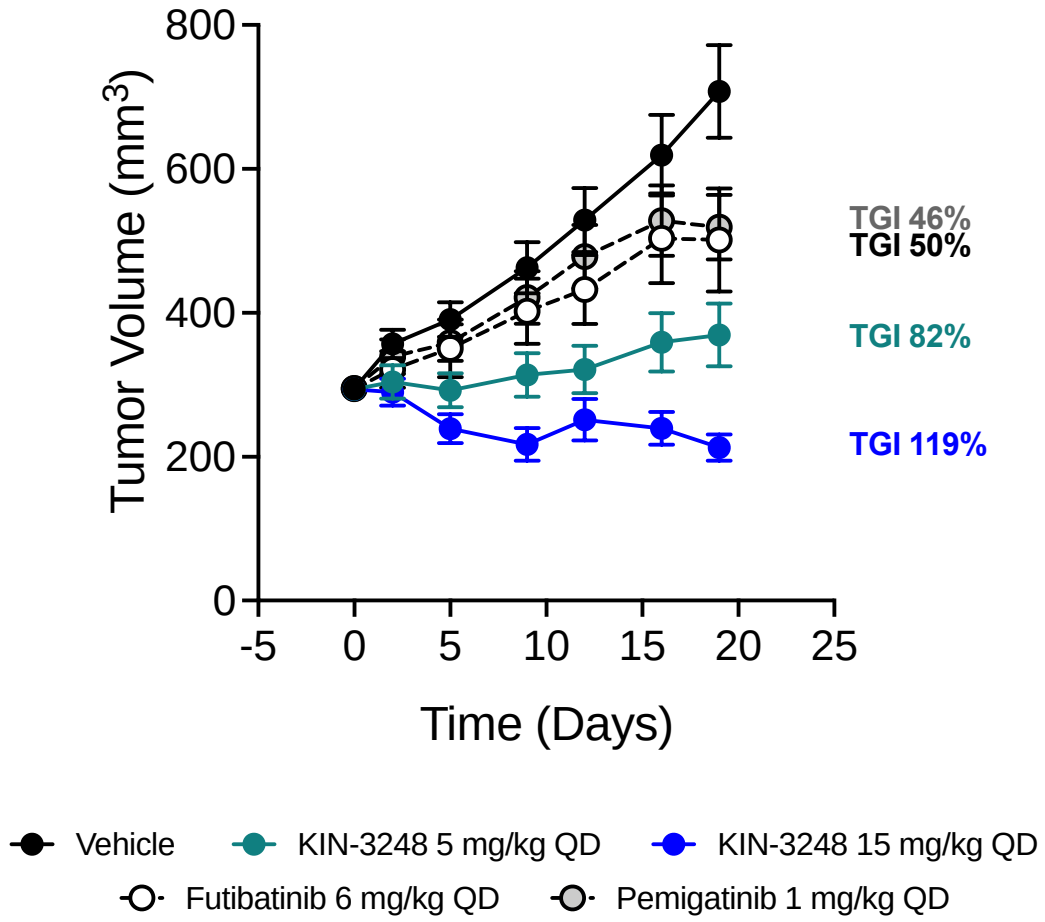


Vehicle — KIN-3248 5 mg/kg QD — KIN-3248 15 mg/kg QD

- Continuous daily dosing of KIN-3248 is well-tolerated and effective in the treatment of FGFR2- and FGFR3-driven human cancer cell line-derived tumors *in vivo*

Tumor growth inhibition (TGI) was calculated as follows: TGI = $(1 - (TV_f\text{-}TV_i)_{treated} / (TV_f\text{-}TVi)_{control})) \times 100\%$, where TV_f is the final tumor volume and TV_i is the initial tumor volume.

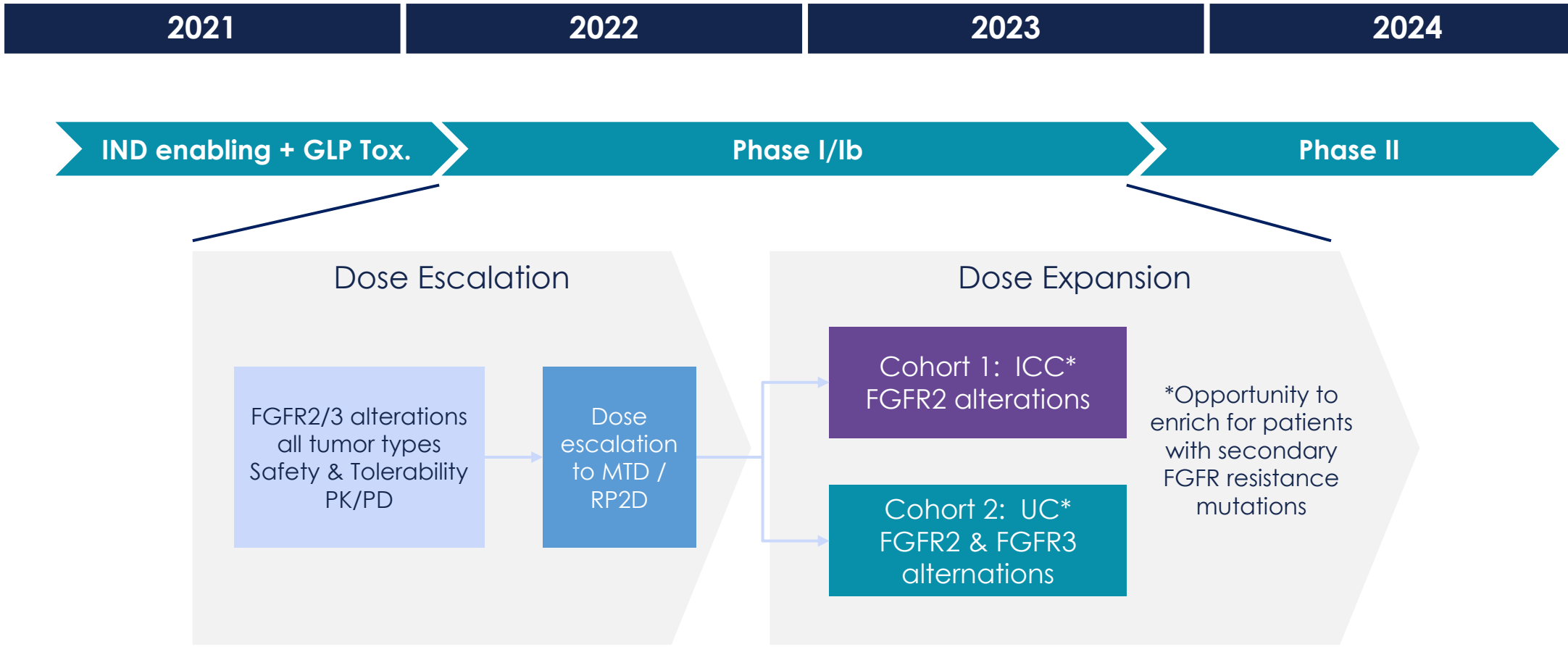
KIN-3248 is Efficacious Against Secondary, Acquired FGFR2 Gatekeeper Resistance Mutation *In Vivo*



- The gatekeeper mutation limits efficacy of approved and clinical stage FGFR inhibitors, pemigatinib and futibatinib, respectively
- Consistent with *in vitro* findings, KIN-3248 led to tumor growth inhibition and regressions in a FGFR2 amplified / V565L gatekeeper mutation-positive gastric cancer patient-derived xenograft model
  - Acquired secondary resistance mutation following treatment with AZD4547 (pan-FGFRi)

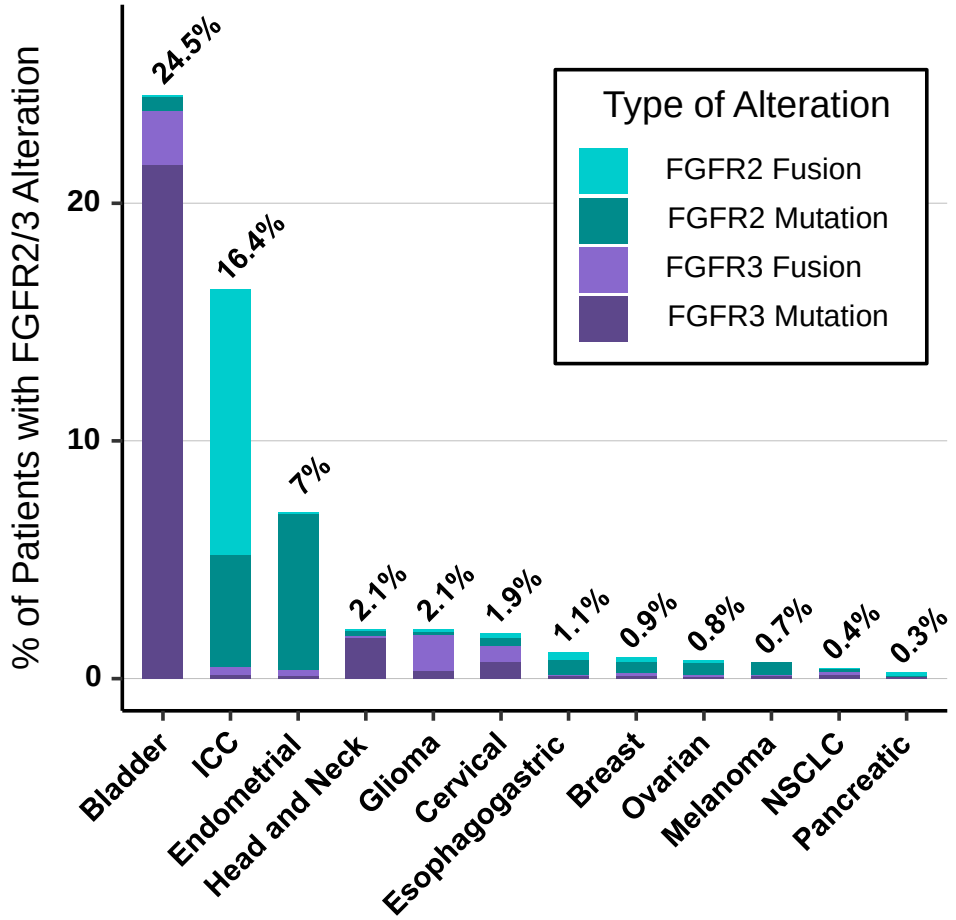
Tumor growth inhibition (TGI) was calculated as follows: TGI = $(1 - (TV_f\text{-}TV_i)_{treated} / (TV_f\text{-}TVi)_{control})) \times 100\%$, where TV_f is the final tumor volume and TV_i is the initial tumor volume.

KIN-3248 Expected Clinical Development Plan



FGFR Market Opportunity

Occurrence Rates of FGFR2 & FGFR3 Alterations by Tumor Types



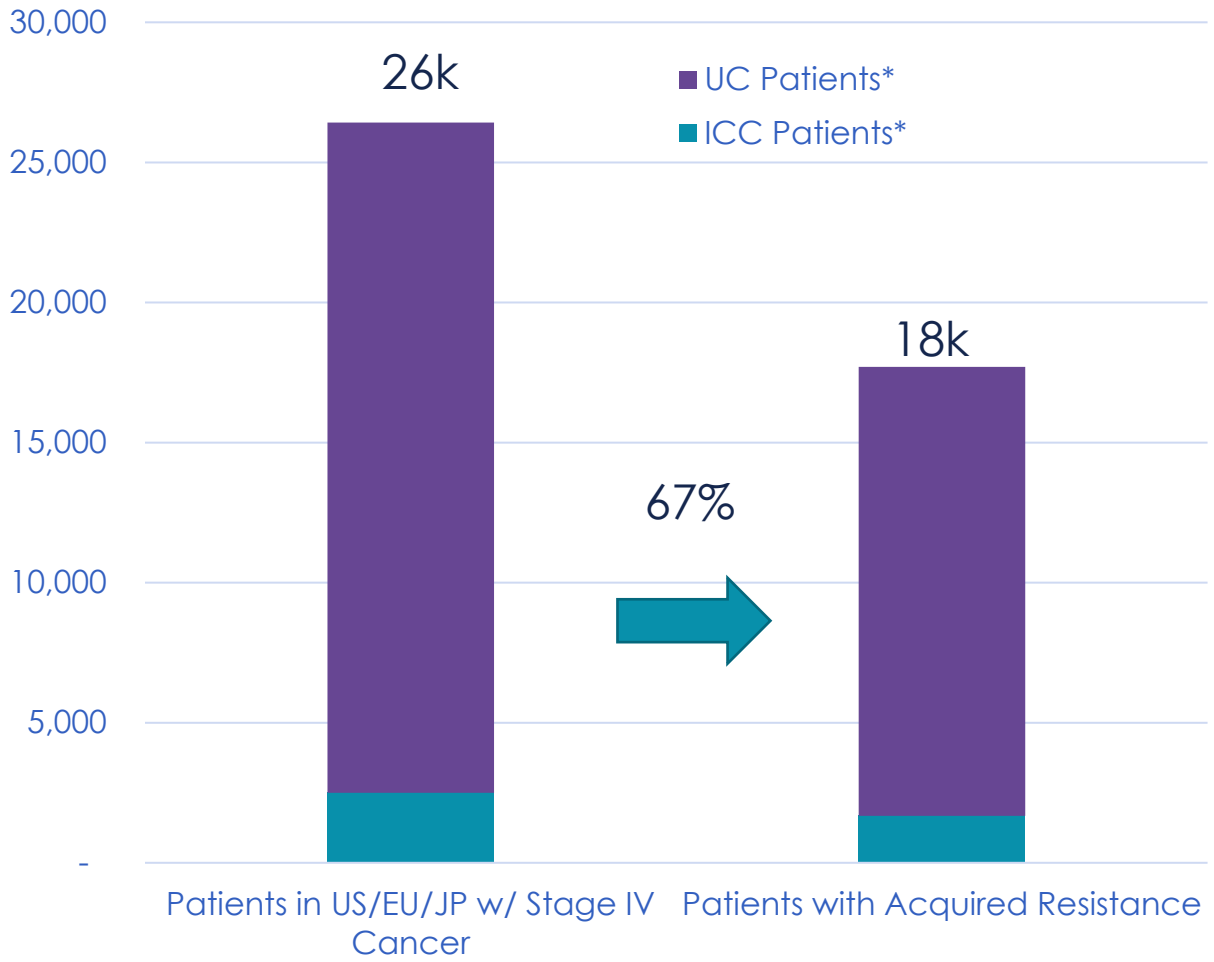
- KIN-3248 has been designed to **target both FGFR2 and FGFR3 alterations** which includes fusions, mutations (indels and SNVs) and other rearrangements which are likely oncogenic drivers of tumors
- While patients with solid tumors do have FGFR2/3 amplifications, they are often not the primary drivers of tumors
- FGFR alterations are most common in Bladder cancer (UC) and ICC which are our primary focus
  - They have also been found in other tumor types like endometrial, breast etc.

Data generated from AACR GENIE Project Data: Version 10.0-public **Powering Precision Medicine Through An International Consortium.** Cancer Discov 7(8): 818-831, 2017 (https://genie.cbioportal.org/)

Analysis includes mutations that are annotated as at least ***Likely Oncogenic*** in oncokb.org and rearrangements including fusions, intergenic and intragenic events. Unknown frame fusions were included, but out-of-frame fusions were not included.

FGFR Market Opportunity – UC & ICC Patients in US, EU & Japan

Patients with Active Disease



*Reflects FGFR2 or FGFR3 Alterations

Opportunities for Growth

- FGFR alterations have been found in other tumors (e.g. breast)
- NGS technologies identifying additional patients with FGFR alterations
- Geographic expansion (e.g. China)

Kinnate calculations based on Kantar data and data generated from AACR GENIE Project Data: Version 10.0-public **Powering Precision Medicine Through An International Consortium.** Cancer Discov 7(8): 818-831, 2017 (https://genie.cbioportal.org/); Adapted from: Goyal et al., Landscape of Acquired Resistance to Selective FGFR Inhibitors in FGFR2 Fusion or Rearrangement+ Cholangiocarcinoma. **EORTC-NCI-AACR Symposium (October 2020).**

Kinnate Discovery Engine

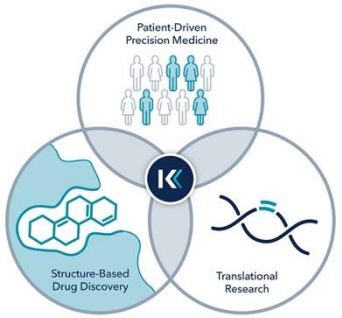
Research Capabilities

KIN004 – CDK12 Program

Cancer Biology & Genomics Drives Drug Discovery Opportunities

Continued Advancements of our Understanding of Disease Reveal our Next Generation Drug Targets

Our Focus: We remain focused on validated oncogenic drivers that directly inform patient selection strategies and are associated with enhanced probabilities of technical, clinical & regulatory success

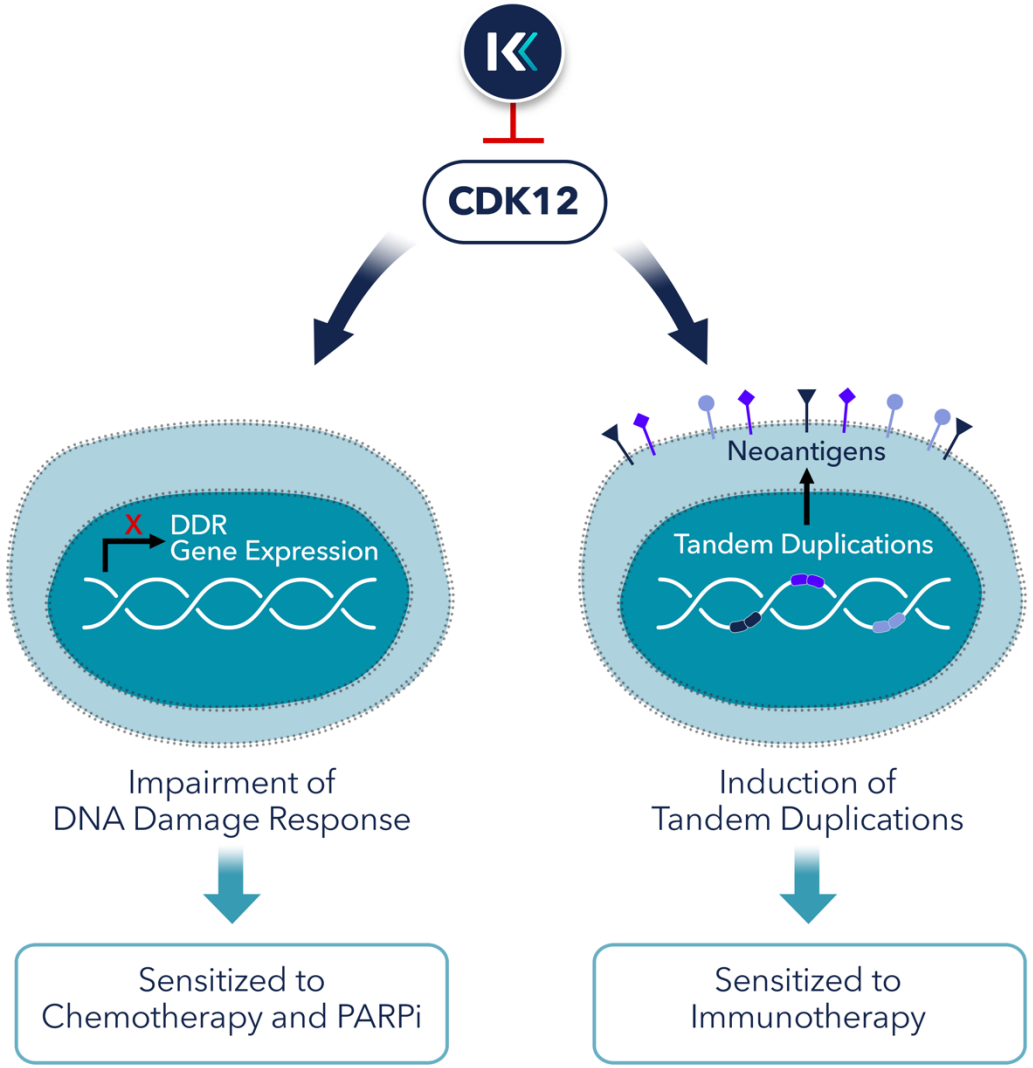


Our Approach: Our Kinnate Discovery Engine, fueled by our small molecule structure-based drug design capabilities and translational research strategies, will serve as the foundation for continued success

Not Kinnate's Focus: 'Pure-play' Immuno-oncology approaches, tumor micro-environment directed strategies, microbiome-based therapies, cellular therapies & cancer vaccines, and biology that is non-tractable with current technology

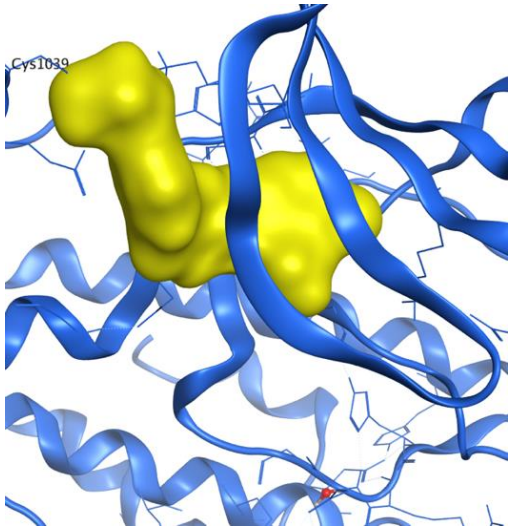
CDK12 Inactivation Impairs DNA Damage Response and Induces Tandem Duplications

- CDK12, a RNA polymerase II C-Terminal Domain (CTD) kinase, is an essential regulator of various DNA damage response (DDR) genes
- Inhibition of CDK12 sensitizes tumors to DNA damaging agents and induces synthetic lethality in both DDR-deficient and the greater unmet need in DDR-proficient tumors
- CDK12-mutant ovarian and prostate cancers demonstrate an accumulation of large Tandem Duplications (TDs) resulting in accumulation of fusion-induced neoantigens (FINAs) in cancer cells

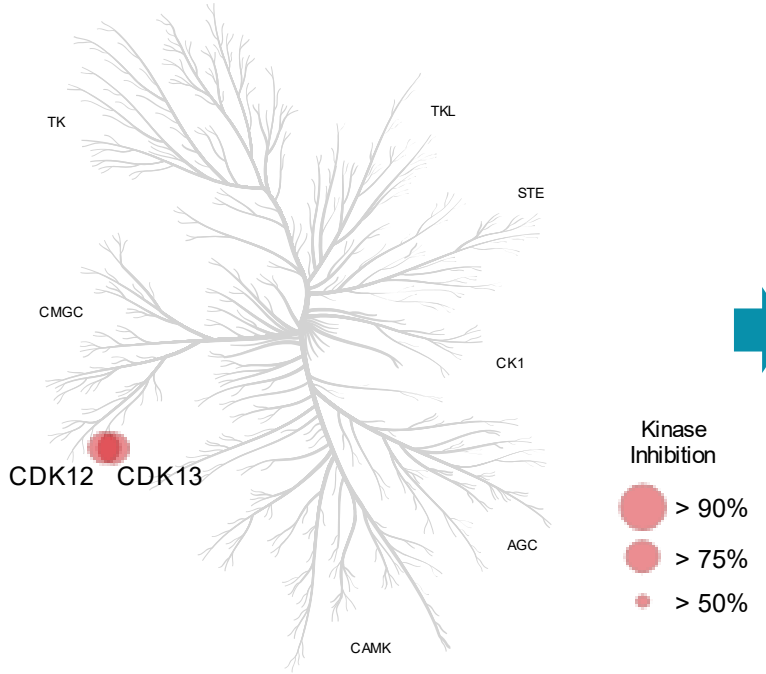


Program Has Demonstrated Selective CDK12 Inhibition

KIN004
Co-Crystal Structure



Kinome Profile – KIN004



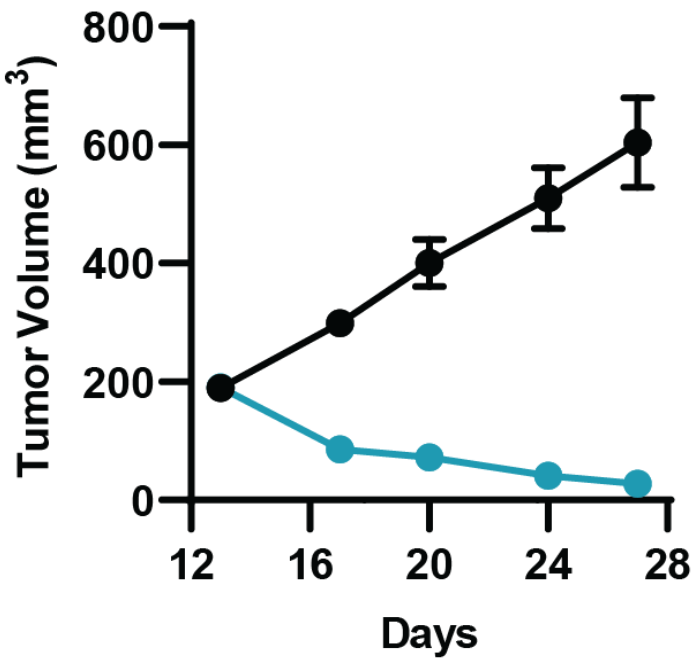
CDK Family Selectivity – KIN004

Kinase	KIN004 IC_{50} (nM)
CDK12	**97**
CDK2	**5104**
CDK7	**3913**
CDK9	**3952**
Ratios (Fold Difference in IC_{50})	
CDK12/CDK2	>50X
CDK12/CDK7	>40X
CDK12/CDK9	>40X

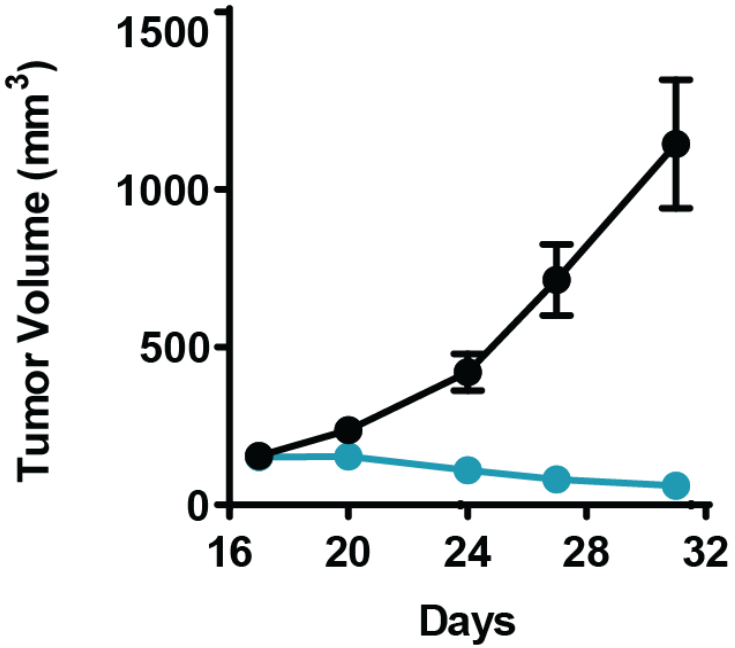
- KIN004 demonstrated selective CDK12 inhibition compared to highly homologous CDK2, CDK7 and CDK9 family members
- Structure-based design enabled by Kinnate proprietary co-crystal structure

Tumor Regressions Demonstrated with Selective Inhibition of CDK12

***In Vivo* Efficacy – HCC70 (BRCA^{WT})**



***In Vivo* Efficacy – OVCAR3 (BRCA^{WT})**



Note: HCC70 breast tumors (left) and OVCAR-3 ovarian tumors (right) represent BRCA 1/2 WT cancers that were DDR-proficient and were not sensitized to PARP inhibitor treatment

Kinnate: Kinase Inhibitors for Genomically Defined Cancers

Striving to Expand the Promise of Precision Medicine in Oncology

Programs

- Multiple compounds advancing to the clinic
- Lead RAF program in unserved population – Phase I initiated
- FGFR program targeting resistance
- R&D pipeline of additional undisclosed discovery programs

Platform

- Productive Kinnate Drug Discovery Engine

People

- Experienced management team
- Strong scientific collaborations
- Diverse board with biopharma expertise