RELAY®
THERAPEUTICS

New Program & Platform Event

June 2024

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This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, including, without limitation, implied and express statements regarding the progress and timing of the clinical development of the programs across our portfolio, including the expected therapeutic benefits of our programs, and potential efficacy and tolerability; the timing of clinical data updates across our pipeline, including the progress of doublet and triplet combinations for RLY-2608, the timing of clinical updates for RLY-2608, and the timing of a clinical data and regulatory update for lirafugratinib; the timing of clinical initiation of our various programs, including a potential pivotal trial for RLY-2608, clinical development in vascular malformations, clinical development of our non-inhibitory chaperone for Fabry disease, and clinical development of our NRAS-selective inhibitor; the potential of our product candidates to address a major unmet medical need; expectations regarding our pipeline, operating plan, use of capital, expenses and other financial results; our cash runway projection; the competitive landscape and potential market opportunities for our product candidates; the expected strategic benefits under our collaborations; our ability to successfully establish or maintain collaborations or strategic relationships for our product candidates; expectations regarding current and future interactions with the U.S. Food and Drug Administration (FDA); our ability to manufacture our product candidates in conformity with the FDA's requirements; the capabilities and development of our Dynamo™ platform, including its role in identifying product candidates; our plans to develop, manufacture and commercialize our current product candidates and any future product candidates; and the implementation of our business model and strategic plans for our business, current product candidates and any future product candidates. The words "may," "might," "will," "could," "would," "should," "plan," "anticipate," "intend," "believe," "expect," "estimate," "seek," "predict," "future," "project," "potential," "continue," "target" and similar words or expressions, or the negative thereof, are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words.

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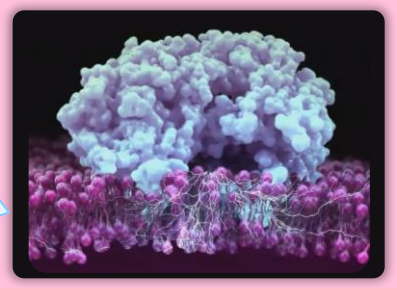
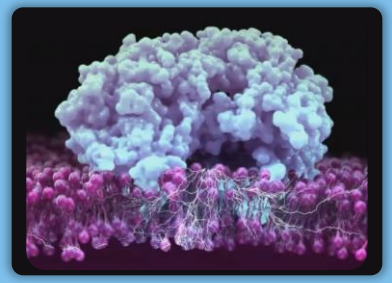
Relay Tx – Today's Updates

	BREAST CANCER	GENETIC DISEASE		SOLID TUMORS
	1 PI3Kα-Driven Breast Cancer	2 PI3Kα-Driven Vascular Malformations	3 Fabry Disease	4 NRAS-Driven Solid tumors
	 Ongoing mono, doublet, triplet; Data update YE24			
Program Updates	1st PI3Kαi + ET + CDK4i combination in clinic (Pfizer)	1st mutant-selective PI3Kα inhibitor	1st non-inhibitory αGal chaperone	1st NRAS-selective inhibitor
Large US opportunity	~150,000 pts[1]	~170,000 pts[2] *(chronic treatment)*	~8,000 pts[3] *(chronic treatment)* $2B current market[5]	~28,000 pts[4]
Milestones	CDK4i clinical start by YE 2024	Clinical start in 1Q 2025	Clinical start in 2H 2025	Clinical start in 2H 2025

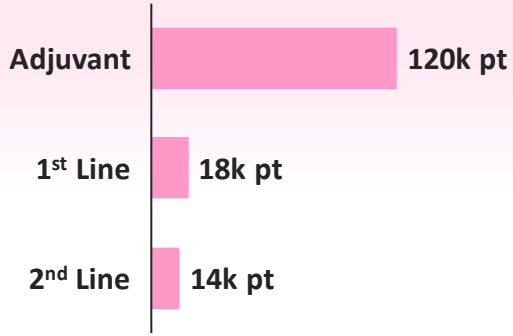
1. Prevalent US patient population with a PIK3CA mutation in adjuvant, first line metastatic and second line metastatic settings (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalence of vascular malformations with a PIK3CA mutation (Gallagher et al 2022 and several other sources); 3. Prevalence of Fabry patients (National Fabry Disease Foundation, Jan 2024); 4. Newly diagnosed (incident) solid tumors with an NRAS mutation, excluding melanoma stages 0-II (SEER, 3rd party source for alteration rate, Jan 2024); 5. Fabry disease forecasted 2024 market size per EvaluatePharma, includes Galafold® and ERTs (May 2024)

PI3Kα – Large Opportunity Across Indications and Therapeutic Areas

PIK3CA mutant HR+/HER2- Breast Cancer

~150k Patients
(US prevalence)[1]

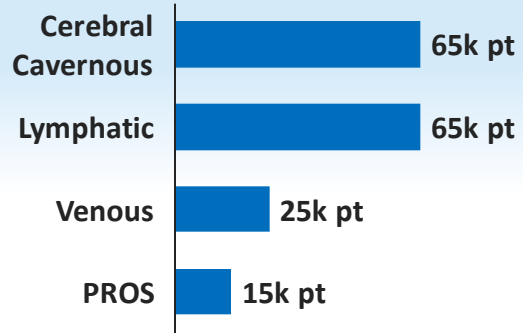
RLY-2608



PIK3CA mutant Vascular Malformations

~170k Patients
(US prevalence)[2]

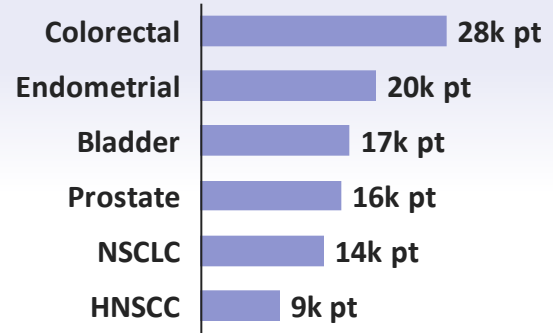
Potential for rapid POC with RLY-2608, then distinct molecule for pivotal



PIK3CA mutant Other Solid Tumors

~160k Patients
(US incidence)[3]

RLY-2608



Relay Tx's PI3Kα Franchise has the potential to address wide range of large disease indications

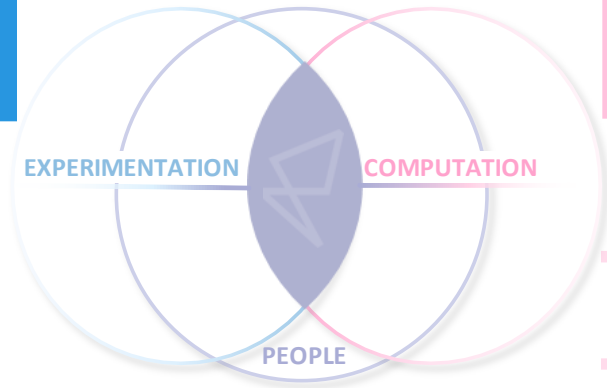
1. Prevalent US patient population with a PIK3CA mutation in each line of therapy (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalent US patient population of vascular malformation patients with a PIK3CA mutation (multiple sources); 3. Incident US patient population solid tumors annually with a PIK3CA mutation (SEER; 3rd party source for alteration rate, May 2024)

Relay Tx – Productive Platform

2016 | 2024 | FUTURE EXPECTATIONS

PRODUCTIVE PLATFORM

8 DCs | **4** INDs | **2** Clinical POC



CONTINUED PLATFORM PRODUCTIVITY

+3 New DCs | **+3** New INDs | **+3** New clinical starts

Expected platform production by YE 2025

- ✓ Built computationally enabled platform
- ✓ Solid Tumors
- ✓ Small Molecule Inhibitors & Degraders

- \+ Internalize, integrate & expand platform
- \+ Genetic Disease
- \+ New Modalities: Chaperones

~$750M Cash as of end Q1 2024
Expected to fund current operating plan into 2H 2026

Relay Tx's Dynamo™ – Productive Computationally Enabled Platform

2024 Dynamo™ Platform

EXPERIMENTATION

- NMR
- Mechanistic enzymology
- HDX-MS
- Cryo-EM
- X-ray fragment screening
- REL-DEL
- Structure ensembles
- Integrated pharmacology
- Protein design and engineering
- Automated Ligand ID System (ALIS)
- Ambient temp. X-Ray crystallography
- High throughput automated chemistry

COMPUTATION

- Free energy calculations
- Long time-scale MD
- Giga-scale virtual screening
- Differential dynamics
- Digitally encoded libraries
- ML-DEL + AI models for DEL
- ADME/PK models
- Active learning
- Generative design
- Automated Chemical Design
- Computational fragment merging

PEOPLE

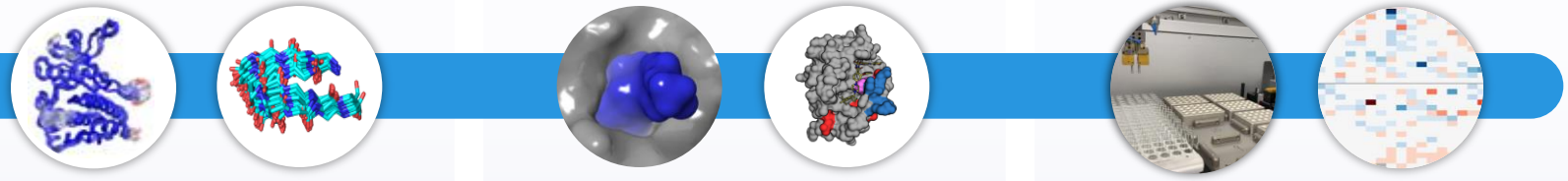
Relay Tx's Dynamo™ Platform – Experimentation Closely Integrated with Computation

Focus of the Dynamo™ Platform

Target Identification | Target Modulation Hypothesis | Hit Identification | Lead Optimization | Development & Commercial

Experimentation

Computation



Develop novel modulation hypotheses

Rapidly generate multiple lead compounds

Optimize lead compounds swiftly and cost-effectively

Dynamo™ Platform Enabled Rapid Creation of First Selective NRAS Inhibitor

Compounds evaluated computationally

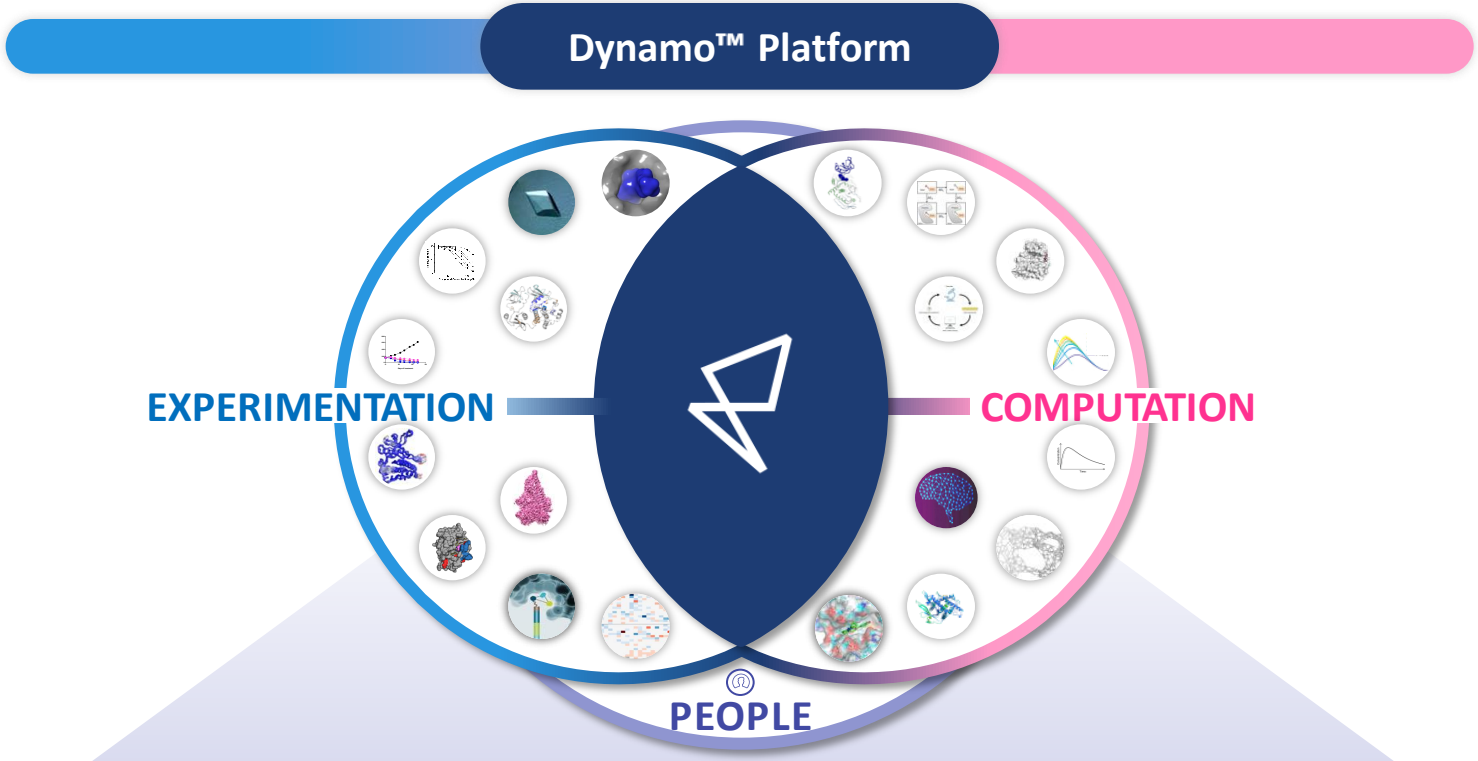
29
compounds synthesized in the lab

1st compound synthesized achieved potent NRAS inhibition

In Vivo PoC

120 days

Relay Tx's Dynamo™ – Focused on Output



Outputs

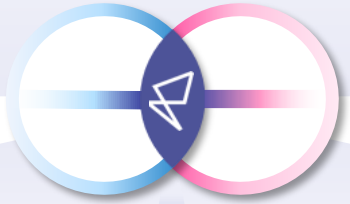
RLY-2608 | RLY-5836 | RLY-2139 | RLY-1013 | Lirafugratinib | Migoprotafib

+ Multiple unnamed earlier stage research programs

Relay Tx – Consistent Focus on Validated, Low Translational Risk Programs

Target Selection Focus

- Genetically Defined
- Clinically Validated
- Unmet Medical Need
- Commercially Attractive
- Amenable to Dynamo™ Platform



BREAST CANCER

- PI3Kα
- CDK2
- ERα

GENETIC DISEASE

- αGal (Fabry Disease)
- PI3Kα (Vascular Malformations)

SOLID TUMORS

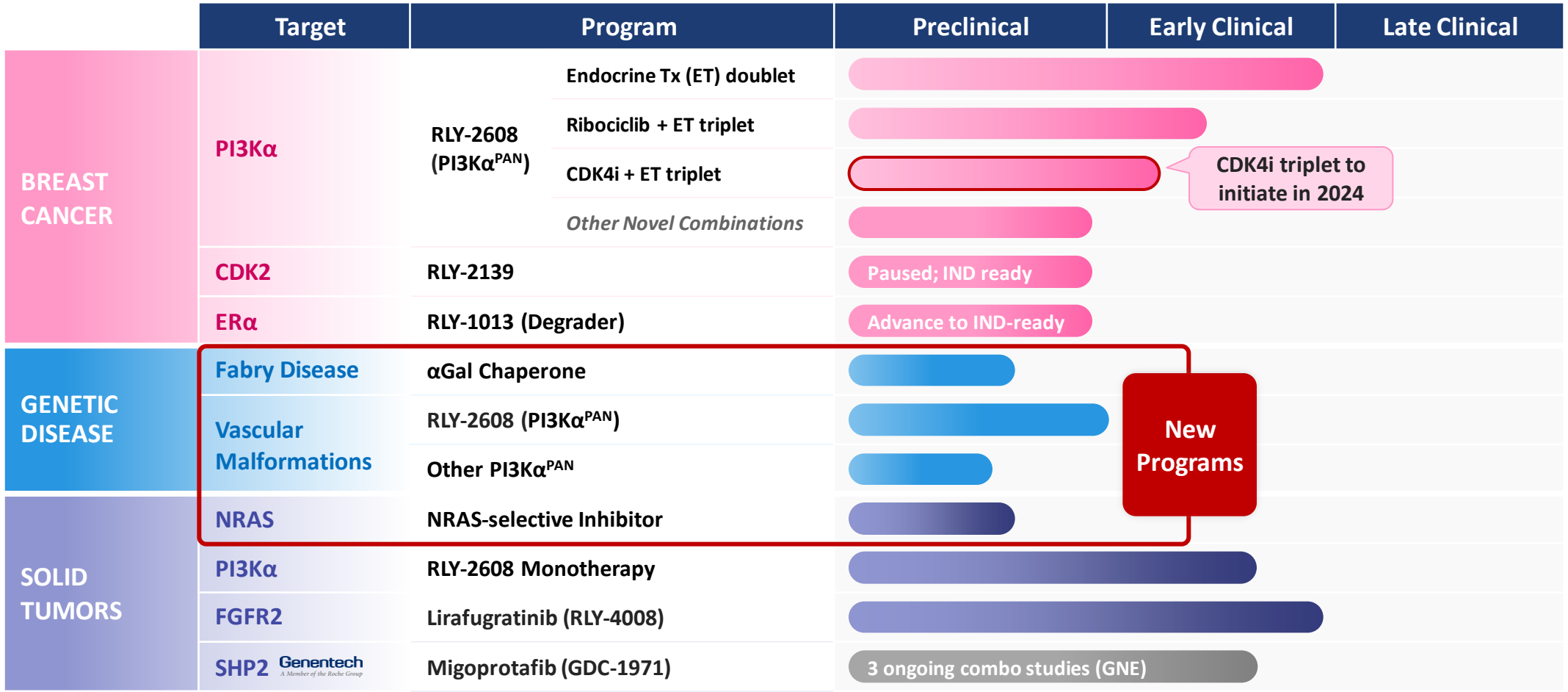
- NRAS
- PI3Kα
- FGFR2
- SHP2

PRODUCTIVE DYNAMO™ RESEARCH ENGINE

Multiple unnamed research stage programs

New Programs

Relay Tx – Broad Precision Medicine Pipeline



5+ additional unnamed research programs

Relay Tx – Milestones 2024-25

BREAST CANCER PORTFOLIO MILESTONES

PI3Kα	RLY-2608	• Data update in 4Q 2024
		  • Doublet safety & efficacy data
		  • Initial triplet data
		• CDK4i triplet clinic start by YE 2024
		• Potential pivotal trial start in 2025

CDK2	RLY-2139	IND-ready
ERα	RLY-1013	• IND-ready in 2025

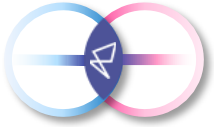
GENETIC DISEASE PORTFOLIO MILESTONES

Fabry	New Program	• Clinical start in 2H 2025
VM	New Program	• Clinical start in 1Q 2025

SOLID TUMORS PORTFOLIO MILESTONES

NRAS	New Program	• Clinical start in 2H 2025
FGFR2	Lirafugratinib	• Tumor agnostic data & regulatory update in 2H 2024
SHP2	Migoprotafib	• Three ongoing combo trials*

** Genentech controls data disclosures*



DYNAMO™ PLATFORM | **5+ unnamed research programs**

~$750M cash as of end Q1 2024
Expected to fund current operating plan into 2H 2026

PI3Kα – Large Opportunity Across Indications and Therapeutic Areas

PIK3CA mutant HR+/HER2- Breast Cancer

~150k Patients
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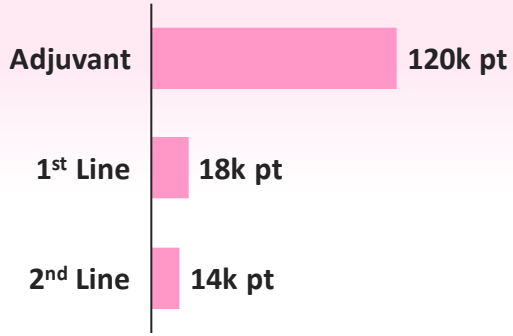
RLY-2608



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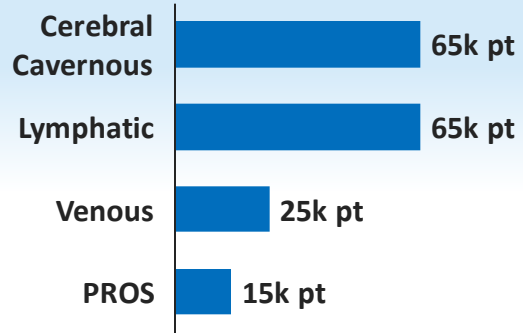

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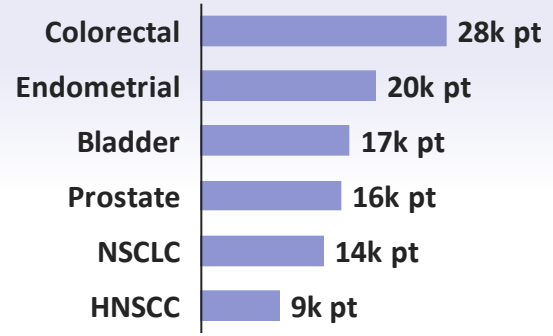


Relay Tx's PI3Kα Franchise has the potential to address wide range of large disease indications

1. Prevalent US patient population with a PIK3CA mutation in each line of therapy (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalent US patient population of vascular malformation patients with a PIK3CA mutation (multiple sources); 3. Incident US patient population solid tumors annually with a PIK3CA mutation (SEER; 3rd party source for alteration rate, May 2024)

Relay Tx – Extensive Breast Cancer Portfolio in Validated Market Expected to Grow to ~$27B by 2030[1]

HR+/HER2- Breast Cancer is a very large patient population…

35% of Breast Cancer Pt with PI3Kα mutation
(14% of all solid tumors)

~150k
US prevalence HR+/HER2- Breast Cancer Patients with PI3Kα mutation[2]

~120k	~18k	~14k
Adjuvant	*1L*	*2L*

…for which Relay Tx's broad next generation ER+/HER2- BC Portfolio is designed to address

Current SoC Tx	→ Evolving SoC	RELAY THERAPEUTICS
PI3Kα Pathway Non-Selective Inhibitors	→ PI3Kα Mutant-specific	RLY-2608 (pan)
ET Backbone: AI / fulvestrant	→ ER Degraders & other SERDs	RLY-1013 (ERα)
CDK4/6 inhibitors	→ CDK4 or CDK2 selective	RLY-2139 (CDK2)[3] Atirmo (CDK4) Pfizer

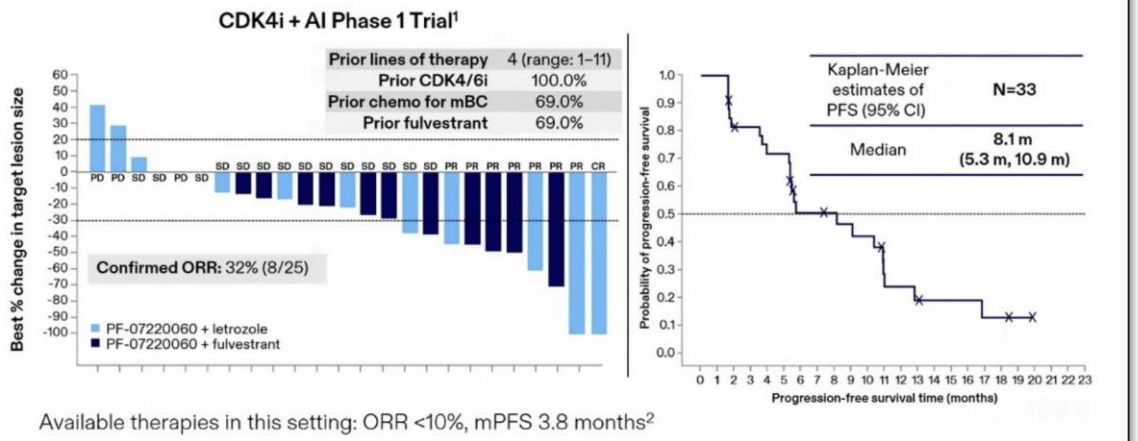
1. Decision Resources Group – Breast Cancer Disease Landscape & Forecast (Nov 2023); 2. Prevalent US patient population with a PIK3CA mutation in each line of therapy (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 3. RLY-2139 is paused and IND-ready

RLY-2608 + Atirmociclib (CDK4i): Pfizer – Relay Tx Clinical Trial Collaboration

Encouraging Efficacy Data in Heavily Pre-Treated Patients

UPDATED DATA

Atirmociclib: Encouraging Efficacy in Heavily Pre-Treated Patients



Available therapies in this setting: ORR <10%, mPFS 3.8 months[2]

ClinicalTrials.gov: NCT04557449.
[1]Data on file source: Ph1 study C4391001, database as of 01 NOV 23.; [2]EMERALD study (*J Clin Oncol* 2022 Oct 1;40(28):3246-3256).
AI, aromatase inhibitor; CDK4/6i, cyclin-dependent kinase 4/6 inhibitor; CI, confidence interval; mBC, metastatic breast cancer; mPFS, median progression-free survival; ORR, objective response rate; PFS, progression-free survival.

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Potentially Differentiated Safety and Tolerability Profile

Atirmociclib: Potentially Differentiated Safety and Tolerability Profile

May Enable More Complete and Continuous Dosing Relative to CDK4/6 Inhibitors

Treatment-Related AEs	Atirmociclib + FUL[1] (N=36)		Palbociclib + FUL[2,3,4] (N=345)		Ribociclib + FUL[5,6] (N=483)		Abemaciclib + FUL[7,8] (N=446)	
	All Grades %	Grade ≥3 %	All Grades %	Grade ≥3 %	All Grades %	Grade ≥3 %	All Grades %	Grade ≥3 %
Neutropenia	36	11	83	66	69	53	46	27
Diarrhea	19	0	24	0	29	<1	86	13
Dose reductions due to AE	8		34		33		43	
Drug discontinuation due to AE	3		4		9		16	

No head-to-head trials between these medicines. Caution is advised when comparing results of different clinical studies

[1]Data on file (Ph1 study C4391001, database as of 01 NOV 23.). Pfizer Inc., New York, NY. [2]Turner NC, et al. SABCS 2016. [3]IBRANCE [prescribing information]. New York, NY: Pfizer Inc.; 2023. [4]Cristofanilli M, et al. *Lancet Oncol.* 2016. [5]Slamon DJ, et al. *J Clin Oncol.* 2018. [6]KISQALI [prescribing information]. East Hanover, NJ: Novartis Pharmaceuticals Corporation; 2023. [7]Sledge GW, et al. *J Clin Oncol.* 2017. [8]VERZENIO [prescribing information]. Indianapolis, IN: Lilly USA, LLC; 2023.
AE, adverse event; CDK4/6, cyclin dependent kinase 4/6; FUL, fulvestrant.

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Source: Pfizer Oncology Innovation Day presentation, Feb 2024

RLY-2608 – Broad Development Program: Doublet

ReDiscover Trial

	Dose Escalation	Dose Expansion	
RLY-2608 + Fulvestrant Doublet	Completed *(Started Apr 2022)* 600mg BID (N=17) 400mg BID (N=10)	20 pt enrolled before YE 2023 600mg BID (n=60)	Enrolling
		400mg BID (n=20)	Enrolling
RLY-2608 + Fulvestrant + CDKi Triplet	Initiated 2024		Enrolling
Mono	Ongoing		Enrolling

Data Expectations for YE2024 Disclosure

Doublet

	Safety Evaluable	6+ mo Follow-up
600mg BID	>60 pts	>40 pts
All Doses	>100 pts	

Landmark PFS analysis

Triplet:
Early safety and tolerability data

RLY-2608 – Broad Development Program: Triplet

ReDiscover Trial

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		400mg BID (n=20)	Enrolling
RLY-2608 + Fulvestrant + CDKi Triplet	Initiated 2024	Ribo (CDK4/6)	Enrolling
		Atirmo (CDK4)	Clinical start in 2024
Mono	Ongoing		Enrolling

Data Expectations for YE2024 Disclosure

Doublet	Safety Evaluable	6+ mo Follow-up
600mg BID	>60 pts	>40 pts
All Doses	>100 pts	Landmark PFS analysis

Triplet:
Early safety and tolerability data

RLY-2608 – Broad Development Program: Mono

ReDiscover Trial

	Dose Escalation	Dose Expansion	
RLY-2608 + Fulvestrant Doublet	Completed *(Started Apr 2022)*: 600mg BID (N=17); 400mg BID (N=10)	20 pt enrolled before YE 2023: 600mg BID (n=60)	Enrolling
		400mg BID (n=20)	Enrolling
RLY-2608 + Fulvestrant + CDKi Triplet	Initiated 2024	Ribo (CDK4/6)	Enrolling
		Atirmo (CDK4)	Clinical start in 2024
Mono	Ongoing	Enrolling	PRs seen in multiple tumor types*

Data Expectations for YE2024 Disclosure

Doublet	Safety Evaluable	6+ mo Follow-up
600mg BID	>60 pts	>40 pts
All Doses	>100 pts	Landmark PFS analysis

Triplet:
Early safety and tolerability data

* PRs include both confirmed and unconfirmed partial responses

RLY-2608 – Broad Development Program

ReDiscover Trial

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Data Expectations for YE2024 Disclosure

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All Doses	>100 pts	

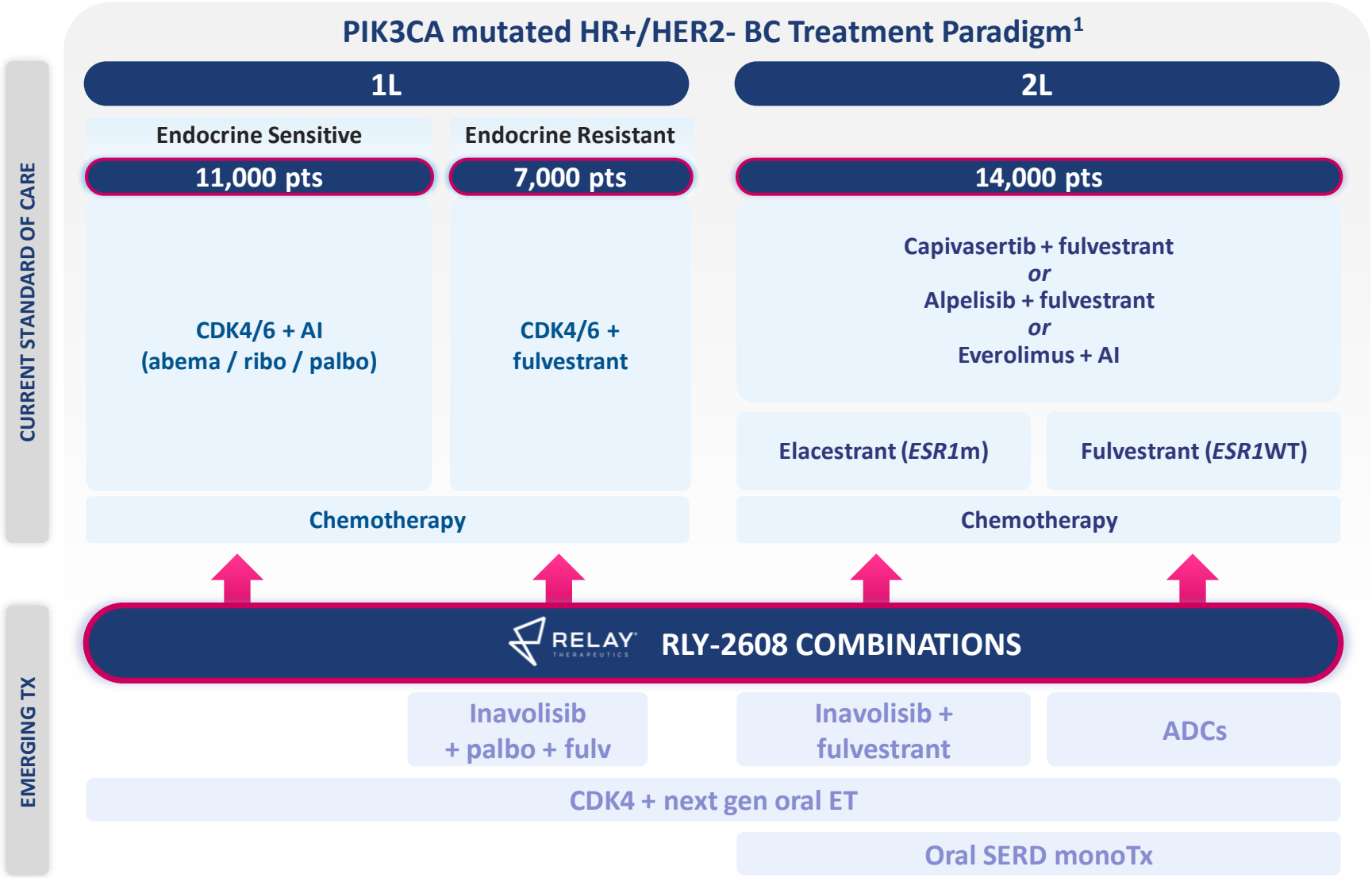
Landmark PFS analysis

Triplet:
Early safety and tolerability data

ReDiscover trial continues broad enrollment across ~25 sites in ~5 countries

* PRs include both confirmed and unconfirmed partial responses

Breast Cancer – Large Market in Current and Emerging Standards of Care



$6B+

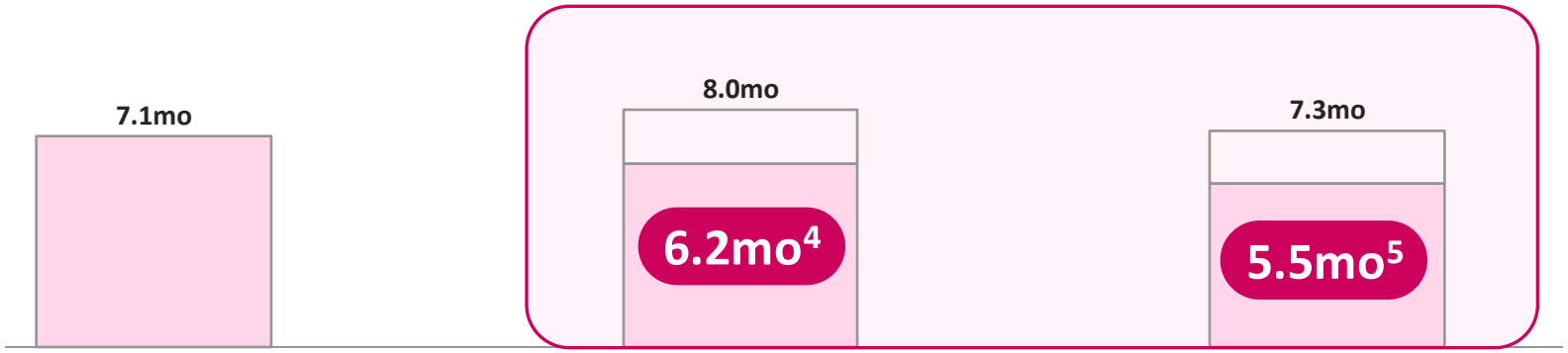
Current PI3Kα Pathway Total Addressable Market[2]

(Metastatic HR+/HER2- Breast Cancer)

1. Prevalent US patient population with a PIK3CA mutation in each line of therapy (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Relay Tx PIK3CA internal market forecast (patient-based – US, EU5, Japan). Forecast includes estimates for genetic testing, class share, market access, compliance, duration of therapy and assumes current PIK3CA therapy net price (primary sources: SEER; GloboCan; Global Data; Evaluate Pharma; DRG Market Forecast; PIK3CAi PIs)

RLY-2608 – 2L PFS Pivotal Benchmark: ~6 Months

	Doublet Combination Regimens		
	Inavolisib + fulvestrant	Alpelisib + fulvestrant	Capivasertib + fulvestrant
FDA Approval	Not approved	Approved 2019	Approved 2023
Data Benchmark	Ph1b Arm D[1]	BYLieve[2]	CAPItello-291[3]
N	60	127	355
% prior fulv	47%	0%	0%
mPFS	7.1mo	6.2mo[4]	5.5mo[5]
CBR	48%	46%	56%
ORR	19%	19%	29%



Capi ORR & CBR include 30% of pts who are CDK4/6-naïve

1. SABCS 2021 #P5-17-05; 2. Rugo 2021 Lancet Oncol 22:489, ASCO 2023 1078; 3. Turner N Engl J Med 2023; 388:2058-2070; 4. Based on 4.0-6.2mo mPFS reported in Novartis-sponsored real-world evidence study for alpelisib + fulvestrant (ASCO 2022 #1055); 5. 5.5mo mPFS reported in CDK4/6-experienced patient sub-population of CAPItello-291
Note: These data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

Relay Tx – Milestones 2024-25

BREAST CANCER PORTFOLIO MILESTONES

PI3Kα	RLY-2608	• Data update in 4Q 2024
		  • Doublet safety & efficacy data
		  • Initial triplet data
		• CDK4i triplet clinic start by YE 2024
		• Potential pivotal trial start in 2025

CDK2	RLY-2139	IND-ready
ERα	RLY-1013	• IND-ready in 2025

GENETIC DISEASE PORTFOLIO MILESTONES

Fabry	New Program	• Clinical start in 2H 2025
VM	New Program	• Clinical start in 1Q 2025

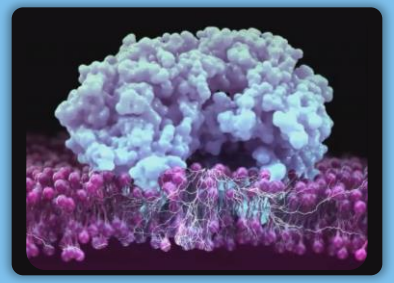
SOLID TUMORS PORTFOLIO MILESTONES

NRAS	New Program	• Clinical start in 2H 2025
FGFR2	Lirafugratinib	• Tumor agnostic data & regulatory update in 2H 2024
SHP2	Migoprotafib	• Three ongoing combo trials*

** Genentech controls data disclosures*

DYNAMO™ PLATFORM | 5+ unnamed research programs

Relay Tx – Today's Updates

	BREAST CANCER	GENETIC DISEASE		SOLID TUMORS
	1 PI3Kα-Driven Breast Cancer	2 PI3Kα-Driven Vascular Malformations	3 Fabry Disease	4 NRAS-Driven Solid tumors
				
Program Updates	1st PI3Kαi + ET + CDK4i combination in clinic	1st mutant-selective PI3Kα inhibitor	1st non-inhibitory αGal chaperone	1st NRAS-selective inhibitor
Large US opportunity	~150,000 pts[1]	~170,000 pts[2] *(chronic treatment)*	~8,000 pts[3] *(chronic treatment)*	~28,000 pts[4]
Milestones	CDK4i clinical start by YE 2024	Clinical start in 1Q 2025	Clinical start in 2H 2025	Clinical start in 2H 2025

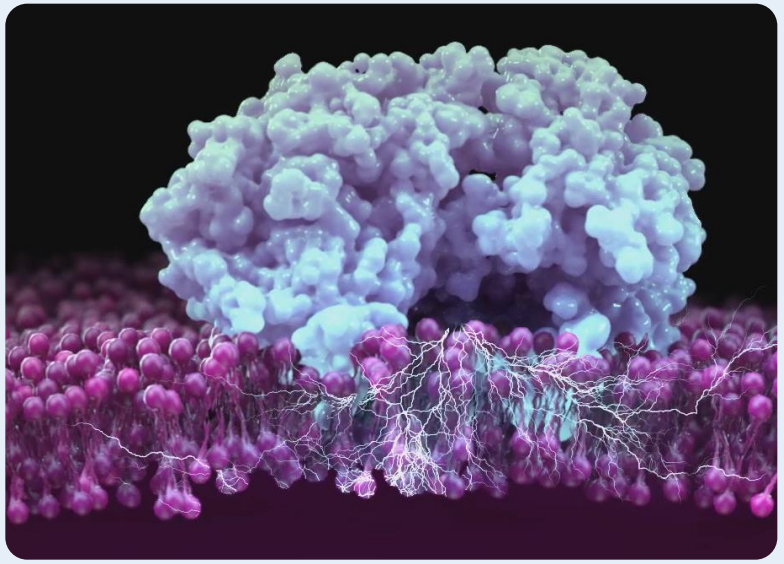
1. Prevalent US patient population with a PIK3CA mutation in adjuvant, first line metastatic and second line metastatic settings (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalence of vascular malformations with a PIK3CA mutation (Gallagher et al 2022 and several other sources); 3. Prevalence of Fabry patients (National Fabry Disease Foundation, Jan 2024); 4. Newly diagnosed (incident) solid tumors with an NRAS mutation, excluding melanoma stages 0-II (SEER, 3rd party source for alteration rate, Jan 2024)

PI3Kα-Driven Vascular Malformations – Significant Unmet Need

	GENETIC DISEASE PI3Kα-Driven Vascular Malformations
Novel Approach	1st mutant-selective PI3Kα inhibitor
Genetically Defined	PIK3CAmut
Clinically Validated	Vijoice® (alpelisib) approved
Unmet Medical Need	• Limited efficacy • Lack of selectivity • Approved Tx for PROS only
Commercially Attractive	~170,000 patients[1] *(chronic treatment)*

DYNAMO™ PLATFORM

First Mutant Selective Inhibitor



1. Prevalence of Vascular Malformations with a PIK3CA mutation (sources: Keppler-Noreuil. Am J Med Genet A. 2015; Engel-Nitz. JVA. 2022; Rodriguez-Laguna; OJRD. 2022; Vogel. Ped Derm. 2013; Shah. J Maxillofac Oral Surg. 2010; Poget. Ped Surg Int. 2023; Behravesh; CDT. 2016; Peyre. NEJM. 2021; Fereydooni et al 2019; Penington et al 2023; Gallagher et al 2022; Luks et al 2015; Limaye et al 2015; Stor et al 2023; Broek et al 2019; Choquet et al 2015; Venot et al. 2018; Pagliazzi et al 2021)

Dr. Adrienne Hammill, MD PhD

- **PhD in Cell Regulation**
  - "Asymmetric cell division results in differential apoptotic cell fates in a B-cell lymphoma model of tumor dormancy"
- **Board certified in Pediatrics and Pediatric Hematology-Oncology**
- **Certificate in Clinical and Translational Research**
- **Working in vascular anomalies since 2009**
- **Serving as**
  - **Research Director of the Hemangioma & Vascular Malformations Center (HVMC)**
  - **Director, Cincinnati HHT Center of Excellence**
  - **Director, Cincinnati Sturge-Weber Center of Excellence/Clinical Care Network Center**

Vascular Anomalies

Dr. Adrienne Hammill, MD PhD

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- Anomalies is an umbrella term for many different diagnoses
  - Includes TUMORS "things that grow" and MALFORMATIONS "present since birth"
  - Distinction less clear than it used to be, a few "unclassified"
- Vascular Anomalies overall not rare due to high frequency of hemangiomas
  - But many of the individual diagnoses are quite rare

ISSVA classification for vascular anomalies ©

(Approved at the 20th ISSVA Workshop, Melbourne, April 2014, last revision May 2018)

This classification is intended to evolve as our understanding of the biology and genetics of vascular malformations and tumors continues to grow

Vascular anomalies				
Vascular tumors	Vascular malformations			
	Simple	Combined °	of major named vessels	associated with other anomalies
Benign Locally aggressive or borderline Malignant	Capillary malformations Lymphatic malformations Venous malformations Arteriovenous malformations* Arteriovenous fistula*	CVM, CLM LVM, CLVM CAVM* CLAVM* others	See details	See list

Vascular Malformations

Dr. Adrienne Hammill, MD PhD

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- **Vascular malformations can include a single type of malformed vessel, or combinations of vessels**

ISSVA classification for vascular anomalies ©

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Vascular malformations			
Simple	**Combined °**	**of major named vessels**	**associated with other anomalies**
Capillary malformations Lymphatic malformations Venous malformations Arteriovenous malformations* Arteriovenous fistula*	CVM, CLM LVM, CLVM CAVM* CLAVM* others	See details	See list

Vascular Malformations

Dr. Adrienne Hammill, MD PhD

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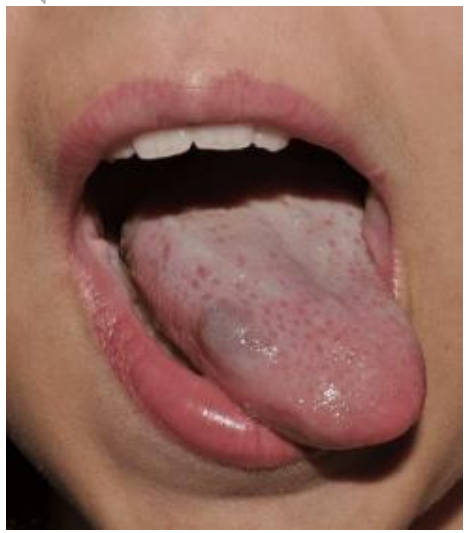
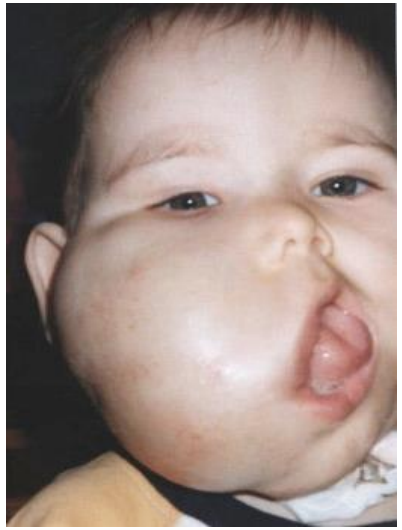
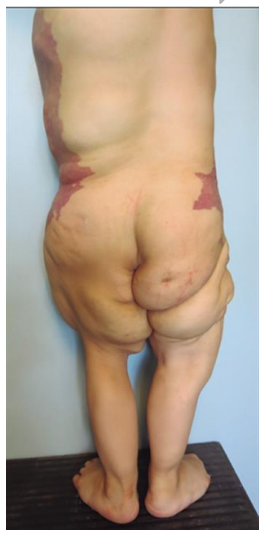
- **Vascular malformations can be localized ("isolated"), diffuse/multifocal, or part of a syndrome with other findings**
  - **Most frequent syndromic association is overgrowth, particularly in "combined vascular malformations"**

A range of severity in Vascular Anomalies







Isolated

Diffuse, Syndromic

Sources: Colmenero 2021; Davidson et al, 2015; de Grazia et al

Genetic Causes of Vascular Malformations – Germline Mutations

Dr. Adrienne Hammill, MD PhD

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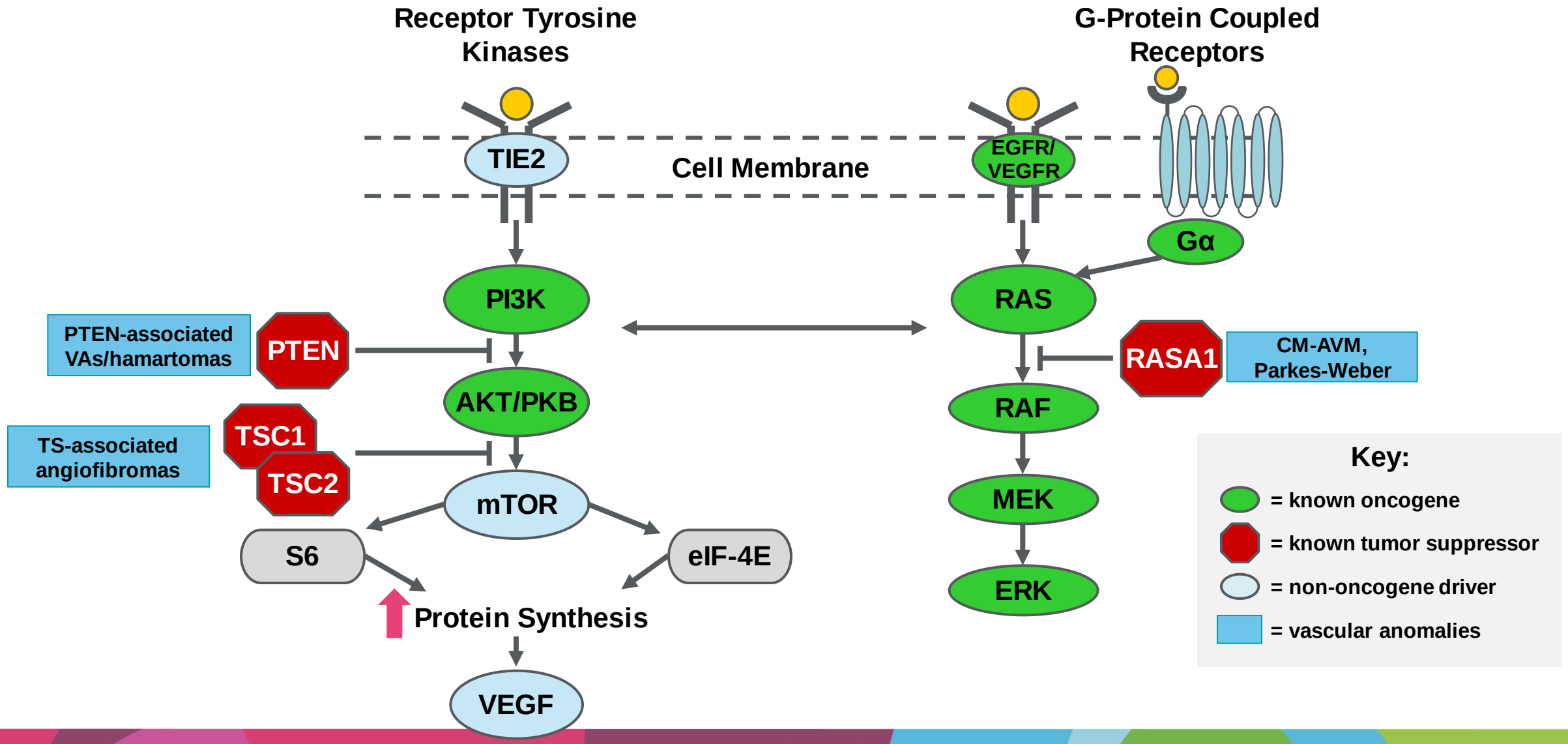

Genetic Causes of Vascular Malformations – Somatic Mutations

Dr. Adrienne Hammill, MD PhD

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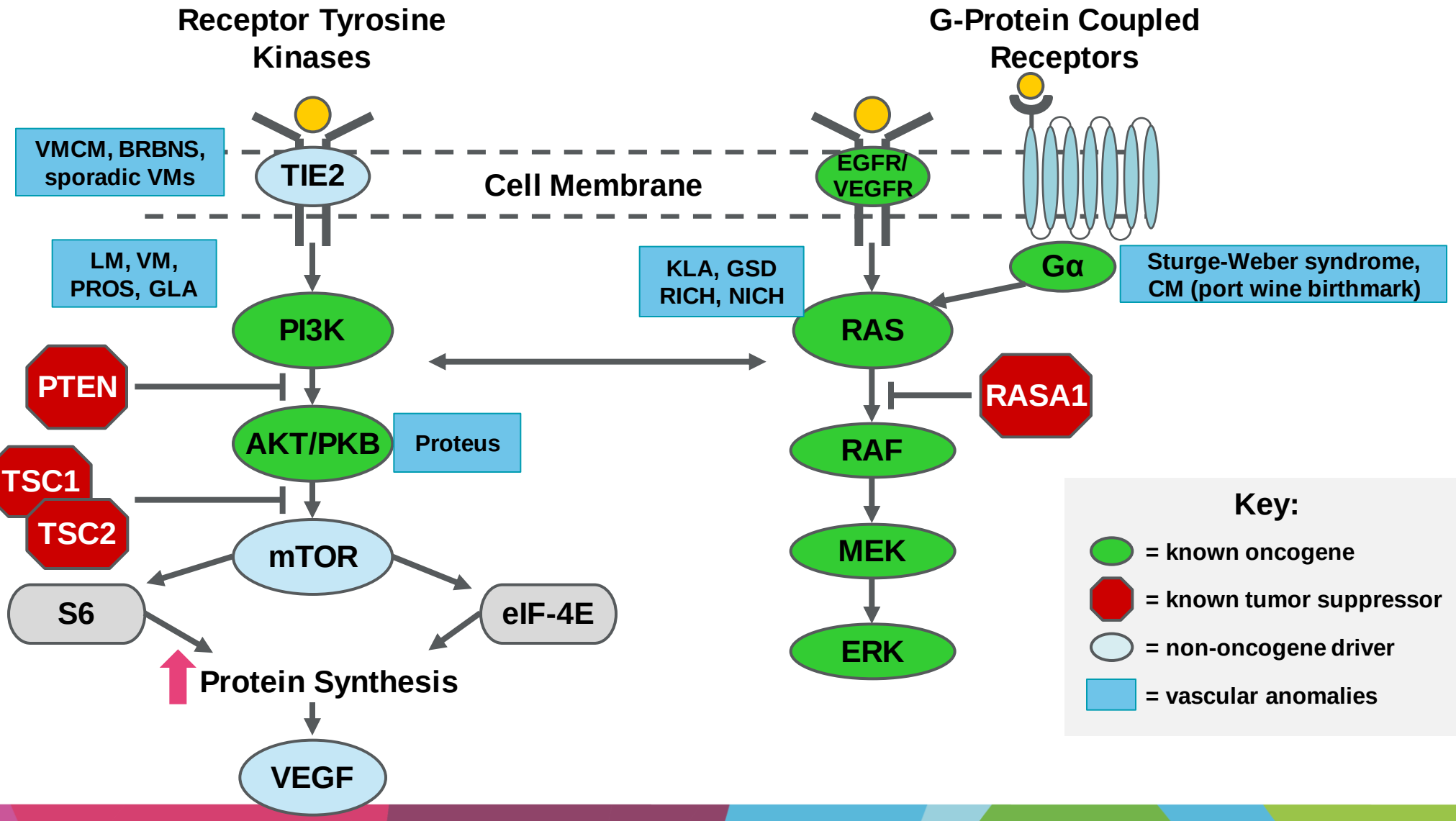

Genetic Causes of Vascular Malformations

Dr. Adrienne Hammill, MD PhD

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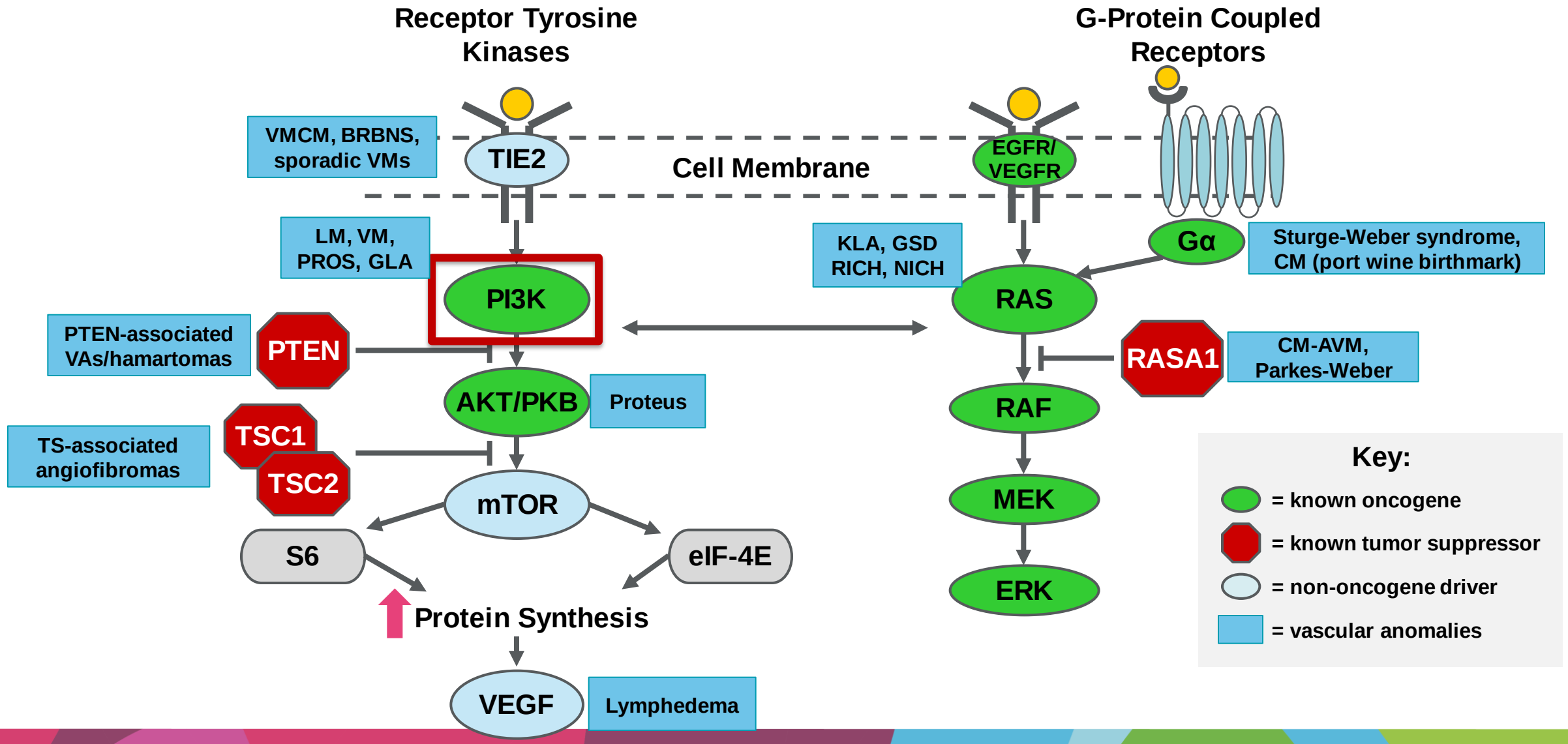

PIK3CA-related Overgrowth Spectrum (PROS) Phenotypes

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- **Megalencephaly-capillary malformation (MCAP) syndrome**
- **Dysplastic megalencephaly (DMEG), hemimegalencephaly (HMEG) and focal cortical dysplasia (FCD)**
- **Congenital lipomatous overgrowth, vascular malformations, epidermal nevi, scoliosis/skeletal and spinal (CLOVES) syndrome**
- **Klippel-Trenaunay syndrome (KTS)**
- **Capillary malformation of the lower lip, Lymphatic malformation of the face and neck, Asymmetry of face and limbs, Partial/generalized Overgrowth (CLAPO) syndrome**
- **Fibroadipose hyperplasia or overgrowth (FAO)**
- **Hemihyperplasia multiple lipomatosis (HHML)**
- **Facial infiltrating lipomatosis (FIL)**
- **Macrodactyly**

- **Isolated tissue dysplasia/overgrowth phenotypes: lymphatic malformations, venous malformations, lipomatosis**

Sources: Mirzaa G et al. PIK3CA-Related Overgrowth Spectrum. 2013 Aug 15 [Updated 2023 Apr 6]. In: GeneReviews® [Internet]

PI3Kα-Driven Vascular Malformations – Overview of Biology

Dr. Adrienne Hammill, MD PhD

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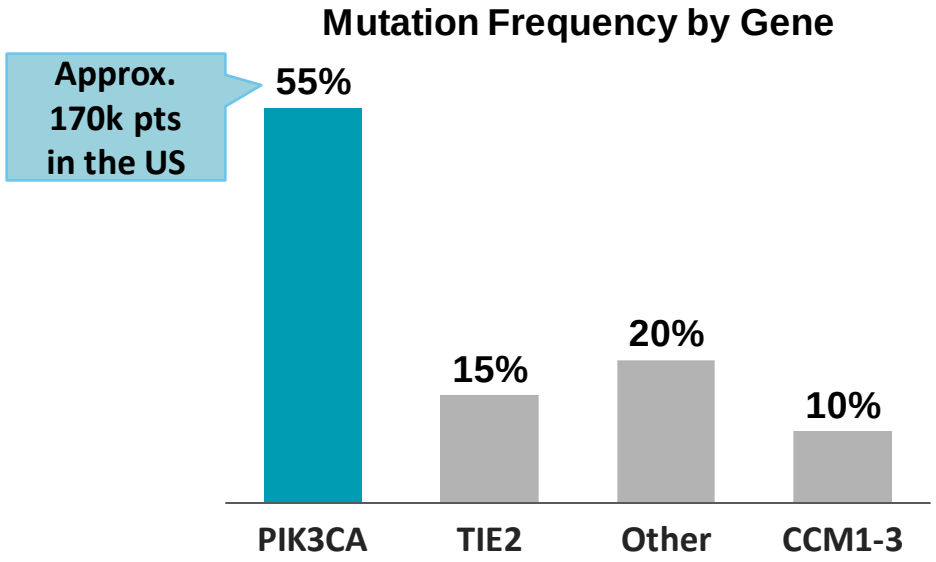
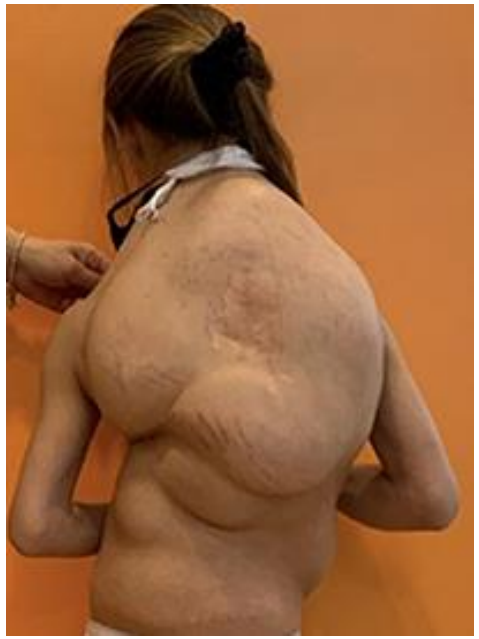
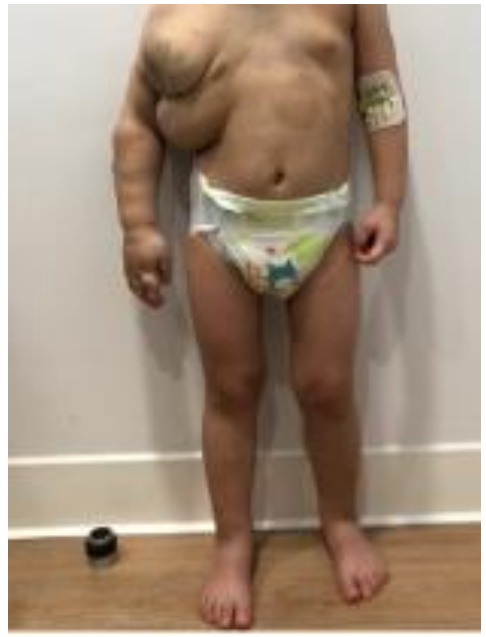
~300k US patients affected by Vascular Malformations, driven by prenatal somatic mutations



Abnormal development of lymphatic and/or blood vessels leads to a wide range of symptoms





Malformations may involve one or more types of vasculature

Sources: Fereydooni et al 2019, Penington et al 2023, Gallagher et al 2022, Luks et al 2015, Limaye et al 2015, Stor et al 2023, Broek et al 2019, Choquet et al 2015, Venot et al. 2018, Pagliazzi et al 2021; Photo sources: Delestre et al 2021, Pagliazzi et al, 2021
Note: TIE2 gene also refers to *TEK* gene

PI3Kα-Driven Vascular Malformations – Patient Treatment Journey

Dr. Adrienne Hammill, MD PhD

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Referral Pathway

Symptom Presentation	PCP, Dermatologist, Surgeon, ENT, etc.
Diagnosis	Geneticist, "Vascular Anomalist"
Treatment	Surgeon, Int. Radiologist, Dermatologist, Heme-Onc

Treatment & Ongoing Management

Frequency of use

Watch and wait; Compression Therapy	Temporary; 50-75% of diagnosed pts receive local or systemic Tx
Local Treatment: Laser, sclerotherapy, surgery	Invasive, recurrence is common (~25-40% recurrence rate)
Systemic Therapy: alpelisib, sirolimus	Incomplete responses, side effects / toxicities limit widespread use

Current unmet need for selective, systemic therapy for Vascular Malformations

Source: primary research

PI3Kα-Driven Vascular Malformations – Over 170,000 US Patients

Dr. Adrienne Hammill, MD PhD

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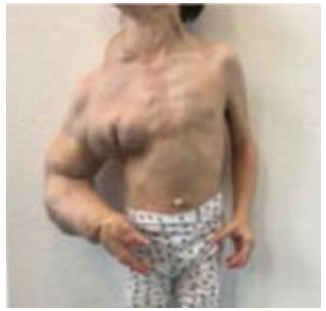
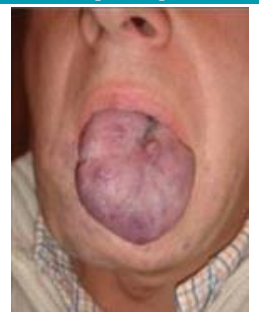
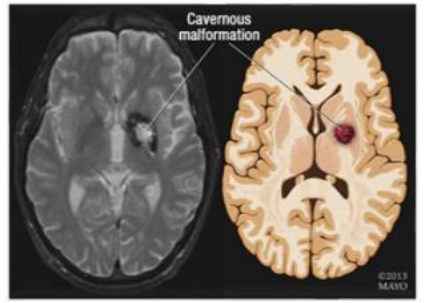
Vascular Malformation Types

	PIK3CA-Related Overgrowth Spectrum (PROS)	Lymphatic Malformation (LM)	Venous Malformation (VM)	Cerebral Cavernous Malformation (CCM)	Total US pt across types
					
US Patients	**~5-15k**	**~80k**	**~100k**	**~120k**	**>300k pt**
% *PIK3CA*mut	**100%** *~5-15k pt*	**80%** *~65k pt*	**~20-25%** *~20-25k pt*	**40-55%** *~50-65k pt*	**~170k pt** ***PIK3CA*mut**
Approved Therapies	**Vijoice® (alpelisib)**	**No approved systemic therapy**			

Sources: ISSVA classification, NORD, Mayo Clinic, Novartis, Penington et al 2023, Gallagher et al 2022, Luks et al 2015, Limaye et al 2015, Peyre et al 2021, Hong et al 2021.
Photo sources: Venot et al. Nature 2018, Wenger et al Genet Med 2022, Limaye et al Nature Genetics 2008, Mayo Clinic

PI3Kα-Driven Vascular Malformations – Systemic Tx Limited by Non-Selective SoC

***PIK3CA*-Related Overgrowth Spectrum (PROS)**

Lymphatic Malformations (LM)

Venous Malformations (VM)

Cerebral Cavernous Malformations (CCM)

Alpelisib

- Limited Efficacy - 27% ORR[1]
- Non-Selective
- Limited scope, approved in PROS only

Sirolimus

- Immunosuppressive
- Non-Selective
- Not approved in any Vascular Malformation types

1. ORR defined as radiologic response ≥20% lesion reduction; Source: FDA label for VIJOICE®

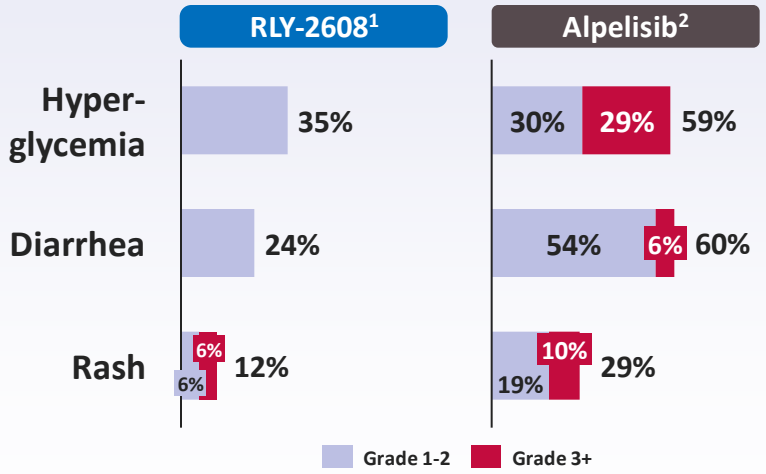
PI3Kα-Driven Vascular Malformations – Relay Tx Mutant Selective Approach

Favorable Selectivity

Selective for mutant PI3Kα

Favorable Tolerability

Fewer key common PI3K class AEs*



Favorable Efficacy

Reduction of mutant *PIK3CA* ctDNA*

***interim data from oncology trials[1-2]**

Potential for rapid POC with RLY-2608, then use a distinct molecule for pivotal studies

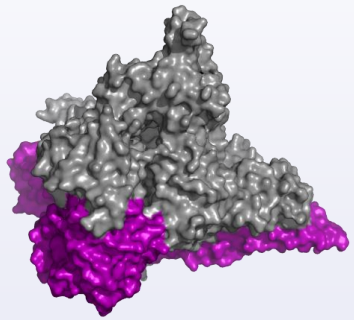
- ***PIK3CA*-Related Overgrowth Spectrum (PROS)**
- **Lymphatic Malformations (LM)**
- **Venous Malformations (VM)**
- **Cerebral Cavernous Malformations (CCM)**

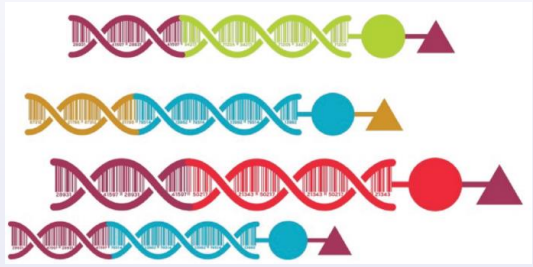
1. Interim AE rates and ctDNA for RLY-2608 part of ongoing ReDiscover trial studying solid tumors (oncology), data as of 07/24/23; 2. Alpelisib AE rates from BYLieve study (oncology), Rugo 2021 Lancet Oncol 22:489

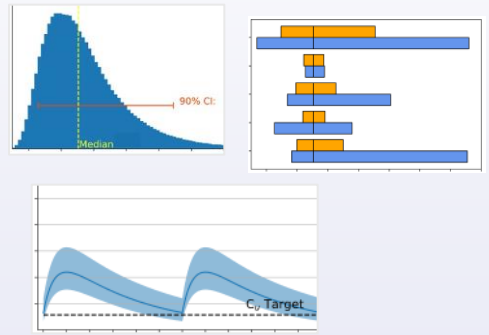
PI3Kα – Dynamo™: Integration of Experimental and Computational Tools

Discovery of 1st mutant-selective PI3Kα inhibitor

Target Modulation Hypothesis	Hit Identification	Lead Optimization
1 Solved 1st full-length structures & novel pocket of PI3Kα	2 Identified early chemical matter for mutant selectivity	3 Rapidly designed the 1st mutant-selective inhibitor of PI3Kα







Platform Tool Examples

- CryoEM & X-ray Crystallography
- Long Time-scale MD
- DNA-Encoded Libraries (DEL)
- Differential Dynamics
- Integrated Pharmacology
- PK / PD Dose Modeling

Experimental tool

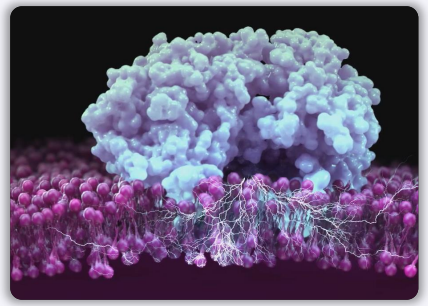
Computational tool

PI3Kα-Driven Vascular Malformations – Significant Unmet Need

	GENETIC DISEASE
	PI3Kα-Driven Vascular Malformations
Novel Approach	1st mutant-selective PI3Kα inhibitor
Genetically Defined	PIK3CAmut
Clinically Validated	Vijoice® (alpelisib) approved
Unmet Medical Need	• Limited efficacy • Lack of selectivity • Approved Tx for PROS only
Commercially Attractive	~170,000 patients[1] *(chronic treatment)*

GENETIC DISEASE PORTFOLIO MILESTONES

Clinical Start in Q1 2025



1. Prevalence of Vascular Malformations with a PIK3CA mutation (Gallagher et al 2022 and several other sources)

Relay Tx – Today's Updates

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			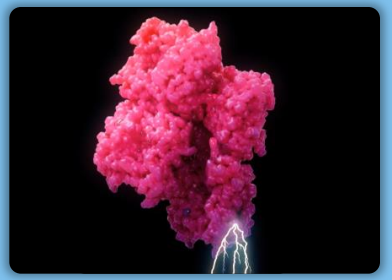	
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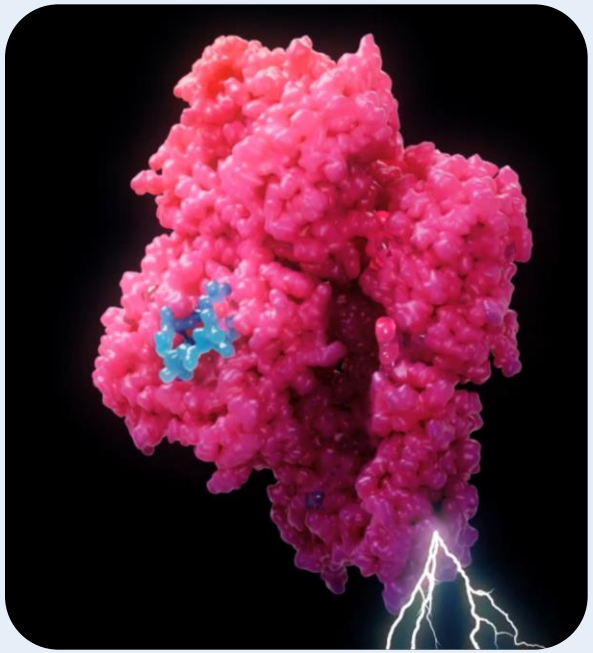
Fabry Disease – Large Validated Market With Significant Unmet Need

	GENETIC DISEASE
	Fabry Disease
Novel Approach	1st non-inhibitory αGal chaperone
Genetically Defined	*GLA* mutations
Clinically Validated	Galafold® (migalastat) approved in Fabry Disease
Unmet Medical Need	Limited αGal activation & limited mutational coverage
Commercially Attractive	~8,000 patients[1] *(chronic treatment)*

DYNAMO™ PLATFORM

First Non-Inhibitory αGal Chaperone



1. Prevalence of Fabry patients (National Fabry Disease Foundation, Jan 2024)

Fabry Disease – Large Validated Market With Significant Unmet Need

Fabry disease is a lysosomal storage disorder affecting ~8,000 patients in US

Over 1,000 different *GLA* gene mutations

Reduces αGal protein levels

Leads to accumulation of toxic Gb3 substrate

Broad clinical manifestations; Life threatening cardiac & renal disfunction

Current therapies have established a market but have key limitations

Current Therapies

Enzyme Replacement Therapy (ERT, intravenous)

~$1.6B peak sales[1]

Inhibitory Chaperone Therapy (migalastat)

40% of pts

~$780M peak sales[2]

Limitations of Inhibitory Chaperone

1. Limited αGal activation
2. Limited mutational coverage
3. Not combined with ERT

Need for a non-inhibitory αGal chaperone

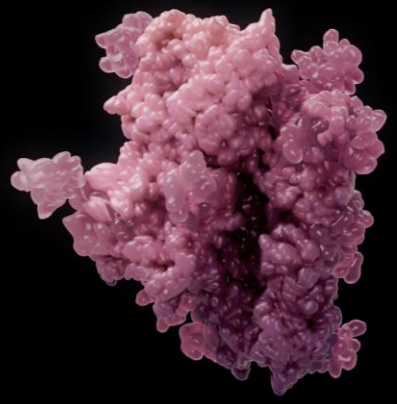
1. Combination of Fabrazyme® (~$1,100M) and Replagal® (~$480M) 2030 forecasted WW sales per EvaluatePharma, April 2024; 2. Galafold® 2030 forecasted WW sales per EvaluatePharma, April 2024

Fabry Disease – Dynamo™ Discovered a Novel Allosteric Pocket

αGal

Normal

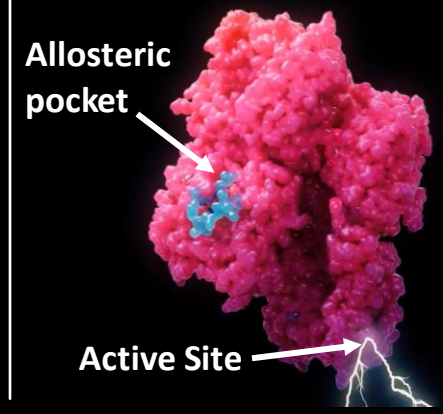
Mutant



Inhibitory vs Non-Inhibitory Chaperone

Inhibitory

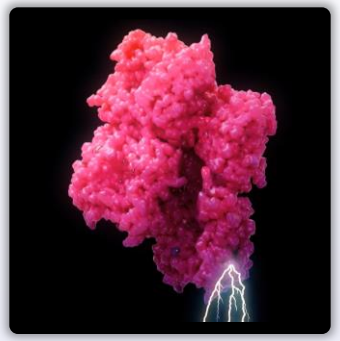
Non-Inhibitory

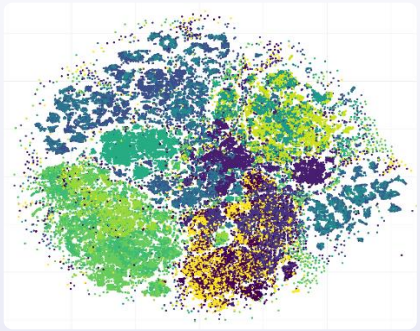


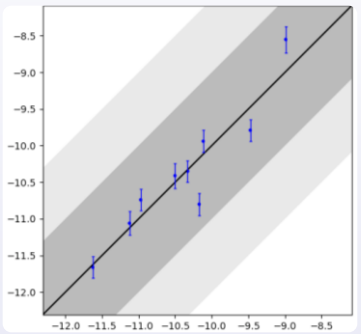
Fabry Disease – Dynamo™: Integration of Experimental and Computational Tools

Discovery of 1st non-inhibitory αGal chaperone

Target Modulation Hypothesis	Hit Identification	Lead Optimization
1 Discovered & validated novel allosteric pocket	2 Identified & validated initial hits that stabilized	3 Achieved potent αGal non-inhibitory chaperones







Platform Tool Examples

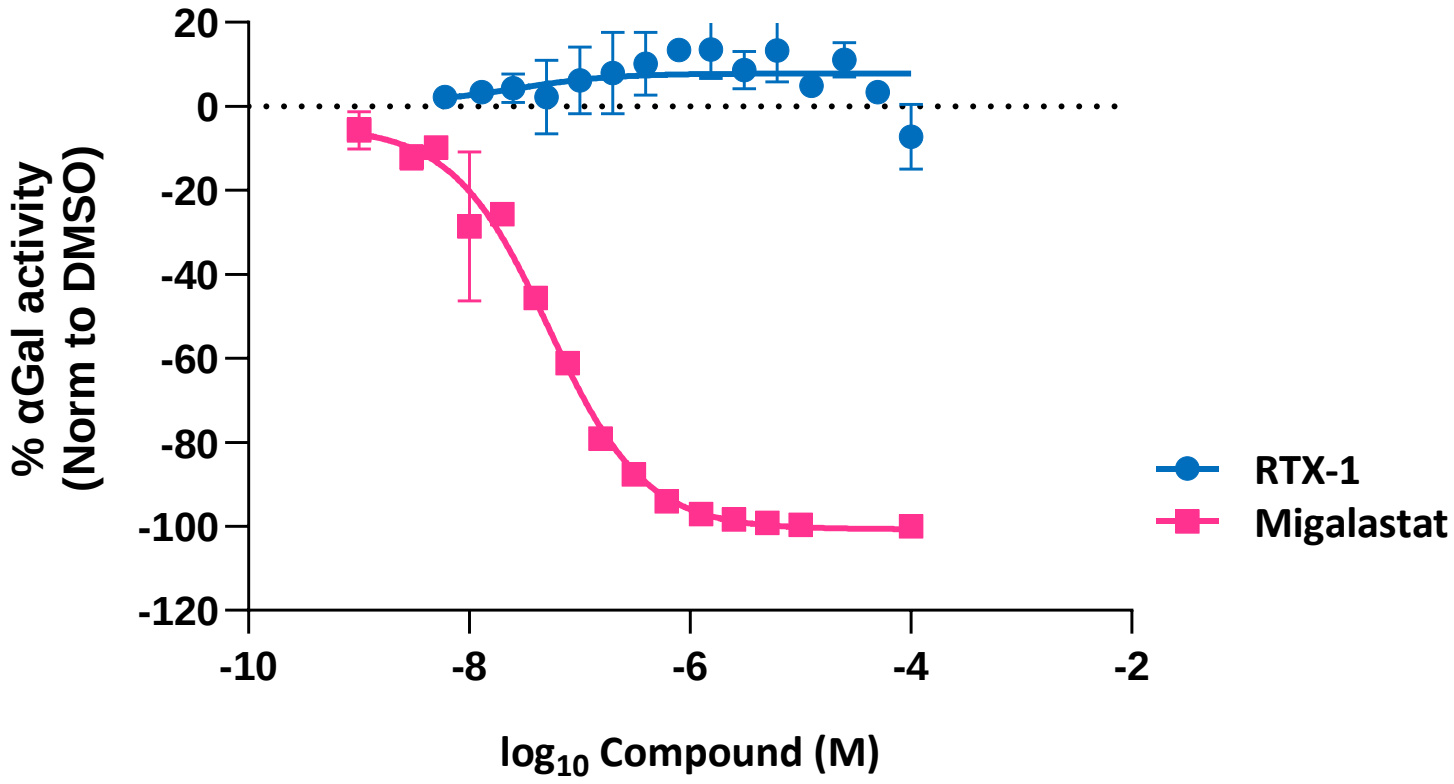
Structure Ensembles	NMR	HTP Automated Chem.
Long Time-Scale MD	Virtual Screening	ADME/PK Models

Experimental tool

Computational tool

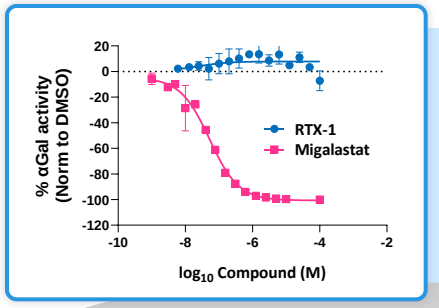
Fabry Disease – Relay Tx Compounds are Non-Inhibitory Chaperones

Migalastat inhibited αGal function while Relay Tx compounds did not



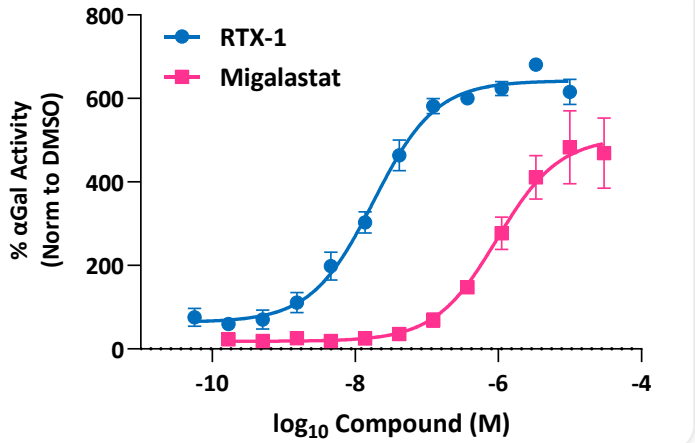
Note: Biochemical assay run with WT αGal; 1 hr incubation at room temperature

Fabry Disease – Potential Benefits of Non-Inhibitory Chaperone Approach

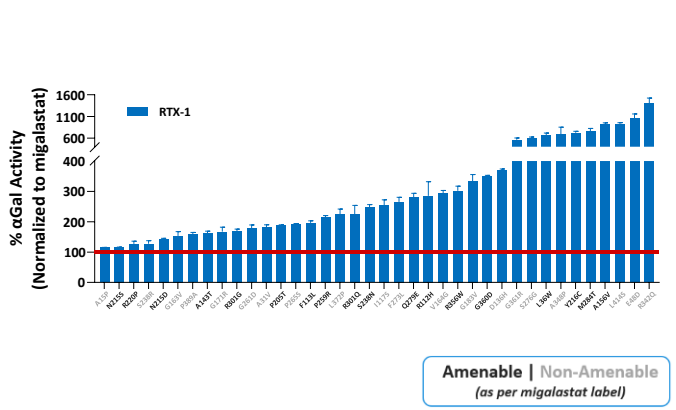


Relay Tx Solution:
Non-Inhibitory Chaperone to Stabilize Protein and Increase Activity

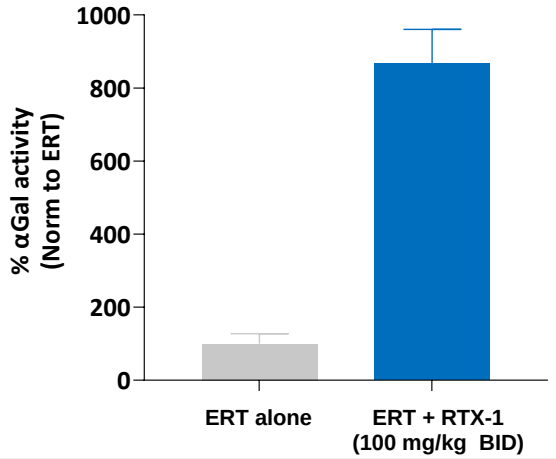
1 Superior αGal activation[1]



2 Broad mutational coverage[2]



3 Combinable with ERT[3]

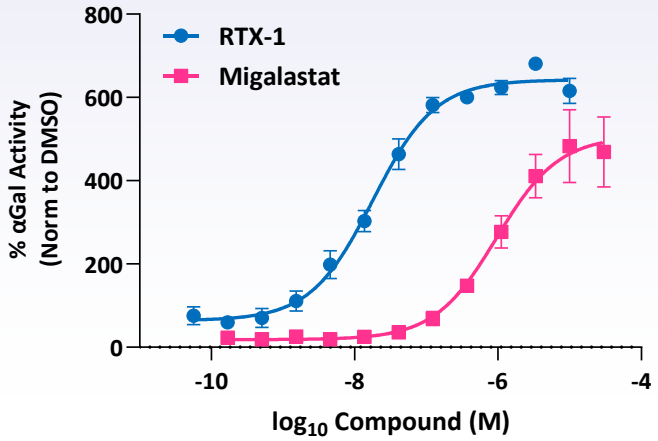


Notes: 1. R301Q mut αGal (2hr post compound washout, expressed in *GLA* KO HEK293 cells); In vitro αGal activity assay (4MU) across multiple GLA mutations expressed in HEK293 GLA KO cells (assessed at 1uM); 3. *GLA* KO mouse model, activity assessed following single dose of ERT and 14-day treatment with RTX-1

Relay Tx Non-Inhibitory Chaperones Can Lead to Higher Levels of *In Vivo* Activity

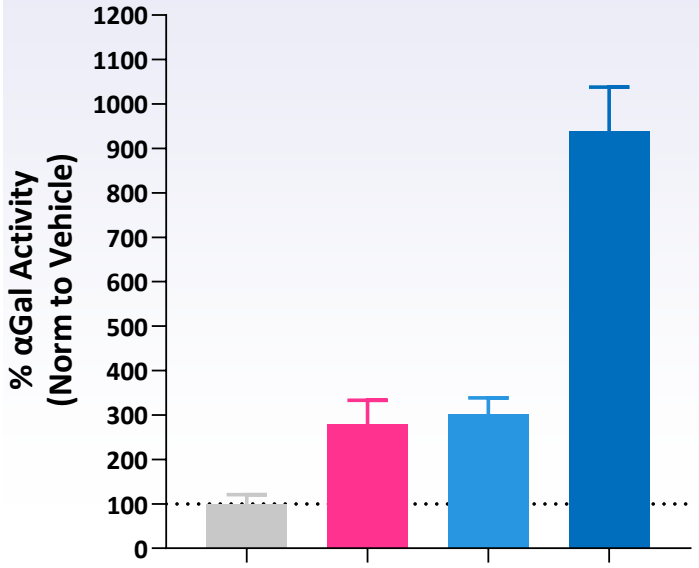
RTX-1 maintains higher levels of αGal activity *in vitro*

R301Q mut αGal
(2hr post compound washout,
expressed in *GLA* KO HEK293 cells)



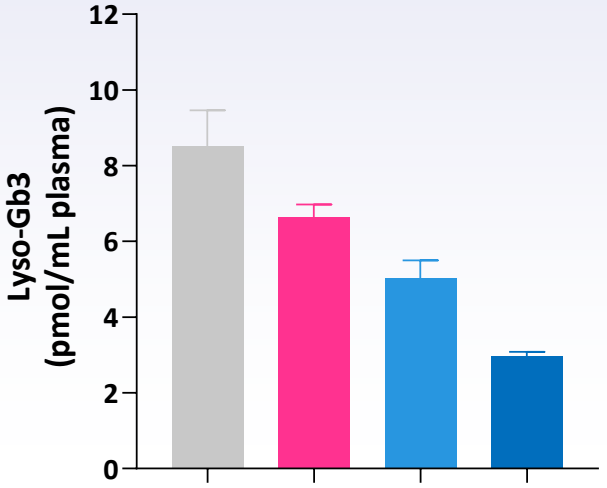
…which translates to greater *in vivo* kidney αGal activity

Activity levels measured at 28 days in humanized *GLA* R301Q mutant mouse model



… and greater substrate reduction

Lyso-Gb3 levels measured at 28 days in humanized *GLA* R301Q mutant mouse model

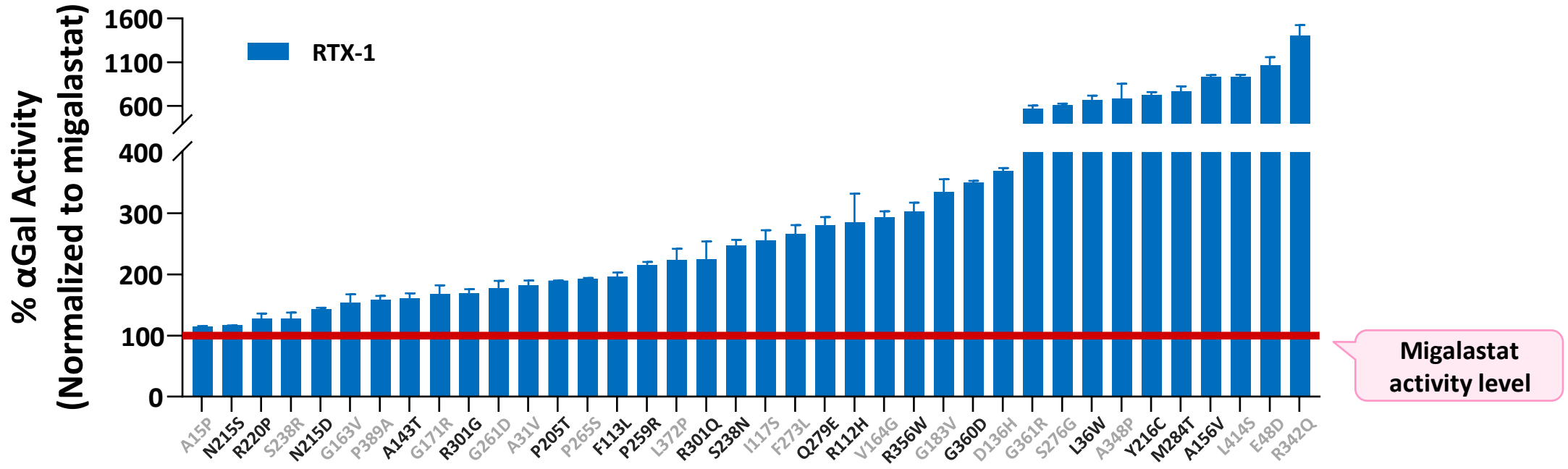


Estimated clinically relevant dose

There were no adverse findings in an exploratory rat toxicology study of RTX-1 at exposures equivalent to 100 mg/kg BID

Relay Tx Non-Inhibitory Chaperones Have Broad Mutational Coverage

In vitro αGal activity assay (4MU) across multiple *GLA* mutations expressed in HEK293 *GLA* KO cells

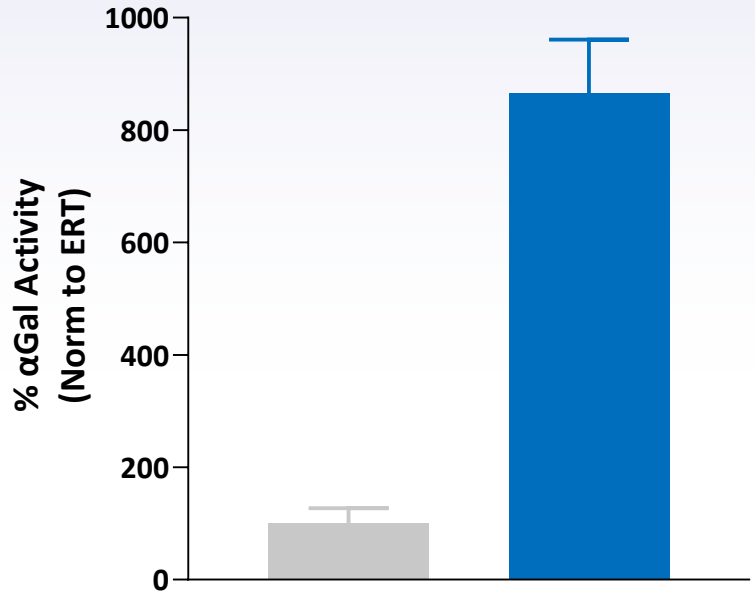


Note: αGal activity evaluated at 1uM of migalastat and RTX-1

Relay Tx Non-Inhibitory Chaperones Combinable with ERT

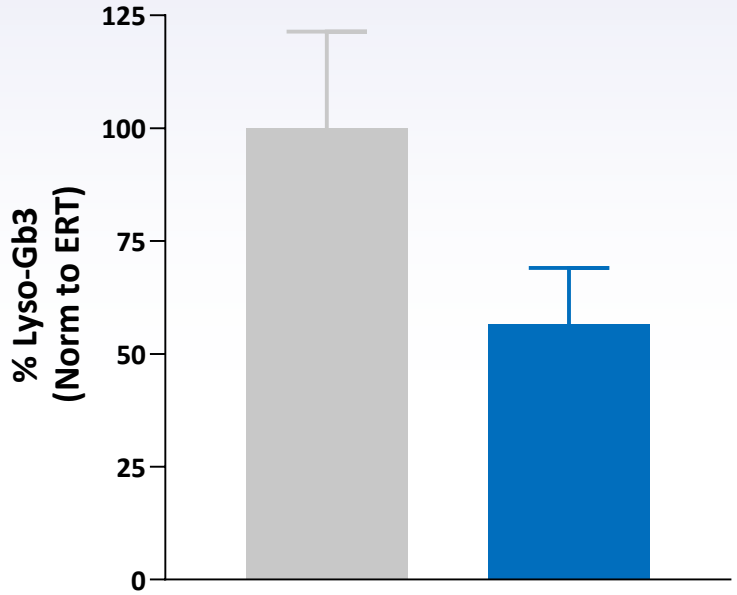
In vivo αGal activity

Activity in kidney following single dose of ERT and 14-day treatment with RTX-1 (*GLA* KO mouse model)



In vivo lyso-Gb3 reduction

Plasma lyso-Gb3 levels following single dose of ERT and 14-day treatment with RTX-1 (*GLA* KO mouse model)



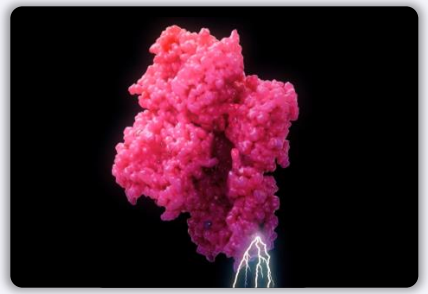
ERT alone ERT + RTX-1 (100 mg/kg BID)

Fabry Disease – Large Validated Market With Significant Unmet Need

	GENETIC DISEASE
	Fabry Disease
Novel Approach	1st non-inhibitory αGal chaperone
Genetically Defined	*GLA* mutations
Clinically Validated	Galafold® (migalastat) approved in Fabry Disease
Unmet Medical Need	Limited αGal activation & limited mutational coverage
Commercially Attractive	~8,000 patients[1] *(chronic treatment)*

GENETIC DISEASE PORTFOLIO MILESTONES

Clinical Start in 2H 2025



1. Prevalence of Fabry patients (National Fabry Disease Foundation, Jan 2024)

Relay Tx – Today's Updates

	BREAST CANCER	GENETIC DISEASE		SOLID TUMORS
	1 PI3Kα-Driven Breast Cancer	2 PI3Kα-Driven Vascular Malformations	3 Fabry Disease	4 NRAS-Driven Solid tumors
				
Program Updates	1st PI3Kαi + ET + CDK4i combination in clinic (Pfizer)	1st mutant-selective PI3Kα inhibitor	1st non-inhibitory αGal chaperone	1st NRAS-selective inhibitor
Large US opportunity	~150,000 pts[1]	~170,000 pts[2] *(chronic treatment)*	~8,000 pts[3] *(chronic treatment)*	~28,000 pts[4]
Milestones	CDK4i clinical start by YE 2024	Clinical start in 1Q 2025	Clinical start in 2H 2025	Clinical start in 2H 2025

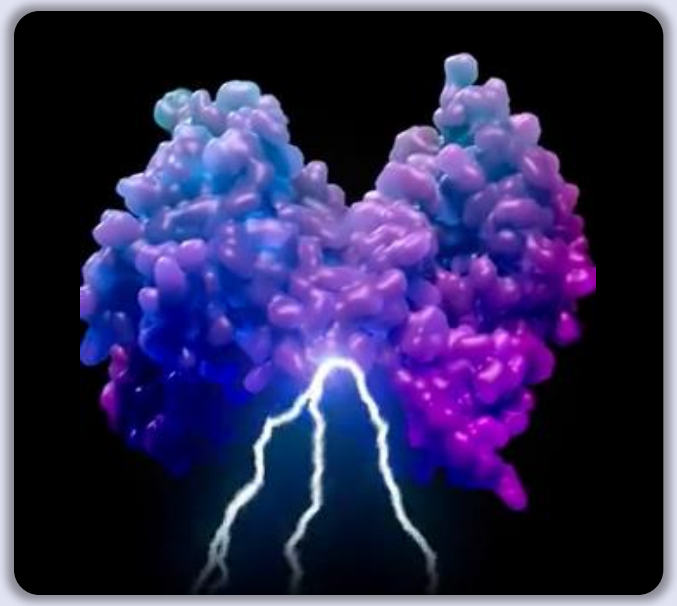
1. Prevalent US patient population with a PIK3CA mutation in adjuvant, first line metastatic and second line metastatic settings (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalence of Vascular Malformations with a PIK3CA mutation (Gallagher et al 2022 and several other sources); 3. Prevalence of Fabry patients (National Fabry Disease Foundation, Jan 2024); 4. Newly diagnosed (incident) solid tumors with an NRAS mutation, excluding melanoma stages 0-II (SEER, 3rd party source for alteration rate, Jan 2024)

NRAS – Large Validated Market With Significant Unmet Need

	Solid Tumors
	NRAS-Driven Solid Tumors
Novel Approach	1st NRAS-selective inhibitor
Genetically Defined	NRASmut
Clinically Validated	Non-selective RAF/MEK/Pan-RAS
Unmet Medical Need	• Lack of selectivity • Challenging AE profile • Limited efficacy
Commercially Attractive	~28,000 patients[1]

DYNAMO™ PLATFORM

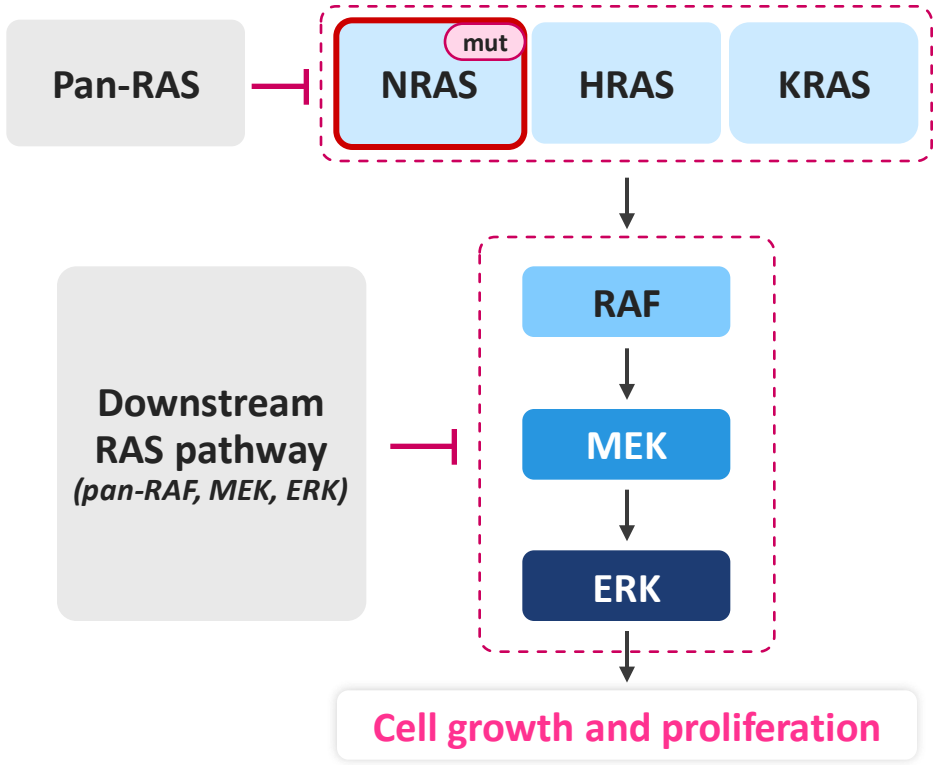
First NRAS Selective Inhibitor



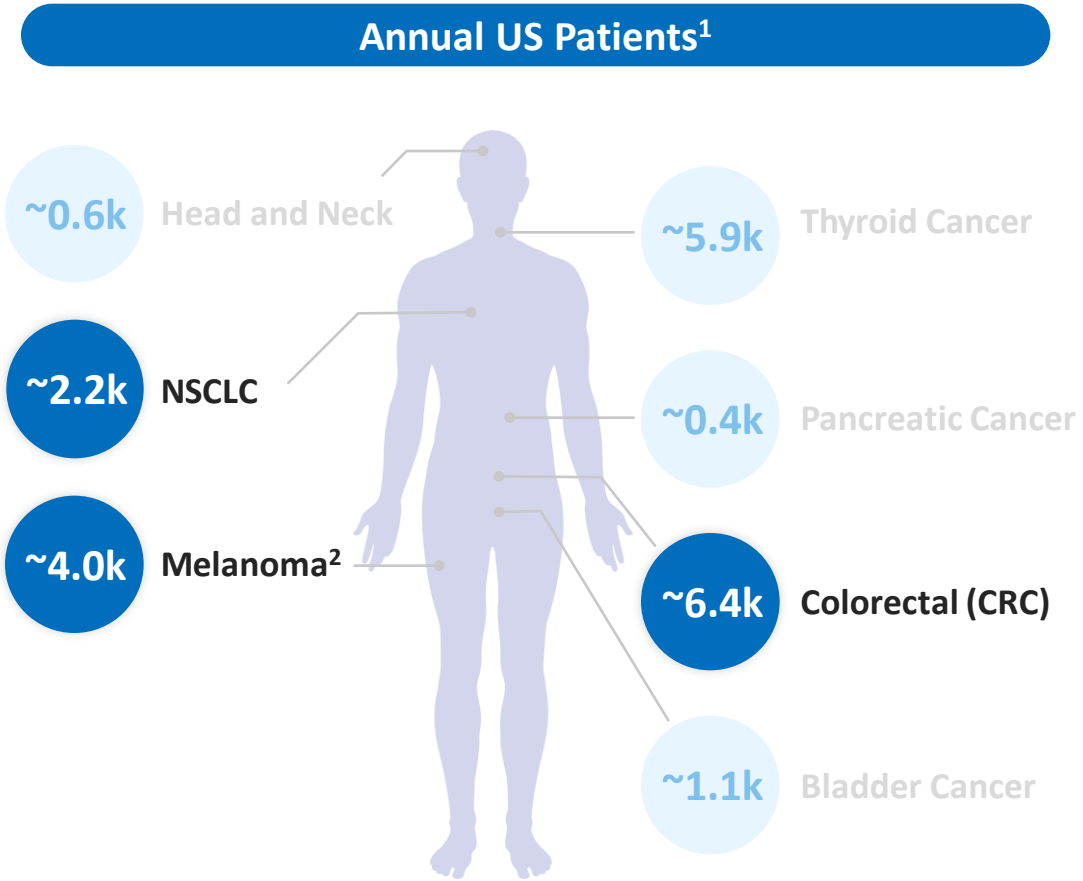
1. Newly diagnosed (incident) solid tumors with an NRAS mutation, excluding stage 0-II newly diagnosed melanoma patients (SEER, 3rd party source for alteration rate, Jan 2024)

NRAS – Large Validated Market With Significant Unmet Need

NRAS mutations are a key driver of solid tumors, though no NRAS-selective agent exists



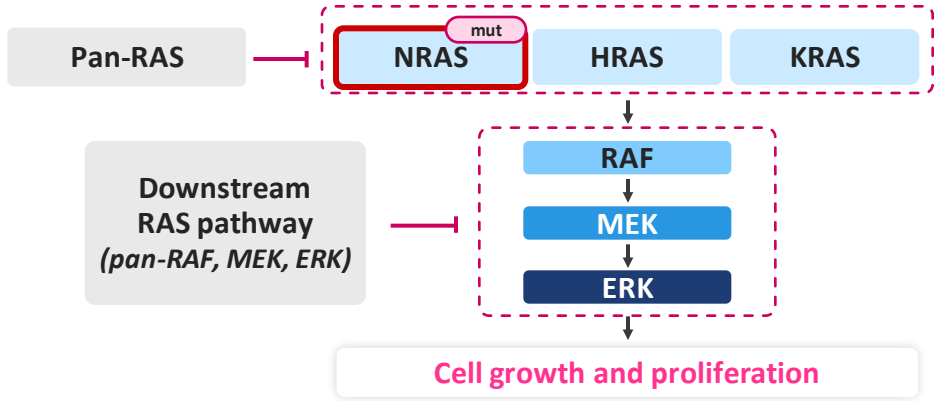
NRAS mutations observed in broad range of tumor types



1. Newly diagnosed (incident) patients with an NRAS mutation for each tumor type (SEER, 3rd party source for alteration rate, Jan 2024); 2. Melanoma includes incident stage III and IV patients only (excludes stage 0-II patients)

Limited Therapeutic Window of Current Agents – Pan-RAF/RAS & MEK Inhibitors

Non-Selective Agents in Development



Limited Tolerability

	Rash	Liver Tox
MEK + RAFi	25-80%	<10-60%
Pan-RAS	81%	7-8%

High rates of skin toxicity driven by off-target pan-RAS pathway inhibition

Limited Target Inhibition

	Dose Red.	Dose Discont.
MEK + RAFi	16-70%	7-28%
Pan-RAS	8%	1%

Limited Efficacy

Regimen (2L *NRAS*mut melanoma)	ORR	PFS (mo)
Naporafenib (RAFi) + trametinib (MEKi)	13-47%	4.2-5.5
Belvarafenib (RAFi) + cobimetinib (MEKi)	39%	7.3

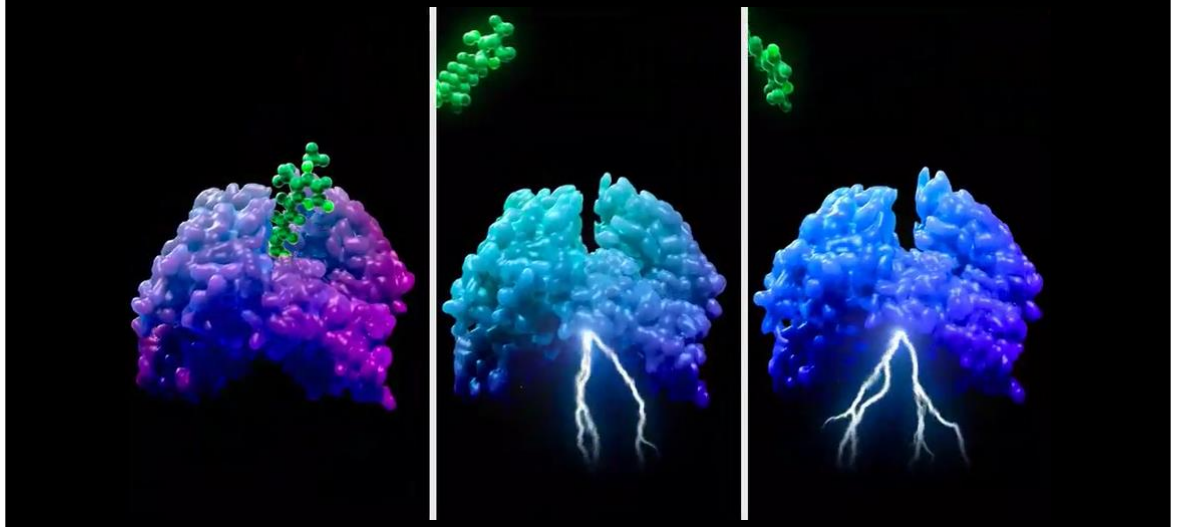
No guidance for Ph3 development

Sources: ASCO 2021 #3007 (Belvarafenib + cobimetinib, n=32 all, 13 for efficacy), de Braud 2023 J Clin Oncol 41:2651 (naporafenib + trametinib, n=30 expansion arm), ASCO 2023 #9510 (tunlametinib, n=95), ESMO 2023 652O (RMC-6236, n=111 pts at ≥80mg; liver tox = elevated ALT/AST

NRAS – Dynamo™ Platform Discovered a Novel Allosteric Pocket

Exploited differences in protein motion…

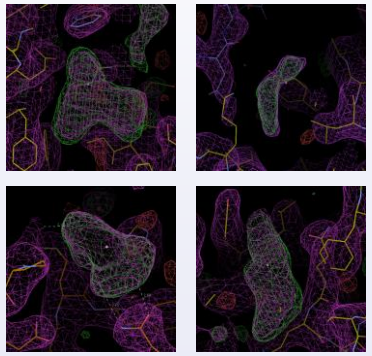
…to design first NRAS-selective inhibitor

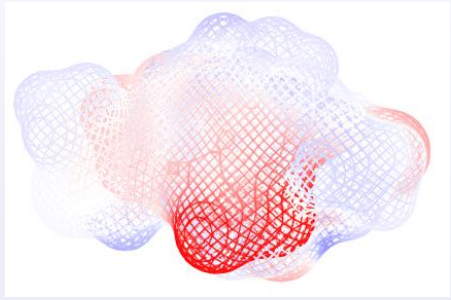


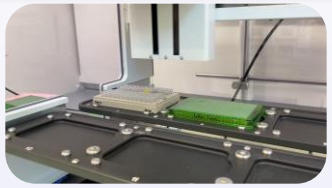
NRAS – Dynamo™: Integration of Experimental and Computational Tools

Discovery of 1st NRAS-selective inhibitor

Target Modulation Hypothesis	Hit Identification	Lead Optimization
1 Discovered a novel cryptic pocket	2 Identified & validated hits selective for NRAS (over H/KRAS)	3 Rapidly designed & prioritized NRAS inhibitors







Platform Tool Examples

X-ray Fragment Screen	2D NMR	High Throughput Automated Chem.
Virtual Screening	Computational Fragment Merging	Free Energy Calculations

Experimental tool

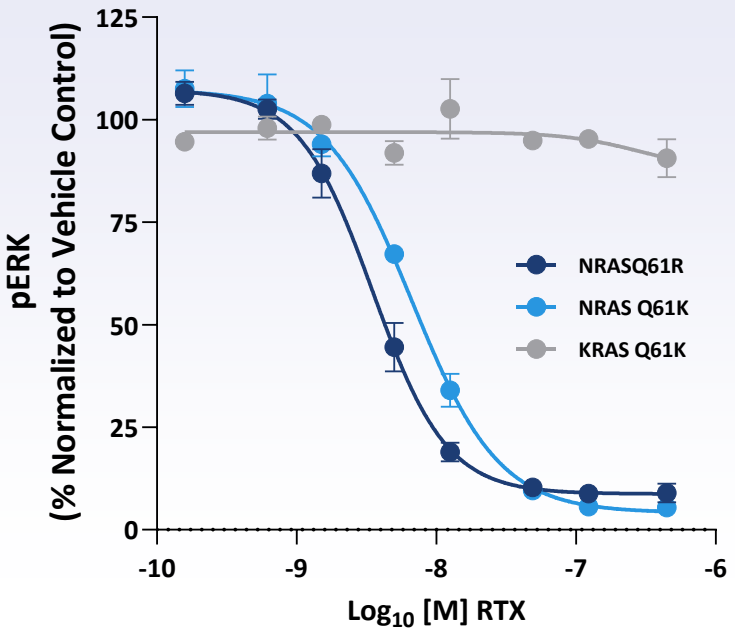
Computational tool

NRAS Inhibitors Are Potent, Selective & Active Across NRAS Mutations

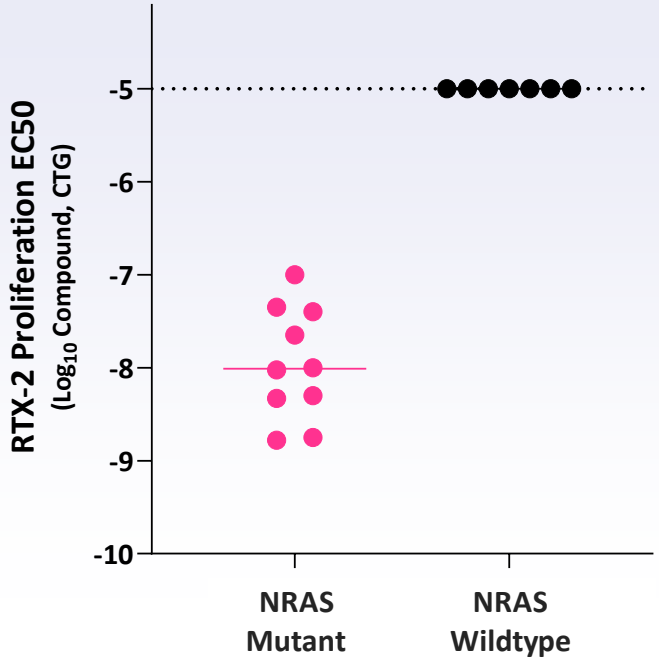
Relay Tx compounds bind to the ON-state with selectivity for NRAS[1]

Binding Affinity (nM)	RTX-2
NRAS Q61R (ON)	7
NRAS Q61K (ON)	9
NRAS Q61L (ON)	10
NRAS WT (ON)	33
NRAS WT (OFF)	100
HRAS Q61K (ON)	No binding observed
KRAS Q61K (ON)	
KRAS WT (ON)	
KRAS WT (OFF)	

…inhibit pathway signaling only in NRAS mutant cells[2]



…inhibiting proliferation of only NRAS mutant cells[3]



NRAS$^{G12C,\ G13R,\ Q61R,\ Q61K,\ Q61L}$

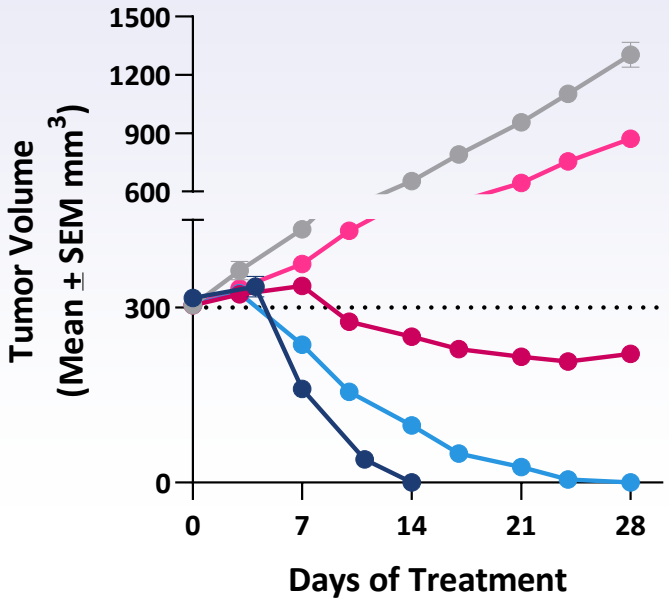
- KRAS$^{WT,\ Q61K,\ G12S}$
- HRAS$^{WT,\ G13D,\ Q61L}$
- BRAF^{V600E}

1. Based on SPR analysis of purified protein; 2. Based on pERK assay of SK-MEL-2, SK-MEL-30, and CALU-6 cell lines evaluated at 24hr timepoint; 3. Based on cell proliferation panel (17 cell lines) evaluated at 3-5d timepoint depending on cell line

NRAS Inhibitors Achieve Complete Regression at Well Tolerated Doses

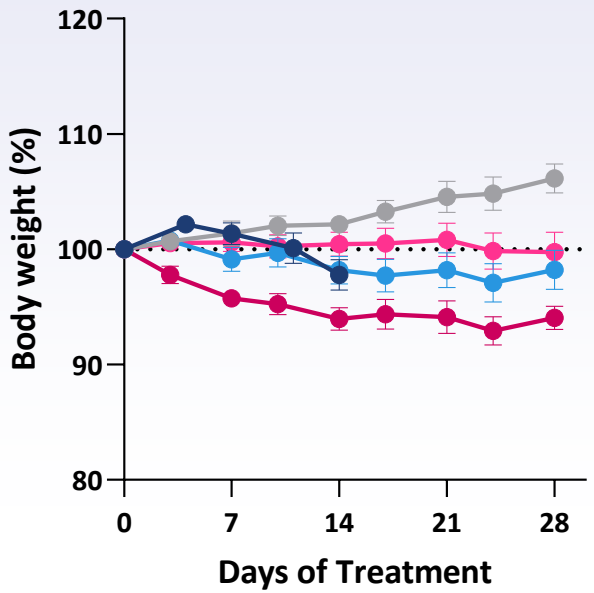
Relay Tx's NRAS inhibitors drove rapid, complete regressions…

SK-MEL-2 ($NRAS^{Q61R}$) CDX*



…and were generally well tolerated

SK-MEL-2 ($NRAS^{Q61R}$) CDX



Estimated clinically relevant doses

Vehicle | RTX-2 100mg/kg QD | RTX-4 10mg/kg QD | Binimetinib 3mg/kg BID | Exarafenib 10mg/kg + Binimetinib 3mg/kg BID

There were no adverse findings in an exploratory rat toxicology study of RTX-2 at exposures equivalent to 100mg/kg QD

*Regressions also achieved with additional NRAS mutant models (NRAS Q61K and NRAS Q61R)

NRAS – Large Validated Market With Significant Unmet Need

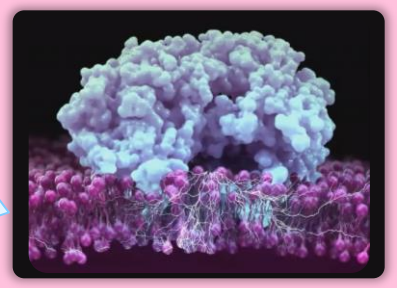
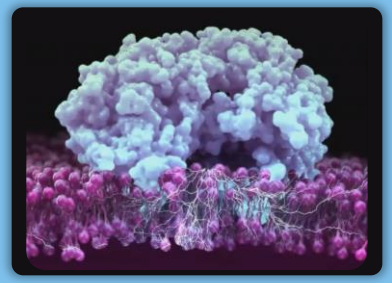
	Solid Tumors NRAS-Driven Solid Tumors
Novel Approach	1st NRAS-selective inhibitor
Genetically Defined	NRASmut
Clinically Validated	Non-selective RAF/MEK/Pan-RAS
Unmet Medical Need	• Lack of selectivity • Challenging AE profile • Limited efficacy
Commercially Attractive	~28,000 patients[1]

SOLID TUMORS PORTFOLIO MILESTONES

Clinical Start in 2H 2025

1. Newly diagnosed (incident) solid tumors with an NRAS mutation, excluding stage 0-II newly diagnosed melanoma patients (SEER, 3rd party source for alteration rate, Jan 2024)

Relay Tx – Today's Updates

	BREAST CANCER	GENETIC DISEASE		SOLID TUMORS
	1 PI3Kα-Driven Breast Cancer	2 PI3Kα-Driven Vascular Malformations	3 Fabry Disease	4 NRAS-Driven Solid tumors
	 Ongoing mono, doublet, triplet; Data update YE24			
Program Updates	1st PI3Kαi + ET + CDK4i combination in clinic (Pfizer)	1st mutant-selective PI3Kα inhibitor	1st non-inhibitory αGal chaperone	1st NRAS-selective inhibitor
Large US opportunity	~150,000 pts[1]	~170,000 pts[2] *(chronic treatment)*	~8,000 pts[3] *(chronic treatment)* $2B current market[5]	~28,000 pts[4]
Milestones	CDK4i clinical start by YE 2024	Clinical start in 1Q 2025	Clinical start in 2H 2025	Clinical start in 2H 2025

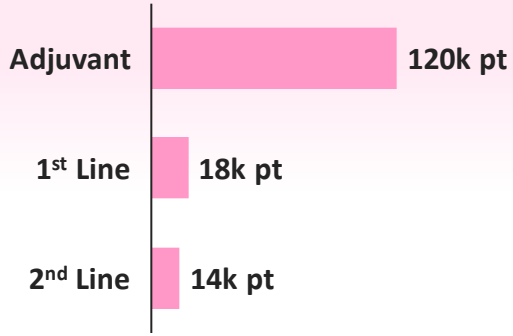
1. Prevalent US patient population with a PIK3CA mutation in adjuvant, first line metastatic and second line metastatic settings (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalence of vascular malformations with a PIK3CA mutation (Gallagher et al 2022 and several other sources); 3. Prevalence of Fabry patients (National Fabry Disease Foundation, Jan 2024); 4. Newly diagnosed (incident) solid tumors with an NRAS mutation, excluding melanoma stages 0-II (SEER, 3rd party source for alteration rate, Jan 2024); 5. Fabry disease forecasted 2024 market size per EvaluatePharma, includes Galafold® and ERTs (May 2024)

PI3Kα – Large Opportunity Across Indications and Therapeutic Areas

PIK3CA mutant HR+/HER2- Breast Cancer

~150k Patients
(US prevalence)[1]

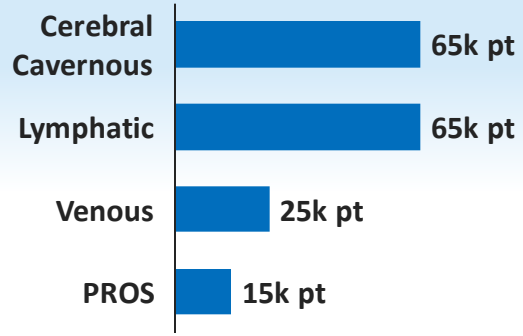
RLY-2608



PIK3CA mutant Vascular Malformations

~170k Patients
(US prevalence)[2]

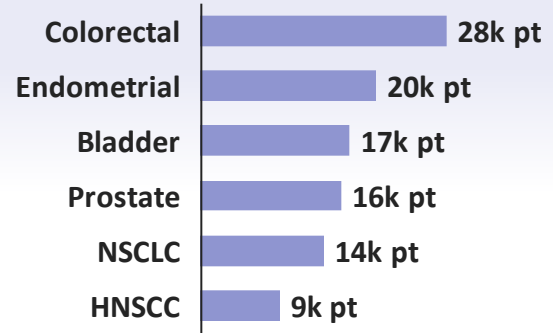
Potential for rapid POC with RLY-2608, then distinct molecule for pivotal



PIK3CA mutant Other Solid Tumors

~160k Patients
(US incidence)[3]

RLY-2608



Relay Tx's PI3Kα Franchise has the potential to address wide range of large disease indications

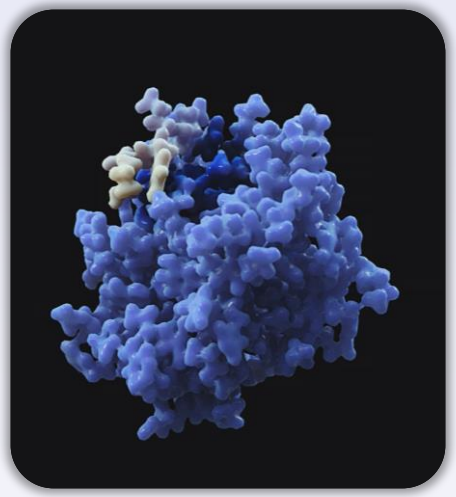
1. Prevalent US patient population with a PIK3CA mutation in each line of therapy (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalent US patient population of vascular malformation patients with a PIK3CA mutation (multiple sources); 3. Incident US patient population solid tumors annually with a PIK3CA mutation (SEER; 3rd party source for alteration rate, May 2024)

Relay Tx Dynamo™ – Internalizing Bespoke Tools

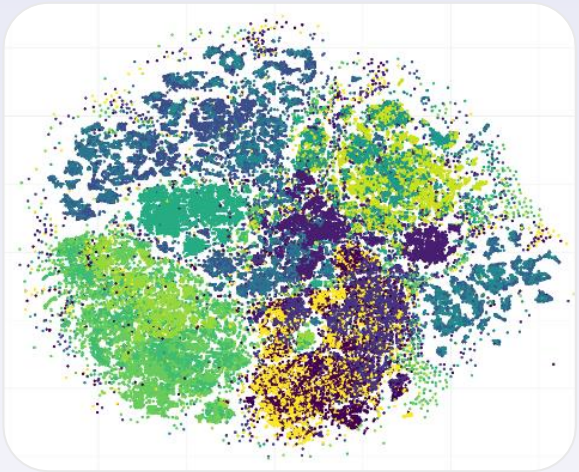
Dynamo™ Platform

Target Identification | Target Modulation Hypothesis | Hit Identification | Lead Optimization | Development & Commercial

In-House Molecular Dynamics (MD)



In-House REL-DEL

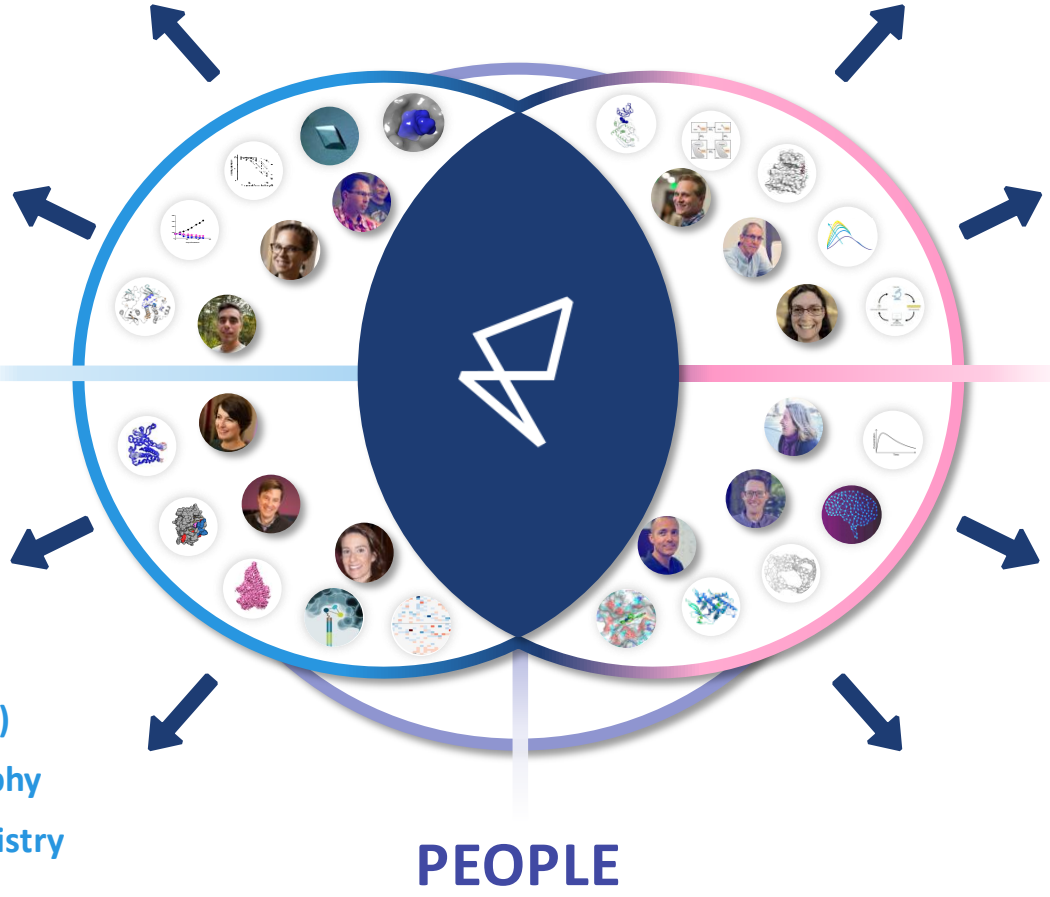


In-House High-Throughput Automated Chemistry

Relay Tx Dynamo™ – Continuing to Focus, Build and Evolve

EXPERIMENTATION

- NMR
- Mechanistic enzymology
- HDX-MS
- Cryo-EM
- X-ray fragment screening
- REL-DEL
- Structure ensembles
- Integrated pharmacology
- Protein design and engineering
- Automated Ligand ID System (ALIS)
- Ambient temp. X-Ray crystallography
- High throughput automated chemistry



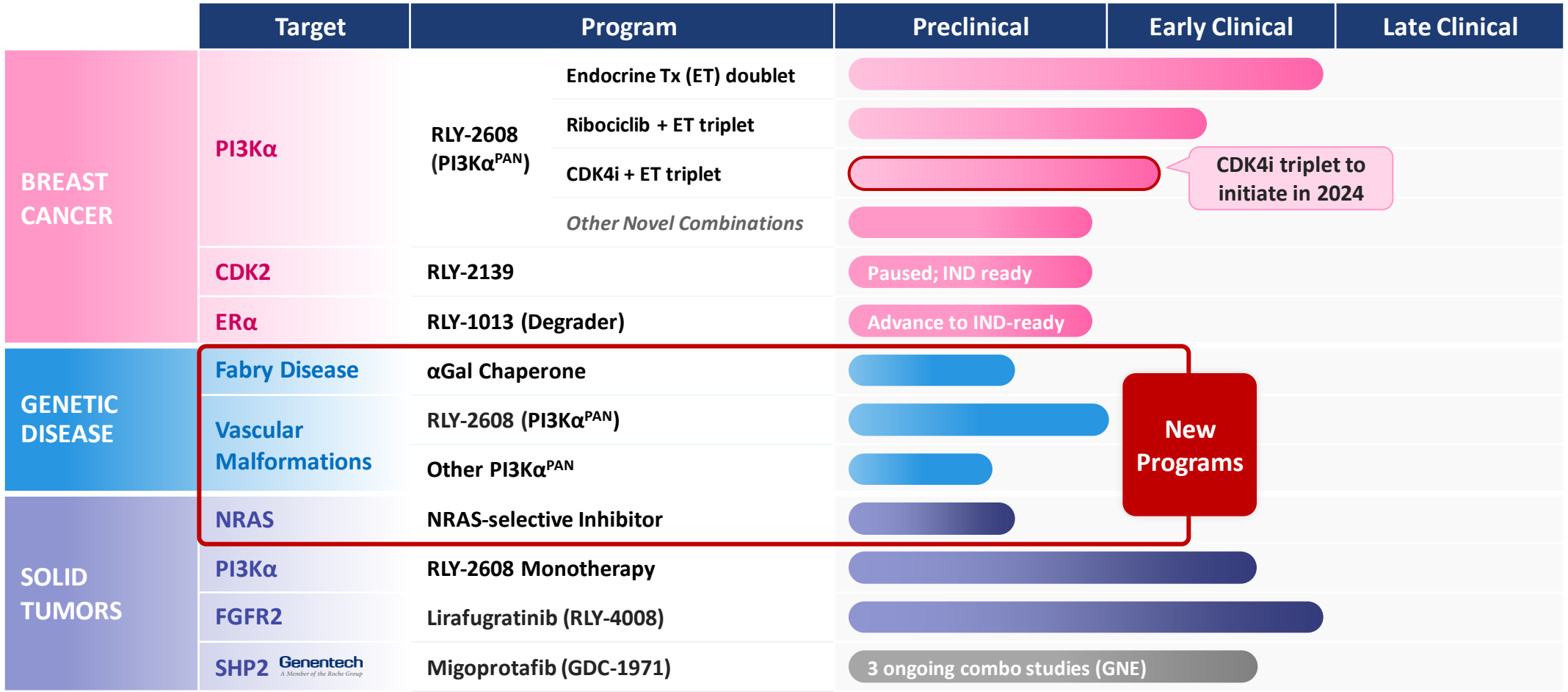
PEOPLE

COMPUTATION

- Free energy calculations
- Long time-scale MD
- Giga-scale virtual screening
- Differential dynamics
- Digitally encoded libraries
- ML-DEL + AI models for DEL
- ADME/PK models
- Active learning
- Generative design
- Automated Chemical Design
- Computational fragment merging

Dynamo™ Platform integrates industry-leading tools and will continue to quickly grow and evolve

Relay Tx – Broad Precision Medicine Pipeline



5+ additional unnamed research programs

Relay Tx – Milestones 2024-25

BREAST CANCER PORTFOLIO MILESTONES

PI3Kα	RLY-2608	• Data update in 4Q 2024
		  • Doublet safety & efficacy data
		  • Initial triplet data
		• CDK4i triplet clinic start by YE 2024
		• Potential pivotal trial start in 2025

CDK2	RLY-2139	IND-ready
ERα	RLY-1013	• IND-ready in 2025

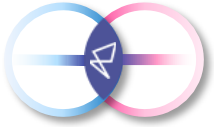
GENETIC DISEASE PORTFOLIO MILESTONES

Fabry	New Program	• Clinical start in 2H 2025
VM	New Program	• Clinical start in 1Q 2025

SOLID TUMORS PORTFOLIO MILESTONES

NRAS	New Program	• Clinical start in 2H 2025
FGFR2	Lirafugratinib	• Tumor agnostic data & regulatory update in 2H 2024
SHP2	Migoprotafib	• Three ongoing combo trials*

** Genentech controls data disclosures*



DYNAMO™ PLATFORM | **5+ unnamed research programs**

~$750M cash as of end Q1 2024
Expected to fund current operating plan into 2H 2026