UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

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

Date of Report (Date of earliest event reported): June 05, 2024

RELAY THERAPEUTICS, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-39385**	**47-3923475**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

399 Binney Street **Cambridge, Massachusetts**	**02139**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (617) 370-8837

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

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

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, par value $0.001 per share	RLAY	Nasdaq Global Market

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

Emerging growth company ☐

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

Item 7.01 Regulation FD Disclosure.

On June 5, 2024, Relay Therapeutics, Inc. (the "Company") issued a press release announcing a clinical trial collaboration between the Company and Pfizer, Inc. ("Pfizer"), a copy of which is furnished herewith as Exhibit 99.1.

On June 6, 2024, the Company issued a press release announcing three new programs from its existing pre-clinical pipeline, a copy of which is being furnished as Exhibit 99.2 to this Current Report on Form 8-K. The Company intends to host a New Program and Platform event on June 6, 2024 from 8:00 to 10:00 a.m. ET to discuss these new programs and describe how the Dynamo™ platform led to these discoveries. The Company has made available a slide presentation to accompany this event, a copy of which is being furnished as Exhibit 99.3 to this Current Report on Form 8-K.

The information in this Item 7.01, including Exhibit 99.1, Exhibit 99.2 and Exhibit 99.3 attached hereto, is intended to be furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 8.01 Other Events.

On June 5, 2024, the Company announced a global clinical trial collaboration with Pfizer for the development of RLY-2608 in combination with fulvestrant and atirmociclib, Pfizer's investigative selective-CDK4 inhibitor, in patients with PI3Kα-mutated, HR+, HER2- metastatic breast cancer. On June 6, 2024, the Company announced three new programs from its existing pre-clinical pipeline, including two novel programs from its genetic disease portfolio to address clinically and commercially validated targets in vascular malformations and Fabry disease, respectively, and an NRAS-selective inhibitor.

Cautionary Note Regarding Forward Looking Statements

This Current Report on Form 8-K and certain of the materials furnished or filed herewith contain 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 Company's strategy, business plans and focus; the progress and timing of updates on the clinical development of the programs across the Company's portfolio, including the expected therapeutic benefits of its programs, and potential efficacy and tolerability, and the timing and success of interactions with and approval of regulatory authorities; the timing of clinical data updates across the Company's 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 the Company's various programs, including a potential pivotal trial for RLY-2608, clinical development in vascular malformations, clinical development of the Company's non-inhibitory chaperone, and clinical development of its NRAS-selective inhibitor; the potential of the Company's product candidates to address a major unmet medical need; the cash runway projection; the competitive landscape and potential market opportunities for the Company's product candidates; the expected strategic benefits under the Company's collaborations, including the clinical trial collaboration with Pfizer; the capabilities and development of the Dynamo platform, including its role in identifying product candidates; the Company's ability to successfully establish or maintain collaborations or strategic relationships for its product candidates; expectations regarding current and future interactions with the U.S. Food and Drug Administration, or FDA; the Company's ability to manufacture its product candidates in conformity with the FDA's requirements; plans to develop, manufacture and commercialize the current product candidates and any future product candidates; and the implementation of the Company's business model and strategic plans for its 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 are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words.

Any forward-looking statements are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements contained in this Current Report on Form 8-K or the materials furnished or filed herewith, including, without limitation, risks associated with: the impact of global economic uncertainty, geopolitical instability, or public health epidemics or outbreaks of an infectious disease on countries or regions in which the Company has operations or does business, as well as on the timing and anticipated results of its clinical trials, strategy, future operations and profitability; the delay of any current or planned clinical trials or the development of the Company's drug candidates; the risk that the preliminary results of its preclinical or clinical trials may not be predictive of future or final results in connection with future clinical trials of its product candidates; the Company's ability to successfully demonstrate the safety and efficacy of its drug candidates; the timing and outcome of its planned interactions with regulatory authorities; and obtaining, maintaining and protecting its intellectual property. These and other risks and uncertainties are described in greater detail in the section entitled "Risk Factors" in the Company's most recent Annual Report on Form 10-K and Quarterly Report on Form 10-Q, as well as any subsequent filings with the Securities and Exchange Commission. In addition, any forward-looking statements represent the Company's views only as of today and should not be relied upon as representing its views as of any subsequent date. the Company explicitly disclaims any obligation to update any forward-looking statements. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements.

Item 9.01 Financial Statements and Exhibits.

99.1	Press release issued by Relay Therapeutics, Inc. on June 5, 2024, furnished herewith.
99.2	Press release issued by Relay Therapeutics, Inc. on June 6, 2024, furnished herewith.
99.3	New Program and Platform event presentation, dated June 2024, furnished herewith.
104	Cover Page Interactive Data File (embedded within Inline XBRL document).

SIGNATURES

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

RELAY THERAPEUTICS, INC.

Date: June 6, 2024

By: /s/ Brian Adams
Brian Adams
Chief Legal Officer

Exhibit 99.1

Relay Therapeutics Announces Clinical Trial Collaboration with Pfizer to Evaluate Atirmociclib in Combination with RLY-2608

Initial triplet combination of RLY-2608 + atirmociclib + fulvestrant to be evaluated in patients with PI3Kα-mutated HR+/HER2- metastatic breast cancer; initiation planned by end of 2024

Cambridge, Mass. – June 5, 2024 – Relay Therapeutics, Inc. (Nasdaq: RLAY), a clinical-stage precision medicine company transforming the drug discovery process by combining leading-edge computational and experimental technologies, today announced a clinical trial collaboration with Pfizer Inc. (NYSE: PFE) to evaluate atirmociclib, Pfizer's investigative selective-CDK4 inhibitor, in combination with RLY-2608 and fulvestrant in patients with PI3Kα-mutated, HR+, HER2- metastatic breast cancer.

"We are very enthusiastic to evaluate Pfizer's novel investigative selective-CDK4 inhibitor atirmociclib in combination with RLY-2608, the first mutant selective PI3Kα inhibitor," said Don Bergstrom, M.D., Ph.D., President of R&D at Relay Therapeutics. "We believe that combining these two selective agents – atirmociclib and RLY-2608 – will avoid key off-target toxicity that comes from hitting CDK6 and wild-type PI3Kα, which has historically significantly limited use of non-selective agents. The breast cancer treatment landscape continues to evolve quickly, and we are pleased that the safety profile RLY-2608 has demonstrated to-date makes it well-positioned to be part of the next generation of therapies."

Under the terms of the agreement, Pfizer will provide atirmociclib for use in the study and Relay will be responsible for conducting the study. The RLY-2608 + atirmociclib + fulvestrant triplet combination is planned to begin by the end of 2024.

About RLY-2608

RLY-2608 is the lead program in Relay Therapeutics' efforts to discover and develop mutant selective inhibitors of PI3Kα, the most frequently mutated kinase in all cancers, with oncogenic mutations detected in about 14% of patients with solid tumors. RLY-2608 has the potential, if approved, to address more than 250,000 patients per year in the United States, one of the largest patient populations for a precision oncology medicine.

Traditionally, the development of PI3Kα inhibitors has focused on the active, or orthosteric, site. The therapeutic index of orthosteric inhibitors is limited by the lack of clinically meaningful selectivity for mutant versus wild-type (WT) PI3Kα and off-isoform activity. Toxicity related to inhibition of WT PI3Kα and other PI3K isoforms results in sub-optimal inhibition of mutant PI3Kα with reductions in dose intensity and frequent discontinuation. The Dynamo™ platform enabled the discovery of RLY-2608, the first known allosteric, pan-mutant, and isoform-selective PI3Kα inhibitor, designed to overcome these limitations. Relay Therapeutics solved the full-length cryo-EM structure of PI3Kα, performed computational long time-scale molecular dynamic simulations to elucidate conformational differences between WT and mutant PI3Kα, and leveraged these insights to support the design of RLY-2608. RLY-2608 is currently being evaluated in a first-in-human trial designed to treat patients with advanced solid tumors with a PIK3CA (PI3Kα) mutation. For more information on RLY-2608, please visit here.

About Relay Therapeutics

Relay Therapeutics is a clinical-stage precision medicine company transforming the drug discovery process by combining leading-edge computational and experimental technologies with the goal of bringing life-changing therapies to patients. As the first of a new breed of biotech created at the intersection of complementary techniques and technologies, Relay Therapeutics aims to push the boundaries of what's possible in drug discovery. Its Dynamo™ platform integrates an array of leading-edge computational and experimental approaches designed to drug protein targets that have previously been intractable or inadequately addressed. Relay Therapeutics' initial focus is on enhancing small molecule therapeutic discovery in targeted oncology and genetic disease indications. For more information, please visit www.relaytx.com or follow us on Twitter.

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Exhibit 99.2

Relay Therapeutics Discloses Three New Programs at New Program & Platform Event

3 new programs include 2 genetic disease programs – vascular malformations & Fabry disease – & 1 precision oncology program – NRAS-specific inhibitor

Cash guidance remains unchanged, and is expected to fund operations into second half of 2026
Relay Therapeutics to host webcast event today, June 6, at 8:00 a.m. ET

Cambridge, Mass. – June 6, 2024 – Relay Therapeutics, Inc. (Nasdaq: RLAY), a clinical-stage precision medicine company transforming the drug discovery process by combining leading-edge computational and experimental technologies, will provide details on the company's portfolio during its New Program & Platform event today, June 6, 2024, from 8:00 to 10:00 a.m. ET. As part of the event, the company will disclose three new programs from its existing pre-clinical pipeline and will review how the Dynamo™ platform led to these discoveries. The new programs include two novel programs from its genetic disease portfolio and a potentially first-in-class NRAS-selective inhibitor. Both genetic disease programs have the potential to provide a unique approach to addressing clinically and commercially validated targets in vascular malformations and Fabry disease. The new programs announced today do not change cash guidance, which is expected to fund operations into the second half of 2026.

"Since Relay Therapeutics was founded eight years ago, our Dynamo platform has been very productive and we have made significant progress advancing our initial set of programs, including four that have entered the clinic. We have successfully created molecules for a variety of targets to-date, have shown clinical proof-of-concept for two of these programs and are aiming to start our first Phase 3 study next year with RLY-2608," said Sanjiv Patel, M.D., President and Chief Executive Officer of Relay Therapeutics. "Today, we are very excited to unveil the next set of innovative programs, which demonstrate the power of our Dynamo platform, and which we believe will drive the next wave of the company's growth. These new programs underscore the breadth of the platform's capabilities with expansion beyond precision oncology into genetic disease and beyond inhibitors to small molecule chaperones."

New Programs Potentially Addressing More Than 200,000 Patients in the United States

The New Program & Platform event presentation will highlight newly disclosed programs in vascular malformations, Fabry disease and NRAS.

Vascular Malformations

- Vascular malformations are a series of rare syndromes that occur due to atypical development of lymphatic and/or blood vessels, which enlarge or form tangles, pockets or shunting vessels that cause abnormal blood flow. They can occur in different parts of the body, vary in severity and may cause symptoms such as pain, swelling, skin discoloration, limb asymmetry and functional limits. The malformations typically grow over time, and, depending on what vessel(s) are involved, can become life-threatening.
- The primary vessel(s) involved determine the sub-type of malformation, which can include venous malformations, cerebral cavernous malformations, lymphatic malformations and PIK3CA-related overgrowth spectrum.

- PI3Kα is the most common driver mutation among these sub-types, causing an estimated 55 percent of these vascular malformations.
- In the U.S., an estimated 170,000 people have one of these sub-types driven by a PI3Kα mutation.
- A mutant selective PI3Kα inhibitor provides the opportunity for greater target coverage, leading to the potential for improved efficacy and better chronic tolerability.
- Relay Therapeutics plans to initiate clinical development of RLY-2608 in vascular malformations in the first quarter of 2025.

Fabry Disease

- In Fabry disease, a defective gene (*GLA*) prohibits the body from producing enough healthy versions of an enzyme called alpha-galactosidase A (αGal), which is responsible for breaking down Gb3 (globotriaosylceramide), a fat-like substance. As a result, harmful levels of Gb3 accumulate in blood cells and tissues throughout the body, which can lead to a range of symptoms, including potentially life-threatening ones such as kidney failure, heart failure and stroke.
- In the U.S., approximately 8,000 people are estimated to have this rare, progressive genetic disorder.
- Relay Therapeutics has created the first investigational non-inhibitory chaperone for Fabry disease, which is designed to stabilize the αGal protein without inhibiting its activity, thus enabling greater Gb3 clearance across organs.
- A non-inhibitory chaperone could potentially serve as a chronic treatment option for people with Fabry disease, either as a monotherapy or in combination with enzyme replacement therapy.
- The company expects its non-inhibitory chaperone to enter the clinic in the second half of 2025.

NRAS

- NRAS is a known oncogene driver that belongs to the RAS family of signaling proteins. It plays an important role in cell division, cell differentiation and programmed cell death. The NRAS protein is responsible for converting GTP to GDP and is turned “on” when it binds to GTP and “off” once the GTP is converted to GDP. When mutated, the NRAS gene creates NRAS proteins that are always “on”, which makes cells grow and divide uncontrollably and can lead to a number of cancers, including melanoma, colorectal and non-small-cell lung.
- In the U.S., an estimated 28,000 people are diagnosed each year with mutated NRAS solid tumors.
- Existing approved and in-development treatments either target all RAS proteins (pan-RAS) or target other downstream parts of the pathway such as RAF and MEK, which leads to significant off-target toxicity and limits efficacy.

- Relay Therapeutics has created the first NRAS-selective inhibitor, which has been designed to address the liabilities of current pan-RAS inhibitors by only binding to NRAS, while sparing KRAS and HRAS.
- The company expects to initiate clinical development of its NRAS-selective inhibitor in the second half of 2025.

Anticipated Milestones

- Breast Cancer
    - RLY-2608 + fulvestrant data update in the fourth quarter of 2024
    - RLY-2608 + fulvestrant + ribociclib initial safety data in the fourth quarter of 2024
    - RLY-2608 + fulvestrant + atirmociclib clinical trial initiation by the end of 2024
    - RLY-2608 + fulvestrant potential Phase 3 trial initiation in 2025
- Genetic Disease
    - Vascular malformations: RLY-2608 clinical trial initiation in the first quarter of 2025
    - Fabry disease: clinical start in the second half of 2025
- Precision Oncology
    - Lirafugratinib: tumor agnostic data and regulatory update in the second half of 2024
    - NRAS: clinical start in the second half of 2025

Platform Productivity

Since the founding of Relay Therapeutics in 2016, the company has built and grown its Dynamo drug discovery platform, which combines experimental and computational techniques, tools and team members. Over the last eight years, Dynamo has been very productive, resulting in eight drug candidates (DCs) and four Investigational New Drug Applications (INDs), including two programs that have demonstrated clinical proof-of-concept. By the end of 2025, Relay Therapeutics expects three new clinical starts from the additional novel programs announced today. Collectively, over the first decade of the company's history, that would be 11 DCs, seven INDs and seven programs that have entered the clinic.

Cash Runway

The three new programs disclosed today are from Relay Therapeutics' existing pre-clinical pipeline. The continued advancement of these programs has already been accounted for in the company's existing cash runway guidance. As of March 31, 2024, cash, cash equivalents and investments totaled approximately $750 million and are expected to fund the current operating plan into the second half of 2026.

Event Information

Relay Therapeutics' New Program & Platform event will begin at 8:00 a.m. ET and is expected to conclude at approximately 10:00 a.m. ET. The live webcast can be accessed here or on Relay Therapeutics' website under Events in the News & Events section through the following link: https://ir.relaytx.com/news-events/events-presentations. An archived replay of the webcast will be available following the event. It is recommended that participants register at least 15 minutes in advance of the event.

About Relay Therapeutics

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Exhibit 99.3

RELAY®
THERAPEUTICS

New Program & Platform Event

June 2024

Disclaimer

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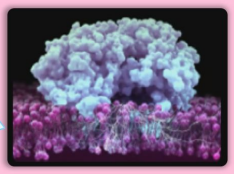
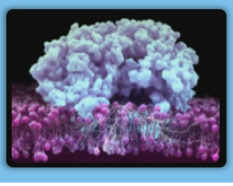
Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and our own internal estimates and research. While we believe these third-party studies, publications, surveys and other data to be reliable as of the date of this presentation, we have not independently verified, and make no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, no independent source has evaluated the reasonableness or accuracy of our internal estimates or research and no reliance should be made on any information or statements made in this presentation relating to or based on such internal estimates and research.

This presentation contains trademarks, trade names and service marks of other companies, which are the property of their respective owners.

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	Pfizer 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

Ongoing mono, doublet, triplet; Data update YE24

$2B current market[5]

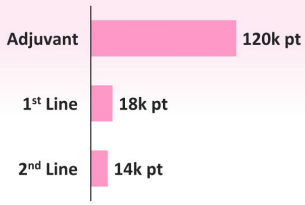
1. Prevalent US patient population with a PIK3CA mutation in adjuvant, first line metastatic and second line metastatic settings (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalence of vascular malformations with a PIK3CA mutation (Gallagher 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]

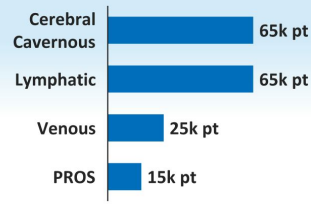
RLY-2608



PIK3CA mutant Vascular Malformations

~170k Patients
(US prevalence)[2]

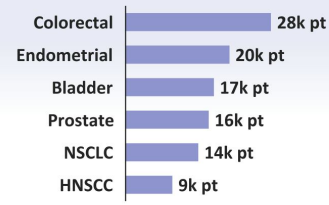
Potential for rapid POC with RLY-2608, then distinct molecule for pivotal



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)

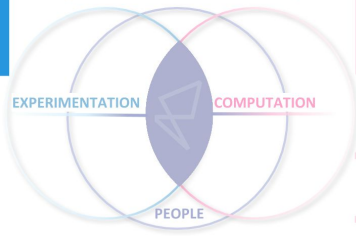
Relay Tx – Productive Platform

2016 | 2024 | FUTURE EXPECTATIONS

PRODUCTIVE PLATFORM

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

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



CONTINUED PLATFORM PRODUCTIVITY

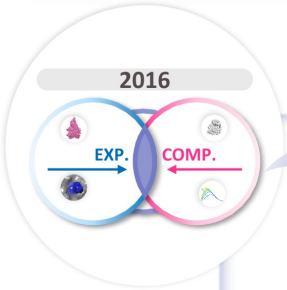
+3 New DCs | **+3** New INDs | **+3** New clinical starts

Expected platform production by YE 2025

- + 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

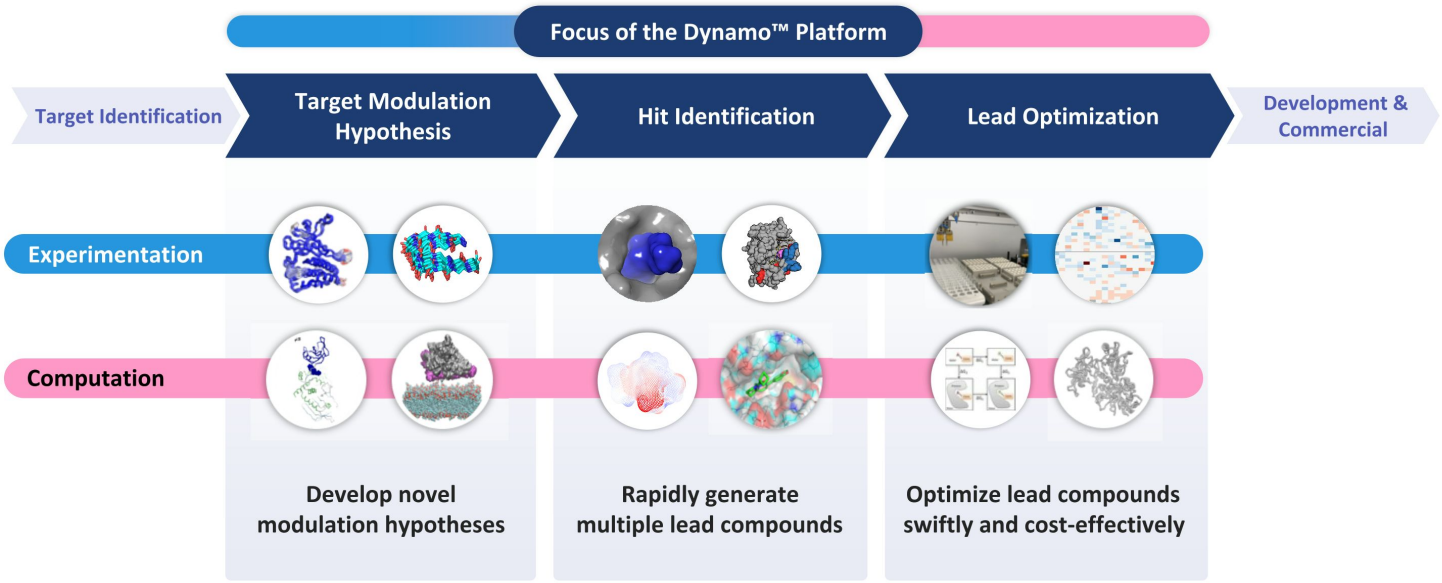
- NMR
- Mechanistic enzymology
- HDX-MS
- Cryo-EM
- X-ray fragment screening
- REL-DEL
- Structure ensembles
- Integrated pharmacology
- Protein design and engineering
- Automated Ligand ID System (ALIS)
- Ambient temp. X-Ray crystallography
- High throughput automated chemistry

PEOPLE

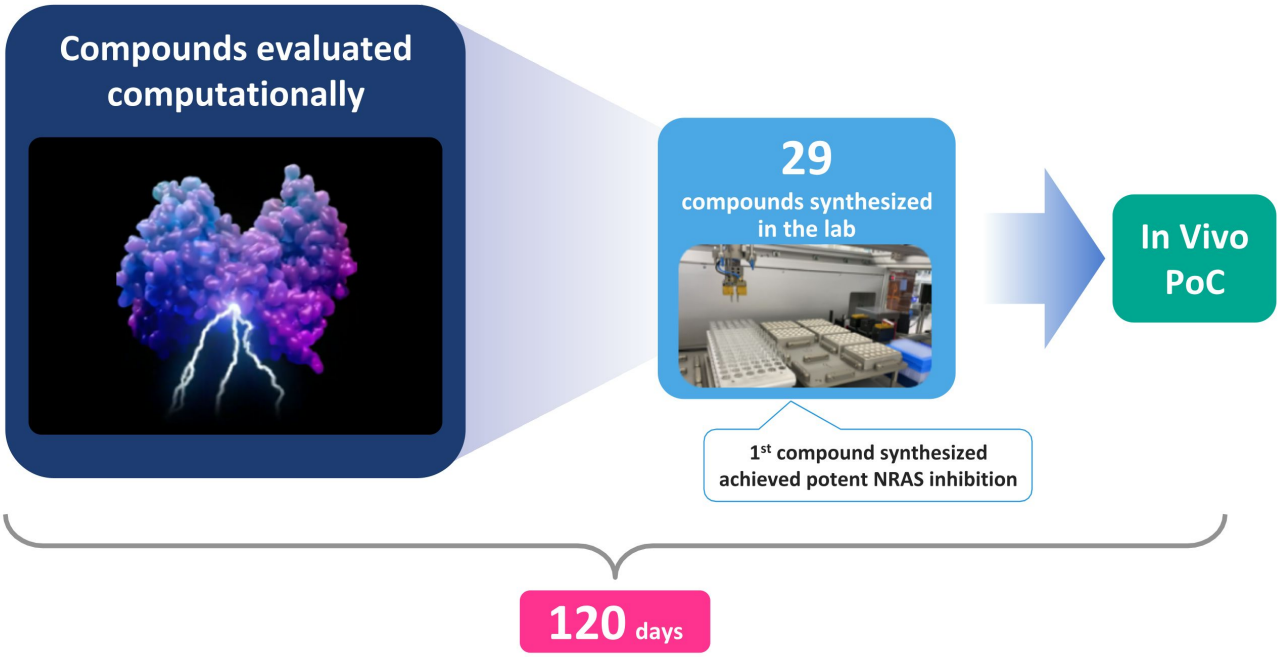
COMPUTATION

- 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

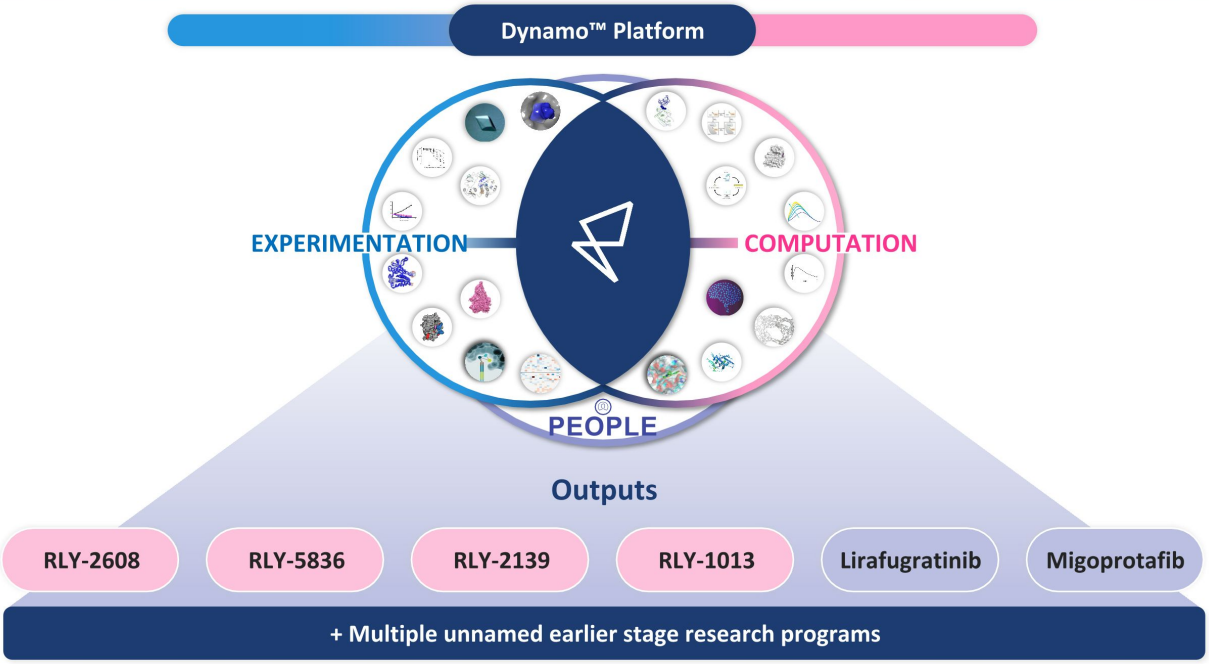
Relay Tx's Dynamo™ Platform – Experimentation Closely Integrated with Computation



Dynamo™ Platform Enabled Rapid Creation of First Selective NRAS Inhibitor



Relay Tx's Dynamo™ – Focused on Output

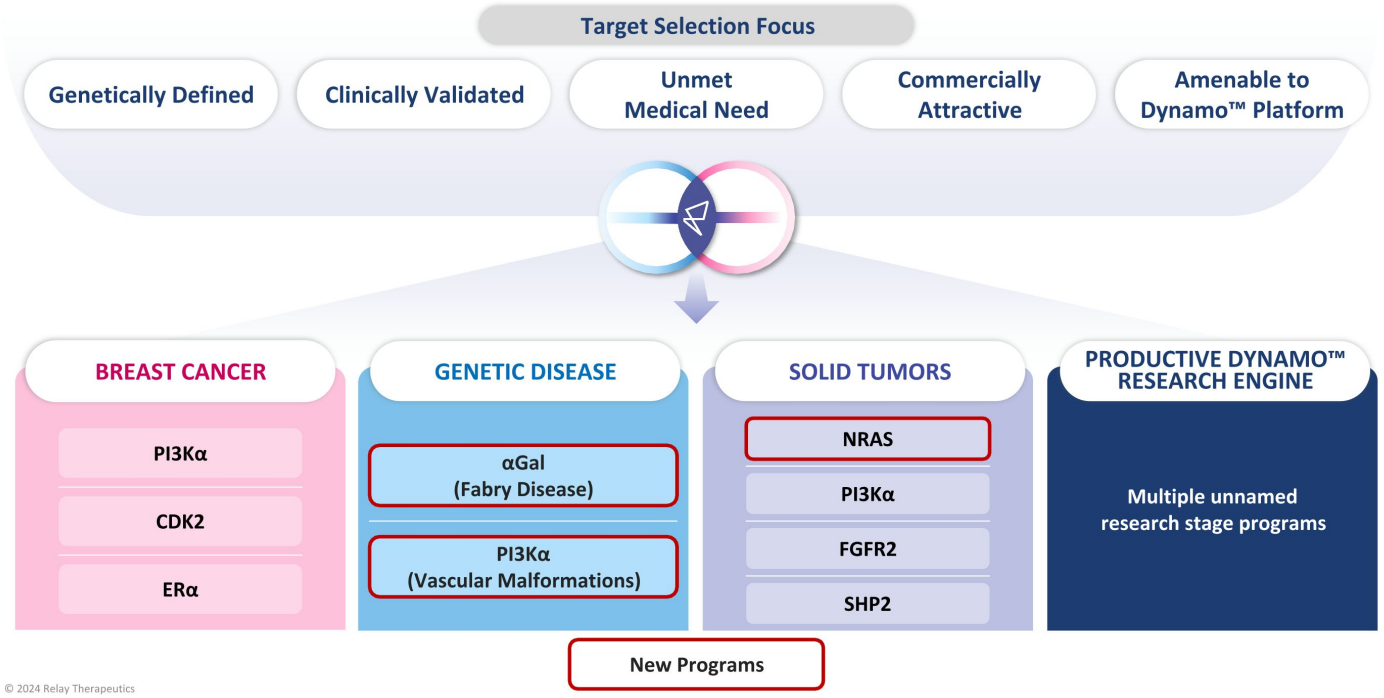


Outputs

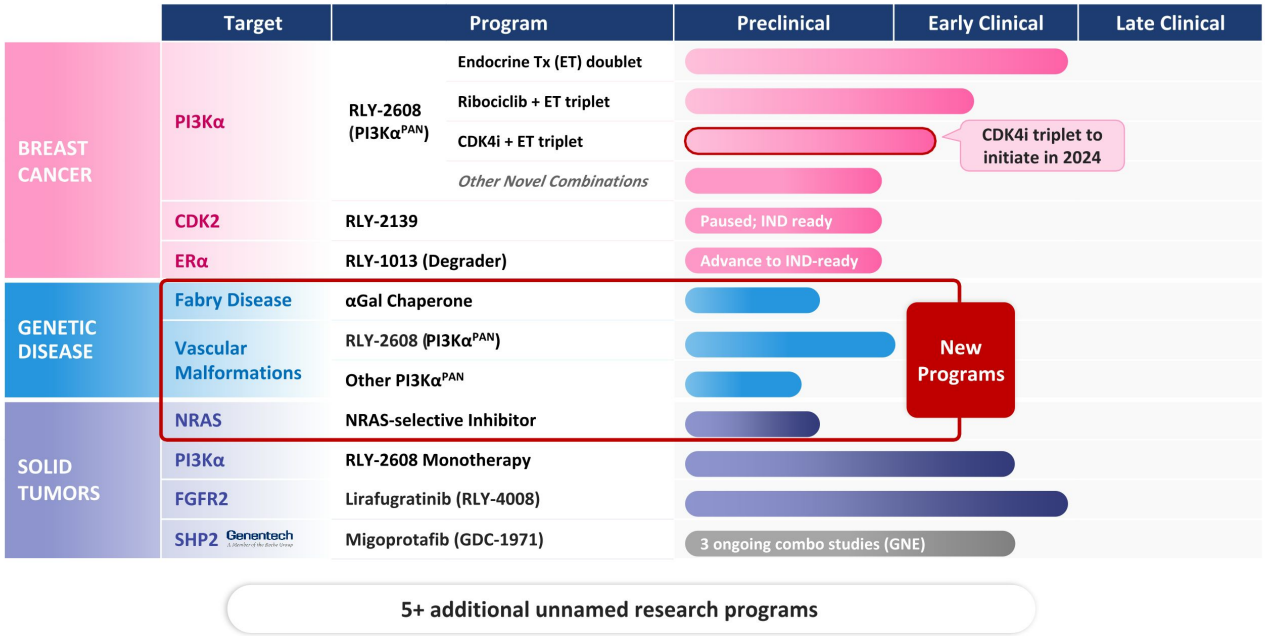
RLY-2608 | RLY-5836 | RLY-2139 | RLY-1013 | Lirafugratinib | Migoprotafib

+ Multiple unnamed earlier stage research programs

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



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

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PI3Kα – Large Opportunity Across Indications and Therapeutic Areas

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~150k Patients
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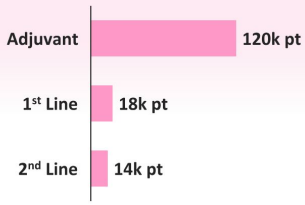
RLY-2608



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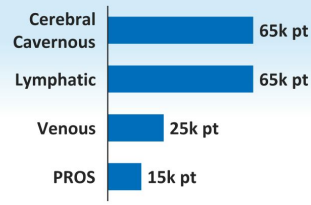

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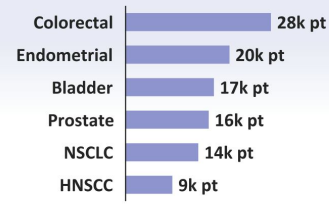
RLY-2608



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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*

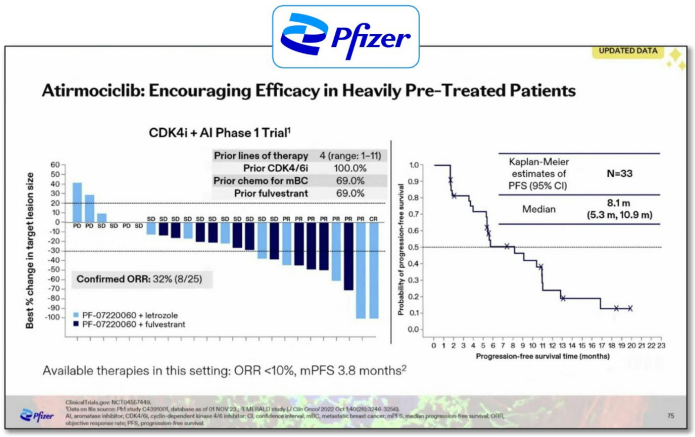
…for which Relay Tx's broad next generation ER+/HER2- BC Portfolio is designed to address

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)

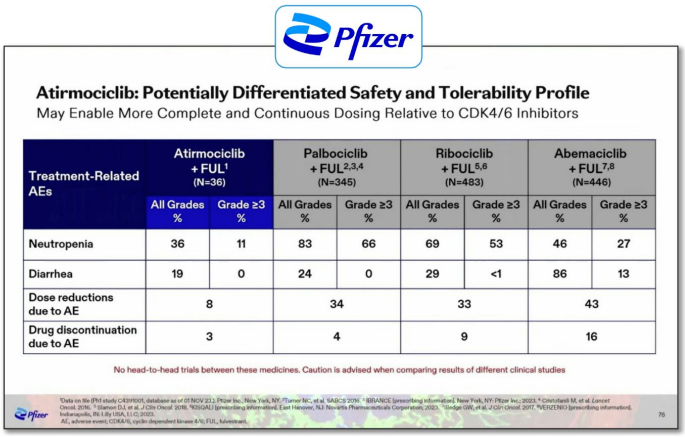
1. Decision Resources Group – Breast Cancer Disease Landscape & Forecast (Nov 2023); 2. Prevalent US patient population with a PIK3CA mutation in each line of therapy (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 3. RLY-2139 is paused and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

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

Encouraging Efficacy Data in Heavily Pre-Treated Patients



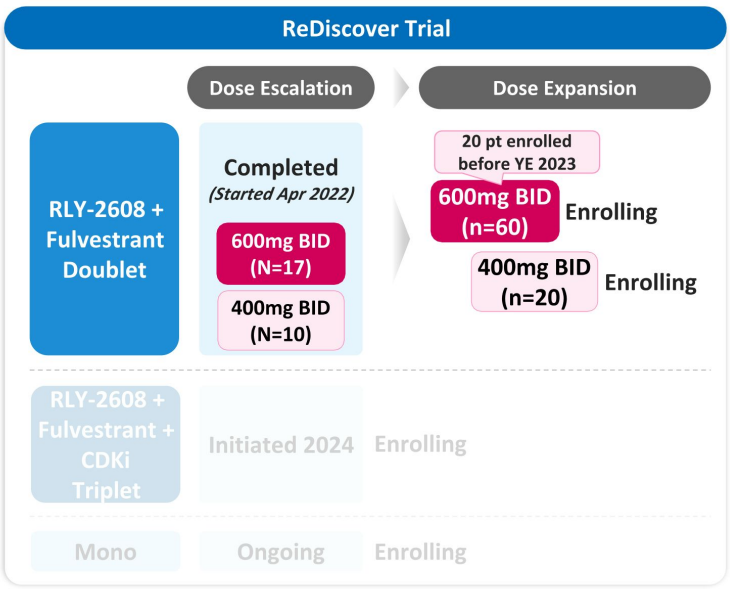
Potentially Differentiated Safety and Tolerability Profile

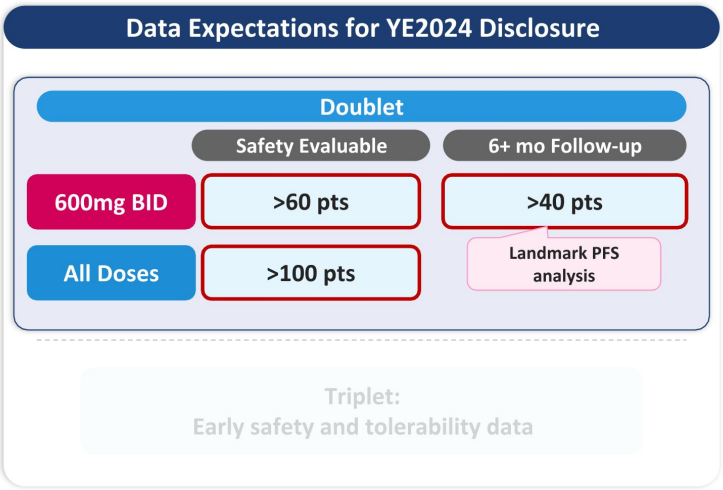


Treatment-Related AEs	Atirmociclib + FUL[1] (N=36)		Palbociclib + FUL[2,3,4] (N=345)		Ribociclib + FUL[5,6] (N=483)		Abemaciclib + FUL[7,8] (N=446)	
	All Grades %	Grade ≥3 %	All Grades %	Grade ≥3 %	All Grades %	Grade ≥3 %	All Grades %	Grade ≥3 %
Neutropenia	36	11	83	66	69	53	46	27
Diarrhea	19	0	24	0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

Source: Pfizer Oncology Innovation Day presentation, Feb 2024

RLY-2608 – Broad Development Program: Doublet





RLY-2608 – Broad Development Program: Triplet

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

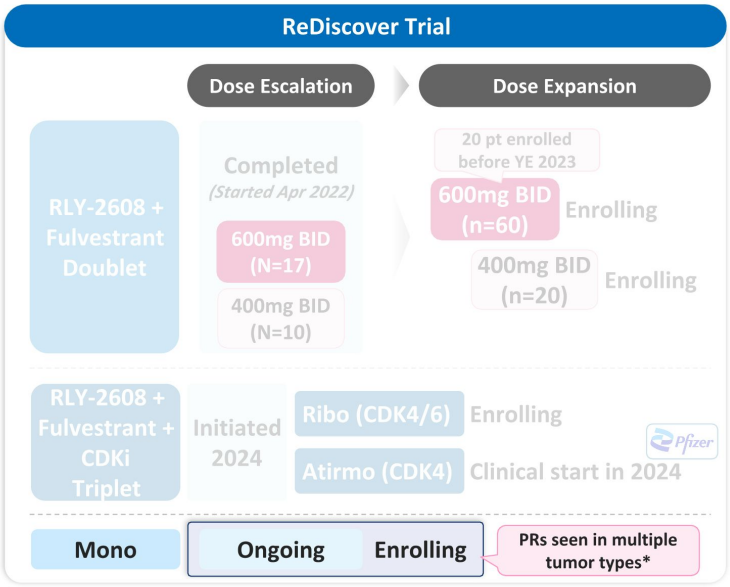
Data Expectations for YE2024 Disclosure

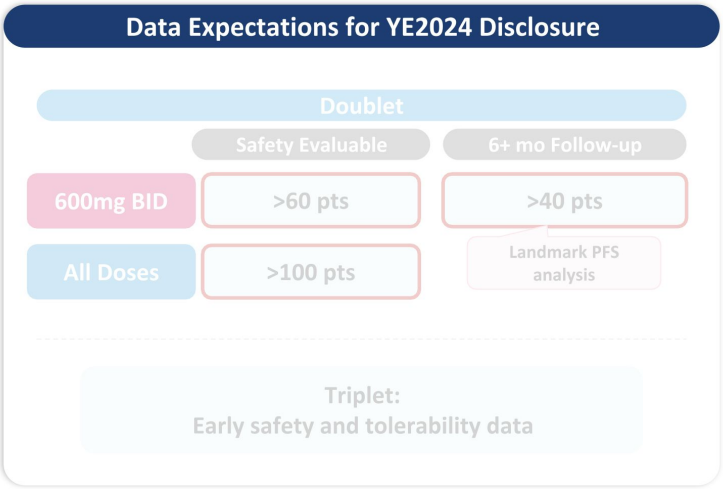
Doublet	Safety Evaluable	6+ mo Follow-up
600mg BID	>60 pts	>40 pts
All Doses	>100 pts	

Landmark PFS analysis

Triplet:
Early safety and tolerability data

RLY-2608 – Broad Development Program: Mono





* PRs include both confirmed and unconfirmed partial responses

RLY-2608 – Broad Development Program

ReDiscover Trial

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		Atirmo (CDK4)	Clinical start in 2024
Mono	Ongoing		Enrolling

PRs seen in multiple tumor types*

Pfizer

Data Expectations for YE2024 Disclosure

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

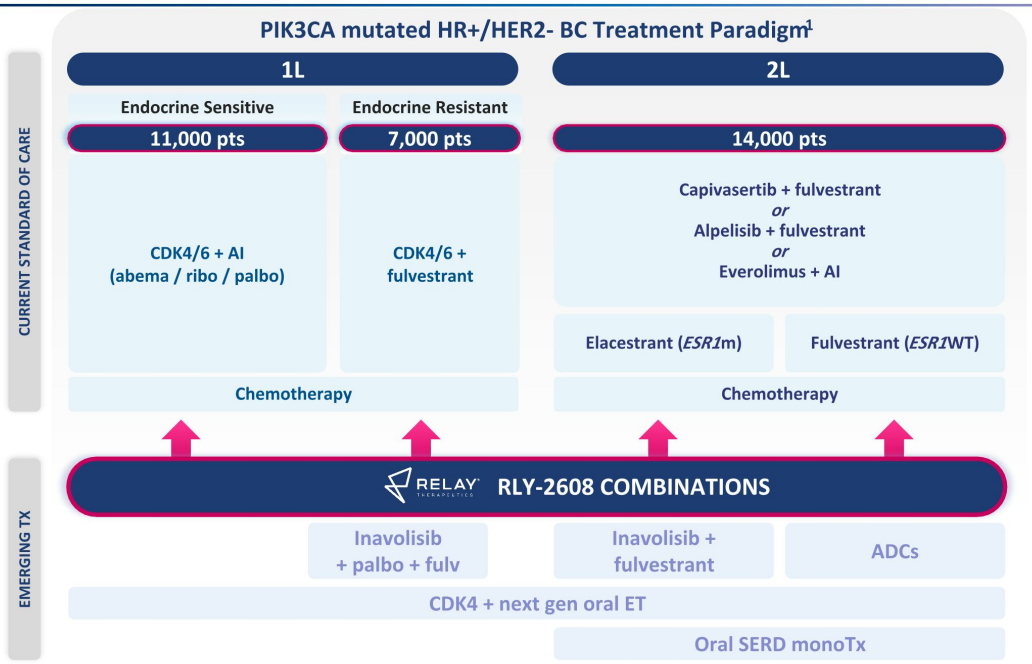
Landmark PFS analysis

Triplet:
Early safety and tolerability data

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	8.0mo, 6.2mo[4]	7.3mo, 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 #PS-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

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		  • Doublet safety & efficacy data
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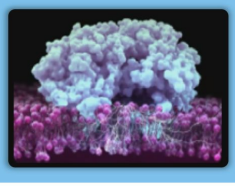
** Genentech controls data disclosures*

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 (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

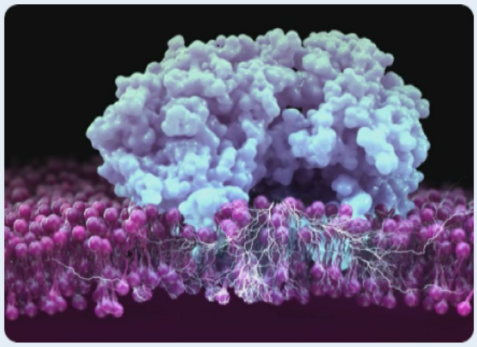
1. Prevalent US patient population with a PIK3CA mutation in adjuvant, first line metastatic and second line metastatic settings (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalence of vascular malformations with a PIK3CA mutation (Gallagher 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)

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**

Dr. Adrienne Hammill, MD PhD
Cincinnati Children's
changing the outcome together

- 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
Cincinnati Children's
changing the outcome together

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

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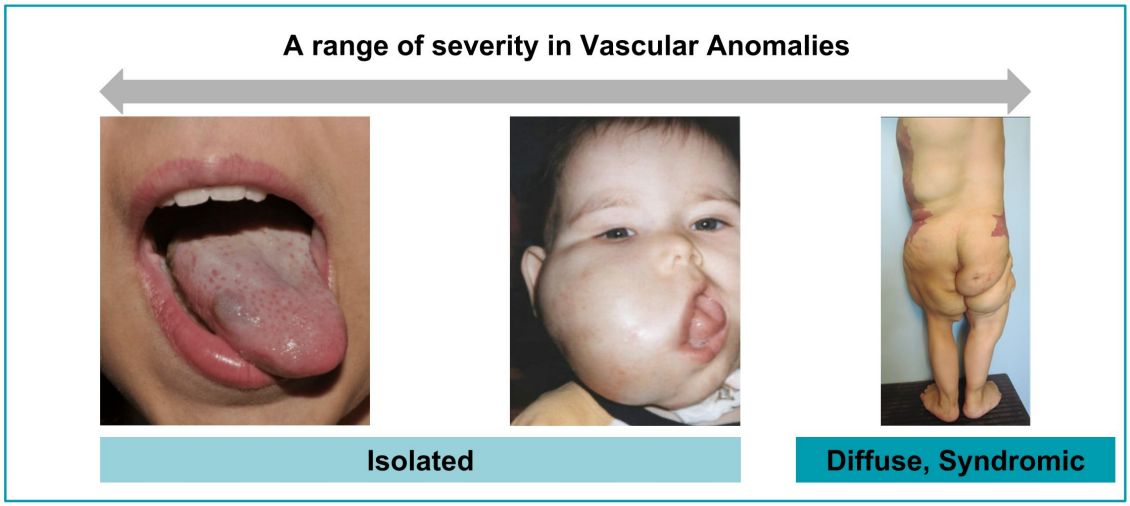
Simple	**Combined °**	**of major named vessels**	**associated with other anomalies**
Capillary malformations Lymphatic malformations Venous malformations Arteriovenous malformations* Arteriovenous fistula*	CVM, CLM LVM, CLVM CAVM* CLAVM* others	See details	See list

Dr. Adrienne Hammill, MD PhD
Cincinnati Children's
changing the outcome together

- **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"**



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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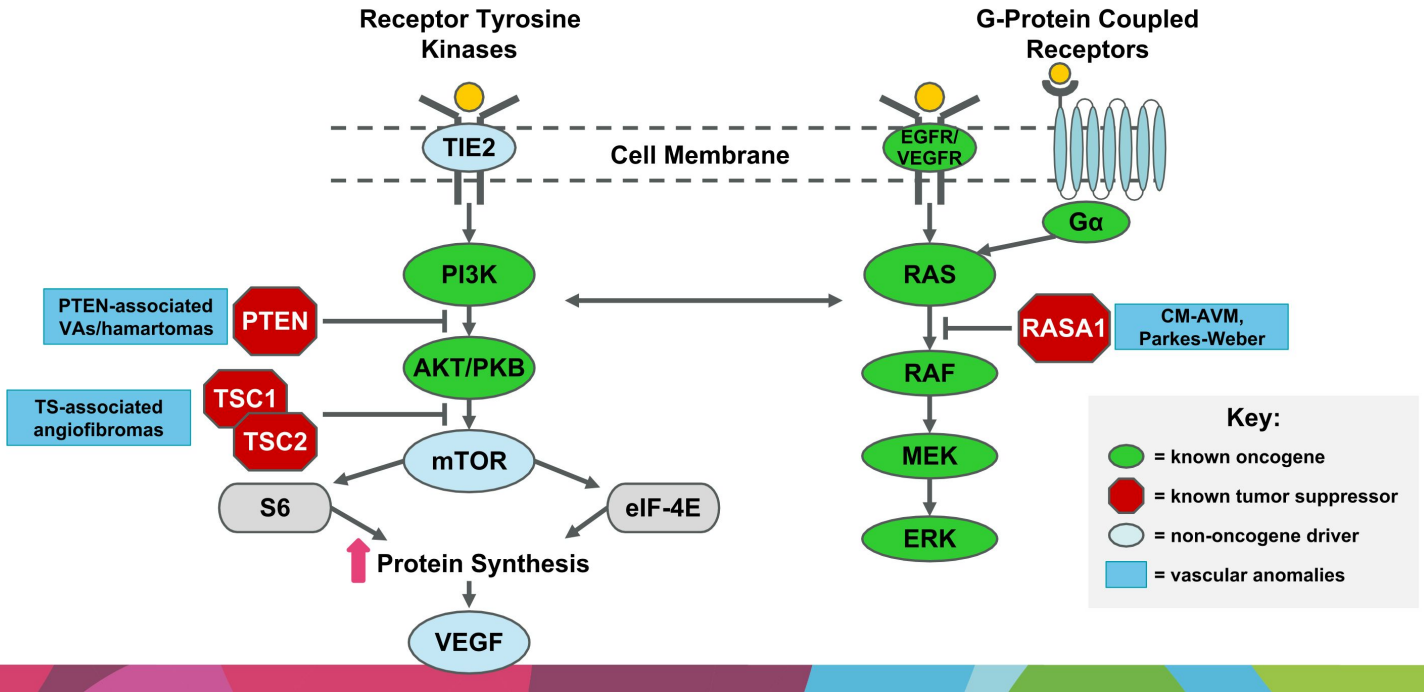

Genetic Causes of Vascular Malformations – Somatic Mutations

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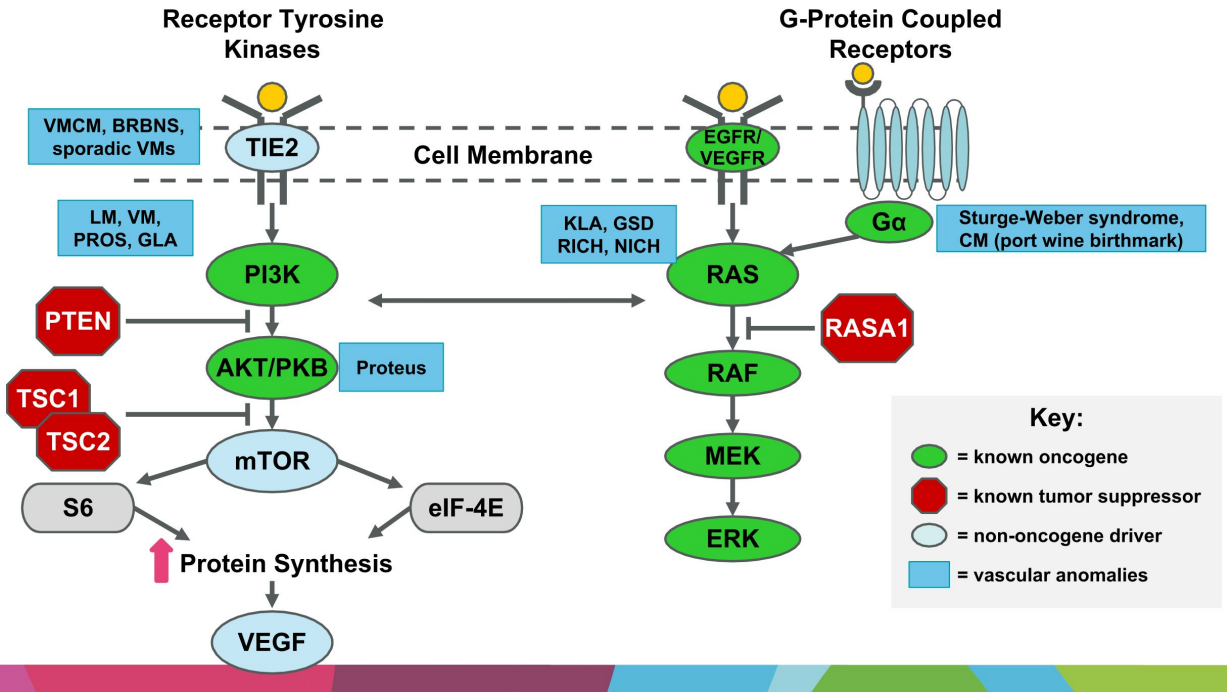

Genetic Causes of Vascular Malformations

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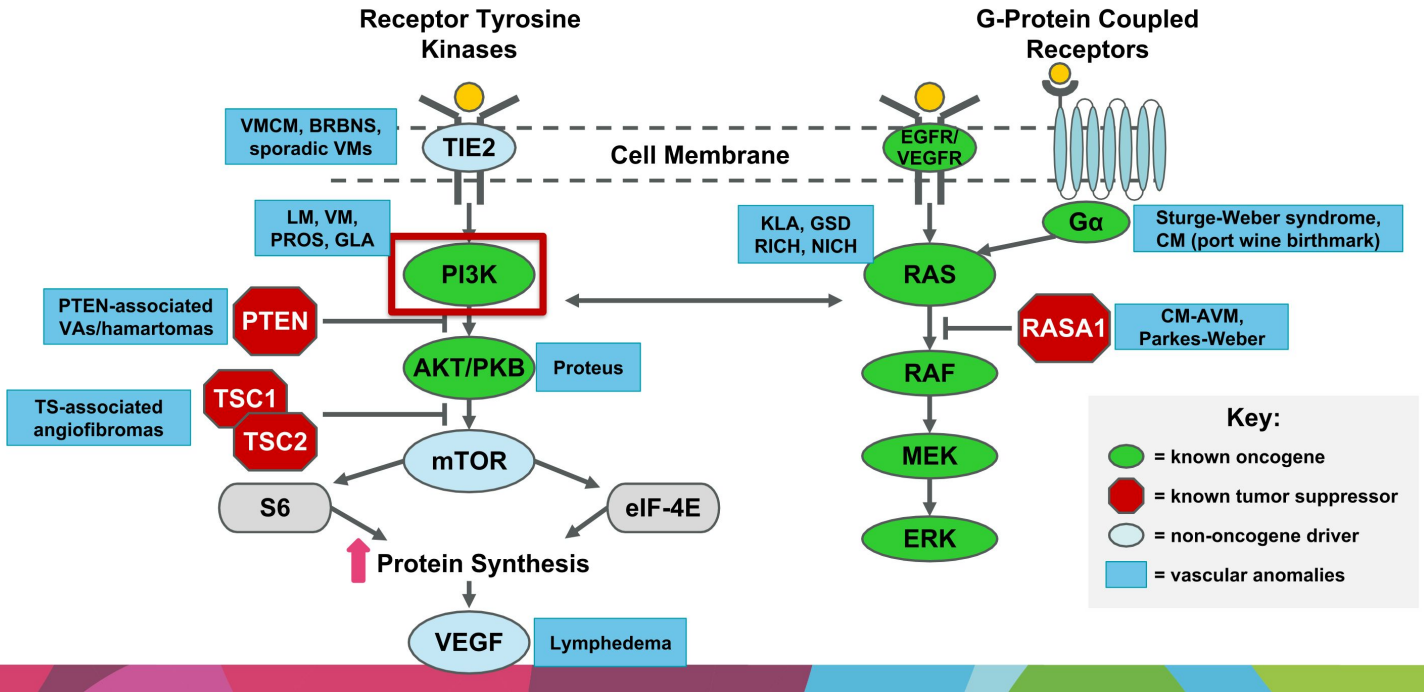

PIK3CA-related Overgrowth Spectrum (PROS) Phenotypes

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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

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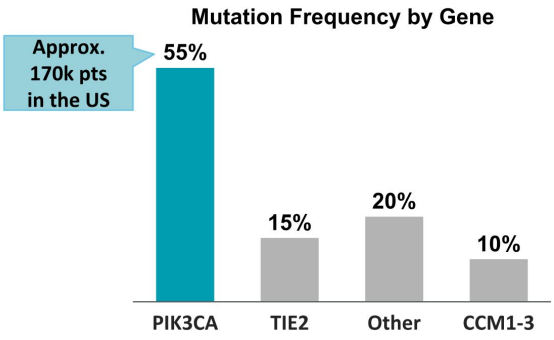
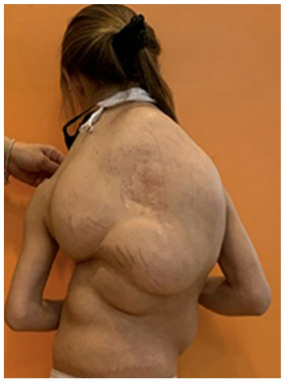
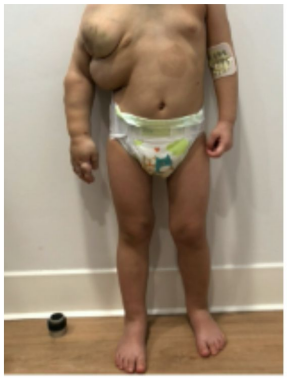
~300k US patients affected by Vascular Malformations, driven by prenatal somatic mutations



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

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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

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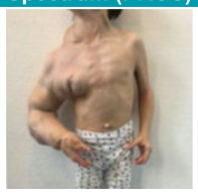
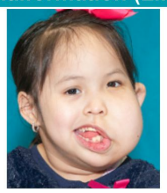
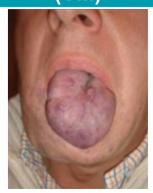
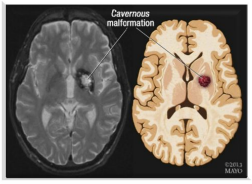
Vascular Malformation Types

	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

RELAY THERAPEUTICS

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

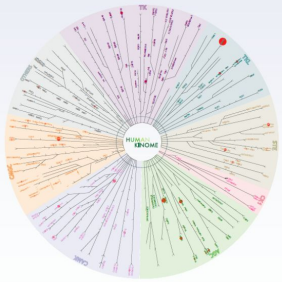
- Immunosuppressive
- Non-Selective
- Not approved in any Vascular Malformation types

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

PI3Kα-Driven Vascular Malformations – Relay Tx Mutant Selective Approach

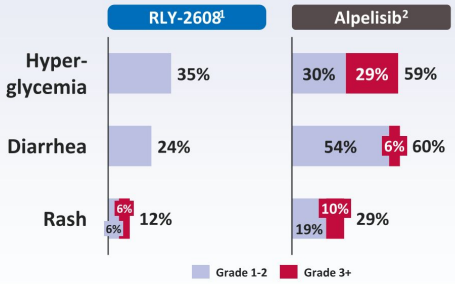
Favorable Selectivity

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

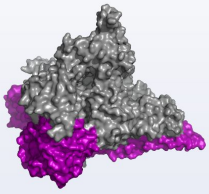
- *PIK3CA*-Related Overgrowth Spectrum (PROS)
- Lymphatic Malformations (LM)
- Venous Malformations (VM)
- Cerebral Cavernous Malformations (CCM)

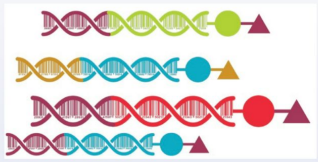
1. Interim AE rates and ctDNA for RLY-2608 part of ongoing ReDiscover trial studying solid tumors (oncology), data as of 07/24/23; 2. Alpelisib AE rates from BYLieve study (oncology), Rugo 2021 Lancet Oncol 22:489

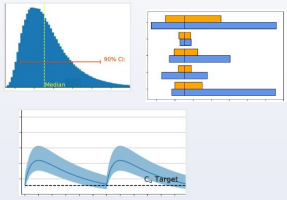
PI3Kα – Dynamo™: Integration of Experimental and Computational Tools

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

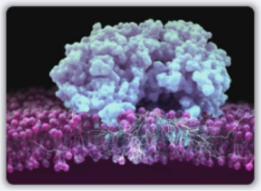
GENETIC DISEASE

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

	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

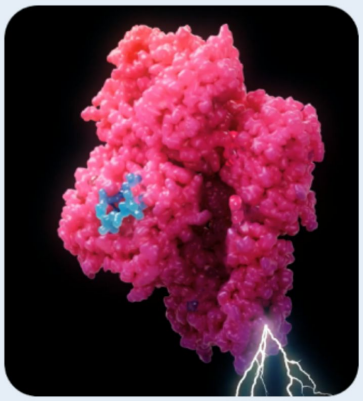
1. Prevalent US patient population with a PIK3CA mutation in adjuvant, first line metastatic and second line metastatic settings (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalence of vascular malformations with a PIK3CA mutation (Gallagher 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)

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

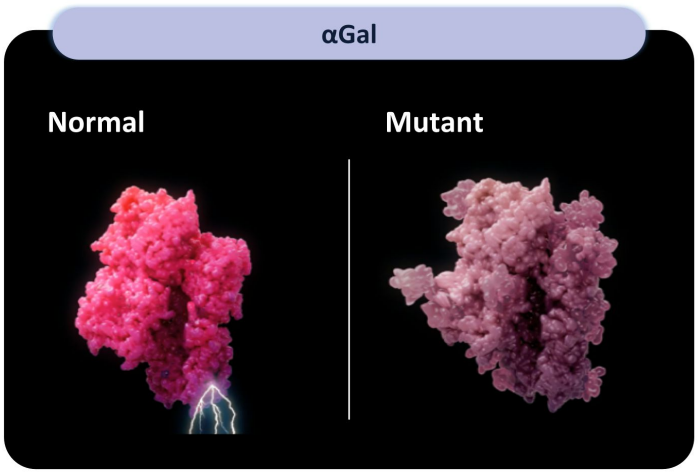
Current therapies have established a market but have key limitations

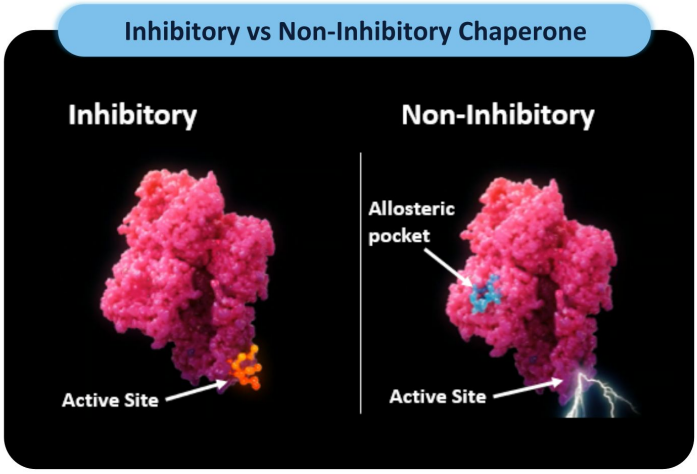
Current Therapies	Limitations of Inhibitory Chaperone
Enzyme Replacement Therapy (ERT, intravenous) ~$1.6B peak sales[1]	
Inhibitory Chaperone Therapy (migalastat) 40% of pts → ~$780M peak sales[2]	1. Limited αGal activation 2. Limited mutational coverage 3. Not combined with ERT

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

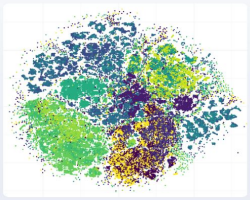
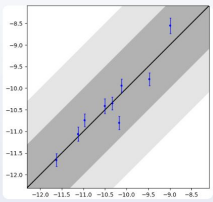
Fabry Disease – Dynamo™ Discovered a Novel Allosteric Pocket





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

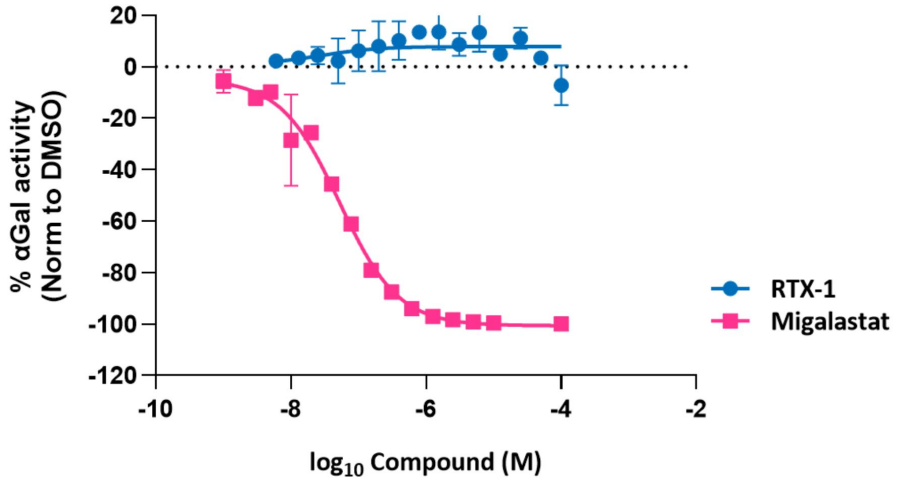
Platform Tool Examples

Experimental tool

Computational tool

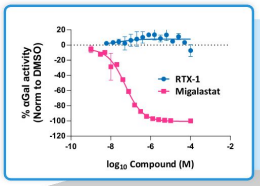
Fabry Disease – Relay Tx Compounds are Non-Inhibitory Chaperones

Migalastat inhibited αGal function while Relay Tx compounds did not



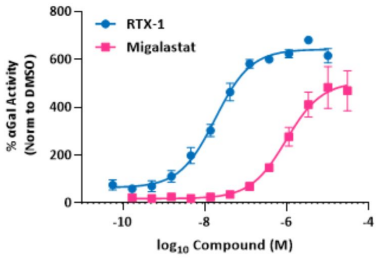
Note: Biochemical assay run with WT αGal; 1 hr incubation at room temperature

Fabry Disease – Potential Benefits of Non-Inhibitory Chaperone Approach

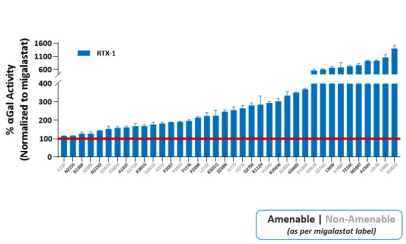


Relay Tx Solution:
Non-Inhibitory Chaperone to Stabilize Protein and Increase Activity

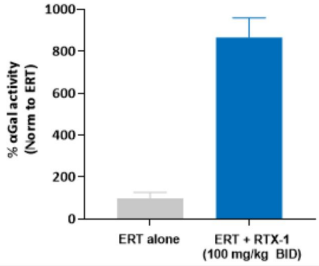
1 Superior αGal activation[1]



2 Broad mutational coverage[2]



3 Combinable with ERT[3]

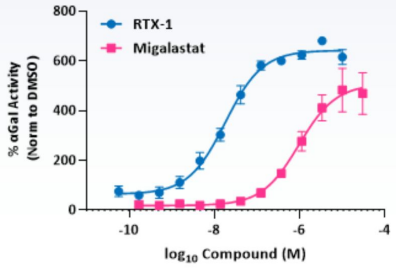


Notes: 1. R301Q mut αGal (2hr post compound washout, expressed in *GLA* KO HEK293 cells); In vitro αGal activity assay (4MU) across multiple GLA mutations expressed in HEK293 GLA KO cells (assessed at 1uM); 3. *GLA* KO mouse model, activity assessed following single dose of ERT and 14-day treatment with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

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

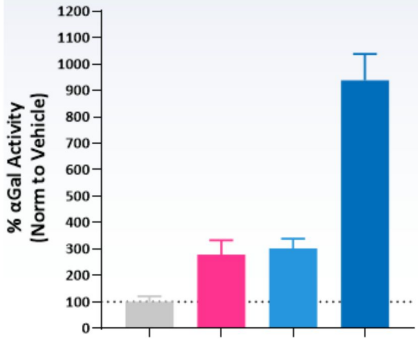
RTX-1 maintains higher levels of αGal activity *in vitro*

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



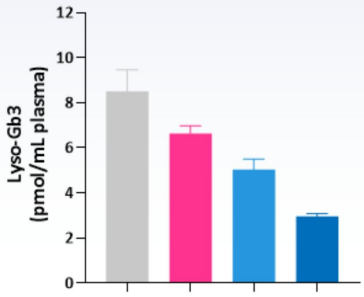
...which translates to greater *in vivo* kidney αGal activity

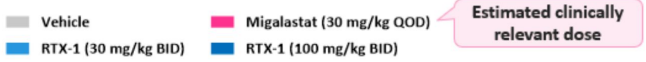
Activity levels measured at 28 days in humanized *GLA* R301Q mutant mouse model



... and greater substrate reduction

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

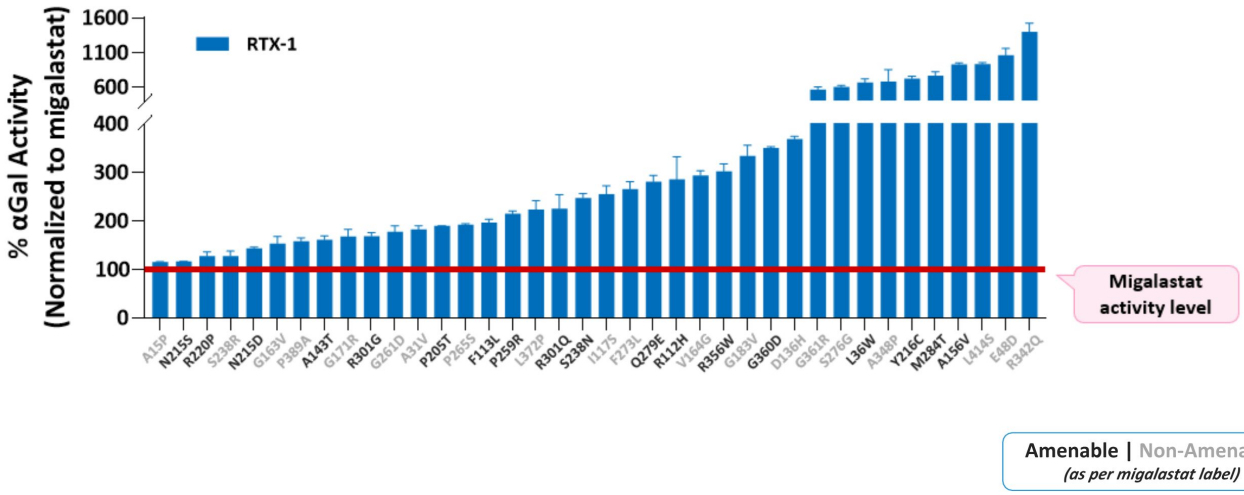




There were no adverse findings in an exploratory rat toxicology study of RTX-1 at exposures equivalent to 100 mg/kg BID

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**



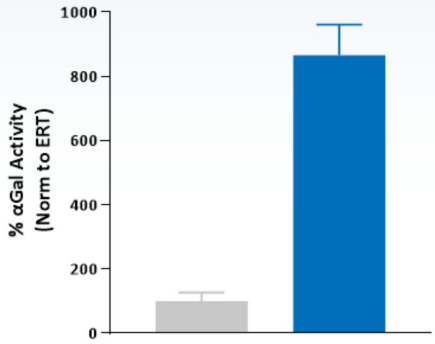
Amenable | Non-Amenable
(as per migalastat label)

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

Relay Tx Non-Inhibitory Chaperones Combinable with ERT

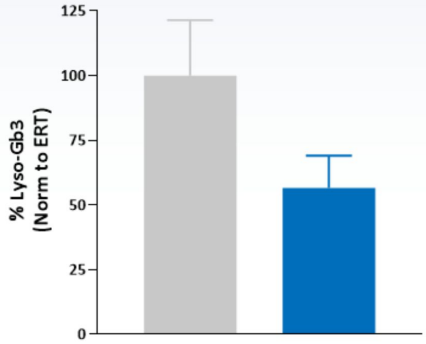
In vivo αGal activity

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

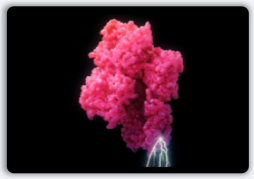
GENETIC DISEASE

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	Pfizer 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)

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]

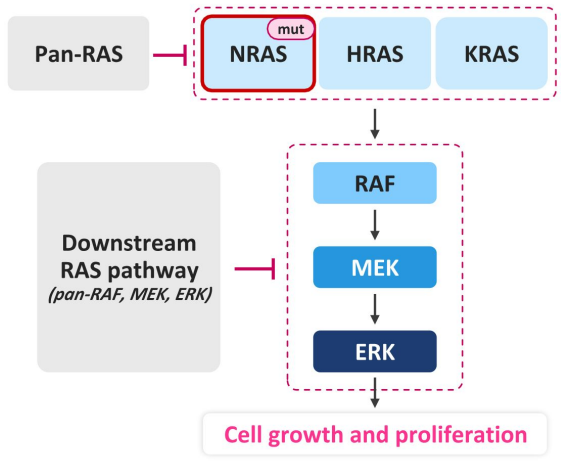
DYNAMO™ PLATFORM

First NRAS Selective Inhibitor

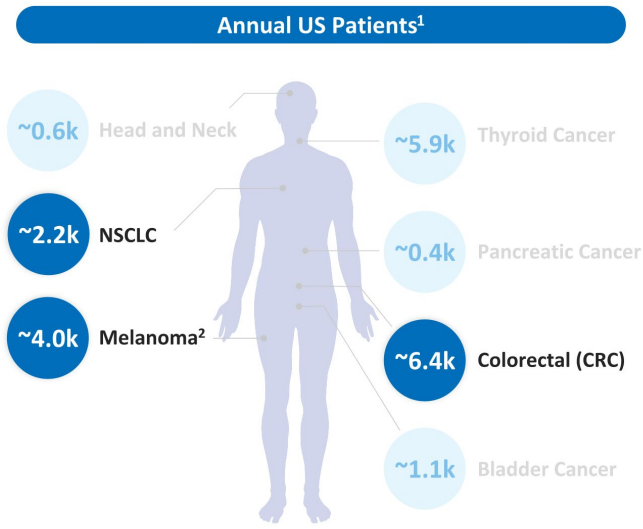
1. Newly diagnosed (incident) solid tumors with an NRAS mutation, excluding stage 0-II newly diagnosed melanoma patients (SEER, 3rd party source for alteration rate, Jan 2024)

NRAS – Large Validated Market With Significant Unmet Need

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

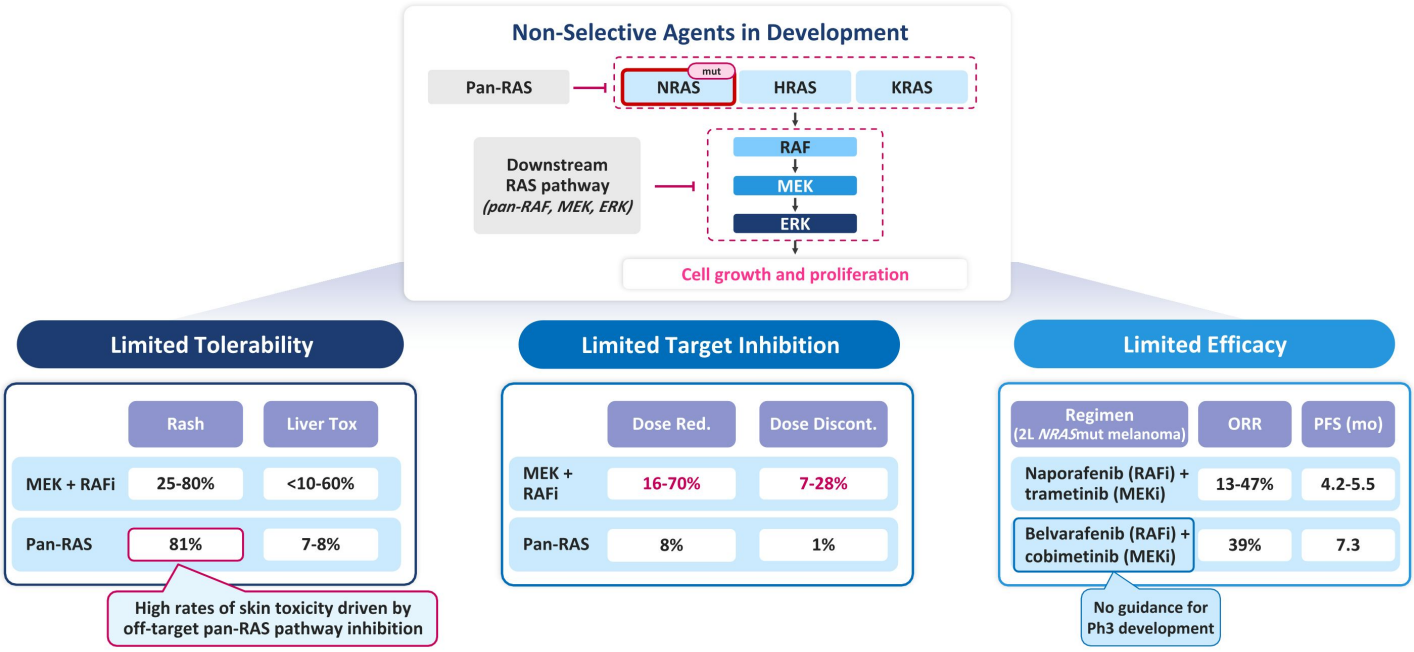


NRAS mutations observed in broad range of tumor types



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



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%

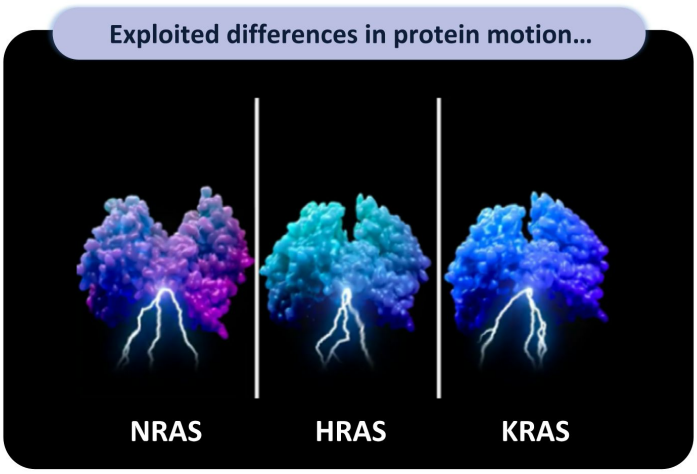
Limited Efficacy

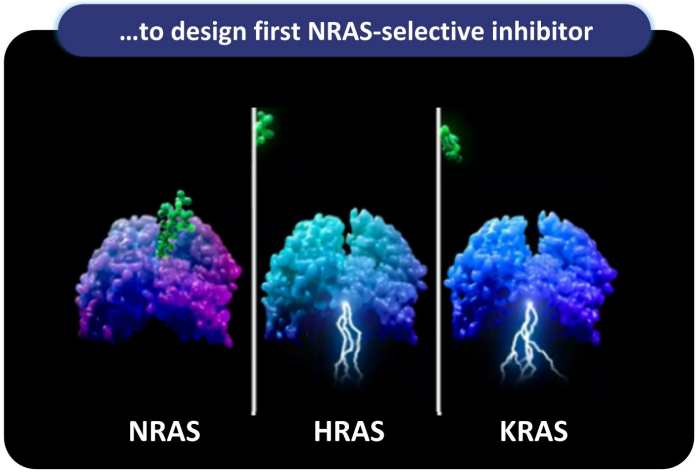
Regimen (2L *NRAS*mut melanoma)	ORR	PFS (mo)
Naporafenib (RAFi) + trametinib (MEKi)	13-47%	4.2-5.5
Belvarafenib (RAFi) + cobimetinib (MEKi)	39%	7.3

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

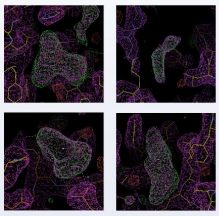


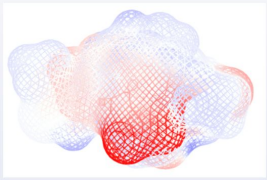


NRAS – Dynamo™: Integration of Experimental and Computational Tools

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

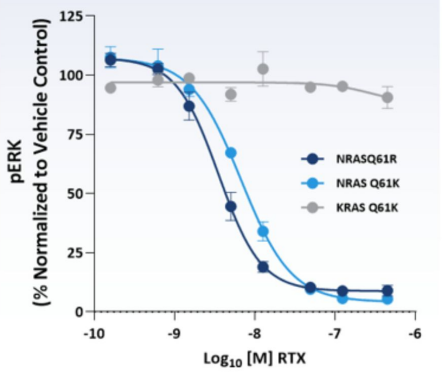
Computational tool

NRAS Inhibitors Are Potent, Selective & Active Across NRAS Mutations

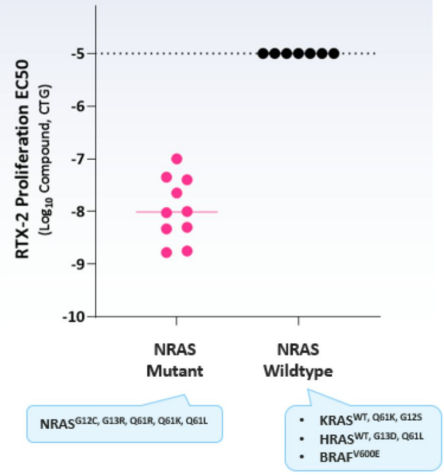
Relay Tx compounds bind to the ON-state with selectivity for NRAS[1]

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]

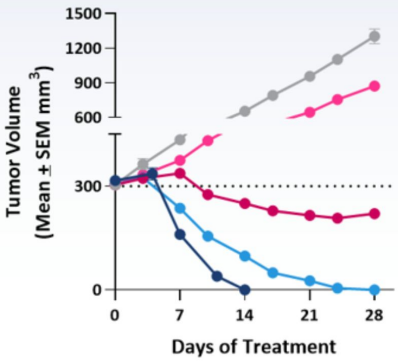


1. Based on SPR analysis of purified protein; 2. Based on pERK assay of SK-MEL-2, SK-MEL-30, and CALU-6 cell lines evaluated at 24hr timepoint; 3. Based on cell proliferation panel (17 cell lines) evaluated at 3-5d timepoint depending on cell line

NRAS Inhibitors Achieve Complete Regression at Well Tolerated Doses

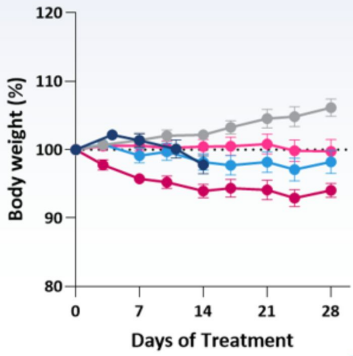
Relay Tx's NRAS inhibitors drove rapid, complete regressions...

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)**

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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)

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	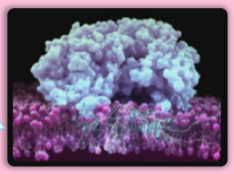	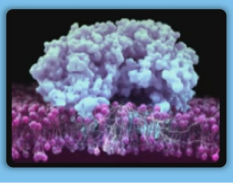		
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Milestones	CDK4i clinical start by YE 2024	Clinical start in 1Q 2025	Clinical start in 2H 2025	Clinical start in 2H 2025

Ongoing mono, doublet, triplet; Data update YE24

$2B current market[5]

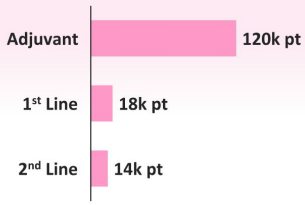
1. Prevalent US patient population with a PIK3CA mutation in adjuvant, first line metastatic and second line metastatic settings (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalence of vascular malformations with a PIK3CA mutation (Gallagher 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]

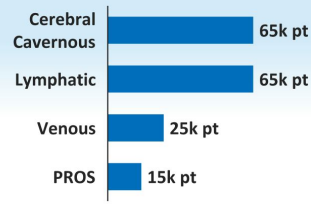
RLY-2608



PIK3CA mutant Vascular Malformations

~170k Patients
(US prevalence)[2]

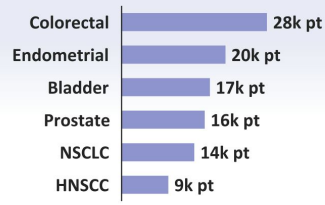
Potential for rapid POC with RLY-2608, then distinct molecule for pivotal



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

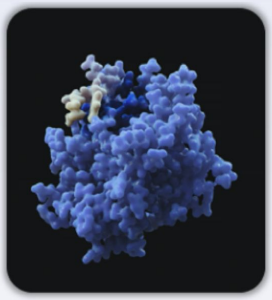
1. Prevalent US patient population with a PIK3CA mutation in each line of therapy (Global Data HR+/HER2- Breast Cancer Global Forecast, November 2023; 3rd party source for alteration rate); 2. Prevalent US patient population of vascular malformation patients with a PIK3CA mutation (multiple sources);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

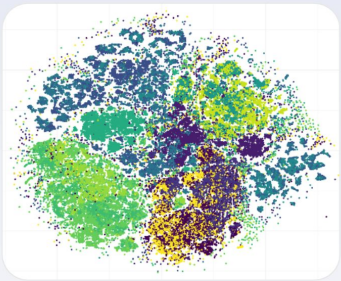
Dynamo™ Platform

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

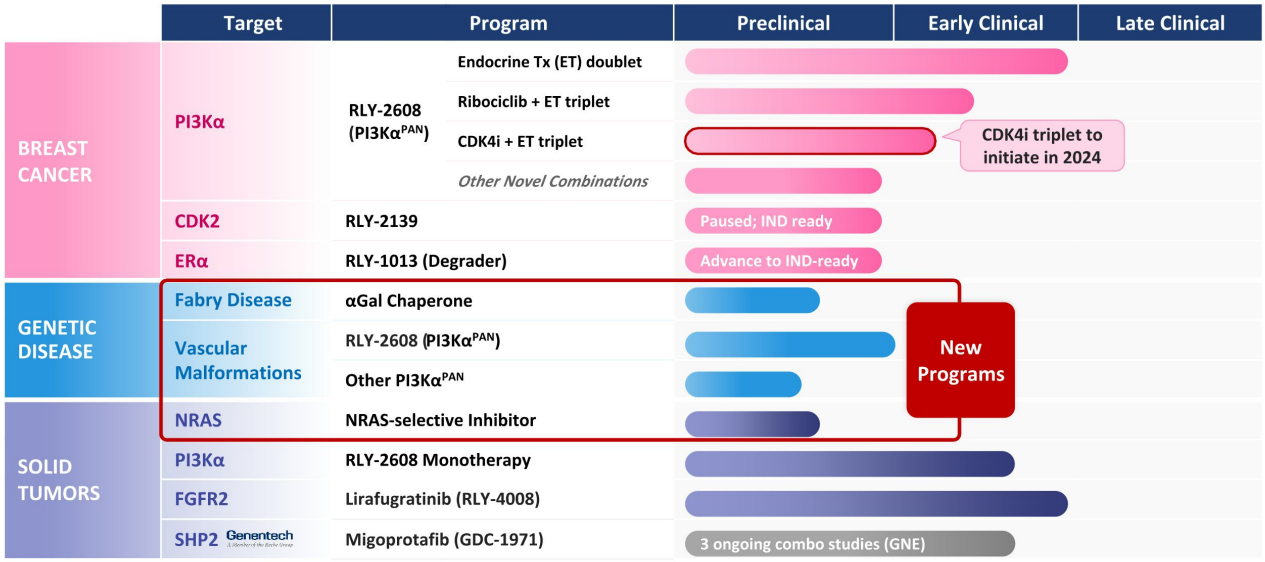
PEOPLE

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