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 ongoing and planned preclinical studies and 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. You may view our filings with the Securities Exchange Commission at their website (www.sec.gov). 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: Expanding on the Promise of Precision Medicine in Oncology

Kinase Inhibitors for Genomically Defined Cancers

Programs

- Clinical and preclinical assets targeting validated oncogenic drivers
- Lead RAF program (KIN-2787) targets large population not served by approved targeted therapy
  - Phase 1 initiated; Expansion of ongoing KN-8701 trial to include $NRAS^{Mut}$ Melanoma
- FGFR program (KIN-3248) targets significant unmet need of resistance to current FGFR inhibitors
  - Expect Phase 1 trial initiation in 1H 2022
- Multiple other compounds in pipeline, including CDK12 inhibitor
  - All programs developed in house with IP & commercial rights fully retained*

Drug Discovery Engine

- Productive drug discovery engine designed to optimize for speed and probability of success
  - 3 Years from inception to initial IND clearance
  - Goal of 1 IND a year

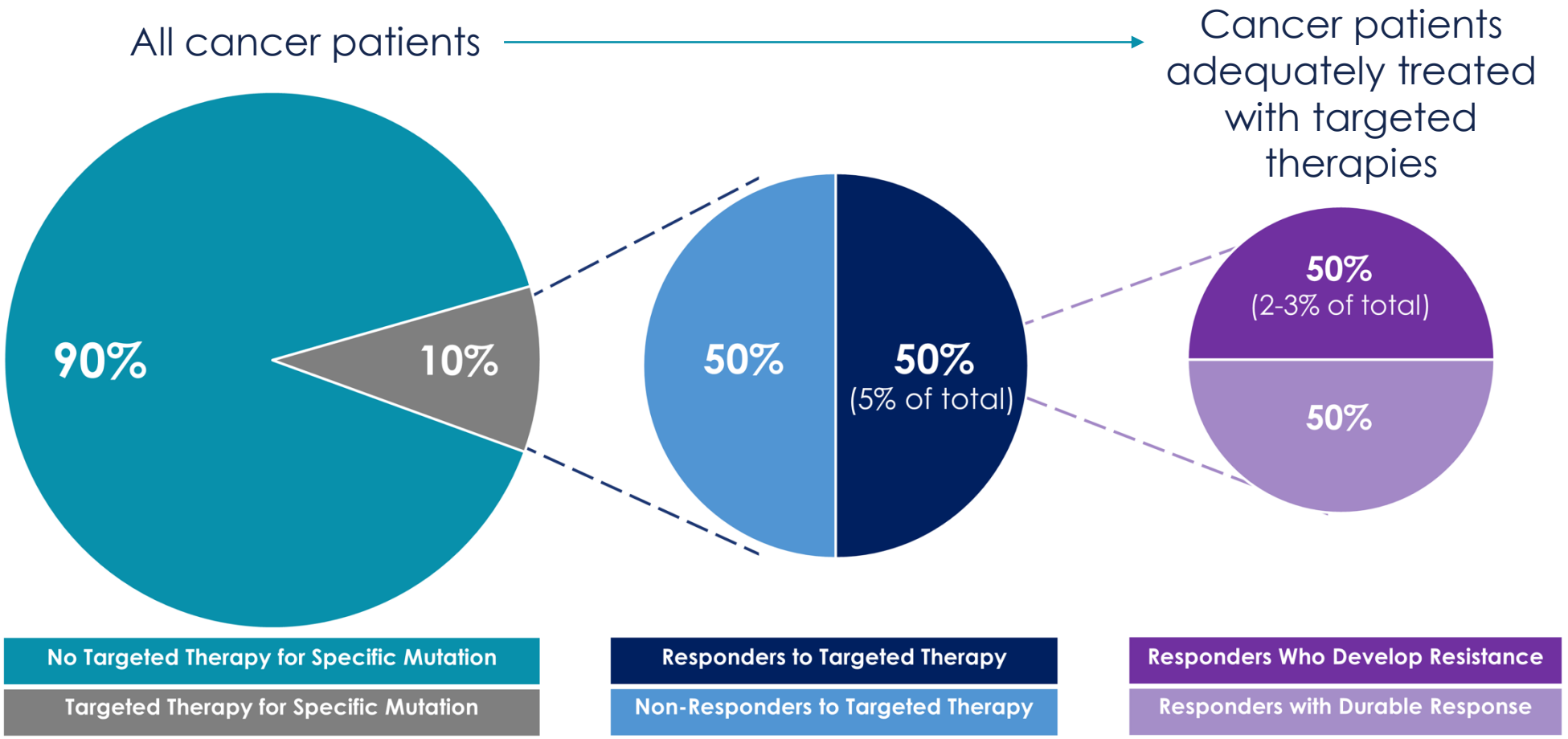
Drug Development Strategy

- Focused drug development strategy designed to maximize potential for success
  - Biomarker-driven approach
  - Continual translational research
  - Early global expansion

Well-Funded with ~$325MM on Hand**

*Greater China rights exclusively licensed to the China joint venture Kinnjiu Biopharma Inc. ("Kinnjiu"), of which Kinnate is the majority shareholder
**Cash and cash equivalents & investments as of December 31, 2021, exclusive of Kinnjiu's cash

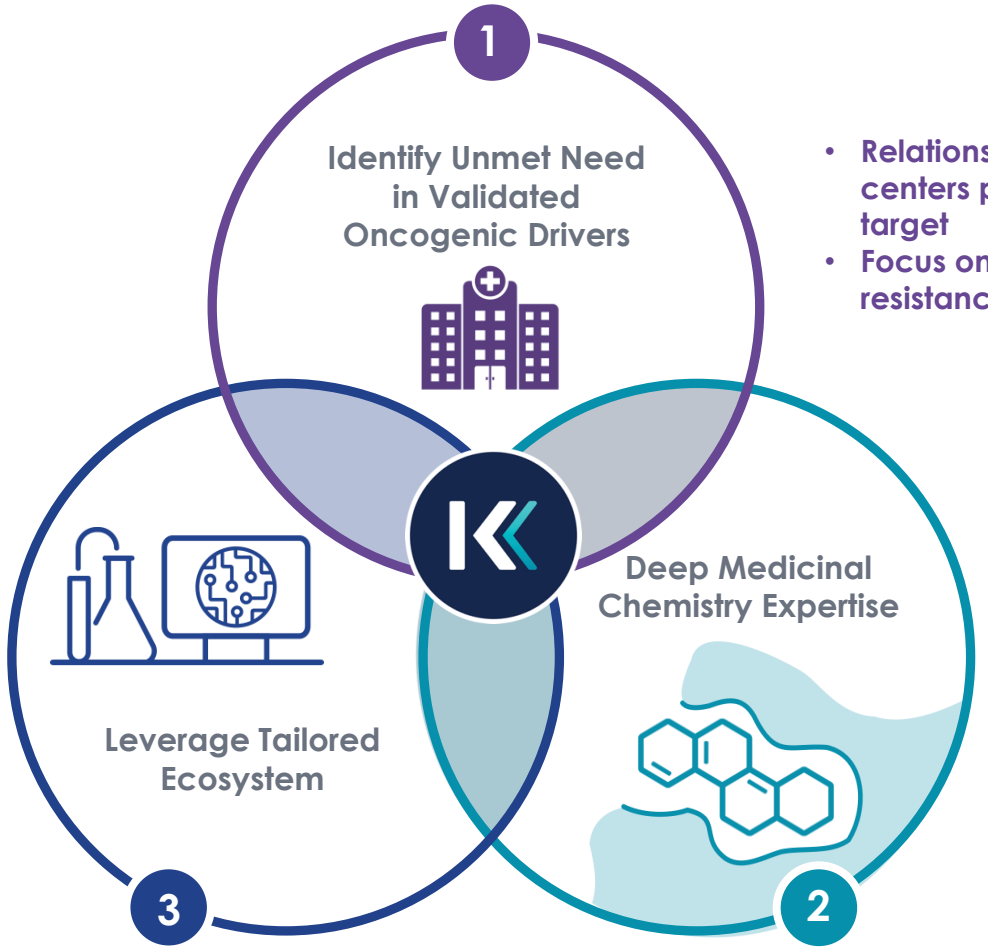
Expanding on the Promise of Precision Oncology



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

Reinventing Success in Drug Discovery

Improving Speed and Probability of Success through the Kinnate Discovery Engine



- **Relationships with premier academic centers provide insight into the drug target**
- **Focus on innate and acquired resistance and new patient populations**

- Over **70 Contracted Medicinal Chemists at CROs**
- Sponsored Research Agreements (SRA) with **prominent academic centers**
- Technologies include: bioinformatics, crystallography, organoid & xenograft models

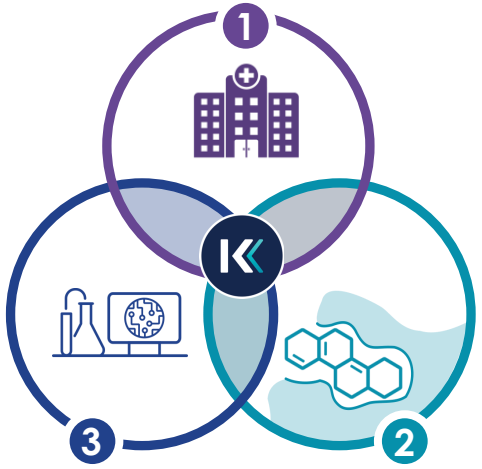
- **Structure based** small molecule drug discovery
- Deep understanding of **binding modes & biology** to achieve best in class product profile spanning:
  - **Alteration coverage**
  - **Selectivity**
  - **Pharmaceutical properties (ex. PK, DDI)**

Since 2018, 1 Product Candidate in Clinic (2021); 1 Other IND Cleared (2022)

Kinnate Discovery Engine in Action: KIN-3248 Case Study

Challenge: Design a FGFR Targeted Therapy that Covers Known FGFR2/3 Resistance Mutations

1 Existing relationships academic centers and KOLs provided insights into clinical resistance mutations to 1st gen. FGFR inhibitors prior to 2020 ENA publication

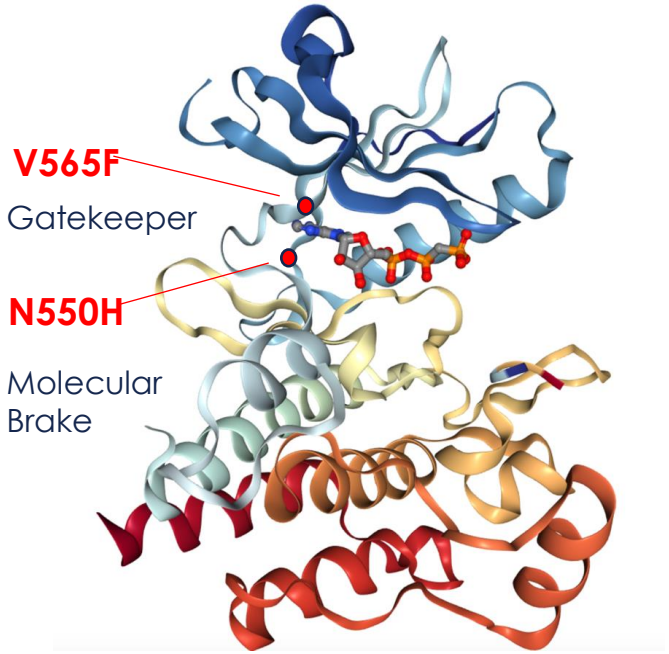


3 Leveraged:
- 20 Medicinal Chemist CRO FTEs
- SRA with MGH explored more than 20 in vitro and in vivo common resistance mutations
- Molecular modeling software: MOE, Schrodinger, Spotfire
- Molecular dynamics simulation servers

2 Drug Design Strategy for Broad Resistance Mutation Potency & Selectivity
- Potency: Compatible with Gatekeeper Mutation and accommodates Molecular Brake Loop movement
- Selectivity: Type II back pocket binding & irreversible Cysteine warhead

~2Yrs later*

FGFR Receptor & Product Candidate: KIN-3248

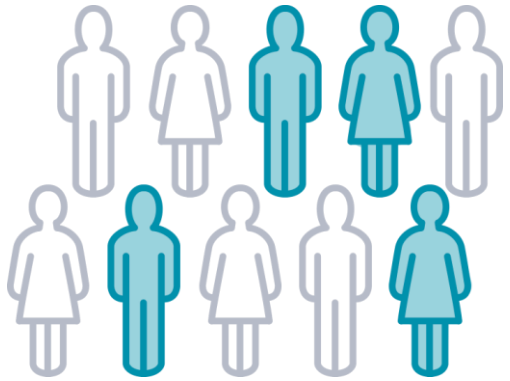


3248: Differentiated Profile
- Covers all common resistance mutations to FGFR2 & R3
- Highly selective, even against closely related kinases

*From start to Drug Candidate nomination

Reinventing Success in Drug Development

Focused Drug Development Strategy Designed to Maximize Potential for Success



Biomarker-driven Approach

- Focus on oncogenic driver as the biomarker
- Enrich for patient populations with a higher likelihood of response
- Potentially enables accelerated regulatory path

Continual Translational Research

- Investment in molecular landscape collaborations
- Identify new patient populations, responsive subsets and resistance mechanisms
- Input into combination strategies

Early Global Expansion

- Accelerate enrollment in geographies with high unmet need
- Formation of Kinnjiu in Greater China

Kinnate Pipeline

Multiple programs advancing towards clinical stage with single agent and combination opportunities

Target, Program	Indications	Discovery	Lead Optimization	IND-Enabling	Phase 1	Phase 2/3	Next Anticipated Milestones
KIN-2787 RAF-Driven and Dependent (KN-8701)	BRAF Class II & III Driven Advanced Adult Solid Tumors (NSCLC, Melanoma etc.) & $NRAS^{Mut}$ Melanoma	**Monotherapy**					**Initial Clinical Data in Q3 2022**
	Advanced $NRAS^{Mut}$ Melanoma	**Combination with Binimetinib**					**Initiate Combination Portion of Trial in H1 2022**
KIN-3248 FGFR2/3 Driven	Naïve and pre-treated FGFR 2 / 3 Driven Advanced Adult Solid Tumors (UC, ICC etc.)						**Initiate Phase 1 in H1 2022**
CDK12 KIN004	Adult Solid Tumors (ex. Ovarian / Breast)						

Multiple undisclosed targets in Research Stage
Goal of 1 IND a Year

Note: Greater China rights exclusively licensed to Kinnjiu, of which Kinnate is the majority shareholder

Team Comprised of Leaders in the Field of Precision Oncology

Leadership	Board of Directors	Scientific Advisory Board
Nima Farzan CEO & Board Member *PaxVax, Novartis*	**Dean Mitchell (Chairman)** Independent	**Keith Flaherty, MD** MGH, Harvard Medical School
Richard Williams, MBBS, PhD CMO *Amgen, GRAIL, WuXi NextCODE*	**Jim Tananbaum, MD** Foresite	**Ryan Corcoran, MD, PhD** MGH, Harvard Medical School
Neha Krishnamohan CFO *Goldman Sachs*	**Michael Rome, PhD** Foresite	**Luis Diaz, MD** MSKCC
Mark Meltz COO & GC *Audentes, PaxVax, Novartis*	**Carl Gordon, PhD** OrbiMed	**Andy Lowy, MD** UCSD Moores Cancer Center
Rob Kania, PhD SVP, Drug Discovery *Pfizer*	**Laurie Smaldone Alsup, MD** Independent	**Ezra Cohen, MD** UCSD Moores Cancer Center
Ken Kobayashi, MD SVP, Clinical Development *Pfizer, J&J, FDA*	**Keith Flaherty, MD** Independent	**John Iafrate, MD** MGH, Harvard Medical School
	Melissa Epperly Independent	**Eric Murphy, PhD** Scientific Advisor, Co-Founder, Kinnate
	Helen Sabzevari, PhD Independent	

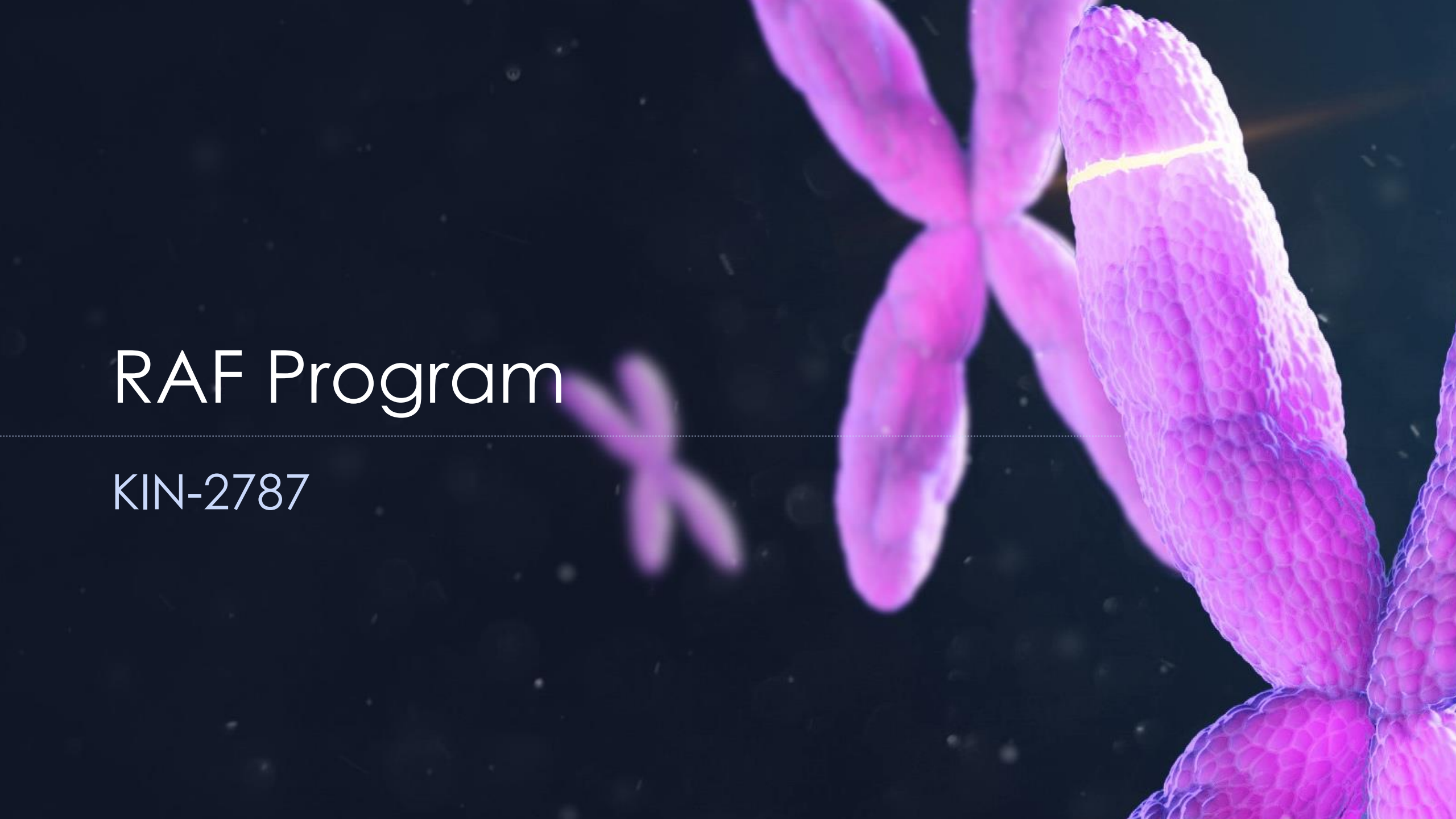
Kinnate Expansion into Greater China

Joint Venture Kinnjiu Established with Experienced China Investor OrbiMed Asia Partners

- $35M Series A Financing for Kinnjiu based in Shanghai
- Investor OrbiMed Asia Partners brings tremendous expertise and connections in China to Kinnjiu
  - OrbiMed Private Investments and Foresite Capital also participated in round
- Kinnate is the majority shareholder of Kinnjiu
  - Kinnjiu 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)
  - Kinnjiu 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 Kinnjiu
  - 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

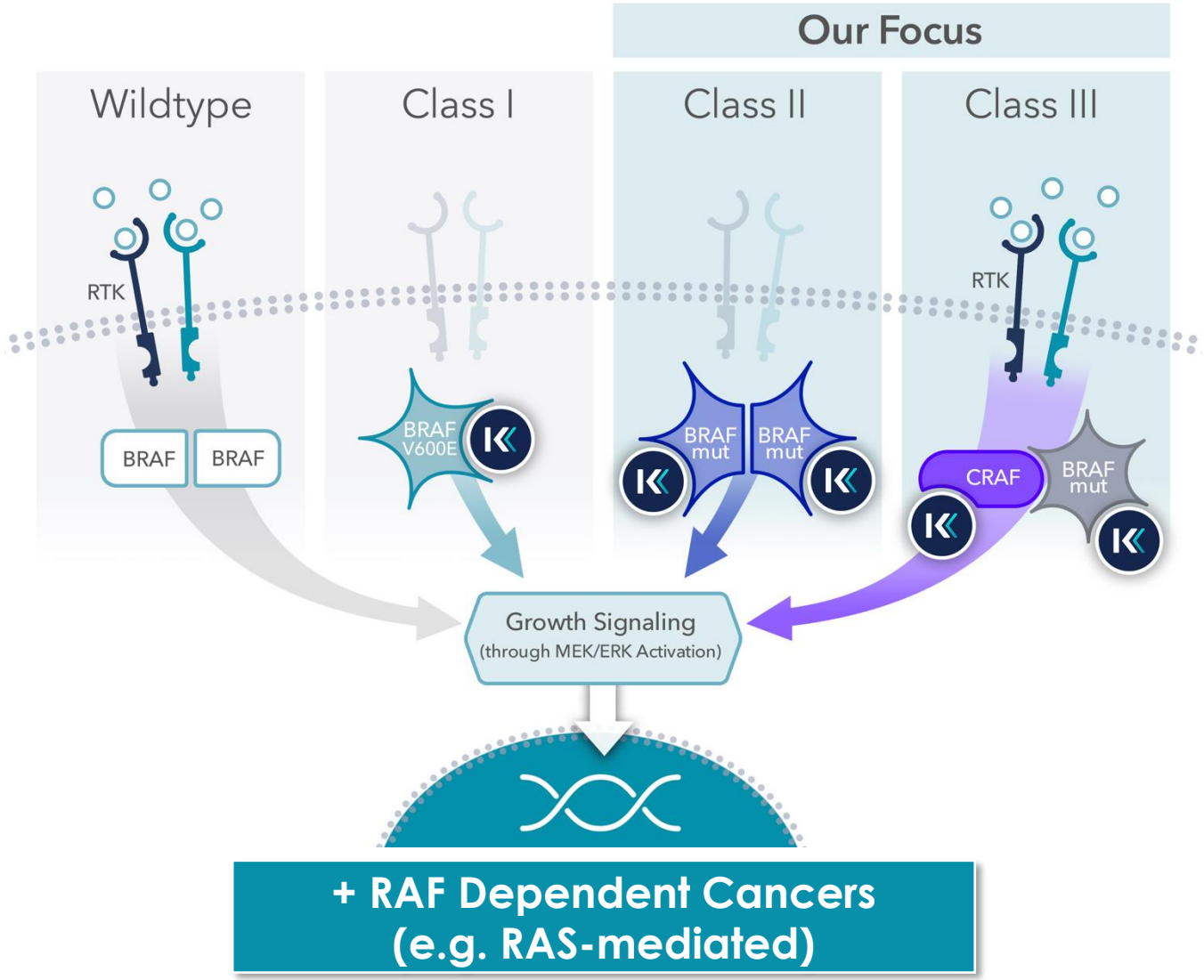
KIN-2787



The RAF Opportunity

Targeting RAF Mutant-Driven and Dependent Cancers Without Approved Precision Therapies

- Approved Class I BRAF inhibitors include Vemurafenib, Dabrafenib, Encorafenib
- The Class II and Class III BRAF alterations 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

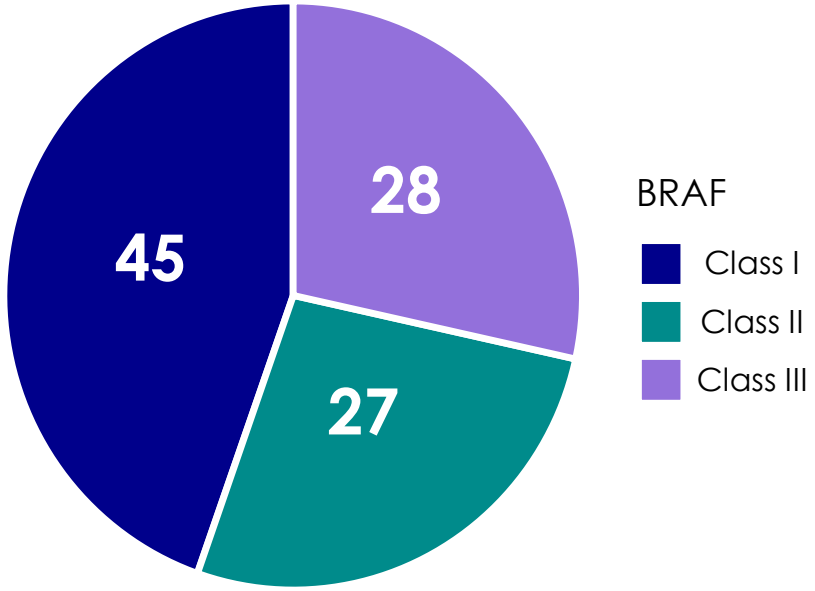


Pan-Cancer Prevalence of Patients Bearing BRAF Alterations

Majority of oncogenic BRAF alterations (~55%) are Class II or III Without Any Approved Drugs

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

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

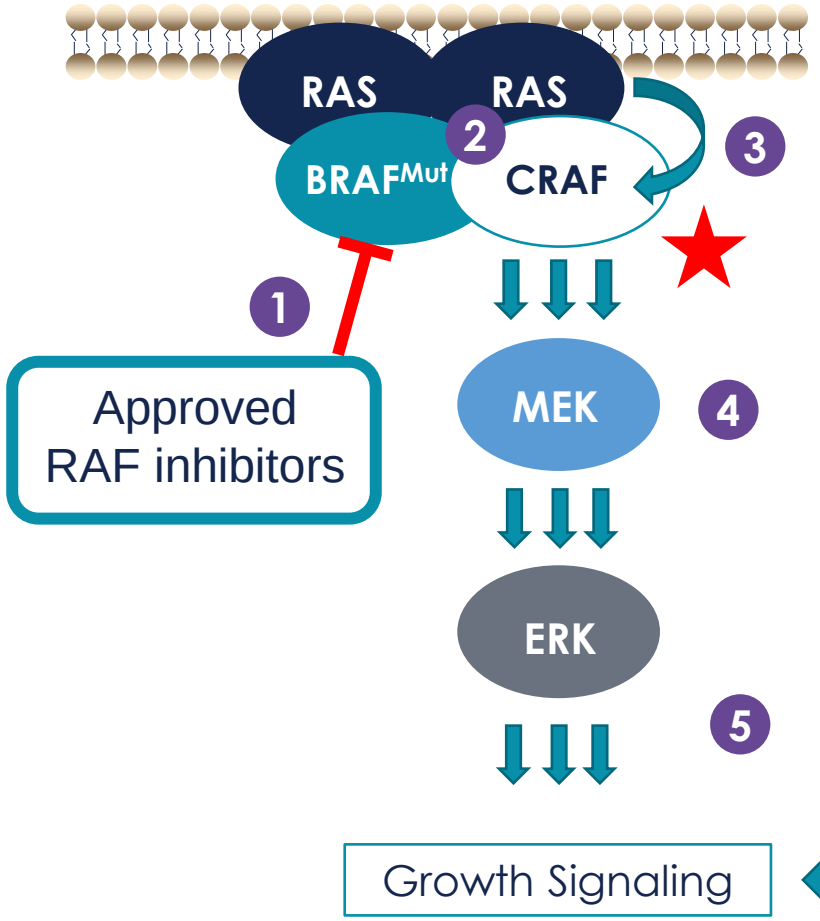


% of Oncogenic RAF Alterations

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

Inhibition of Both RAF Kinases in Dimer is Required for Class II & Class III

Otherwise Transactivation drives MAPK signaling and Pathway Activation



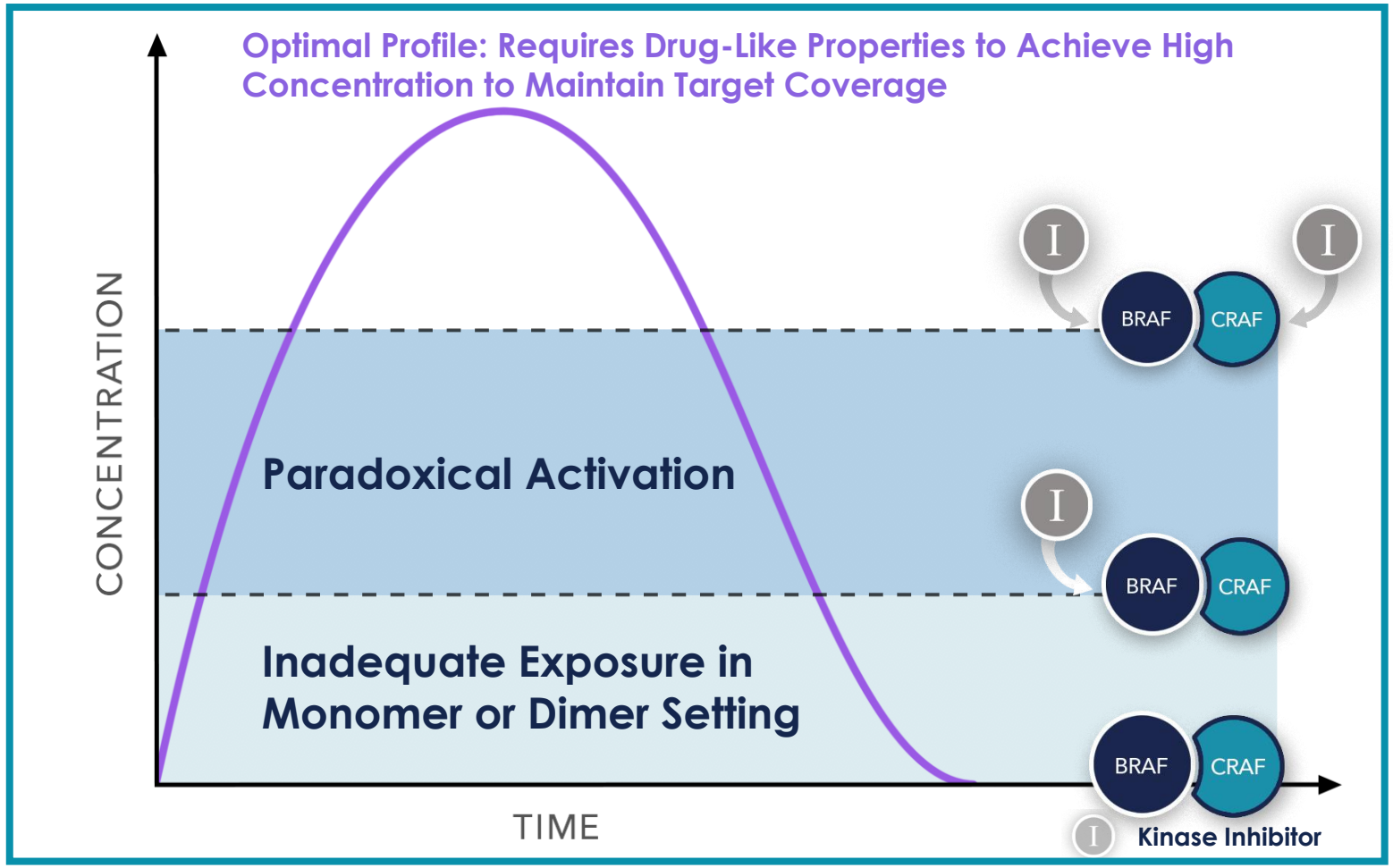
1. BRAF Inhibitor binds to BRAF Target
2. BRAF + CRAF heterodimerize, recruited to RAS
3. Transactivation of CRAF via RAS binding
4. MEK and ERK activation
5. Elevated growth promoting signaling

★
- 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 & Equipotent Inhibition of Both RAF Kinases in Dimer is Required to Avoid Paradoxical Activation



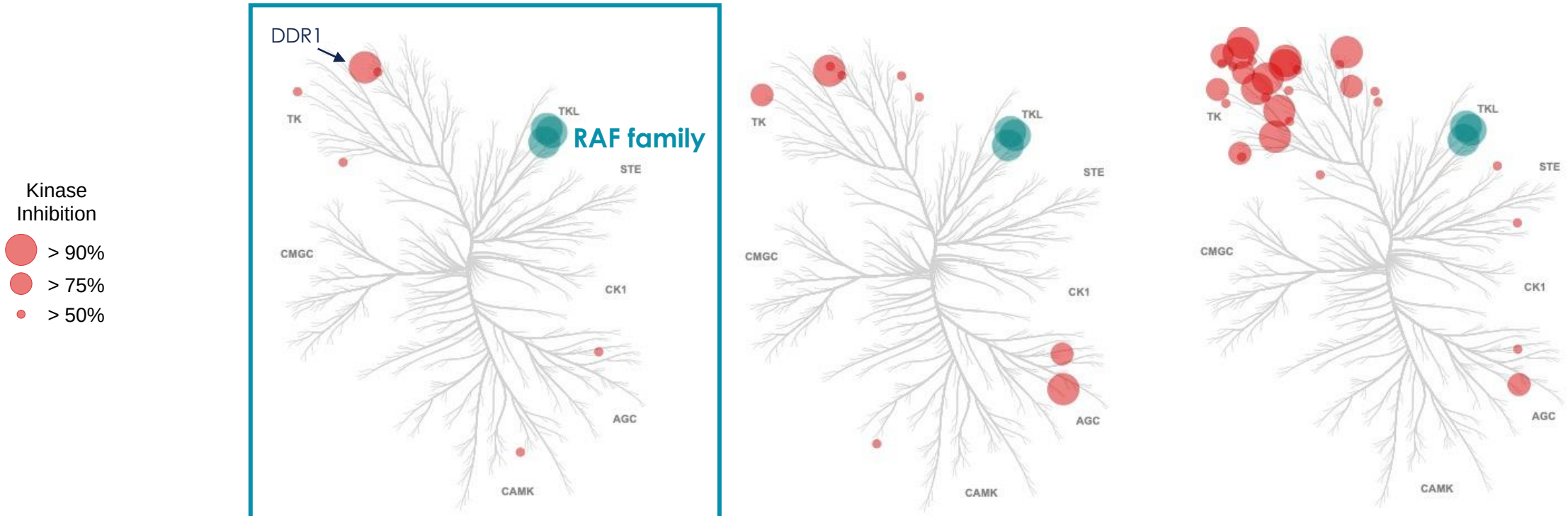
- Paradoxical activation occurs when the non-inhibited RAF molecule in the RAF dimer is activated
- This can occur when the RAF molecule is in a homodimer (BRAF-BRAF) or a heterodimer conformation (BRAF-CRAF), depicted in the figure on the left
- Can occur:
  - As drug concentrations approach & dip below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

Optimal Product Profile to Target Class II & Class III BRAF Driven Cancers

- Selective drug with activity limited off target activity
- Broad alteration coverage
  - Pan-RAF approach with the ability to cover heterogenous nature of Class II & Class III BRAF alterations
- Equipotent inhibition across both RAF kinases in the Dimer
  - Narrows the concentration levels at which paradoxical activation occurs
- Superior pharmaceutical properties:
  - Robust PK to stay above the paradoxical activation zone
  - High free fraction to enable more drug available to bind to the target

KIN-2787 is designed to achieve the optimal product profile needed to target Class II & Class III BRAF Driven Cancers

KIN-2787 Offers Differentiated Selectivity versus Other Pan-RAF Approaches



Non-RAF Kinase Inhibition	Kinnate KIN-2787	Novartis LXH-254	Hanmi / Genentech Belvarafenib
# inhibited > 50%	**7**	10	42
# inhibited > 75%	**1**	5	23
# inhibited > 90%	**1**	2	14

- Kinome profiling at 1 µM across > 600 kinases at Reaction Biology (including wild type, atypical, mutant)
- Only wild type kinases pictured in kinome trees

KIN-2787

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	Hanmi / Genentech Belvarafenib	**Kinnate KIN-2787**
Class I	A-375	Melanoma	$BRAF^{V600E}$	7	171	67	**67**
	Colo800	Melanoma	$BRAF^{V600E}$	6	242	108	**112**
Class II	BxPC-3	Pancreatic	$BRAF^{indel(VTAPTP)}$	3	32	42	**51**
	OV-90	Ovarian	$BRAF^{indel(NVTAP)}$	4	24	22	**26**
	NCI-H2405	NSCLC	$BRAF^{indel(LNVTAP)}$	6	5	8	**10**
Class III	WM3629	Melanoma	$BRAF^{D594G}$, $NRAS^{G12D}$	5	6	4	**9**
	CAL-12T	NSCLC	$BRAF^{G466V}$	3	19	41	**18**
Wild Type	NCI-H358	NSCLC	$BRAF^{WT}$, $KRAS^{G12C}$	1	153	303	351
	CHL-1	Melanoma	$BRAF^{WT}$, $NRAS^{WT}$	5	291	443	580
	BJ	Normal fibroblast	Wild type	31	4686	2923	7963

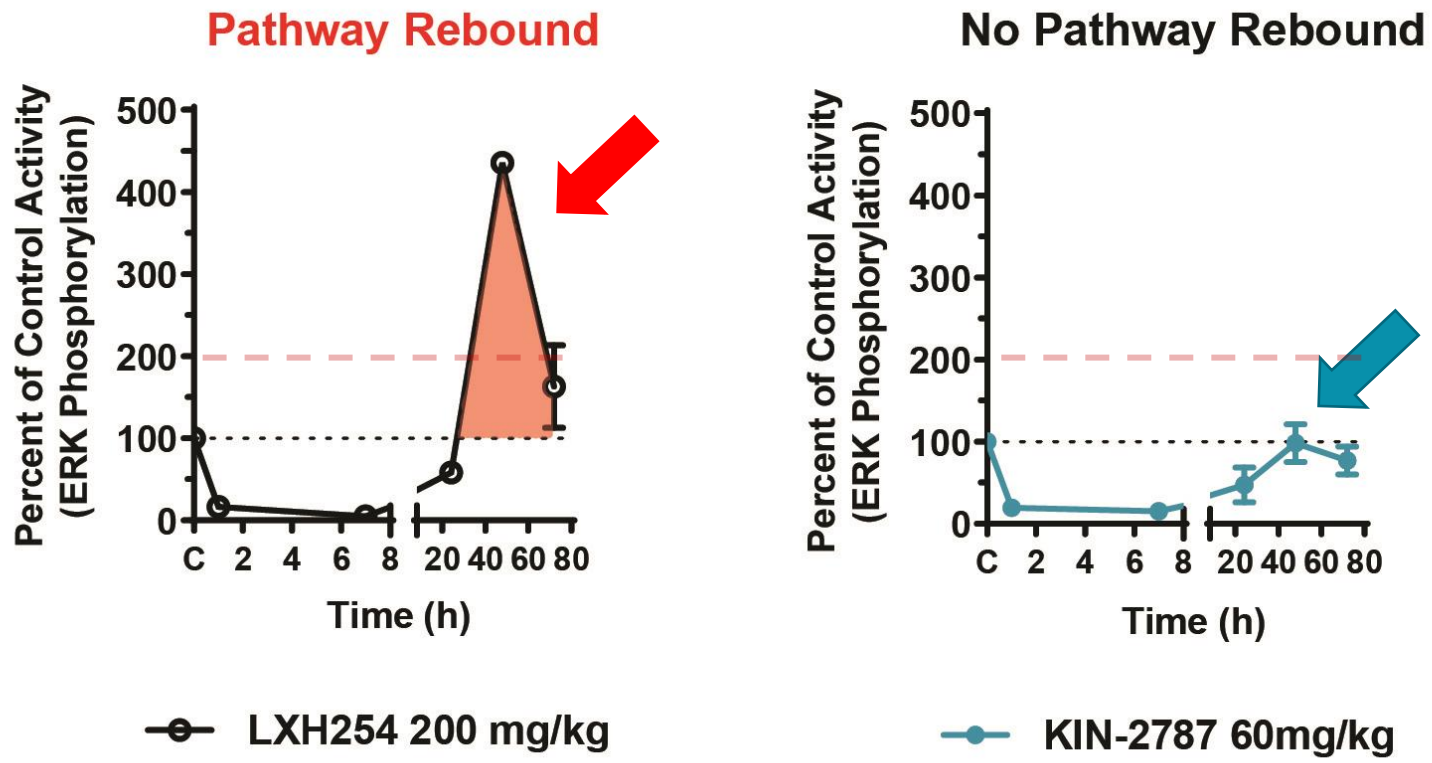
Note: More potent inhibition is reflected by a lower EC_{50} number presented in nM concentration

- Clear differentiation from MEK inhibitors that do not differentiate against WT (wild type)
- LXH-254 & Belvarafenib have 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, BRAFD594G/NRASG12D) xenografts compared to >400% pERK levels observed with LXH254 at 48 hours post-dose

Superior Pharmaceutical Properties for KIN-2787 May Enhance *In Vivo* Target Exposure

Greater than 7-fold Less Total Plasma Concentrations Needed for Similar Target Coverage

	Novartis LXH-254	Hanmi / Genentech Belvarafenib	**Kinnate KIN-2787**
Class II / III Cell Potency (nM)*	**5 to 32 nM**	**4 to 42 nM**	**9 to 51 nM**
Human Plasma Free Fraction (%)	**<1**	**<1**	**7**
Aqueous Solubility (uM):			
pH = 2	**50**	**266**	**312**
pH = 4.5	**7**	**0.4**	**196**
pH = 7.4	**6**	**0.1**	**29**

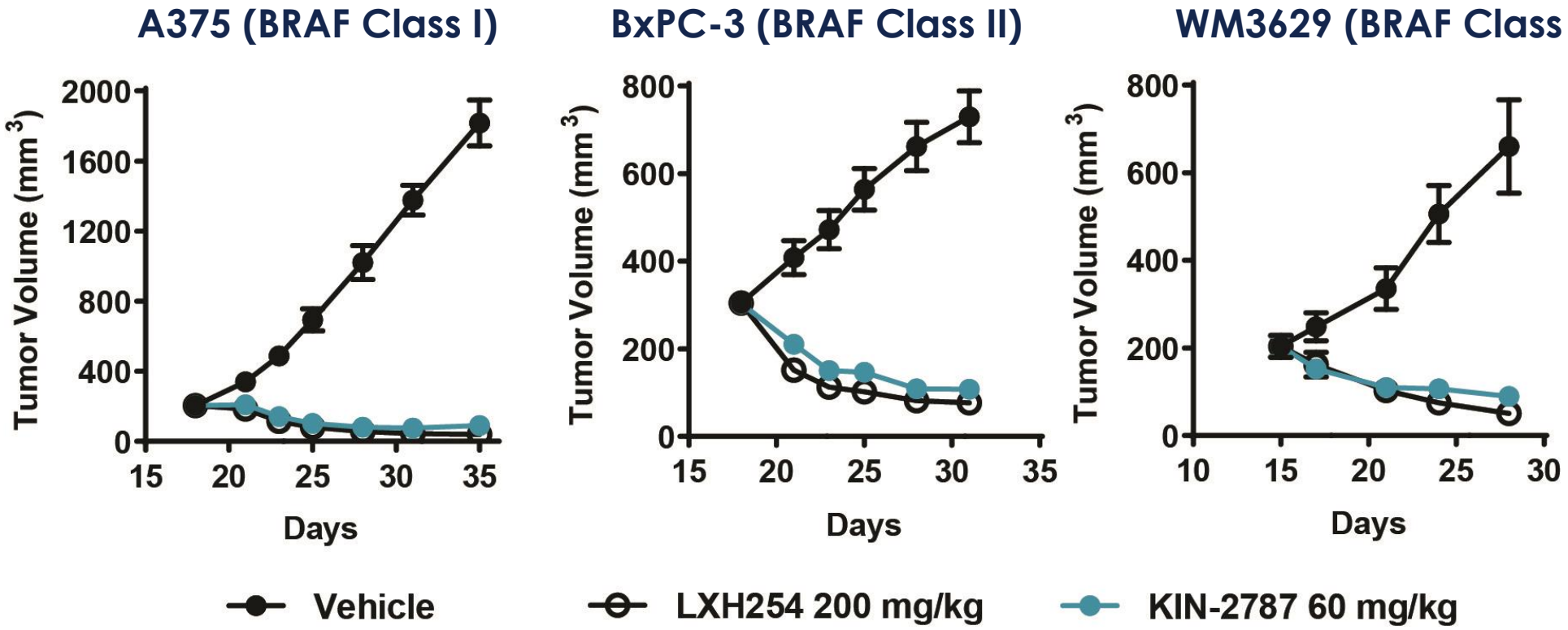
} **Relevant physiological pH**

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

*pERK Inhibition EC50 (nM)

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

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



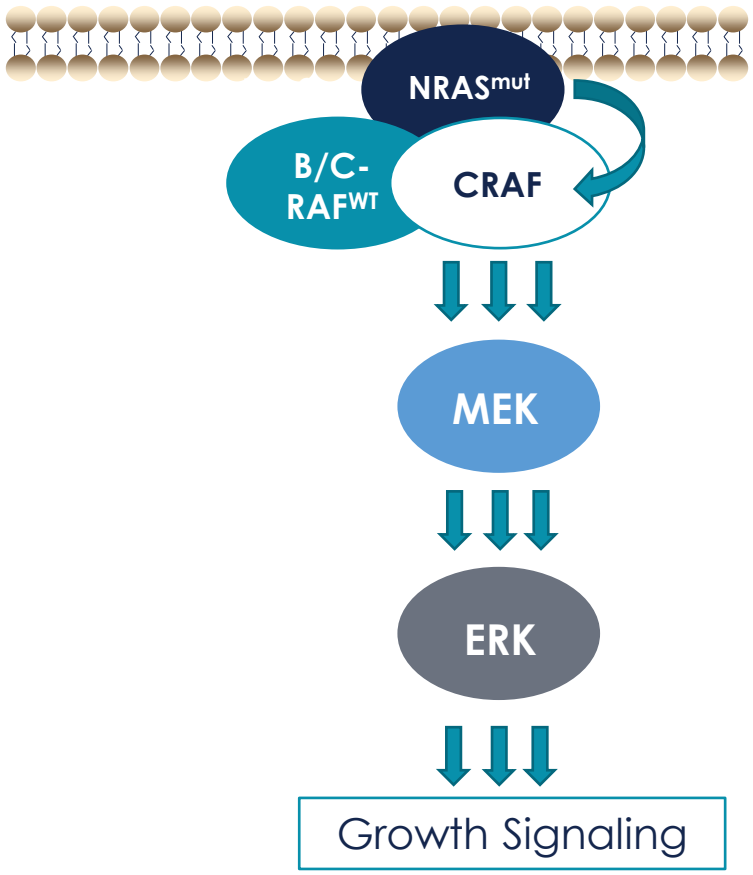
- KIN-2787 (60 mg/kg QD) and LXH254 (200 mg/kg QD) both demonstrated tumor regressions
- 200 mg/kg LXH254 is >4-fold increased free drug exposure relative to the highest clinical dose (600 mg BID)

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

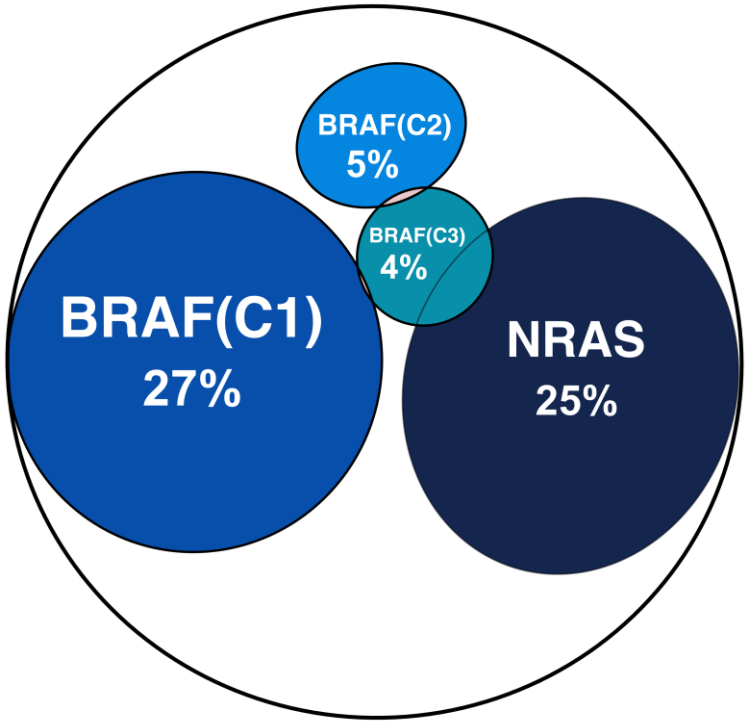
NRAS^{mut} Melanoma Opportunity

RAF-Dependent Signaling

NRAS Signaling Pathway[1]



Prevalence of NRAS^{mut} relative to BRAF Driven Melanoma[2]

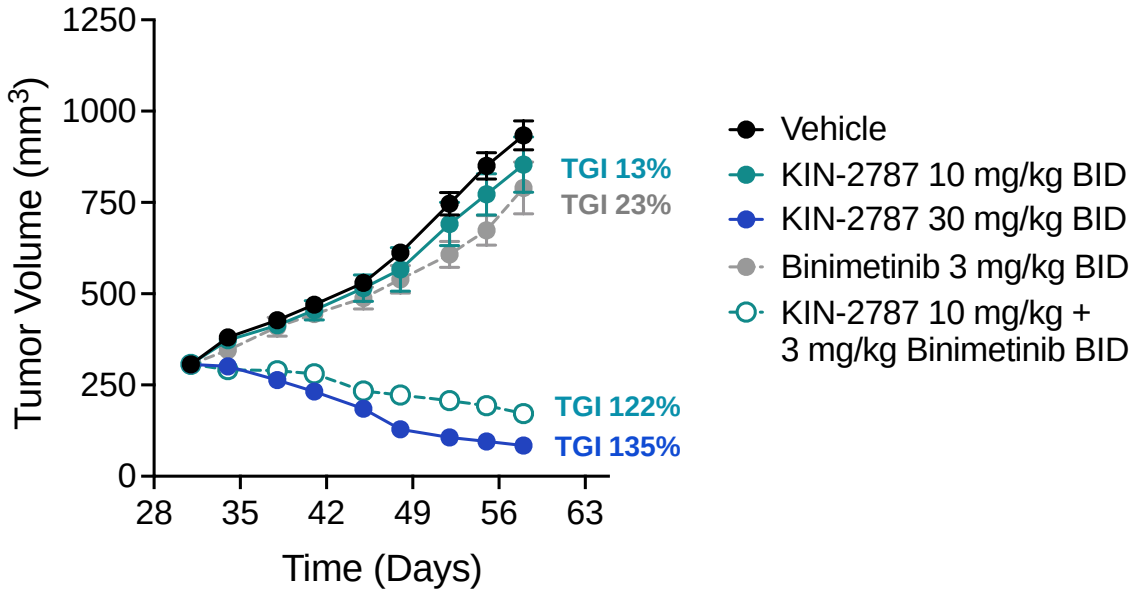


- NRAS^{mut} melanoma signaling has shown to be highly CRAF-dependent
- Approved Class I BRAF Inhibitors have not shown activity in NRAS^{mut} melanoma
- There are currently no targeted therapies approved for this population
- Recent clinical data from Belvarafenib+Cobimetinib provide clinical validation of pan RAFi+MEKi approach
  - PRs observed in 33% of evaluable patients with NRAS^{mut} melanoma[3]
- NRAS^{mut} melanoma represents ~20-25%[2] of melanoma, with limited presence of co-occurring mutations with RAF-driven Class II & III alterations

[1]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
[2]Genomic 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/);
[3]Kim TW et al. ESMO 2021 Congress, 16-21 September 2021, Poster #529P. https://clinicaltrials.gov/ct2/show/NCT03284502

KIN-2787 Preclinical Validation in Combination with Binimetinib in NRAS-Mutant Melanoma

Evidence for Synergy *in vitro* and suggestive *in vivo* in NRAS ^{Q61R}, BRAF WT Melanoma

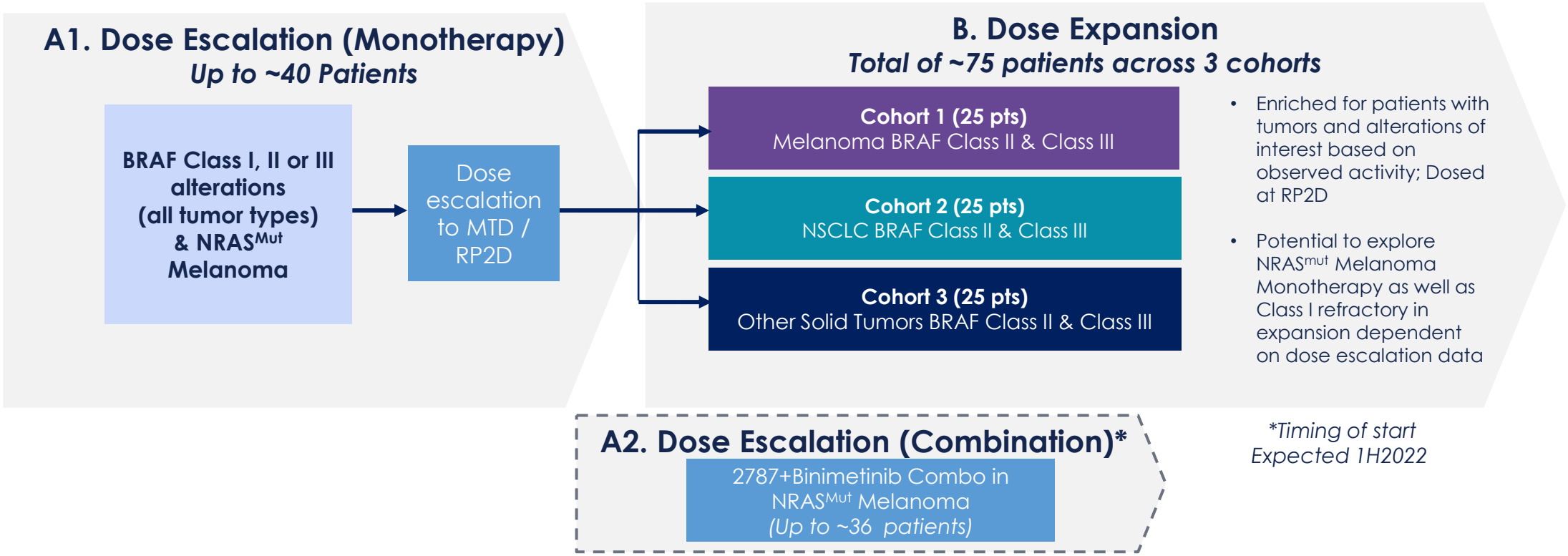


- KIN-2787 10 mg/kg BID dose is in line with clinical dosing strategy
- Binimetinib exposure is consistent with human clinical exposure at approved dose
- Synergy with binimetinib, as indicated by *in vitro* data, potentially enables deeper and more sustained target coverage than monotherapy
- Preclinical data shows that KIN-2787 plus binimetinib combination treatment demonstrated meaningful tumor reductions, is well-tolerated and enhances anti-tumor activity compared to monotherapy

Note: Reflects salt form of KIN-2787

KIN-2787 Development Plan: Ongoing Phase 1 Trial

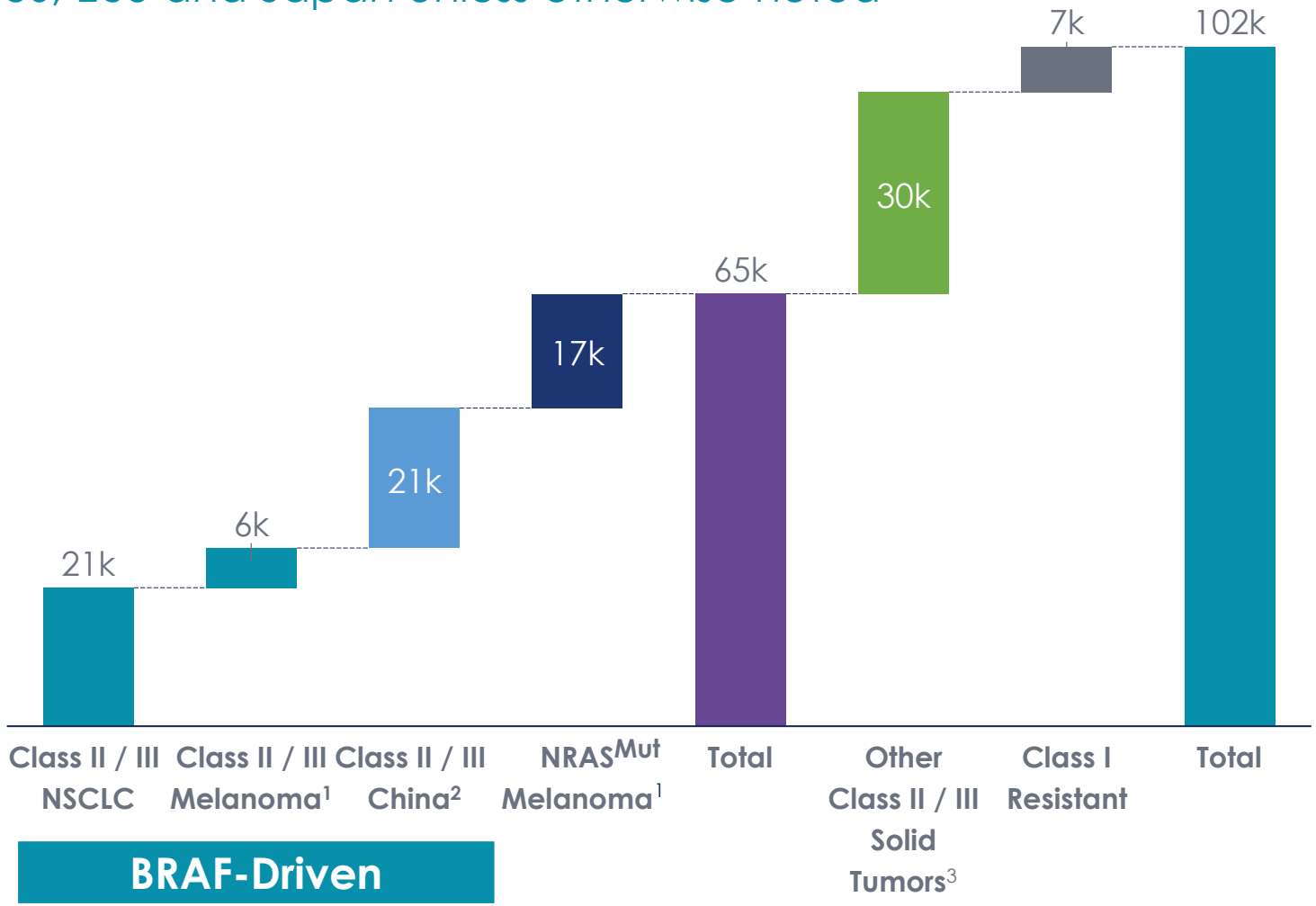
Initial Data Expected Q3 2022; Currently Enrolling Dose Escalation



- Currently enrolling Pt A1. Dose Escalation phase with multiple sites in the US and expansion planned globally
- Addition of NRAS^{Mut} Melanoma for Monotherapy and in Combination with Binimetinib
- **Trial Objectives:** Evaluate Safety. PK & PD, Establish MTD/RP2D, Evaluate preliminary anti-tumor activity
- **Population:** Adult Solid Tumor patients with advanced & unresectable or metastatic disease
- In Part A1: Class I mutation-positive patients would be pre-treated by an approved Class I BRAF inhibitor
- In Part A1: Single patient cohorts for first two Dose Levels; 3+3 design thereafter
- Dose Level 1 for Dose Escalation at 50 mg/day (25 mg BID)

Potential Expansion of Market Opportunity with the Addition of NRAS^{Mut} Melanoma: Over 100k Prevalence

US, EU5 and Japan unless otherwise noted

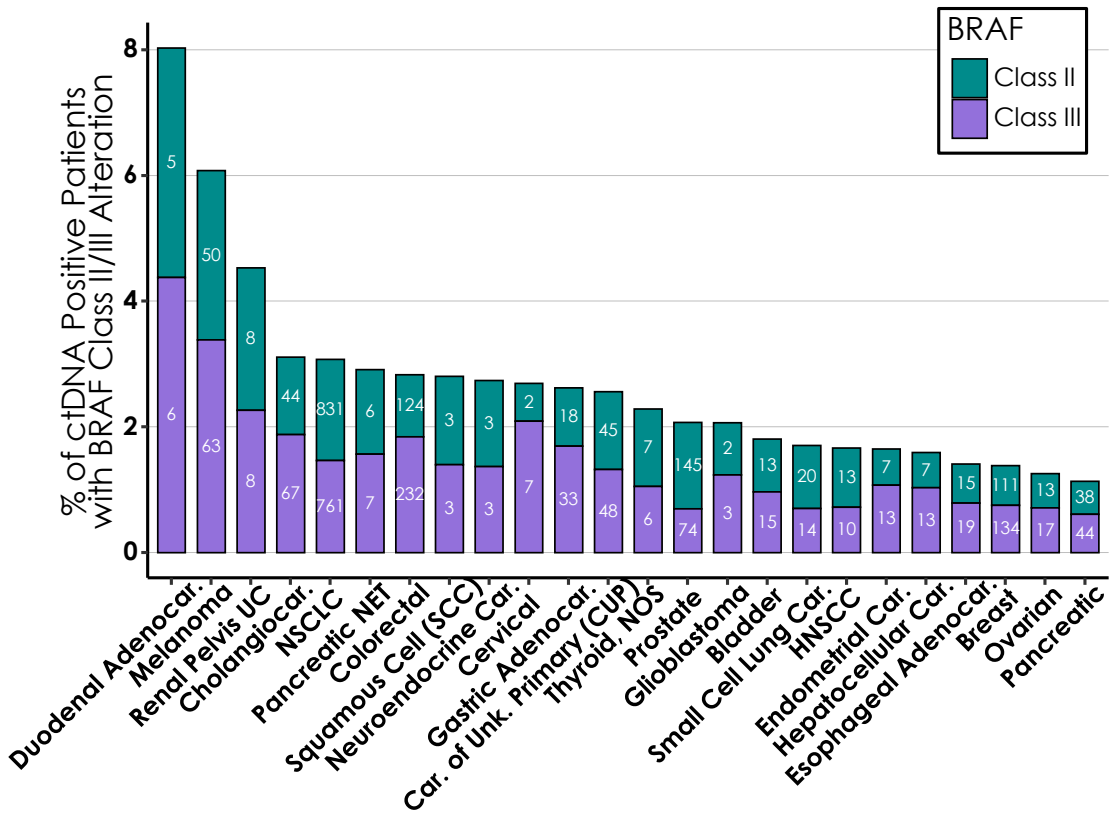


- 2020 sales of the 3 approved products for Class I BRAF alterations were $1.8B
  - 20% growth from 2019 sales
- Substantial opportunities for growth in Class II & Class III alone
- Additional opportunities in various cancer types beyond NSCLC & Melanoma with Class II / Class III alterations (30k+)
- Class I BRAF alterations, including both first line and second line for intrinsic and acquired resistance
  - ~27k – 30k patients have advanced NSCLC and Melanoma with Class I alterations + China
  - ~25% of acquired resistance may be dimer based
- Potential for expansion in earlier lines of treatment (7k for Stage III NSCLC & Melanoma) and other geographies with high disease burden

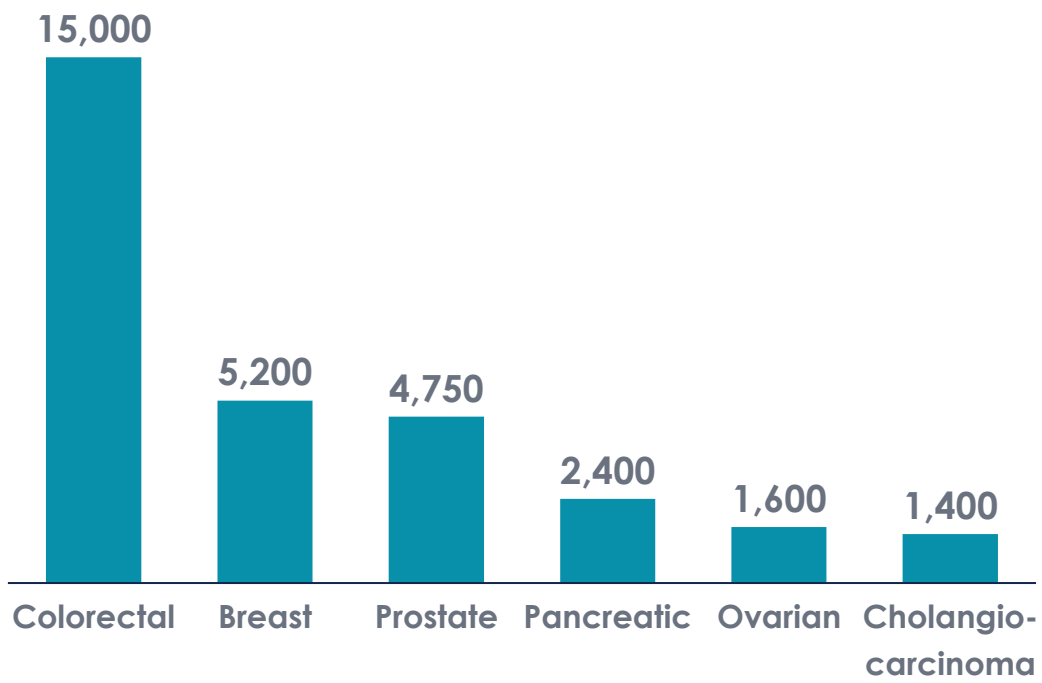
Kinnate prevalence calculations based on Kantar data, 2021 DRG data and data generated from 2021 genomic landscape study with Guardant Health utilizing GuardantINFORM™ unless otherwised noted
Note: Stages IIIb and IV for NSCLC US, EU5 and Japan, Stage IV for Melanoma in US, EU5 and Japan
[1] Prevalence of alteration based on 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/)
[2] Stage IIIb and IV NSCLC in Urban Markets only.
[3] Assumes unresectable or advanced metastatic disease for Colorectal, Breast, Prostate, Pancreatic, Ovarian and Cholangiocarcinoma.

Broad Range of Class II or Class III Driven Solid Tumors Could Expand Market Opportunity Beyond NSCLC and Melanoma

GuardantINFORM Data



Prevalence 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

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™

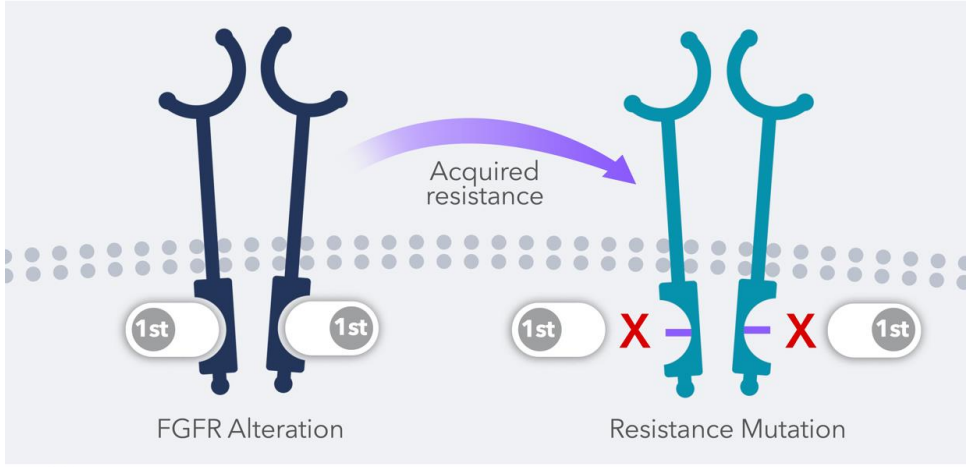
FGFR2/3 Program

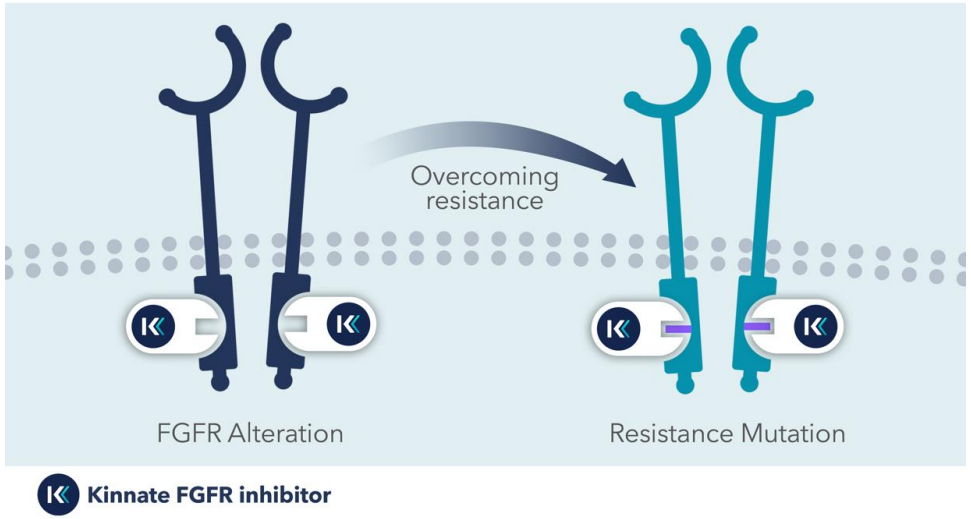
KIN-3248

Kinnate FGFR 2/3 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



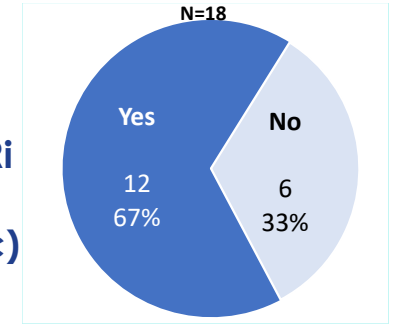


Meaningful On-Target Acquired Resistance to FGFR Inhibitors

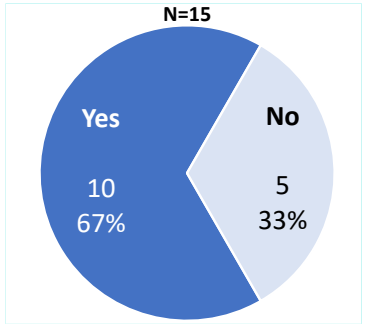
67% of FGFRi-treated ICC Patients Developed FGFR2 Kinase Domain (KD) Mutations at Progression

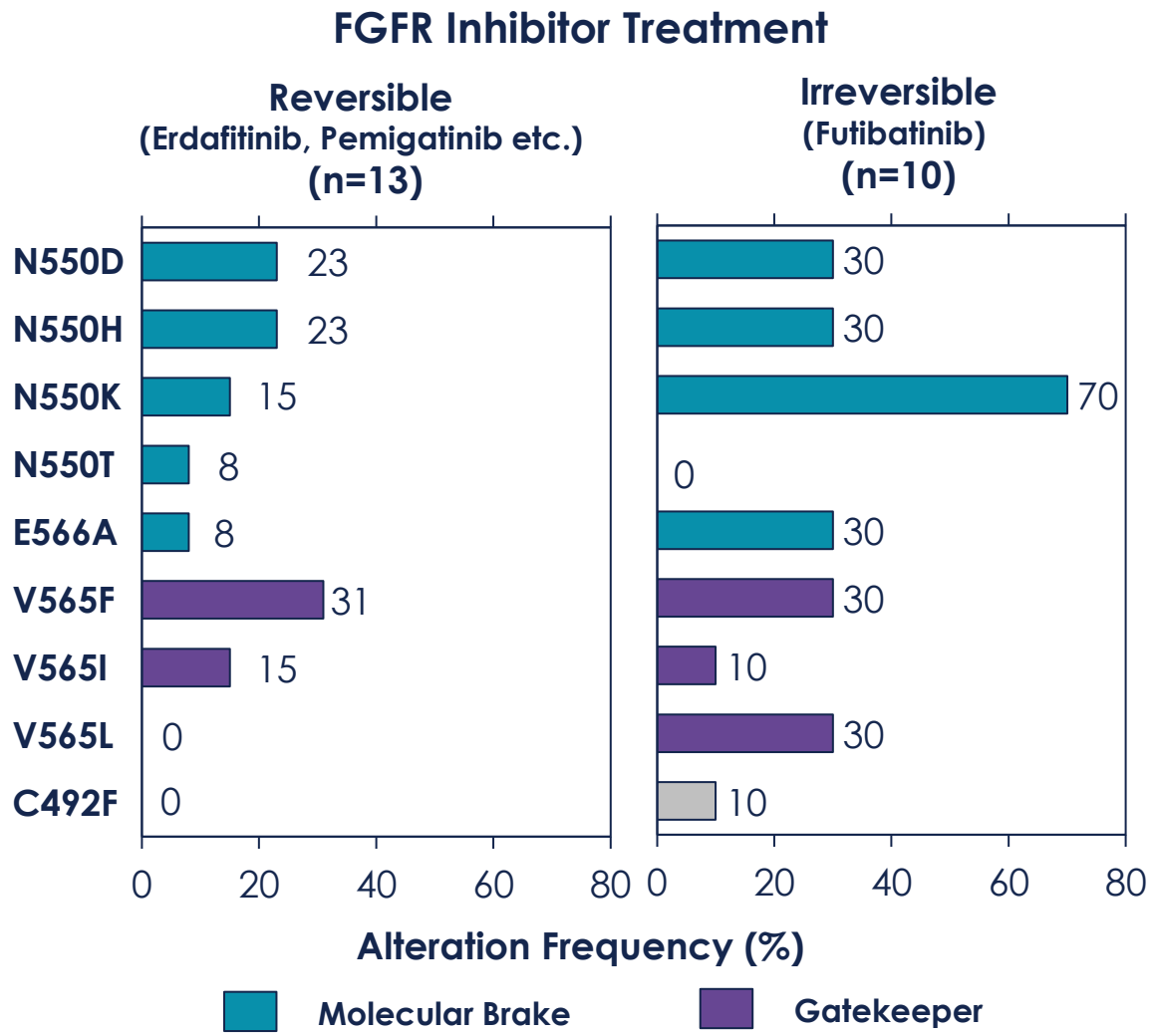
FGFR2 Kinase Domain Mutation

Reversible FGFRi (Erdafitinib, Pemigatinib, etc)



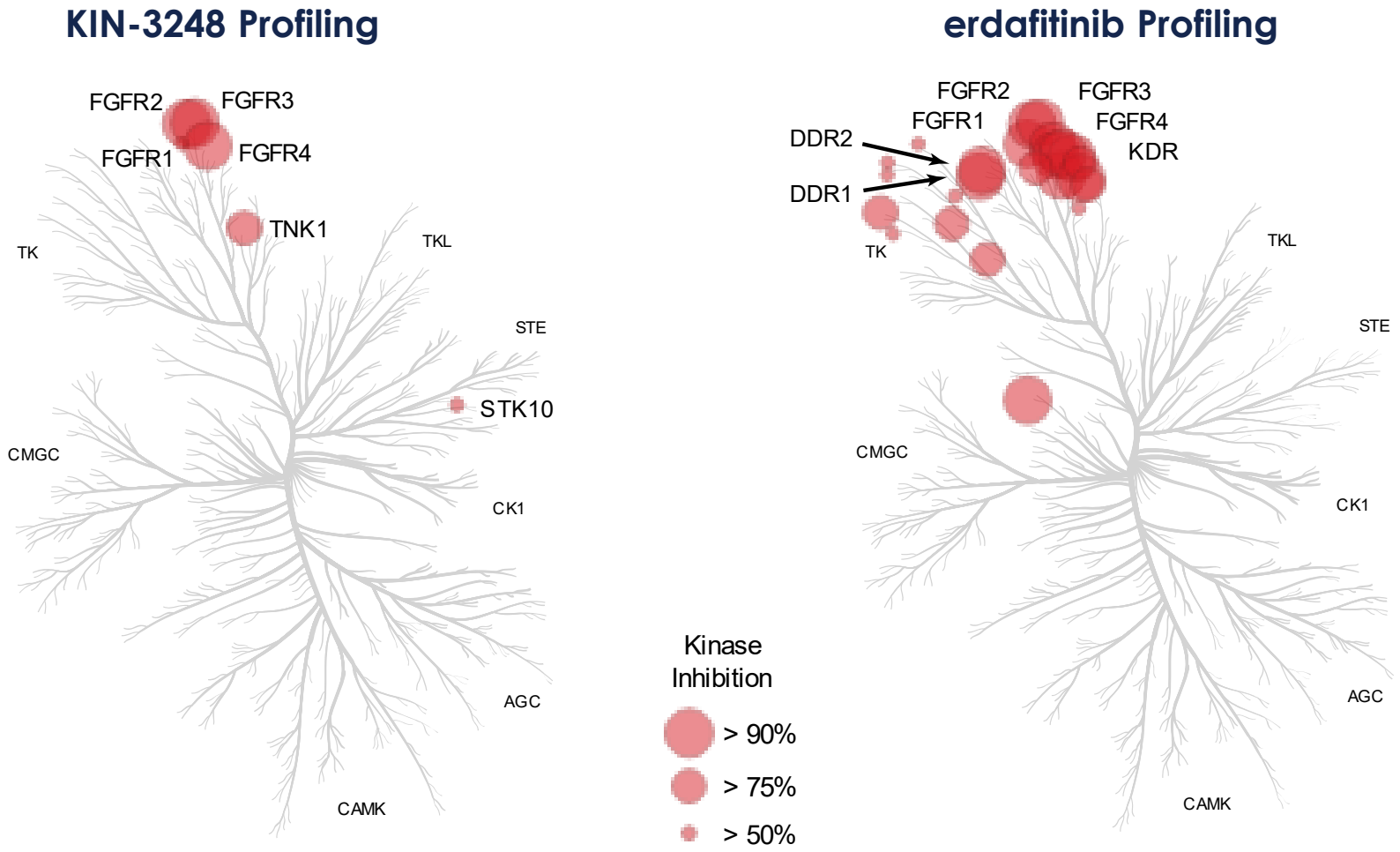
Irreversible FGFRi (Futibatinib)





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)

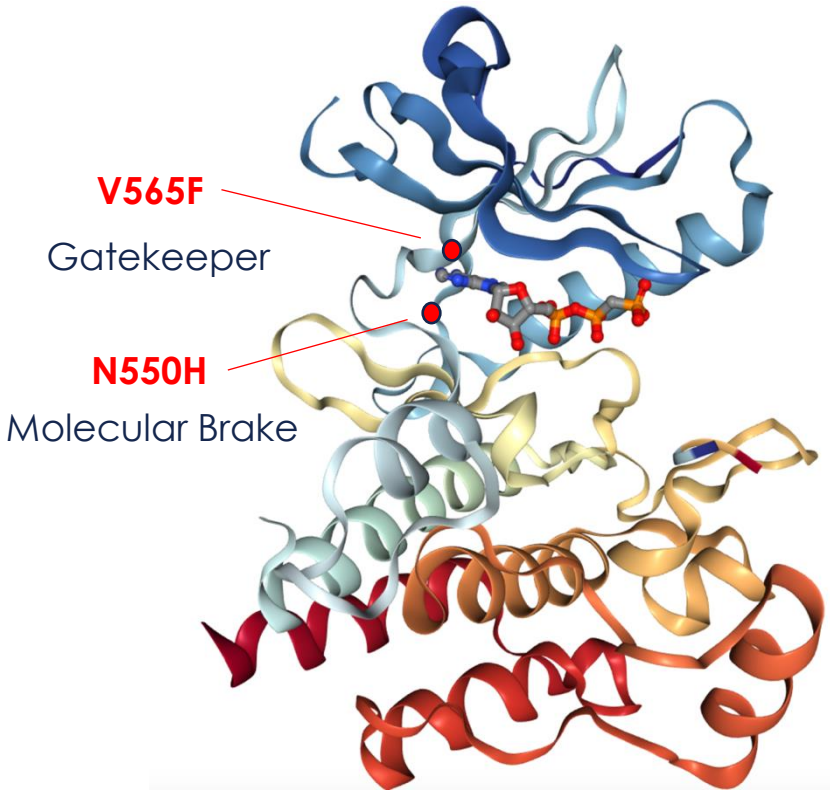
KIN-3248 Displays a Selective & Differentiated Kinase Profile



- 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 and 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	1.7	3.9
FGFR2 WT FGFR2 V565F FGFR2 N550H	- Gatekeeper Mol. Brake	0.15 330 4.1	0.5 492 18.9	2.2 >500 33.4	5.3 20.8 22.8
FGFR3 WT FGFR3 V555M FGFR3 K650M	- Gatekeeper Activ. Mut.	0.7 137 3.5	1.4 494 20	5.6 408 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	984X 38X	227X 15X	**4X** **4X**
R3 V555M / WT R3 K650M / WT	Gatekeeper Activ. Mut.	188X 5X	353X 14X	73X 1.5X	**3X** **0.5X**

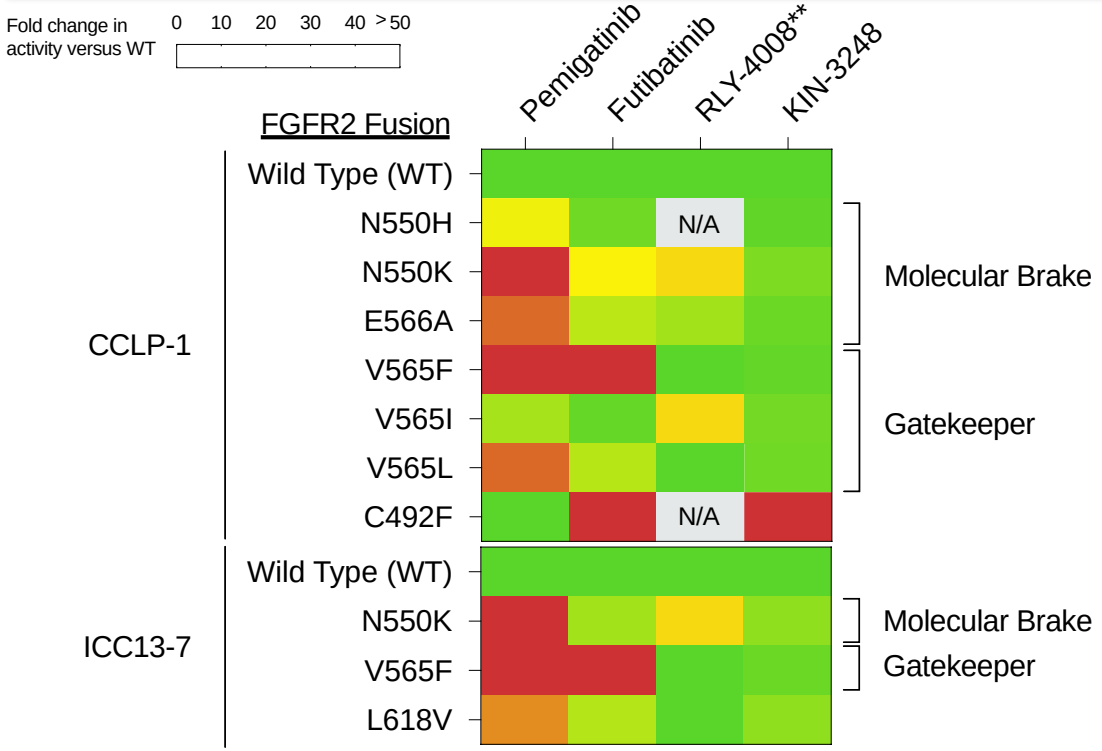
Note: Ratios <10x = equivalent kinase inhibition of either the resistance mutations or corresponding WT kinase. Ratios > 10x = substantial loss of activity against the indicated resistance mutations compared to the corresponding WT kinase

- 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

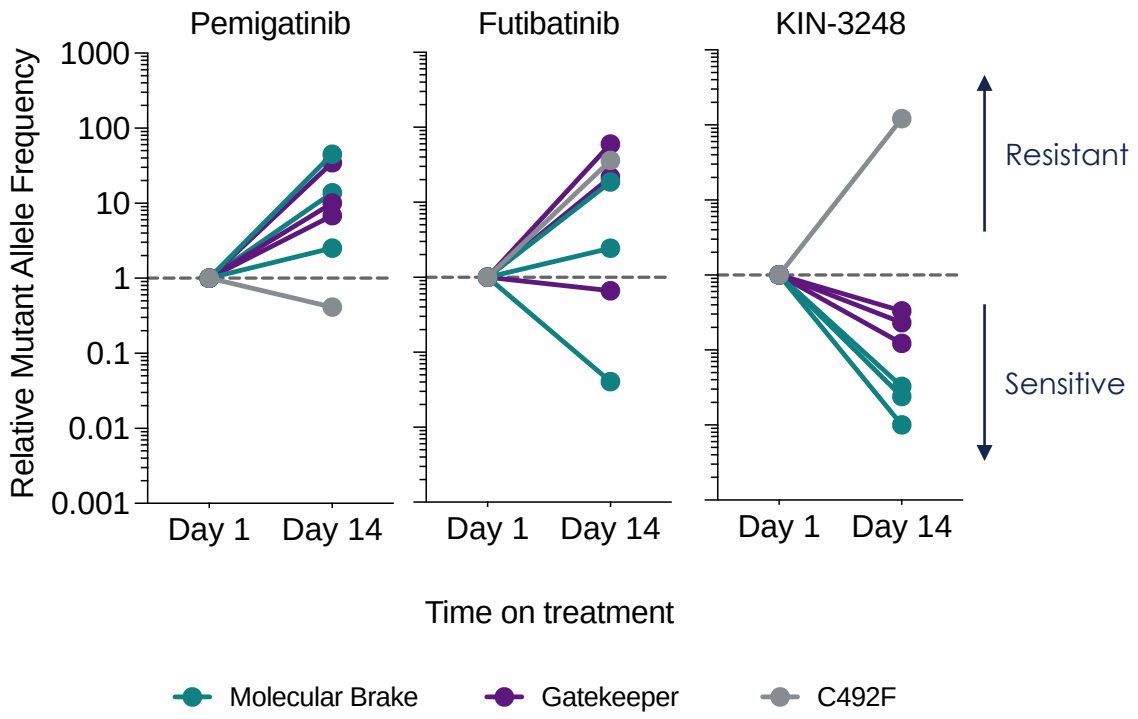
Inhibition of key mutations that drive resistance to first generation FGFR inhibitors

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

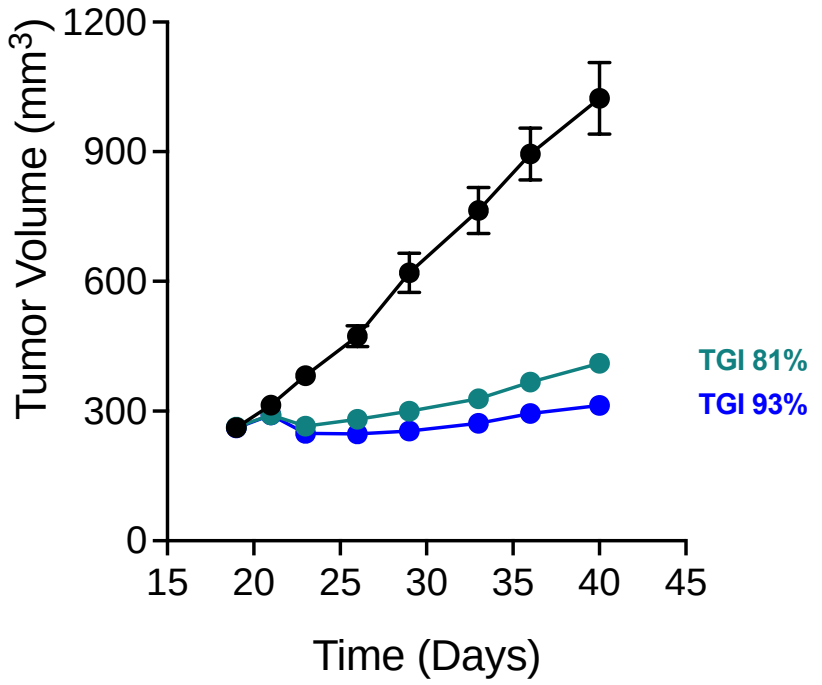
KIN-3248 inhibits the growth of FGFR3 fusion-positive UC cells harboring secondary resistance mutations

FGFR3 Fusion		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					

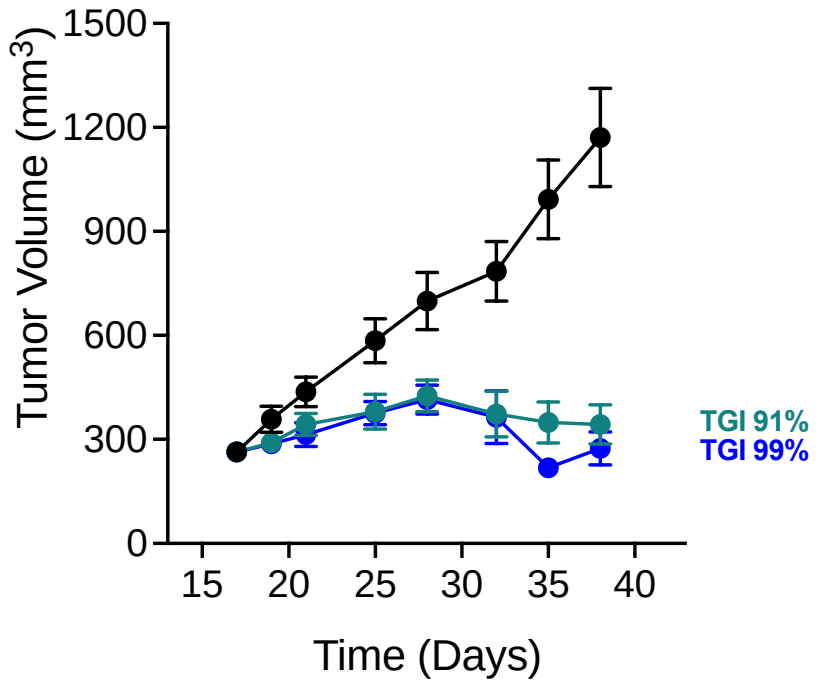
- KIN-3248 showed inhibition of both FGFR3 gatekeeper, molecular brake and activation loop resistant mutations when compared to the FDA approved and clinical candidate FGFR inhibitors

KIN-3248 Demonstrated Tumor Reductions Against Primary FGFR2 & FGFR3 Oncogenic Driver Alterations *In Vivo*

FGFR2 Amplified / Fusion+ Gastric Cancer



FGFR3 Fusion+ Urothelial Cancer

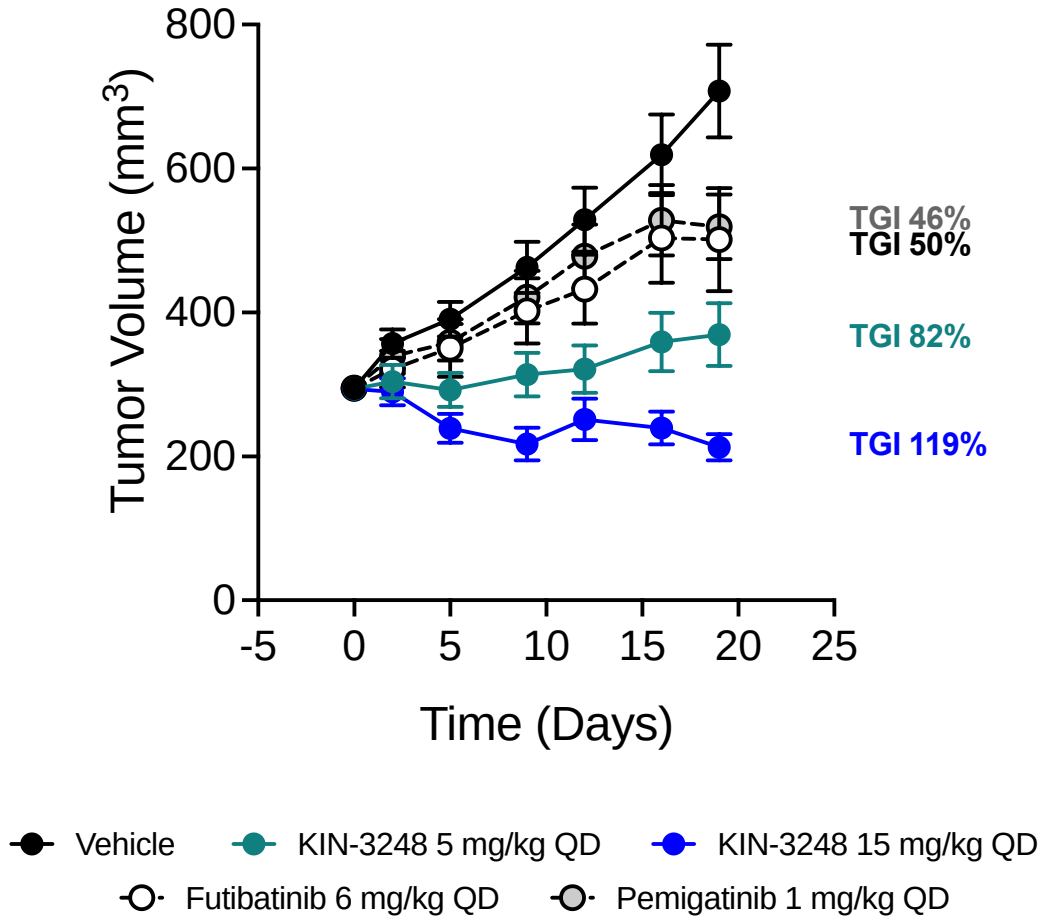


Vehicle — KIN-3248 5 mg/kg QD — KIN-3248 15 mg/kg QD

- Continuous daily dosing of KIN-3248 is **well-tolerated and demonstrated anti-tumor activity** 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 Demonstrated Tumor Reductions Against Secondary, Acquired FGFR2 Gatekeeper Resistance Mutation *In Vivo*

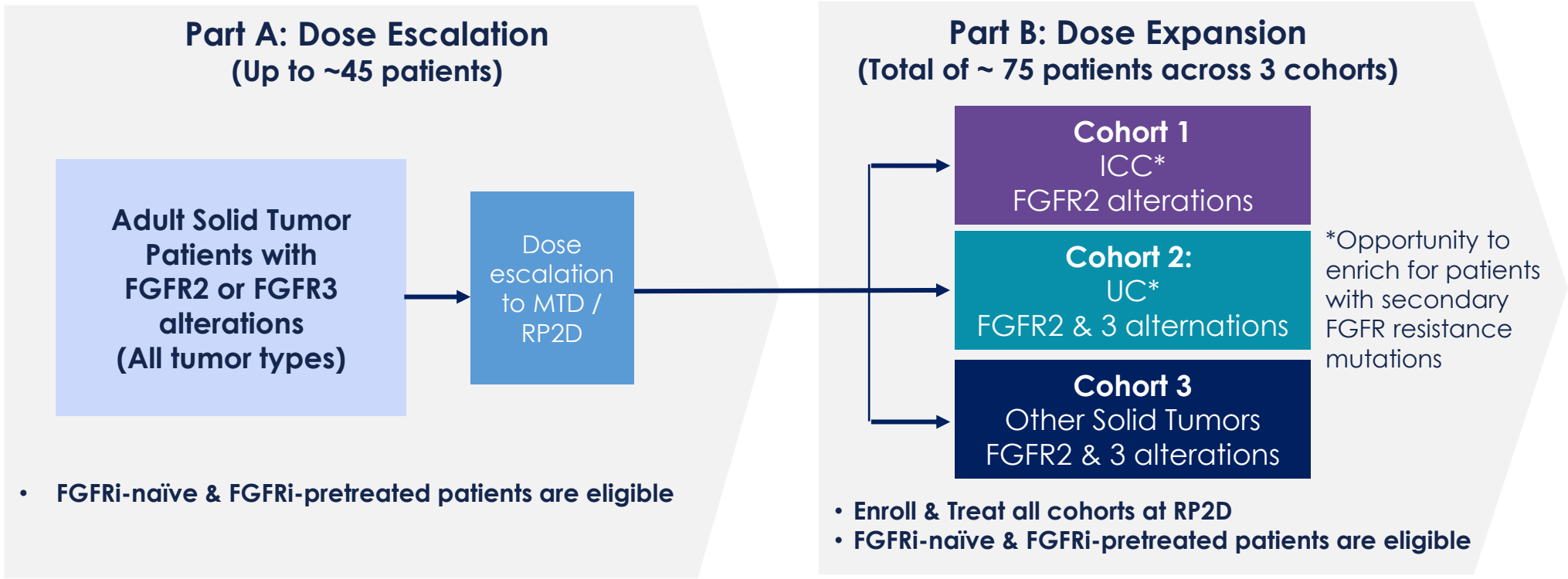


- The **gatekeeper mutation limits efficacy of approved and clinical stage FGFR inhibitors**, pemigatinib and futibatinib, respectively
- Consistent with *in vitro* findings, **KIN-3248 led to tumor growth inhibition and regressions** in a FGFR2-amplified / **V565L gatekeeper mutation-positive** gastric 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

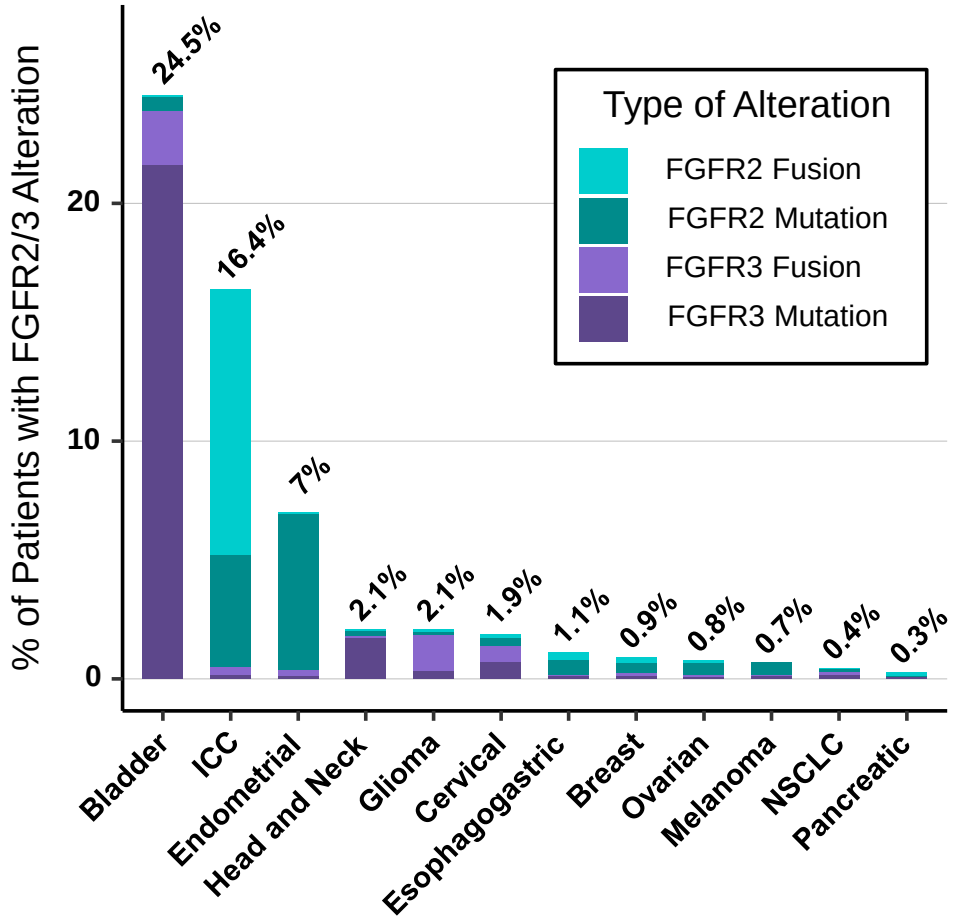
Phase 1 Trial Initiation Expected in 1H 2022



- **Trial Objectives:** Evaluate Safety, PK & PD, Establish MTD/RP2D, Evaluate preliminary anti-tumor activity
- **Population:** Adult Solid Tumor patients with advanced or metastatic disease
- FGFR2 & FGFR3 gene alterations previously detected in tissue-based or blood-based genomic testing

FGFR Inhibitor Market Opportunity

Occurrence Rates of FGFR2 & FGFR3 Alterations by Tumor Types



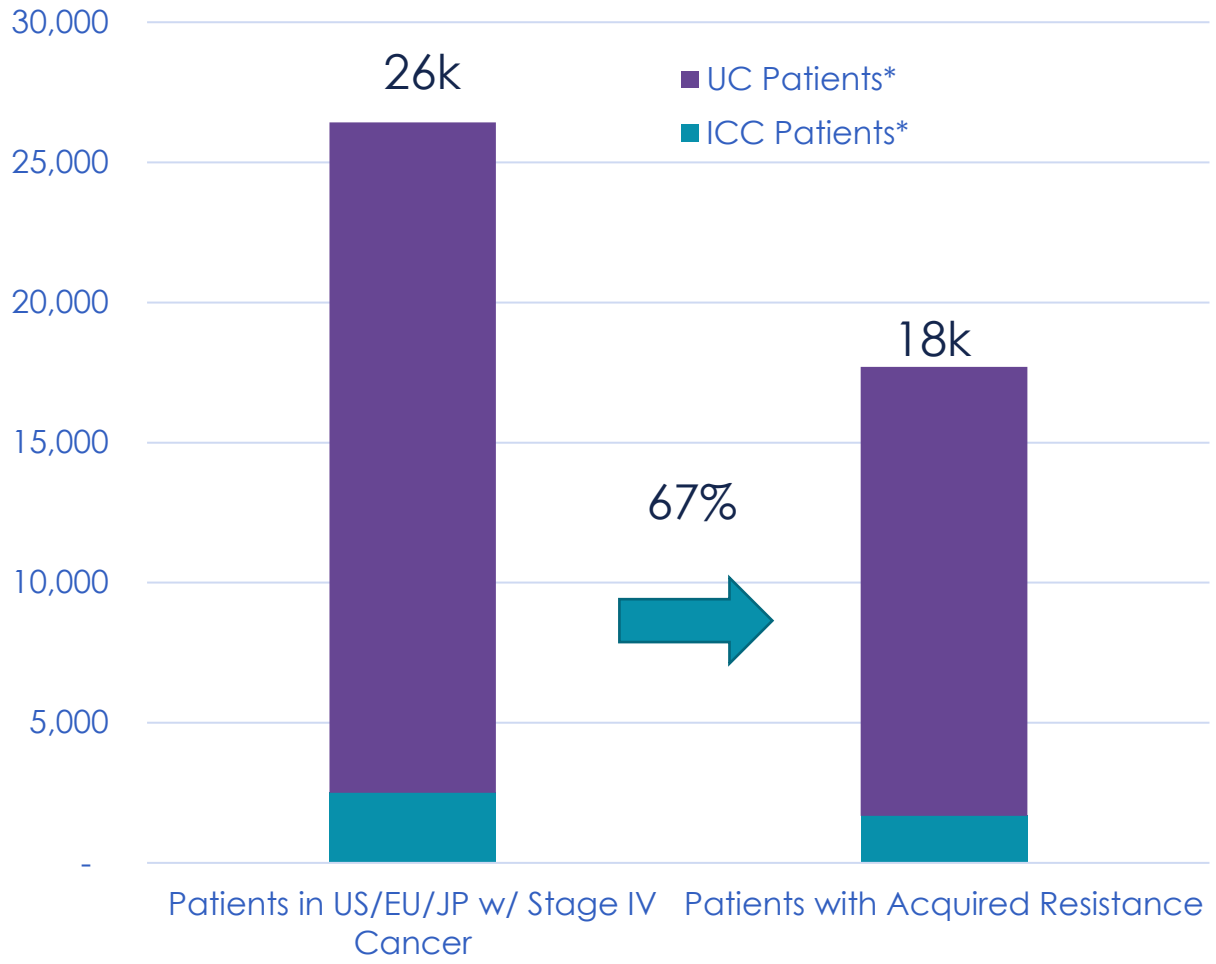
- KIN-3248 has been designed to target both FGFR2 and FGFR3 alterations which includes fusions, mutations (indels and SNVs) and other rearrangements which are likely oncogenic drivers of tumors
- While patients with solid tumors do have FGFR2/3 amplifications, they are often not the primary drivers of 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 Inhibitor Market Opportunity – UC & ICC Patients

US, EU and Japan Patients with Active Disease



*Reflects FGFR2 or FGFR3 Alterations

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).**

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)

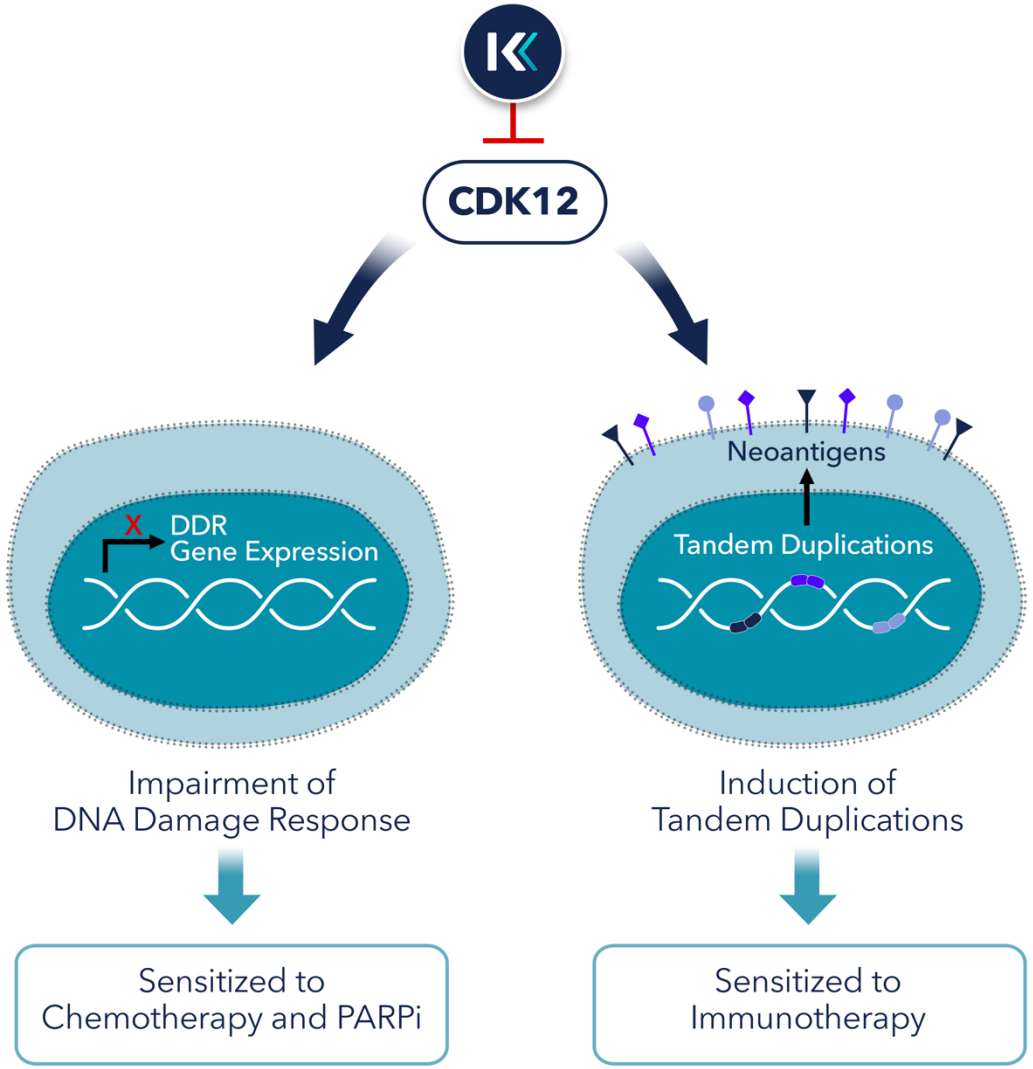
Kinnate Discovery Engine

Research Capabilities

KIN004 – CDK12 Program

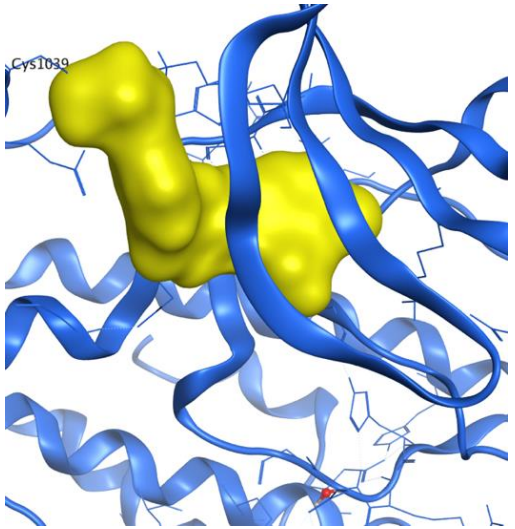
CDK12 Inactivation Impairs DNA Damage Response and Induces Tandem Duplications

- 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

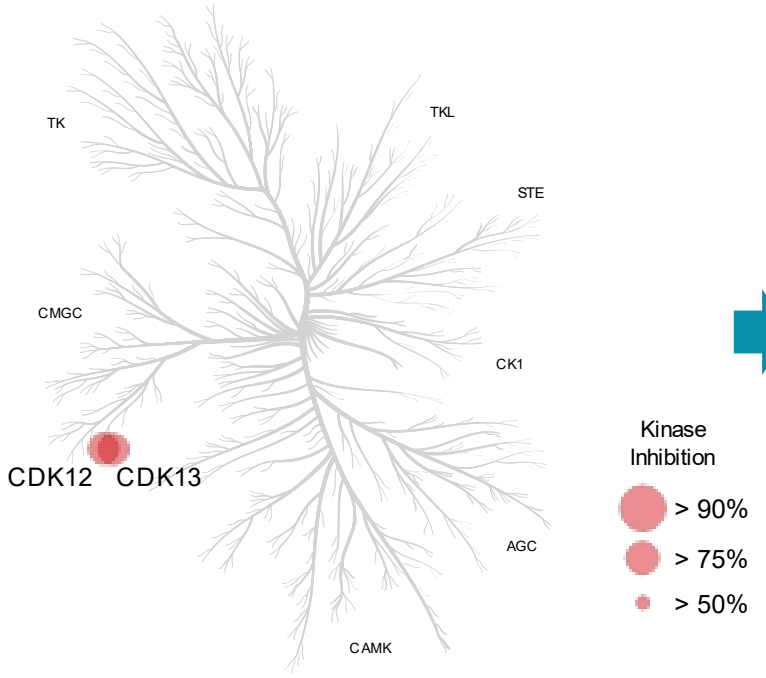


Program Has Demonstrated Selective CDK12 Inhibition

KIN004
Co-Crystal Structure



Kinome Profile – KIN004



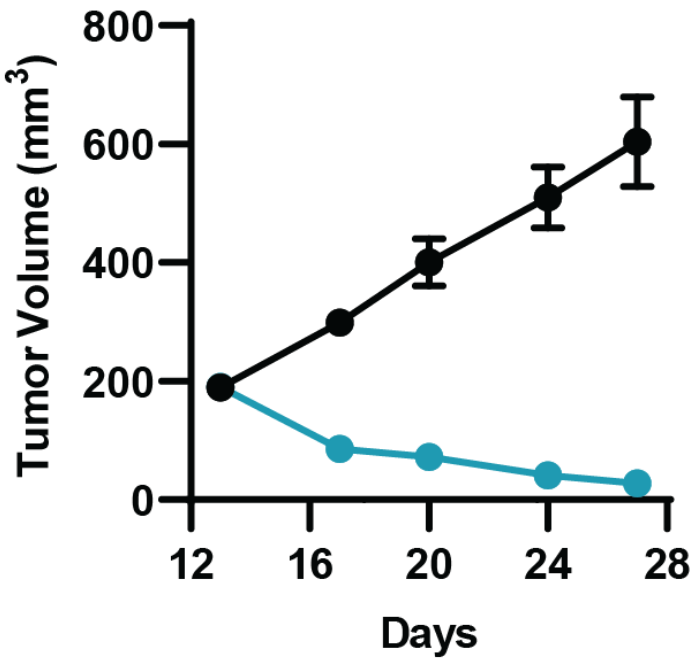
CDK Family Selectivity – KIN004

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

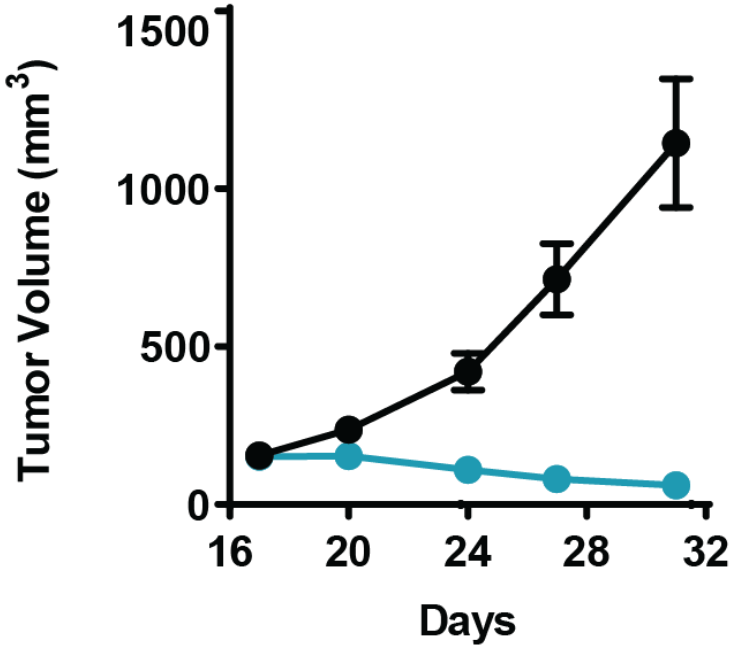
- KIN004 demonstrated selective CDK12 inhibition compared to highly homologous CDK2, CDK7 and CDK9 family members
- Structure-based design enabled by Kinnate proprietary co-crystal structure

Tumor Regressions Demonstrated with Selective Inhibition of CDK12

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



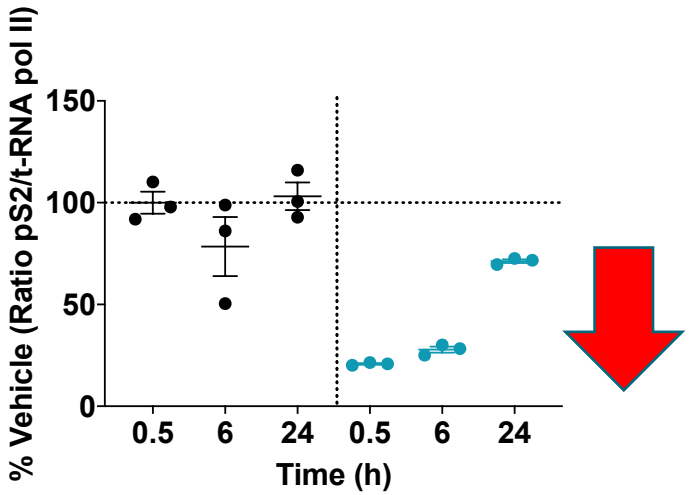
***In Vivo* Efficacy – OVCAR3 (BRCA^{WT})**



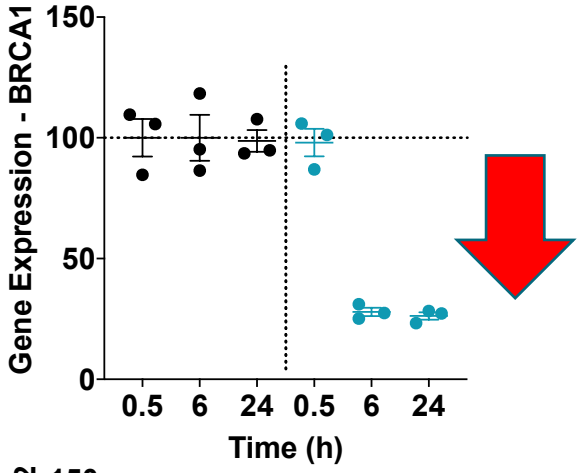
Note: HCC70 breast tumors (left) and OVCAR-3 ovarian tumors (right) represent BRCA 1/2 WT cancers that were DDR-proficient and were not sensitized to PARP inhibitor treatment

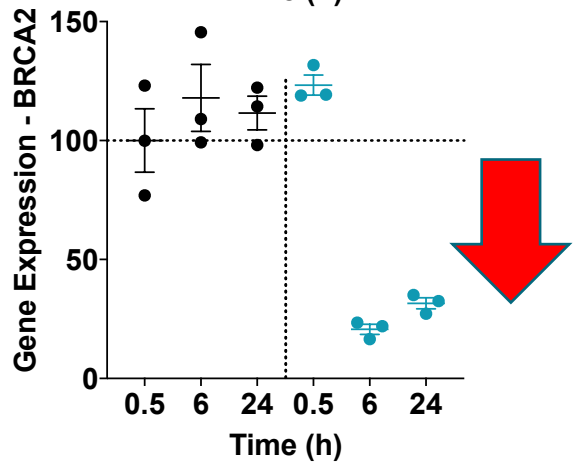
Selective Inhibition of CDK12 *In Vivo* Produces DNA Damage Response Gene Downregulation in HCC70 Xenografts

Biomarker of CDK12 Activity

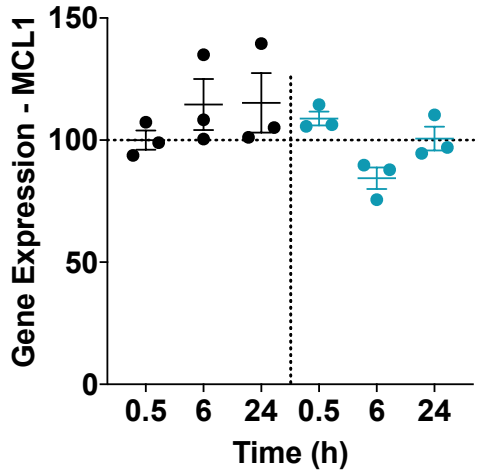


DDR Gene Expression





Control Gene Expression



Vehicle KIN-CDK12 25 mg/kg

Key Milestones

KIN-2787	Initiate Phase 1 Trial in Class II & III BRAF-driven advanced adult solid tumors (KN-8701)	✓
	Initiate Phase 1/2 binimetinib (bini) combination portion of KN-8701 in $NRAS^{mut}$ Melanoma	**H1 2022**
	Initial monotherapy data from ongoing Phase 1 trial (KN-8701)	**Q3 2022**
	Initial Phase 1/2 bini combination data in $NRAS^{mut}$ Melanoma from KN-8701	**YE 2022**
KIN-3248	Initiate Phase 1 Trial in FGFR2 & FGFR3 driven, FGFR inhibitor naïve and pretreated advanced adult solid tumors	**H1 2022**
Pipeline	Announce next pipeline target	**H2 2022**
Kinnjiu	Form Joint Venture in China (Kinnjiu)	✓
	Initiate KIN-2787 Phase 1 Trial in Greater China	**mid 2022**