



Corporate Overview

January 2026

Company Disclaimer

Certain statements set forth in this presentation by Pasithea Therapeutics Corp's (the "Company") contain forward-looking statements, within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, that reflect the Company's plans, beliefs, expectations and current views with respect to, among other things, future events and financial performance (collectively referred to herein as "forward-looking statements") including plans to develop, manufacture and commercialize its product candidates, the treatment potential of PAS-004, the translation of preclinical data into human clinical data, the design, enrollment criteria and conduct of the ongoing Phase 1 clinical trial of PAS-004 in advanced cancer patients and the ongoing Phase 1/b clinical trial of PAS-004 in adult neurofibromatosis type 1 (NF1) patients, the ability of initial clinical data to de-risk PAS-004 and be confirmed as studies progress, including the safety, tolerability, pharmacokinetics, pharmacodynamics and potential efficacy of PAS-004, 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 the Company in future clinical studies, the filing with, and approval by, regulatory authorities of our product candidates, the sufficiency of funds to operate the business of the Company, statements regarding the Company's ability to advance its pipeline and further diversify its portfolio, the Company's cash needs and availability, including our projected cash runway and current operating plans, and the plans and objectives of management for future operations. Forward-looking statements involve known and unknown risks, uncertainties, assumptions and other important factors that could cause our actual results, performance or achievements or industry results to differ materially from historical results or any future results, performance or achievements expressed, suggested or implied by such forward-looking statements.

These include, but are not limited to, statements about the Company's ability to develop, obtain regulatory approval for and commercialize its product candidates; our ability to submit Investigational New Drug applications ("IND"), or IND amendments or comparable documents in foreign jurisdictions in order to commence clinical trials on the timelines we expect; initiation of preclinical studies and clinical trials, and results of preclinical studies and clinical trials for our product candidates; the Company's success in early preclinical studies, which may not be indicative of results obtained in later studies or clinical trials; the potential benefits of our product candidates, including efficacy and safety profiles of our product candidates; the Company's ability to obtain regulatory approval to commercialize our existing or any future product candidates; the Company's ability to identify patients with the diseases treated by our product candidates, and to enroll patients in clinical trials; the Company's expectations regarding collaborations and other agreements with third parties and their potential benefits; the Company's ability to obtain, maintain and protect our intellectual property; the Company's ability to identify, recruit and retain key personnel; the Company's expected use of cash and cash equivalents to fund its operations; the Company's financial performance; developments or projections relating to the Company's competitors or industry; the impact of laws and regulations; the Company's expectations regarding the time during which it will be an emerging growth company under the JOBS Act; and other factors and assumptions described in the Company's public filings with the Securities and Exchange Commission ("SEC") and other documents we file from time to time with the SEC.

These statements are based on the Company's historical performance and on its current plans, estimates and projections in light of information currently available to the Company, and therefore, you should not place undue reliance on them. The inclusion of forward-looking information should not be regarded as a representation by the Company or any other person that the future plans, estimates or expectations contemplated by us will be achieved. Forward-looking statements made in this presentation speak only as of the date of this presentation, and the Company undertakes no obligation to update them in light of new information or future events, except as required by law.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, 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.

Investment Highlights



PAS-004 is a next-generation macrocyclic MEK inhibitor

- Designed to address the limitations and liabilities associated with earlier generation MEK inhibitors
- Macrocyclic molecules typically exhibit superior target binding, oral bioavailability, potency and pharmacokinetics (PK) properties



Encouraging interim safety, PK and early signs of monotherapy efficacy demonstrates differentiated profile ¹

- No Dose Toxicities (DLTs), ocular retinal abnormalities or cardio toxicities; All AEs are Grade 1 / 2, through cohort 7
- PK profile supports long half-life (~60 hours in human) with dose proportional PK as well as cohort 7 Cmax and Cmin above the IC50 from our cellular assay
- Extensive exposures at steady state with the potential for constant suppression of the MAPK pathway
- Partial response observed in MEK rechallenged patient with BRAF V600E mutated tumor after 11 months of treatment, remains on trial



Initial indication is Neurofibromatosis Type 1 (NF1)-plexiform neurofibromas (NF1-PN)

- NF1-PN to be followed by NF1-cutaneous neurofibromas (NF1-CN)
- Multiple development opportunities as MAPK pathway is critical in multiple diseases



Large market opportunities

- MEK inhibitors generated ~\$2.4B in sales in 2023 ²
- NF1-PN is a \$2+ billion Total Addressable Market (TAM): Koselugo (selumetinib) worldwide sales of \$531M in 2024 in pediatric NF1-PN patients ³
- NF1-CN is a potential \$1+ billion TAM per management estimates

Leadership team with broad range of experience and success



Dr. Lawrence Steinman

- Executive Chairman & Co-Founder

- Endowed Chair in the Neurology Dept. at Stanford University. Member of the National Academy of Sciences.
- Founded and served on board of successful biotech companies, including Neurocrine Biosciences Inc. (Founder and Board Member) and Centocor (Board Member and head of SAB) until sold to J&J.
- Drug development pioneer in MS, with research that led to the development of the drug Tysabri.



Dr. Tiago Reis Marques

- Chief Executive Officer & Co-Founder

- Fellow at Imperial College and lecturer at King's College London.
- Renowned psychiatric researcher and lecturer with decades of experience in the biological mechanisms of mental health and brain disorders.



Daniel Schneiderman

- Chief Financial Officer

- 20+ years of experience in the capital markets and operations.
- Senior financial roles at translational biotech companies, including, MetaStat, Inc., Biophytis SA and First Wave BioPharma, Inc.



Dr. Rebecca Brown

- Scientific Advisory Board Member

- Associate Professor of Neuro – Oncology at UAB Medicine
- Research investigates novel treatments for tumors including NF1-PNs, NF1-CNs, gliomas, and meningiomas

Pipeline focused on developing MEKi for multiple indications

Program	Drug modality	Target	Indication	Target ID / Validation	Lead Selection	IND Enabling	Phase I	Next Steps
PAS-004	Macrocyclic Small molecule	MEK 1/2	RAS, RAF, NF1 mutated cancers					Updated interim data in Q2 2026
			NF1-PN (adult)					Interim data in 2H 2026
PAS-003	Monoclonal antibody	$\alpha 5\beta 1$ Integrin	Amyotrophic Lateral Sclerosis (ALS)					Partnership opportunity
PAS-001	Small molecule	C4A	Schizophrenia					Partnership opportunity

PAS-004

Next Generation MEK Inhibitor for
The Treatment of Neurofibromatosis
Type 1 (NF1) and Solid Tumors

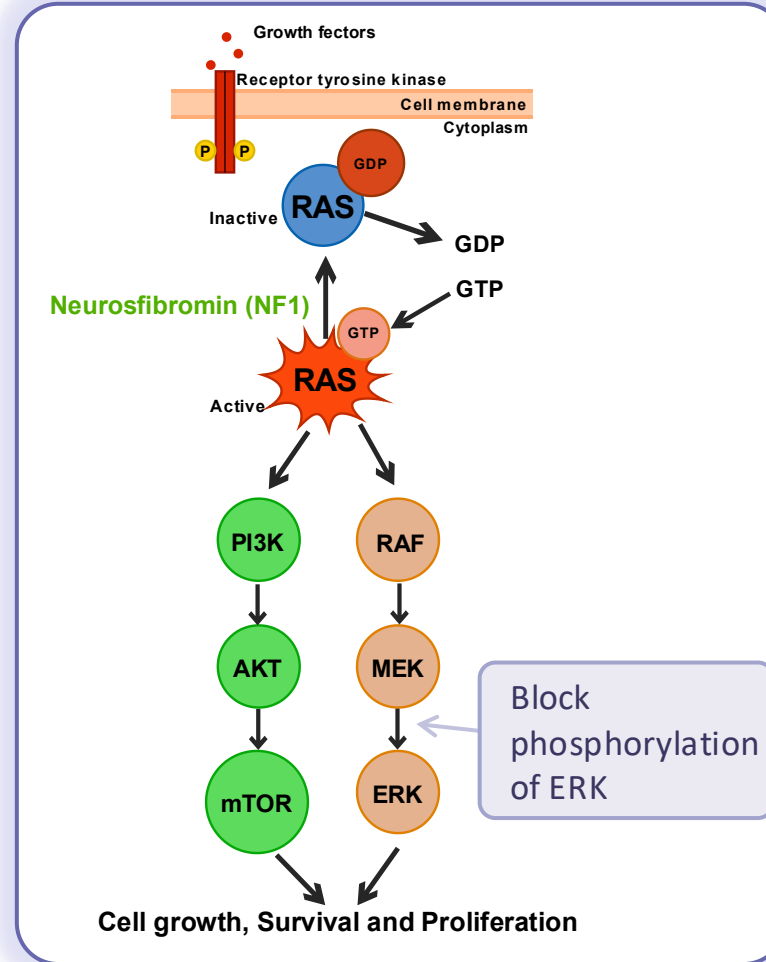
MAPK Pathway dysregulation is implicated in cancer and NF1/RASopathies



The mitogen-activated protein kinase (**MAPK**) pathway is a chain of proteins that are essential for cell function by regulating cellular transcription, proliferation, survival and other functions.



When abnormally activated, the MAPK pathway is critical for the formation and progression of tumors, fibrosis and other diseases (Rasopathies).



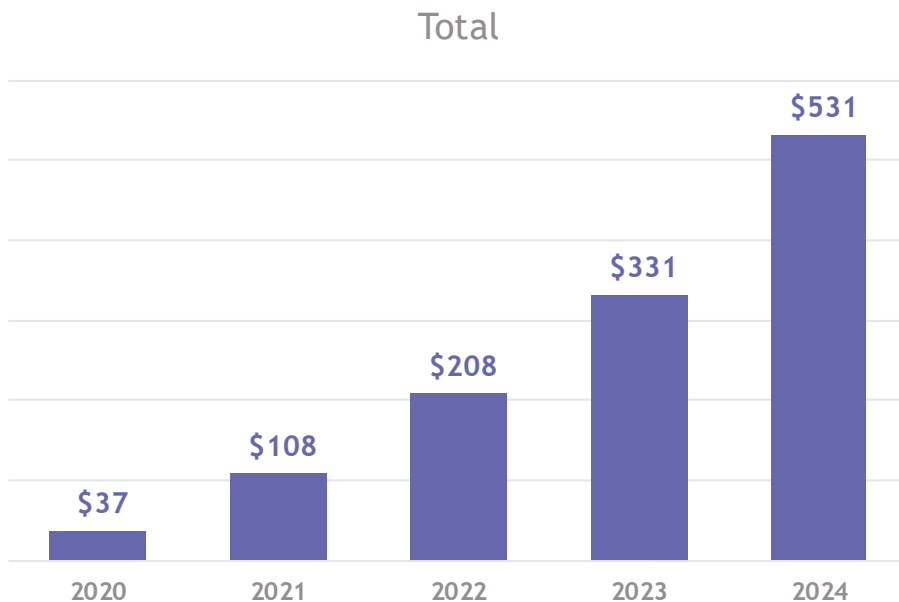
Alterations in RAS or RAF have been described in many cancers, including melanoma and colorectal where MEK inhibitors are approved.



NF1 arises from mutations in the NF1 gene, which encodes for neurofibromin, a key negative regulator of MAPK pathway by inactivating RAS.

NF1: Large unmet medical need and market opportunity

Koselugo (selumetinib) Worldwide Sales in Pediatrics ¹



NF1 is an autosomal dominant genetic disorder.



Affects approximately one in 3,000 newborns worldwide with ~100,000 patients living in U.S. with NF1².



30-50% of NF1 patients develop plexiform neurofibromas (NF1-PN).
>95% develop cutaneous neurofibromas (NF1-CN).



PNs are benign peripheral nerve sheath tumors that can cause severe complications, including disfigurement, pain, motor dysfunction, and neurological impairment and have malignant transformation potential.



Surgical resection of PN is challenging. Currently there are 2 MEKi approved for NF1-PN, Koselugo (pediatric) and Gomekli (adults and pediatric). Neither are approved for NF1-CN.



CNs usually presents as skin bumps and can cause disfigurement, irritation, pain and quality of life challenges. There are currently no approved treatments.

NF1 Tumor Conditions



Neurofibromas are noncancerous (benign) tumors that are derived from Schwann cell lineage



Plexiform neurofibromas can undergo malignant transformation at a rate of approximately 8-13%

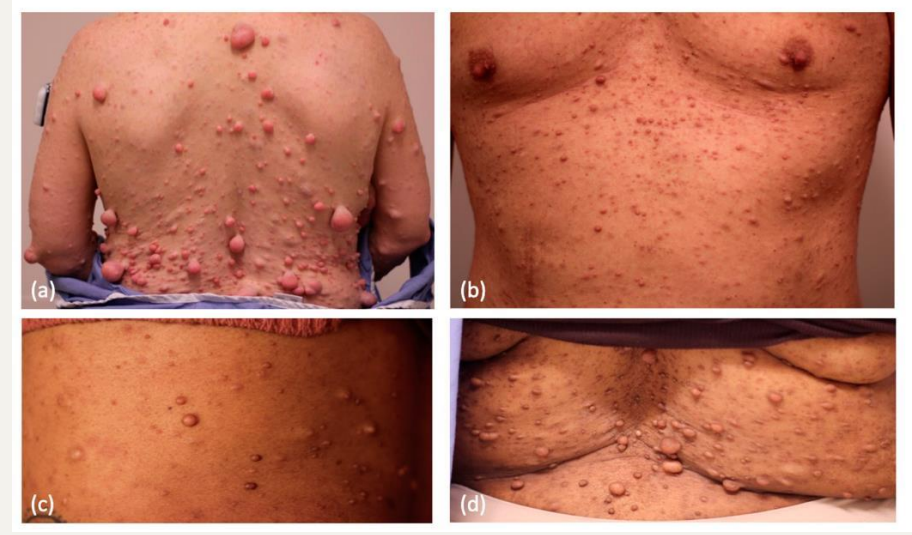


Cutaneous neurofibromas are disfiguring, and typically cause irritation, pain and discomfort

Plexiform Neurofibromas ¹



Cutaneous Neurofibromas ²



Where and how to improve on FDA-approved MEKi for NF1 treatment



Suboptimal profile



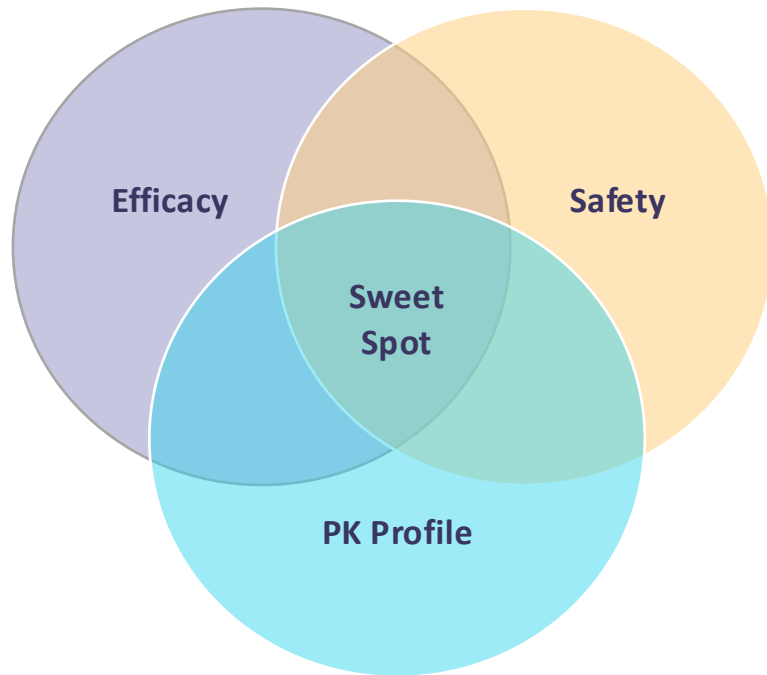
Long time to generate responses



Poor tolerability and compliance

WARNING AND PRECAUTIONS	Koselugo ¹	Gomekli ²	Gomekli ²	Gomekli ²
ADVERSE REACTIONS	Pediatric	Adult	Pediatric	Pooled
SELECT SAFETY AND AE INFORMATION				
Ocular Toxicity	15%	28%	19%	25%
Cardiomyopathy (left ventricle dysfunction)	17%	16%	25%	20%
Dermatologic/Skin (Rash)	68%	92%	72%	84%
Gastrointestinal Toxicity (Diarrhea)	59%	59%	55%	57%
Increased Creatinine Phosphokinase (CPK)	55%	55%	59%	57%
Increased Levels of Vitamin E and Risk of Bleeding	Yes	N/A	N/A	N/A
Vomiting	64%	38%	39%	39%
Nausea	49%	52%	27%	40%
OTHER EFFICACY AND KEY INFORMATION				
Time to generate response (months)	7.2	7.8	7.9	N/A
ORR	44%	41%	52%	N/A
Dosing Frequency	BID continuous	BID 3 weeks on/1 week off		

Macrocycle structure may position PAS-004 into the “Sweet Spot” for NF1



Efficacy



- Evidence of monotherapy activity in MEK rechallenged patients
- No plateau effect observed in cell lines
- Sustained pathway inhibition

Safety



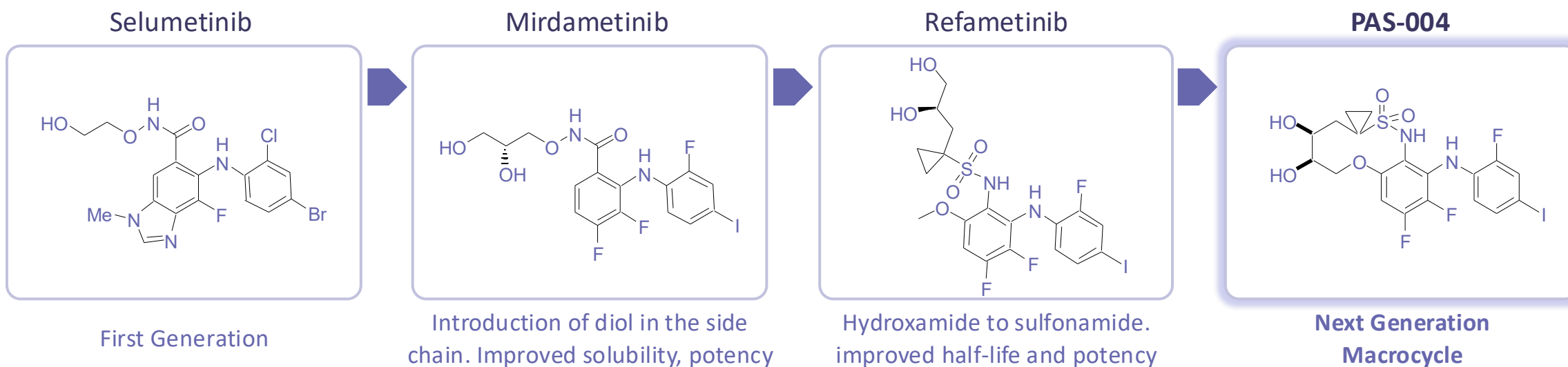
- Macrocyclic molecules are more rigid with possible less “off target” side-effects
- Favourable AE profile, All TRAEs to date have been Grade 1 or 2
- Dosing convenience – once per day or less frequent dosing expected to enhance compliance

PK Profile



- >60 Hour half-life enables pathway titration and continuous suppression
- C_{max} to C_{min} ratio below 2 at steady state – demonstrating relatively flat PK curve
- Cohort 7 C_{max} and C_{min} above the IC₅₀ from our cellular assay
- Differentiated PK profile compared to FDA-approved MEKi for NF1-PN

PAS-004 was designed to address the liabilities of previous MEK inhibitors



- PAS-004 is the first MEK inhibitor with a Macrocyclic structure ($IC_{50} = 2 \text{ nM}$)
- Modifications in chemical structures such as removing metabolically unstable groups, including removal of a primary alcohol may have a big impact on drug properties:



Stronger binding with drug target receptors



Reduced potential for active metabolites



Improved oral bioavailability, PK properties and potency



Better safety and tolerability profile

Constant suppression of the MAPK pathway may lead to better outcomes

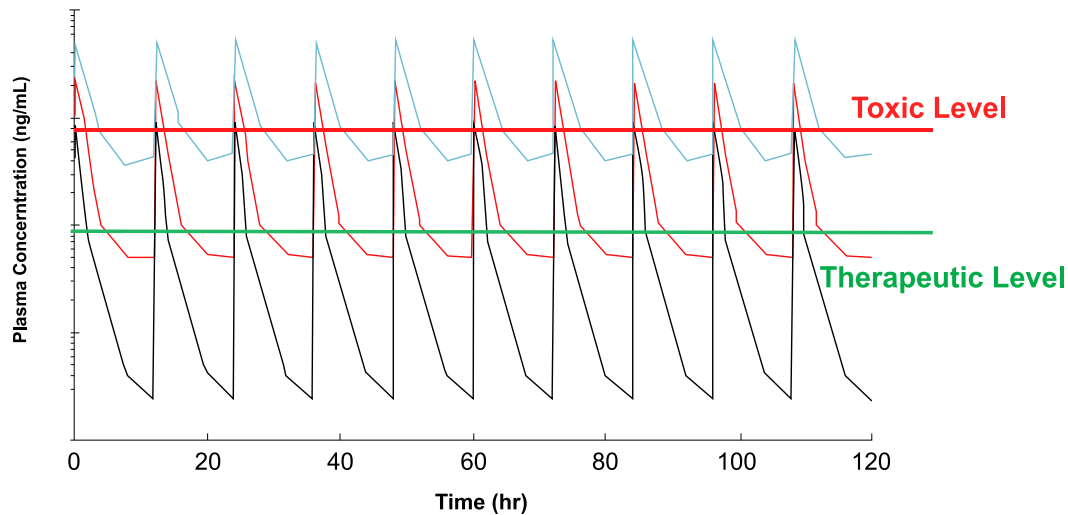
PAS-004 was designed to have a PK profile suited to enhance efficacy and limit toxicity

Short half-life:

May lead to significant fluctuations in plasma concentrations

Target is not continuously inhibited

Toxicities may be associated with Cmax spikes

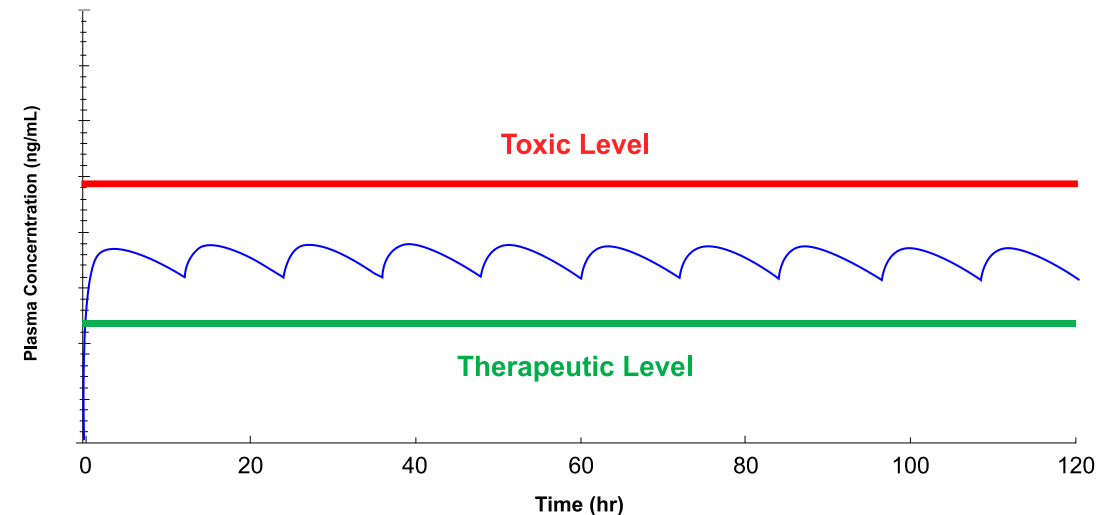


Long half-life:

Achieve steady-state levels and maintain continuous inhibition

Allows for pathway titration

Low Cmax to Cmin ratio avoids Cmax spikes



Not actual data; For illustrative purposes only

Approved MEK inhibitors

Typical liabilities associated with approved MEK Inhibitors:



High toxicity and dose limiting side effects / Selective MEKi have short half lives



Toxicity and PK profile limits MEKi use in combination therapy

Drug	Company	Development Approach	Tumor Type	Dosing
Selumetinib (Koselugo)	AstraZeneca 	Monotherapy (pediatric + adult)	NF1-PN (pediatric)	BID dosing
Mirdametinib (Gomekli)	Spring Works 	Monotherapy (pediatric + adult)	NF1-PN (adult & pediatric)	- BID Dosing 3 week on 1 week off Interval
Binimetinib (Mektovi)	Pfizer 	+ B-Raf inhibitors	Melanoma, NSCLC, Thyroid cancer, BRAF V600E	BID Dosing
Cobimetinib (Cotellic)	Genentech 	+ B-Raf inhibitors	Melanoma	QD Dosing
Trametinib (Mekinist)	Novartis 	+ B-Raf inhibitors	Melanoma	QD dosing
Avutometinib	VeraStem 	+ Defactinib (FAK) combination	LGSOC with KRAS mutation	1st and 4th day

Dose Dependent inhibition of pERK correlates with clinical efficacy / PAS-004 is more potent than Selumetinib in *In Vitro* NF1 model



PAS-004 dose dependent tumor response correlates with dose dependent pERK reduction
Partial pERK reduction may be sufficient in NF1



PAS-004 is more potent in all 3 NF1 mutated cell lines than Selumetinib

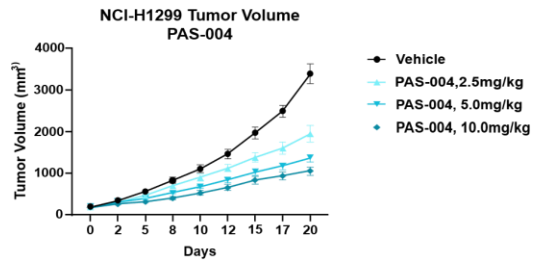


No plateau effect was observed for PAS-004 = potential for deeper clinical activity



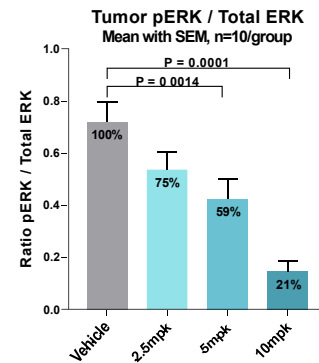
Limited activity against the control NF1 WT cells = potential for enhanced safety profile

In Vivo Dose dependent efficacy (NCI-H1299 xenograft)



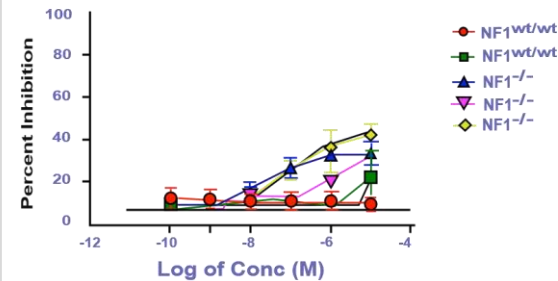
Non-published study conducted at Wuxi AppTec

In Vivo Dose dependent pERK reduction (NCI-H1299 xenograft)



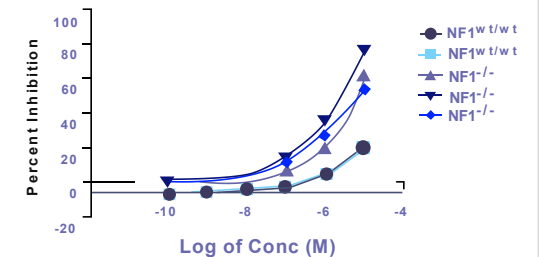
Non-published study conducted at Wuxi AppTec

Selumetinib



Non-published study conducted at Raymond Mattingly's lab, Wayne State Univ.

PAS-004

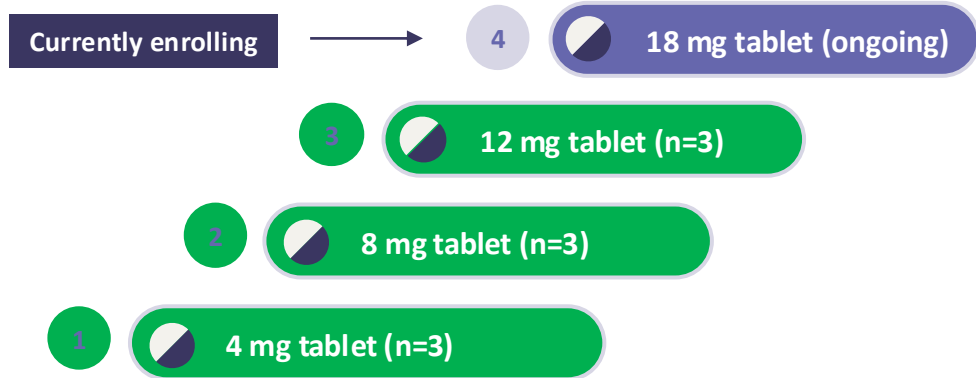


Non-published study conducted at Raymond Mattingly's lab, Wayne State Univ.

PAS-004: Ongoing NF1 open-label dose escalation Phase I/IB clinical trial

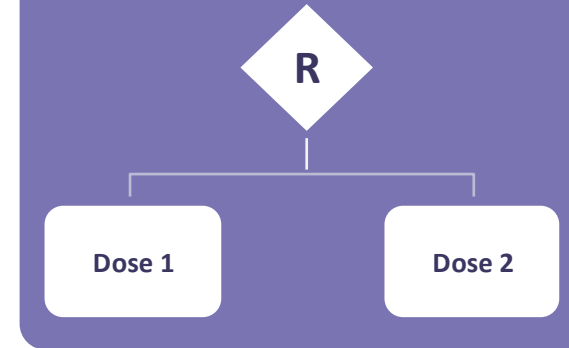
Part A Modified 3+3 Dose Escalation Study Design

Four dose cohorts in up to 24 patients to determine Recommended Part B Dose



Part B Dose Expansion

Two Part A dose levels will be selected and randomized (12 patients per cohort) to determine RP2D



Recommended Phase 2 Dose

Patient Population
(n= up to 48)

Adult participants with NF1 with symptomatic and inoperable, incompletely resected, or recurrent PNs



5 sites: US, Australia and South Korea



TRIAL OBJECTIVES

Primary

- Assess the safety, tolerability, PK and PD of PAS-004, in adult participants with NF1-PN

Secondary

- Identify the RP2D
- Characterize PK and PD profile in NF1
- Evaluate the preliminary efficacy of PAS-004 on target PN volume
- Evaluate the preliminary efficacy of PAS-004 on the size, appearance and associated symptoms of CNs

PAS-004: Ongoing open-label dose escalation Phase I clinical trial in cancer

Patient Population (n=~36)



Patients with MAPK pathway driven solid tumors with a documented RAS, NF1, or RAF mutations or patients who have failed BRAF/MEK inhibition



7 sites in US and Eastern Europe



TRIAL OBJECTIVES

Primary

- To evaluate the safety and tolerability of PAS-004 in patients with MAPK pathway driven advanced solid tumors.

Secondary

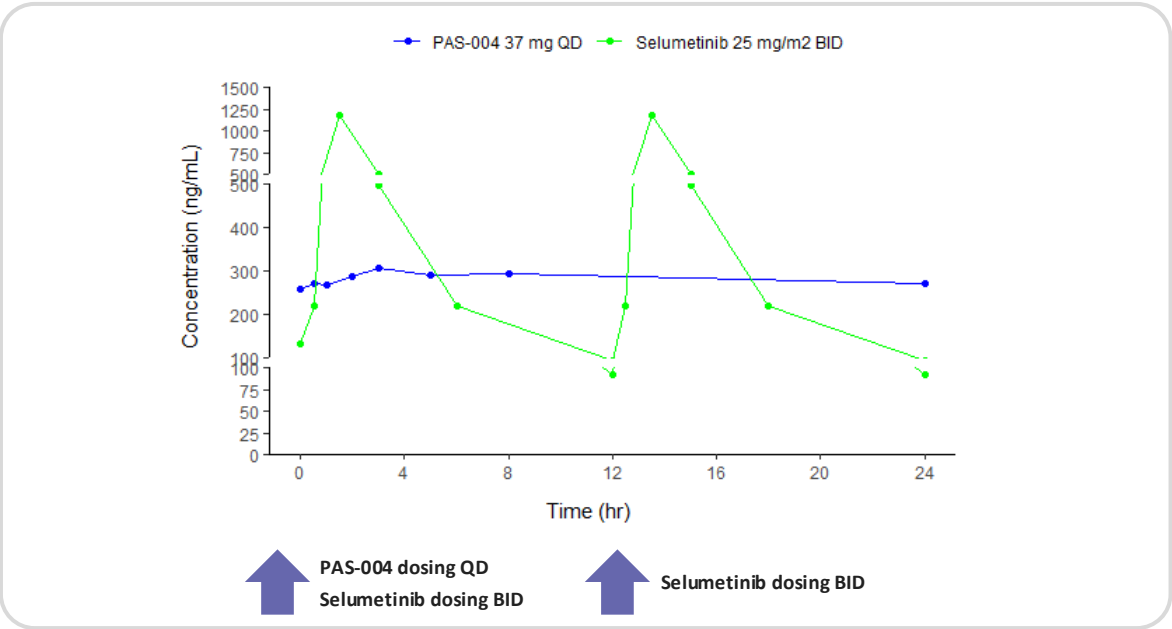
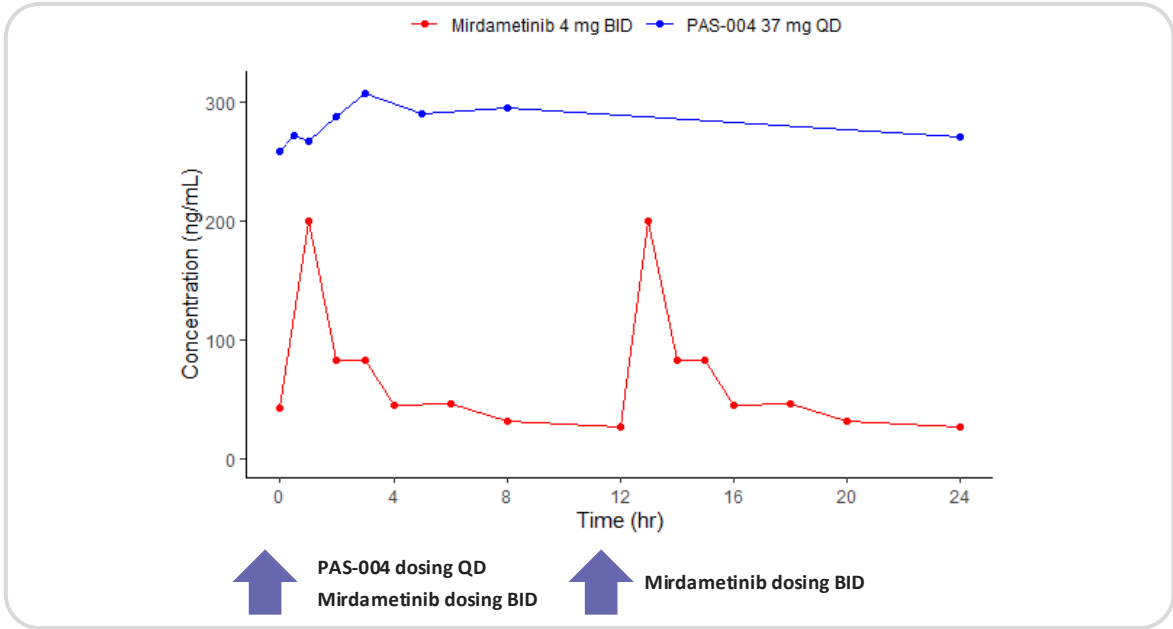
- Pharmacokinetic (PK) profile
- Pharmacodynamic (PD) effects (ERK phosphorylation)
- Define the recommended Phase 2 dose
- To evaluate the preliminary anticancer activity

Modified 3+3 Dose Escalation Study Design

Cohort

1	2 mg capsule n=3
2	4 mg capsule n=3
3	8 mg capsule n=3
4a	15 mg capsule n=3
4b	4 mg tablet n=5
5	22 mg capsule n=3
6	30 mg capsule n=6
7	37 mg capsule n=3
8	45 mg capsule (ongoing)

PAS-004 Demonstrates Favorable Human PK Properties in Advanced Cancer Study

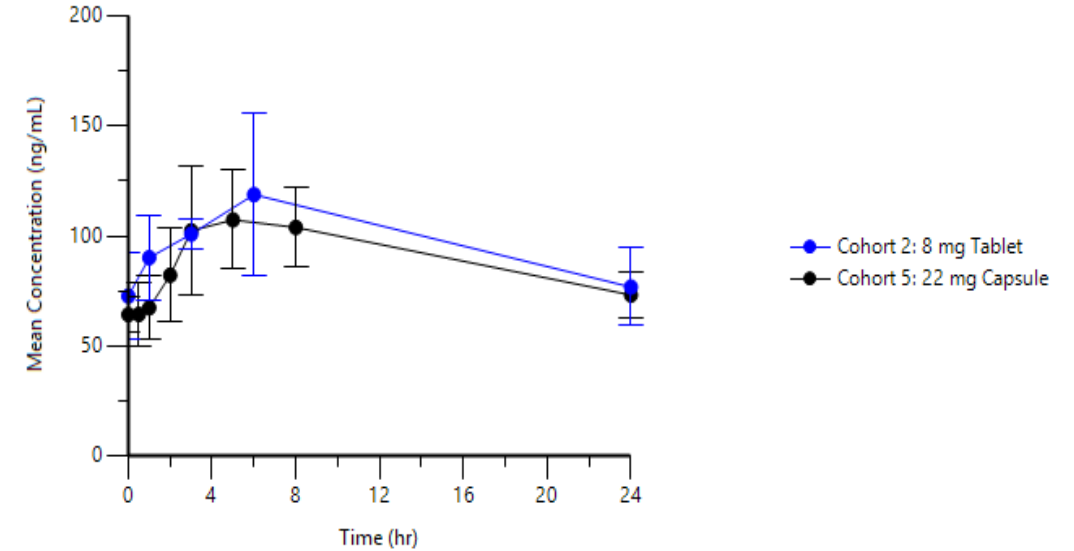
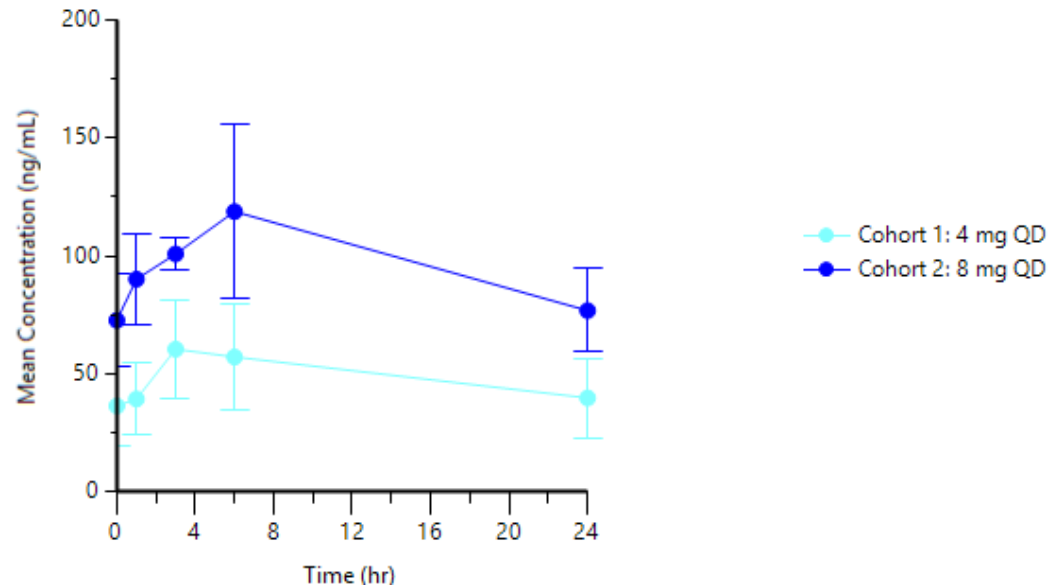


Steady State Parameter	PAS-004	Mirdametinib ¹	Selumetinib ²
Dosing regimen	37 mg QD	4 mg BID	25 mg/m ² BID
C _{max} (ng/mL)	313	198	1,168
AUC ₀₋₂₄ or AUC ₀₋₁₂ x2 (ng*h/mL)	6690	1,382	7,864
C _{tau} (ng/mL)	260	~30	~90



1 - LoRusso PM et al. Phase I pharmacokinetic and pharmacodynamic study of the oral MAPK/ERK kinase inhibitor PD-0325901 in patients with advanced cancers. Clin Cancer Res. 2010 Mar 15;16(6):1924-37.
2 -Wang Z, et al. Safety, pharmacokinetics and efficacy of selumetinib in Chinese adult and paediatric patients with neurofibromatosis type 1 and inoperable plexiform neurofibromas: The primary analysis of a phase 1 open-label study. Clin Transl Med. 2024 Mar;14(3): e1589.

Favorable PK Properties Replicated in Ongoing Phase 1/1b Adult NF1-PN Study

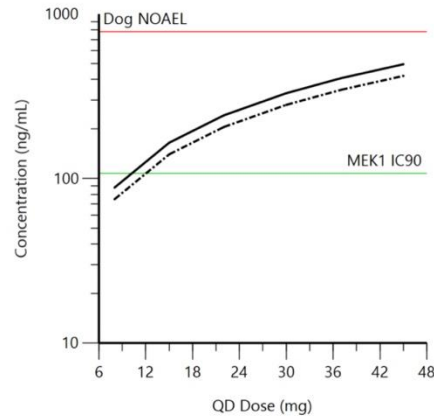


Steady State Parameter	4mg Tablet QD	8mg Tablet QD	22mg Capsule QD
Study Cohort	NF1 Cohort 1	NF1 Cohort 2	Advanced Cancer Cohort 5
C _{max} (ng/mL)	58.1	118	108
AUC ₀₋₂₄ (ng*h/mL)	1,120	2,290	2,160
C _{tau} (ng/mL)	37.6	75.4	72.7

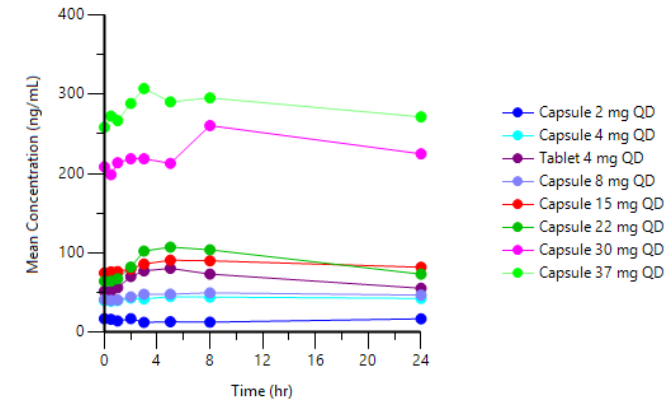
PK profile differences (PAS-004 vs Selumetinib)

PAS-004 > 60 hour half life and C_{max} / C_{min} ratio below 2

C_{max} & PD Modeling

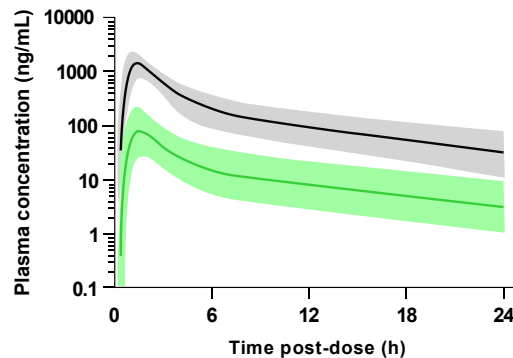


PAS-004 Day 22

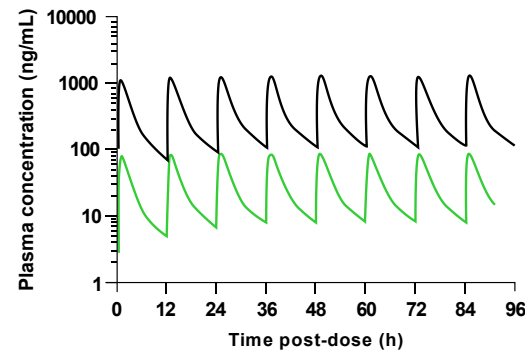


Selumetinib PK Curves ¹

A] Population profiles - Adults - 75 mg SD



B] Mean steady state profiles - Adults - 75 mg bid



— Selumetinib
— N-Desmethyl-Selumetinib
(active metabolite)

AUC correlates with treatment responses in NF1



Positive relationship between treatment response and mirdametininb exposure (AUC_{0-12h}) ($p = 0.052$)



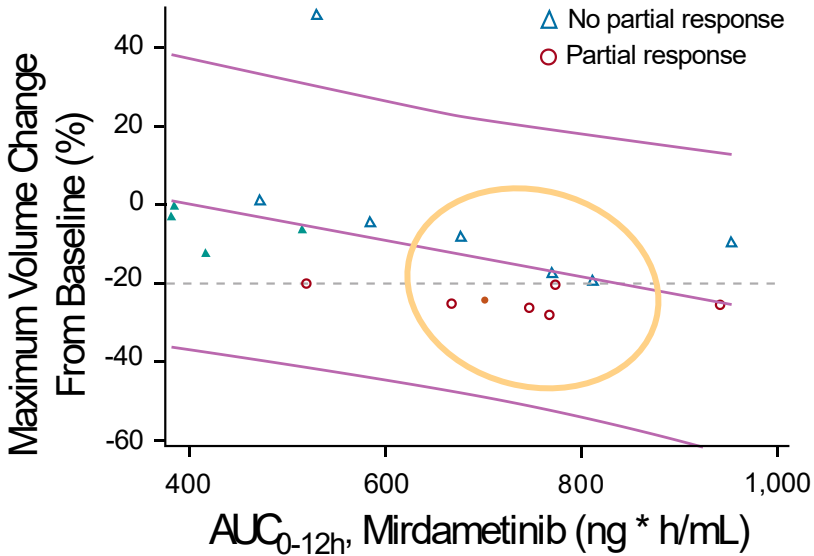
The majority of Gomekli responses were seen in patients whose AUC was ≥ 600 ng*h/ml



PAS-004 dose of 30mg capsule at steady state (AUC_{0-24h}) over 6,690 ng*h/ml

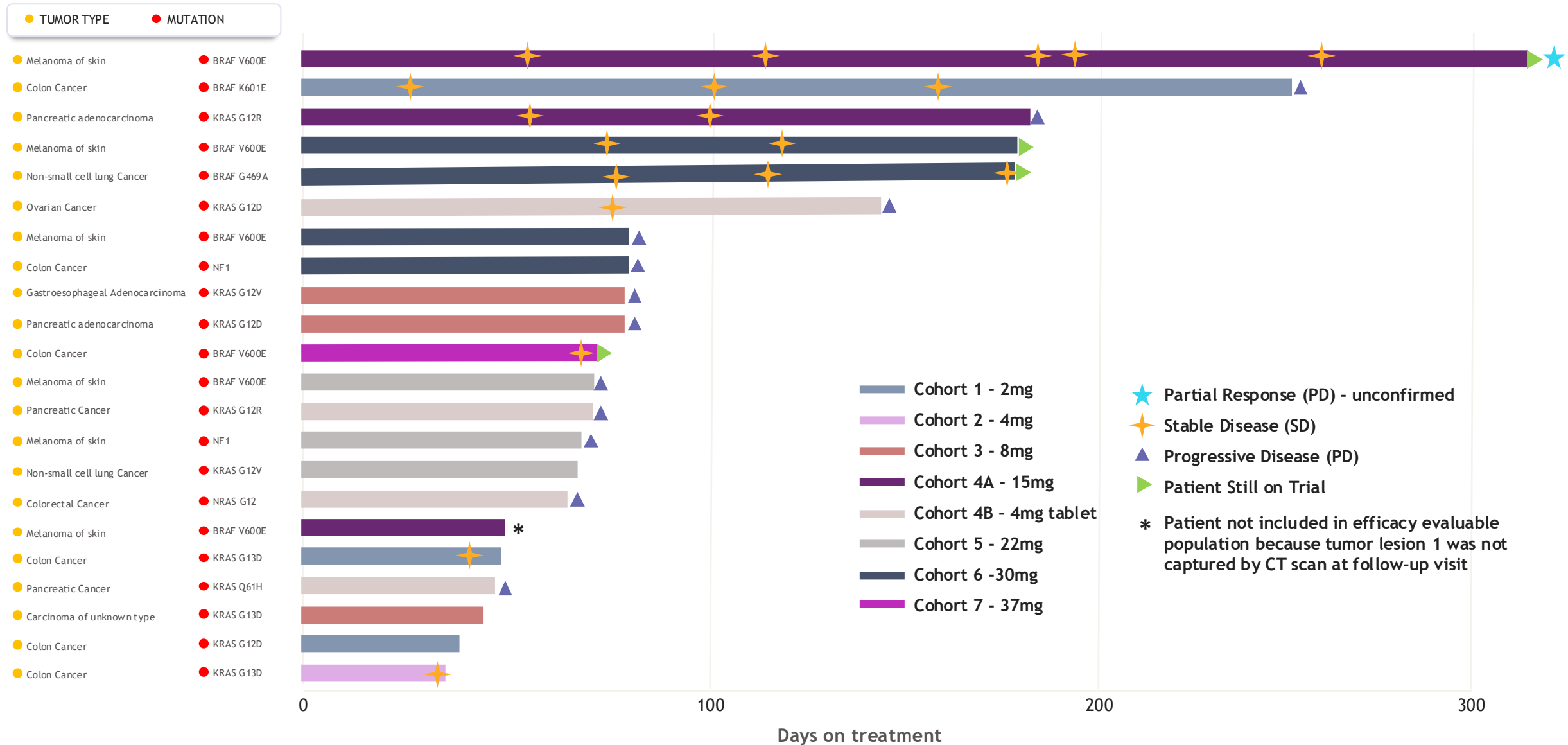


PAS-004 dose of 8mg tablet (AUC_{0-24h}) over 2,290 ng*h/ml



PAS-004 Study Cohort	C _{max} (ng/mL) [mean CV%]	AUC ₀₋₂₄ (ng*h/mL) [mean CV%]
37 mg Capsule (n=3)	313 (26.3)	6690 (28.1)
8 mg Tablet (n=3)	118 (28.0)	2290 (24.8)

Efficacy evaluable patients in Phase 1 Advanced Cancer Study (n=22)



Cohort 1 - 2mg

Cohort 2 - 4mg

Cohort 3 - 8mg

Cohort 4A - 15mg

Cohort 4B - 4mg tablet

Cohort 5 - 22mg

Cohort 6 - 30mg

Cohort 7 - 37mg

Partial Response (PD) - unconfirmed

Stable Disease (SD)

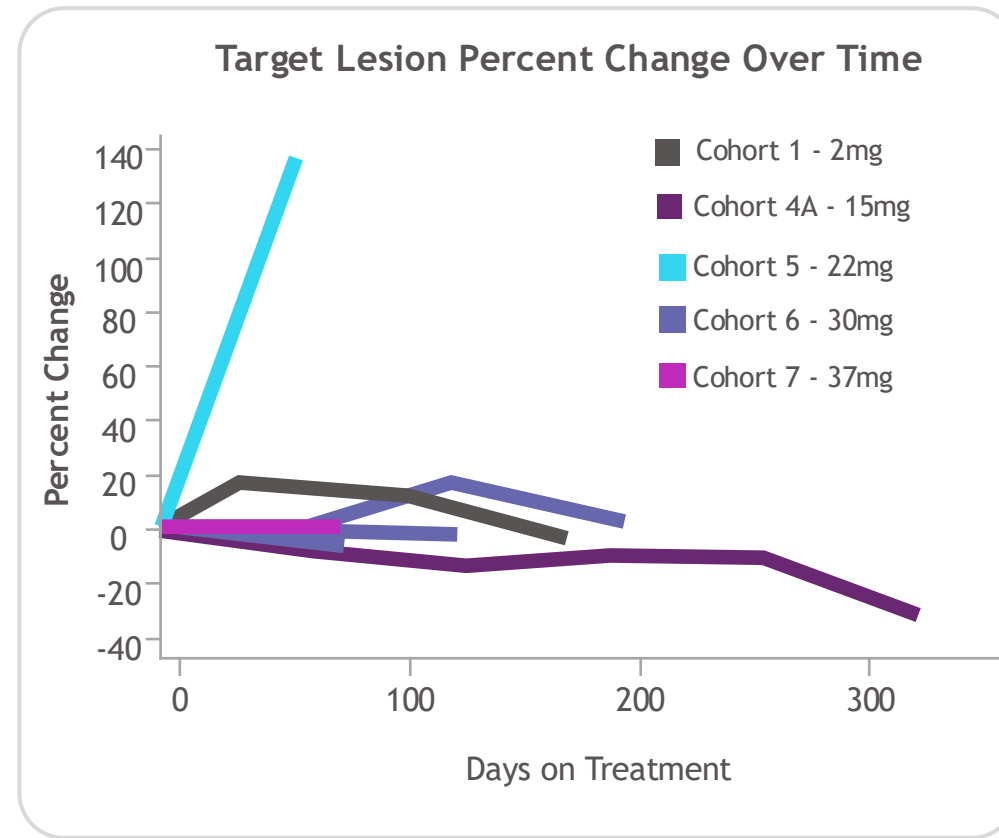
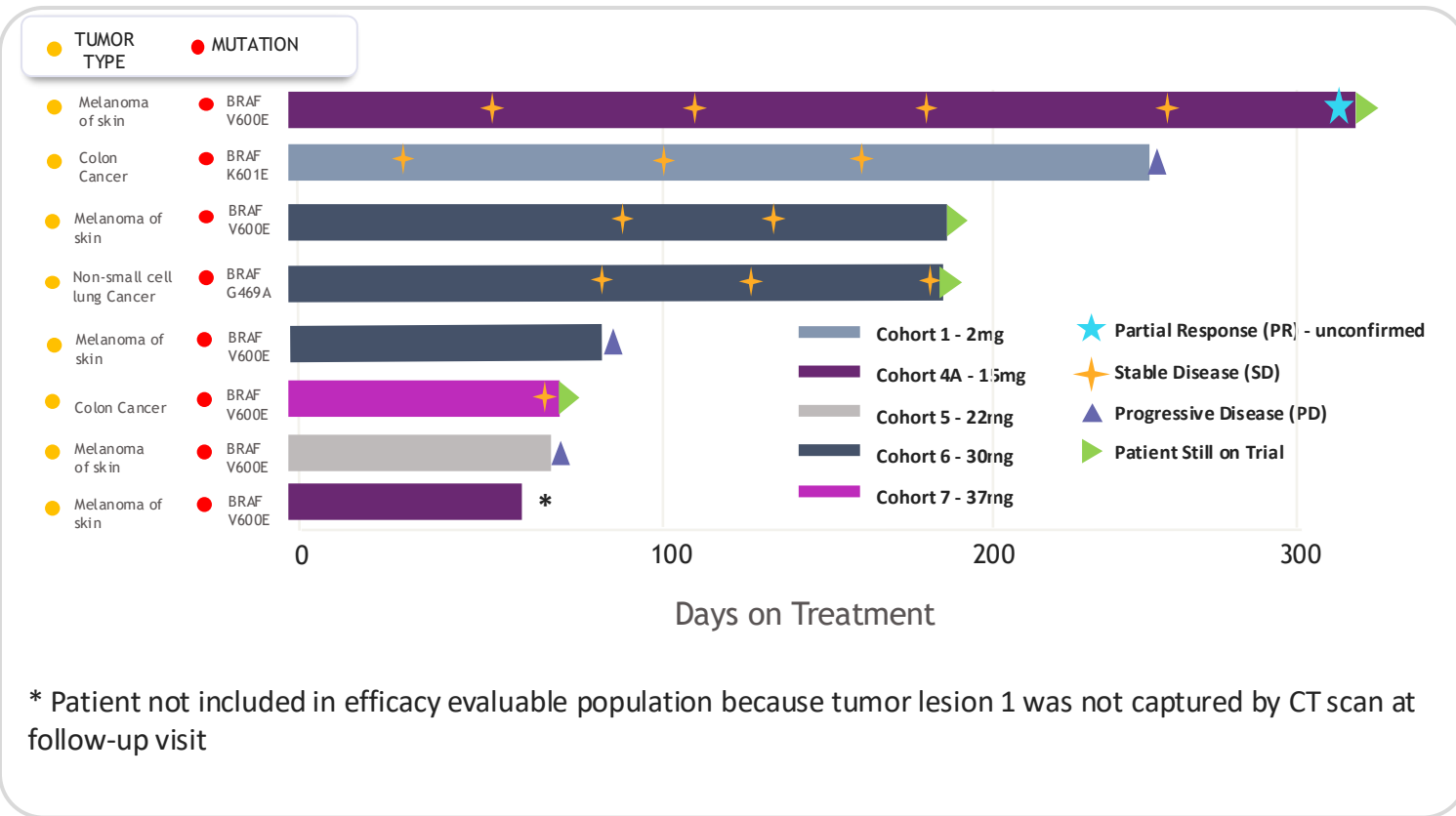
Progressive Disease (PD)

Patient Still on Trial

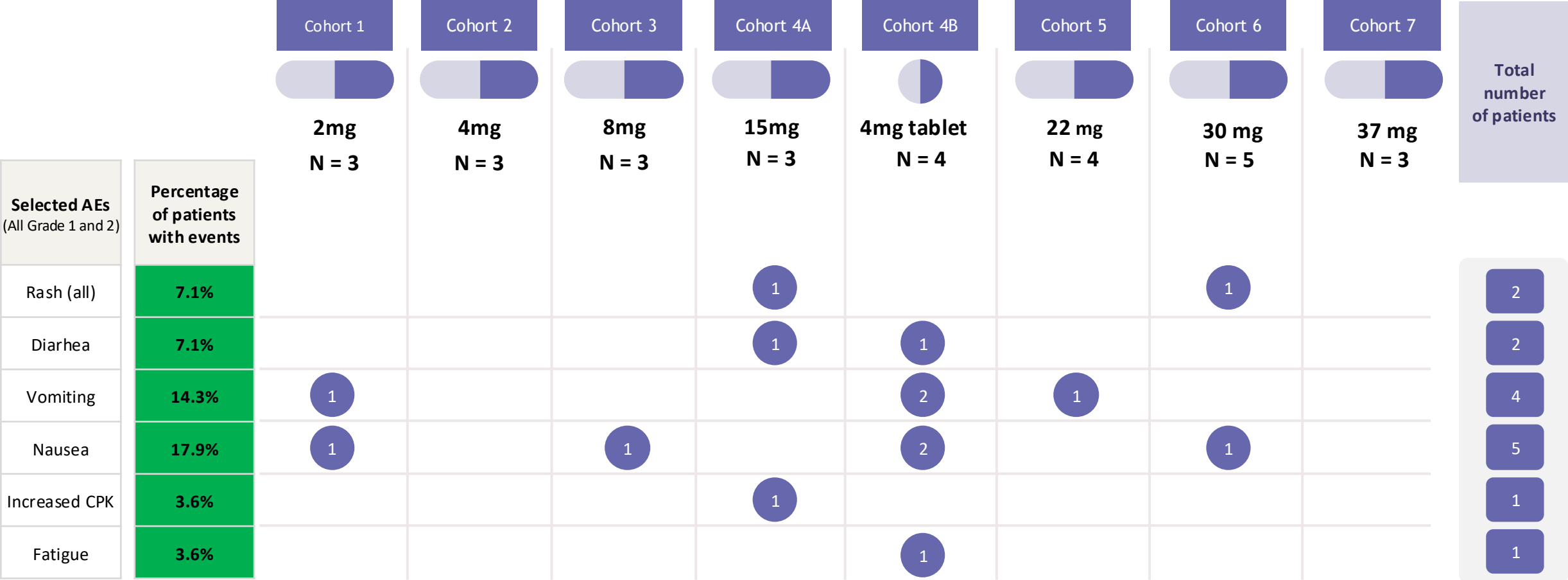
* Patient not included in efficacy evaluable population because tumor lesion 1 was not captured by CT scan at follow-up visit

Preliminary efficacy signals in BRAF mutated tumors (n=7 evaluable patients)

- ✓ Initial Disease Control Rate (DCR) of 71.4% in BRAF-mutated tumors: 5 of 7 patients achieved stable disease
- ✓ Patient in cohort 4A with BRAF V600 mutated melanoma cancer who previously received a MEK + BRAF inhibitor combination therapy achieves partial response with -31.9% tumor shrinkage and remains on trial for over 11 months



Number of patients with at least possibly related selected AEs ¹



1. Based on 28 patients completing DLT period / DLT evaluable.

Mirdametininib PK and AE profile from its Phase 1 trial in advanced cancer



PK profile with large Cmax to Cmin variability¹



Adverse events observed even at the lowest dose level, including rash and diarrhea¹

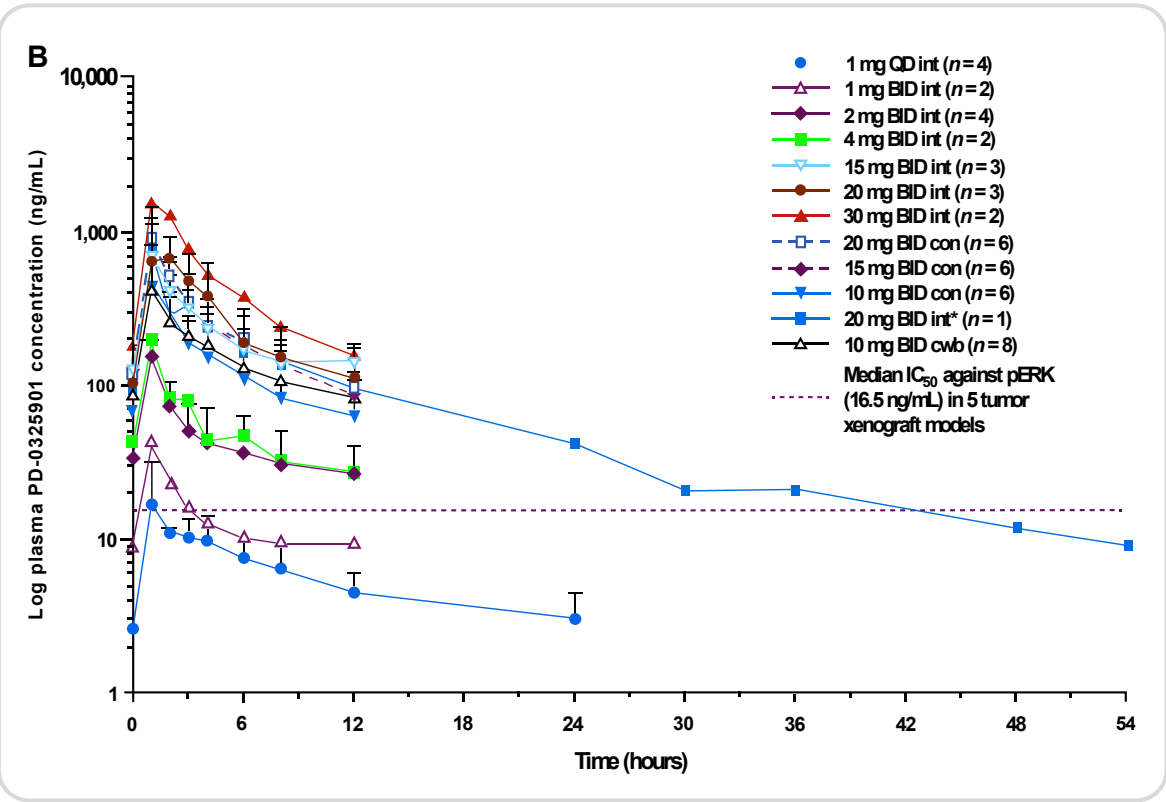
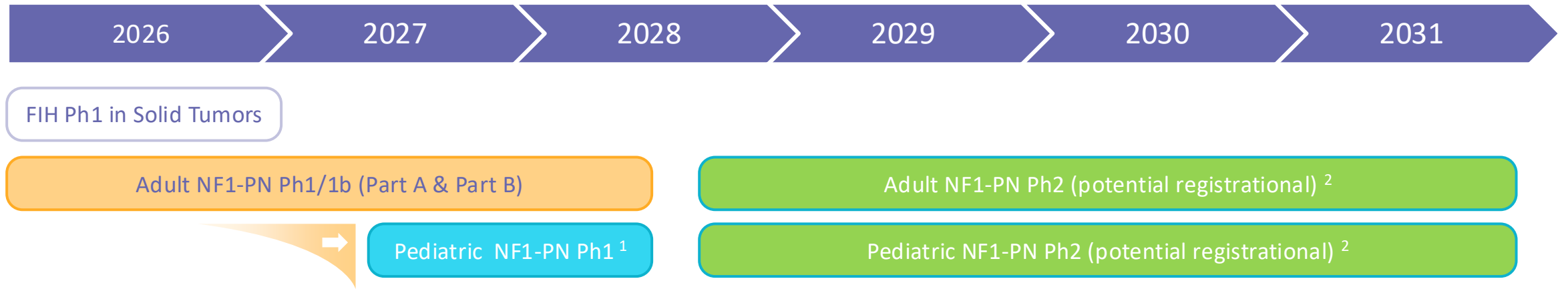


Table 3. Most common treatment-related AEs (maximum grade, all cycles)

AE	Total no. patients with events	PD-0325901 schedules and dose levels (no. patients)											
		Intermittent											
		1 mg QD (n = 4)		1 mg BID (n = 4)		2 mg BID (n = 4)		4 mg BID (n = 4)		8 mg BID (n = 3)		15 mg BID (n = 4)	
		All	G≥3	All	G≥3	All	G≥3	All	G≥3	All	G≥3	All	G≥3
Rash	52	—	—	2	—	1	—	3	—	1	—	2	—
Diarrhea	36	—	—	—	—	2	—	4	—	2	—	3	—
Fatigue	40	2	—	1	—	3	—	1	—	—	—	2	—
Nausea	25	1	—	1	—	3	—	2	—	—	—	1	—
Peripheral edema	17	—	—	—	—	—	—	—	—	—	—	—	—
Balance and gait disorders*	15	—	—	—	—	—	—	—	—	—	—	1	—
Pruritus	15	—	—	1	—	3	—	—	—	—	—	—	—
Vomiting	14	—	—	—	—	1	—	1	—	1	—	—	—
Visual disturbance	9	—	—	—	—	1	—	—	—	—	—	2	—
Blurred vision	7	—	—	—	—	—	—	1	—	—	—	1	—
Periorbital edema	6	—	—	—	—	—	—	1	—	—	—	—	—

*Includes ataxia, dizziness, and gait disturbance.

Clinical Program Timelines to Registration and Near-term Milestones



2H 2026

Initial safety and efficacy
data in NF1-PN and
NF1-CN

H1 2026

Additional safety and
efficacy in advanced
cancer study

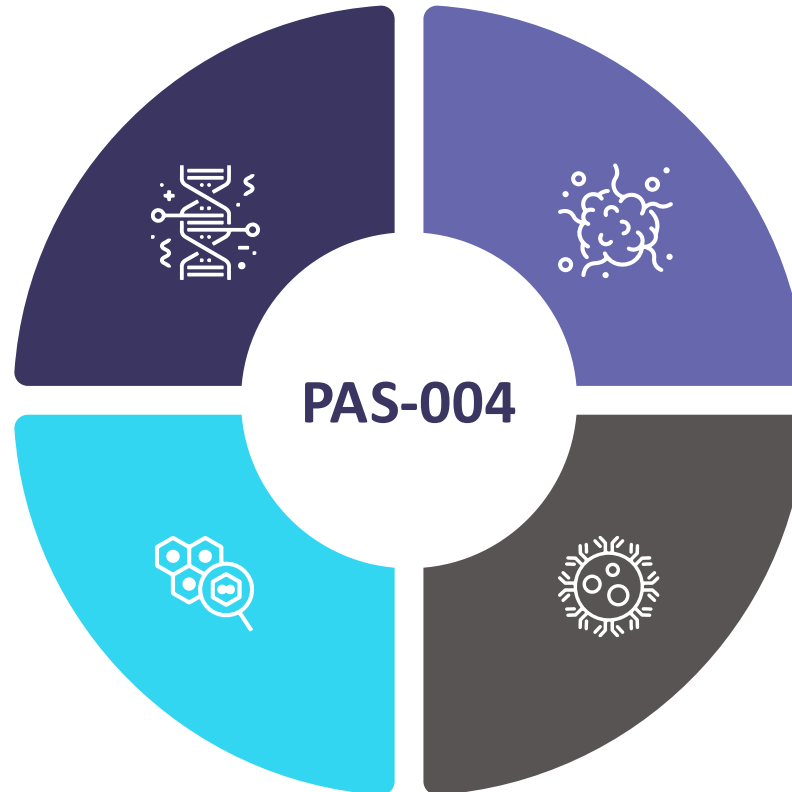
MEK inhibitors have broad potential opportunity

****Rasopathies****

- NF1 Plexiform
 - Adults
 - Pediatric
- NF1 Cutaneous
- Noonan Syndrome
- Costello Syndrome
- CM-AVM

****Rare Disease****

- ALS
- TSC (tuberous Sclerosis)
- LMNA Cardiomyopathy
- Hypertrophic Cardiomyopathy



****Cancer****

- V600 mutations
- Melanoma
- Colorectal
- Pancreatic
- BRAF Fusions
- Glioma and Glioblastoma
- AML – Ras Mutated
- MPNST – Malignant Peripheral Nerve Sheath Tumor

****ETS2 Diseases****

- Irritable Bowel Disorder (IBD)
- Ulcerative Colitis (UC)
- Crohn's Disease (CD)
- Primary Sclerosing Colingitis (PSC)
- Ankylosing Spondylitis

MEK inhibitors have broad opportunity - modulate the ETS2 pathway¹



ETS2 gene is a central regulator of human inflammatory macrophages



MEKi as a class are the strongest known ETS2 inhibitors



MEKi modulation provides potent anti-inflammatory activity, phenocopying ETS2 knock-out, modulating multiple cytokines

Article

A disease-associated gene desert directs macrophage inflammation through ETS2

<https://doi.org/10.1038/s41586-024-07501-1>

Received: 17 April 2023

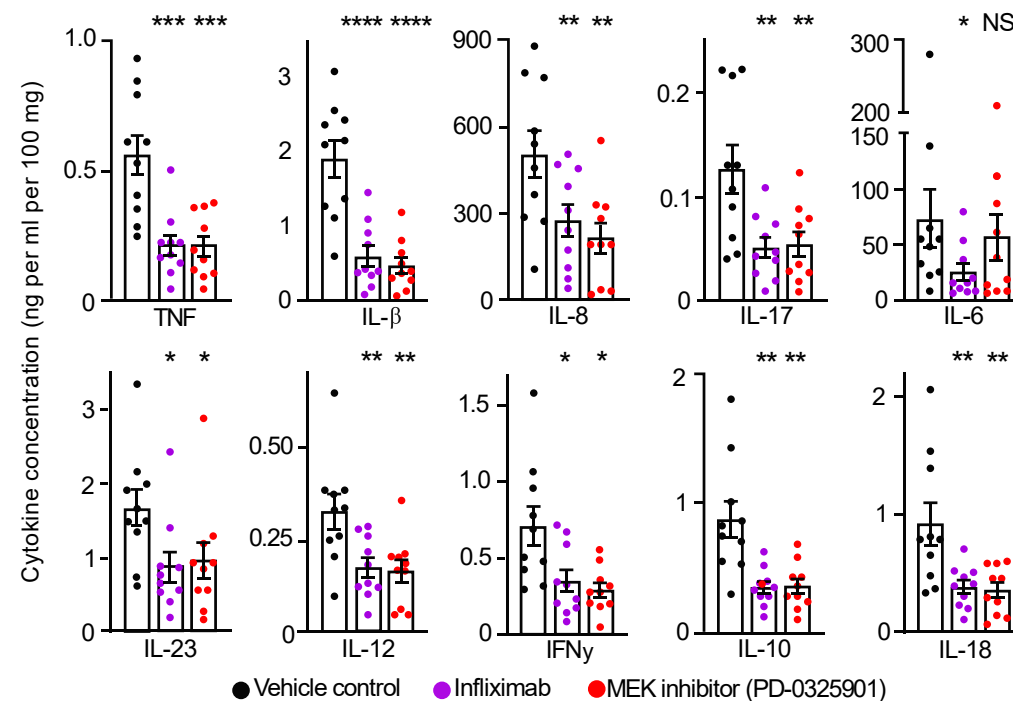
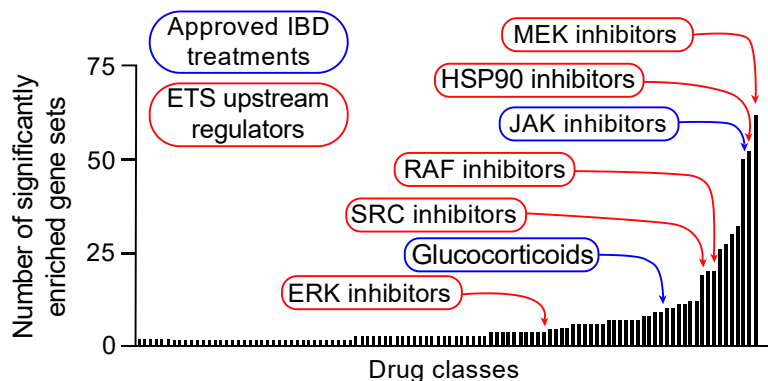
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Intellectual Property



Composition of Matter patent issued

- US patent 9034861, Exclusivity protection until Sept 2032 with extension estimated to be March 2037
- Additional 6-month exclusivity for pediatric application
- Patent issued in multiple geographies



New Composition of Matter patents filed in Jan 2024

- Based on identification of a stable crystalline form
- Anticipated patent protection at least until 2045



Orphan exclusivity

- PAS-004 received orphan-drug designation from the FDA for the treatment of NF1
- For rare diseases: 7 years exclusivity in U.S. and 10 years in European Union



Pursuing patent extension strategy

- Process patents, dosing, delivery & formulation development, follow-up compounds

Financial Overview

	Founded / IPO	2020 / 2021
	Nasdaq	KTТА
	Market Cap	\$29.7 M
	Primary Market Cap ⁽²⁾	\$113.8 M
	52 Week Low – High ⁽¹⁾	\$0.292 / \$2.82

	Stock Price ⁽¹⁾	\$1.29
	Common Shares Outstanding ⁽¹⁾	23.09 M
	Primary Shares Outstanding ⁽²⁾	88.24 M
	Fully Diluted Shares Outstanding ^(2,3)	103.02 M
	Cash Balance ⁽⁴⁾	\$59.1 M

1. As of market close on 12/31/2025.

2. Includes 65,153,335 shares of common stock issuable upon exercise of pre-funded warrants outstanding, at an exercise price of \$0.001 per share

3. Includes 13,090,120 warrants (WAEP: \$4.84) and 1,685,843 stock options (WAEP: \$2.77)

4. Cash and cash equivalents as of 9/30/25 plus proforma estimate of approximately \$54.9 million of net proceeds from the December 2025 public offering



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