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Roche Pharma Day 2026

London, 28 September 2026

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Welcome

Bruno Eschli
Head of Investor Relations

Agenda

	Introduction	
	09:30 BST	Bruno Eschli, Head of Investor Relations
Strategy	**Pharma Strategy and Commercial Growth Drivers**	
		Teresa Graham, CEO Roche Pharmaceuticals
	R&D Excellence	
		Levi Garraway, CMO and Global Head of Product Development
	Revolutionizing drug R&D with Lab-in-a-Loop	
		Aviv Regev, Head of Genentech Research and Early Development (gRED)
	Financial Excellence and Digitalization	
		Alan Hippe, CFO Roche Group
	11:15-11:40	**Q&A – Strategy**
	11:40-12:30	**Lunch Break**
Pipeline	**Oncology/Hematology**	
	12:30 BST	Levi Garraway & Matt Hellman, SVP and Global Head of Oncology and Hematology Product Development
	Neurology	
		Hideki Garren, SVP and Global Head of Neurology Product Development
	Immunology	
		Larry Tsai, SVP and Global Head of Immunology Product Development
	Ophthalmology	
		Christopher Brittain, SVP and Global Head of Ophthalmology Product Development
	Cardiovascular, Renal and Metabolism	
		Manu Chakravarthy, SVP and Global Head of Cardiovascular, Renal and Metabolism Product Development
	14:10-14:40	**Q&A - Pipeline**
	14:40-15:00	**Buffet reception**

Pharma Strategy and Commercial Growth Drivers

Teresa Graham
CEO Roche Pharmaceuticals

Progress since Pharma Day 2025

Our strategy and capabilities

Our commercialization excellence

Future growth opportunities

Significant progress made since Pharma Day 2025

Continued strong operational efficiency, supporting delivery of the next wave of innovation

Strong financial performance[1]

+6% HY 26 sales growth

+10% HY 26 COP growth

+2.0%p HY 26 COP margin growth

+11% HY 26 operating free cash flow growth

Positive Ph III readouts

giredestrant	Adj. ER+/HER2- eBC
giredestrant	Post CDKi ER+/HER2- mBC
divarasib	2L+ KRAS G12C+ NSCLC
Lunsumio	2L+ FL
fenebrutinib	RMS
fenebrutinib	PPMS
Enspryng	MOGAD
Gazyva	SLE
Gazyva	MN
sefaxersen[2]	IgAN

✓ = Positive Ph III readout

Significant pipeline progress

HER2 TKI[3]	HER2+ BC
tambotatug pelitecan	Lung cancer
bexobrutideg[4]	CLL
CD-20 Brainshuttle™	MS
NLRP3 inhibitors	AD, PD, CAD
VEGF x IL6 DutaFab	DME
petrelintide[5]	Obesity ± T2D
CT-996	Obesity ± T2D
enicepatide + petrelintide	Obesity ± T2D

» = Moving to Ph II »» = Moving to Ph III

Oncology/Hematology *Neurology* *Immunology* *Ophthalmology* *CVRM*

1. At CER: Constant exchange rates (avg. full year 2025); 2. Positive Ph III interim data, study ongoing, in collaboration with Ionis; 3. Currently in Ph II/III study; 4. In collaboration with Nurix Therapeutics; 5. In collaboration with Zealand Pharma; COP: Core operating profit

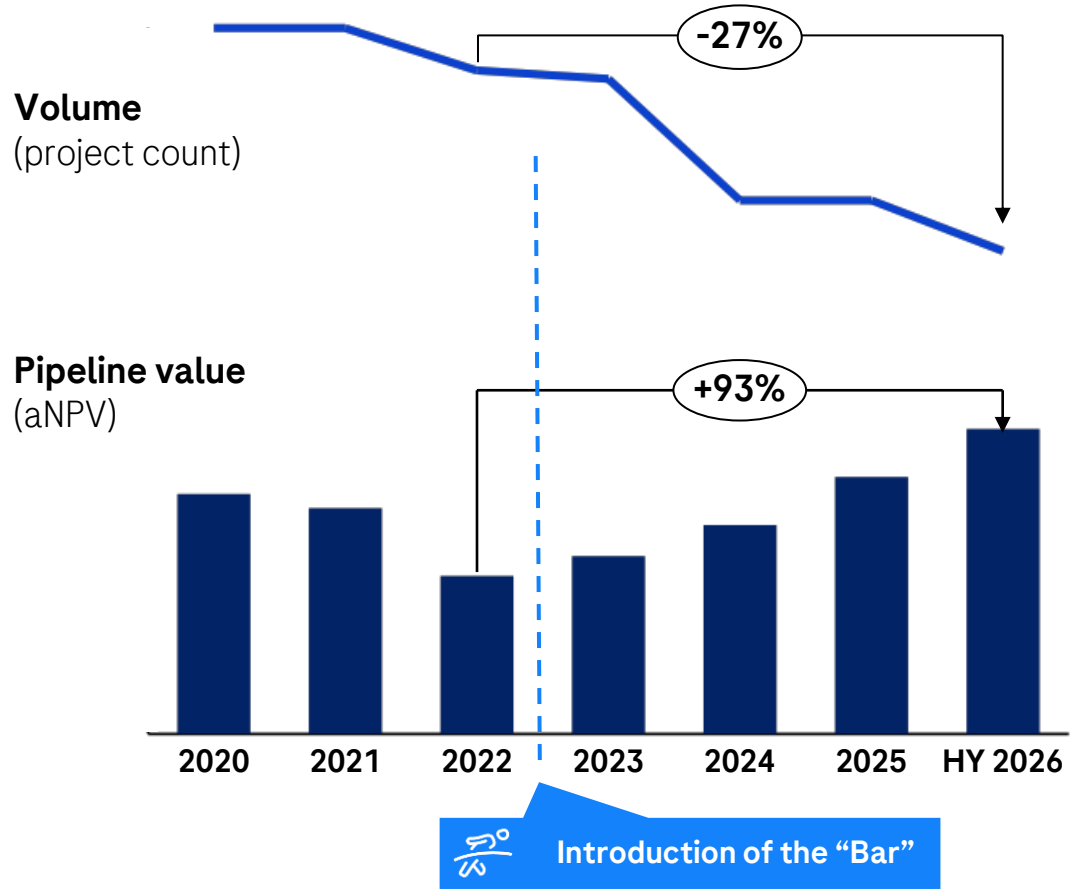
Substantial progress on Pharma R&D Excellence achieved

R&D Excellence and strategic M&A driving pipeline value to all-time high

Achievements[1]

- Introduction of the "Bar": Portfolio value +93% since YE 2022
- Improvement of the Ph3 success rate: 65% in 2025, >80% in 2026 YTD
- Shortened R&D cycle times by ~19 months until YE 2026[2] vs. YE 2023
- Re-allocation of R&D resources with CHF ~1.6 bn savings reinvested since YE 2023
- Increased early stage research productivity with LI GOs growing to 104 at YE '25 vs. 51 YE '22
- Disciplined BD complementing in-house R&D

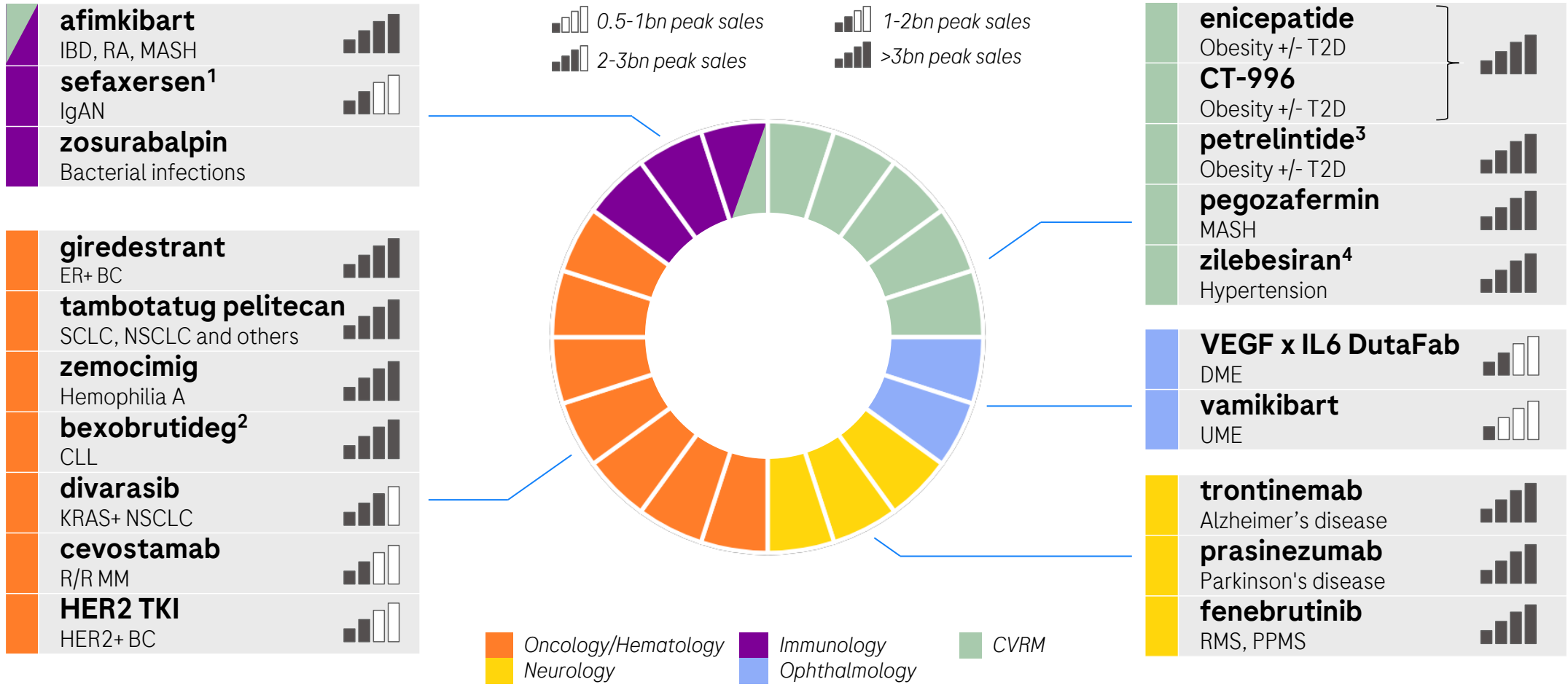
Pipeline evolution



1. As of HY 2026; 2. From Lead Identification to end of Phase 3; aNPV: adjusted net present value; LI Go: Lead identification Go

Up to 20 NMEs with launch potential by 2030

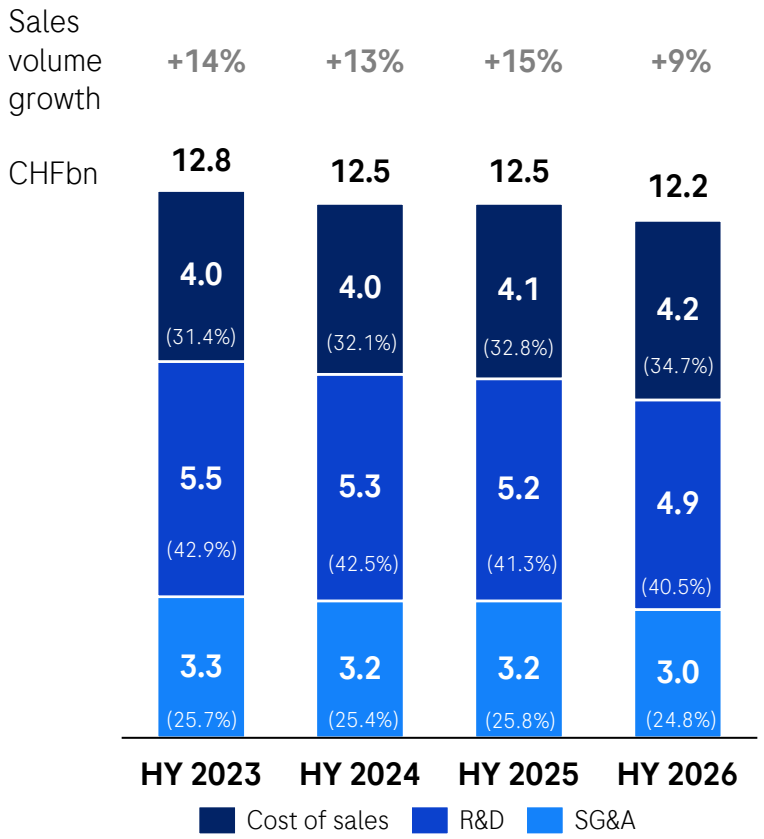
Two recent additions: Tam-Peli in SCLC and NSCLC, and VEGF x IL6 DutaFab in DME



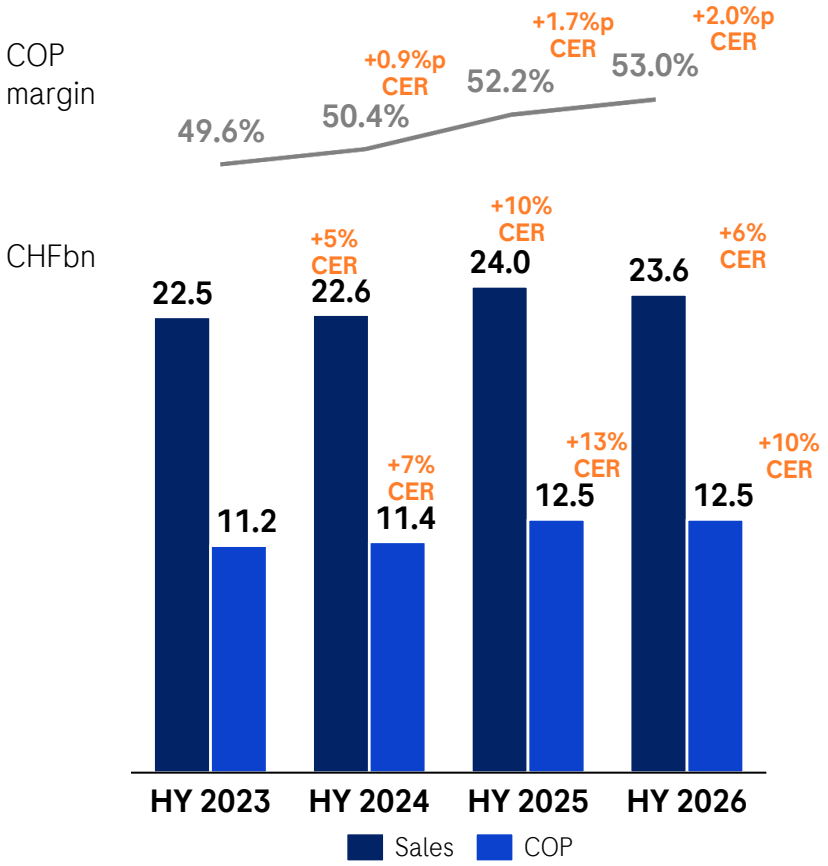
Peak sales shown unadjusted; 1. In collaboration with Ionis; 2. In collaboration with Nurix Therapeutics; 3. In collaboration with Zealand Pharma; 4. In collaboration with Alnylam; CVRM: Cardiovascular, renal and metabolism; NME: New molecular entity

Efficiency gains & cost control driving Pharma COP margin improvement

HY Pharma OPEX development (% of OPEX)



HY Pharma sales & profit development

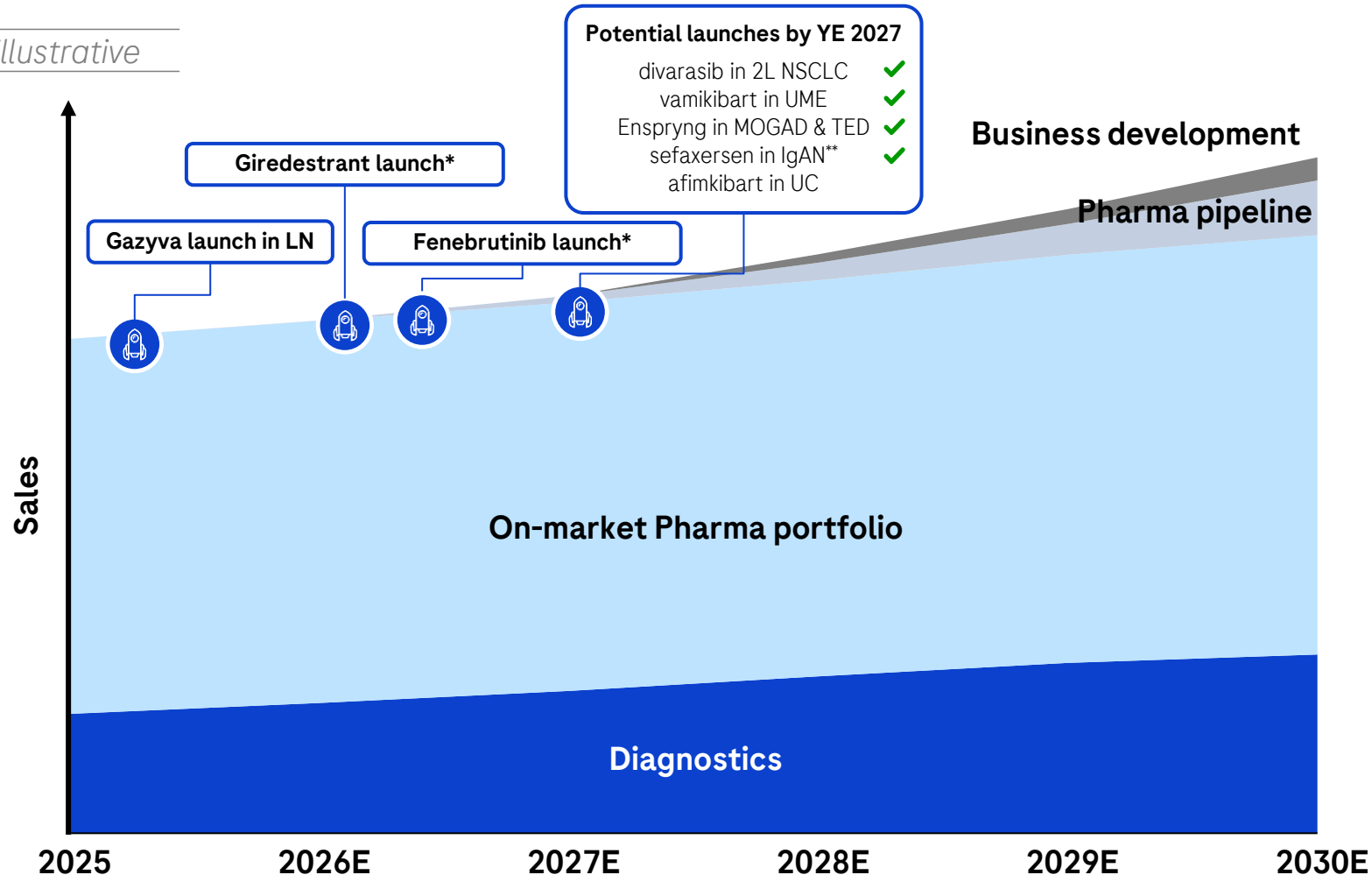


Note: Totals & subtotals may not add up due to rounding. Absolutes in CHF bn. Growth rates at CER; CER: Constant exchange rates of given year; COP: Core operating profit; OPEX: Operating expenditure; SG&A: Selling, general and administrative expenses

A strong base to deliver long-lasting future growth

Year end 2025 pipeline successes provide a clear path for sustained and strong growth beyond 2030

illustrative



- BD to further accelerate pipeline growth
- Late-stage pipeline with major readouts in new diseases from 2027 to 2030 including IBD, AD, MASH, PD, obesity and uHTN
- On-market Pharma portfolio (including Gazyva in immunology, giredestrant and fenebrutinib) to deliver strong growth overcompensating for any generic erosion
- No patent cliff ahead
- Pharma COP margin at least stable
- Diagnostics growth driven by on-market portfolio and key launches
- Unique blockbuster opportunities with SBX sequencing, mass spec and CGM
- Mid- to high-single digit sales growth
- COP growth ahead of sales growth

✓ Primary endpoint met

*Pending regulatory approval; **Positive Ph III interim data, study ongoing; Note: Graph is purely conceptual to outline portfolio trends; AD: Alzheimer's disease; CGM: Continuous glucose monitor; COP: Core operating profit; IBD: Inflammatory bowel disease; MASH: Metabolic dysfunction-associated steatohepatitis; PD: Parkinson's disease; SBX: Sequencing by expansion; uHTN: Uncontrolled hypertension

Progress since Pharma Day 2025

Our strategy and capabilities

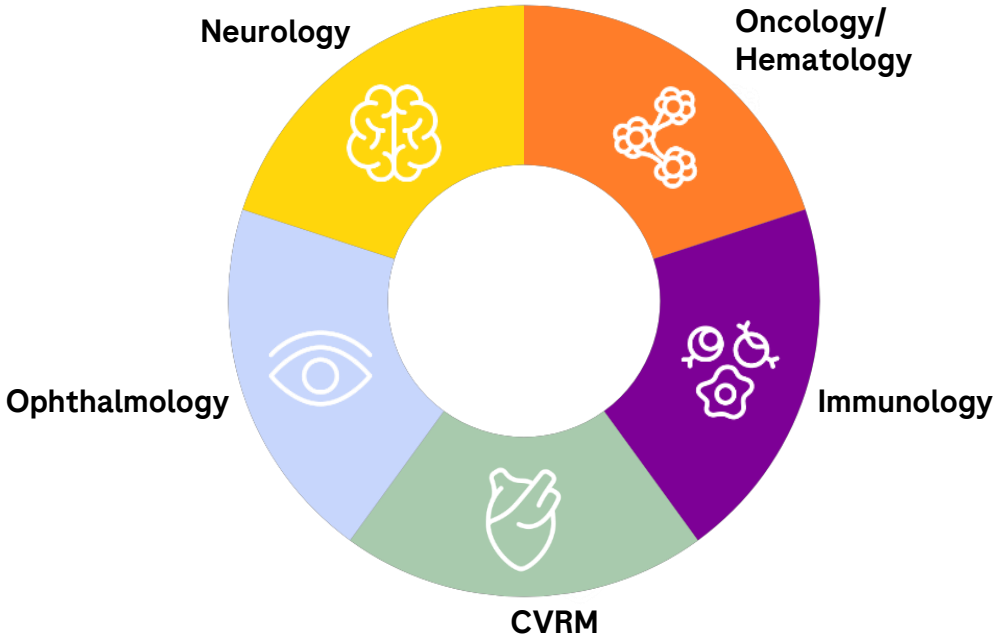
Our commercialization excellence

Future growth opportunities

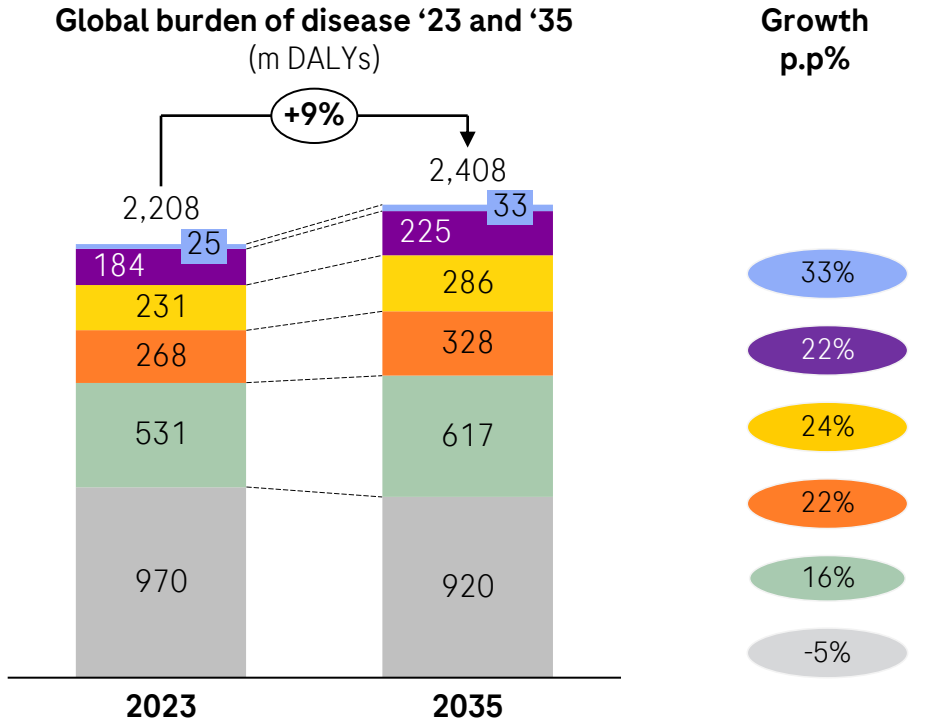
Committed to innovate in our five TAs

Providing clarity and intentional focus to leverage our scientific strengths and impact patients globally

Our five Therapeutic Areas



Covering 60% of total global burden of disease



Currently 11 disease areas where we invest E2E from discovery to commercial

CVRM: Cardiovascular, renal and metabolism; DALY: Disability-adjusted life years; E2E: End-to-end; TA: Therapeutic areas

Clear strategic focus across our five TAs

Recent pipeline successes driving updated Oncology/Hematology strategy

Updated

Oncology/Hematology	Neurology	Immunology	Ophthalmology	CVRM
Achieve leadership through transformative anchors	Extend #1 position in neurology post 2030	Maximize the value of our pipeline via multi-indication expansion	Establish a strong position in Ophthalmology by becoming #1 in retina	Secure a strong entry before 2030 with competitive products
Establish & expand position in key patient segments	Broaden portfolio and lead in preventive neurology	Leverage pan-immuno pathways to create value & cross-TA synergies	Broadly diversified and differentiated pipeline with transformational potential	Differentiate our offering by unique Roche capabilities
Secure position with next wave of innovations	Transform patient journey E2E via integrated Pharma + Dia approach	Pursue curative therapies for sustained remission	Expand portfolio beyond RVD into Dry eye disease and Glaucoma	Become a top 3 player in obesity

CVRM: Cardiovascular, renal and metabolism; E2E: End-to-end; RVD: Retinal vascular diseases; TA: Therapeutic areas

Core capabilities to succeed

Modalities & technologies



Focus on approaches with breakthrough potential in focus TAs & diseases

Devices



Making devices an integral part of our assets, from R&D to commercialization

Manufacturing



Optimizing and future-proofing our manufacturing network

Customer experience & access



Providing a holistic customer experience & enabling rapid, broad & sustainable access

Data & AI



Leveraging data and generative AI to improve process efficiency

Our People



Creating a culture that allows our people to thrive in our Pharma division

AI: Artificial intelligence; TA: Therapeutic area

Broad toolbox of modalities driving the next wave of innovation

Leveraging internal capabilities and external partnerships

- Small molecules
- Antibodies
- Bispecifics/ trispecifics
- Antibody-drug conjugate
- Peptides
- Cyclic peptides
- Fusion proteins
- Protein degraders
- RNA-targeting therapeutics
- Brainshuttle™
- Radioligand therapy
- Gene therapy
- Stem cell therapy
- Allogeneic CAR-Ts
- Neoantigen vaccines
- Port delivery platform

Pipeline deals and platform collaborations

Zion Pharma, Alnylam Pharmaceuticals, Telavant, CARMOT Therapeutics, HANSOH Pharma, C4 Therapeutics, ORIONIS BIOSCIENCES, DualityBio 映恩生物, MediLink Therapeutics, Monte Rosa Therapeutics, Innovent, POSEIDA THERAPEUTICS, ZEALAND PHARMA, 89bio, NURIX, Hanmi*

*Pending deal closure; CAR-T: Chimeric antigen receptor T- cell

Taking our global manufacturing network to the next level

Accelerating facility readiness through virtual validation of manufacturing designs before implementation

Next-gen. manufacturing platform to increase yields

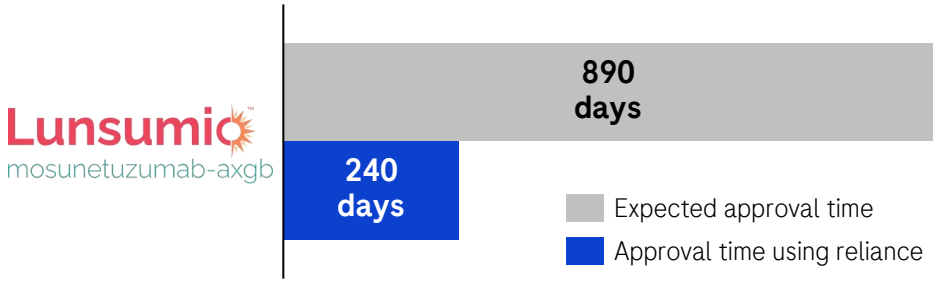
PERJETA® pertuzumab

5x yield increase

OCREVUS® ocrelizumab

2x yield increase

Regulatory innovation to accelerate medicines to patients[1]



Regulatory Reliance[2] pilots: Achieved simultaneous FDA & EMA manufacturing expansion approvals for Lunsumio on the same day.

Fully integrated digital twin

Integrated virtual model of the entire facility enables end-to-end testing of equipment, processes and system interactions before implementation



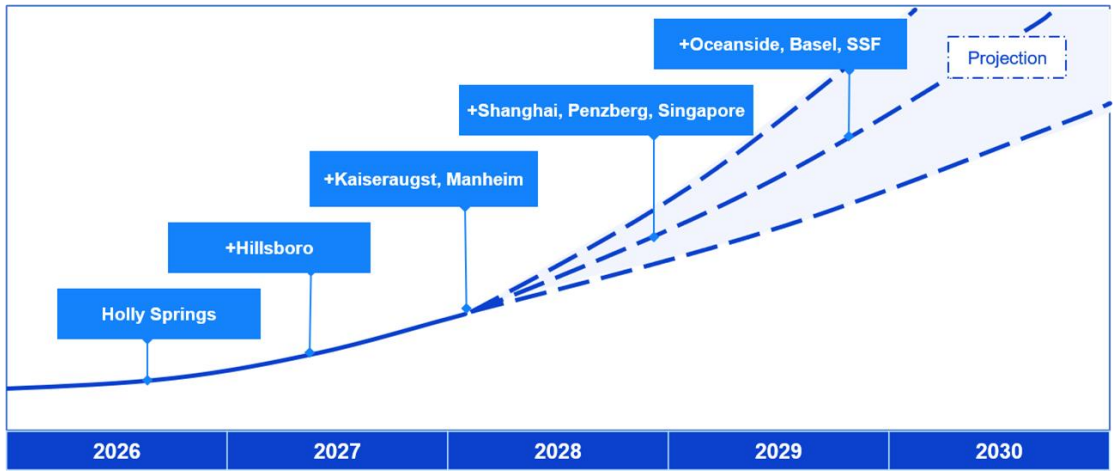
Optimizing manufacturing performance to improve capital efficiency and accelerate facility readiness

1. Approval Time Using Reliance (240 days) was calculated as an average of the time taken to get 80% approvals from participating health authorities in the pilot. 2. Reliance: the act whereby a regulatory authority in one jurisdiction takes into account, and gives significant weight to, assessments performed by another regulatory authority or trusted institution, or to any other authoritative information, in reaching its own decision. The relying authority remains independent, responsible and accountable for the decisions taken, even when it relies on the decisions, assessments and information of others. (WHO Good Regulatory Practices: guidelines for national regulatory authorities for medical products, 2021)

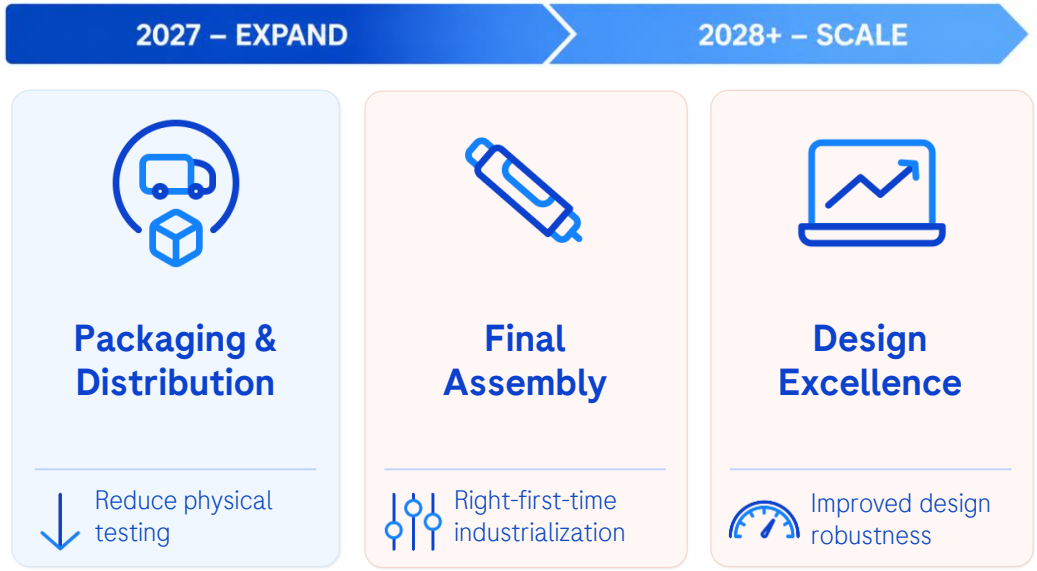
Scaling digital twin capabilities enterprise-wide

Unlocking value through repeatable deployment and expansion into device lifecycle innovation

Scaling digital twins enterprise-wide



A repeatable deployment approach brings new sites onboard faster and unlocks value across Roche

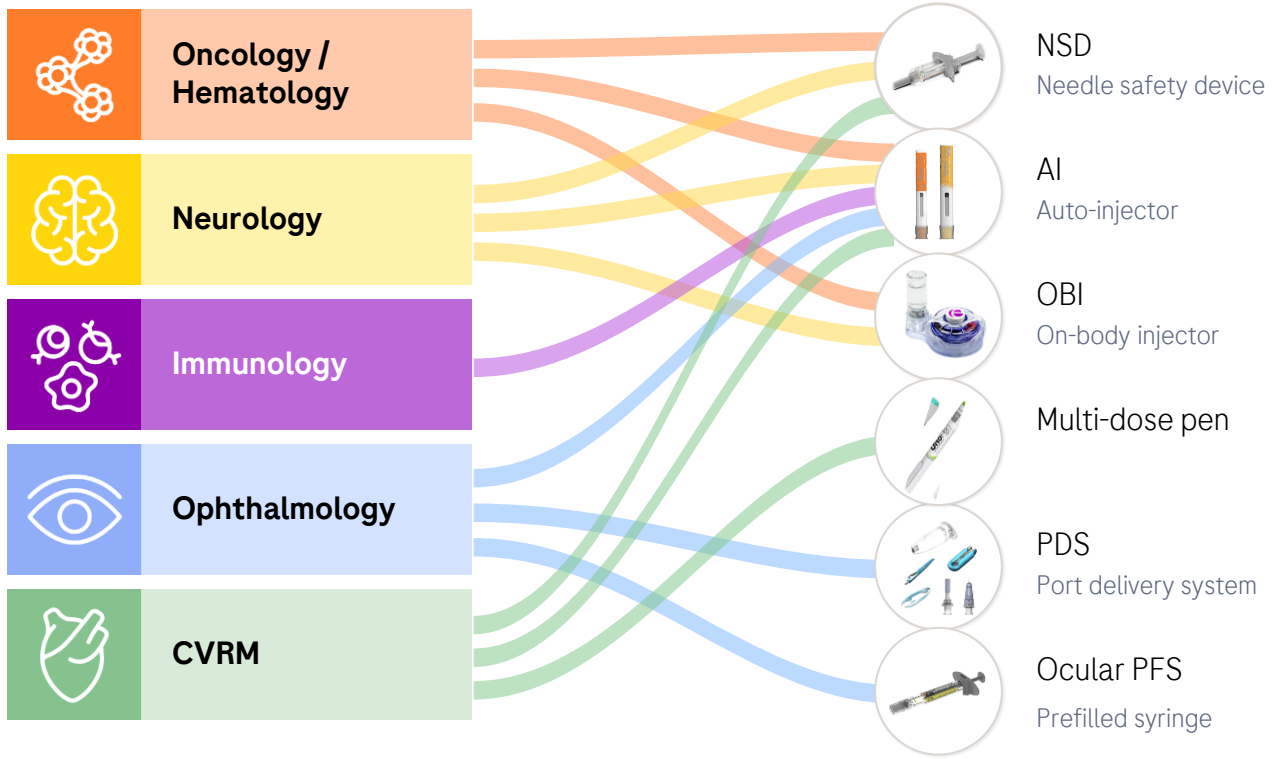


"Partnering with Roche brings end-to-end digital twins to the forefront of pharmaceutical manufacturing. Roche's deployment of NVIDIA Physical AI stack sets a new industry standard." - Kimberly Powell, Vice President / General Manager, Healthcare

Building and scaling for the portfolio of tomorrow

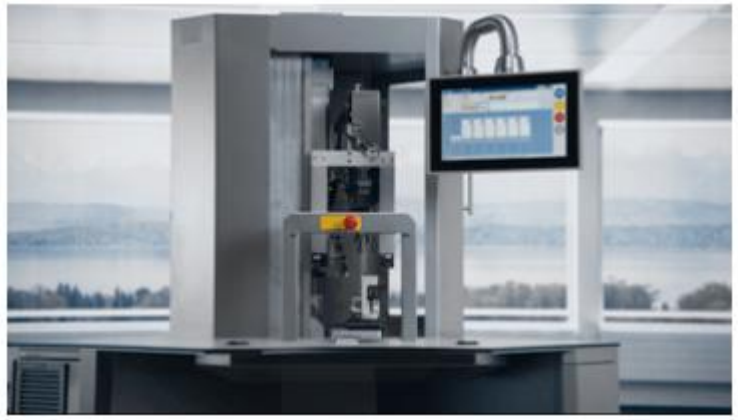
Novel device platforms & capacity investments enabling upcoming launches and improving patient experience

Device technology platforms



6 device platforms will cover **90%** of our anticipated future device needs

Device pilot plant in Basel



Global drug product manufacturing network



Digital excellence to enhance our marketing performance

Investing in AI-driven digital initiatives to drive speed, reduce costs, and increase effectiveness

Accelerated Go-To-Market

Our content engine[1] is driving efficiency in content creation:

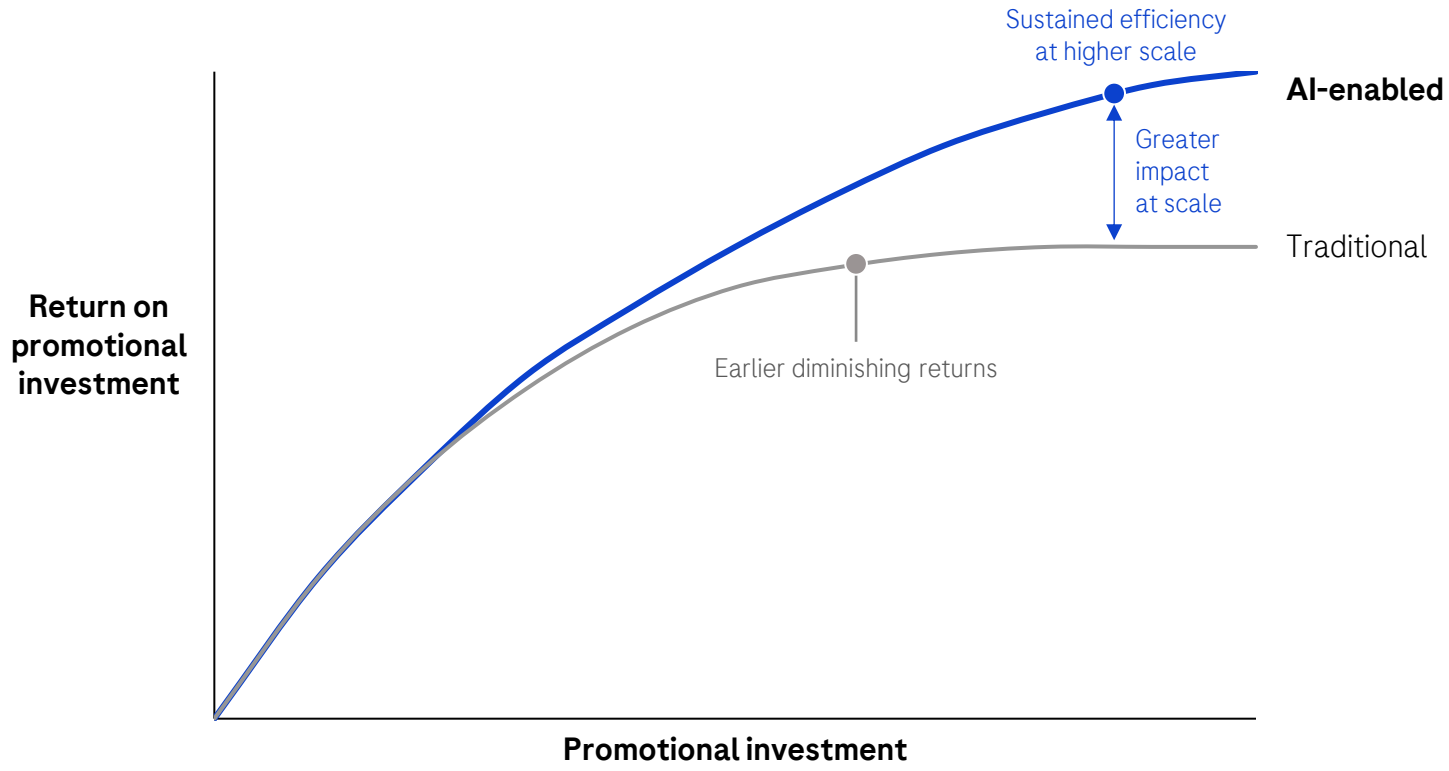
-50% Avg. content creation time

-83% Cost per asset

+7% Content volume output

leading to faster creation + approval cycles enabling highly personalized experiences

Increasing the return on promotional investments over time

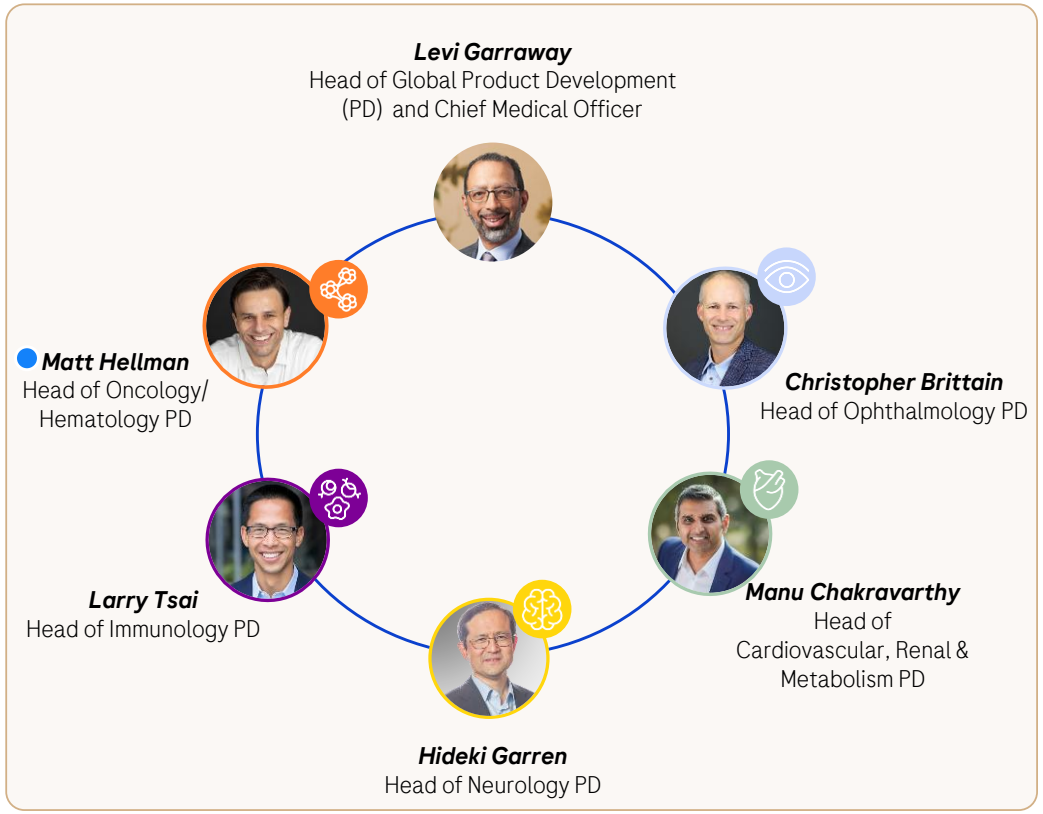


Incremental revenue driven by AI-enabled **M**arketing **A**utomation & **R**evenue **S**imulation (MARS) tool

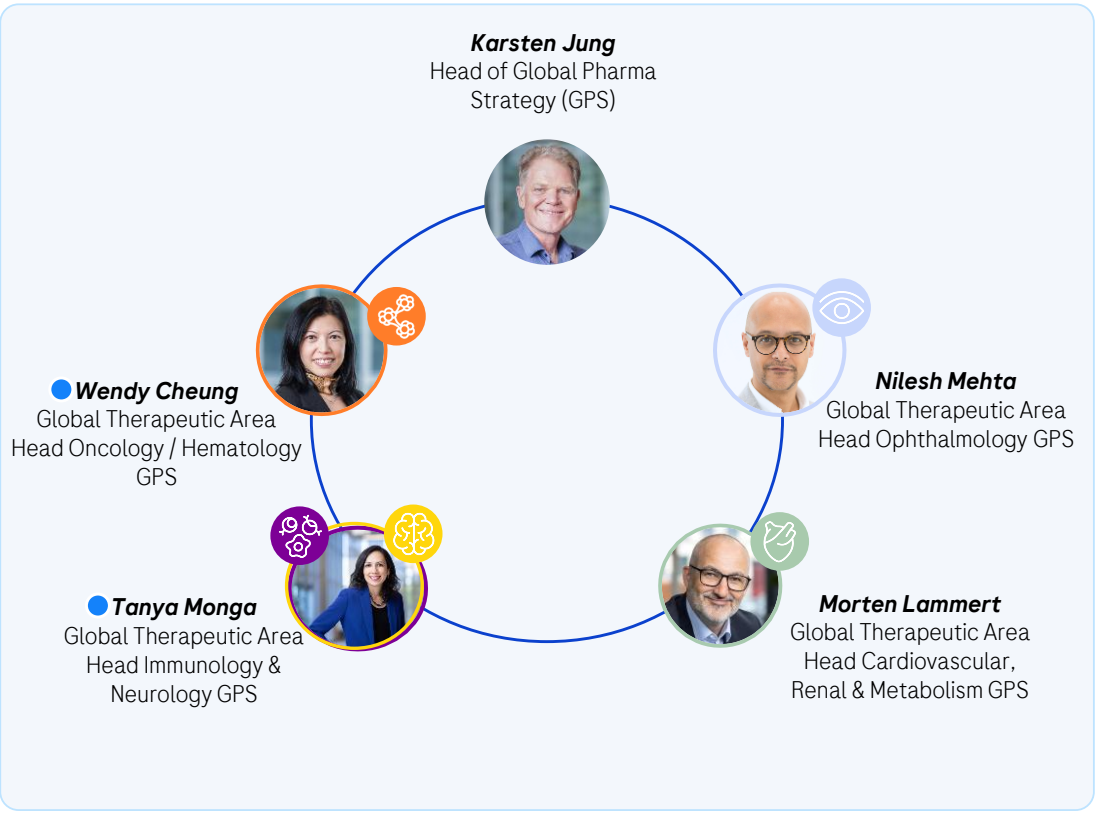
1. Content Engine currently live in the US market with global roll-out ongoing.

Strengthening our R&D and GPS teams

Product Development Therapeutic Area Heads



Global Pharma Strategy Therapeutic Area Heads



Strengthening our Capabilities in Early Research & Development



Mark Dawson
Head of Pharma Research and Early Development (pRED)



Saswata Talukdar
VP Head of CVRM Discovery, pRED



Kory Lavine
VP Head of CVRM Research, gRED



Louis Vermeulen
SVP Head of Oncology, pRED

● Key new appointments since Pharma Day 2025

Progress since Pharma Day 2025

Our strategy and capabilities

Our commercialization excellence

Future growth opportunities

Commercialization archetypes: Entrench, expand, establish

Leveraging our commercial execution and innovative pipeline to disrupt existing treatment paradigms

Entrench



Maximize leadership positions in key disease areas

- Line extensions into adjacent indications
- Devices to boost convenience & adherence
- Commercial execution & customer experience

Expand



Leveraging established disease areas by launching NMEs and expanding indications

- Build on in-depth scientific and medical knowledge & KOL networks
- Leverage market expertise to address further unmet needs
- Drive commercial synergies while broadening market access

Establish



Entering into disease areas new to Roche by reshaping the current SoC

- Capabilities and infrastructure building
- Market understanding & value proposition articulation
- Disease awareness & unmet need scoping

KOL: Key opinion leader; NME: New molecular entity; SoC: Standard of care

Portfolio mapping across the commercialization archetypes

Entrench

- Phesgo
- Tecentriq
- Itovebi
- Hemlibra
- Columvi
- Lunsumio
- Polivy
- Ocrevus
- Evrysdi
- Xolair
- Vabysmo
- Susvimo

Expand

- giredestrant
- divarasib
- tambotatug pelitecan
- HER2 TKI
- bexobrutideg
- cevostamab
- zemocimig
- fenebrutinib
- Elevidys
- Enspryng
- vamikibart
- VEGF x IL6 DutaFab

Establish

- trontinemab
- prasinezumab
- Gazyva
- sefaxersen
- afimkibart
- petrelintide
- enicepatide
- zilebesiran
- pegozafermin
- CT-996

Oncology/Hematology *Neurology* *Immunology* *Ophthalmology* *CVRM*

Entrench: Maximize potential by LEs, devices & commercial excellence

Entrench

- Phesgo
- Tecentriq
- Itovebi
- Hemlibra
- Columvi
- Lunsumio
- Polivy
- Ocrevus
- Evrysdi
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Establish

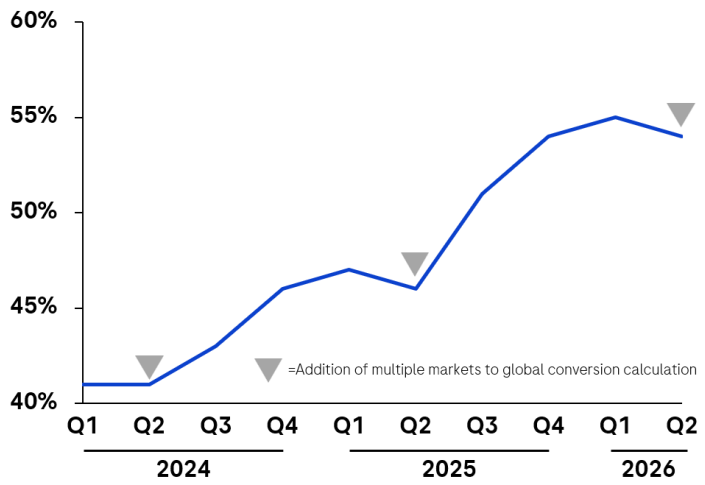
- trontinemab
- prasinezumab
- Gazyva
- sefaxersen
- afimkibart
- petrelintide
- enicepatide
- zilebesiran
- pegozafermin
- CT-996

Oncology/Hematology *Neurology* *Immunology* *Ophthalmology* *CVRM*

Breast cancer: Well-positioned to serve evolving needs in HER2+

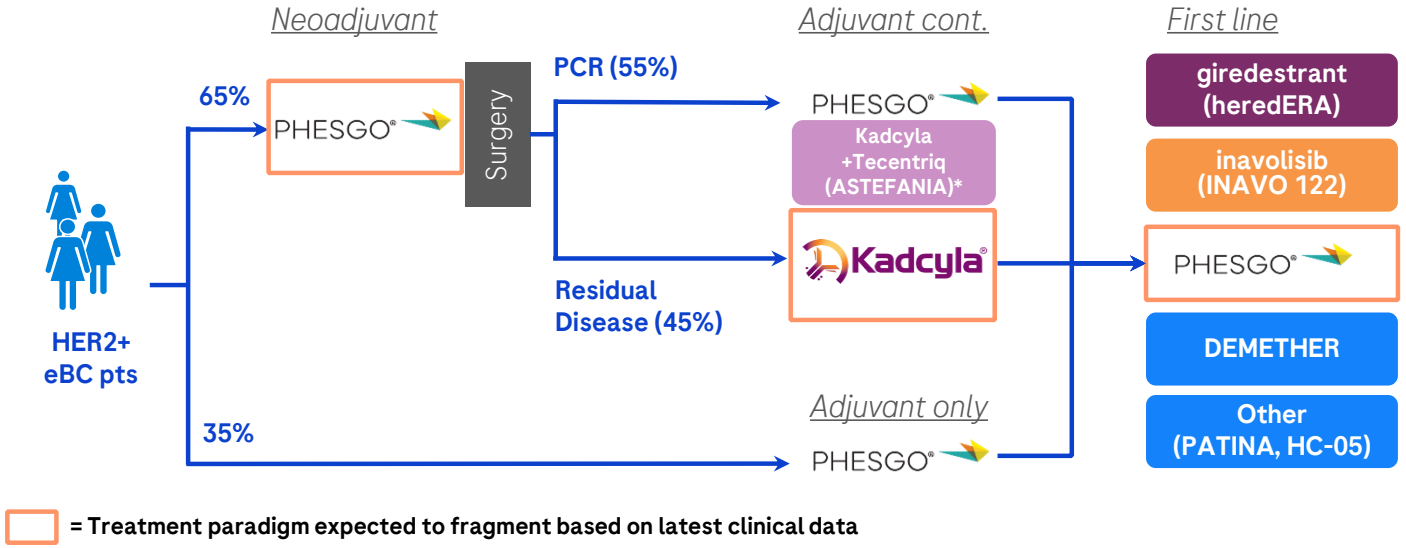
Treatment landscape is becoming increasingly segmented as novel therapies enter the market

Phesgo conversion rate



- Phesgo global conversion rate climbing to 54%*
- Expecting to achieve >60% conversion rate at peak

HER2+ BC treatment paradigm to evolve further



- eBC: Phesgo and Kadcyla expected to remain SoC in the majority of patients
- mBC: treatment strategy to evolve from 'one-size-fits-all' to tailored approaches anchored in disease biology and patient risk; Phesgo expected to remain a key treatment option
- HER2 franchise expected to peak in 2026, with a solid tail remaining by 2030
- Innovative pipeline of combinations (e.g. Phesgo + giredestrant) and NMEs (e.g. HER2 TKI)

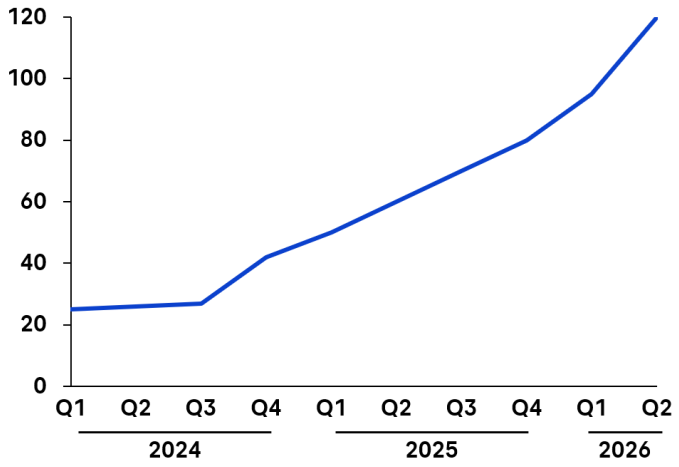
*Perjeta/Phesgo conversion rate calculated using volumes, currently taking 85 launch countries into account; e/mBC: Early/metastatic breast cancer; HER2: Human epidermal growth factor receptor 2; PCR: Pathological complete response; SoC: Standard of care

NHL: Building on leadership with novel combinations

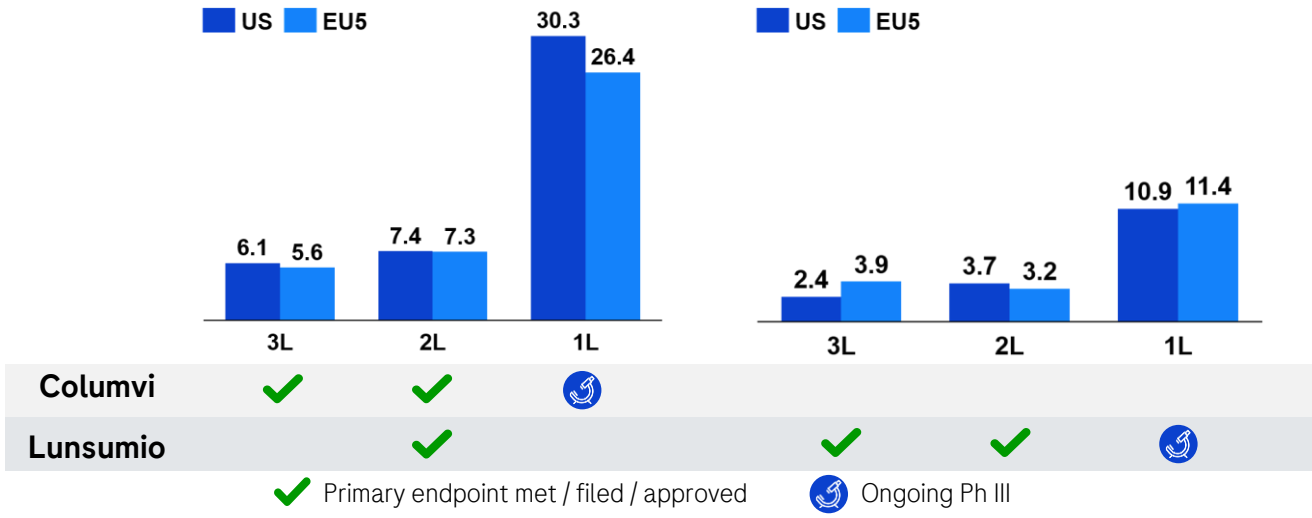
Columvi and Lunsumio expanding into earlier lines

Polivy in 1L DLBCL

Global patient count (1L DLBCL, thousands)



Columvi & Lunsumio in NHL



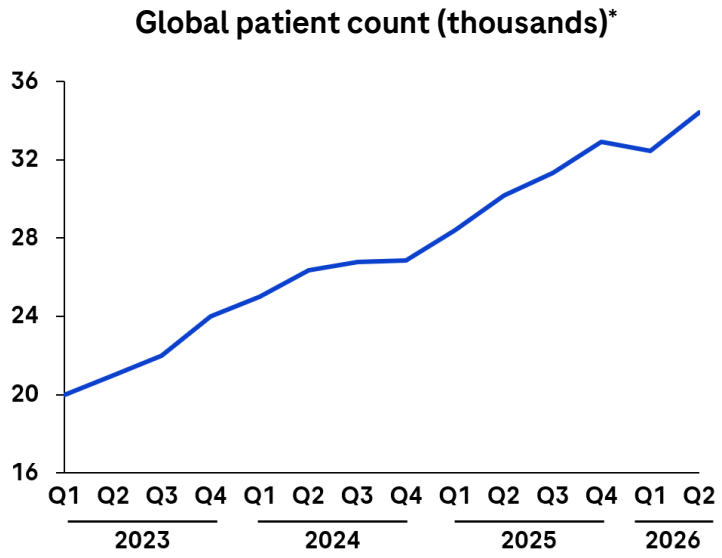
- Polivy established as SoC in 1L DLBCL; >120k pts treated globally
- Columvi: Leading bispecific in 2L+ DLBCL, supported by proven long-term OS benefit; Ph III SKYGLO combination with Polivy in 1L DLBCL with potential to bring well-tolerated outpatient regimen to larger patient population
- Lunsumio: Positive Ph III studies in 2L+ setting for both aggressive (DLBCL) and indolent (FL) lymphomas; outpatient community oriented doublet regimens with BIC tolerability and simplified dosing schedule

BIC: Best in class; DLBCL: Diffuse large B-cell lymphoma; FL: Follicular lymphoma; NHL: Non-Hodgkin's lymphoma; IPI: International prognostic index; SoC: Standard of care

Hemophilia A: Setting the SoC today, tomorrow and in the future

Potential for Hemlibra autoinjector launch and Ph III zemocimig readout by YE 2027

Hemlibra in Hemophilia A



Upcoming autoinjector



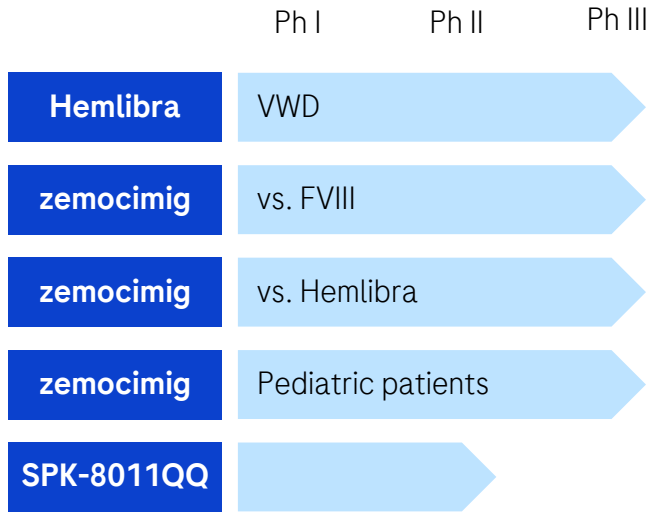
Illustrative

Time-saving administration

Improved comfort & non-visible needle

Ensures optimal therapeutic exposure

Non-malignant heme pipeline



- Growth driven by rising adoption and increased penetration in adult population
- Clinical confidence: ~80% of pts achieving zero treated bleeds** and without inducing FVIII inhibitors; RWD over >10 years in diverse patient populations, severities and clinical settings
- Autoinjectors in development to further improve convenience for Hemlibra and zemocimig

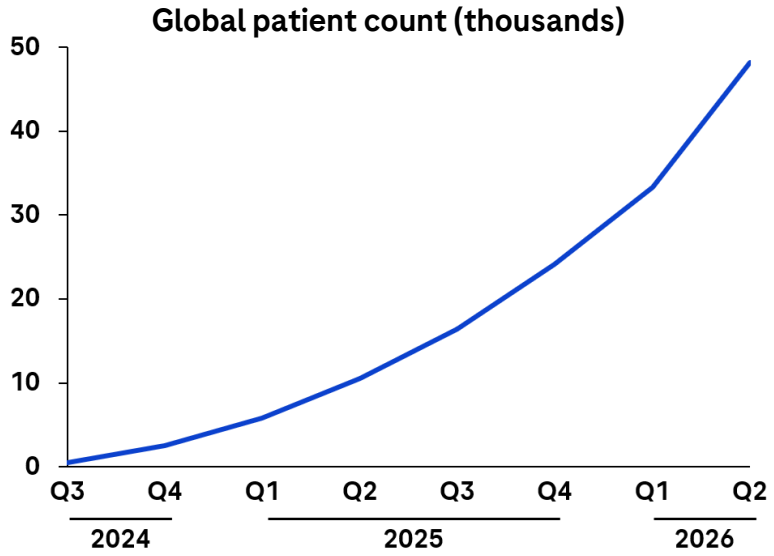
- Three Ph III trials of zemocimig in Hem A, incl. H2H vs. Hemlibra; data expected YE 2027
- Zemocimig with potential to achieve hemostatic normalization

*Commercial and WFH HA program; **Based RWD from McCary I, et al. Haemophilia 2020, Wall C, et al. ISTH 2020, Poon M-C, et al. ASH 2022 and Khairnar R, et al. ASH 2021; H2H: Head-to-head; Q2W/Q4W: Once every 2/4 weeks; RCT: Randomized-controlled trial; RWD: Real-world data; SC: Subcutaneous; SoC: Standard of care; VWD: Von Willebrand disease

Ocrevus: Expanding leadership to community and at-home settings

#1 aCD20 in MS with >525k patients globally; Roche ranked #1 amongst MS patient organizations[1]

Ocrevus SC



Upcoming Ocrevus OBI (on-body injector)



Illustrative

Novel high concentration formulation for lower injection volume vs. Ocrevus SC

Twice-yearly at-home self-administration

9.6/10 preference rating amongst investigators

Proven device: Partnership with Enable Injections

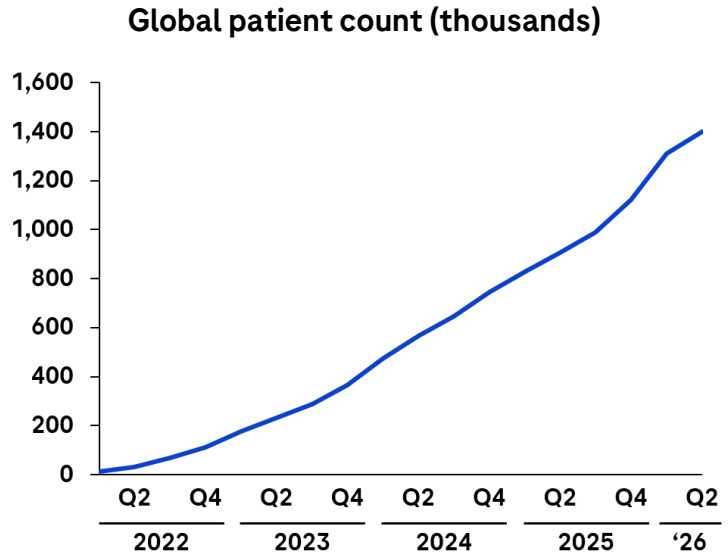
- Ocrevus proven efficacy & safety backed by 11+ years of data from clinical studies and 7+ years of experience from real-world studies
- Ocrevus Zunovo (SC) is the fastest growing aCD20 in the US, driven by uptake in community practices
- OBI is in development with the potential for convenient twice-yearly at-home self-administration; data expected in 2027

1. PatientView Corporate Reputation of Pharma - Multiple-Sclerosis Edition 2025; MS: Multiple sclerosis; OBI: On-body injector; SC: Subcutaneous; Ocrevus Zunovo with Halozyme's rHuPH20/ Halozyme's human hyaluronidase; Ocrevus OBI in partnership with Enable Injections

Vabysmo: Expecting continued global market share gains

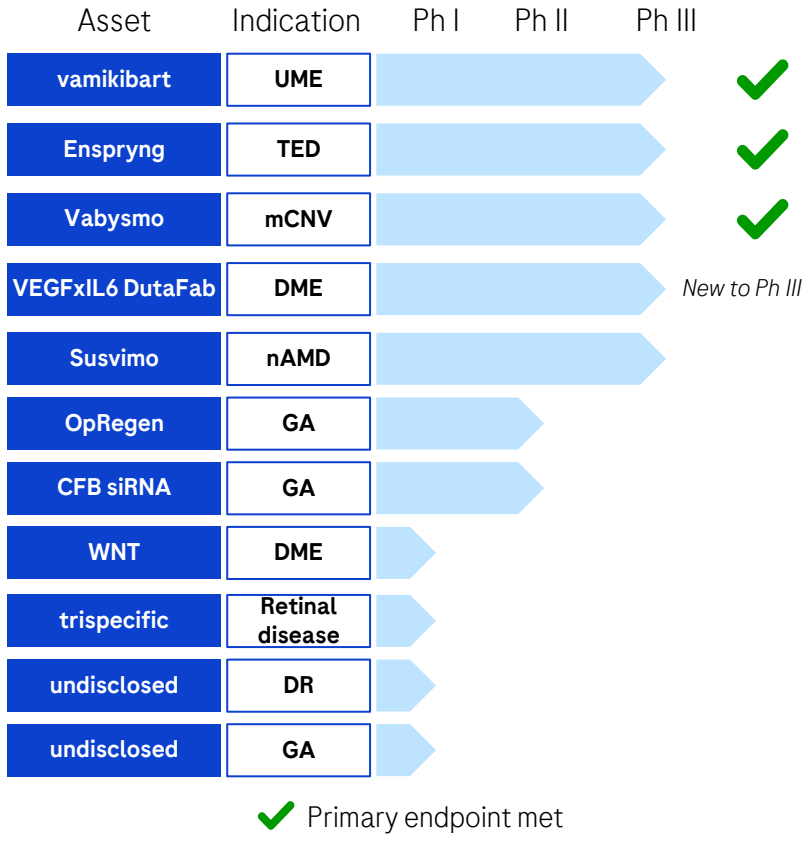
Industry-leading Ophthalmology pipeline to further build on Vabysmo's commercial success

Vabysmo in RVD (nAMD, DME and RVO)



- US branded market has stabilized; continued branded market share gains expected
- EU with strong volume growth overcompensating pricing effects
- ROW with strong growth driven by market share expansion; significant growth potential remaining

Ophthalmology pipeline



DME: Diabetic macular edema; DR: Diabetic retinopathy; GA: Geographic atrophy; nAMD: Neovascular age-related macular degeneration; RVO: Retinal vein occlusion

Expand: Leverage existing footprint for NME launches

Entrench



- Phesgo
- Tecentriq
- Itovebi
- Hemlibra
- Columvi
- Lunsumio
- Polivy
- Ocrevus
- Evrysdi
- Xolair
- Vabysmo
- Susvimo

Expand



- giredestrant
- divarasib
- tambotatug pelitecan
- HER2 TKI
- bexobrutideg
- cevostamab
- zemocimig
- fenebrutinib
- Elevidys
- Enspryng
- vamikibart
- VEGF x IL6 DutaFab

Establish



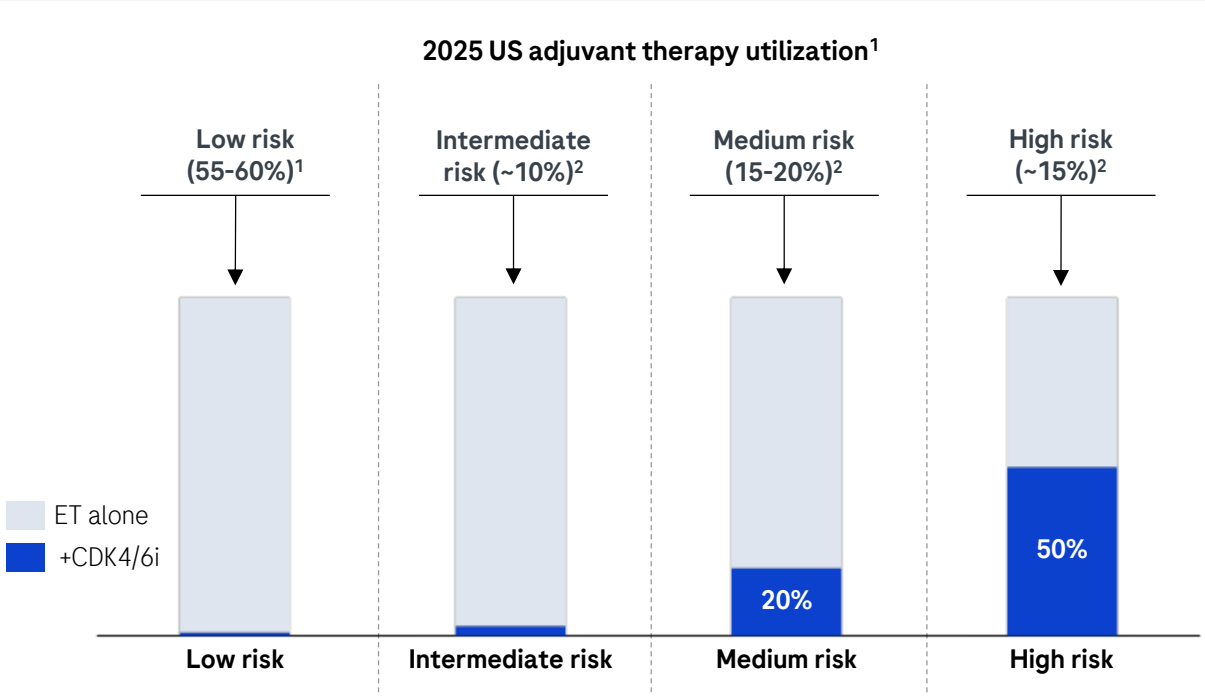
- trontinemab
- prasinezumab
- Gazyva
- sefaxersen
- afimkibart
- petrelintide
- enicepatide
- zilebesiran
- pegozafermin
- CT-996

Oncology/Hematology *Neurology* *Immunology* *Ophthalmology* *CVRM*

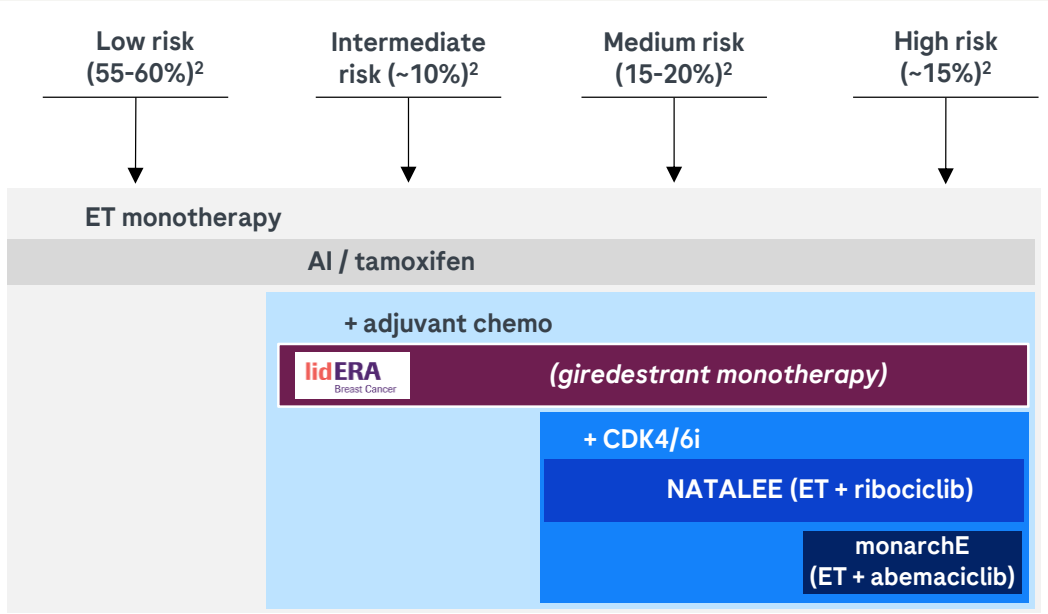
Giredestrant: Potential new SoC in adjuvant HR+ breast cancer

Giredestrant to potentially overcome limitations of current treatment options

Treatment of HR+ HER2- eBC



lidERA population vs. current treatments in adj. HR+ BC



Potential key opportunities

- *Pts. initiating ET monotherapy*
- *Pts. discontinuing/not tolerating ET*
- *Pts. discontinuing/not tolerating CDKi*
- *Future combos with CDKi*
- *Future use in low risk patients*

1. Estimates based on IQVIA and Flatiron data; 2. Risk definitions vary according to guidelines and tools used: stage at diagnosis based on internal estimates using SEER data; intermediate risk encompasses stage I, N0 included in the lidERA criteria, but excluded in NATALEE; AI: Aromatase inhibitor; (e)BC: (Early) breast cancer; CDKi: Cyclin dependent kinase inhibitor; ET: Endocrine therapy; HER2: Human epidermal growth factor receptor 2; HR: Hormone receptor; SoC: Standard of care

Giredestrant: Enabling access for rapid patient uptake

Generating clinical evidence to establish giredestrant as the new ET backbone across adjuvant settings

Strong BC legacy and US launch readiness

>5 million pts

treated globally across Roche's BC portfolio[1]

>50%

of target US HCPs engaged to date, with ongoing expansion

US Access

Robust patient support programs to reduce access barriers and enable rapid clinically-driven adoption

Awareness

Patient-centered, unbranded campaign to launch in the US

Development in adj. HR+ eBC

Pts population	Regimen	Ph I	Ph II	Ph III
Intermediate to high risk	Giredestrant mono	*lidERA*		✓
All risk, AI-intolerant	Giredestrant mono[2]	*Ph IIIb (novERA)*		
All risk, upfront, switch from CDKi	Giredestrant mono[2]	*Ph IIIb (kalitERA)*		
MRD-guided	Giredestrant mono vs. ET + CDKi[2,3]	*AFT-70 NAVIGATE IIS*		
Intermediate to high risk	Giredestrant + atirmociclib[4]			

✓ Primary endpoint met

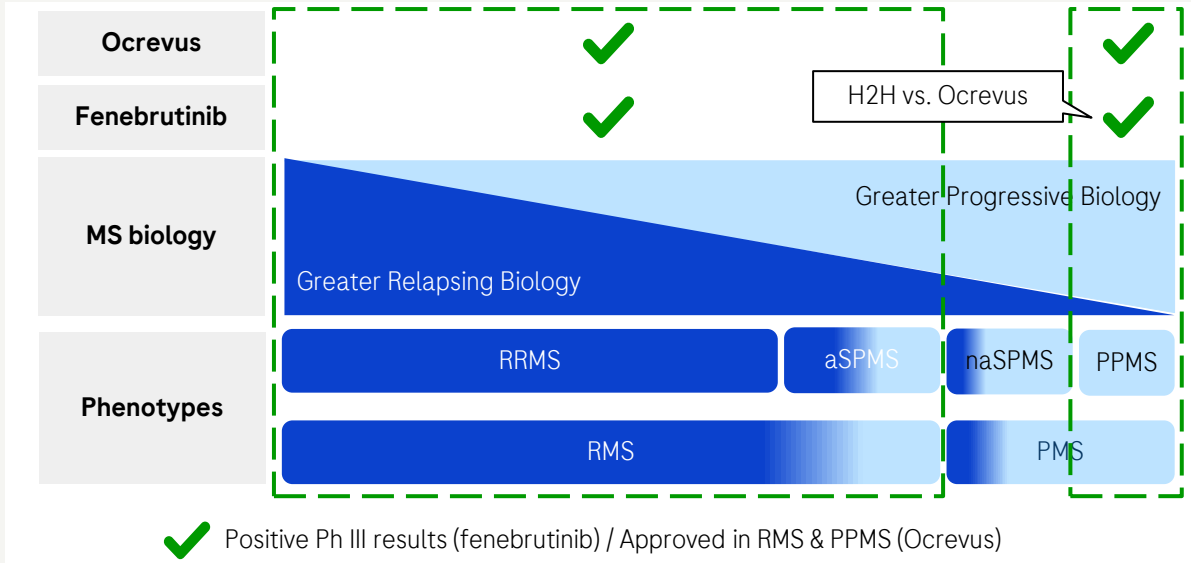
- Adjuvant HR+/HER2- eBC (lidERA): US filing acceptance under priority review, PDUFA set for Nov 30th
- Post CDKi HR+/HER2- mBC (evERA): US PDUFA set for Dec 18th
- Generating additional evidence across eBC populations (AI-intolerant, CDKi-intolerant, low-risk) and regimens (ctDNA-guided de-escalation)
- CDKi combination studies in eBC: Ph I/III with atirmociclib (CDK4i); single-arm lidERA substudies with abemaciclib and ribociclib

1. Since 1998 and until April 2026; 2. non-registrational study; 3. Pending final agreement, in collaboration with Natera and Alliance Foundation Trials; 4. Pfizer-managed, Ph III gated on positive Ph I; AI: Aromatase inhibitor; (e)BC: (Early) breast cancer; CDKi: Cyclin dependent kinase inhibitor; ET: Endocrine therapy; HCP: Healthcare professional; HER2: Human epidermal growth factor receptor; HR: Hormone receptor; MRD: Minimal residual disease; PDUFA: Prescription drug user fee act

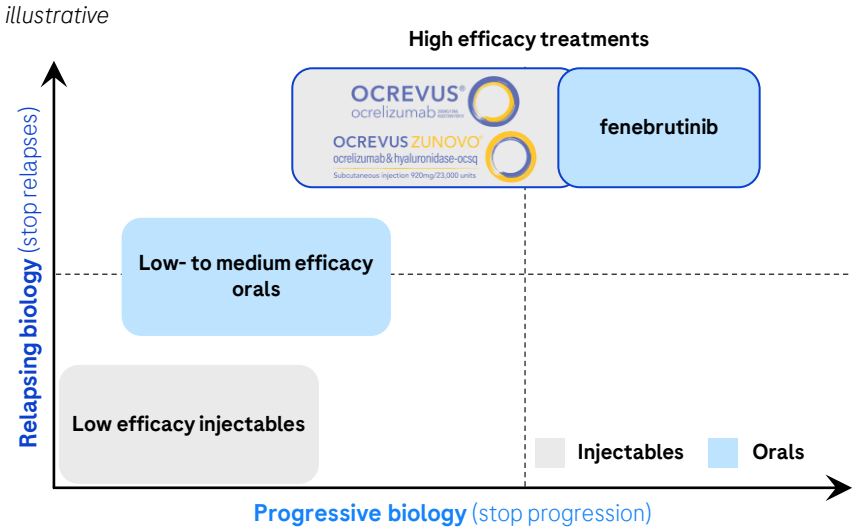
Fenebrutinib: First & only BTKi with positive Ph III in RMS/PPMS, filed in US

Potential to be the first high efficacy oral therapy for RMS and PPMS, treating both relapses and progression

Fenebrutinib & Ocrevus in relapsing and progressive MS



MS treatment landscape[1,2]



- Only BTKi with positive Ph III results in both RMS and PPMS, and only BTKi with positive H2H data vs. Ocrevus in PPMS; FIC potential in RMS and PPMS; planning real-world switch studies evaluating fenebrutinib post low or high efficacy therapies
- RMS: Significant reduction in relapses, down to one relapse every ~17 years, with the lowest ARR seen in Ph III MS trials; consistently positive trend in reducing disability progression vs. teriflunomide, incl. nominal significance in pooled cCDP12
- PPMS: First and only medicine to reduce disability progression (cCDP12) with numerical benefit vs. Ocrevus

1. Hauser SL & Cree BAC, Am J Med, 2020: 133(12):1380-1390; 2. Singer BA et al., J Neurol, 2024: 271(6):3116-3130; ARR: Annual relapse rate; BTK: Bruton's tyrosine kinase; cCDP12: 12-week composite confirmed disability progression; FIC: First in class; H2H: Head-to-head; MS: Multiple sclerosis; (n)aSPMS: (Non-)active secondary progressive multiple sclerosis; PPMS: Primary progressive multiple sclerosis; RRMS: Relapsing-remitting multiple sclerosis; SPMS: Secondary progressive multiple sclerosis

Enspryng: Differentiated risk-benefit profile in MOGAD and TED

Potentially first approved treatment in MOGAD and first IL-6R inhibitor in active and inactive TED

Enspryng addressing unmet needs in MOGAD & TED

MOGAD

- Potentially first approved treatment in MOGAD
- Significant, clinically meaningful reduced risk of new relapses
- Highly favorable safety profile

TED

- Potentially first at-home SC treatment in TED
- Consistent, clinically meaningful improvements across key TED symptoms
- Highly favorable safety profile

Enspryng development program

Indication	Ph I	Ph II	Ph III	Status
NMOSD	*SAkuraSTAR / SAkuraSky*		✓	Launched in US/EU
MOGAD	*METEOROID*		✓	Filed in US/EU
TED	*SatraGO-1/2*		✓	Filed in US
AIE	*CIELO*			2027 readout

✓ Filed or approved

- Enspryng engineered for maximal inhibition of IL-6 signaling, a key pathway across Neurology and Ophthalmology indications
- MOGAD: Highly significant reductions in annualized relapse rate, active MRI lesions, and rescue therapy use; US & EU filing achieved; US priority review granted with PDUFA Jan 10th
- TED: Totality of Ph III results show consistent, clinically meaningful improvements across key TED signs and symptoms; US priority review granted with PDUFA Oct 15th
- Ongoing Ph III in AIE with data expected 2027

AIE: Autoimmune encephalitis; MOGAD: Myelin oligodendrocyte glycoprotein antibody-associated disease; NMOSD: Neuromyelitis optica spectrum disorder; PDUFA: Prescription drug user fee act; TED: Thyroid eye disease

Establish: Enter disease areas new to Roche

Entrench	Expand	Establish
Phesgo	giredestrant	trontinemab
Tecentriq	divarasib	prasinezumab
Itovebi	tambotatug pelitecan	Gazyva
Hemlibra	HER2 TKI	sefaxersen
Columvi	bexobrutideg	afimkibart
Lunsumio	cevostamab	petrelintide
Polivy	zemocimig	enicepatide
Ocrevus	fenebrutinib	zilebesiran
Evrysdi	Elevidys	pegozafermin
Xolair	Enspryng	CT-996
Vabysmo	vamikibart	
Susvimo	VEGF x IL6 DutaFab	

Oncology/Hematology *Neurology* *Immunology* *Ophthalmology* *CVRM*

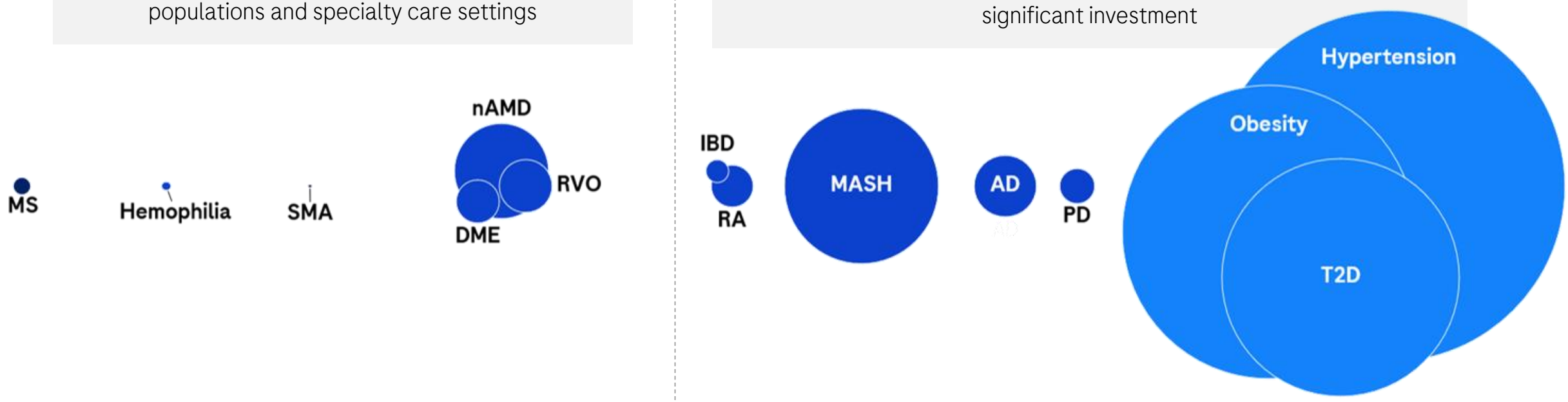
Entry into new disease areas to address global burden of disease

We deploy targeted investments and new approaches to ensure launch excellence in new disease areas

2015 → **2025** → **2035**

Over the past 10 years, Roche has successfully entered new Disease Areas, primarily within targeted patient populations and specialty care settings

In the next 10 years, Roche will enter broad-scale Disease Areas like obesity, T2D and Hypertension, necessitating different approaches and significant investment



Ocrevus *Hemlibra* *Evrysdi* *Vabysmo* *afimkibart* *pegozafermin* *afimkibart* *trontinemab* *prasinezumab* *enicepatide, CT-996* *petrelintide* *zilebesiran*

Bubble size is illustrative and represents prevalence of disease

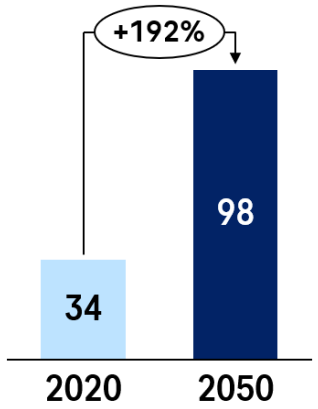
Care setting for majority of patients: Hospital Clinic Patient-driven

AD: Alzheimer's disease; DME: Diabetic macular edema; IBD: Inflammatory bowel disease, MASH: Metabolic dysfunction-associated steatohepatitis; MS: Multiple sclerosis; nAMD: Neovascular age-related macular degeneration; PD: Parkinson's disease; RA: Rheumatoid arthritis; RVO: Retinal vein occlusion; SMA: Spinal muscular atrophy; T2D: Type-2 diabetes

Trontinemab with BID potential in Alzheimer's disease

Alzheimer's disease overview

Global prevalence
(Mn patients)[1]



Key unmet needs

Diagnosis
Invasive (PET/CSF), costly and not widely accessible

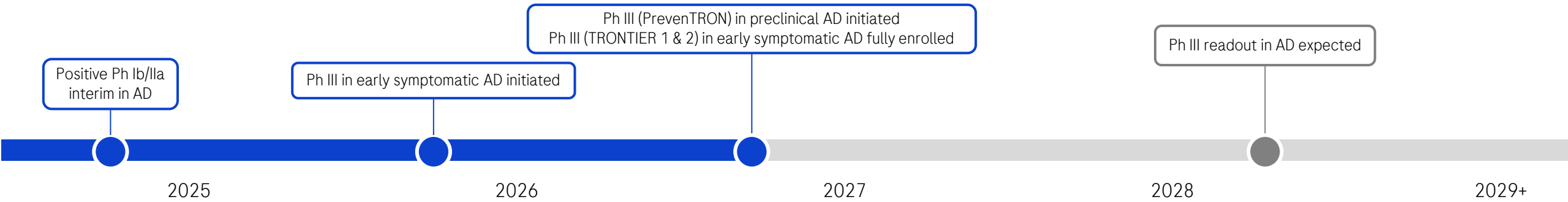
Efficacy
Faster and more complete amyloid clearance needed

Safety
ARIA-E and high monitoring needs

Roche in Alzheimer's disease

- Opportunity for trontinemab to differentiate on efficacy and safety as next gen Aβ antibody
- New Elecsys pTau217 blood test offers more accessible, reliable & faster diagnosis

Indication	Asset	Ph I	Ph II	Ph III
Early symptomatic AD	trontinemab	TRONTIER 1 & 2		
Preclinical AD	trontinemab	PrevenTRON		
AD	nivegacetor	GABRIELLA		
AD	NLRP3i			
AD	APOE ASO			

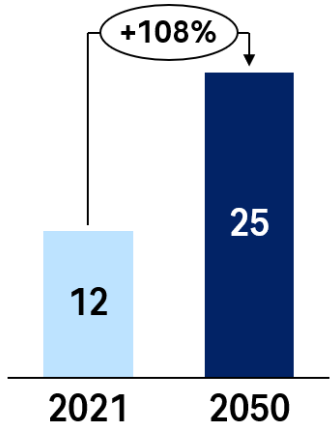


1. DRG (data available till 2036, extrapolated till 2050); AD: Alzheimer's disease; APOE: Apolipoprotein E; ASO: Antisense oligonucleotide; BID: Best-in-disease; CSF: Cerebrospinal fluid; PET: Positron emission tomography

Prasinezumab with potential to become first DMT in Parkinson's disease

Parkinson's disease overview

Global prevalence,
(Mn patients)[1]



Quality of life impact
(% of patients)[2-4]

41%

of initially independent patients lose functional independence with 5 years

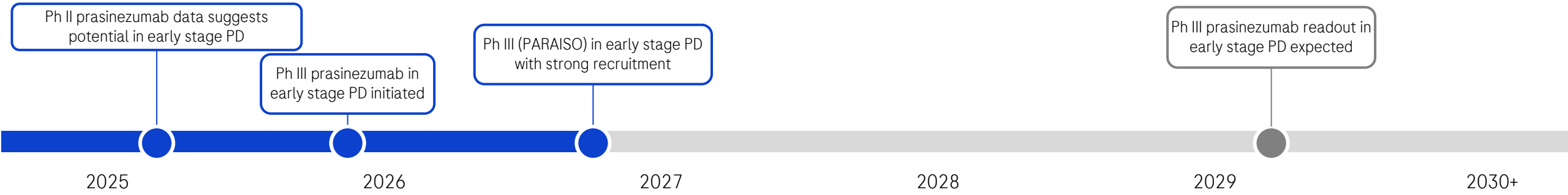
46%

stop working within 5 years of diagnosis

Roche in Parkinson's disease

- Symptomatic treatments temporarily relieve motor symptoms, but benefits decline over time
- Multiple endpoints from Ph II (PASADENA & PADOVA) suggest potential to delay motor progression
- Ph III (PARAISO) in early stage PD ongoing; trial design optimized based on Ph II insights

Indication	Asset	Ph I	Ph II	Ph III
PD	prasinezumab			*PARAISO*
PD	NLRP3i			

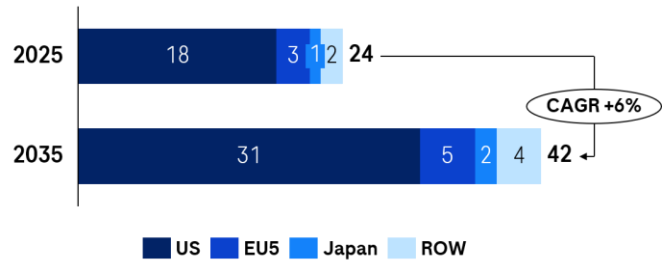


1. Shalash A, et al., J Parkinsons Dis. 2026 Jun 23:1877718X261459875; 2. Bjornestad et al., Neurology 2016 Oct 11;87(15):1599-1606; 3. J Parkinsons Dis 2018, The Emerging Evidence of the Parkinson Pandemic Dorsey et al. 4. Global, regional, national epidemiology and trends of Parkinson's disease from 1990 to 2021: findings from the Global Burden of Disease Study 2021 Luo et al.; DMT: Disease modifying treatment; PD: Parkinson's disease

Afimkibart spearheading our IBD entry, backed by next-gen MoAs

IBD overview

Global IBD market (CHF bn)[1]



Key unmet needs

Efficacy ceiling

Initial response and remission limits of current Tx options

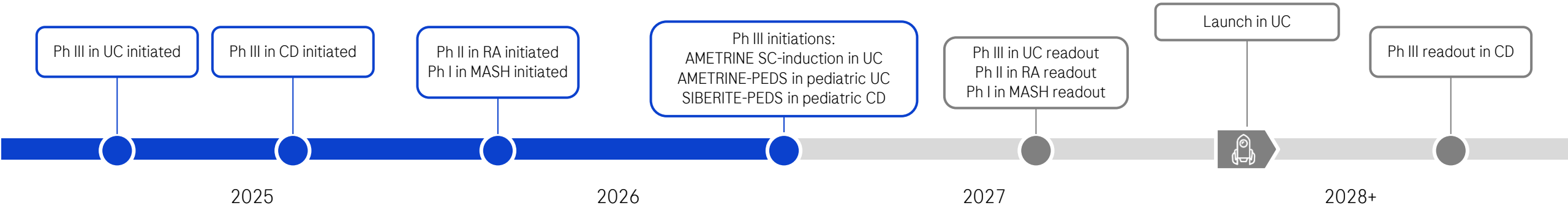
Biologics sequencing

Inconsistent guidelines, multiple cycles through Tx impacting QoL

Roche in IBD

- Afimkibart, the only TL1A offering the possibility of SC administration for induction & maintenance
- Exploring TL1A in multiple immunological diseases, incl. MASH and RA, in mono/combo Tx
- Developing next-gen IBD assets, e.g. bispecifics

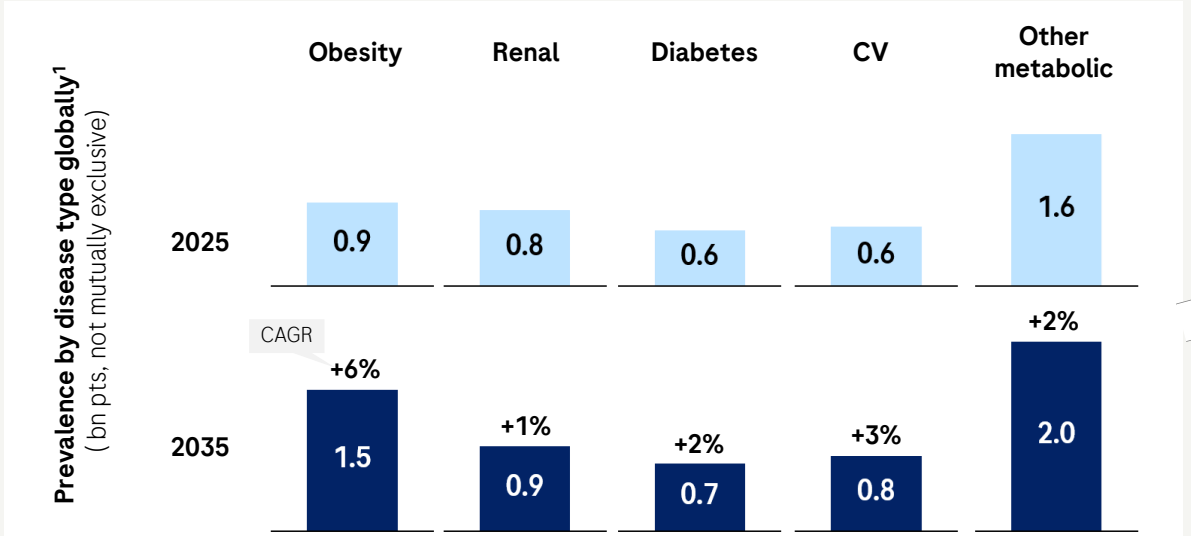
Indication	Asset	Ph I	Ph II	Ph III
UC	afimkibart	*AMETRINE-1/2*		
CD	afimkibart	*SIBERITE-1/2*		
IBD	p40 x TL1A	*SUNCREST*		
IBD	TL1A bispecific			



1. Evaluate Pharma; CD: Crohn's Disease; IBD: Inflammatory bowel disease; MASH: Metabolic dysfunction-associated steatohepatitis; MoA: Mode of action; QoL: Quality of life; RA: Rheumatoid arthritis; SC: Subcutaneous; Tx: Treatment; UC: Ulcerative colitis

CVRM pipeline advancing, primed to address growing disease burden

CVRM overview

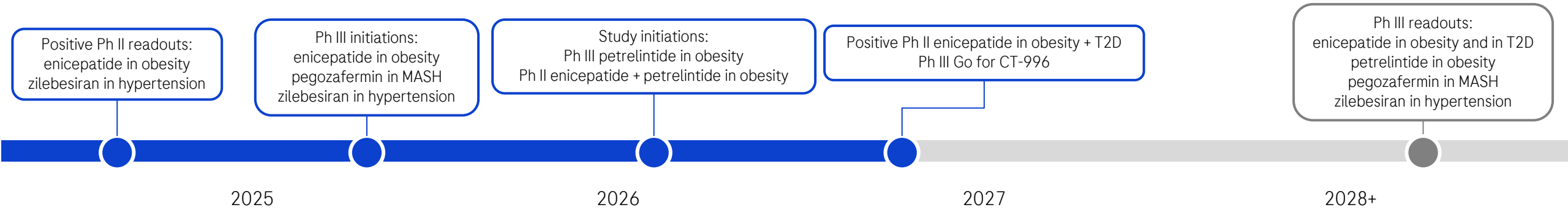


Roche in CVRM

Obesity/ diabetes	▪ Positioned to capture market expansion & fragmentation ▪ Pts-centric care to meet evolving obesity/diabetes needs
MASH	▪ Leveraging advanced NITs for earlier diagnosis ▪ Potential BID innovative NME in a nascent market
uHTN	▪ Potential to address poor oral adherence to boost control ▪ Leading the paradigm shift to continuous BP control

not exhaustive

Disease/Indication	Asset	Ph I	Ph II	Ph III
Obesity, T2D, HFrEF, ASCVD	enicepatide			
Obesity	petrelintide			
Obesity	CT-996			
MASH	pegozafermin			
uHTN	zilebesiran			
Obesity	UCN2 analog*			



1. Sources: Hu SS et al. Chin Med J, 2025; Wang L et al. JAMA Intern Med, 2023; Zhou J et al. Clin Gastroenterol Hepatol, 2025; Chen Y et al. J Clin Transl Hepatol, 2024; Estes C et al. J Hepatol, 2018; IDF Diabetes Atlas, 11th ed., 2025; Zhou B et al. Lancet, 2024; Phelps NH et al. Lancet, 2024; Wang L et al. Lancet, 2021; Dandona L et al. Lancet Glob Health, 2018; Kalra A et al. Int J Environ Res Public Health, 2025; GBD 2023 CKD Collaborators Lancet, 2025; Duseja A et al. J Hepatol, 2024; Kalra S et al. J Gastroenterol Hepatol, 2024; Misra A et al. Diabetes Ther, 2025; Anjana RM et al. Lancet Diabetes Endocrinol, 2023; Srivastava S et al. BMJ Open, 2025; Yusuf S et al. Lancet, 2004; *pending deal closure; ASCVD: Atherosclerotic cardiovascular disease; BID: Best In disease; BP: Blood pressure; CVRM: Cardiovascular, renal and metabolism; HFrEF: Heart Failure with reduced ejection fraction; MASH: Metabolic dysfunction-associated steatohepatitis; NIT: Non-invasive tests; T2D: Type-2 diabetes; uHTN: uncontrolled hypertension

Progress since Pharma Day 2025

Our strategy and capabilities

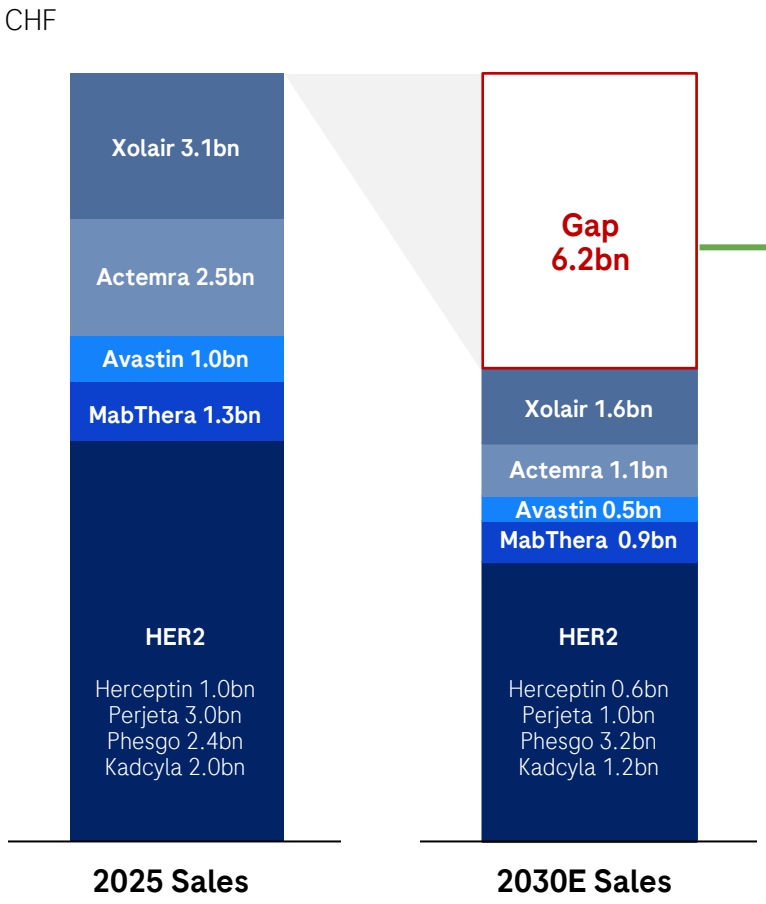
Our commercialization excellence

Future growth opportunities

Updated 2025-30 consensus outlook*

Long-term growth outlook improved; significant upside potential remains on the late-stage pipeline

Biosimilar gap (25-30)



Consensus sales growth (25-30)

Vabysmo	1.6bn
Itovebi	1.0bn
Columvi	0.9bn
Polivy	0.9bn
Gazyva	0.8bn
Evrysdi	0.6bn
Lunsumio	0.5bn
Hemlibra	0.4bn
Enspryng	0.3bn
Ocrevus	-0.1bn
Other in-market[1]	0.1bn
Pipeline Ph III[2]	**9.2bn**
thereof giredestrant	3.5bn
thereof fenebrutinib	1.1bn
thereof enicepatide (CT-388)	0.8bn
thereof afimkibart (TL1A)	0.7bn
thereof divarasib	0.5bn
thereof pegozafermin	0.4bn
thereof petrelintide	0.4bn
thereof trontinemab	0.4bn
thereof vamikibart	0.3bn
thereof sefaxersen	0.3bn
thereof zemocimig (NXT007)	0.3bn
thereof prasinezumab	0.3bn
thereof zilebesiran	0.2bn
Total	**16.3bn**

Potential upside

Assets with low to no coverage in current sell side models:

Cardiovascular & Metabolism: CT-996 in obesity +/- T2D; enicepatide + petrelintide in obesity +/- T2D; enicepatide in T2D

Oncology/Hematology: cevostamab in r/r MM; bexobrutideg in r/r CLL; CDK4/2i in BC; HER2 TKI in HER2+ BC; tambotatug pelitecan in lung cancer; P-BCMA-ALLO1 in r/r MM

Neurology: Enspryng in MOGAD & AIE; nivegacetor in AD; NLRP3i in AD/PD; CD-20 Brainshuttle™ in MS

Immunology: Gazyva in immune-mediated kidney diseases; p40 x TL1A bispecific in IBD; Lunsumio and cevostamab in autoimmune diseases

Ophthalmology: Enspryng in TED; VEGF x IL6 DutaFab in DME

*All estimates are based on Post HY 2026 consensus collected by FTI Consulting on behalf of Roche (n=17) differences may occur due to rounding; 1. Tecentriq, Alecensa, Elevidys, Susvimo, PiaSky, Activase/TnKase, Pulmozyme, CellCept, Xofluza, Rozlytrek: 2. included in >50% of sell-side models

2027: Key newsflow outlook*

	Compound	Indication	Milestone
Regulatory	divarasib	2L KRAS G12C+ NSCLC	US/EU approval
	Lunsumio + Polivy	2L+ DLBCL	US approval
	Lunsumio	2L+ FL	US/EU filing
	Hemlibra	Autoinjector	US/EU approval
	PiaSky	aHUS	US/EU approval
	fenebrutinib	RMS	US approval
	fenebrutinib	PPMS	US approval
	Enspryng	MOGAD	US/EU approval
	sefaxersen	IgAN	US filing
	Gazyva	SLE	EU approval
	Gazyva	MN	EU approval
	vamikibart	UME	US/EU approval
	Susvimo	DME	EU filing
Clinical results	Itovebi + Phesgo	1L *PIK3CA*-mut HER2+ mBC	Ph III INAVO122
	Columvi + Polivy + R-CHP	1L DLBCL	Ph III SKYGLO
	zemocimig	Hemophilia A	Ph III ZEBRHA 1/2
	Hemlibra	VWD	Ph III WILL-EMI
	Enspryng	AIE	Ph III CIELO
	Ocrevus OBI	RMS/PPMS	Ph III OCTAVIBE
	afimkibart	UC	Ph III AMETRINE-1/2
	Susvimo	AMD Q9M	Ph III VELODROME
	nivegacetor	AD	Ph II GABriella
	afimkibart	RA	Ph II dRAvite
	p40 x TL1A	UC	Ph II SUNCREST

*Outcome studies are event-driven: timelines may change

R&D Excellence

Levi Garraway
CMO and Global Head of Product Development

Recap | Our 2030 ambition

Deliver 20 transformative medicines[1] by 2030, addressing disease areas with the highest societal burden

Reach top-quartile performance in R&D productivity across the biopharma industry to enable competitive end-to-end advantage



1. Reaching ‘Bar’ criteria: Future medicines that can have high impact for patients, high revenue potential, and optimized risk.

Roche is on a path to materially elevate R&D productivity

Prioritizing transformative assets by applying the Bar



Answers a clear & addressable unmet need



Engages a 'foundational target'



Possesses worthy pharmacologic & developability characteristics



Achieves meaningful therapeutic differentiation



Unlocks a path to value (e.g., robust revenue potential)

Full adoption and early impact seen

100% adoption in Research and Development, enforced via Governance Boards

Sharper focus on critical aspects

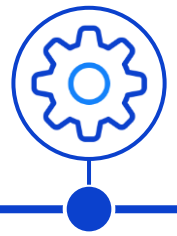
Recent updates sharpen the focus on the 'crux' (single most important risk of program), path to value & therapeutic differentiation



2023
Diagnosis



2024
Implementation



2025
Execution



2026
Operationalization

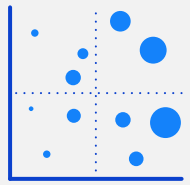


2027+
Augmentation

R&D Excellence: Our solutions

Moving from implementation to “business as usual”

Adopt a unified portfolio framework ✓



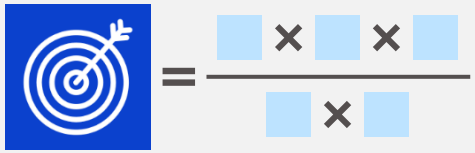
Transform our portfolio management & governance ✓



Access the best external innovation ✓



Embrace ambitious R&D objectives ✓



Evolve our R&D engine and invest in its excellence ✓



Align our incentives with the new R&D strategy ✓



Build a simplified system landscape and data foundation ✓

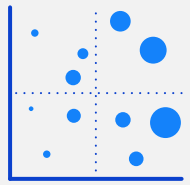
✓ Implemented and moving into business as usual

✓ Implementation in progress

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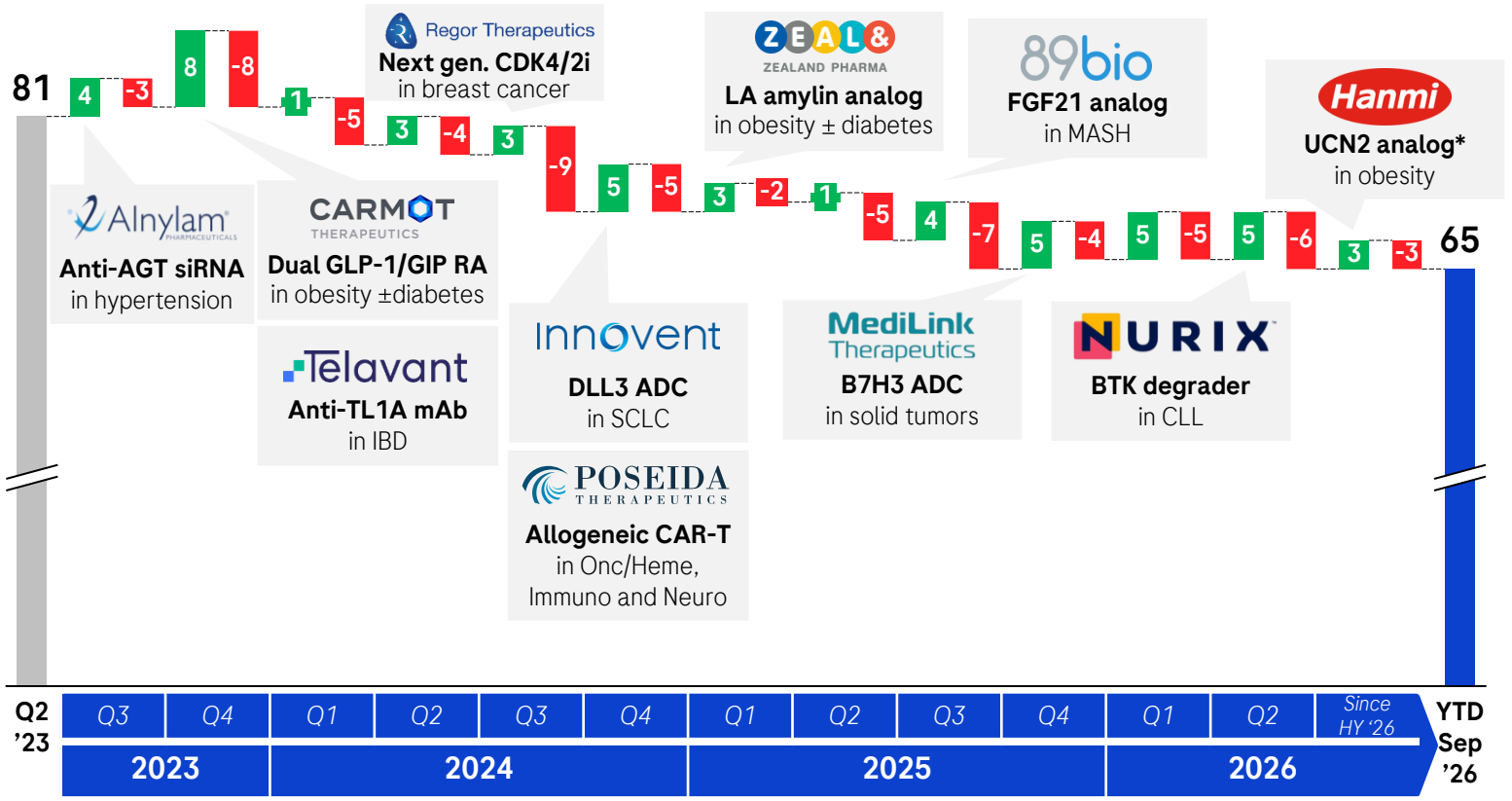
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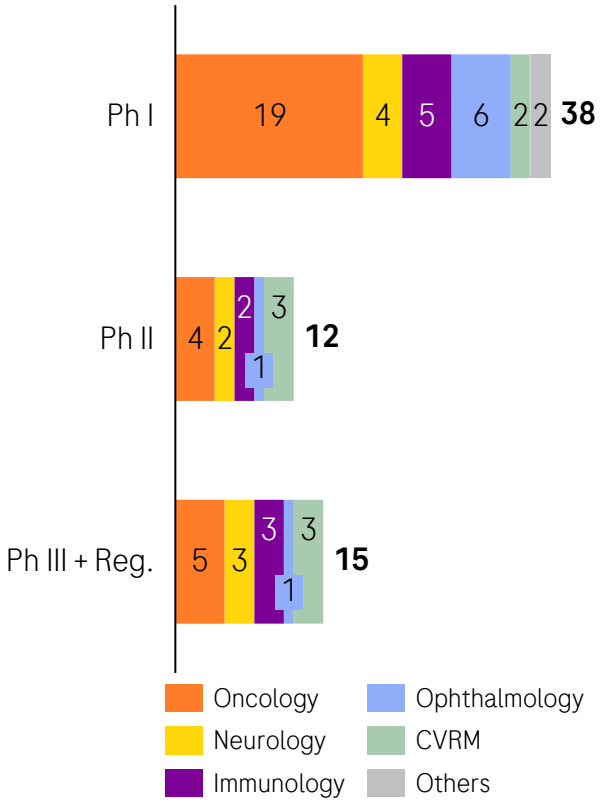
Pipeline prioritization since start of R&D Excellence

Focus on high-impact projects and partnerships

Portfolio evolution (NME additions / removals)



Pipeline NMEs by TA

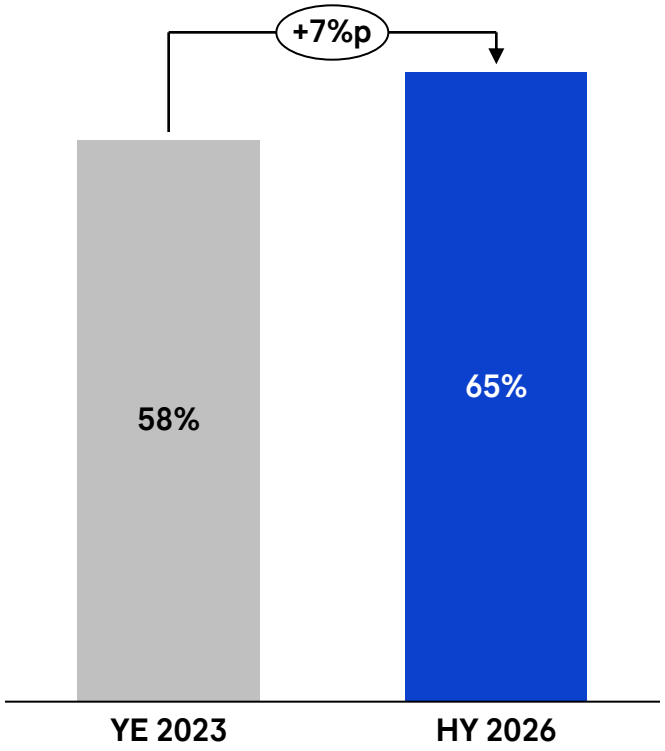


1. Source: internal data, 28th September 2026; *pending deal closure; ADC: Antibody-drug conjugate; AGT: Angiotensinogen; BTK: Bruton's tyrosine kinase; CAR-T: Chimeric antigen receptor T-cell; CDK4/2i: Cyclin dependent kinase-4/2 inhibitor; CSU: Chronic spontaneous urticaria; DLL3: Delta-like ligand 3; FGF21: Fibroblast growth factor 21; GIP: Glucose-dependent insulinotropic polypeptide; GLP-1: Glucagon-like peptide-1; IBD: inflammatory bowel disease; LA: Long acting; mAb: Monoclonal antibody; MASH: Metabolic dysfunction-associated steatohepatitis; NME: New molecular entity; RA: Receptor agonist; SCLC: Small-cell lung cancer; siRNA: Small interfering RNA; TL1A: Tumor necrosis factor-like cytokine 1A; UCN2: Urocortin 2; Note: Chart Includes all assets from Ph I to Registration

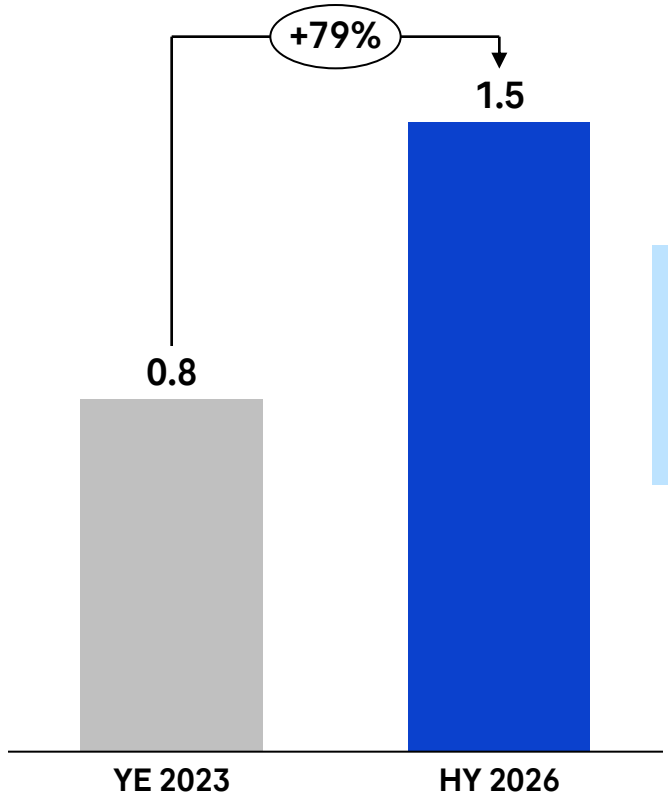
Pipeline evolution since YE 2023

Growing share of potential best in disease assets and increasing peak sales for pipeline projects

Share of late-stage projects with BID potential[1]



Average peak sales per pipeline project, CHFbn[1]



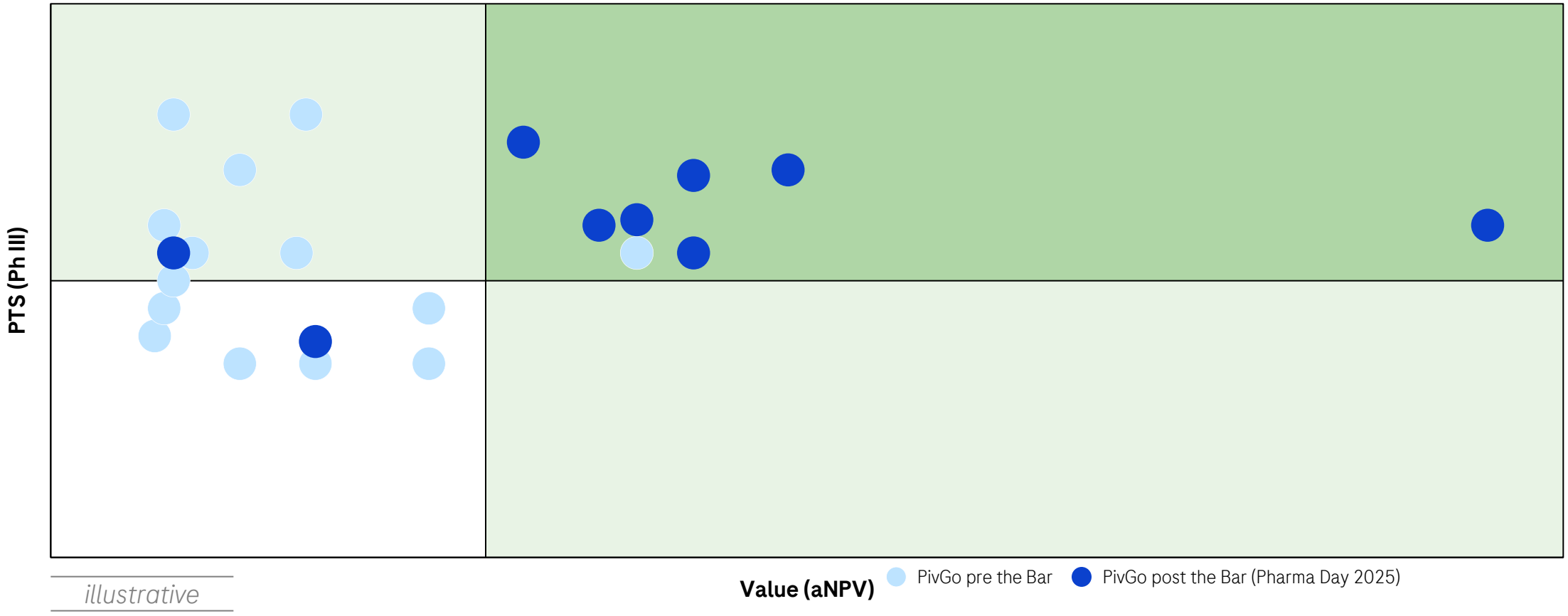
Total portfolio value +72% since YE 2023

1. Source: Internal data; BID: Best-in-disease; YE: Year end

Overall portfolio shift after introduction of the Bar

Shifting portfolio to higher value and more balanced risk-reward

Risk-reward profile of assets with PivGo decisions post introduction of the Bar vs pre the Bar[1], NMEs only

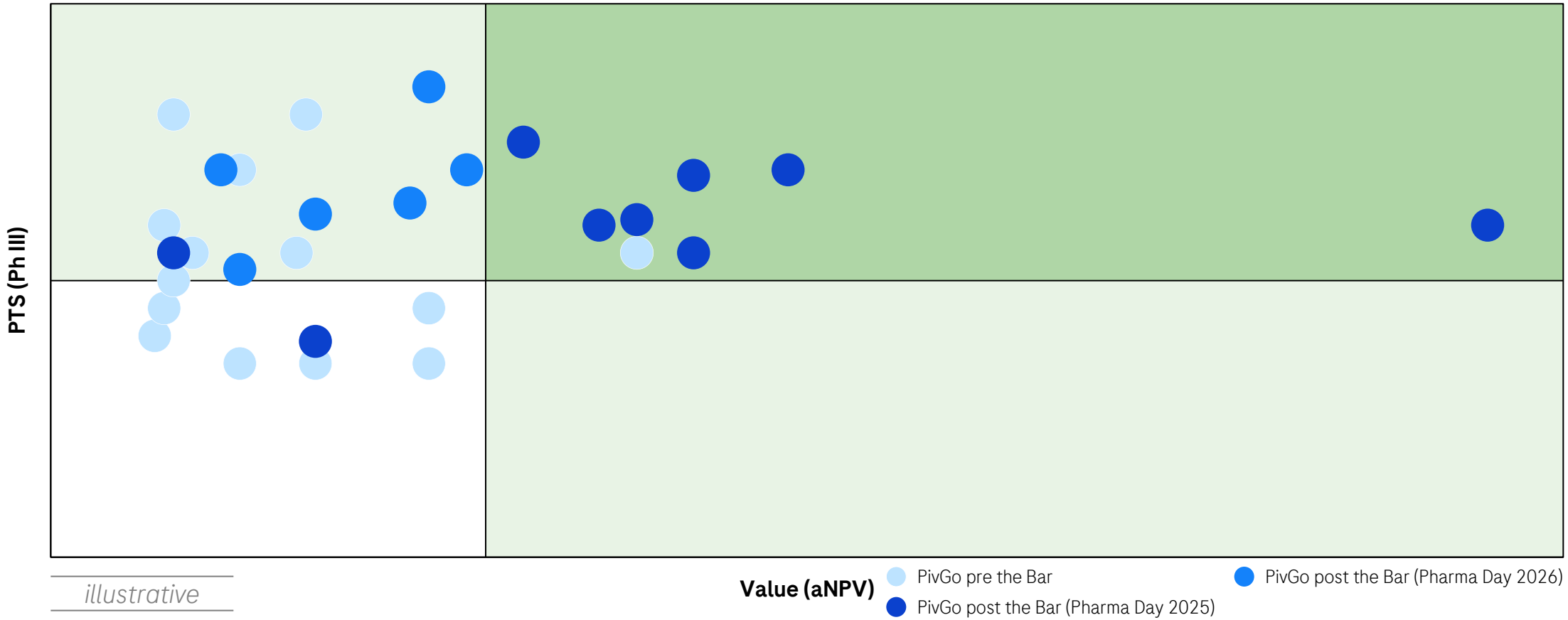


1. Based on all NMEs with PivGo decision since 2019, cut-off for pre the Bar at start of 2024; Values as of Ph III Go decision; aNPV: Risk-adjusted net present value (accounting for probability of launch); NME: New molecular entity; PivGo: Pivotal Go (pivotal study initiation decision point); PTS: Probability of technical success

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Access the best external innovation



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Evolve our R&D engine and invest in its excellence

Align our incentives with the new R&D strategy

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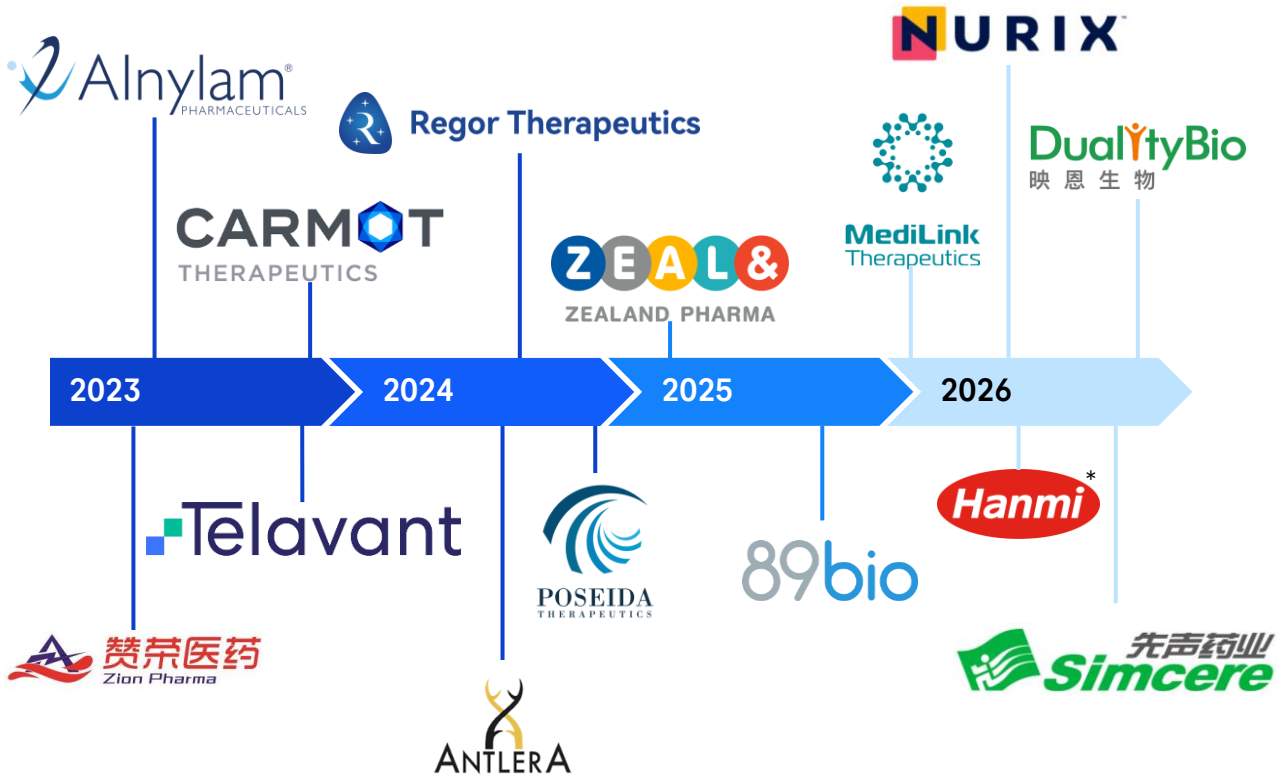
Implemented and moving into business as usual

Implementation in progress

Pipeline acceleration through partnering and acquisitions

Key deals completed to complement our pipeline across our five therapeutic areas

Key deals completed since start of R&D Excellence



External innovation to catalyze portfolio rejuvenation

- Deals focusing on clinical stage assets (e.g. Ph III asset bexobrutideg in CLL) and platform technologies (e.g. access to DualityBio's ADC platform for lead compound generation)
- Focusing on efficient integration processes and capabilities to remain the 'Partner of choice' for biotech
- Business development in combination with stringent R&D budget control to catalyze portfolio rejuvenation

*pending deal closure; CLL: Chronic lymphocytic leukemia

R&D Excellence: Our solutions

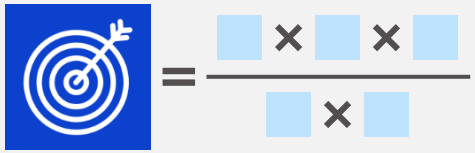
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Our cumulative impact (2024 - YTD 2026)

Added **47 assets**[1] total from early R&D and high-value partnerships (+18 in 2026)

CHF +0.64bn[2] average peak year sales across late-stage portfolio

Team-assessed **Ph III PTS has increased** since R&D Excellence roll-out

R&D Excellence =

(**Volume** × **Value** × **Success rate**) → **Effectiveness**

(**Costs** × **Cycle time**) → **Efficiency**

Reinvested **CHF ~1.6bn** of savings from R&D back into the portfolio

On track to achieve **~19 months acceleration** by end of 2026

On track | Longer follow-up needed

1. Assets entering EIGLP (entry into GLP toxicology) studies; 2. From CHF 0.81 bn CHF to nearly CHF 1.45 bn; PTS: Probability of technical success

Significant progress made since Pharma Day 2025

+18 assets added from early R&D and high-value partnerships

Average peak year sales across late-stage portfolio raised by **CHF +0.19bn**[1]

Ph 3 success rate at **>80%** in 2026 YTD

R&D Excellence =

Effectiveness: Volume × Value × Success rate

Efficiency: Costs × Cycle time

Increased savings by **CHF ~0.5bn**

Further accelerated cycle times by **~8 months**

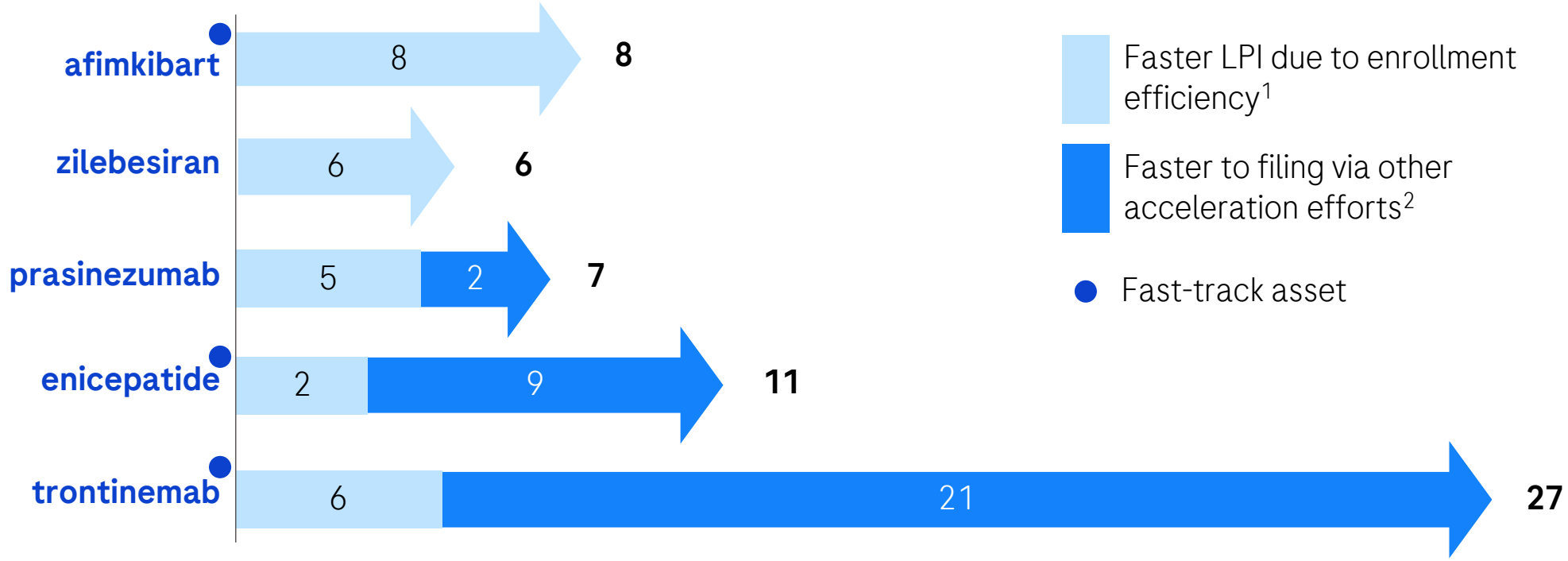
On track | Longer follow-up needed

1. From CHF 0.81 bn CHF to nearly CHF 1.45 bn

Significant acceleration achieved across late-stage pipeline

Optimized R&D efficiency driving cycle time acceleration beyond fast-tracked assets

Acceleration from initial projections (months)



Source: Internal data; 1: Faster LPI not guaranteed to accelerate filing submission timelines for event-driven trials. 2: Other acceleration efforts include changes in study design/clinical development plan, Ph III ungating at interim and PoC biomarker analyses, and frontloading Ph III activities; LPI: Last patient in

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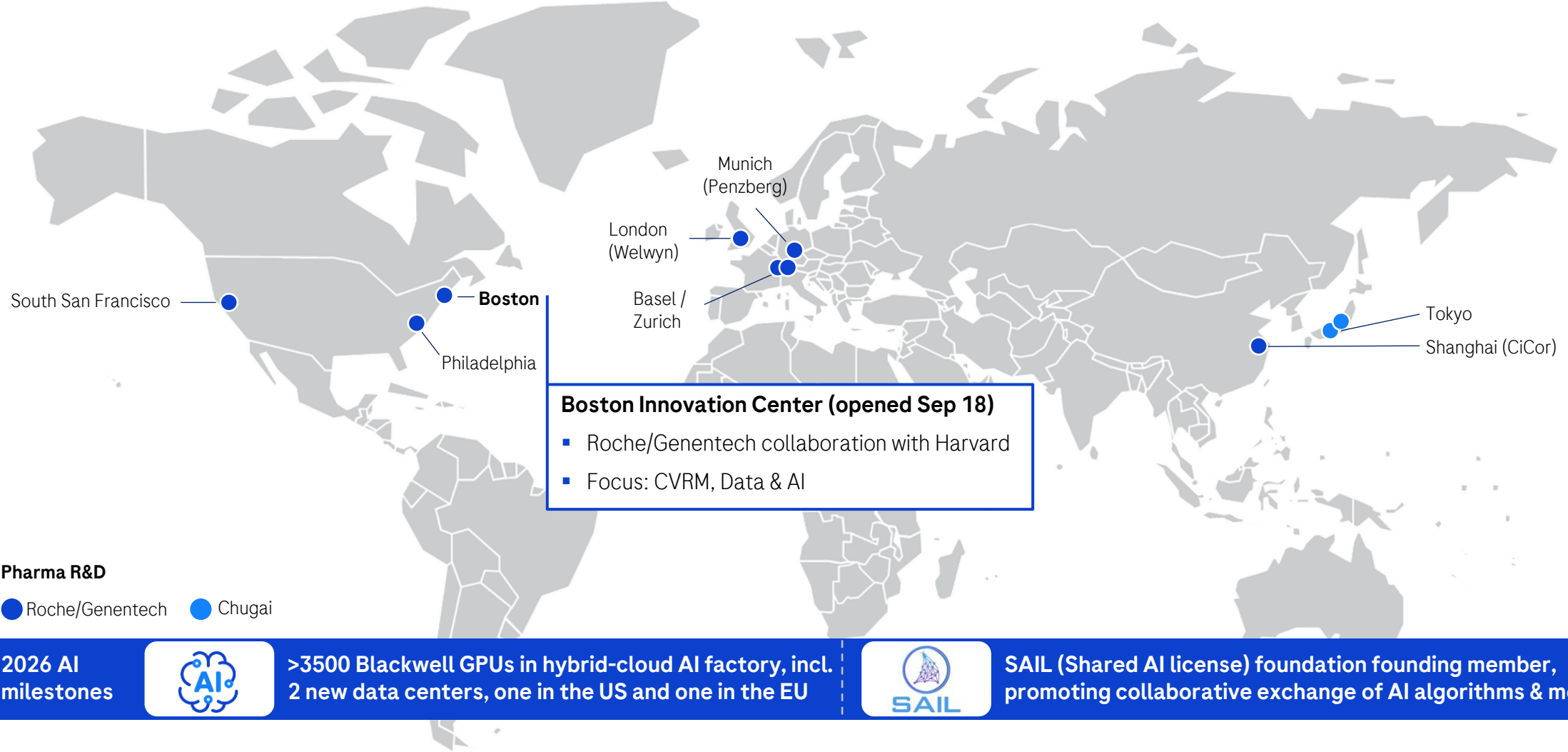
Build a simplified system landscape and data foundation ✓

✓ Implemented and moving into business as usual

✓ Implementation in progress

Ongoing R&D investments

Boston Innovation Center focusing on CVRM and AI in drug development opened



Boston Innovation Center (opened Sep 18)

- Roche/Genentech collaboration with Harvard
- Focus: CVRM, Data & AI

2026 AI milestones

>3500 Blackwell GPUs in hybrid-cloud AI factory, incl. 2 new data centers, one in the US and one in the EU

SAIL (Shared AI license) foundation founding member, promoting collaborative exchange of AI algorithms & models

AI: Artificial intelligence; CVRM: Cardiovascular, renal and metabolism

R&D Excellence delivering the next wave of innovation

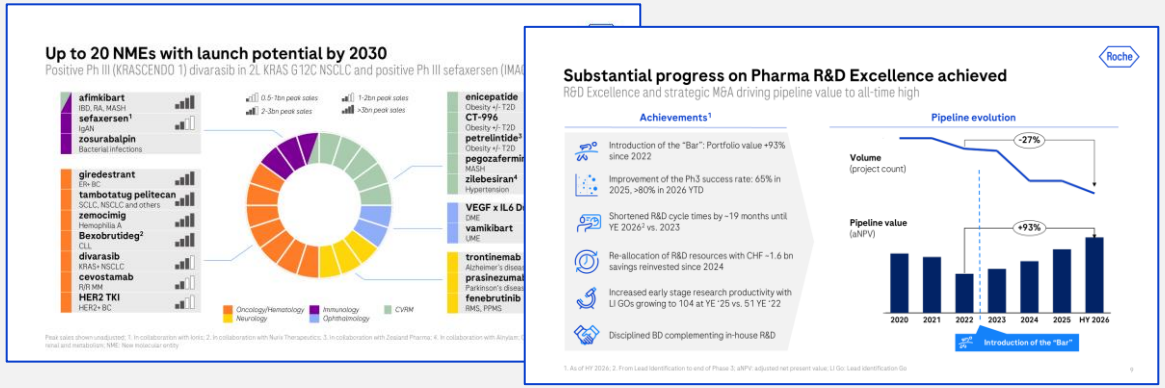
On-track to reach our 2030 ambition

R&D Excellence drives...



- Majority of solutions fully implemented, as tracked by R&D productivity KPIs
- Systematically applying the Bar to all in-house developed and partnered assets
- Continued augmentation of our R&D engine

...our next wave of innovation



- Up to 20 NMEs with launch potential by 2030, majority with a peak sales potential of CHF 3bn+
- Balanced portfolio mix allowing for future development optionality of NMEs in the five focus TAs, including in disease areas new to Roche
- Disciplined resource reallocation leading to an all-time high in the total portfolio value (aNPV)

aNPV: adjusted net present value; BD: Business development; KPI: Key performance indicator; NME: New molecular entity

Revolutionizing drug R&D with Lab-in-a-Loop

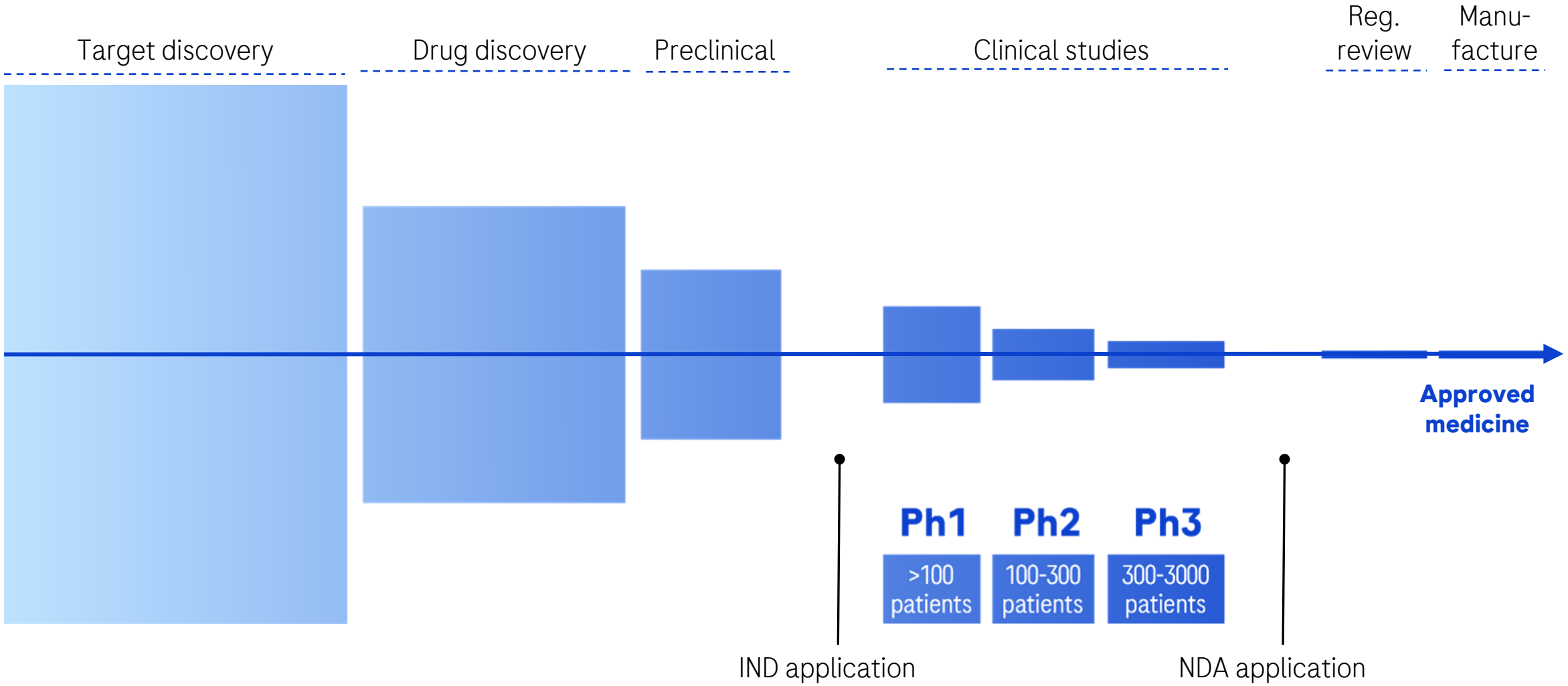
Aviv Regev
Genentech Research and Early Development (gRED)

Genentech
A Member of the Roche Group

September 28th, 2026

Drug R&D is a challenging process with a low success rate

Typically, only ~1-2 of 100 programs initiated in research will lead to a launch



Why is drug R&D so difficult?

BIG NUMBERS

Enormous search spaces

MULTI-SCALE

Atoms, genes, proteins, cells, tissues, organs, patients

MULTI-MODAL

Multiple measurements / views of each entity

VARIED

No two research paths are quite the same

AI has the potential to tackle each of these challenges

BIG NUMBERS

Enormous search spaces

MULTI-SCALE

Atoms, genes, proteins, cells, tissues, organs, patients

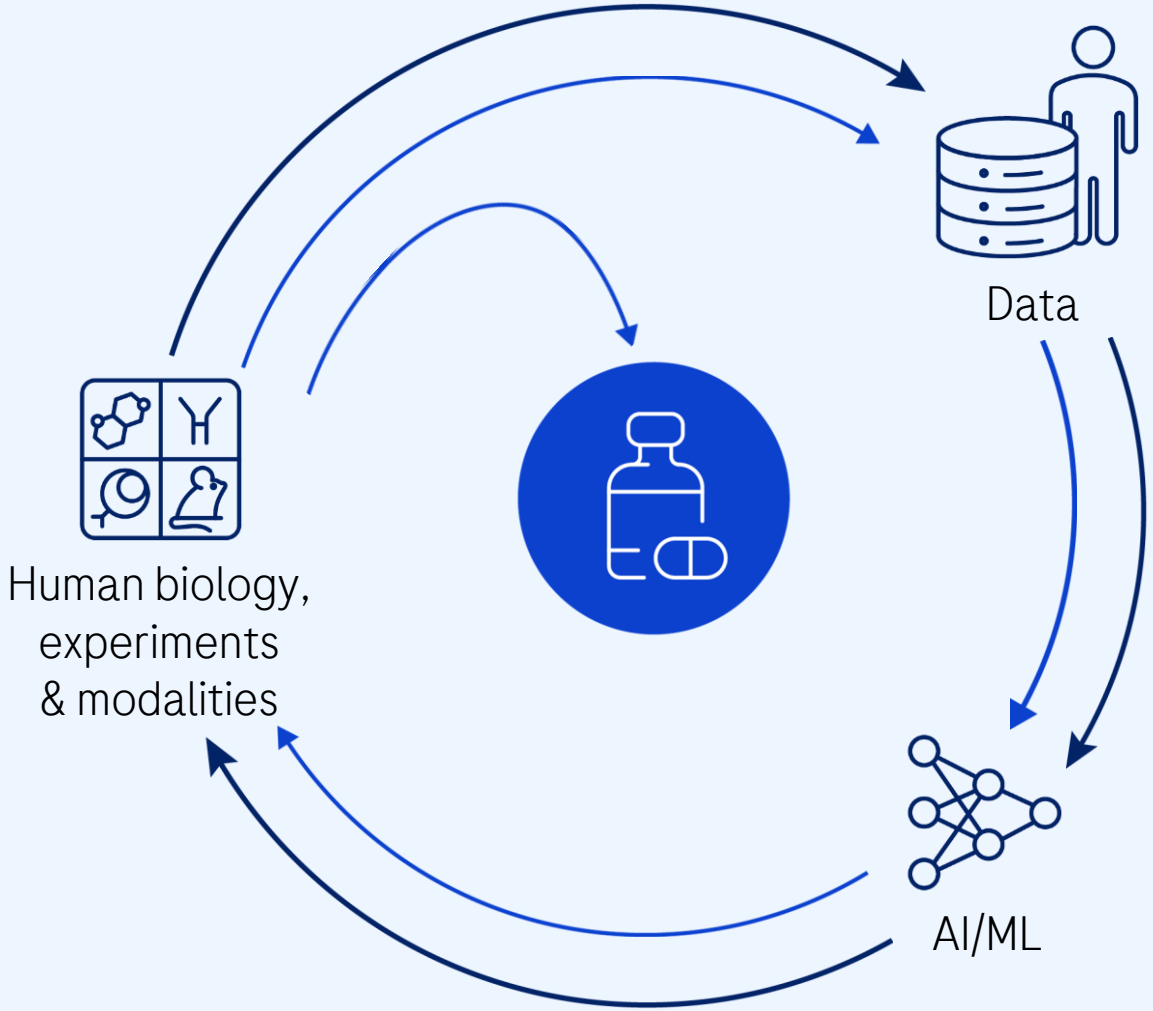


MULTI-MODAL

Multiple measurements / views of each entity

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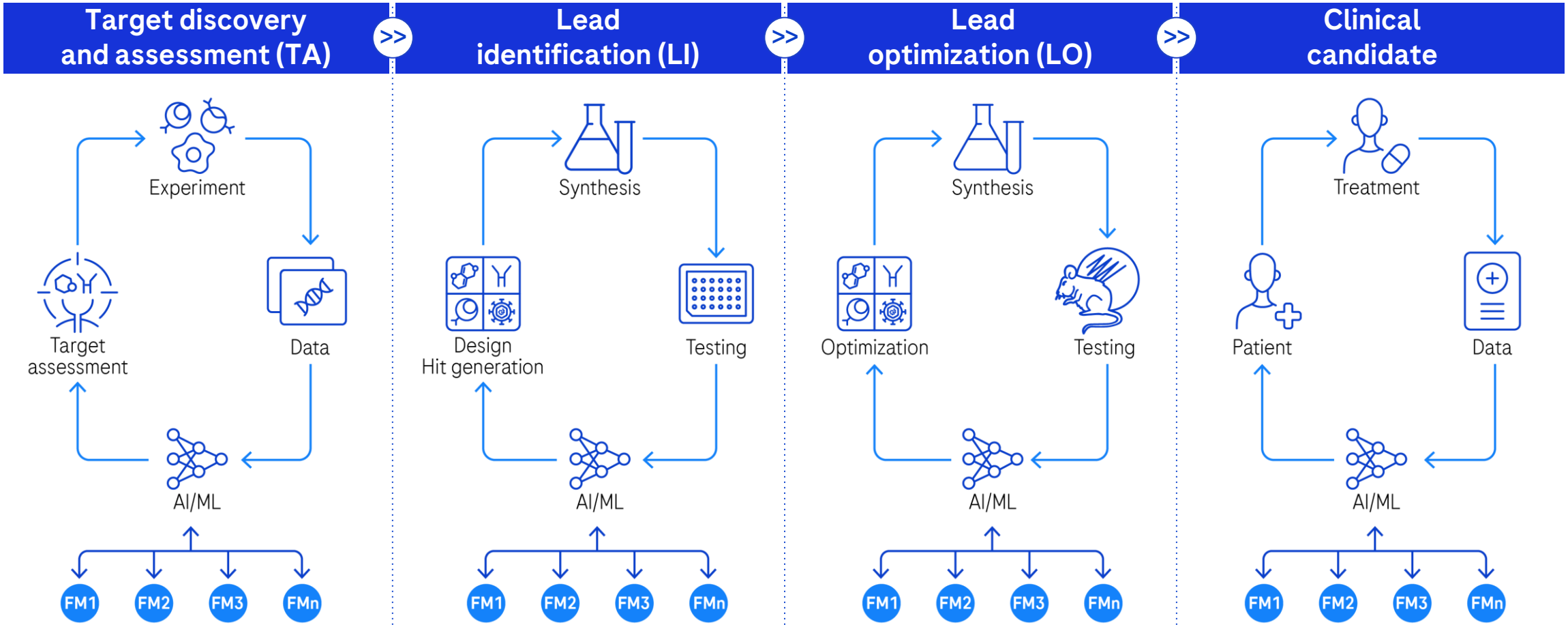
No two research paths are quite the same



LAB
(OR CLINIC)
IN THE LOOP

Lab-in-the-Loop in drug R&D

Iterations with AI embedded E2E, across each component, from target discovery to the clinic



AI/ML, Artificial Intelligence and Machine Learning; FM, Foundation model; LI, Lead identification; LO, Lead optimization; R&D, Research and development; TA, Target assessment

Our journey from build to delivery



Our journey from build to delivery

Strategy-driven investment across talent, AI capabilities, org design, data generation capacity & compute

2020 2021 2022 2023 2024 2025 2026

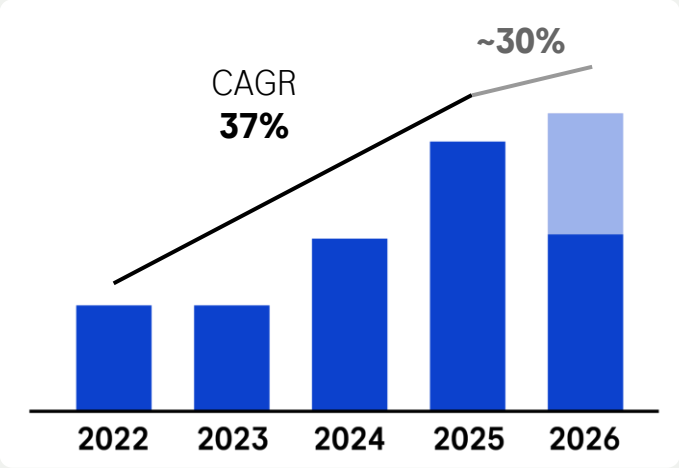
	2020–2022 **Build the foundation**	2023–2024 **Scale…**	2025–2026 **Transform…**
Strategy & capability	• Lab-in-the-Loop strategy • Multiplicative Levers	• Prescient expansion (all molecules) • Herophilus[1] automation software • AWS Research Agent collaboration	• Medra physical AI, MHS (Anthropic[1]) • Autonomous Lab Accelerator
Organization & talent	• BRAID (biology AI) • PrescientDesign[1] (antibodies AI)	• gRED Computational Sciences (gCS) consolidated	• Computational Sciences Center of Excellence (serves both REDs)
Data generation	• Cell & Tissue Genomics; Translational Genomics • Recursion collaboration[1]	• A-alpha Bio collaboration[1]	• Foundational Datasets for FMs initiative • Caris collaboration • LitL2.0 initiative
Compute & software		• NVIDIA partnership[1]	• OpenAI pilot partnership[1] • AI factory (NVIDIA)
Example impact milestones	1st LitL small molecule cycle 1st LitL large molecule cycle	1st LLM trained on Roche data 1st IBD pathobiology map + 15 LI targets 1st LO small molecule scaffold with AI-supported design	1st LI target enters with AI agent support 1st Antibody with AI support enters development portfolio (FPI) AI participating: 100% large molecules / 50% small molecules[2]

1. Partnering/acquisition; 2. ~90% of eligible projects | Notes: BRAID: Biology Research AI Department; AI: artificial intelligence; LLM: large language model; LitL: Lab in the loop; LI: lead initiation; LO: lead optimization; SM: small molecules; IBD: Inflammatory bowel disease; gCS: gRED computational sciences;

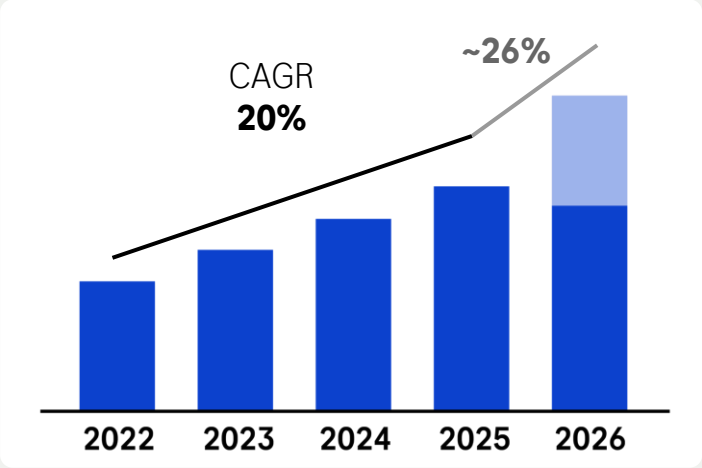
Steady increase in research portfolio productivity 2022-2026

Increase from LI-Go to LO-Go to development candidates under nominally flat resources and halving LI CT

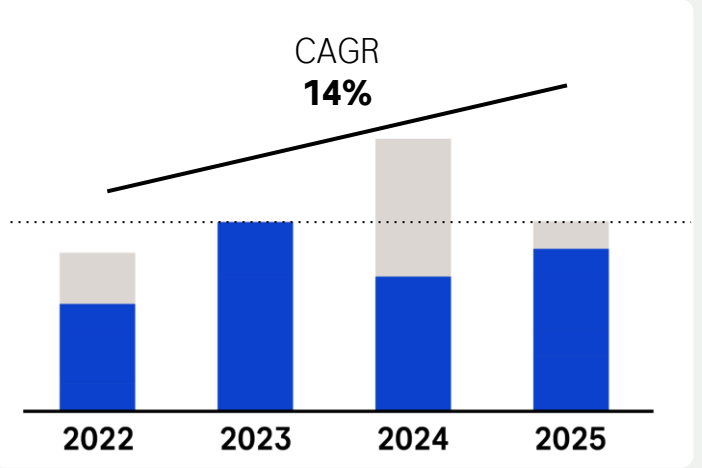
Lead Identification Go



Lead Optimization Go



Internal Development Go



2022-2025 growth

+155% LI Go

+75% LO Go

+50% internal DevGo

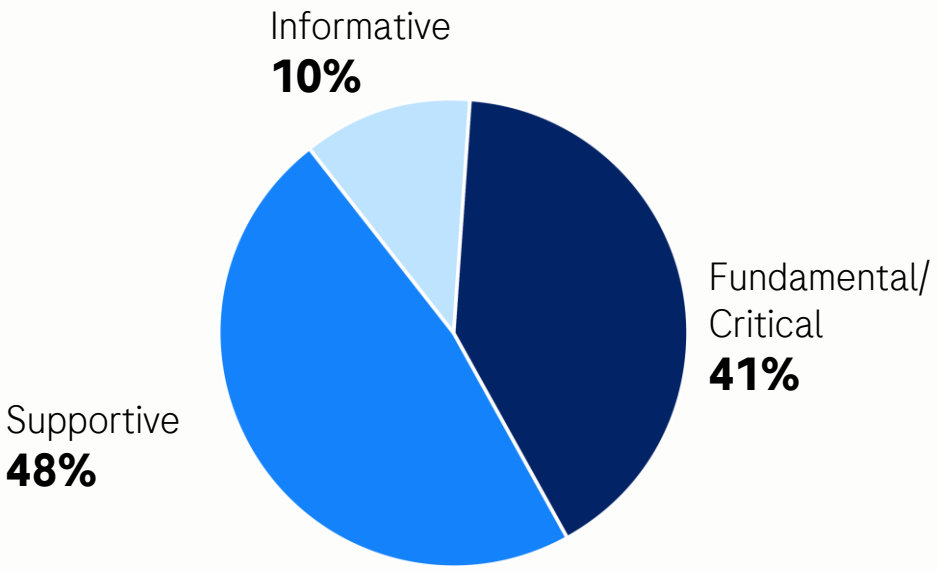
Actual · Forecasted · Business deals · ····· Historical number

CAGR, Compound annual growth rate; LI, Lead identification; LO, Lead optimization

AI + computation contribute across the pipeline

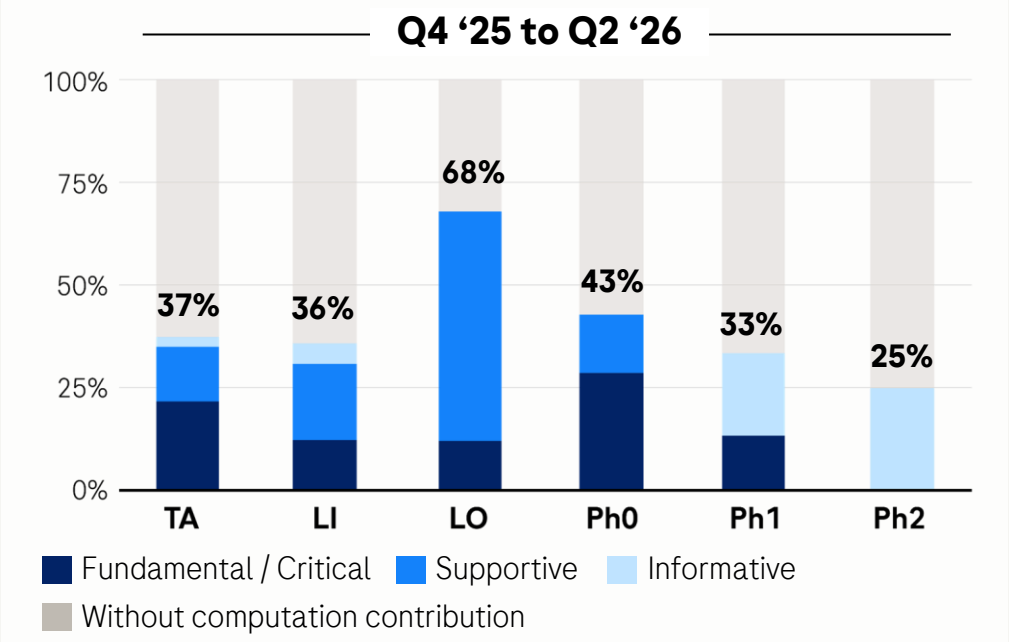
40% of Q4 ’25 through Q2 ’26 pipeline decisions had a tracked AI / computational contribution

Role of contributions in decision making



- **Critical:** Materially influenced
- **Supportive:** Meaningfully impacted decision confidence
- **Informative:** Informed thinking

Impact on pipeline decisions by phase

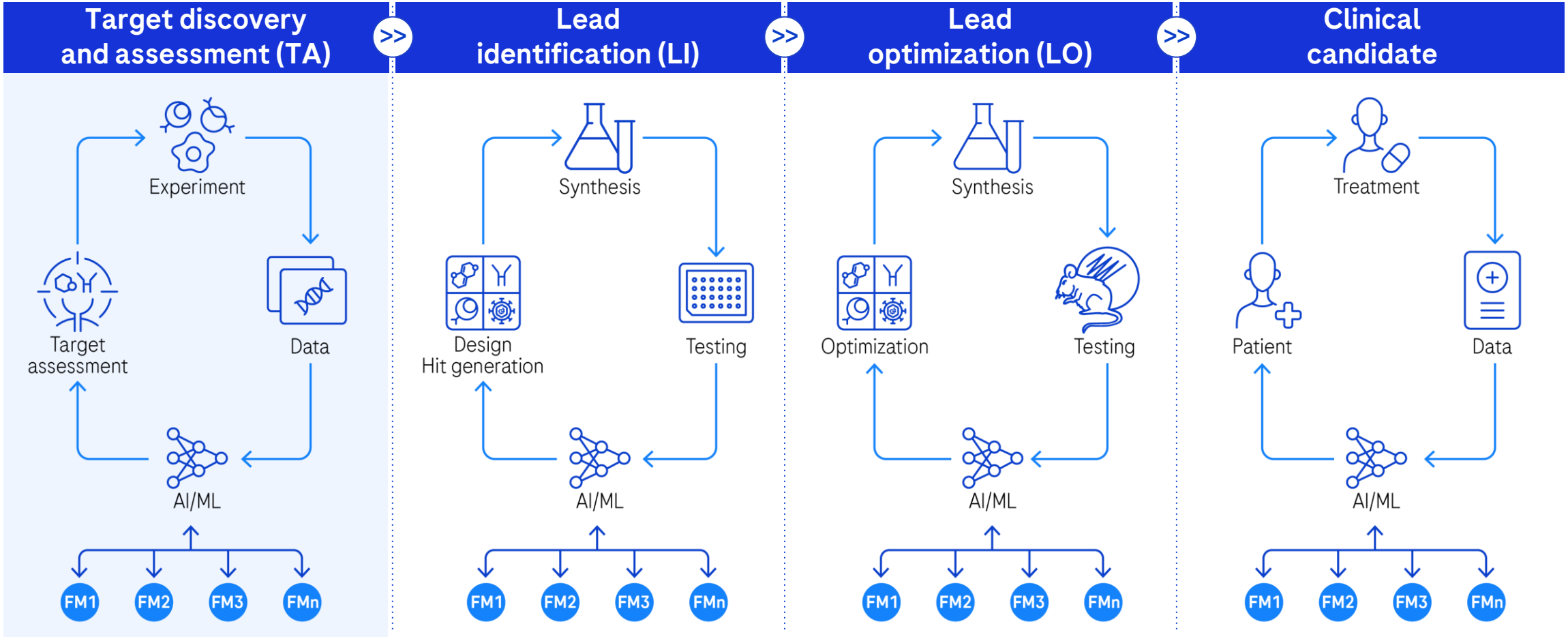


Cumulative coverage: 40%
(Q2 coverage reached 44%)

AI, Artificial intelligence; CS CoE, Computational sciences center of excellence; LI, Lead identification; LO, Lead optimization; Ph, Phase; TA, Target assessment

Lab-in-the-Loop in drug R&D

Iterations with AI embedded E2E, across each component, from target discovery to the clinic

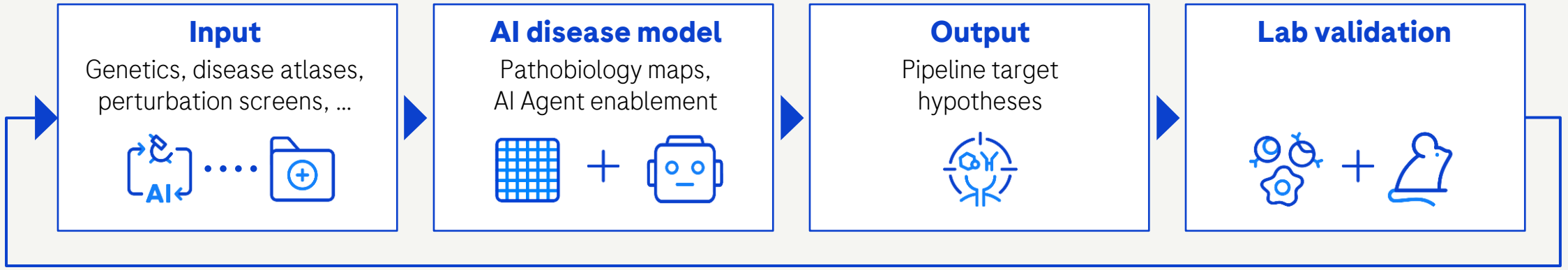


AI/ML, Artificial Intelligence and Machine Learning; FM, Foundation model; LI, Lead identification; LO, Lead optimization; R&D, Research and development; TA, Target assessment

Increase in high quality volume of research portfolio entries

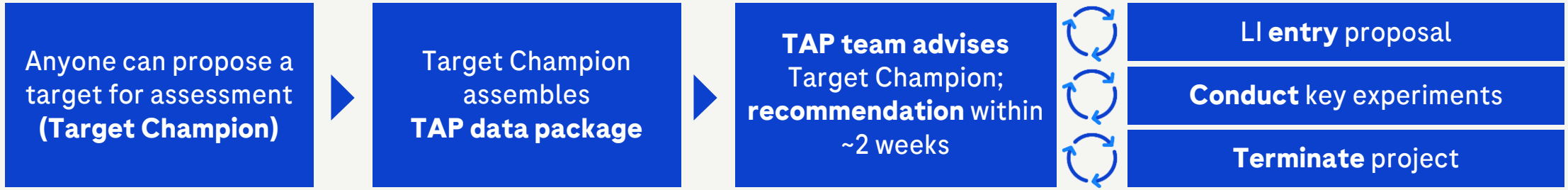
Large-scale human and lab data, AI, and new processes increase high-quality volume and speed

Innovative scientific foundation: AI-enhanced pathobiology maps for target discovery



New enabling process: Target Assessment Portfolio (TAP) Teams

Cross-functional system to ensure speedy target assessment, expert guidance and prioritized resourcing

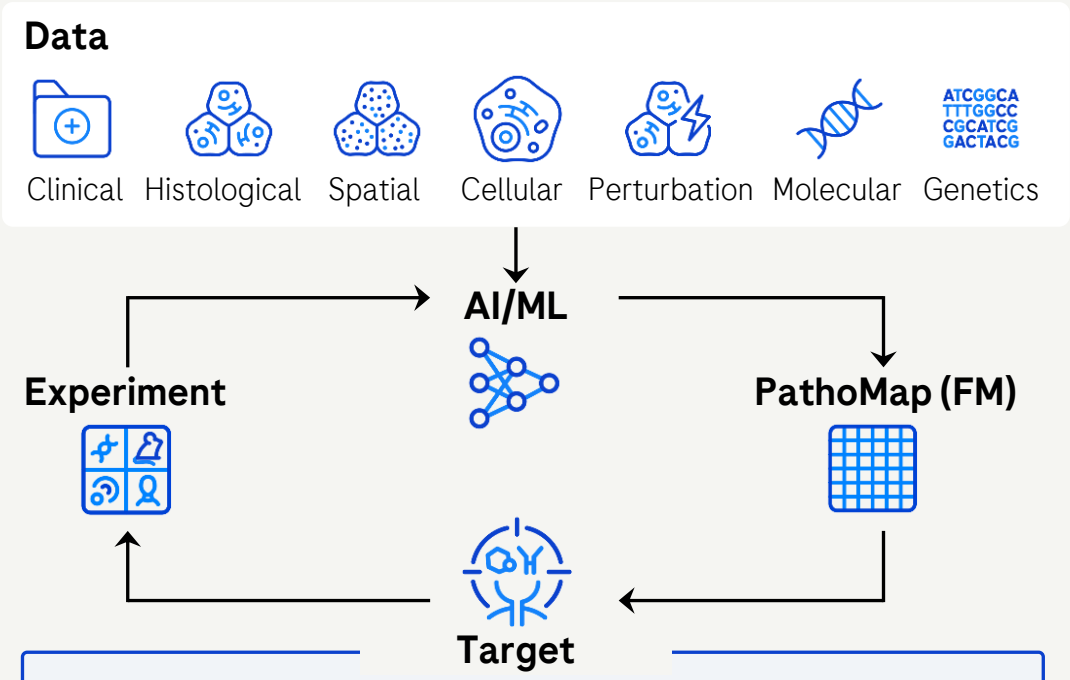


AI, Artificial intelligence; LI, Lead identification; TAP, Target assessment portfolio

Pathobiology maps have scaled and accelerated our pipeline

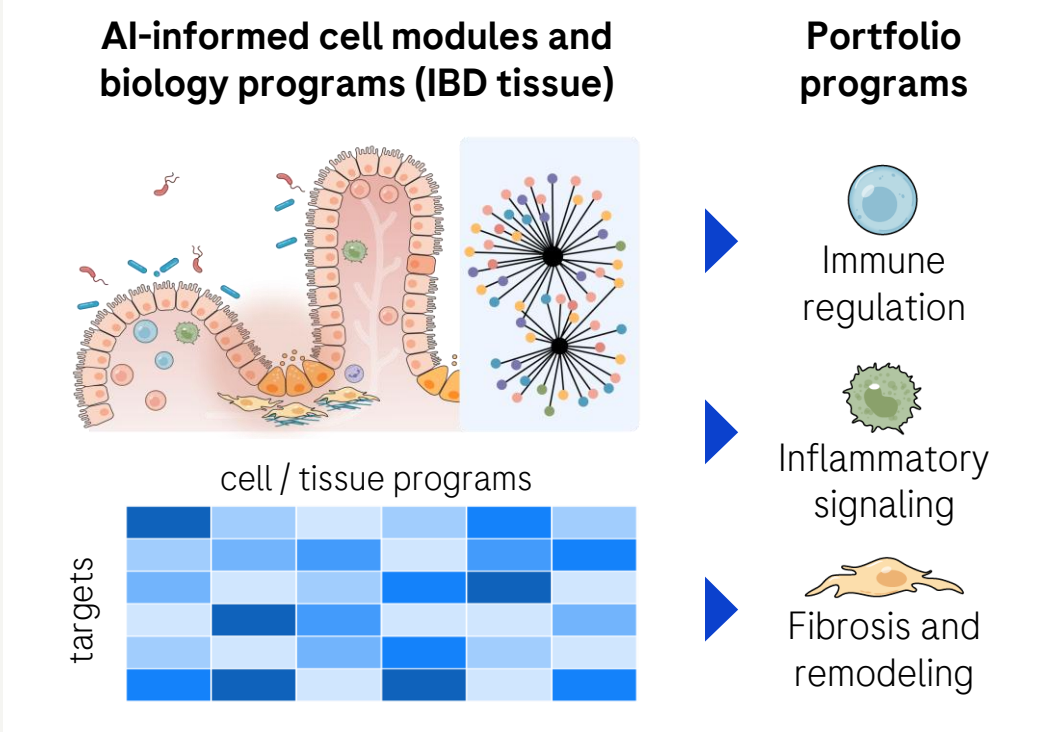
Example: IBD pathobiology maps

Goal: Data & FMs to help break the efficacy ceiling



- New drug targets
- Molecule discovery
- New biomarkers
- New combination therapies

Example: IBD Pathobiology Map v 2.0



- Example map: cells x spatial context x human genetics
- Multi-modal data power IBD target discovery

AI, Artificial intelligence; IBD, Inflammatory bowel disease; FM, Foundation model; PathoMap, Patho-biology map

AI-enhanced pathobiology maps across our TAs

Oncology	Immunology	Neurology	Ophthalmology
Cell surface targets across tumors	COPD IBD	Alzheimer's MS Parkinson's ALS	AMD

1 YEAR

First map deployed (IBD)

6 WEEKS

4 neurology maps deployed

+155%

increase in LI entries in 2 years

ALS, Amyotrophic lateral sclerosis; AMD, Age-related macular degeneration; COPD, Chronic obstructive pulmonary disease; IBD, Inflammatory bowel disease; LI, Lead identification; MS, Multiple sclerosis

Foundational Datasets for Foundation Models initiative

Large-scale data generation dedicated for training of AI models of relevant disease biology

Foundational datasets (FD)

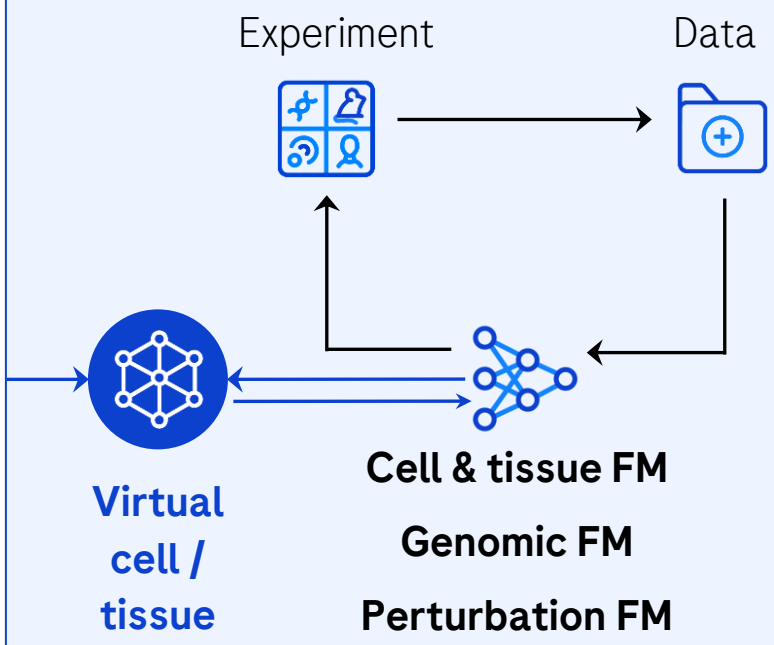
Sequence-to-phenotype
Human genetics
Massive scale lab assays

Perturbations
Genetics and compounds
Cells, organoids, tissues

Patient tissues
Healthy & disease

Clinico-genomics
Mechanisms & patient outcomes

Foundation models (FM)



Target discovery and prediction
→ Find causal drivers

Mechanistic insight
→ Understand how targets alter cell or tissue state

Target-hopping
→ Find alternative therapeutic targets

Target deconvolution
→ Identify what drives an observed compound's effect

Safety
→ Detect on- and of-target effects

FD, Foundational datasets; FM, Foundation models

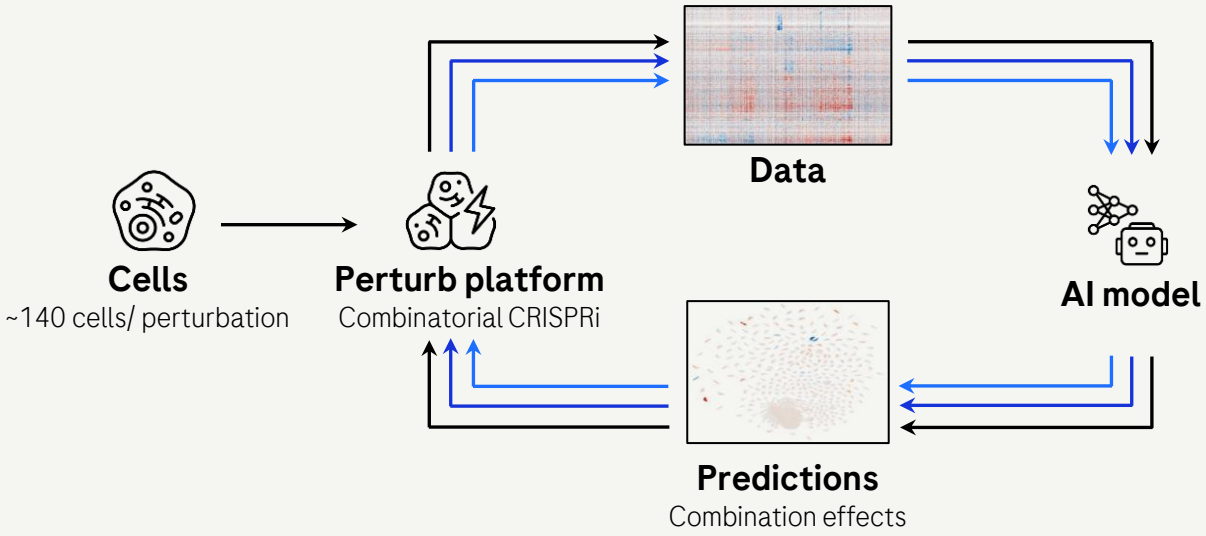
Tackling biological search spaces with active learning

Iterative AI-driven sampling efficiently explores the search space

Example: Iterative Perturb-Seq loop

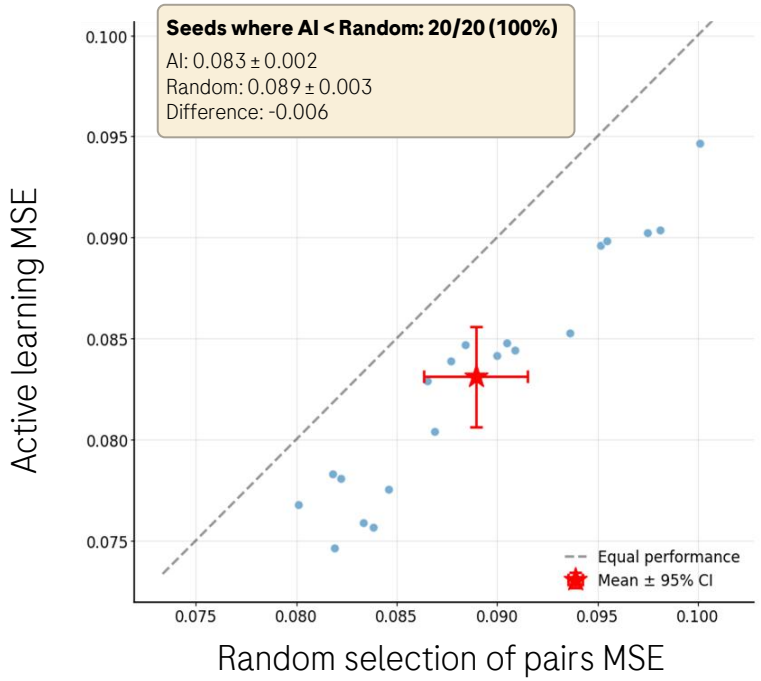
Search space: ~180k pairs over 600 impactful genes

Sampling: ~600 pairs / round



Round #1	Round #2	Round #3
No AI **~700** selected pairs	AI selected **600** pairs +Baseline: **400** randomly selected pairs	AI selected **600** pairs +Baseline: **400** randomly selected pairs

Active learning *vs.* baseline selection



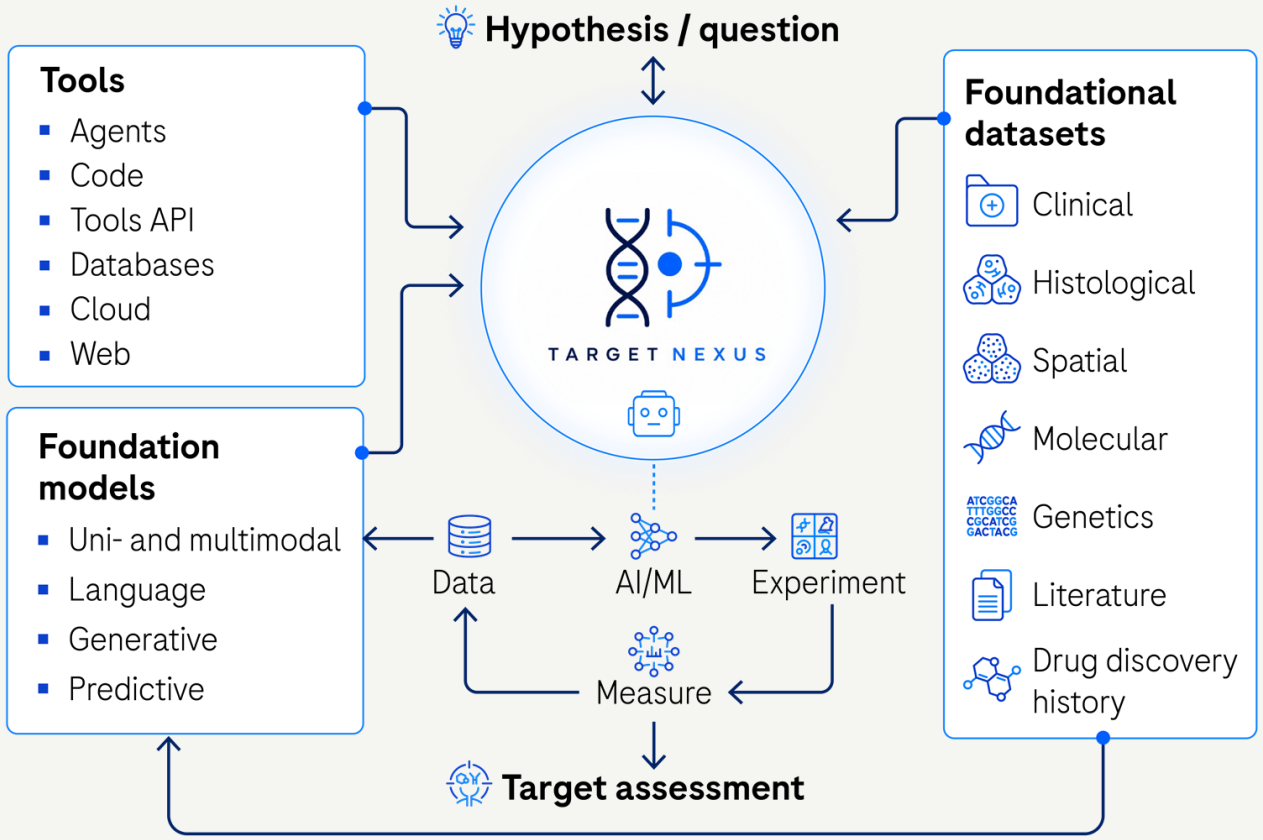
Active learning delivers better predicting model than strong baseline selection (random pairs over 600 impactful genes)

AI, Artificial intelligence; k, Thousands; MSE, Mean squared error

TargetNexus

Our AI agent for target assessment on track to be part of 80% of research portfolio decisions by Q4

Target discovery and assessment



Coverage across the pipeline

Target Biology

- Causal mechanisms
- Disease biology
- Disease models

Drug Discovery

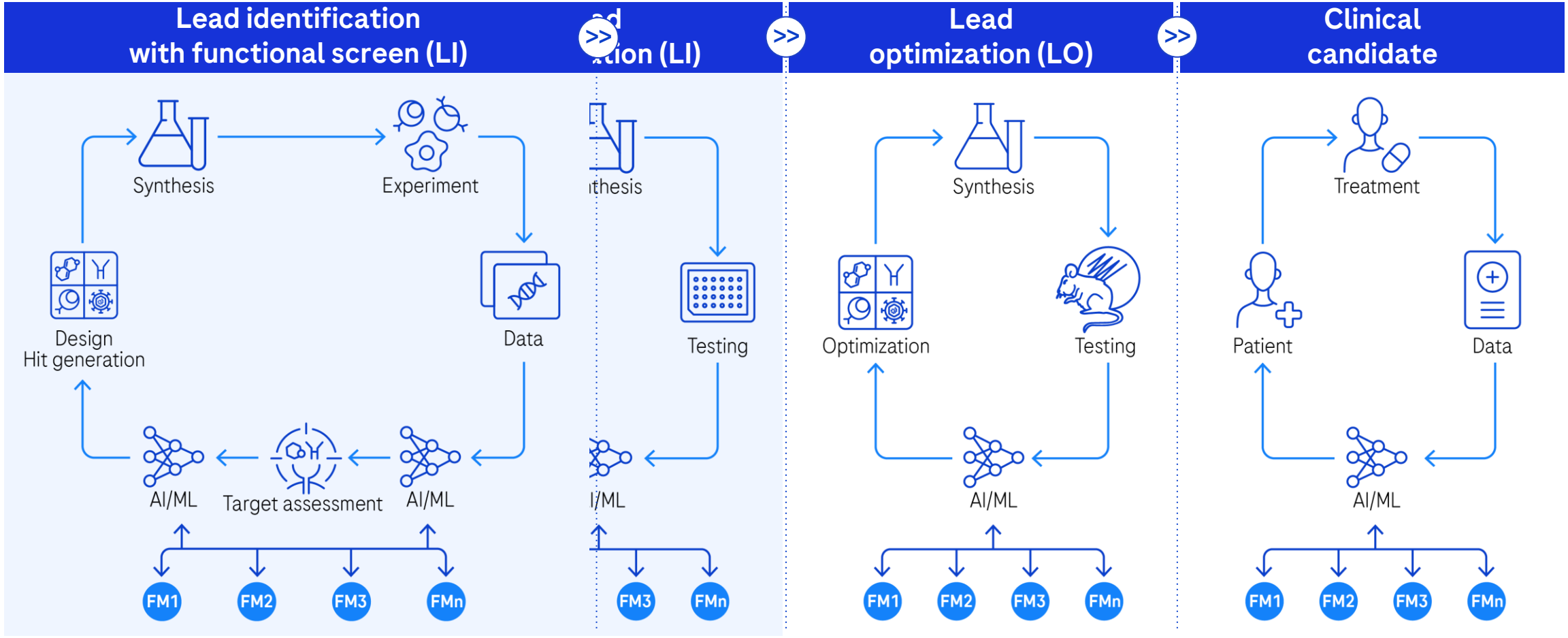
- Druggability
- Tractability
- Safety
- Modality

Clinical & Portfolio

- Patient population
- Biomarkers
- Portfolio fit
- Competitive landscape

Lab-in-the-Loop in drug R&D

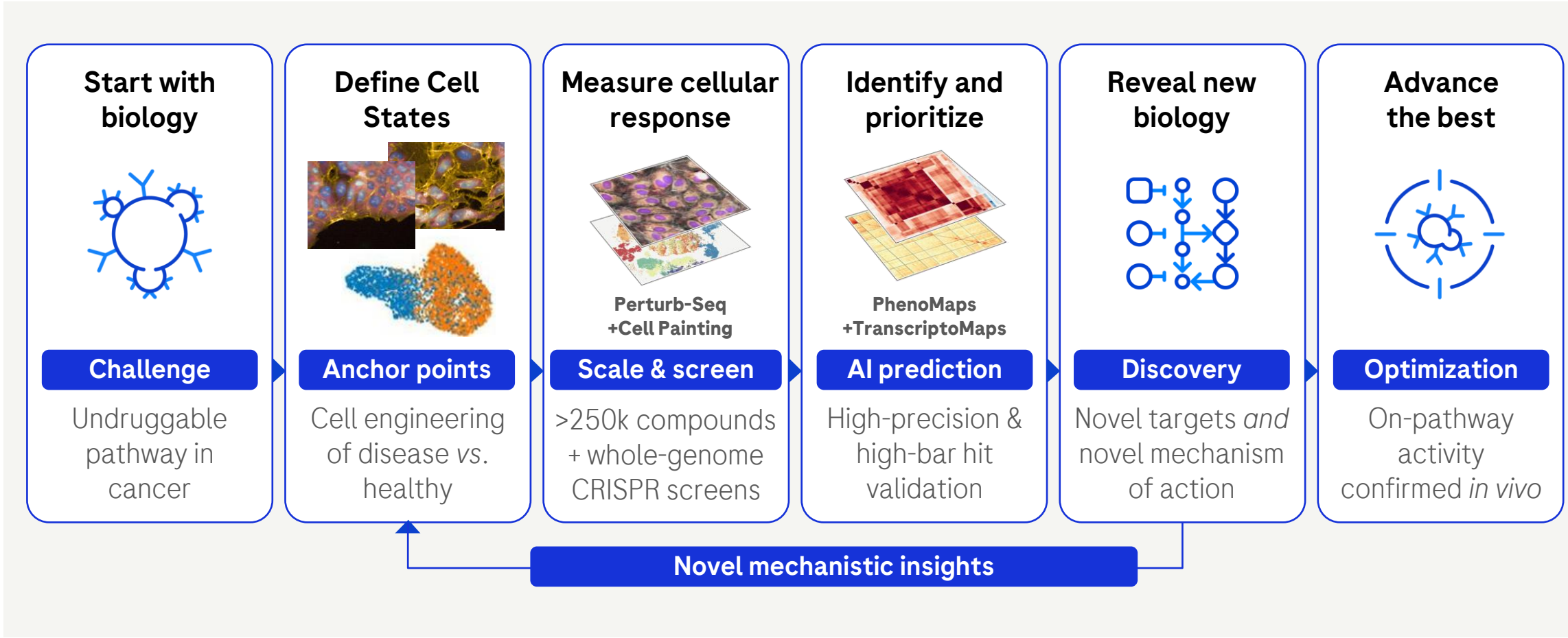
Iterations with AI embedded E2E, across each component, from target discovery to the clinic



AI/ML, Artificial Intelligence and Machine Learning; FM, Foundation model; LI, Lead identification; LO, Lead optimization; R&D, Research and development; TA, Target assessment

Lab-in-the-Loop applied to cancer target & drug discovery

Discovery of unique druggable MoA for a previously 'undruggable' pathway in oncology



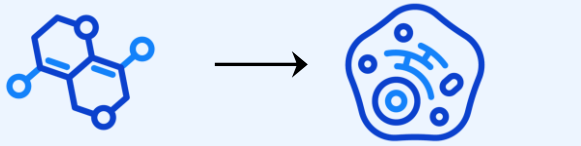
In collaboration with RECURSION

Virtual high-content small molecule phenotypic screens

Scale discovery from constrained lab process to dramatically larger virtual scale

~220K compounds screened in a lab, 90B (10^5-fold) space in ENAMINE to explore

Train foundation model

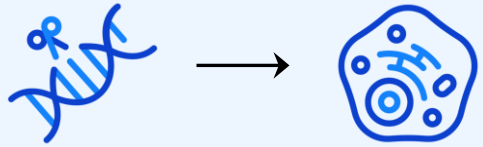


Input compound structure

Output measured phenotype

Example public dataset
116K compounds

Induce phenotype of interest in lab

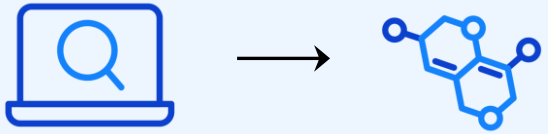


Anchor perturbation

Measure phenotype

Anchor perturbation

Conduct phenocopy virtual screen



Query the model virtually

Discover anchor phenocopies

Enamine virtual library
4B compounds screened

Prospective validation of a virtual cell phenotypic screen

mTOR/PI3K pathway inhibitors

Virtual screen and lab validation assays

Enamine REAL library

~4B compounds

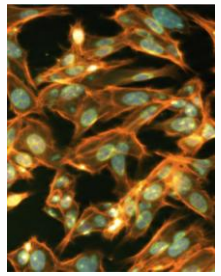
PhenoCompass

Predicted mTOR/PI3K pathway inhibitors

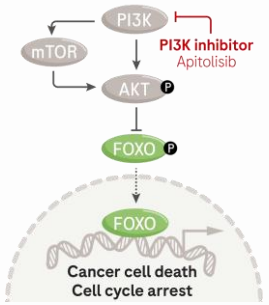
~500 compounds ordered (400 synthesized)

3 rounds of **experimental validation** with increasing specificity

Morphology

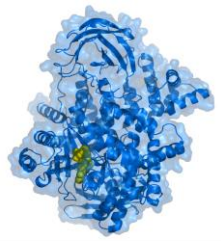


Pathway activity

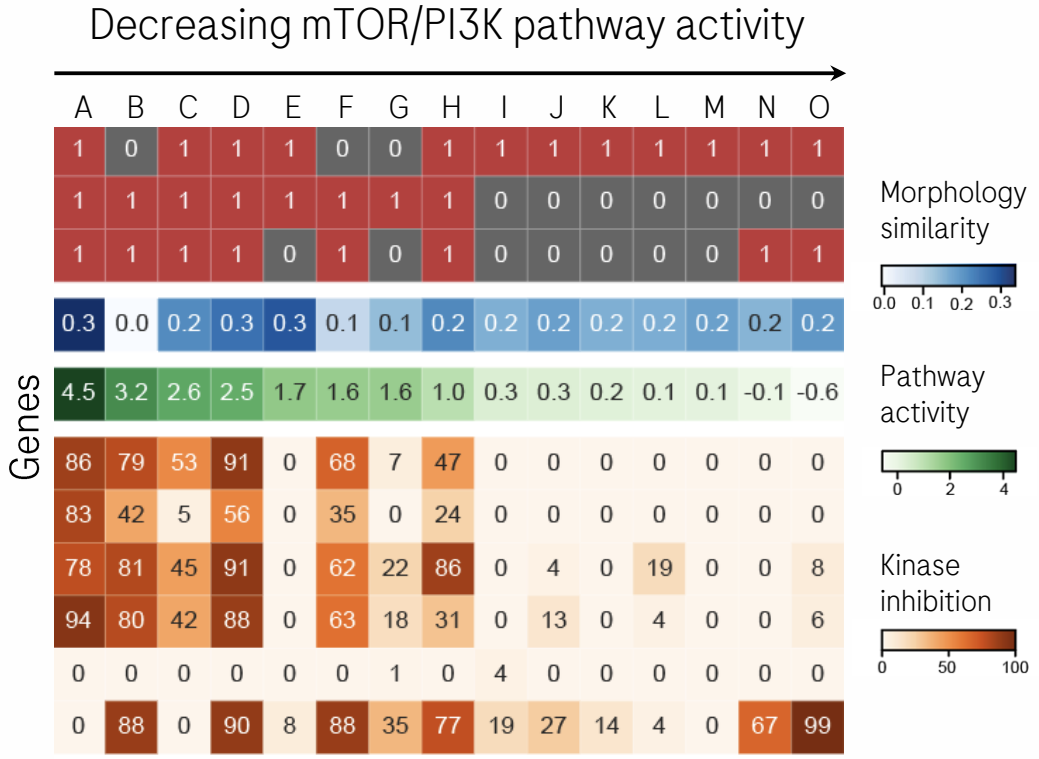




Kinase inhibition

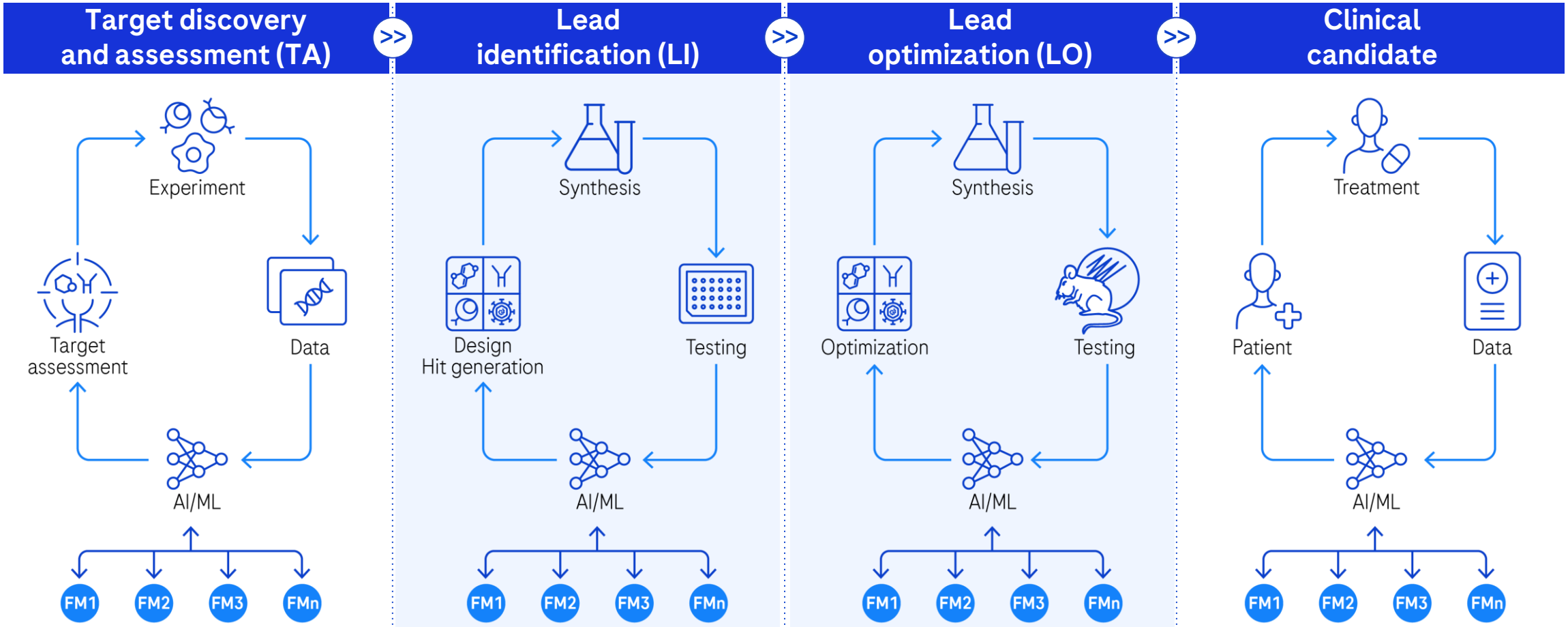


Hits with confirmed kinase inhibition; novel scaffolds



Lab-in-the-Loop in drug R&D

Iterations with AI embedded E2E, across each component, from target discovery to the clinic



AI/ML, Artificial Intelligence and Machine Learning; FM, Foundation model; LI, Lead identification; LO, Lead optimization; R&D, Research and development; TA, Target assessment

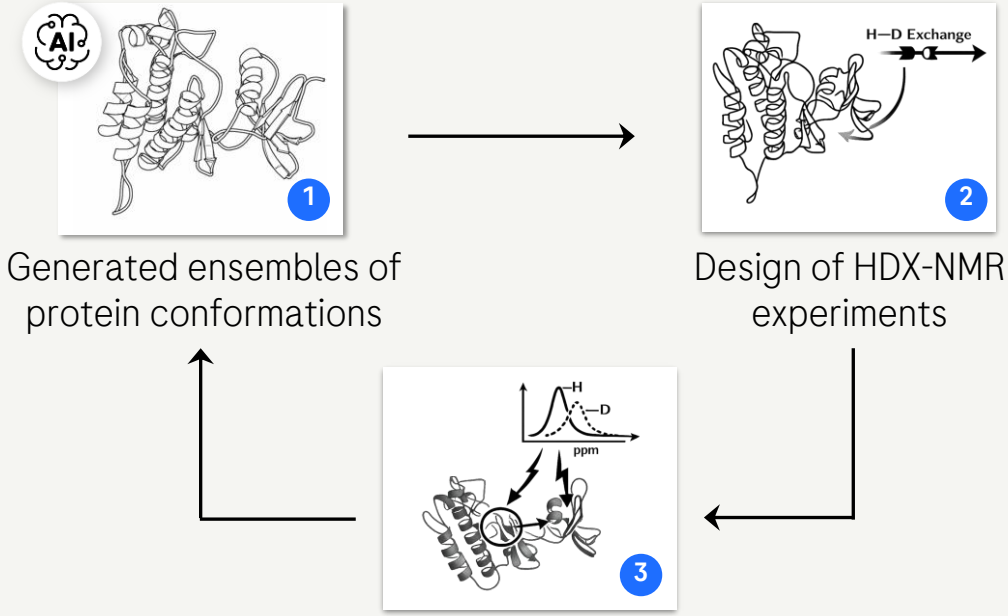
Small and large molecule drug discovery

AI can impact every stage of drug discovery

	Target assessment	Lead identification	Lead optimization
Small molecules	Druggability	Hit generation	Test → Analyze → Design → Make
Biologics	Lead discovery	Hit expansion	Test → Analyze → Design → Make

AI-powered structural biology accelerates hit finding

Identify druggable binding pockets



Generated ensembles of protein conformations

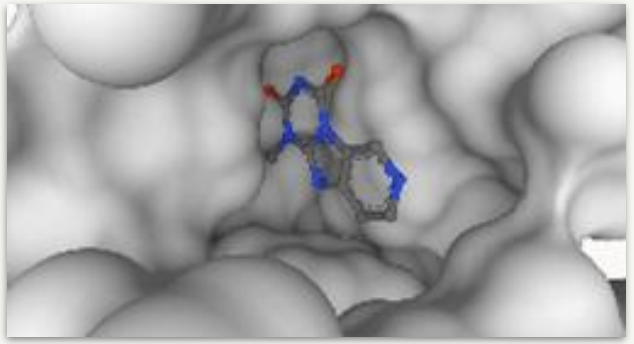
Design of HDX-NMR experiments

HDX-NMR maps solvent-exposed regions

Molecular hit finding

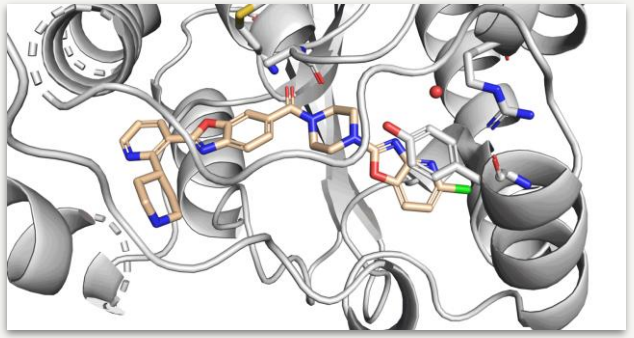
VoxBind

Pocket-conditioned voxel-denoising generative model



SAGE

Multi-property small molecule ligand design



Generic timeline — **X** months

First scaffold (New lab-based approach) — X **- 40%** months

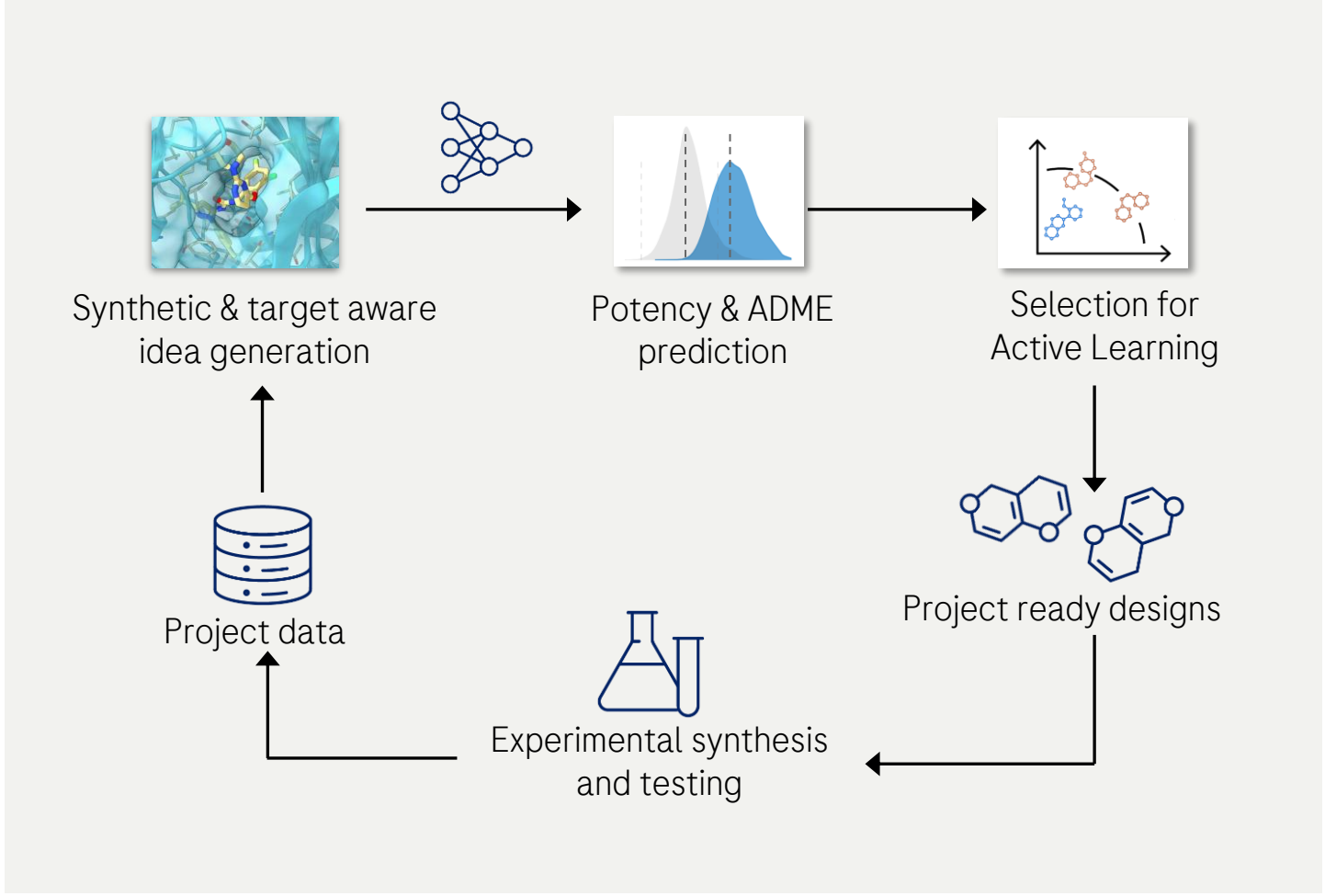
First scaffold (AI-enabled virtual screen) — X **- 80%** months — X **- 90%** months

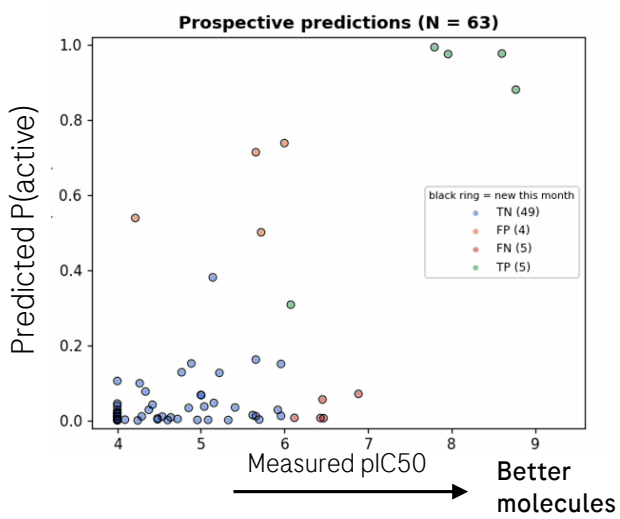
Hit #1 (first synthesis) — **Hit #2**

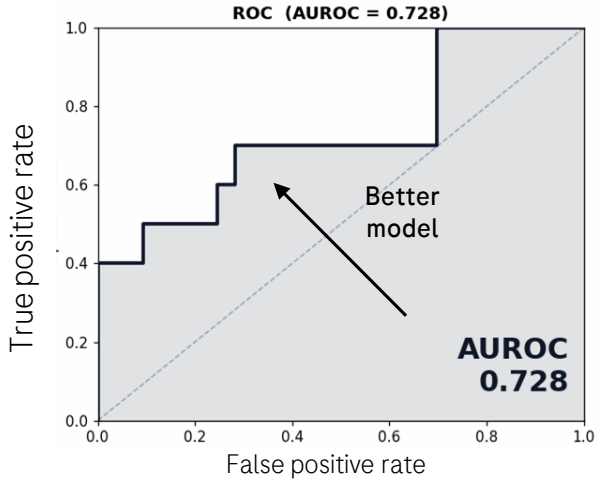
ICML 2026 GenBio Workshop Spotlight: Boltz-Jump: Accelerated Sampling of the Conformational Landscape of Biomolecular Structure Prediction Models. Daigavane *et al.*

Lab-in-the-Loop for small molecule optimization

Weekly active learning improves both molecules and models

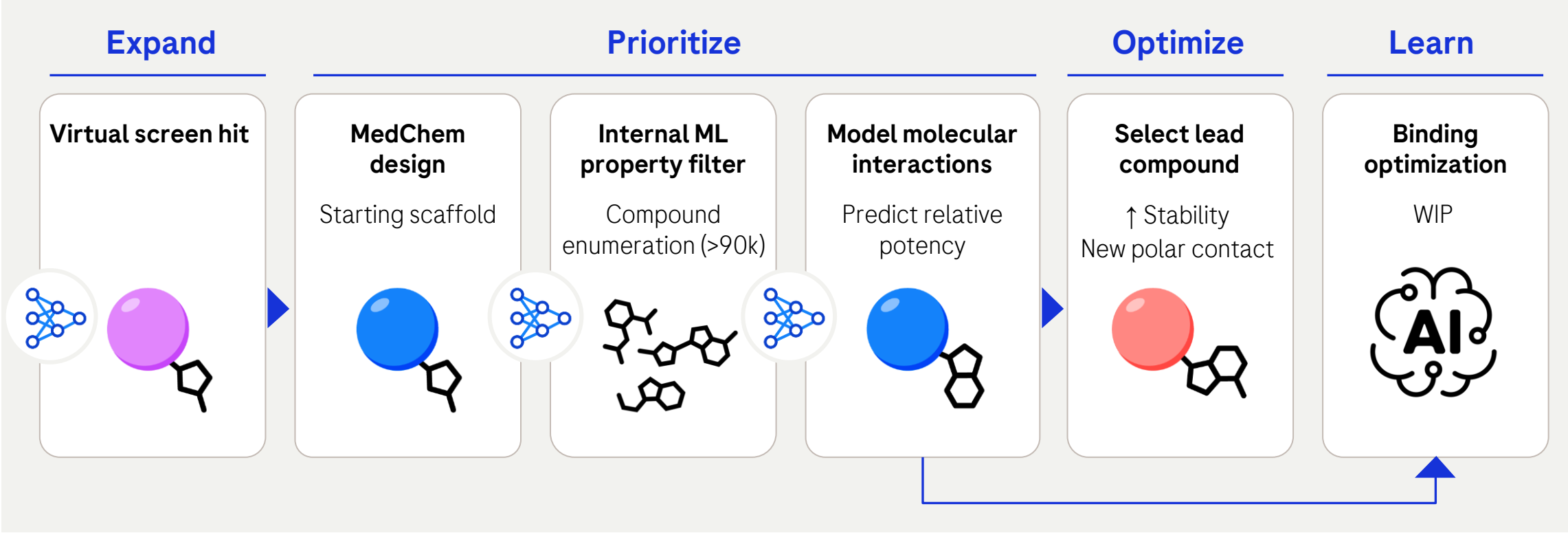






AI+physics-guided scaffold hopping accelerates LO

Optimizing metabolic stability while maintaining potency



Time
<35 min to generate & filter ~100K structures

Scale
>1k compounds

Potency
~40x improvement

An AI Agent supports SM drug discovery

Connecting context, SAR, design and computation to accelerate decisions

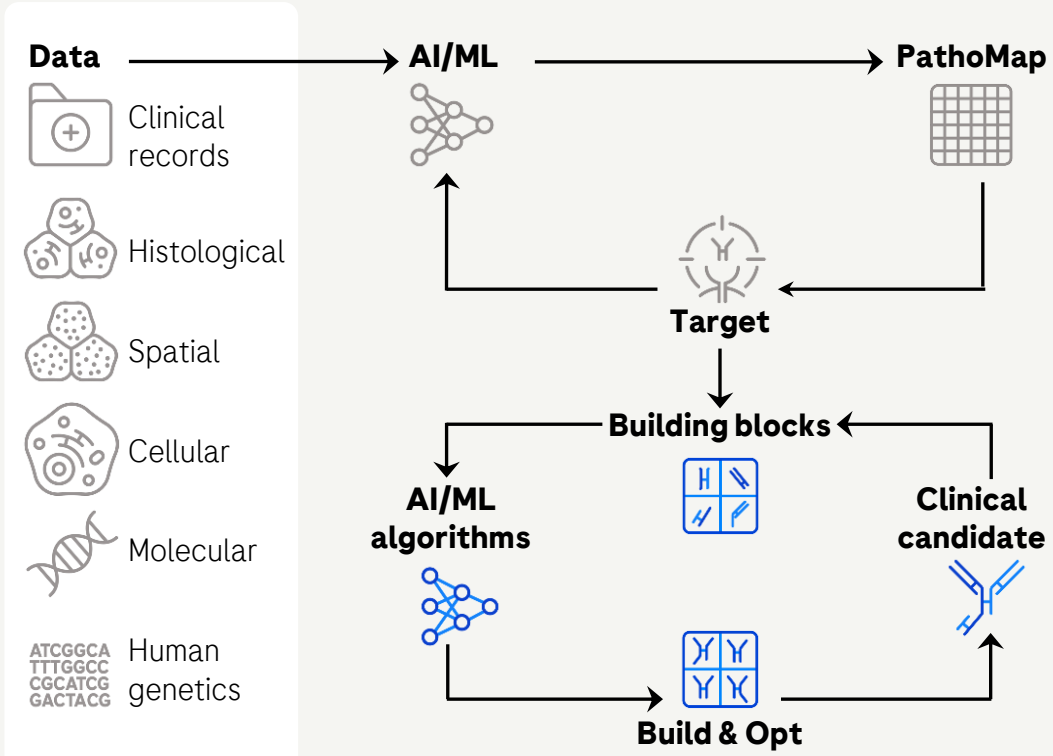
	Scientific foundation	AI support
Models & tools	Co-folding, docking, ADME and potency models, free energy perturbation, enumeration, etc.	**LLM + Harness**
Skills	Structural biology, structure-activity analysis, model evaluations, plotting and visualizations, etc.	**Reasoning** about project science and compound design
Data	Structure-activity project data, experimental structures, DesignHub, SM building blocks, etc.	**Orchestration** tools, models and workflows
Context	Project-specific context, documentations / history, medchem knowledge	

ADME, Absorption, distribution, metabolism, excretion; LLM, Large language model; SAR, Structure-activity relationship; SM, Small molecule

Building next-gen multispecifics to improve outcomes

Antibody building blocks and multi-program execution boost PTS, speed biologics, reduce costs

Data & AI driven target discovery



Foundational building blocks for next-gen multi-specifics against relevant targets

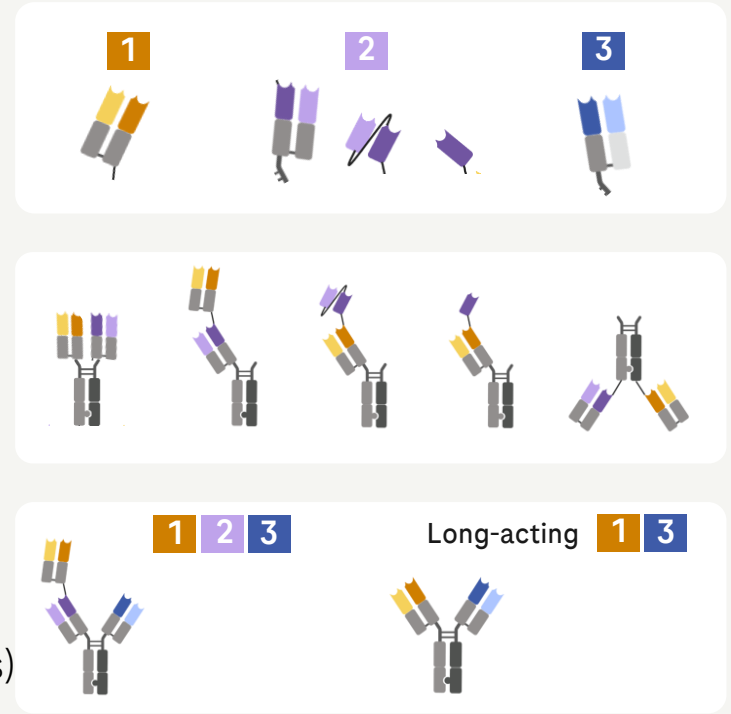
Foundational building blocks (e.g. 100+)

↓

Assembly and optimization (e.g., 5 programs)

↓

Rapid generation of clinical candidate (e.g., 2 of 5 programs)



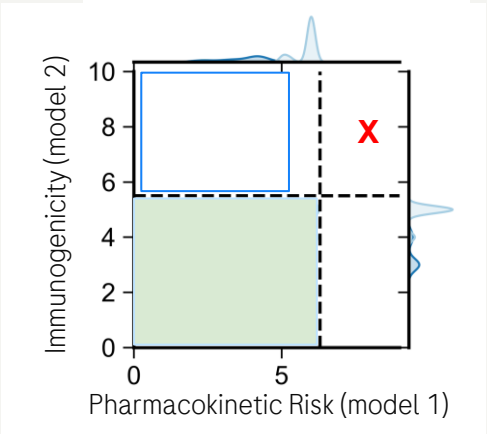
Integrated AI to optimize lead arms with low developability risk

Improved multi-specifics from building blocks

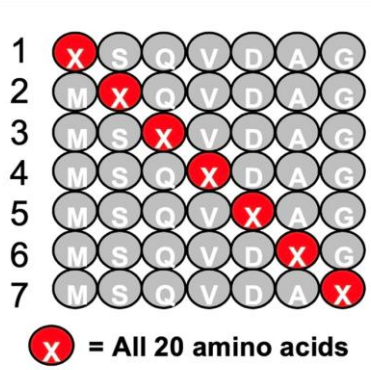
Example: Parallel efforts for each of 4 targets, for multiple bi- and tri-specifics

Rat, mice, rabbit, llama → High affinity and cross-reactivity → Potent function as monovalent arm → Lead and backup arms for each target

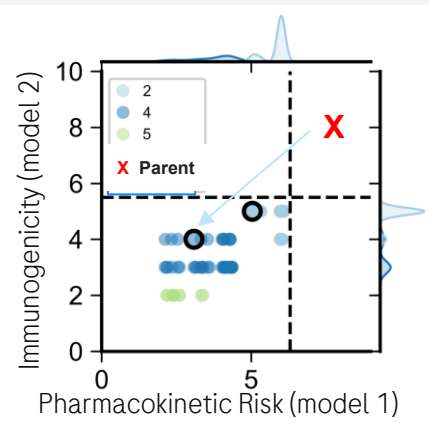
Integrated experiments and computation help optimize lead arms to lower developability risk

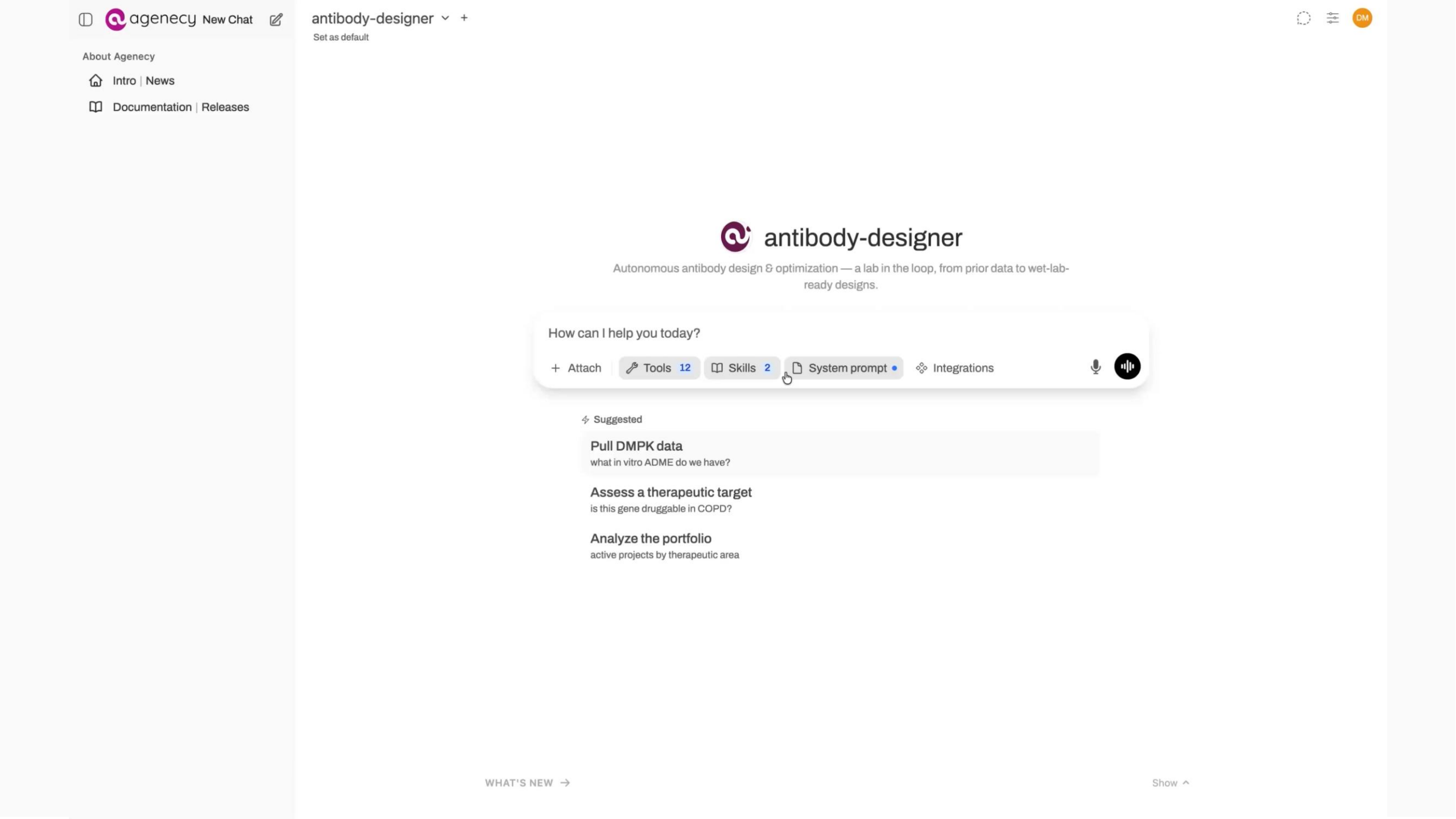


High-throughput experimental affinity data set: ~1,200 single mutants



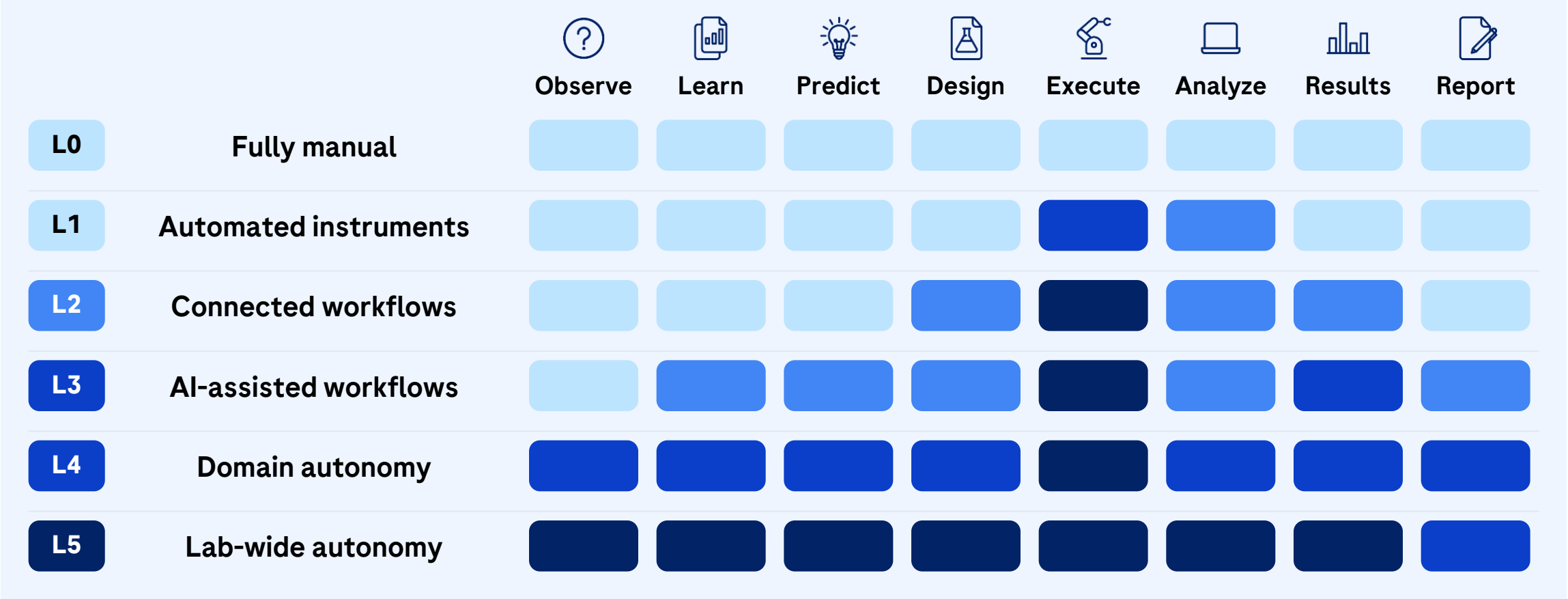
Sequence optimization




agenecy New Chat
antibody-designer
Set as default
DM
About Agenecy
Intro | News
Documentation | Releases
antibody-designer
Autonomous antibody design & optimization — a lab in the loop, from prior data to wet-lab-ready designs.
How can I help you today?
Attach
Tools 12
Skills 2
System prompt
Integrations
Suggested
Pull DMPK data
what in vitro ADME do we have?
Assess a therapeutic target
is this gene druggable in COPD?
Analyze the portfolio
active projects by therapeutic area
WHAT'S NEW
Show

Towards autonomous labs for drug R&D

Our Autonomous Lab Accelerator to enhance the loop



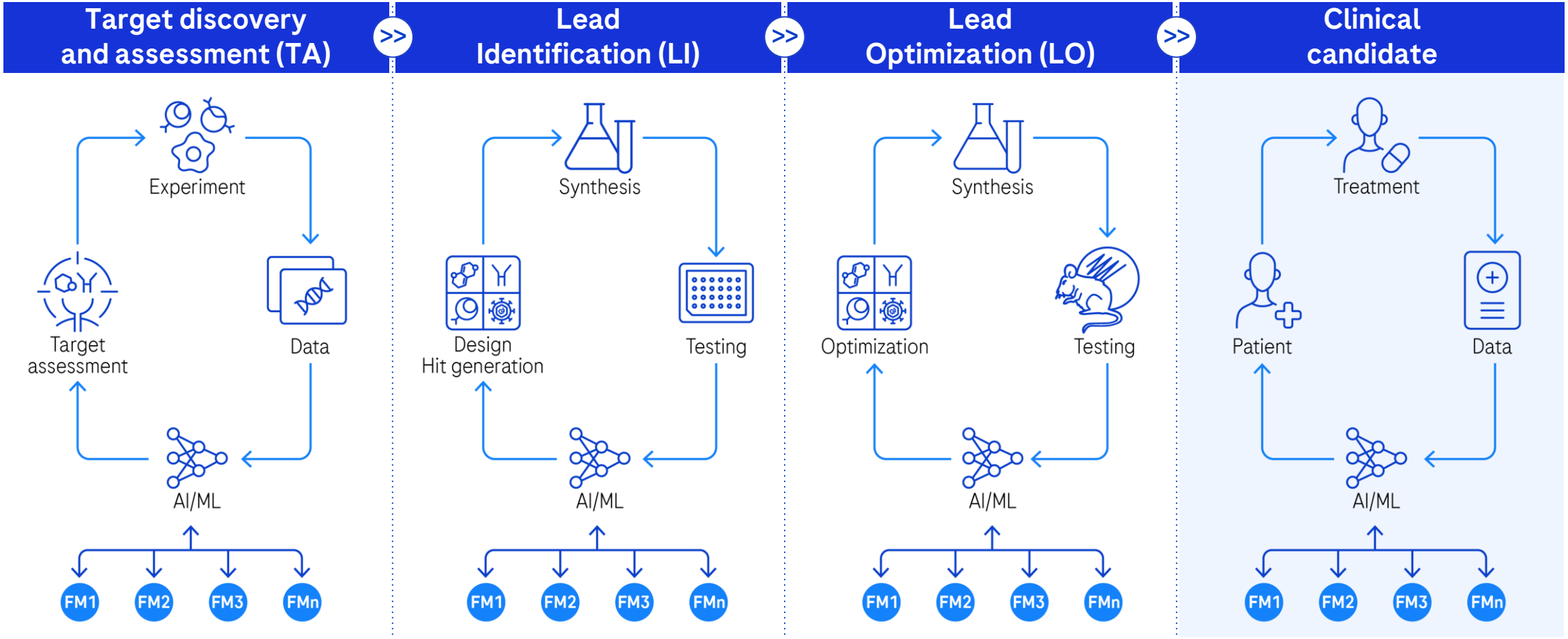
Human role: Operator | Assistant | Approver | Auditor

Including collaborations with Medra ANTHROP\C

Cheng et al. *Preprints.org* 2026 (in review)

Lab-in-the-Loop in drug R&D

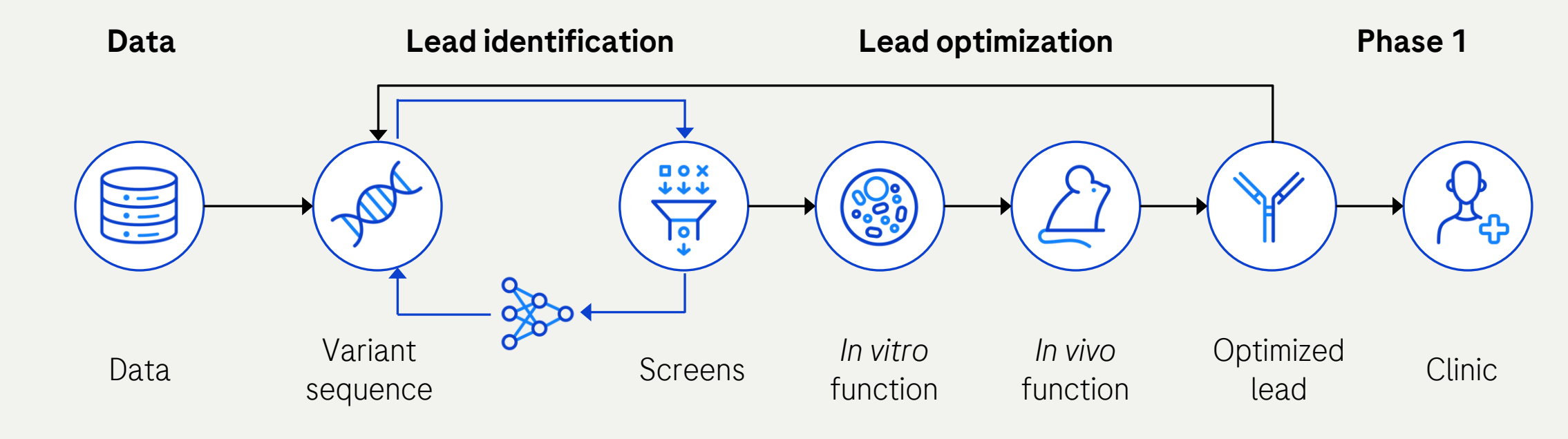
Iterations with AI embedded E2E, across each component, from target discovery to the clinic



AI/ML, Artificial Intelligence and Machine Learning; FM, Foundation model; LI, Lead identification; LO, Lead optimization; R&D, Research and development; TA, Target assessment

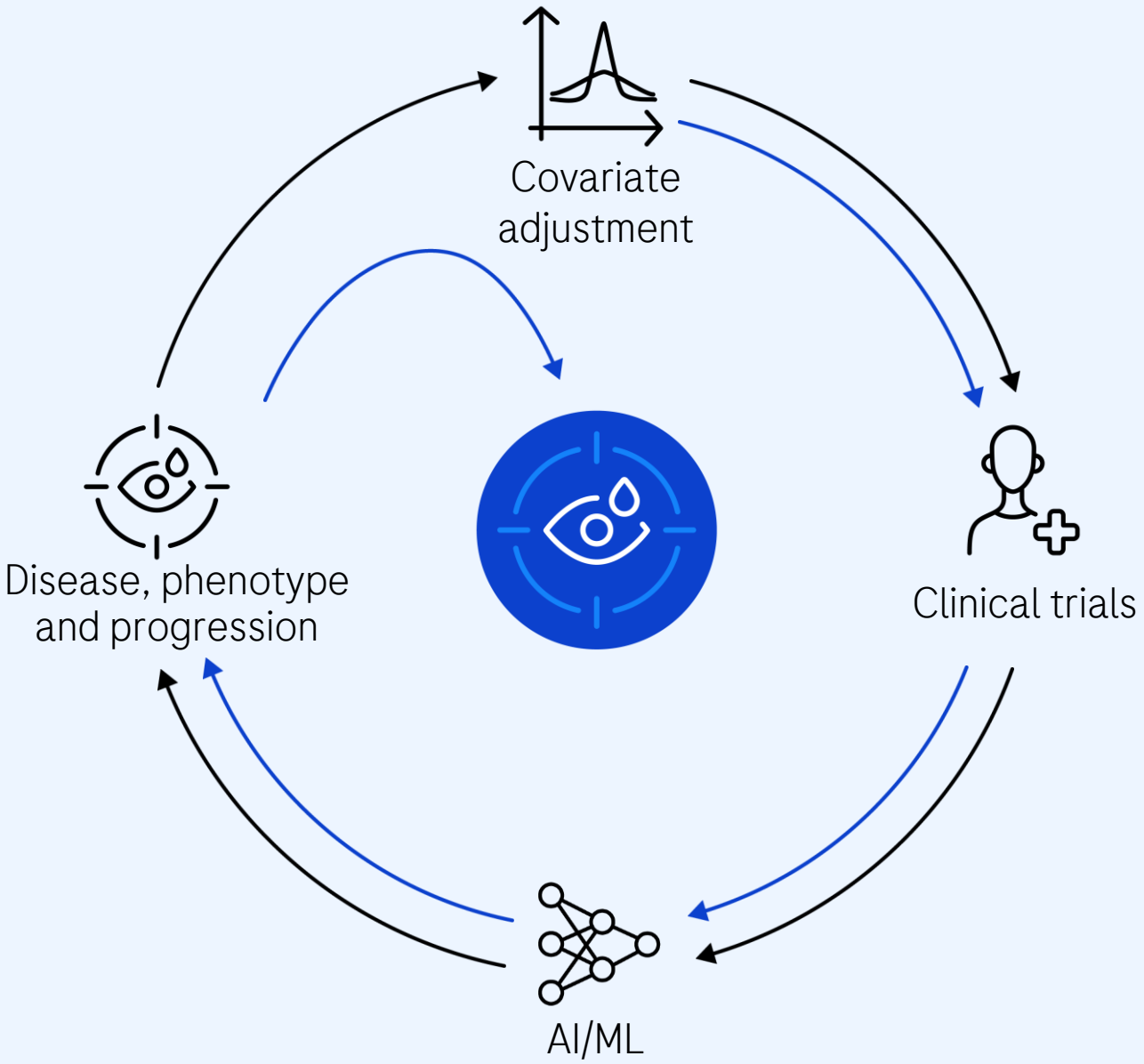
Lab-in-the-Loop helps discover better molecules, faster

Clinical-stage molecule with desired properties and reduced risk



LI to clinical candidate in 18 months: 2x faster, reduced immunogenicity risk

2022	2023	2024	2025	2026
Target identification (novel target)	Lead identification Go decision	Lead optimization Go decision	Clinical candidate	First patient in



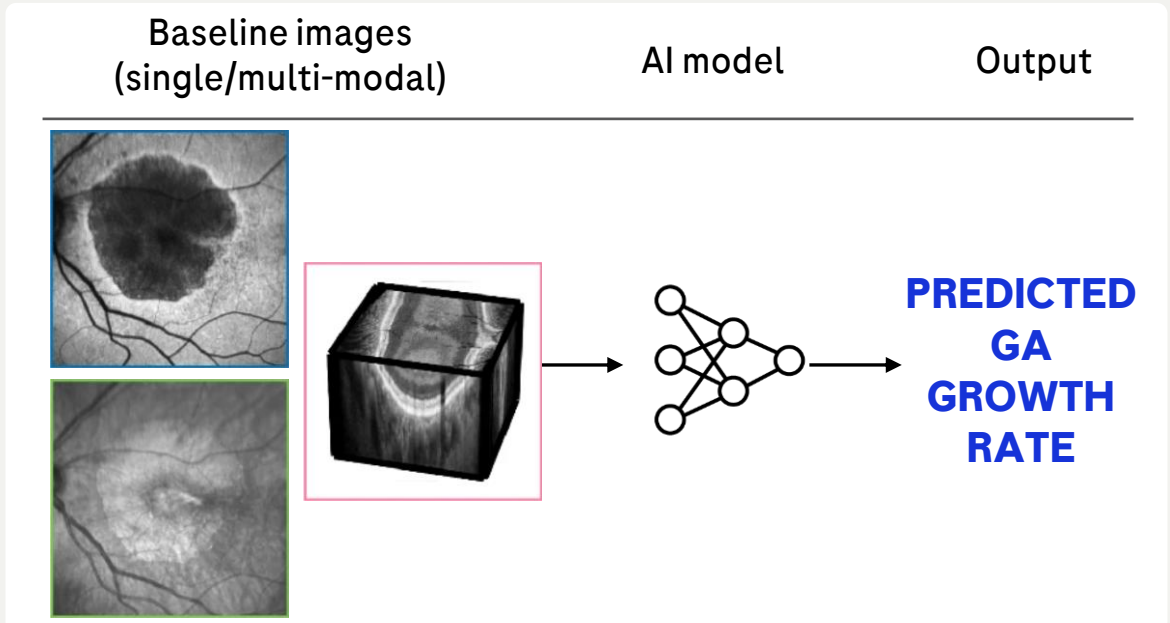
CLINIC IN THE LOOP

Lab-in-the-Loop to predict disease progression

AI outperforms in predicting lesion growth

AI-based models allows covariate adjustment and better clinical decision making

"E2E" AI to predict lesion growth rate

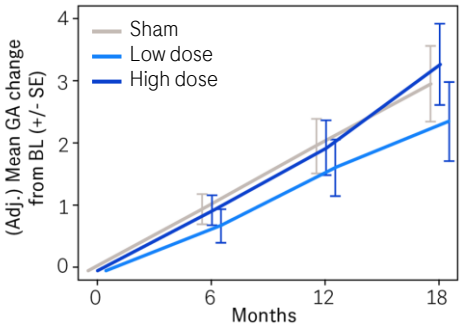


Trained and validated on >2,000 images from our past clinical trials in GA

Covariate adjustment with AI model

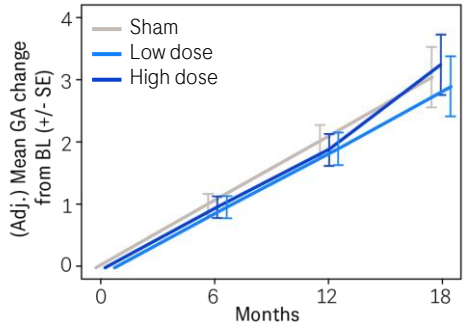
Mahalo **unadjusted** analysis

False-positive effect
~20% (p=0.16)



Mahalo **adjusted** analysis using deep-learning FAF model

No effect
~4.5% (p=0.6)



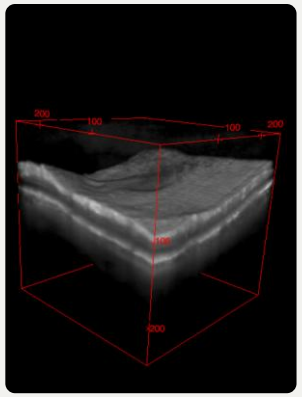
- Covariate Adjustment with AI model (which wasn't available at that time) changes Ph2 trial interpretation
- Precision of treatment effect estimate after adjustment is like running a trial >2x the size in this example

Anegondi N et al. *Ophthalmology Retina*. 2023 Mar;7(3):243-252.
E2E, End-to-end; FAF, Fundus autofluorescence; GA, Geographic atrophy; Ph2, Phase 2

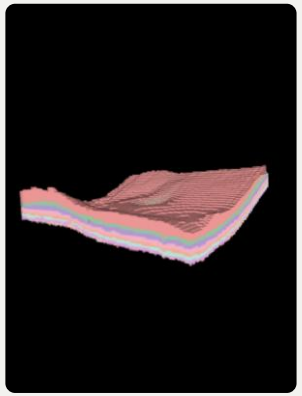
Clinic-in-the-Loop

GWAS of novel AI-based outcomes identifies loci not detected in traditional risk studies

OCT image

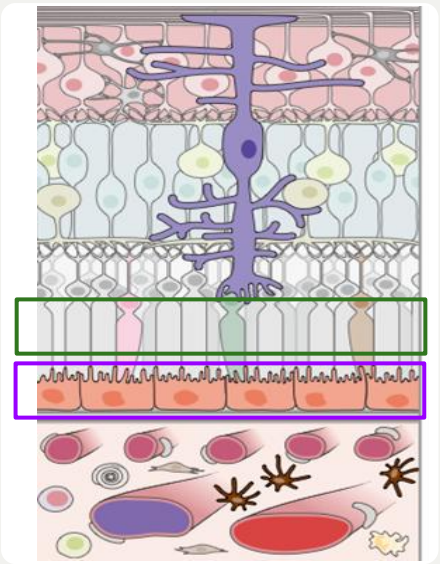


AI layer segmentation

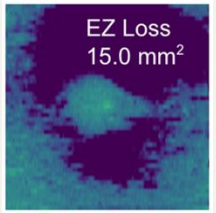


Ellipsoid zone (EZ):
photoreceptor health
RPE supports the photoreceptors

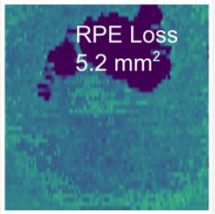
Retinal zone mapping



EZ loss
15.0 mm^2



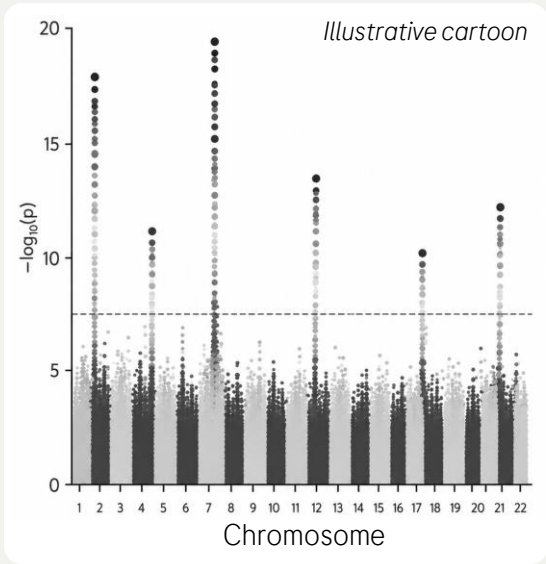
RPE loss
5.2 mm^2



$= \frac{15.0}{5.2}$

High **EZ**/**RPE**
= Poor patient prognosis

GWAS of novel outcomes

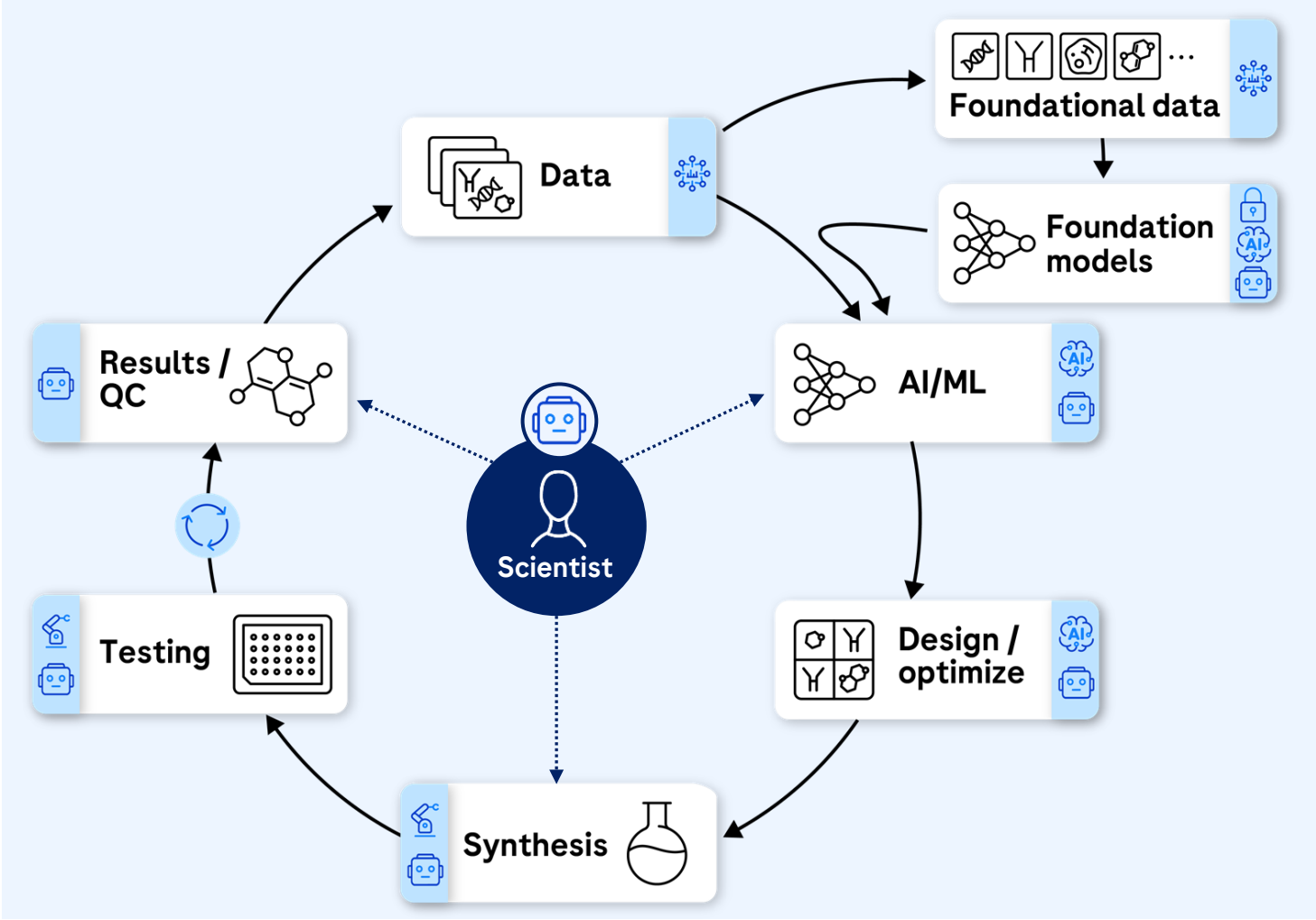


Closing the loop E2E: added as new input for GA pathobiology map driving **target discovery**

E2E, End-to-end; EZ, Ellipsoid zone; GWAS, Genome-wide association study; RPE, Retinal pigment epithelial cells; OCT, Optical coherence tomography

AI independence: owning the loop, learnings, improvements

Data generation and AI independence needed to maximize competitive benefit of our learnings



Capabilities

- Proprietary Foundation models
- Expert domain agents and harness
- AI factory
- Multiple data generation streams at scale
- Ability to iterate (lab and AL/RL)
- Autonomous lab capabilities

Partnerships

NVIDIA

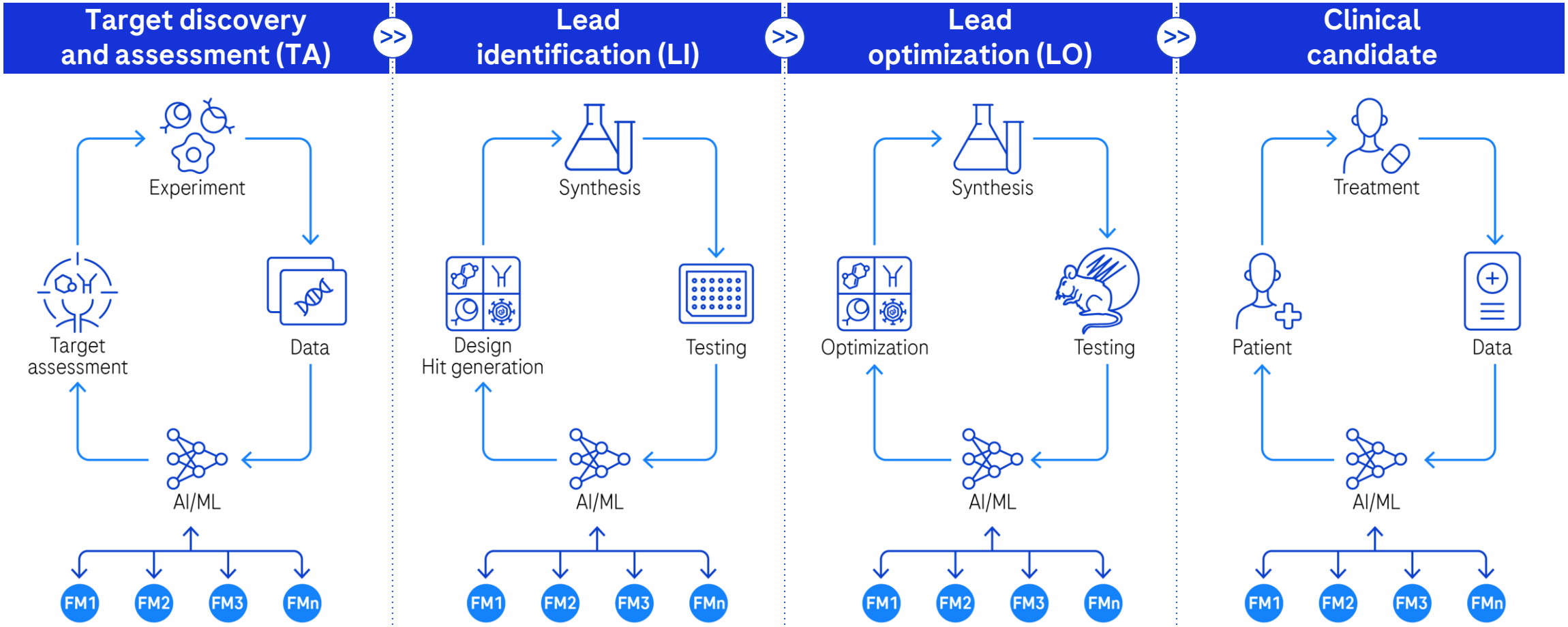
RECURSION

Medra

ANTHROP\C

Lab-in-the-Loop in drug R&D

Iterations with AI embedded E2E, across each component, from target discovery to the clinic



AI/ML, Artificial Intelligence and Machine Learning; FM, Foundation model; LI, Lead identification; LO, Lead optimization; R&D, Research and development; TA, Target assessment

Financial Excellence and Digitalization

Alan Hippe
CFO Roche Group

Overview of key priorities

Scalable automation and AI implementation driven by business process transformation



Scalable automation and AI implementation driven by business process transformation

Disciplined capital allocation to maximize long-term value creation

Efficiency in R&D through operational excellence and resource allocation

AI strategic framework

Leverage AI to increase effectiveness, transform business processes and realize value at scale

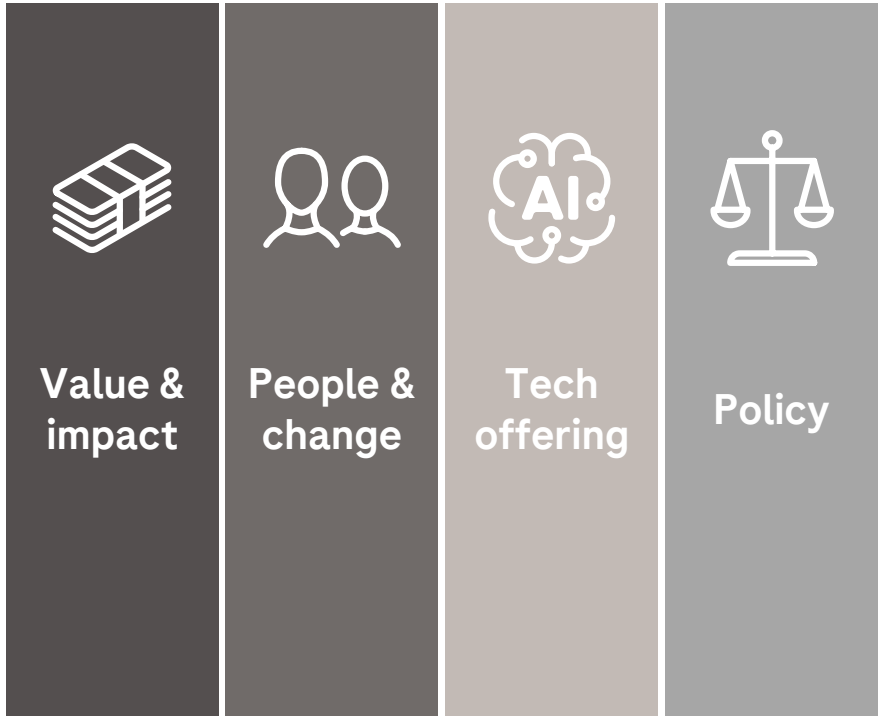
Strategy developed for three levels of AI...

EverydayAI — Embed Everyday AI technologies in daily work to increase effectiveness

Reshape — Reshape processes to realize value at scale from existing AI technologies

Big Ideas — Invest in Big Ideas that use AI to disrupt the industry with emerging technologies

...supported by four strategic pillars



ASPIRE driving business transformation

Integrated and standardized system enabling advanced analytics and data-driven decisions

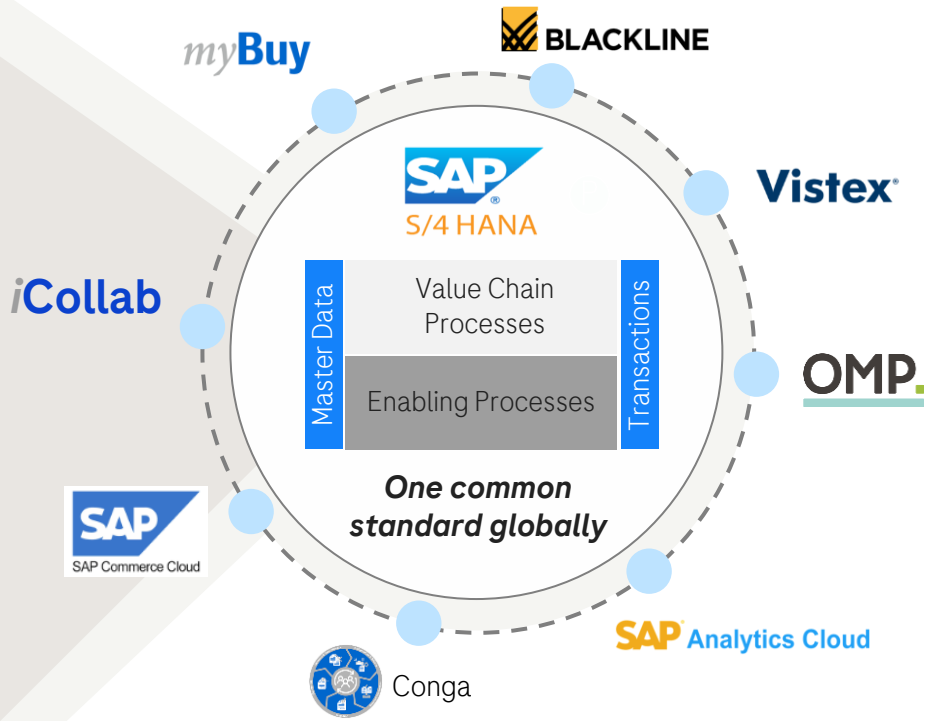
Value chain processes

- Idea to product (Lifecycle)
- Marketing plan to cash (Commercial)
- Plan to deliver products (Manufacturing & Supply Chain)
- Subscribe / license to retire digital products
- Plan to deliver services

Enabling Processes

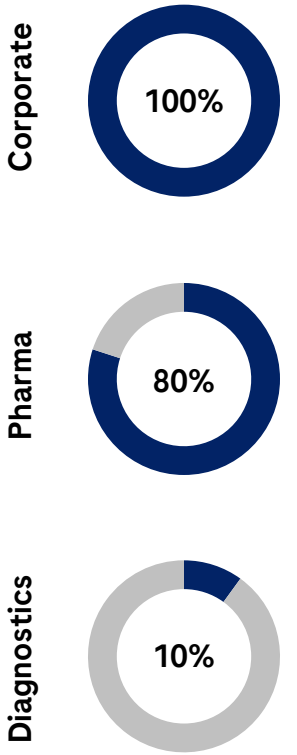
- Demand to pay (Procurement)
- Record to analyze (Finance)
- Plan to retire fixed assets
- Manage IT
- Manage quality & operational compliance
- Develop and manage business capabilities

● In scope ● Partially in scope



Selection of solutions fully integrated in the ASPIRE portfolio

ASPIRE implementation (by year end 2026)



ASPIRE enabling productivity and efficiency

Automation and agentic workflows to drive meaningful gains post-deployment

Deploying ASPIRE (Status Sep 2026)

- Complex deployment of greenfield ASPIRE template and ecosystem:
  - 118 company codes (44% deployed)
  - ~950 process activities enabled[1]
  - 98% adherence to template
- Early value deployment and first benefits realized
- Full roll-out by 2029

Sustained productivity and efficiency enabled by ASPIRE

Harmonization and standardization:

- **Data**
- **Processes**
- **Systems**

Automation and AI enablement:

- **Broad and simplified application**
- **Business insights at speed**

Gains to be realized 2-5 years post-deployment

1. Process classification framework according to APQC (American Productivity & Quality Center): Process activities are the key steps within the process chain that enable the 11 end-to-end processes, e.g. “Plan to deliver products (Manufacturing & Supply Chain)”

Overview of key priorities

Disciplined capital allocation to maximize long-term value creation

Scalable automation and AI implementation driven by business process transformation



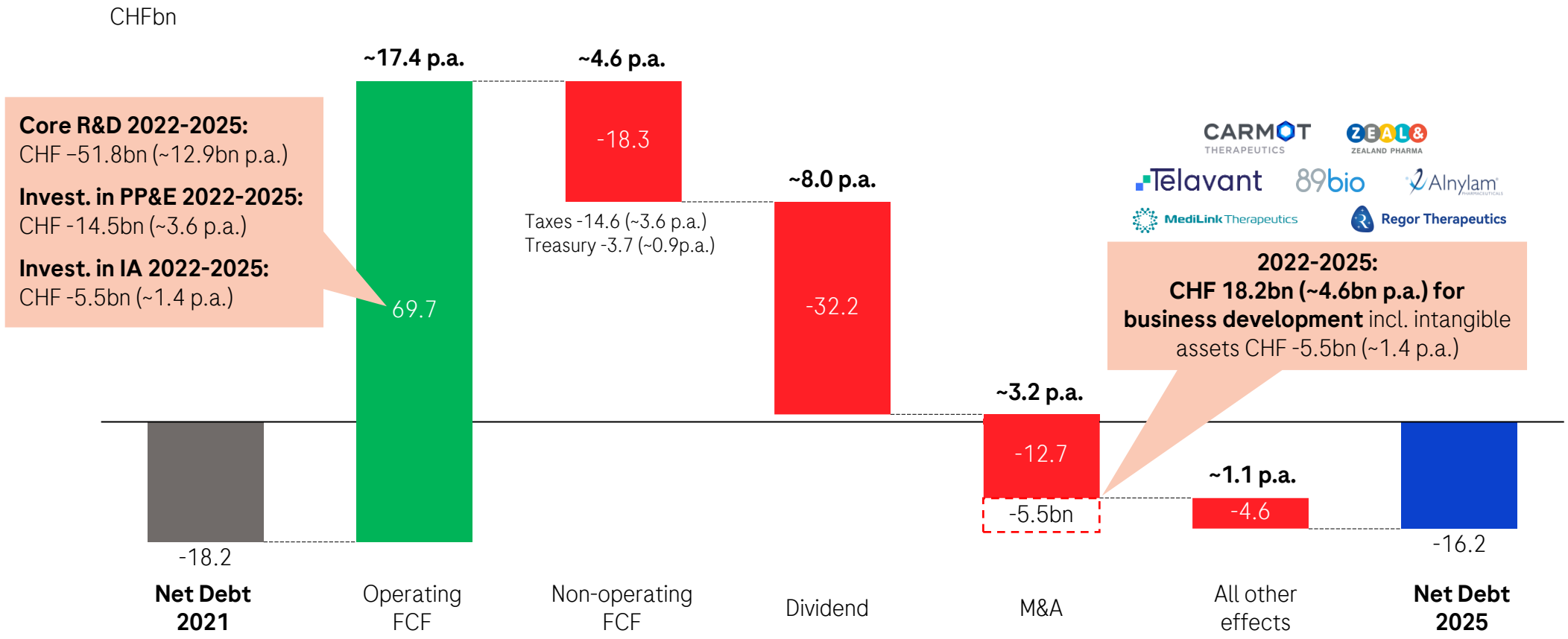
Disciplined capital allocation to maximize long-term value creation

Efficiency in R&D through operational excellence and resource allocation

Sustainable financial development creating financial flexibility

Balanced investments to support next wave of innovation while maintaining attractive shareholder returns

Roche net debt development 2021-2025

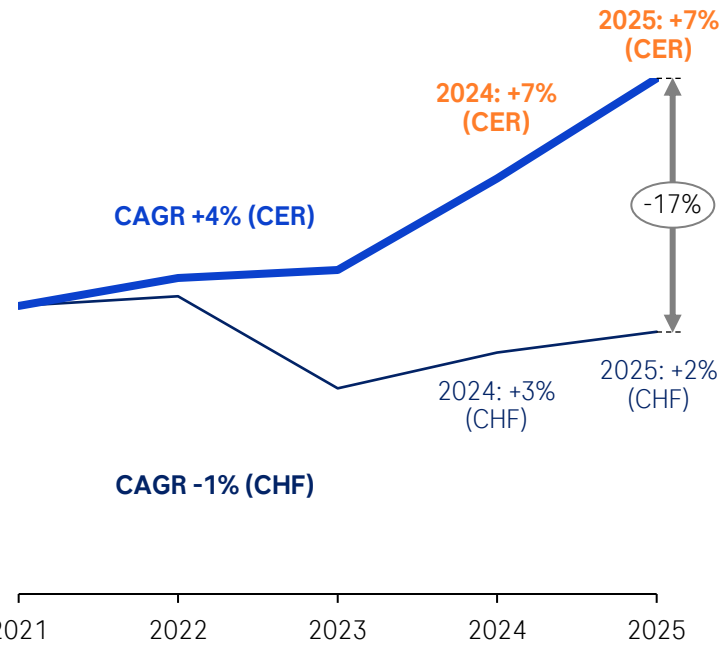


Totals and subtotals may include differences due to rounding

Group sales, Core OP and COP margin momentum past 5 years

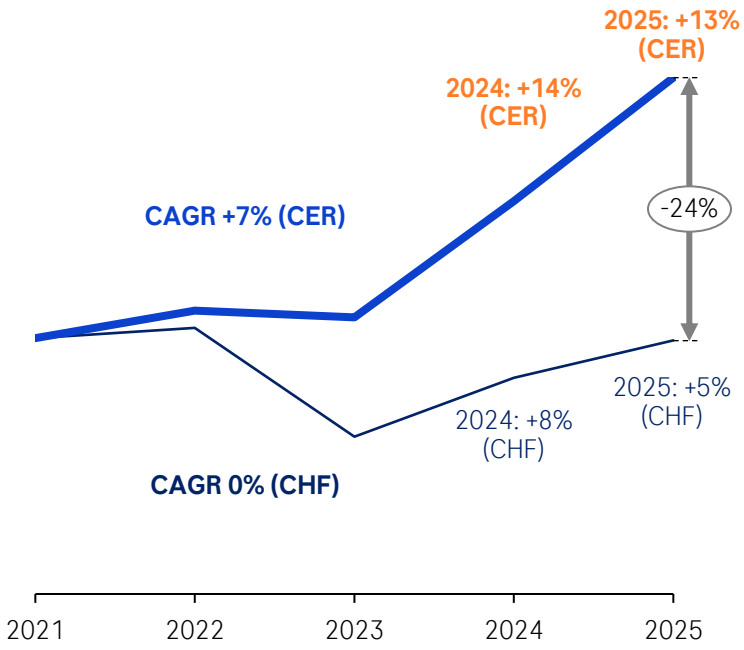
Accelerating growth after successfully navigating AHR cliff and COVID-19 pandemic

Sales growth CHF rep vs CER



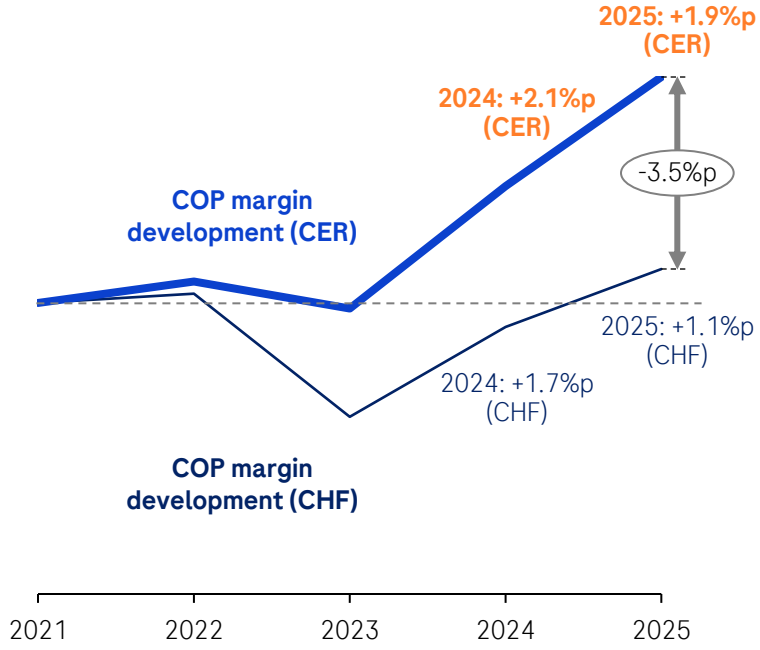
Group sales growth CAGR 2011-25: CER: +5%; CHF +3%

Core OP growth CHF rep vs CER



Group Core OP growth CAGR 2011-25: CER: +6%; CHF +3%

COP margin growth CHF rep vs CER

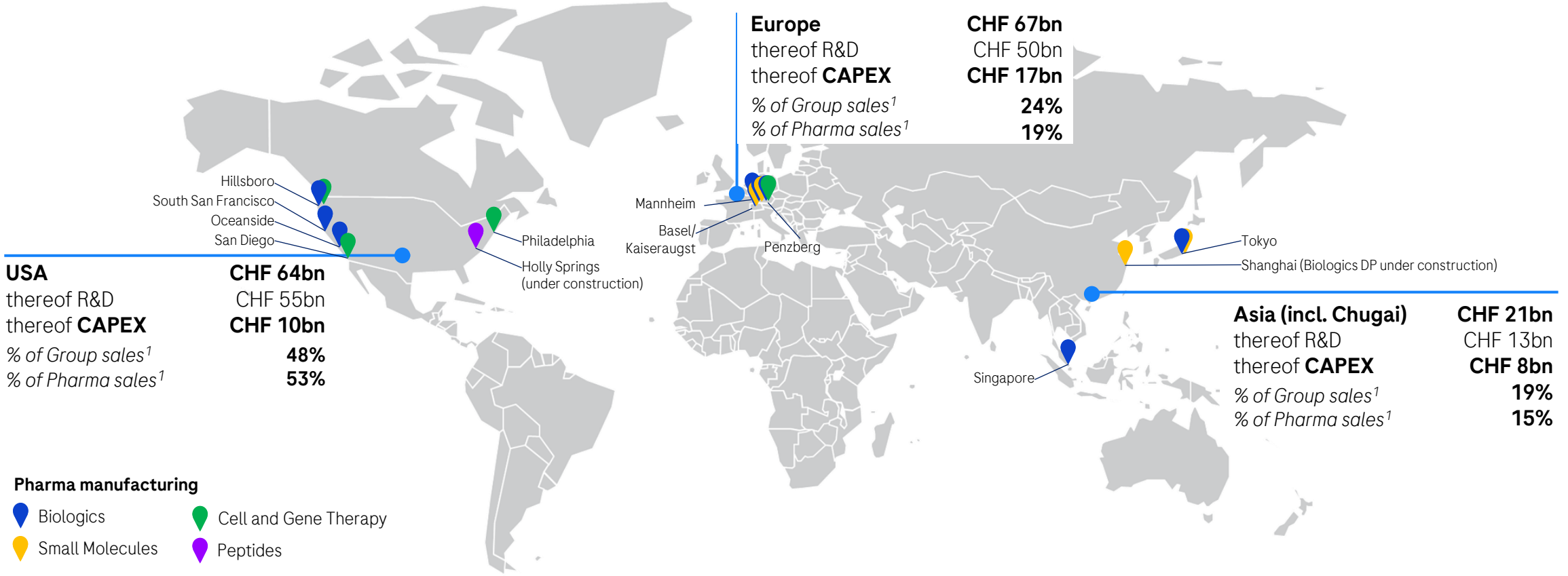


Group COP margin development 2011-25: CER: +6.4%p; CHF -0.1%p

Group sales and Core OP indexed at baseline, growing with CER/CHF rep growth rates of each year; CAGR calculated based on CER/CHF rep growth rates of each year; CER=Constant Exchange Rates; AHR: Avastin, Herceptin and Rituxan/MabThera; CAGR=Compound Annual Growth Rate; Source: Roche Finance Report 2021-2025

Roche investments worldwide (2016-2025)

Strategic investments in R&D and manufacturing with complete value chain in key markets to ensure resilience



USA	**CHF 64bn**
thereof R&D	CHF 55bn
thereof **CAPEX**	**CHF 10bn**
% of Group sales[1]	**48%**
% of Pharma sales[1]	**53%**

Europe	**CHF 67bn**
thereof R&D	CHF 50bn
thereof **CAPEX**	**CHF 17bn**
% of Group sales[1]	**24%**
% of Pharma sales[1]	**19%**

Asia (incl. Chugai)	**CHF 21bn**
thereof R&D	CHF 13bn
thereof **CAPEX**	**CHF 8bn**
% of Group sales[1]	**19%**
% of Pharma sales[1]	**15%**

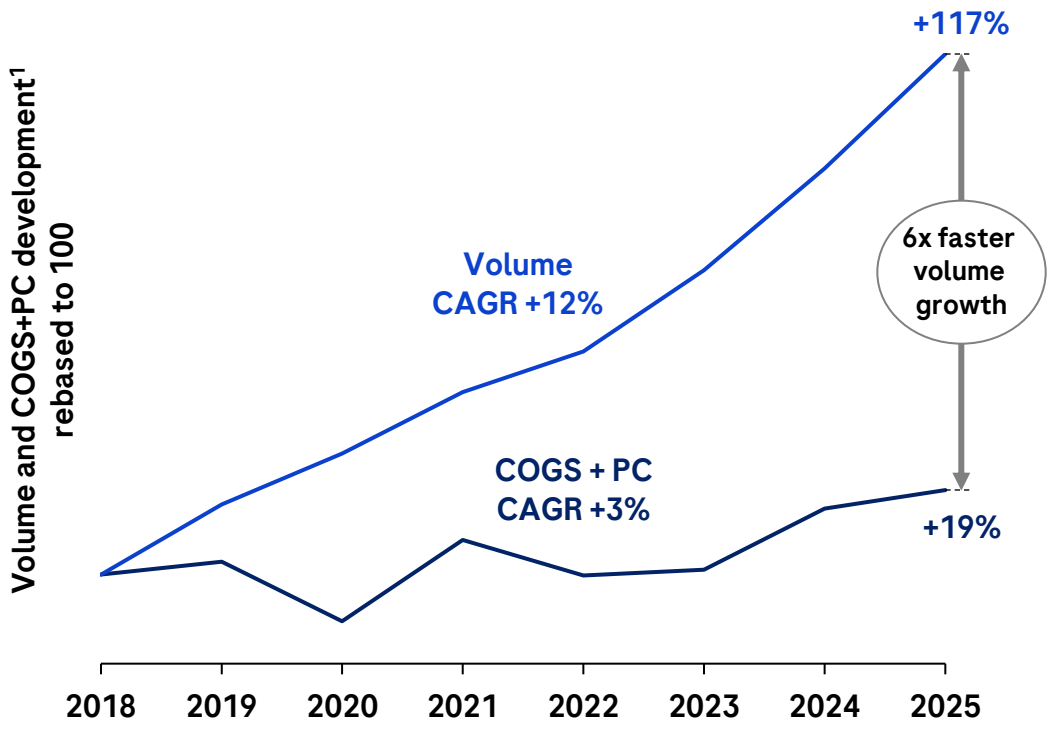
CHF 158bn	investments in R&D and CAPEX (2016-2025)[2]	**USD 50bn**	commitment into R&D and PP&E in the U.S. by 2029	**CHF 1.4bn**	further investments planned at Basel site

1. Percentage of Roche sales in 2025; totals and subtotals may include differences due to rounding; 2. Investments rest of world 2016-2025: CHF 6bn; DP: Drug product

Manufacturing: Network optimization and productivity improvement

Freeing up resources to invest in new modalities, including peptides within existing budget

Biologics network optimization since 2018



CVRM manufacturing strategy

- Clinical supply: Secured through several CDMOs
- Commercial supply: Optimized mix of in-house capacity and CDMOs, building resilient supply chains (regional & dual-sourced)
  - Initial capacity secured through CDMO network
  - Internal capacity (Holly Springs) operational by 2029, funded out of existing CAPEX budget

Efficient site ramp-up via digital twins, reusable templates and standardized & harmonized data, processes, and systems.

CER: Constant Exchange Rates (avg. full year 2025 as basis calculating back with the CER growth rate of the respective year); COGS + PC: Manufacturing cost of goods sold and period costs

Overview of key priorities

Efficiency in R&D through operational excellence and resource allocation

Scalable automation and AI implementation driven by business process transformation

Disciplined capital allocation to maximize long-term value creation

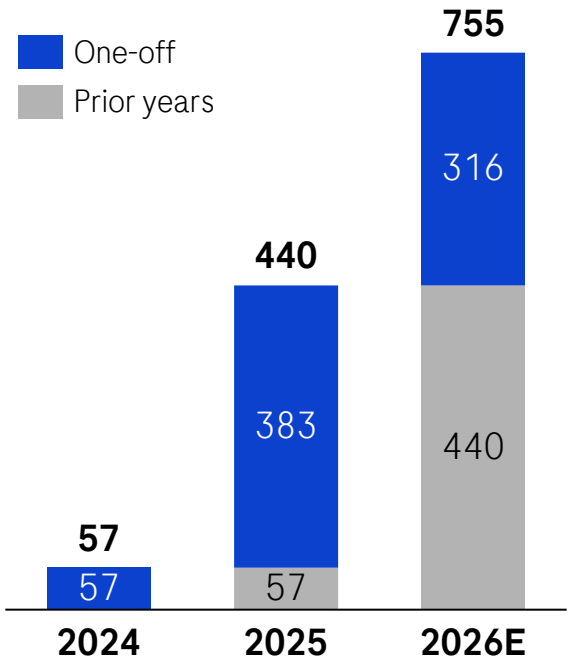


Efficiency in R&D through operational excellence and resource allocation

R&D Excellence: Optimization across portfolio and operations

R&D savings achieved since 2024; existing initiatives expected to deliver additional savings

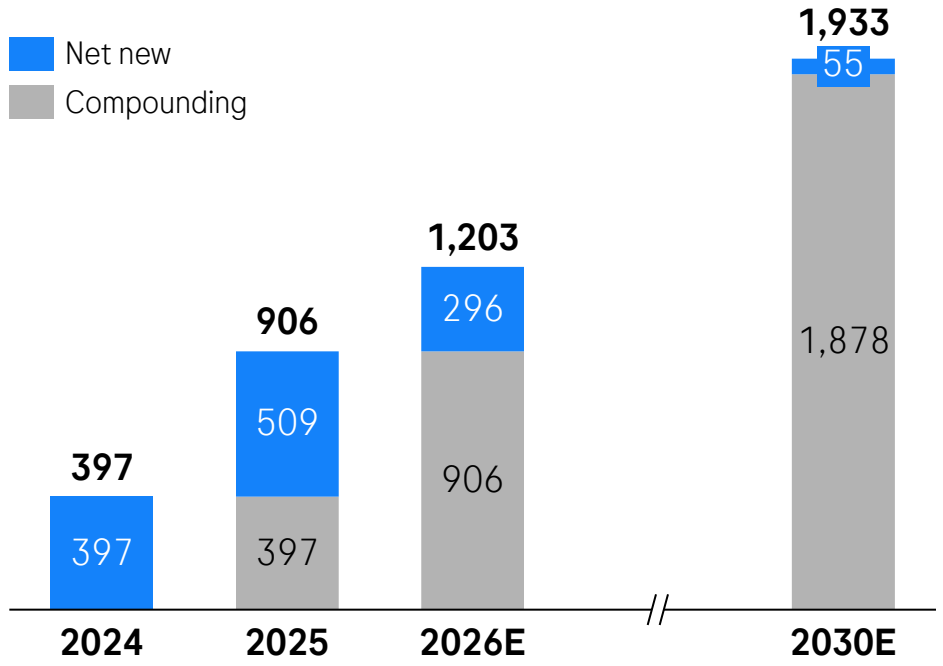
One-off savings in CHFm[1]



Non-recurring cost reductions

e.g. portfolio prioritization (stopping programs early)

Recurring savings (net new + compounding) in CHFm[1]



Savings with long-lasting impact, reducing the cost base

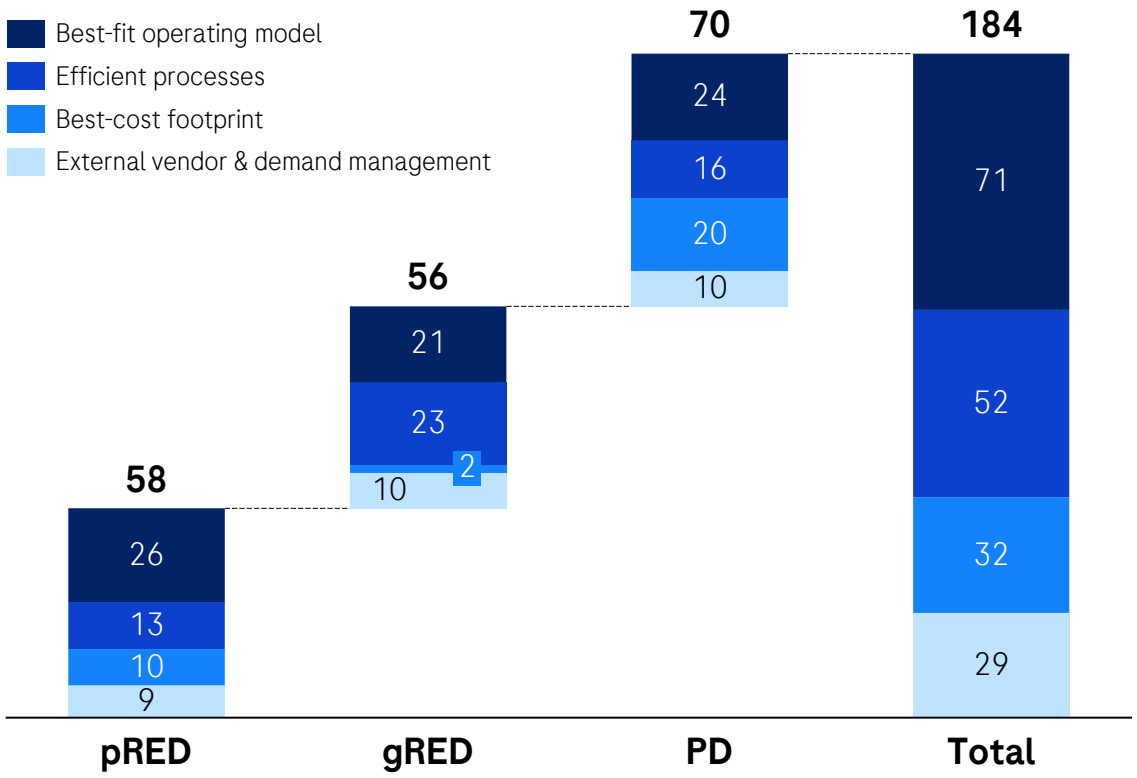
e.g. PD capacity optimization, clinical sourcing (vendor consolidation)

1. Source: Internal data including Spark, Flatiron, RMCS, PHC; E: Estimated savings by year-end

R&D Excellence: 184 cost optimization initiatives identified

Continued focus on excellence to deliver future portfolio in the most efficient way

184 cost optimization initiatives identified



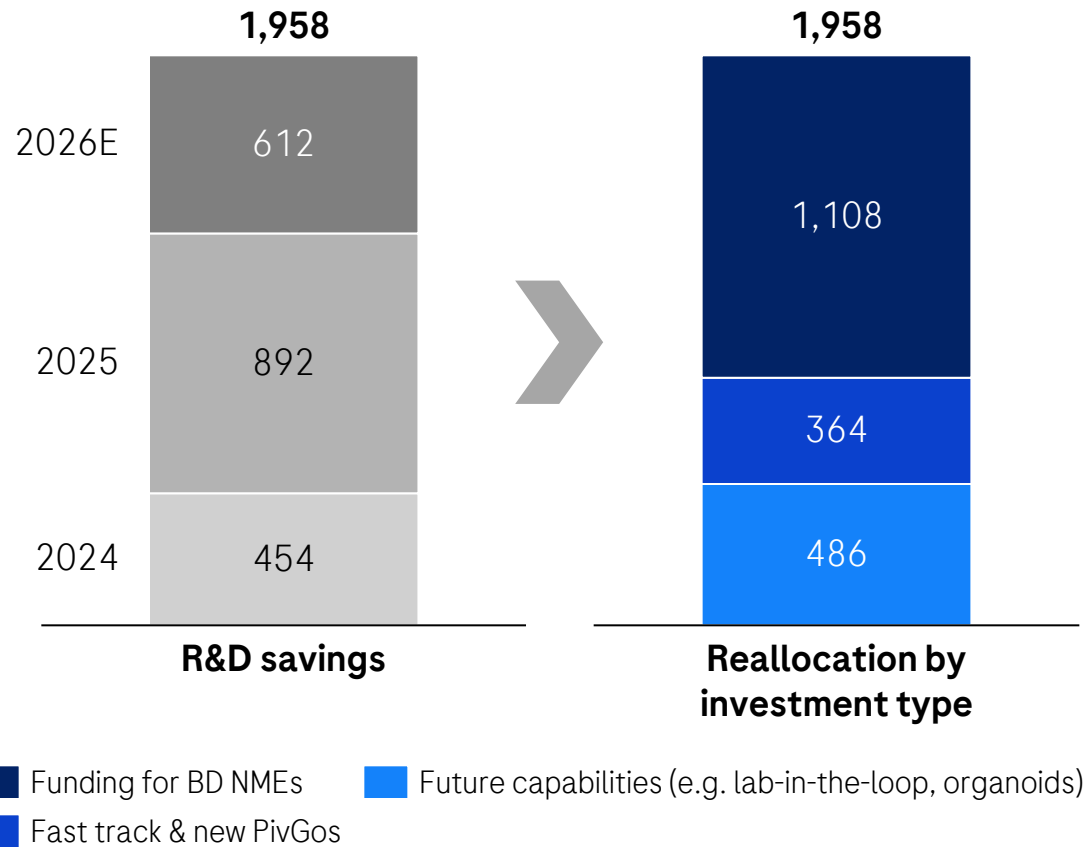
Key levers and opportunity areas across R&D

- Best-fit operating model; improve organizational structure and right-size capacity aligned to portfolio planning
- Efficient processes; reduce non-portfolio work and optimize processes through standardization and automation
- Best-cost footprint for repeatable standard task
- Vendor and demand management

R&D Excellence: Resource reallocation to higher ROI projects

CHF ~2bn will be reallocated to transformative programs and productivity initiatives by YE 2026

Reallocation of R&D budget in 2024-2026E (CHFm)[1]



Outcomes achieved through resource reallocation

- Enter CVRM as new therapeutic area via external innovation
- Maximize assets with transformational potential in existing therapeutic areas *(e.g. giredestrant)*
- Accelerate "fast track" and other key programs to ensure timely delivery *(e.g. trontinemab, afimkibart, enicepatide, zilebesiran, prasinezumab)*
- Increase investment in future capabilities and technology to enable sustained R&D efficiencies

1. Source: Internal data including Spark, Flatiron, RMCS, PHC; AI: Artificial intelligence; BD: Business development; E: Estimated savings by year-end; NME: New molecular entity; Note: Fast-track assets: afimkibart, enicepatide, trontinemab

Oncology/Hematology

Levi Garraway
CMO and Global Head of Product Development

Significant pipeline progress since Pharma Day 2025

Leveraging both internal capabilities and external innovation to rejuvenate Oncology/Hematology pipeline

Key late-stage successes

5 positive Ph III readouts

- ✓ **giredestrant *(lidERA)*** Adj. HR+/HER2- eBC
- ✓ **divarasib *(KRASCENDO 1)*** 2L+ KRAS G12C+ NSCLC
- ✓ **Tecentriq *(IMvigor011)*** MIBC
- ✓ **Lunsumio *(CELESTIMO)*** 2L+ FL
- ✓ **PiaSky *(COMMUTE-a/p)*** aHUS

Pipeline progress

2 NME Ph III decisions

- **tambotatug pelitecan** SCLC, NSCLC
- **bexobrutideg** R/R CLL

18 Ongoing Ph III studies

28 NMEs in clinical development

71% FIC/BIC assets

External innovation

4 assets added through BD

Simcere	HANSOH PHARMA	MediLink Therapeutics	NURIX
Hem	**CRC**	**Lung**	**Hem**
CD79a x CD19 x CD3 TCE	**CDH17 ADC**	**Tam-Peli (B7-H3 ADC)**	**bexobrutideg (BTK degrader)**
Preclinical	**Ph I**	**Ph III***	**Ph III**

6 platform deals / research collaborations**

DualityBio 映恩生物	C4 Therapeutics	Earendil Labs	Ventus THERAPEUTICS	astex	CARIS LIFE SCIENCES
ADC platform	**DAC platform**	**Bispecific antibodies**	**Small molecules**	**Drug discovery**	**Target discovery**
Next-gen ADCs with novel cytotoxic payloads	Degrader-Antibody Conjugates (DACs)	Novel antibody target combinations	Novel candidates for challenging targets	Targeting cell cycle regulators in breast cancer	Next-gen breast cancer targets

*Phase III to be initiated; **including deals pending closure; aHUS: Atypical hemolytic uremic syndrome; ADC: Antibody-drug conjugate; BD: Business development; BIC: Best in class; BTK: Bruton's tyrosine kinase; CLL: Chronic lymphocytic leukemia; (e/m)BC: (Early/metastatic) breast cancer; FIC: First in class; FL: Follicular lymphoma; HER2: Human Epidermal Growth Factor Receptor 2; HR: Hormone receptor; MIBC: Muscle-invasive bladder cancer; NSCLC: Non-small cell lung cancer; SCLC: Small cell lung cancer; TCE: T-cell engager

Oncology solid tumor pipeline

Focus on breast and lung cancer as end-to-end investment areas

Phase I

RG6171	giredestrant
RG6330	divarasib
RG6505	Pan-RAS inhibitor
RG6537	AR Degrader
RG6620	KRAS G12D inhibitor
RG6648	cMET ADC[1]
RG6810	DLL3 ADC[2]
RG6852	undisclosed
RG6975	CDH17 ADC[3]
CHU	codrituzumab
CHU	ROSE12 (anti-CTLA-4 switch Ab)
CHU	MINT91
CHU	AUBE00 (Pan-KRAS inhibitor)
CHU	clesitamig (DLL3 trispecific Ab)
CHU	AQUA07 (allosteric ALK inhibitor)

Phase II

RG6180	**autogene cevumeran**[4] PDAC
RG6114	**Itovebi** eBC HR+
RG6114	**Itovebi** CRPC
RG6114	**Itovebi** HR+ BC *PIK3CA* wt
RG6794	**CDK4/2 inhibitor** eBC HR+
RG6596	**ZN-1041 (HER2 TKI)** eBC HR+

Phase III

RG6171	**giredestrant** multiple HR+ BC indications
RG6330	**divarasib** NSCLC
RG3502	**Kadcyla** HER2+ eBC high risk
RG7446	**Tecentriq** multiple indications
RG6114	**Itovebi** multiple BC indications
RG7853	**Alecensa** LA stage III NSCLC
RG6919	**tambotatug pelitecan**[1] SCLC**

Registration

RG6171	**giredestrant** HR+ BC adj and metastatic
RG7446	**Tecentriq** stage III dMMR/MSI-H colon cancer

Breast cancer · Colon cancer · Prostate cancer · Lung cancer · HCC · Pancreatic cancer · Multiple indications

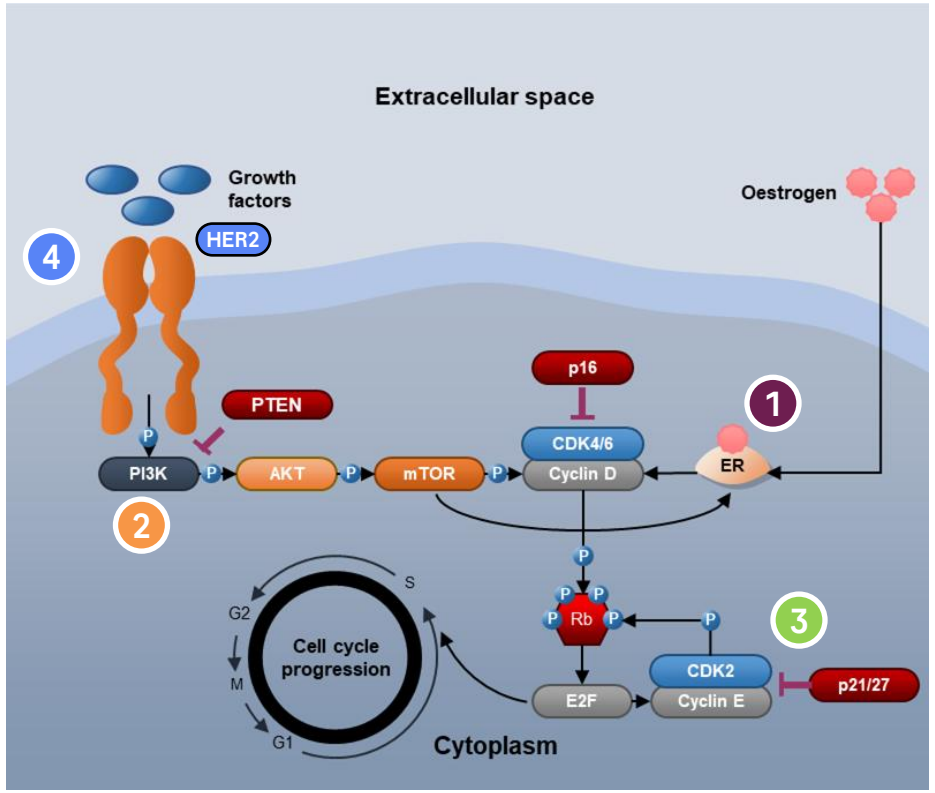
Small molecule · Antibody · Bispecific/trispecific · Neoantigen vaccine · Antibody-drug conjugate · Cyclic peptides · Protein degraders

**Phase III to be initiated; RG-No: Roche/Genentech; CHU: Chugai managed; 1. MediLink managed; 2. Innovent managed; 3. In collaboration with Hansoh Pharma; 4. In collaboration with BioNTech; AR: Androgen receptor; (e)BC: (Early) breast cancer; CDK: cyclin-dependent kinase; (Early)breast cancer; HCC: Hepatocellular carcinoma; HER2: Human epidermal growth factor receptor 2; HR+: Hormone receptor positive; (N)SCLC: (Non)small cell lung cancer; PDAC: pancreatic ductal adenocarcinoma; TKI: tyrosine kinase inhibitor

Breast cancer: Portfolio of potentially BIC molecules to improve SoC

Targeting key signaling nodes that drive etiology and progression of BC

Differentiated molecules with a potential to redefine BC treatment paradigm as monotherapy and in combinations



1 **ER**

Giredestrant: Next generation SERD with the potential to become the ET backbone treatment

2 **PI3K AKT mTOR**

Itovebi: PI3K inhibitor with potentially greater safety margins allowing for combination with ET and CDK4/6i at standard doses with proven substantial benefit

3 **CDK**

RG6794 (GDC-4198): Highly potent CDK4/2 inhibitor with substantial activity against CDK2 to address a key mechanism of resistance to CDK4/6i

4 **HER2**

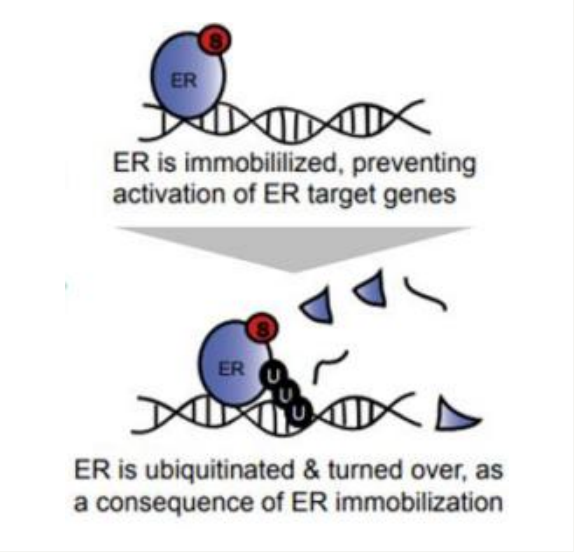
RG6596 (ZN-1041): Highly selective, brain-penetrant HER2 TKI, potentially combinable with established anti-HER2 backbones

Adapted from: Brufsky AM & Dickler MN. Oncologist 2018; BC: Breast cancer; BIC: Best-in-class; CDK: Cyclin-dependent kinase; ET: Endocrine therapy; HER2: Human epidermal growth factor receptor 2; HR+: Hormone receptor positive; PI3K: Phosphatidylinositol 3-kinase; SERD: Selective estrogen receptor degrader; SoC: Standard of care; TKI: Tyrosine kinase inhibitor

Giredestrant: Differentiated SERD with BIC potential

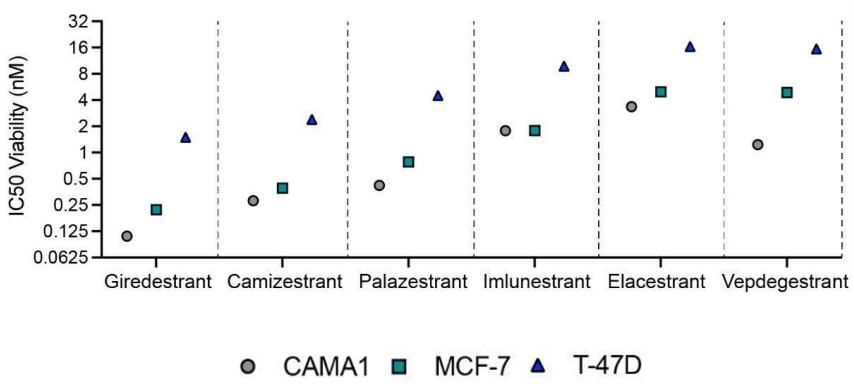
Deep and sustained ER inhibition; superior potency over other SERDs*, allowing lowest therapeutic dose in class

Selective ER degrader (SERD)[1]



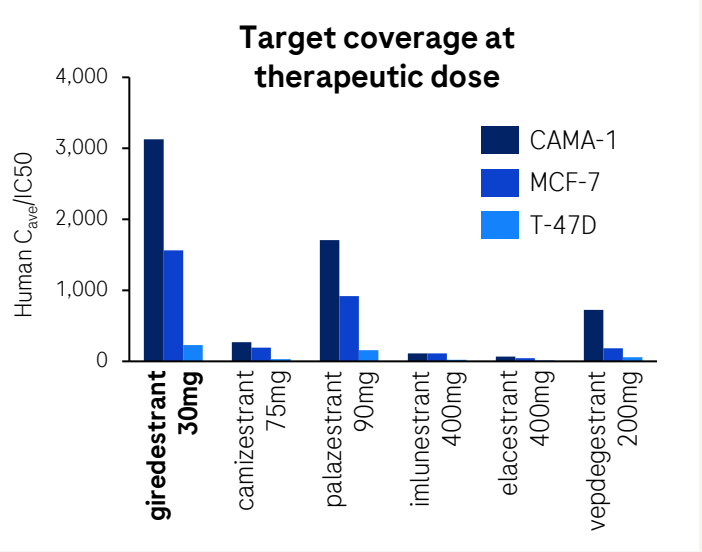
- Giredestrant suppresses ER signaling through ER immobilization and subsequent degradation
- Therapeutic potency is most associated with ER immobilization[2]

Preclinical potency[3-5]



- Highest preclinical potency vs other oral SERDs, allowing lowest therapeutic dose in the class
- Combinable with CDK inhibitors
- Well tolerated at all doses, with no dose-limiting toxicities

Full ER antagonist[2,6,7]



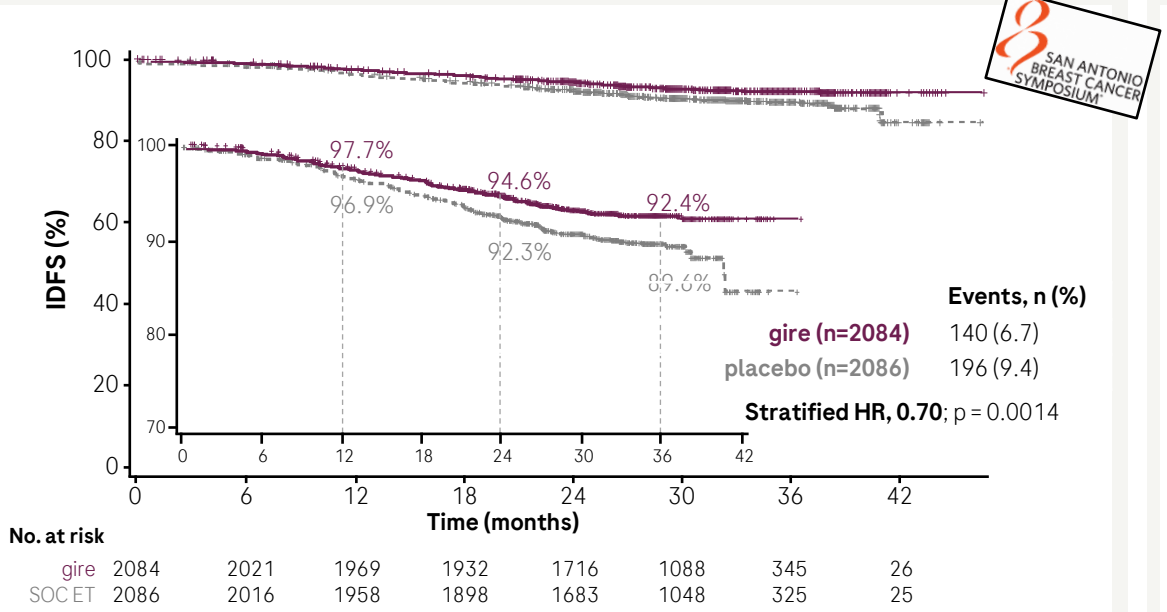
- Full ER antagonist achieving deep and sustained ER inhibition
- Highest target coverage among SERDs

*In preclinical studies; 1. Guan J & Zhou W, et al. Cell 2019; 2. Zhou W, et al. Cell Chemical Biology, 2026; 3. Liang J, et al. J Med Chem 2021; 4. Metcalfe C, et al. Cancer Res 2020; 5. Roche data on file; 6. van Kruchten M, et al. Cancer Discov 2015; 7. Not corrected for plasma protein binding; Human Cave (uM) = AUC0-24 ng*hr/mL/24 hr/MW; giredestrant: AUC0-24 (C2D1), 4320 ng*hr/mL [Cancer Res. Commun. 2023]; camizestrant: AUCτ (C1D15), 862.3 ng*hr/mL [Ann. Oncol. 2024]; palazestrant: Cave,ss (C2D1), 322 ng/mL [Breast Cancer Res. 2025]; vepdegestrant: AUC0-24 (C1D15), 15,459 ng•hr/mL (western patients) [Int. J. Clin. Oncol. 2025]; imlunestrant: AUCτ (C1D15), 2480 ng*hr/mL [J. Clin. Oncol. 2024]; Elacestrant: AUC0-24 (C1D6) 2440 ng*hr/mL [FDA approval label]; CDKi: Cyclin dependent kinase inhibitor; HR+: Hormone receptor positive; SERD: Selective estrogen receptor degrader; SoC: Standard of care

Giredestrant: Positive results in both adjuvant and metastatic HR+ BC

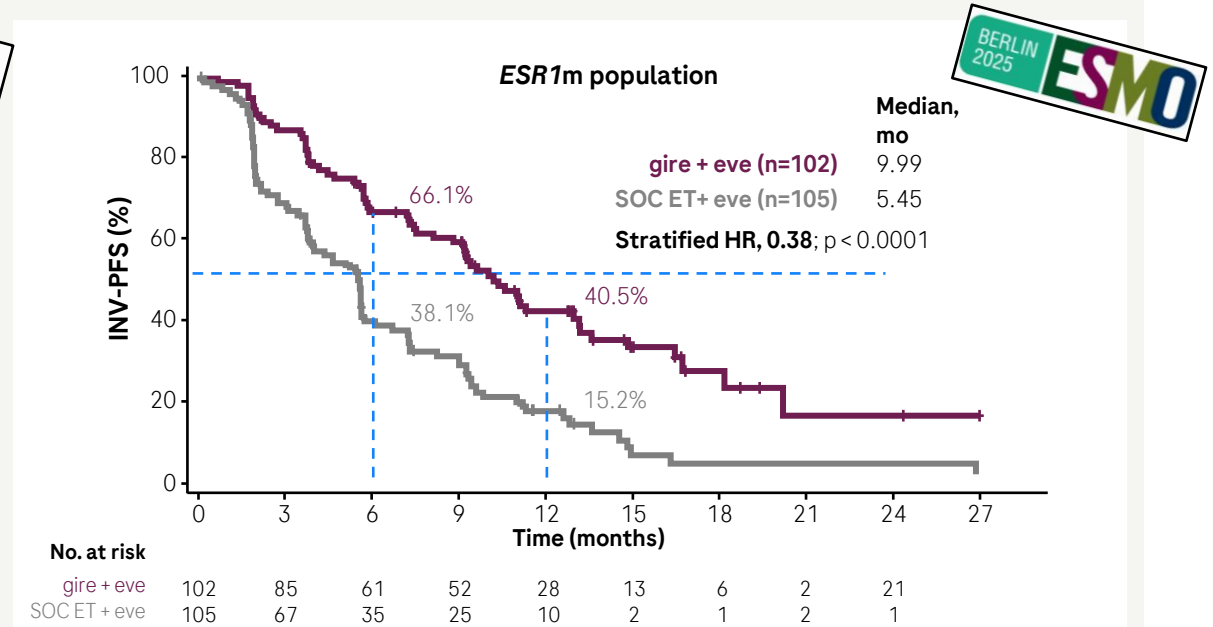
First and only oral SERD to show positive Ph III results in adjuvant HR+ BC

Ph III (lidERA) iDFS results in adjuvant HR+ BC[1]



- 30% reduction in recurrence or death vs SoC ET
- Clear positive OS trend (HR=0.79 at 32m)
- Lower discontinuation rates vs SoC ET in adjuvant BC; no photopsia
- PDUFA date November 30 under priority review

Ph III (evERA) INV-PFS results in post-CDK4/6i HR+ mBC[2]

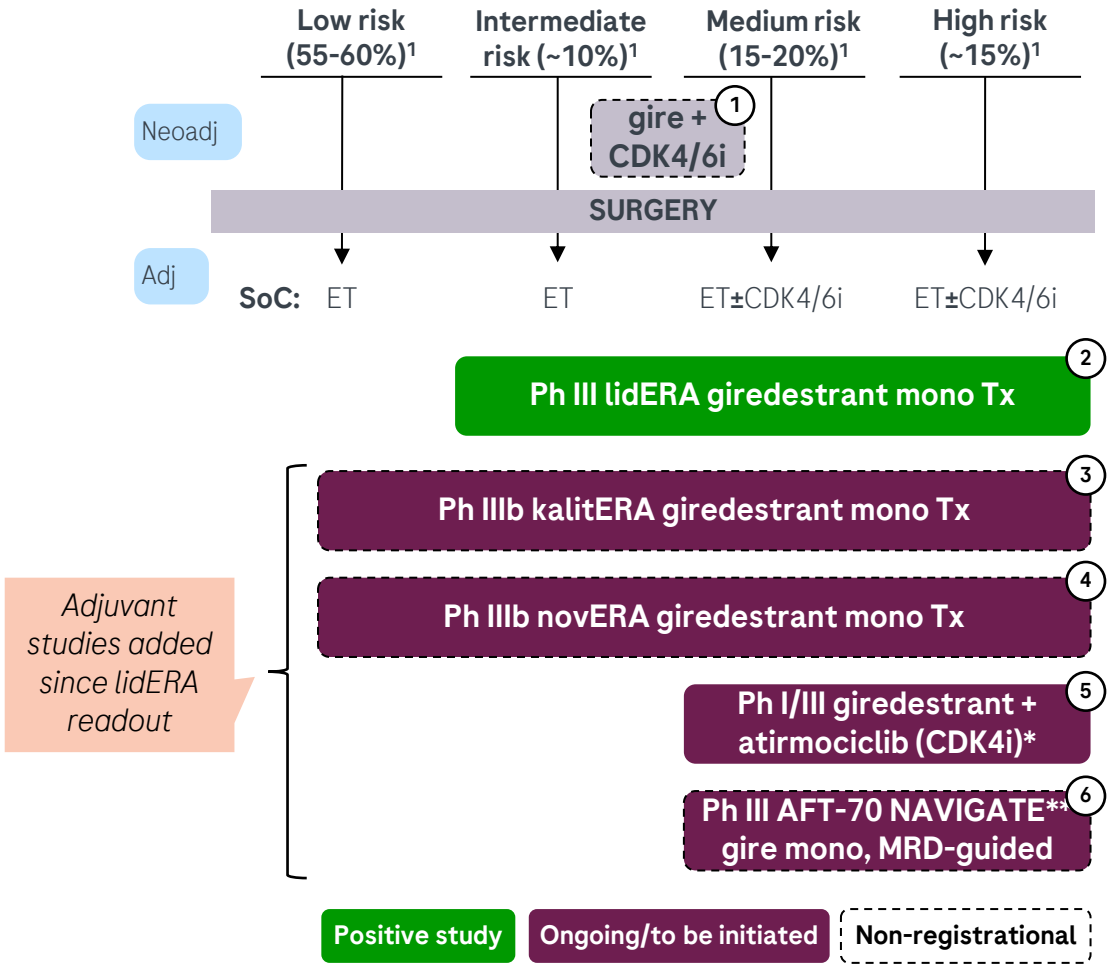


- PFS improvement in both *ESR1*m (HR=0.38) and ITT populations (HR=0.56)
- Clear OS trend favoring giredestrant across subgroups
- PDUFA date December 18 (US filing accepted in *ESR1*m patients)

1. Bardia et al., SABCS 2025; 2. Mayer et al., ESMO 2025; CDKi: Cyclin dependent kinase inhibitor; CI: Confidence interval; DoR: Duration of response; *ESR1*m: ESR1 mutation; ET: Endocrine therapy; HER2: Human epidermal growth factor 2; HR: Hazard ratio; HR+: Hormone receptor positive; iDFS: Invasive disease-free survival; INV-PFS: Investigator-assessed progression free survival; (m)BC: (Metastatic) breast cancer; ORR: Objective response rate; OS: Overall survival; SERD: Selective estrogen receptor degrader; SOC: Standard of care

Giredestrant: Evidence generation in eBC to maximize opportunity

Ph I/III with atirmociclib (CDK4i)* and Ph III in ctDNA-guided** to be initiated; abema combo data expected Q4 '26

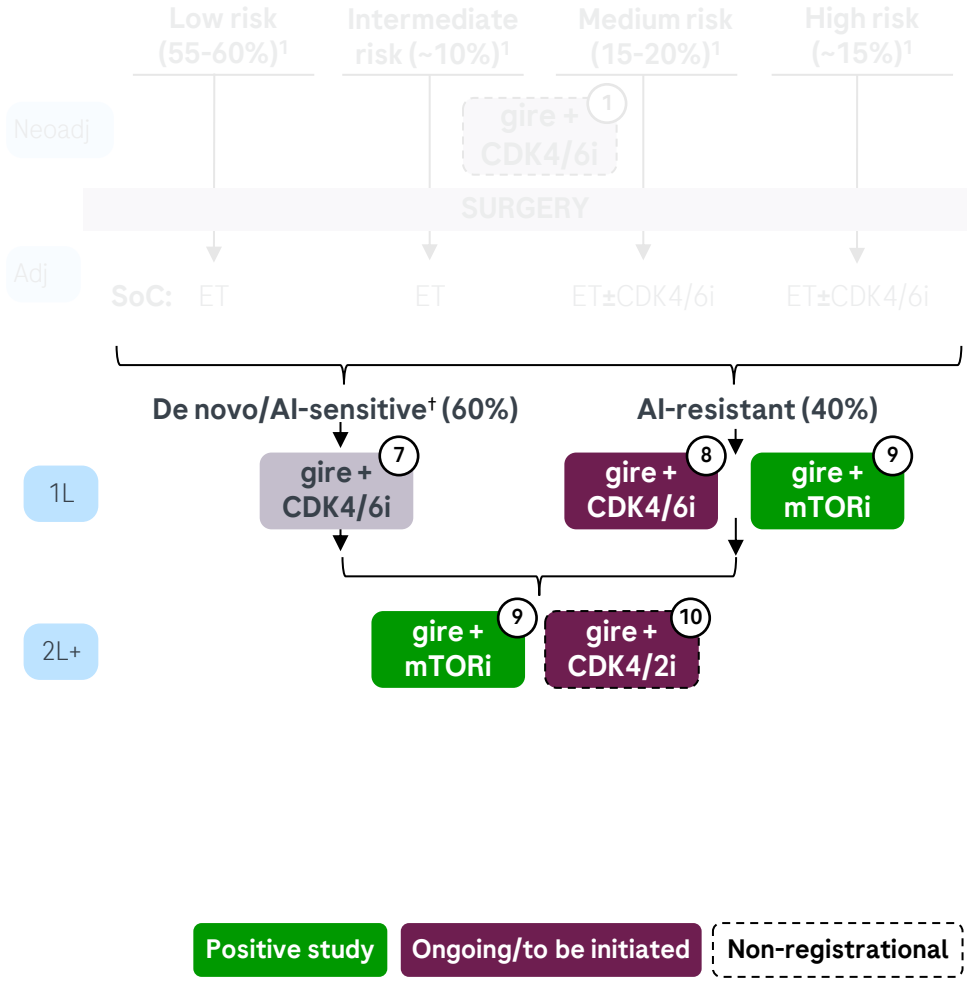


early BC	1	giredestrant (coopERA)	Neodjuvant HR+/HER2- BC	✓
	2	giredestrant (lidERA)	Adjuvant HR+/HER2- BC	✓ Interim OS data 2027
			CDK4/6i single arm substudy	abemaciclib 100 pts (IA Q4 '26); ribociclib 300 pts to be initiated
	3	giredestrant (kalitERA)	Adjuvant HR+/HER2- BC (all risk and switch from CDKi)	Ph IIIb FPI imminent
	4	giredestrant (novERA)	Adjuvant HR+/HER2- BC (AI-intolerant)	Ph IIIb ongoing
	5	giredestrant + atirmociclib*	giredestrant + atirmociclib intermediate to high risk	Ph I/III* to be initiated
	6	giredestrant (AFT-70 NAVIGATE)**	MRD-guided CDKi usage	Ph III (IIS) to be initiated
metastatic BC	7	giredestrant (persevERA)	1L HR+/HER2- mBC (ET-sensitive)	✗ Primary endpoint not met; numerical PFS improvement
	8	giredestrant (pionERA)	1L HR+/HER2- mBC (ET-resistant, enriched for *ESR1*m)	Readout 2028
	9	giredestrant (evERA)	2L+ HR+/HER2- mBC	✓
	10	gire + GDC-4198 (MoonROSE)	2L+ HR+/HER2- mBC	Ph Ib/II ongoing, FPI in Ph II achieved
		gire + Phesgo (heredERA)	1L maintenance HR+/HER2+ mBC	Readout 2028

*Pfizer-managed; Phase III gated on successful outcome of Ph I; **Pending final agreement, in collaboration with Natera and Alliance Foundation Trials 1. Risk definitions vary according to guidelines and tools used: stage at diagnosis based on internal estimates using SEER data; intermediate risk encompasses stage I, N0 included in the lidERA criteria, but excluded in NATALEE; †AI sensitive defined as patients who relapse >1yr after completion of adjuvant therapy; AI: Aromatase inhibitor; CDK: Cyclin-dependent kinase; CDP: Clinical development program; *ESR1m*: *ESR1* mutation; ET: Endocrine therapy; Human Epidermal growth factor receptor 2; HR+: Hormone receptor positive; IIS: Investigator initiated study (m)BC: (Metastatic) breast cancer; MRD: Minimal residual disease; SoC: Standard of care

Giredestrant: Additional opportunity in mBC

Ph III studies in 1L mBC HR+/HER2- (pionERA) and HR+/HER2+ (heredERA) ongoing; FPI for Ph II CDK4/2i combo



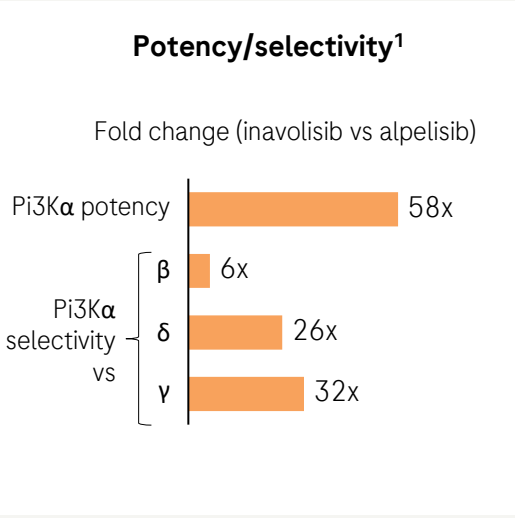
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			CDK4/6i single arm substudy	abemaciclib 100 pts (IA Q4 '26); ribociclib 300 pts to be initiated
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	5	giredestrant + atirmociclib*	giredestrant + atirmociclib intermediate to high risk	Ph I/III* to be initiated
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	8	giredestrant (pionERA)	1L HR+/HER2- mBC (ET-resistant, enriched for *ESR1*m)	Readout 2028
	9	giredestrant (evERA)	Post-CDKi HR+/HER2- mBC	✓
	10	gire + CDK4/2i (MoonROSE)	2L+ HR+/HER2- mBC	Ph Ib/II ongoing, FPI in Ph II achieved
		gire + Phesgo (heredERA)	1L maintenance HR+/HER2+ mBC	Readout 2028

1. Risk definitions vary according to guidelines and tools used: stage at diagnosis based on internal estimates using SEER data; intermediate risk encompasses stage I, N0 included in the lidERA criteria, but excluded in NATALEE; †AI sensitive defined as patients who relapse >1yr after completion of adjuvant therapy; AI: Aromatase inhibitor; CDK: Cyclin-dependent kinase; CDP: Clinical development program; *ESR1m*: *ESR1* mutation; ET: Endocrine therapy; FPI: First patient in; HER2: Human Epidermal growth factor receptor 2; HR: Hormone receptor; (m)BC: (Metastatic) breast cancer; PFS: Progression free survival; SoC: Standard of care

Itovebi: Defining new SoC in *PIK3CA*m HR+ BC

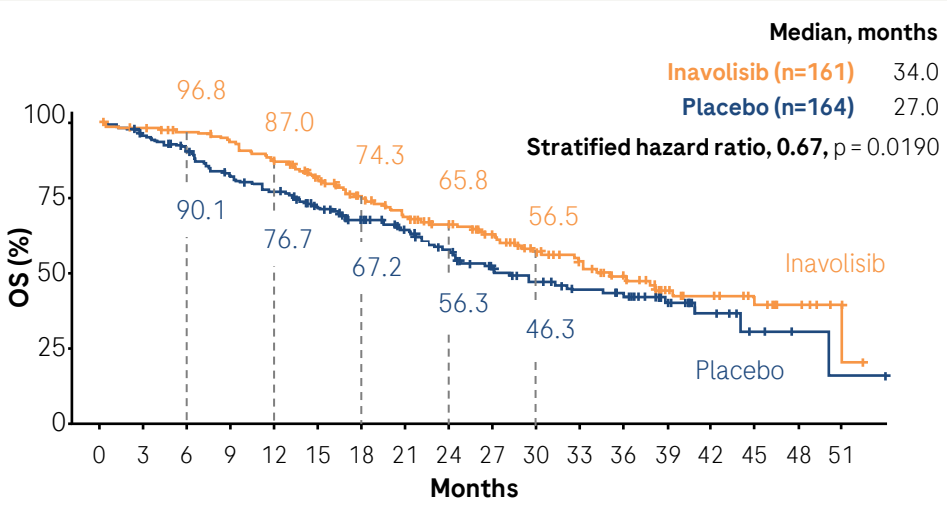
Exploring combinations with giredestrant and CDK inhibitors (CDK4/6i, CDK4/2i, CDK4i)

PI3Kα inh. with dual MoA



- Highly potent and selective PI3Kα inhibition and degradation
- Better *in vivo* efficacy
- Greater safety margins allow for combination with ET and CDK4/6i at standard doses

Ph III (INAVO120) OS results in 1L HR+/HER2- mBC[2]



- Itovebi + palbociclib + fulvestrant reduced risk of death >30% compared with palbociclib + fulvestrant alone
- PFS benefit maintained during longer follow-up (17.2m vs 7.3m; HR=0.42)
- Low discontinuation rates due to AEs (6.8%) and hyperglycemia (1.2%), confirming manageable tolerability

Clinical development program

inavolisib (neoTOV)	untreated, stage II–III HR+/HER2- BC	Ph II ongoing
inavolisib (INAVO123)	1L HR+/HER2- mBC (ET sensitive)	Ongoing
inavolisib (INAVO120)	1L HR+/HER2- mBC (AI resistant)	✔ Approved
inavolisib (INAVO121)	Post-CDK4/6i HR+/HER2- mBC	Readout 2026
inavo + Phesgo (INAVO122)	1L HER2+ mBC	Readout 2027
inavolisib	1L HR+/HER2- *PIK3CA* wt mBC	Ph II ongoing

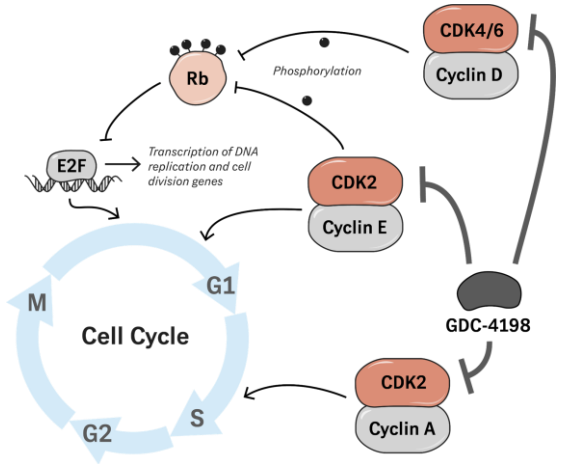
- Exploring combinations with giredestrant and CDK inhibitors (CDK4/6, CDK4, CDK4/2) in Ph Ib/II HR+/HER2- mBC
- Ph II (InavoPC) study in mCRPC ongoing
- Potential to expand into other tumors: Ph Ib/II signal seeking studies ongoing

1. Jhaveri KL et al., SABCS 2023; 2. Turner N et al., ASCO 2025; AE: Adverse event; CDK: AI: Aromatase inhibitor; Cyclin-dependent kinase; ET: Endocrine therapy; HER2: Human epidermal growth factor receptor 2; HR: Hazard ratio; HR+: Hormone receptor positive; (m)BC: (Metastatic) breast cancer; mCRPC: Metastatic castration-resistant prostate cancer; MoA: Mechanism of action; ORR: Objective response rate; OS: Overall survival; PI3K: Phosphoinositide 3-kinase; PIK3CAm: Phosphatidylinositol 3-kinase, catalytic, alpha mutated; PFS: Progression-free survival; SoC: Standard of care

RG6794 (GDC-4198): CDK4/2i to overcome CDK2-driven resistance

Encouraging activity in post-CDK4/6i setting; FPI in Ph II (MoonROSE) in combination with giredestrant

CDK4/6i resistance mechanism[1,2]



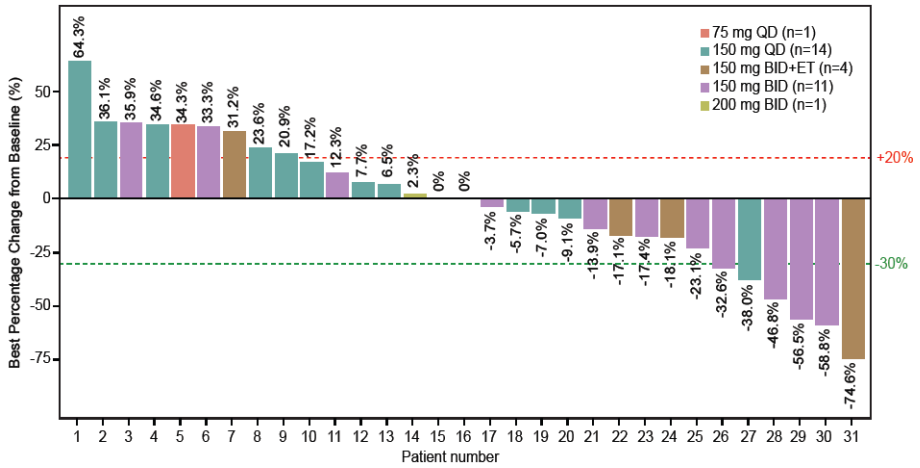
- CDK4/6i resistance remains a challenge; nearly all patients progress
- Data suggest that CDK2 activity is a potential mechanism of resistance

Activity against CDK4 & CDK2[3]

IC_{50} (nM)	CDK4	CDK2
GDC-4198	**0.3**	**4.6**
abemaciclib	0.8	270
palbociclib	2.3	>10^3

- Highly potent CDK4 inhibitor with substantial activity against CDK2
- More durable cell cycle arrest than approved CDK4/6is in preclinical studies

Preliminary activity in post-CDK4/6i setting[4]



- Preliminary evidence of disease control with sustained responses in heavily pre-treated post-CDK4/6i setting
- Well-tolerated as a single agent and in combination with ET; safety profile consistent with approved CDK4/6i
- Achieved FPI in Ph II (MoonROSE) study of GDC-4198 + gire vs abema + gire in CDK4/6i-experienced HR+ mBC

1. Wander SA, et al. Cancer Discov. 2020;10:11174-1193. 2. Turner NC, et al. J Clin Oncol. 2019;37(14):1169-1178. 3. Wander S, et al. Poster presentation (PO3-18-06) at 2023 SABCS; 4. Wander S, et al. Poster presentation (PS1-11-07) at 2025 SABCS; BC: Breast cancer; BIC: Best-in-class; BID: Twice daily; CDK: Cyclin-dependent kinase; HR+ BC: Estrogen receptor positive breast cancer; ET: Endocrine therapy; HER2: Human epidermal growth factor receptor 2; HR+: Hormone receptor positive

RG6596 (ZN-1041): Highly selective, brain-penetrant HER2 TKI in Ph II/III

Potential for combinations to improve HER2+ BC outcomes and prevent brain metastases

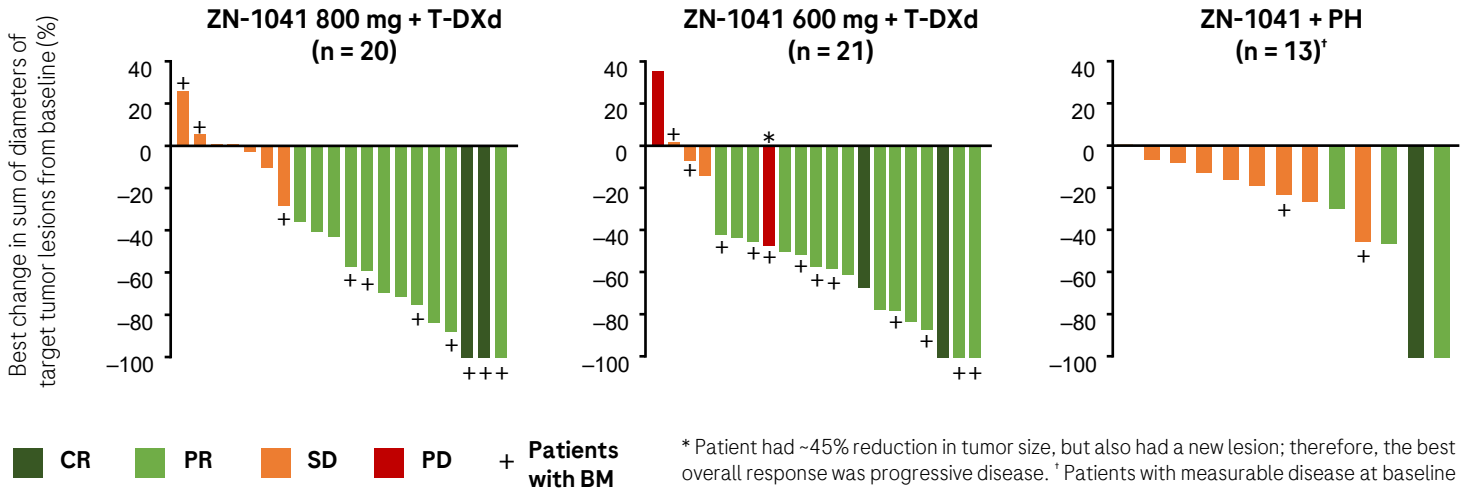
ZN-1041 properties[1]

TKI	$AUC_{br,u}$/ $AUC_{pl,u}$*	Efflux transporter (P-gp/BCRP) substrate?
tucatinib	0.004	Yes
lapatinib	0.010	Yes
neratinib	0.013	Yes
pyrotinib	0.024	Yes
epertinib	0.080	Yes
ZN-1041	**0.470–0.770**	**No**

- Despite recent advances in treating brain metastases, about half of patients still face CNS progression[2]
- ZN-1041 is differentiated by high BBB permeability and CNS retention
- Better pre-clinical activity than tucatinib

Global Phase 1c study results[3]

2026 ASCO ANNUAL MEETING



* Patient had ~45% reduction in tumor size, but also had a new lesion; therefore, the best overall response was progressive disease. † Patients with measurable disease at baseline

- Promising clinical activity in combination with standard anti-HER2 therapies (T-DXd and Phesgo)
- High disease control in combination with T-DXd (ORR >65%; DCR >90%) and notable continued responses in the PH maintenance setting (ORR >30.8; DCR 100%)
- Manageable safety profile, with predictable toxicity and low treatment discontinuations
- Initiated Phase II/III (BREnnA) study vs tucatinib in combination with Herceptin and capecitabine

1. Ma F, et al. ASCO 2023 (Abstract 1040; poster presentation); 2. Andre et al, Ann Oncol 2024; 3. Lin NU, et al. ASCO 2026 (Abstract 1055; poster presentation); * AUCbr,u and AUCpl,u are measurements of the unbound drug concentrations over time in the brain and plasma, respectively; the ratio of these two values is the gold-standard assessment of brain penetration for drugs. A higher AUCbr,u/AUCpl,u ratio indicates a greater ability of the drug to enter and remain in the CNS from the systemic circulation; AUC: Area under the curve; BBB: Blood-brain barrier; BCRP: Breast cancer resistance protein; BC: Breast cancer; BM: Brain metastasis; CNS: Central nervous system; DCR: Disease control rate; HER2: Human epidermal growth factor receptor 2; ORR: Objective response rate; PH: Phesgo; P-gp: P-glycoprotein; T-DXd, trastuzumab deruxtecan; TKI: Tyrosine kinase inhibitor

Multiple approaches to (K)RAS signaling inhibition, incl. combinations

Leveraging Roche/Genentech/Chugai R&D engines to build a portfolio diversified across targets and mechanisms

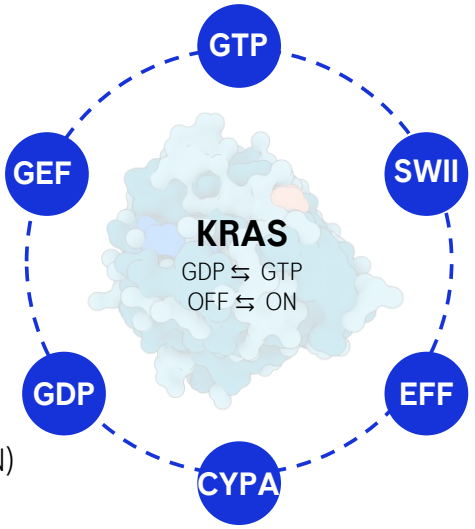
Orthosteric
Blocks GTP binding site directly

Catalytic inhibitors
Catalyzes GTP hydrolysis and conversion to OFF state

Switch II pocket
Specific allosteric site, reshapes GTP binding site

ON vs OFF inhibitors
Stabilize inactive GDP state (OFF) or GTP active state (ON)

Allosteric
Blocks effector docking (e.g. RAF)



Tricomplex
Molecular glue traps; current generation leverage Cyclophilin A

Roche RAS-targeted pipeline

Molecule	Indication	Precl.	Ph I	Ph II	Ph III	Status
divarasib	2L+ mNSCLC H2H vs sotorasib/adagrasib			KRASCENDO 1		✓
divarasib + pembro	1L mNSCLC			KRASCENDO 2		
divarasib	eNSCLC (adjuvant)			KRASCENDO 3		FPI exp. H2'26
KRAS G12Di	solid tumors					
panRASi	solid tumors					
pan-KRASi	solid tumors					
multiple undisclosed	solid tumors					

✓ Primary endpoint met

- Diverse portfolio with mutant-selective and pan-RAS/pan-KRAS inhibitors provide optionality for mono and combination therapy regimens in lung cancer and beyond
- Divarasib (KRAS G12Ci) and RG76620 (KRAS G12Di) potential for differentiation through greater potency and selectivity versus others

(e/m)NSCLC: (Early/metastatics) non small cell lung cancer; H2H: Head-to-head

Divarasib: Potentially BIC oral KRAS G12C inhibitor

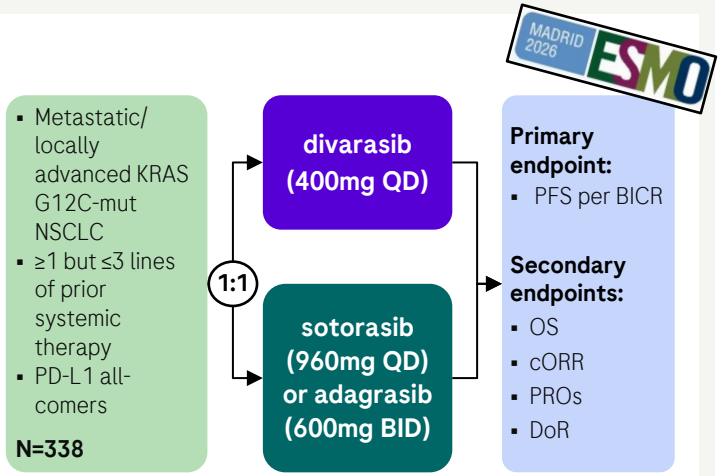
Positive Ph III (KRASCENDO 1) divarasib in 2L KRAS G12C mNSCLC (H2H vs sotorasib and adagrasib)

Potency and selectivity[1]

	IC_{50} (nM) / selectivity
divarasib	**0.18 / 16,000**
sotorasib	3.4 / >1,800
adagrasib	1.0 / 340

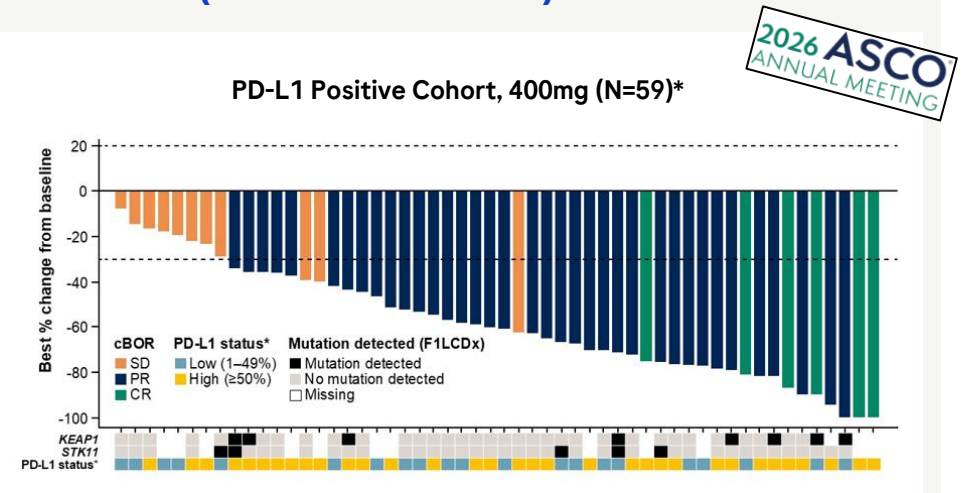
- 5-25 times more potent and 10-50 times more selective *in vitro* than sotorasib and adagrasib

Ph III (KRASCENDO 1) in 2L mNSCLC



- Positive Ph III (KRASCENDO 1) divarasib in 2L+ mNSCLC
- PFS superiority vs approved KRAS G12Ci; OS significance reached at interim analysis
- No new safety signals were observed
- Selected as Presidential Symposium at ESMO 2026

Ph II (KRASCENDO 170) in 1L NSCLC[2]



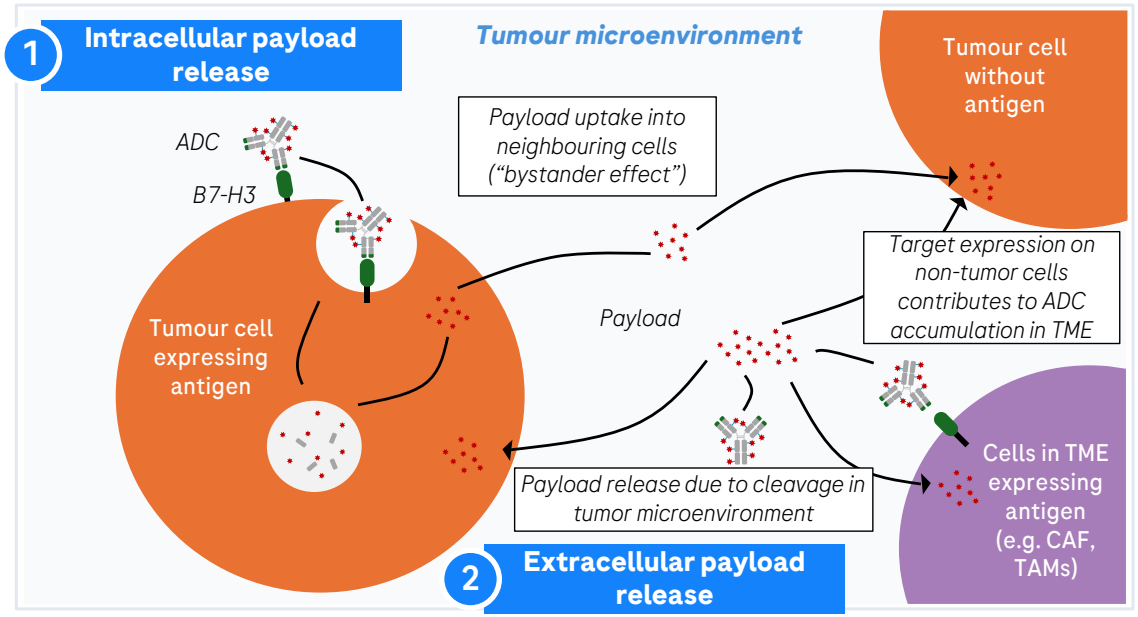
- PD-L1 pos.: reduction in tumor size in all patients with a post-baseline assessment, cORR 73%, mPFS 19.3m; PD-L1 neg.: unconfirmed ORR of 70%
- Divarasib plus pembro had a manageable safety profile
- Potential to be established as first 'chemo-free' regimen in 1L KRAS G12C NSCLC, regardless of PD-L1 status

1. Purkey H. et al., AACR 2022; 2. Skoulidis et al., ASCO 2026 * 3 patients did not receive treatment, 1 patient received divarasib 200 mg; (c)ORR: (Confirmed) objective response rate; CR: Complete response; H2H: Head-to-head; LFT: Liver functional test; (m)NSCLC: (Metastatic) non-small cell lung cancer; (m)PFS: (Median) progression free survival; OS: Overall survival

Tambotatug pelitecan: Potentially BIC B7-H3 ADC

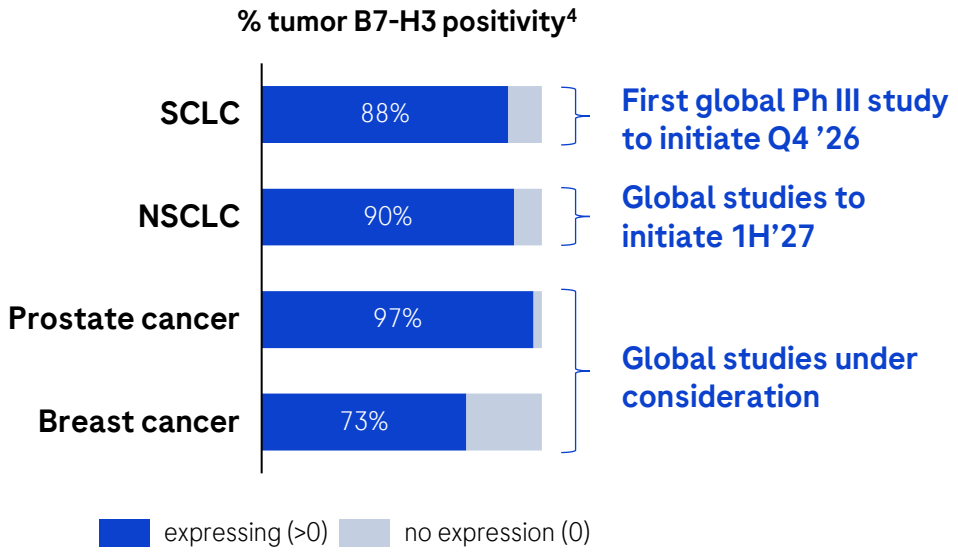
Accelerating global clinical development across key solid tumor indications

Next gen. B7-H3 targeted ADC with dual-release mechanism[1,2,3]



- Differentiated by a potent payload, high DAR=8 and highly hydrophilic and plasma-stable linker to minimize premature payload release and limit off-target toxicity, critical for future combination treatments
- Dual-release of payload: classical internalization amplified by tumor microenvironment activation

Foundational, potentially multi-indication asset



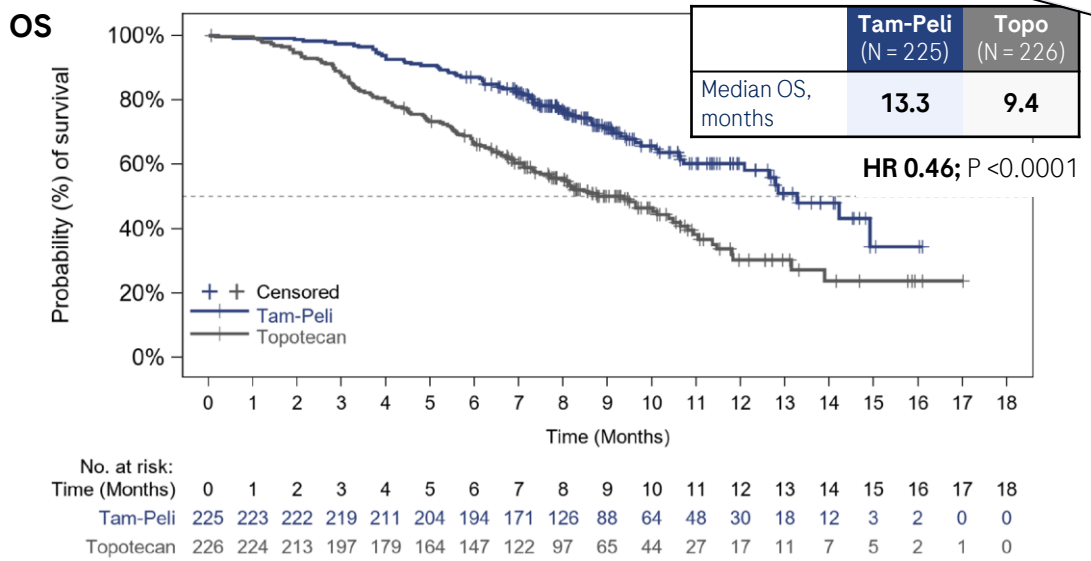
- B7-H3 is broadly expressed across key tumors with limited expression in healthy tissue
- Accelerating clinical development with multiple global trials across indications, starting with SCLC

1. Ashman, et al. Chem Soc Rev 2022; 2. Cazzamalli, et al. Nat Cancer 2025; 3. Cai, et al. AACR 2023 (Abs 596), and adapted from MediLink materials available at MediLink website; 4. Roche internal study results; ADC: Antibody drug conjugate; DAR: Drug-antibody ratio; NSCLC: Non small cell lung cancer; SCLC: Small cell lung cancer

Positive Ph III (TAISHAN-302) results in 2L SCLC in Chinese patients

Statistically significant improvement in PFS and OS; potentially differentiated safety profile with low ILD rates

Ph III (TAISHAN-302) OS results[1]



	Tam-Peli (N = 225)	Topo (N = 226)
Median OS, months	13.3	9.4

HR 0.46; P <0.0001

- Statistically significant and clinically meaningful OS improvement vs topotecan (54% reduction), PFS (71% reduction) and cORR (59.1%)
- Benefit maintained across all subgroups, incl. patients with baseline metastasis; intracranial PFS, OS and DCR superiority vs topotecan
- Significant proportion of patients with poor prognostic factors, e.g. metastatic burden, short chemo-free interval

Ph III (TAISHAN-302) safety[1]

	Tam-Peli (N = 224)	Topotecan (N = 217)
Median duration of exposure, weeks (range)	31.1 (3, 72.9)	10.1 (3, 70.4)
TRAEs, n (%)		
All grades	221 (98.7)	216 (99.5)
Grade ≥3	**104 (46.4)**	**162 (74.7)**
Serious TRAEs	**58 (25.9)**	**79 (36.4)**
Leading to dose interruption	78 (34.8)	80 (36.9)
Leading to dose reduction	**58 (25.9)**	**80 (36.9)**
Leading to discontinuation	13 (5.8)	6 (2.8)
Leading to death	**0**	**1 (0.5)**

- Treatment-emergent ILD/pneumonitis events at 4.9% in the Tam-Peli arm vs 1.4% with topotecan; grade 3 events at 0.9% in each arm, and no grade 4 or 5 events
- Lower Grade ≥3 TRAE vs. topotecan (46.4% vs 74.7%) confirming favorable and manageable safety profile

1. Zhang et al. WCLC 2026; cORR: Complete overall response rate; ILD: Interstitial lung disease; NSCLC: Non-small cell lung cancer; PFS: Progression free survival; OS: Overall survival; SCLC: Small cell lung cancer; TRAE: Treatment-emergent adverse events

Hematology

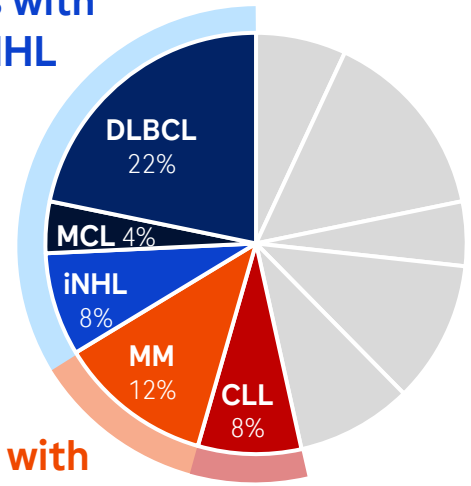
Matthew Hellmann
SVP and Global Head of Oncology and Hematology Product Development

Continuing to redefine the standard of care in hematology

Pioneering new modalities and targets: ADCs, bispecifics, small molecule degraders

mAb	Glycoeng. mAb	SM Inhibitor	ADC	Bispecifics	Combinations & novel modalities
1997	2013	2016	2019	2022/23	2026+
Rituxan Rituximab	GAZYVA obinutuzumab injection	VENCLEXTA venetoclax tablets	POLIVY polatuzumab vedotin	Lunsumio mosunetuzumab	**bexobrutideg** (BTK degrader)
(CD20 mAb)	(CD20 mAb)	(BCL-2i)	(CD79b ADC)	(CD20x CD3)	**cevostamab** (FcRH5 x CD3)
				COLUMVI glofitamab-gxbm (CD20x CD3)	**P-BCMA-ALLO1** (Allogeneic CAR-T)
					CD79a x CD19 x CD3 (Trispecific)

Building on anchor assets with novel combinations in NHL



Expanding into MM with differentiated modalities

Next-generation leadership in CLL

Source: Zhang et al., Global burden of hematologic malignancies and evolution patterns over the past 30 years, Blood Cancer Journal 2023; DLBCL: Diffuse large B-cell lymphoma; FL: Follicular lymphoma; MCL: Mantle cell lymphoma; NHL: Non-hodgkin lymphoma; MM: Multiple myeloma; len: lenalidomide; Pd: pomalidomide and dexamethasone; MDS: myelodysplastic syndrome; MPNs: myeloproliferative neoplasms ; r/r: relapsed/refractory

Hematology R&D focus areas

Building upon our leadership and experience with transformative medicines

Critical Capabilities		Examples
Novel modalities	*Investing in key technologies to engage unique set of targets*	**Degraders:** Bexobrutideg (BTK degrader) has potential to overcome resistance mechanisms with BTK inhibition
Combinations	*Building on top of anchor assets with novel combinations*	**SKYGLO:** Columvi + Polivy –R-CHP builds on top of POLARIX regimen as standard of care in 1L DLBCL **Cevostamab:** Novel target with lower risk of infection enables combinability in earlier lines
Curative potential	*Leverage best-in-disease assets, which offer the potential for cure / normalization of disease*	**Zemocimig (NXT007):** Potential to achieve hemostatic normalization
Improving patient experience	*Developing manageable regimens which can be used across treatment settings*	**Hemlibra/zemocimig autoinjector:** Continuing to improve administration with new delivery options

Hematology pipeline

Ph I

RG6538[1]	**P-BCMA-ALLO1** r/r MM
RG7828	**Lunsumio monotherapy + combos** heme tumors
RG6026	**Columvi monotherapy + combos** heme tumors
RG7473[1]	**bexobrutideg combinations[1]** 1L+ CLL
RG6733	**undisclosed** r/r MM
RG6735	**undisclosed** heme tumors

Ph II

RG6797	**SPK-8011QQ** hemophilia A

Ph III

RG6026	**Columvi** 1L DLBCL, 1L unfit DLBCL, r/r MCL
RG7828	**Lunsumio** 1L FL, 2L+ FL
RG6107	**PiaSky** aHUS
RG6013	**Hemlibra** Type 3 VWD
RG6160	**cevostamab** r/r MM
RG6512	**zemocimig (NXT007)** hemophilia A
RG7473[1]	**bexobrutideg[1]** 2L+ CLL

Registration

RG7828	**Lunsumio + Polivy** 2L+ DLBCL

Small molecule
Antibody
Bispecifics
Gene therapy
Allogeneic CAR-T cells
Protein degraders

NHL
CLL
MM
Non-malignant heme

1. In collaboration with Nurix Therapeutics; aHUS: Atypical hemolytic uremic syndrome; CLL: Chronic lymphocytic leukemia; DLBCL: Diffuse large B-cell lymphoma; FL: Follicular lymphoma; MCL: Mantle cell lymphoma; MDS: Myelodysplastic syndrome; MM: Multiple myeloma; R/r: Relapsed/refractory; VWD: Von Willebrand disease

Extending the standard of care in NHL

Building on anchor assets with novel combinations

Increasing the cure rate in DLBCL

Indication	Regimen		Ph1 / Ph2 / Ph3	
1L DLBCL	Polivy + R-CHP	✓	POLARIX	
1L DLBCL	Columvi + Polivy + R-CHP		SKYGLO	Data expected 2027
1L DLBCL (unfit)	Columvi + Polivy + R		EVERGLO	New Ph III study to be initiated in elderly, unfit, and frail pts
2L+ DLBCL	Lunsumio + Polivy	✓	SUNMO	Filed in US in Q2 (PDUFA: Feb 2027)
2L+ DLBCL	Columvi + GemOx	✓	STARGLO	
3L+ DLBCL	Columvi	✓	NP30179	

First-in-class potential in MCL

Indication	Regimen	Ph1 / Ph2 / Ph3	
2L+ MCL	Columvi	GLOBRYTE	
2L+ MCL	Columvi	Ph I	Pivotal Ph I data expected 2027 (BTD)

Moving into earlier lines in FL

Indication	Regimen		Ph1 / Ph2 / Ph3	
1L FL	Lunsumio + len		MorningLYTE*	LPI achieved
2L+ FL	Lunsumio + len	✓	CELESTIMO	Met Primary Endpoint (PFS)
3L+ FL	Lunsumio	✓	GO29781	

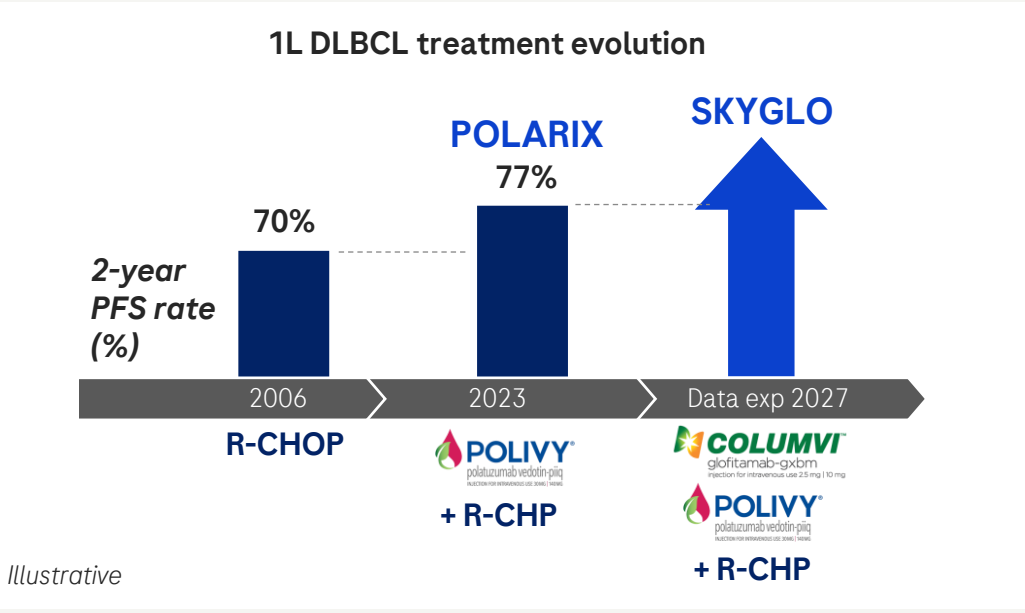
✓ *Approved or primary endpoint met*

1. Zhang et al., Global burden of hematologic malignancies and evolution patterns over the past 30 years, Blood Cancer Journal 2023; * Sponsored by LYASRC; CLL: Chronic lymphocytic leukemia; DLBCL: Diffuse large B-cell lymphoma; FL: Follicular lymphoma; MCL: Mantle cell lymphoma; NHL: Non-hodgkin lymphoma; r/r: relapsed/refractory; BTD: breakthrough therapy designation

1L DLBCL: SKYGLO has potential for BIC efficacy and safety

Continuing to advance the standard of care in a curative setting

SKYGLO builds on POLARIX regimen in 1L DLBCL



- Polivy + R-CHP (POLARIX) is established SOC in 1L DLBCL, most prescribed 1L regimen among IPI 2-5 patients in the US
- Columvi+Polivy-R-CHP (SKYGLO) is the only regimen in Ph III to build on top of POLARIX in 1L DLBCL; readout expected 2027

Ph Ib: 100% ORR with differentiated safety profile[1]

	%	Columvi + R-CHOP (n=56)	Columvi + Polivy+ R-CHP (n=24)
Efficacy Summary	**ORR**	93%	100%
	CMR	86%	96%
Safety Summary	**CRS**	10.7%	12.5%
	Gr 1	7.1%	12.5%
	Gr 2	3.6%	0

- Potential for best-in-class efficacy: Columvi + Polivy-R-CHP 100% ORR, 96% CMR, mPFS/mDOR not reached
- Potential for best-in-class safety: low CRS rates: 12.5% grade 1 CRS; no grade 2-5 CRS; regimen is suitable for outpatient administration

1. Topp M et al, ICML 2025; BIC: Best in class; CMR: Complete metabolic response; DLBCL: Diffuse large B cell lymphoma; FU: Follow-up; HGBCL: High grade B-cell lymphoma; IRC: Independent review committee; NOS: Not otherwise specified; ORR: Overall response rate; PFS: Progression-free survival; Pola-R-CHP: Polivy + Rituxan + cyclophosphamide + hydroxydaunorubicin + prednisone; R-CHOP: Rituxan + cyclophosphamide + doxorubicin + vincristine + prednisone; SoC: Standard of care

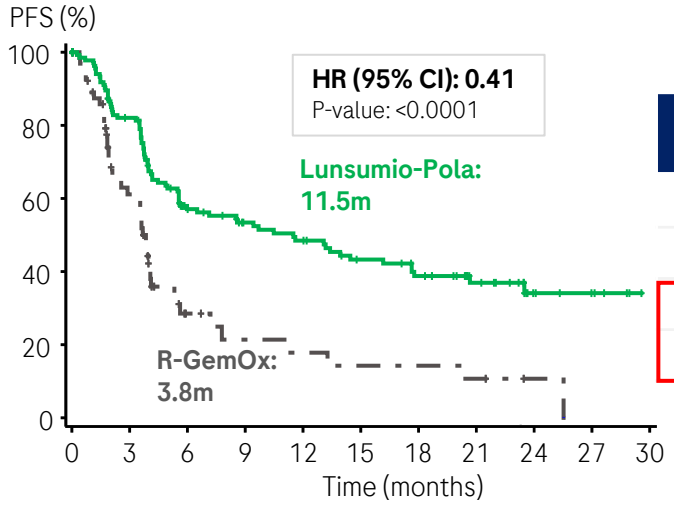
Lunsumio with chemo free combos in 2L+ DLBCL and 2L+ FL

Expanding into earlier lines with positive Phase III SUNMO in 2L+ DLBCL and CELESTIMO in 2L+ FL

2L+ DLBCL: SUNMO shows strong efficacy and lowest CRS

Ph III SUNMO: Lunsumio+Polivy in 2L+ DLBCL[1]

✔ *Filed with FDA (PDUFA Feb 9)*



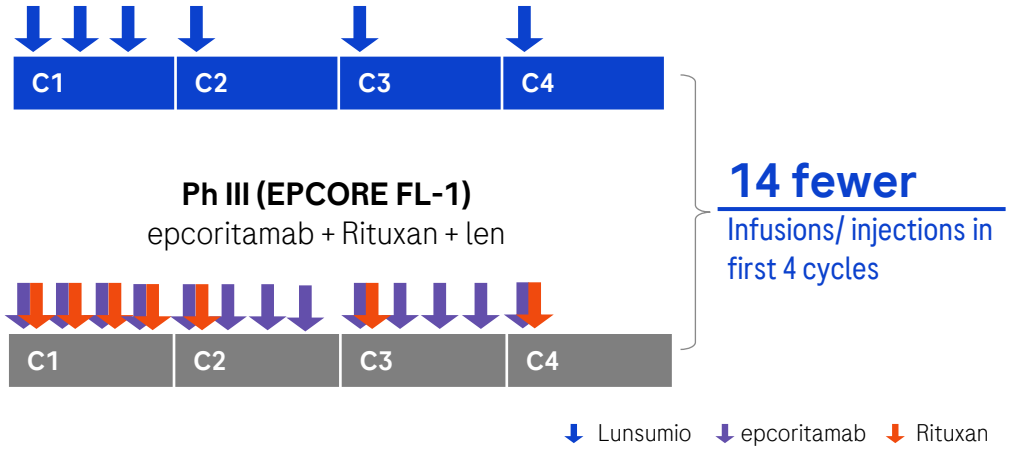
Patients with ≥1 CRS AE (%)	Lunsumio+Pola (n=135)
Any grade	26%
Grade 1	22%
Grade 2	4%
Grade 3	<1%

- Lunsumio + Polivy tripled median PFS vs. R-GemOx
- Chemo-free, fixed duration regimen, suitable for outpatient use
- Lowest CRS incidence and severity among T-cell bispecifics; <5% of patients with Gr 2+ CRS

2L+ FL: CELESTIMO differentiated by convenience

Ph III (CELESTIMO): 2L+ FL
Lunsumio + len

✔ *Met primary endpoint*



14 fewer
Infusions/ injections in first 4 cycles

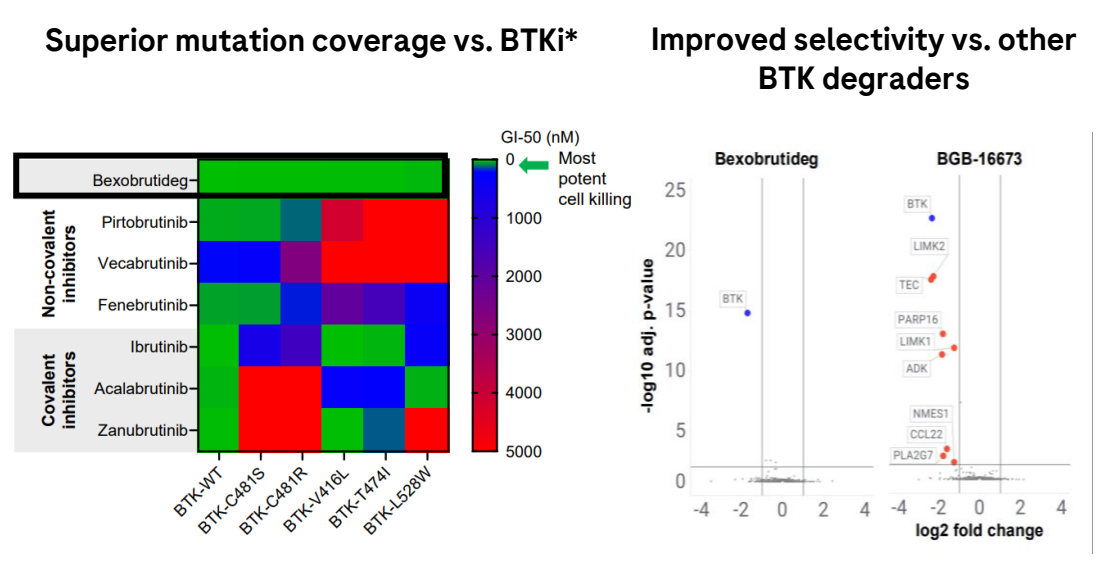
- Doublet regimen (Lunsumio + len) with simple dosing schedule, fixed duration
- Minimal steroid pre-medication required (3 days vs. 16 in C1)
- Suitable for outpatient administration, community

1. Westin et al. ICML 2025; GemOx: Gemcitabine + oxaliplatin; PFS: Progression-free survival; R/R: relapsed/refractory

Bexobrutideg: Potential best-in-class BTK-targeted therapy

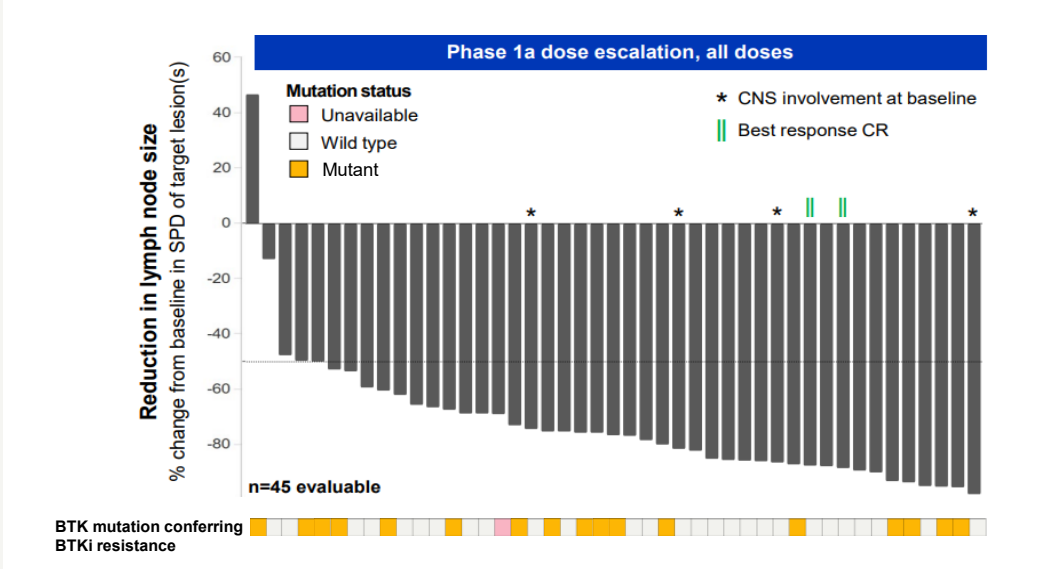
Ph III in 2L+ CLL has been initiated; planned combinations in CLL (1L & R/R), MCL, and WM

BTK degradation addresses resistance to BTKi



- Complete target ablation and scaffold removal yield deeper pathway inhibition than BTK inhibitors
- Superior *in vitro* potency against both wildtype and mutant BTK
- Best-in-class potential, with improved selectivity vs. other BTK degraders, supporting differentiated tolerability profile
- Exceptional CNS penetrance

Strong Ph I efficacy in late-line r/r CLL



- ORR: 83%, PFS of 22.1m in heavily pre-tx pts incl. difficult-to-treat subgroups (BTK mutations, high-risk mol. features, CNS disease)
- Potential for BIC safety with low rates of SAEs, ~5% D/C rate
- Ph III DAYBreak-CLL 306 in r/r CLL achieved FPI in Q3
- Ph I/II combo with Venclexta +/- CD20 in CLL initiated

Bexobrutideg in collaboration with Nurix Therapeutics; Source: Phase 1a, ASH 2025 update (Sep 2025 cutoff); *In CRISPR-engineered TMD8 cells; GI-50, growth inhibition 50%; r/r: relapsed /refractory; CLL: Chronic Lymphocytic Leukemia; MCL: Mantle Cell Lymphoma; WM: Waldenström's Macroglobulinemia

Cevostamab: First-in-class FcRH5-targeted therapy in MM

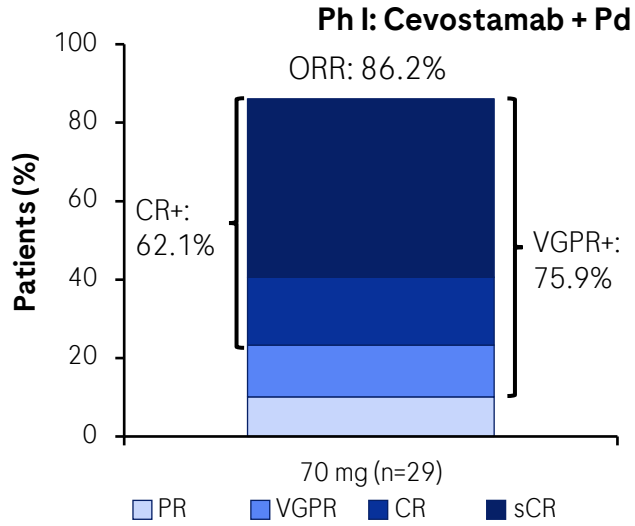
FcRH5 x CD3 bispecific, potential combination partner of choice with differentiated safety profile

FcRH5: high unmet need for new targets in MM



- FcRH5 expressed in ~100% of MM cells, independent of BCMA or GPRC5D expression
- Unlike BCMAs, cevostamab not associated with persistent depletion of B-cells; lower risk of severe infection
- Unlike GPRC5Ds, cevostamab not associated with GI/skin toxicity

Strong efficacy and differentiated safety in r/r MM



MRD- CR rates* (CR+ evaluable pts)	**93.8%**
18m PFS	**67.4%**
mPFS	**NR**

Median follow-up: 20m

- Differentiated and favorable safety profile: At 70mg dose:
  - 17.2% gr 3 infection rate, no gr 4, one gr 5
  - Restoration of humoral immunity without continuous IVIG
- Ph III (CEVOLUTION) cevostamab + Pd in r/r MM ongoing
- Initiating combinations in 1L

Source: 1. Spencer et al., IMS 2026; *MRD-negative CR rates at the 10^{-5} level among MRD-evaluable CR+ patients; MRD by next generation sequencing (ClonoSEQ); MRD-evaluable patients were those who achieved CR+ and had ≥1 valid MRD result (positive or negative) at any time; BCMA: B cell maturation antigen; FcRH5: Fc receptor-homolog 5; IVIG: Intravenous immunoglobulin; MM: Multiple myeloma; ORR: Overall response rate; Pd: Pomalidomide and dexamethasone; r/r: Relapsed/refractory; VGPR: Very good partial response; MRD: minimal residual disease

Roche is set to again transform care in Hemophilia A

Zemocimig is the only molecule going head-to-head vs. Hemlibra in Ph III

Hemlibra: 2026 Lasker award

Roche is leading evolution of the Hemophilia A standard of care

	Dosing/admin	Efficacy
FVIII prophylaxis	Daily to weekly IV	Common breakthrough bleeds
HEMLIBRA® emicizumab-kxwh	Weekly to Q4W SC (autoinjector)	~80% bleed-free[1]
zemocimig (NXT007)	Monthly SC (autoinjector)	Potential for Hemostatic normalization
SPK-8011QQ	One-time treatment	Potential for Hemostatic normalization

Non-malignant hematology: Key newsflow

Indication	Regimen	Ph1 / Ph2 / Ph3	
Hemophilia A	zemocimig vs. FVIII	ZEBRHA 1	• Ph III data exp. late 2027 • ZEBRHA-2 is the only head-to-head trial vs. Hemlibra
	zemocimig vs. Hemlibra	ZEBRHA 2	
	zemocimig (pediatric)	ZEBRHA 3	
	SPK-8011QQ	Ph II	• FPI achieved in Ph II in Q3
VWD	Hemlibra	WILL-EMI	
PNH	PiaSky	COMMODORE 1/3 ✓	
aHUS	PiaSKy	COMMUTE a/p ✓	• Filing in aHUS expected 2026



Hemlibra autoinjector potential to launch by year end 2027

✓ *Approved or primary endpoint met*

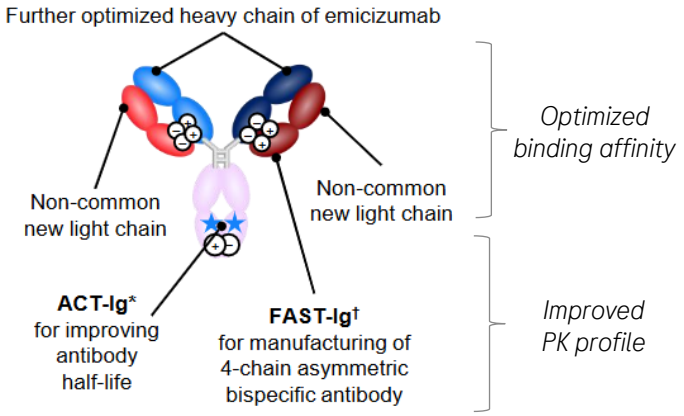
1. Based on RWD from McCary I, et al. Haemophilia 2020, Wall C, et al. ISTH 2020, Poon M-C, et al. ASH 2022 and Khairnar R, et al. ASH 2021; aHUS: atypical hemolytic uremic syndrome; SC: subcutaneous; IV: intravenous

Zemocimig (NXT007): BIC potential in Hemophilia A

Potential to achieve true hemostatic normalization; Ph III data expected late 2027

Next-gen FIXa/FX bispecific

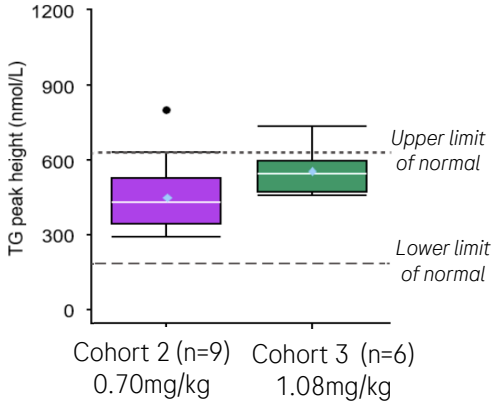
Zemocimig molecule design



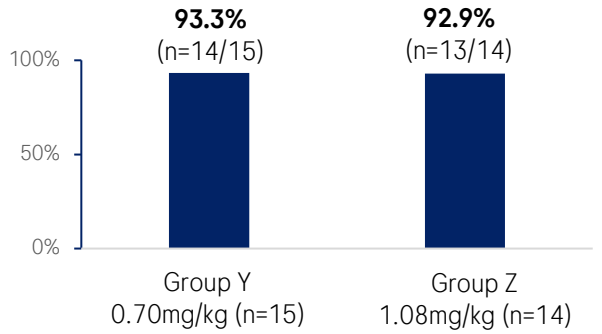
- Zemocimig is a next-gen, bispecific, based on Hemlibra and optimized to provide increased FVIIIa-mimetic activity and a longer half life
- ~30-fold more potent than Hemlibra; low volume monthly injection with autoinjector
- Head-to-head trial vs. Hemlibra (powered for non-inferiority and superiority)

Ph I/II combined analysis[1]

Hemostatic normalization achieved at Ph II doses[2]



Proportion of participants with zero treated bleeds during the NXT007 maintenance period[1]



- >90% of pts with zero treated bleeds in the two highest dose cohorts.[1]
- Thrombin generation at Ph II doses is well into the normal range of people without Hemophilia A**
- Favorable safety profile, with no observed thrombotic events
- Only one case of ADAs partially affecting PK out of 42 pts. NXT007 levels stabilized at ~2.5 µg/mL (considered equivalent to level of protection achieved with Hemlibra). Patient continued treatment with zero treated bleeds during study

1. Mancuso, et al., ISTH 2026; 2. Kiialainen et al., ASH 2025 *One notable outlier in Group Z had four treated spontaneous joint bleeds in the maintenance period. This participant had a leftknee replacement approximately 4 years prior to study entry and a left knee arterial embolization approximately 1 year prior to entry due to recurrent joint bleeds. The bleeds were considered secondary to direct trauma to an aberrant arterial vessel close to the replaced knee surface. **Normal range generated from plasma taken from 120 healthy volunteers; NXT007 TG peak height at steady state. ABR, annualized bleeding rate; CI, confidence interval.

Roche

Neurology

Hideki Garren

SVP and Global Head of Product Development Neurology

Neurology R&D focus areas

End-to-end investment in MS and AD

Critical Capabilities	Examples
Therapeutic modalities	Brainshuttle™ in AD (trontinemab) readout in early 2028 Brainshuttle™ in MS (RG6035, aCD20) moving to Ph II
Pharma + Dia partnership	TRAVELLER prescreening program in AD uses blood-based biomarkers to reduce diagnostic burden (CSF/PET) Elecsys® NfL blood test detects disease activity
Prevention approaches	Initiating Ph III (PrevenTRON) of trontinemab in preclinical AD
E2E investment	Invest end-to-end in Alzheimer's disease and multiple sclerosis from discovery, R&D, to commercialization Invest into breakthrough innovation in Parkinson's disease

AD: Alzheimer's Disease; CSF: Cerebrospinal fluid; E2E: End-to-end; MS: Multiple Sclerosis; NfL: Neurofilament light chain; PD: Parkinson's Disease; PET: Positron emission tomography

Neurology pipeline

Fenebrutinib filed in RMS and PPMS; Enspryng filing accepted in MOGAD with Priority Review

Phase I		Phase II		Phase III		Registration	
RG6035	**Brainshuttle™ CD20** Multiple Sclerosis	RG6182	**MAGLi** Multiple Sclerosis	RG6102	**trontinemab** Early AD, preclinical AD	RG7845	**fenebrutinib** RMS, PPMS
RG6627	**APOE ASO** Alzheimer's	RG6289	**nivegacetor (GSM)** Alzheimer's	RG6168	**Enspryng** AIE RD	RG6168	**Enspryng** MOGAD RD
RG6662	**HTT miRNA GT (SPK-10001)** Huntington's RD	RG6356	**Elevidys[1]** DMD (0-<4 y.o.) RD	RG6356	**Elevidys[1]** DMD (>8 y.o.[2]) RD		
		RG6434	**NLRP3i** Alzheimer's, Parkinson's	RG7935	**prasinezumab** Parkinson's		

Small molecule
Antibody
RNA-targeting therapeutics
Bispecifics
Gene therapy
Brainshuttle™

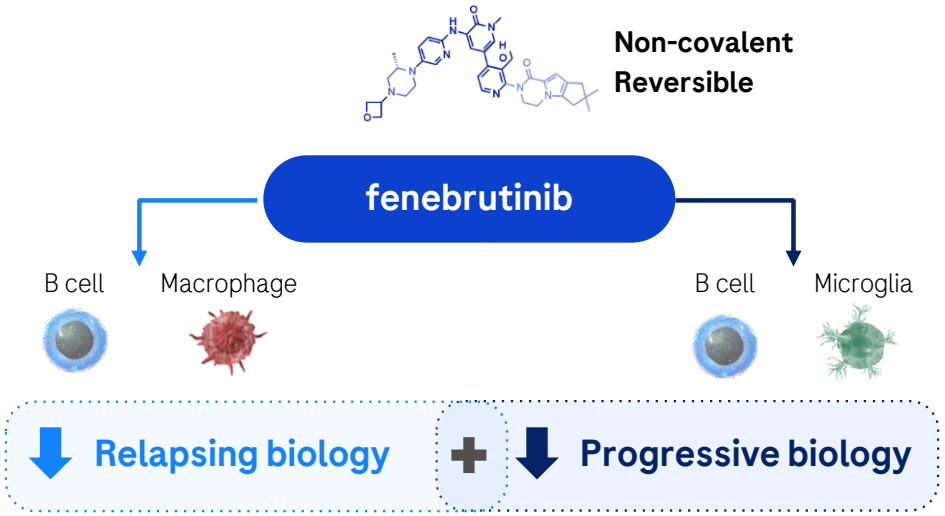
Neuroimmunologic disorders
Neurodegenerative diseases
Neuromuscular disorders
RD = Rare disease

1. Elevidys in partnership with Sarepta Therapeutics; 2. Ambulatory, 8-<18 yrs; non-ambulatory, all ages; AIE: Autoimmune encephalitis; DMD: Duchenne muscular dystrophy; GSM: Gamma-secretase modulator; MAGL: Monoacylglycerol lipase; MOGAD: Myelin oligodendrocyte glycoprotein antibody-associated disease; PPMS: Primary progressive multiple sclerosis; RMS: Relapsing multiple sclerosis; y.o.: year old

Fenebrutinib: Only BTKi with positive results across RMS/PPMS

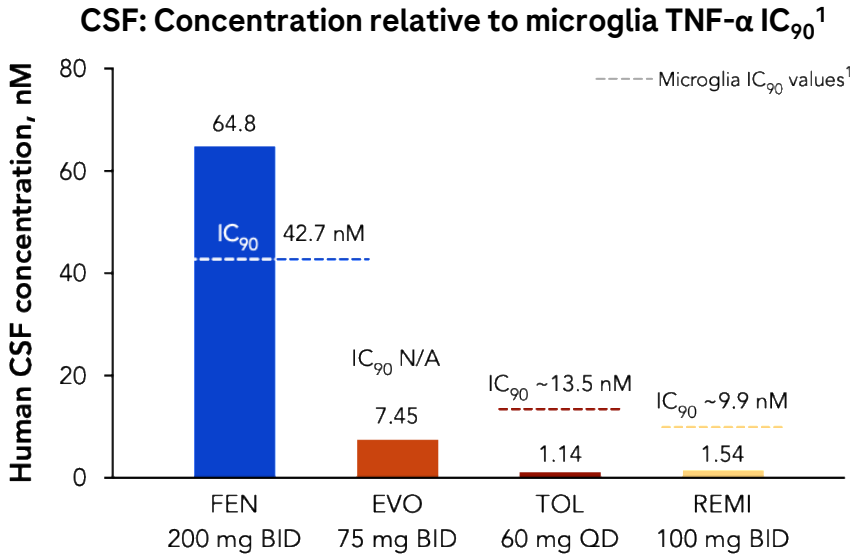
Fenebrutinib US filing completed in RMS and PPMS

Designed to control relapsing and progressive biology



- Fenebrutinib is the only non-covalent, reversible BTKi in MS
- Highly selective with unique structure and binding mode that targets dual drivers of disability in MS

Inhibition of B-cells and microglia in CNS



- Optimized PK profile maintains high systemic and CNS exposure
- Fenebrutinib achieves near maximal inhibition of B-cells and microglia in the CNS (associated with progressive biology)[2,3]

Sources: 1. Johnson A, et al. Presented at ACTRIMS 2025 (Poster P146); 2. Crawford J, et al. J Med Chem 2018;61:2227–452; 3. Bar-Or A, et al. Presented at CMSC 2025 (Oral presentation PLA-A5); CSF: Cerebrospinal fluid; CNS: Central nervous system; PPMS: Primary progressive multiple sclerosis; RMS: Relapsing multiple sclerosis

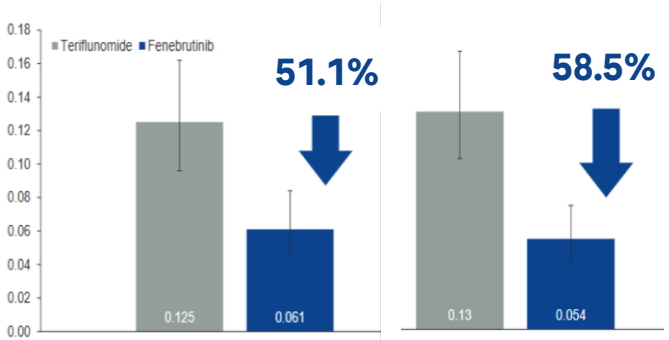
Fenebrutinib in RMS with lowest ARR seen in Phase III trials

>50% improvement in ARR, >70% reduction in in new brain lesions, clinically meaningful improvements in cCDP

Substantial impact on relapsing biology, MRI activity, and disease progression

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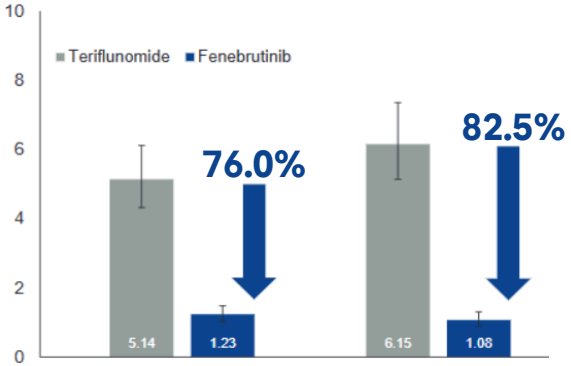
Adjusted ARR (95% CI)[1]



FENhance 1
0.49 (0.33-0.73)
P<0.001

FENhance 2
0.42 (0.28-0.61)
P<0.00001

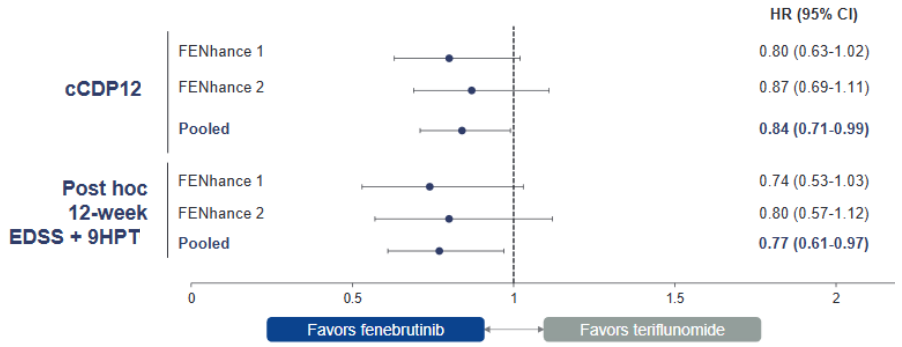
Annualized number of NET2Ls



FENhance 1
0.24 (0.19-0.31)
P<0.0001

FENhance 2
0.17 (0.13-0.23)
P<0.0001

cCDP12[2]



- >50% reduction in ARR vs. teriflunomide: translates to an average of one relapse every ~17 years (lowest ever demonstrated in a Ph III trial)
- >70% reduction in the number of new brain MRI lesions vs. teriflunomide (T1 Gd+ and NET2Ls)
- Ph II FENopta OLE showed sustained suppression of relapses and MRI lesions (Ph III longitudinal data to be presented at MSToronto)
- Clinically meaningful and nominally significant trend in both pooled cCDP12 and CDP12-EDSS+9HPT (composite endpoint used in ORATORIO-HAND study)

Sources: Oh J, et al. AAN 2026, 1. Adjusted for the presence of T1 Gd+ lesions and EDSS score (<4.0 vs ≥ 4.0) at baseline. In FENhance 2, EDSS score was removed due to non-convergence of the model. 2. Defined as ≥ 1.0 or ≥ 0.5-point increase in EDSS, with baseline EDSS ≤5.5 or >5.5, respectively; or ≥20% increase from baseline in T25FWT; or ≥20% increase from baseline in 9HPT. Pooled results shown are not α controlled; Defined as ARR: Annualized relapse rate; cCDP12: 12-week composite confirmed disability progression; EDSS: Expanded disability status scale; NET2L: new or enlarging T2 lesions; RMS: Relapsing multiple sclerosis; 9HPT: Nine-hole peg test

Fenebrutinib ARR reduction positions it as a high efficacy DMT

Fenebrutinib is the first and only oral to demonstrate >50% improvement in ARR in RMS

Neurology®

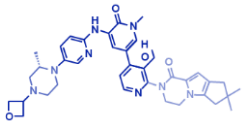
	DMT, class	Comparator	Clinical trial	Reduction of ARR
High efficacy[a]	Anti-CD20 monoclonal antibody			
	Ocrelizumab [27]	IFN beta-1a SC 44 mg	OPERA I/II	46–47%
	Ofatumumab [28]	Teriflunomide 14 mg	ASCLEPIOS I/II	50–60%
	Ublituximab [29]	Teriflunomide 14 mg	ULTIMATE I/II	50–58%
	Anti-CD52 monoclonal antibody			
	Alemtuzumab [30, 31]	IFN beta-1a SC 44 mg	CARE-MS I/II	49–55%
	Anti-a4 integrin receptor monoclonal antibody			
	Natalizumab [32]	Placebo	AFFIRM	68%
Moderate efficacy[b]	S1P receptor modulators			
	Fingolimod [33, 34]	Placebo	FREEDOMS I/II	48–54%
	Siponimod [35]	Placebo	EXPAND	55%
	Ozanimod [36, 37]	IFN beta-1a IM 30 mg	RADIANCE/ SUNBEAM	21–48%
	Ponesimod [38]	Teriflunomide 14 mg	OPTIMUM	31%
	Purine analog			
	Cladribine [39]	Placebo	CLARITY	58%
	Fumarates			
	Dimethyl fumarate [40, 41]	Placebo	CONFIRM/DEFINE	44–53%
	Diroximel fumarate [42]		EVOLVE-MS-1	
Low efficacy[c]	Pyrimidine synthesis inhibitor			
	Teriflunomide [43, 44]	Placebo	TOWER/TEMSO	31.5–36%
	Amino acid copolymer			
	Glatiramer acetate [40, 45]	Placebo	CONFIRM/GALA	29–34%
	IFNs			
	IFN beta-1a [46]	Placebo	PRISMS	33%

High efficacy:
~50-60%
reduction of ARR

Moderate efficacy:
~40-50%
reduction of ARR

Low efficacy:
~30-40%
reduction of ARR

Fenebrutinib

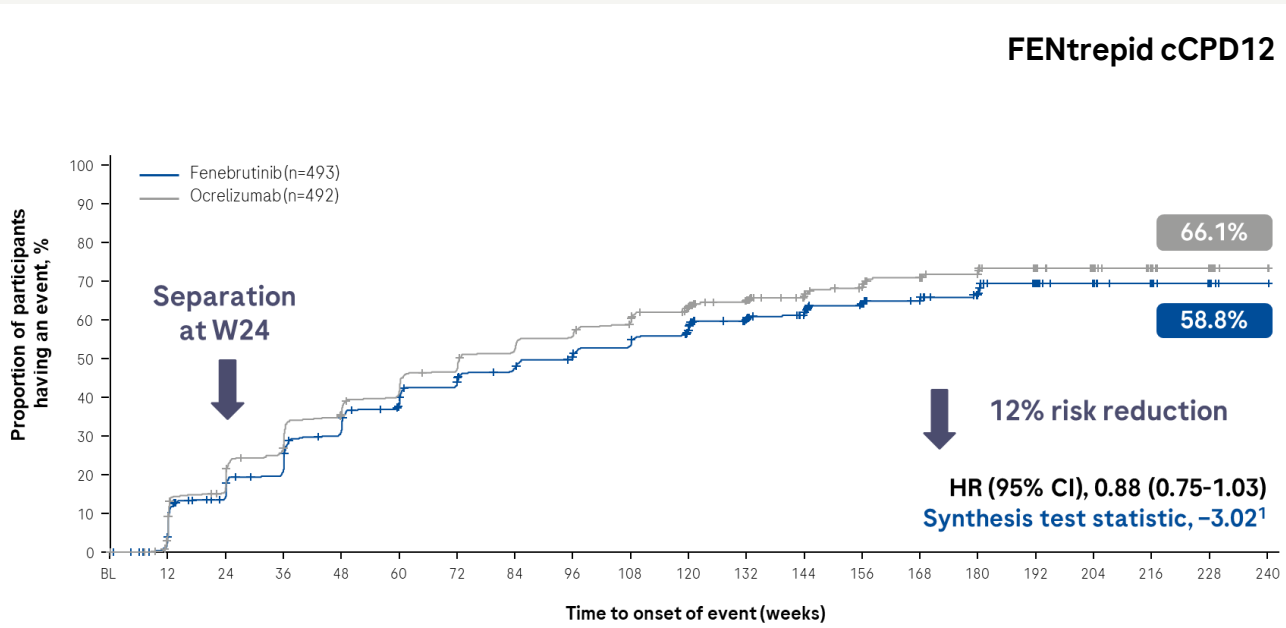


Singer et al., J Neurol. 2024 Apr 14;271(6):3116–3130; ARR: Annualized relapse rate; DMT: Disease modifying therapy; RMS: Relapsing multiple sclerosis

Fenebrutinib: Only BTKi with positive results in PPMS and vs. CD20

Consistent numeric improvements on disease progression observed across all three MS studies

Fenebrutinib is the only molecule with head-to-head data vs. CD20



	Proportion with event, n (%)		HR (95% CI)	Risk reduction with fenebrutinib
	Fenebrutinib (n=493)	Ocrelizumab (n=492)		
cCDP12	290 (58.8)	325 (66.1)	0.88 (0.75-1.03)	12%
CDP12-EDSS	146 (29.6)	174 (35.4)	0.84 (0.68-1.05)	16%
T25FWT	242 (49.1)	263 (53.5)	0.93 (0.78-1.11)	7%
9HPT	82 (16.6)	114 (23.2)	0.74 (0.56-0.98)	26%

Post hoc analysis based on composite endpoint used in ORATORIO-HAND[1]

CDP12-EDSS + 9HPT — HR (95% CI), 0.78 (0.64-0.95) — 22% risk reduction

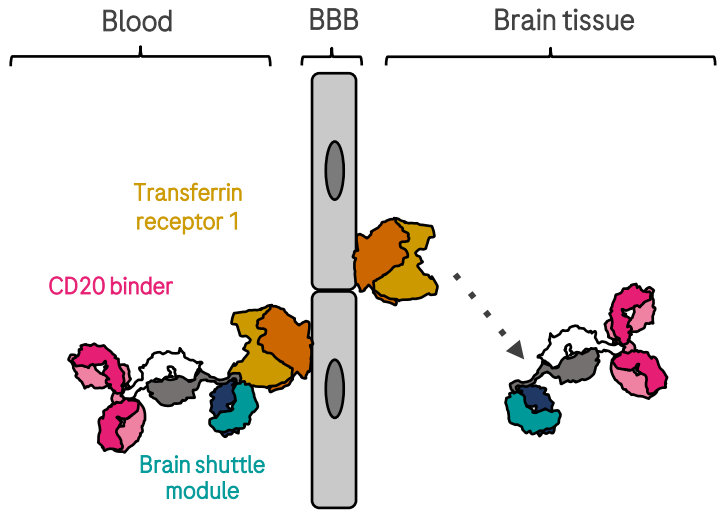
- Curves started to separate by week 24, which lasted throughout the entire treatment duration of >120 weeks
- Fenebrutinib demonstrated nominally significant 22% reduction in disability progression on CDP12-EDSS+9HPT (composite endpoint used in ORATORIO-HAND study)

Sources: Bar-Or A et al. ACTRIMS 2026; 1. Synthesis test statistic <−1.96 indicated noninferiority, with supplementary analysis based on pre-specified 95-95 fixed margin of 1.06 for the HR upper bound; cCDP12:12-week composite confirmed disability progression; EDSS: Expanded disability status scale; MS: Multiple sclerosis; PPMS: Primary progressive multiple sclerosis; 9HPT: Nine-hole peg test

Multiple sclerosis: Early development pipeline

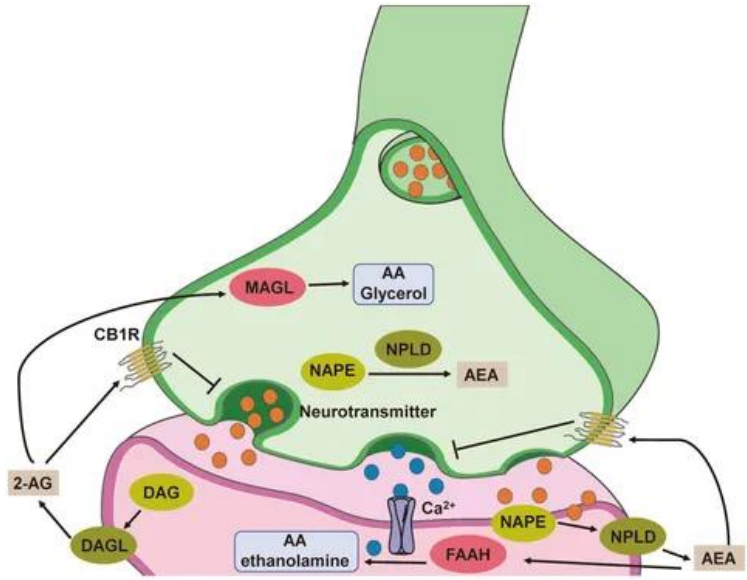
Diverse portfolio of modalities build on Roche's leadership in MS

RG6035: BrainShuttle™ CD20



- Leverages Roche's proprietary BrainShuttle™ technology to address limitations of CD20 antibodies to penetrate CNS
- Advancing to Ph II in MS

RG6182: MAGLi



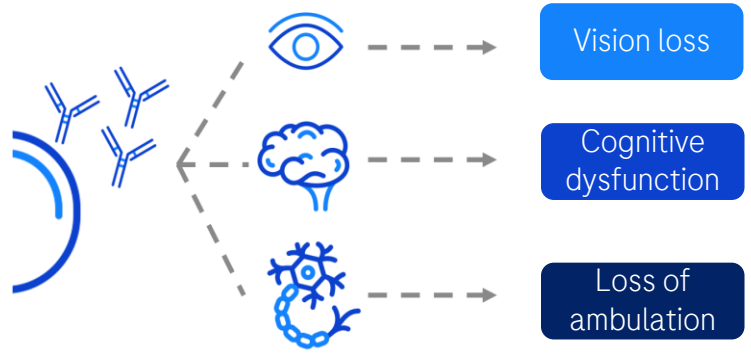
- Unique and multimodal MoA to target compartmentalized inflammation in the CNS
- Ph II (Mintaka) trial in MS initiated; designed to evaluate MAGLi in combination with Ocrevus

BBB: Blood brain barrier; CNS: Central nervous system; DAG: Diacylglycerol; MAGLi: Monoacylglycerol lipase inhibitor; MoA: Mechanism of action; MS: Multiple sclerosis

Enspryng: Potential to be the first approved treatment in MOGAD

US filing in MOGAD accepted with Priority Review (PDUFA Jan 10)

Disability in MOGAD driven by relapses

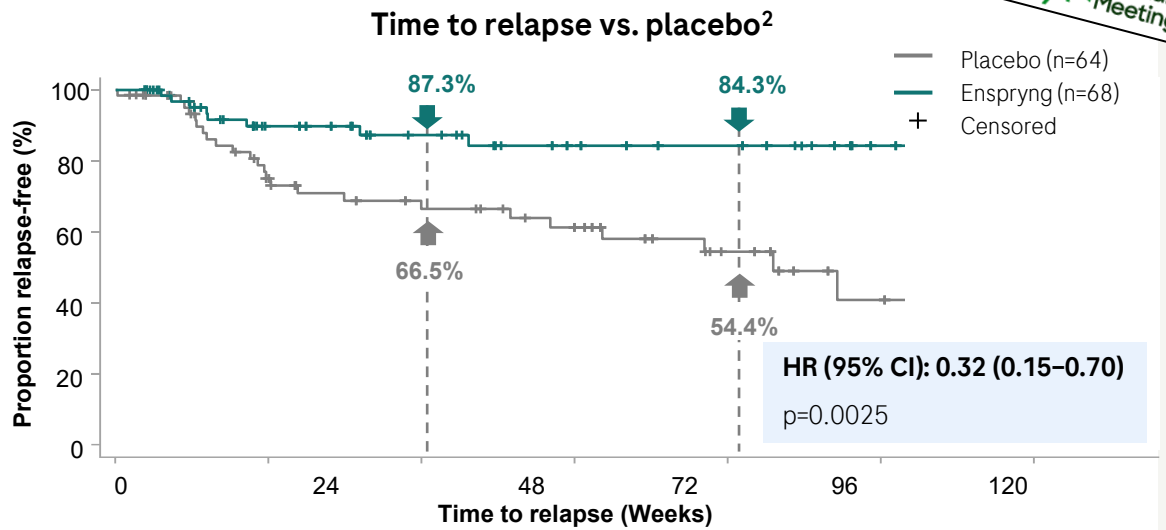


IL-6 levels elevated in CSF and serum of patients with MOGAD, contributing to autoantibody production

- MOGAD is a rare autoimmune condition that presents with inflammatory demyelinating attacks affecting the optic nerve, brain, and spinal cord
- No approved therapies; disease management frequently relies on acute treatment with high-dose corticosteroids[1]; relapse prevention is the main objective of maintenance therapy

Ph III METEOROID in MOGAD

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- Primary endpoint met: 68% reduction in risk of a new relapse vs. placebo; onset of response demonstrated as early as week 8
- Highly significant reductions in annualized relapse rate, active MRI lesions, and rescue therapy use
- Enspryng was well tolerated with a safety profile comparable to placebo
- Enspryng autoinjector 120 mg granted FDA and EMA approval, 180mg under review

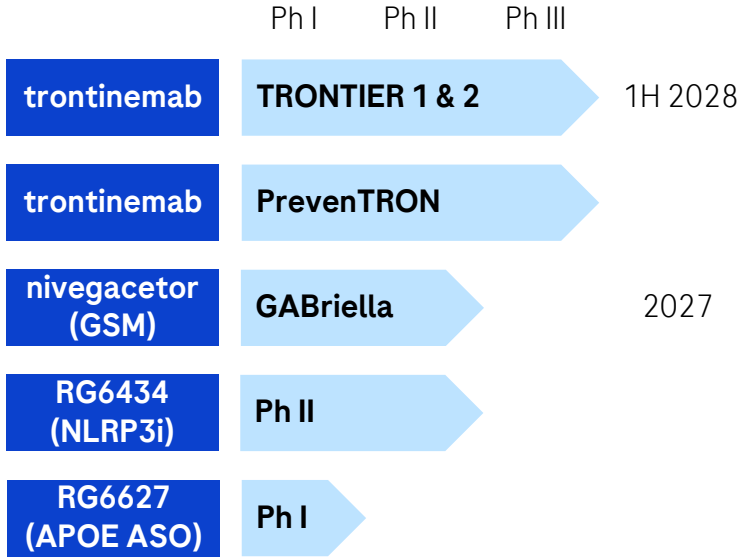
Sources: 1. Marignier R, et al. Lancet Nuerol. 2021;20:762-772; 2. Michael L, et al. AAN 2026. PL5; MOGAD: Myelin oligodendrocyte glycoprotein antibody-associated disease

Alzheimer's disease: Opportunities to unlock patient benefit

Despite approved therapies, significant unmet need remains

	Opportunities
Diagnosis	Blood tests enable early, convenient, at-scale detection, in symptomatic and presymptomatic individuals
Efficacy	Greater clinical benefit via: • Faster and more complete amyloid depletion via Brainshuttle™ • Targeting additional disease mechanisms, alone and in combination
Safety	Improved safety profile with low ARIA-E
Dosing & Administration	Induction/maintenance dosing regimen ensures rapid and robust removal of amyloid while reducing patient burden and further controlling AD pathology

Alzheimer's disease development program



- Ph III (TRONTIER 1 & 2) in early symptomatic AD achieved LPI; data expected 1H '28
- Ph III (PrevenTRON) in preclinical AD initiated

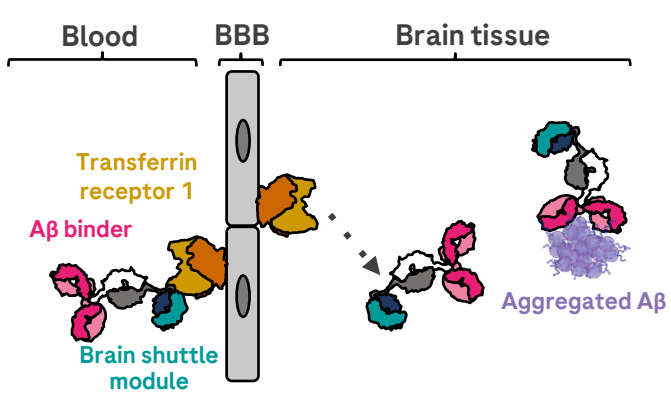
AD: Alzheimer's disease; ARIA: Amyloid-related imaging abnormalities

Trontinemab has best-in-disease potential in Alzheimer's disease

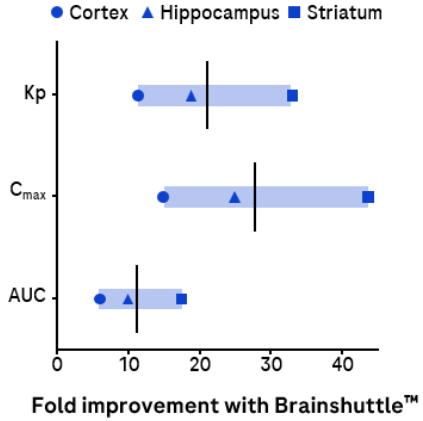
Brainshuttle™ anti-Aβ engineered to increase brain penetration and distribution

Active transport across the blood-brain-barrier enhances brain penetration

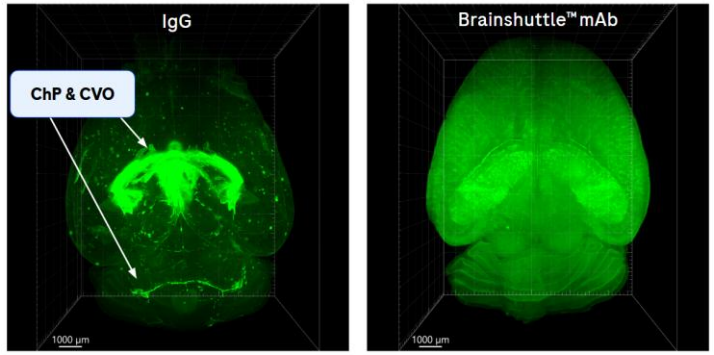
Active TfR1 transport at the capillary level



Concentration of trontinemab in the brain vs a conventional mAb[4,5]



Improved distribution vs. standard IgG mAb[2,3]



- Trontinemab is a novel bispecific 2+1 Brainshuttle™ Aβ-targeting mAb that crosses the BBB via TfR1-mediated transcytosis
- Brainshuttle™ technology enables 6- to 17- fold higher brain exposure with trontinemab observed compared with standard mAb[1], and homogeneous distribution across brain regions[2,3]
- Less engagement with vascular amyloid vs. standard mAb may lower ARIA incidence

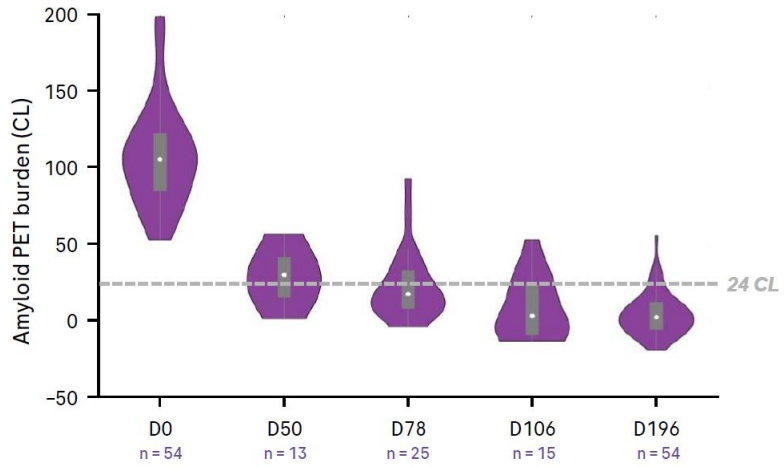
Sources: 1. Kulic L et al. Presented at AAIC 2026; 2. Data on file: Poeschinger Lab, Roche Innovation Center Munich; 3. Villaseñor R, et al. Presented at AD/PD 2026, Copenhagen, Denmark. Whole-brain images show a 3D volume rendering of the whole brain acquired with a light-sheet microscope. Images highlight the distribution of classical IgG vs Brainshuttle™ IgG, targeting neurones in mouse brains. Brains were perfused and chemically fixed 5 days after a single IV dose of fluorescently labelled Brainshuttle™ IgG (3 mg/kg) or two consecutive IV doses of IgG (2× 6 mg/kg); 4. Grimm HP, et al. MAbs 2023;15:2261509; 5. Kulic L, et al. Presented at AD/PD 2025; Aβ: Amyloid β; BBB: Blood-brain barrier; ChP: Choroid plexus; CVO: Circumventricular organs; mAb: Monoclonal antibody

Trontinemab demonstrates best-in-disease plaque removal

Converging evidence suggests the rate and amount of clearance of amyloid is important[1]

Rapid and robust amyloid plaque removal

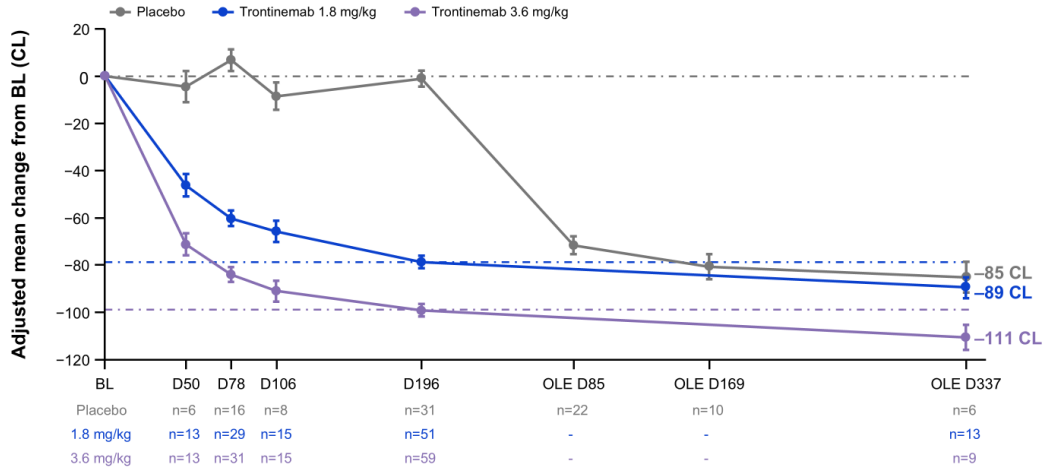
Amyloid PET trontinemab 3.6 mg/kg



- 92% of participants were below the amyloid positivity threshold (≤24 CL) at 28 weeks, and 71% were below 11 CL†
- Substantial amyloid reduction in all patients (minimum -47CL change from baseline)
- An amyloid-negative threshold of approximately 24 CL appears to be critical for clinical response

Trontinemab OLE data

PhIb/II OLE data

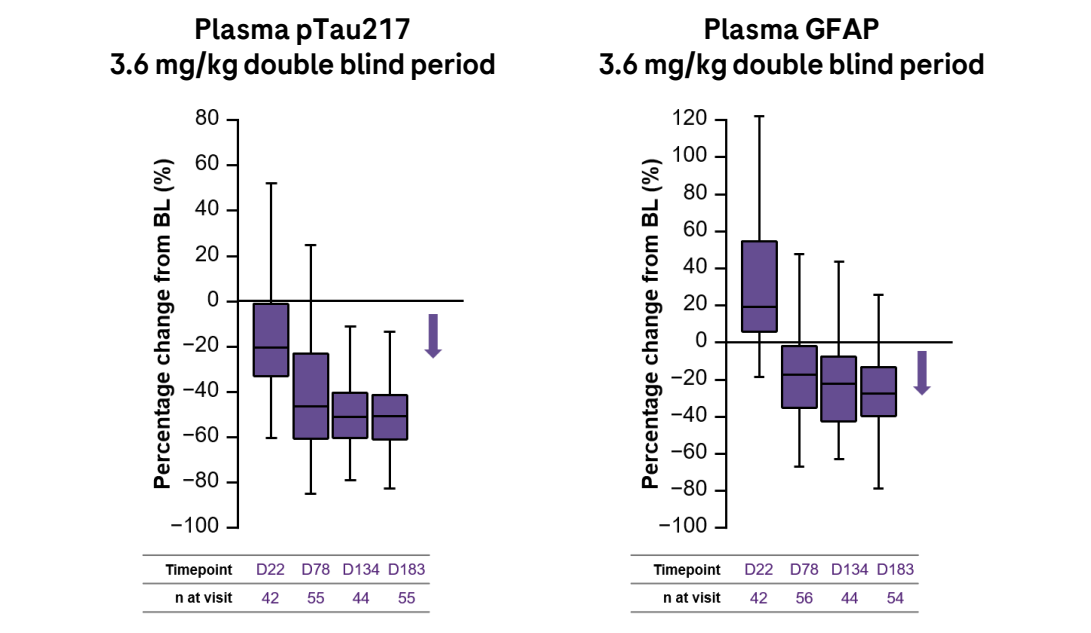


- Patients previously treated with trontinemab assigned either to Q4W or Q12W dosing based on amyloid levels at OLE baseline
- Previously treated patients demonstrated continued amyloid reduction through 1 year of OLE

†Amyloid positivity threshold defined as values ≤ 24; Sources: 1. Kulic L et al. Presented at AAIC 2026; CL: Centiloid unit; OLE: open label extension; PET: Positron emission tomography

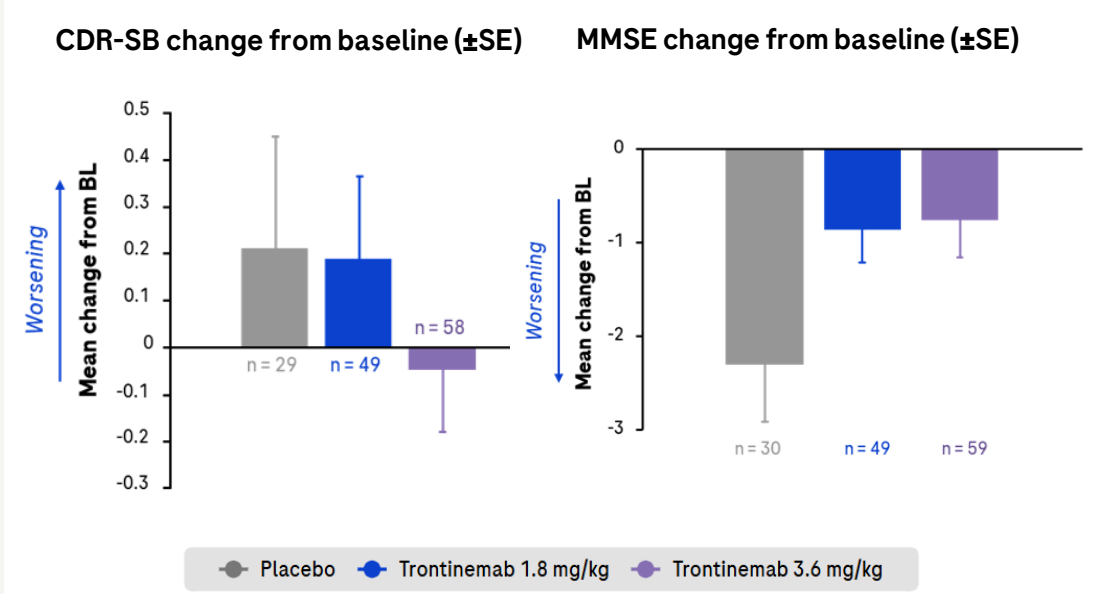
Biomarkers of AD pathology and exploratory clinical outcomes

Effects on downstream biomarkers of AD pathology



- Pronounced effects on key downstream biomarkers including CSF total Tau, CSF pTau181, plasma pTau 217, and plasma GFAP which is associated with AD pathology

Trontinemab exploratory clinical efficacy outcomes[1]



- Numerically less decline on CDR-SB and MMSE with trontinemab compared with placebo at 28 weeks
- The Brainshuttle™AD study design limits a robust quantitative evaluation of clinical scales: short duration, small sample size, heterogeneous population, 4:1 randomization active:placebo

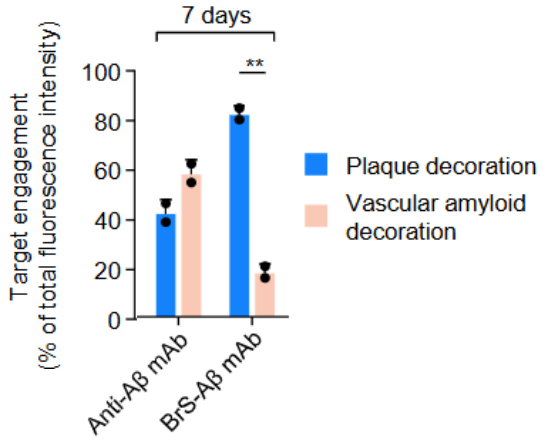
Sources: 1. Kulic L et al. Presented at CTAD 2025, Unblinded exploratory clinical efficacy data by dose levels (snapshot date: September 05, 2025). Completer analysis restricted to participants with available assessment at both BL and Day 196. Exclusion of one participant with ischemic stroke and clinical aphasia in Cohort 4, Part 1, from the analysis; AD: Alzheimer's disease; CDR-SB: Clinical dementia rating- sum of boxes; CSF: Cerebrospinal fluid; GFAP: Glial fibrillary acidic protein; MMSE: Mini-mental state examination; SE: Standard error

Trontinemab has potential for best-in-class safety with <5% ARIA-E

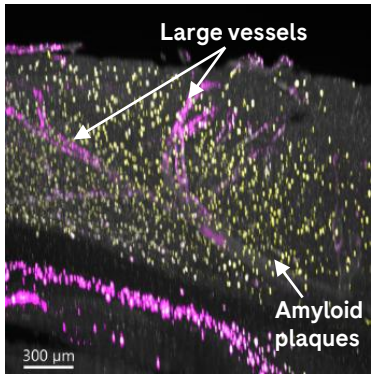
Vascular engagement of standard anti-amyloid mAbs reduced with Brainshuttle™

ARIA associated with vascular engagement

Target engagement with Brainshuttle™ anti-amyloid mAb vs standard mAb



Vascular amyloid decoration 7 days post injection



Standard mAb

BrainShuttle™ anti-amyloid mAb

- In entering via CSF and perivascular spaces, anti-Aß mAbs binding to vascular amyloid pathology may exacerbate inflammation, leading to ARIA[1,2]
- Murine surrogate of trontinemab shows enhanced plaque decoration with reduced vascular engagement compared with a standard mAb

Ph Ib/II Brainshuttle™ safety data[3]

	Part 1 + 2 (combined) (n=151)*	
Total number of participants, n (%)	**Cohort 3 1.8 mg/kg or Placebo (n=76)**	**Cohort 4 3.6 mg/kg or Placebo (n=75)**
ARIA-E†	3 (3.9)	1 (1.3)
Symptomatic ARIA-E	1 (1.3)	0 (0.0)
ARIA-H	5 (6.6)	3 (4.0)
Microhaemorrhage	2 (2.6)	2 (2.7)
Superficial siderosis	3 (3.9)	1 (1.3)
Concurrent ARIA-E + ARIA-H	0 (0.0)	0 (0.0)
Other significant MRI findings		
Cerebral macrohaemorrhage§	1 (1.3)	0 (0.0)

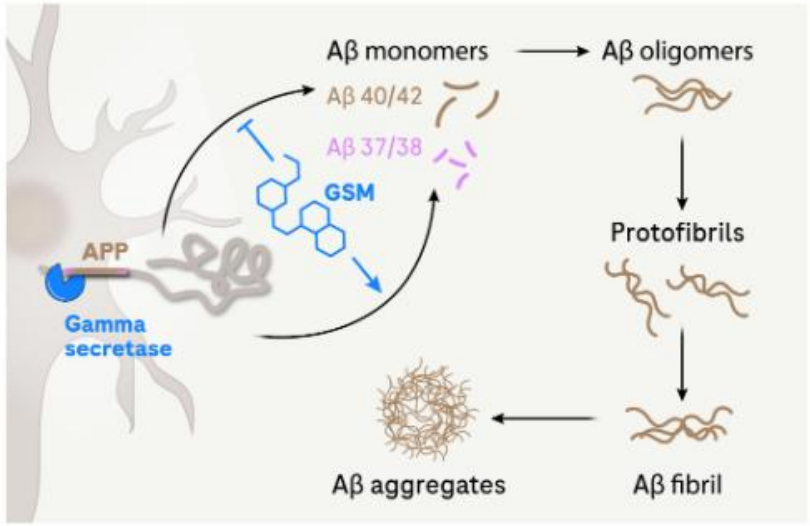
- Interim data demonstrated a consistent overall favorable safety profile with low incidence of ARIA-E (<5%)
- Anemia incidence mostly mild and transient, IRR were mild to moderate with severity and incident reduced with steroid premedication[2]

Sources: 1. Mummery C, et al. Presented at AAIC 2025, Toronto, Canada; 2. Pizzo M, et al. Science 2025;389:eads3204; 3. Kulic L et al. Presented at CTAD 2025, Unblinded exploratory clinical efficacy data by dose levels (snapshot date: September 05, 2025). Completer analysis restricted to participants with available assessment at both BL and Day 196. Exclusion of one participant with ischemic stroke and clinical aphasia in Cohort 4, Part 1, from the analysis; Aβ: Amyloid β; ARIA: Amyloid-related imaging abnormalities; CSF: Cerebral spinal fluid; IRR: Infusion-related reaction; mAb: Monoclonal antibody

Nivegacetor: Gamma-secretase modulator (GSM) in Alzheimer's

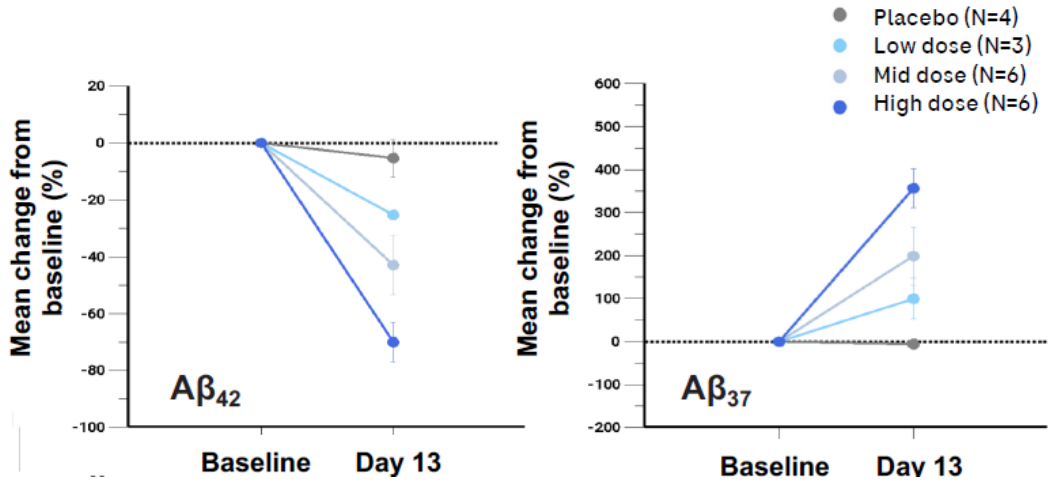
GSM offers complementary approach to target amyloid beta; ideal for intervention at earliest stages

GSM alters APP processing, inhibits Aβ aggregation



- GSMs selectively target processing of amyloid precursor protein (APP) and leave the other substrates (e.g. Notch) unchanged.
- Reduce amyloidogenic long peptides (Aβ40, Aβ42) and increase production of short, non-amyloidogenic peptides (Aβ38,Aβ37). Increased levels of shorter Aβ species reduce the risk of cognitive decline[1,2]

Ph I nivegacetor in healthy volunteers[1]



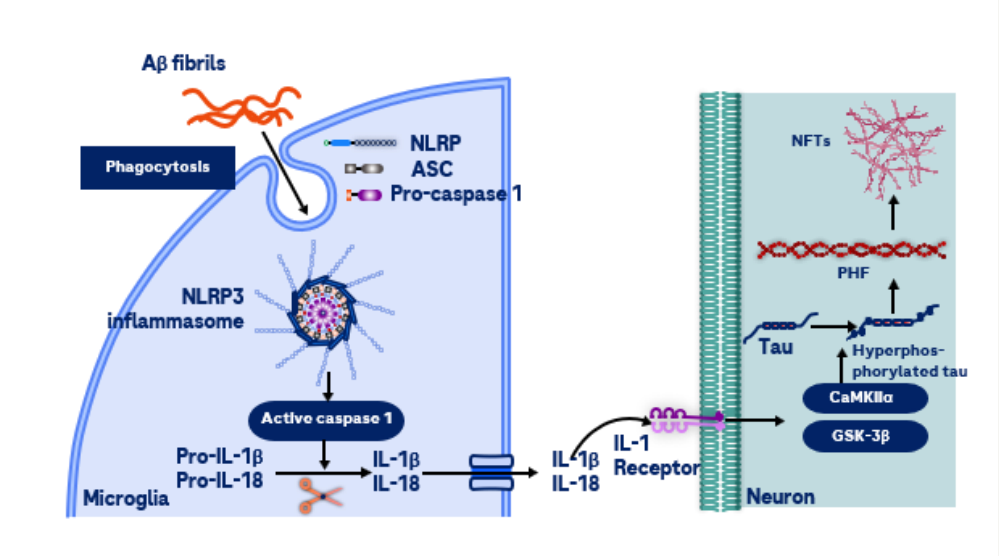
- Ph I healthy volunteers: dose-dependent γ-secretase modulation with up to~70% Aβ42 reduction and strong increase in short Aβ forms. PK profile supports oral once daily dosing
- Ph IIa GABriella in early-AD recruitment completed; data expected 2027

Sources: 1. Cullen et al., Neurology 2022; 2. Von Kienlin et al. presented at CTAD 2018, Barcelona, Spain; Aβ: Amyloid beta; APP: Amyloid precursor protein; GSM: Gamma-secretase modulator

NLRP3i in neurodegenerative disorders

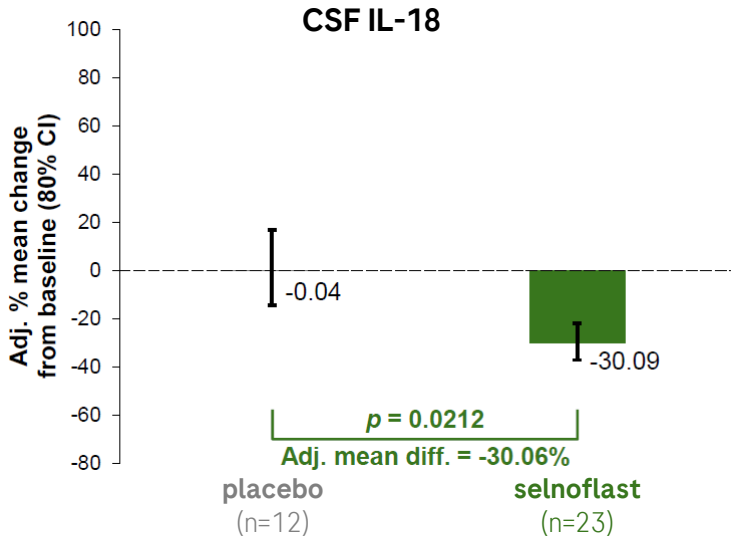
RG6434 advancing to Ph II in Alzheimer's disease and Parkinson's disease

Alzheimer's: NLRP3 links Aβ and Tau pathologies



- Amyloid fibrils activate NLRP3 in microglia; subsequent inflammatory signaling enhances tau phosphorylation[1,2]
- Targeting NLRP3 offers potential to decouple Aβ accumulation from the subsequent neurodegenerative cascade[3]
- Ph II study of RG6434 in Alzheimer's to be initiated

Parkinson's: Ph I pharmacodynamics[4]



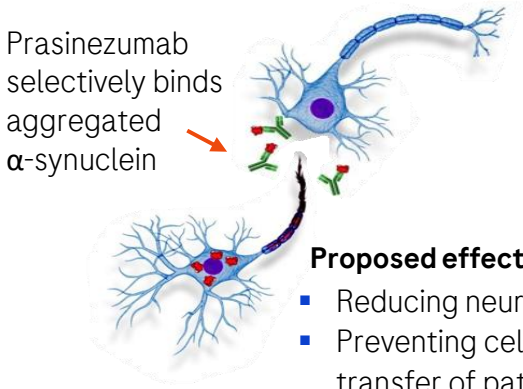
- NLRP3 inflammasome activation associated with downstream dopaminergic neuronal death[5]
- Ph Ib study of another NLRP3i (selnoflast) in early PD show proof of mechanism by downstream inflammasome biomarkers
- Potential to combine NLRP3i with orthogonal mechanisms in PD
- Ph II study of RG6434 in Parkinson's to be initiated

Sources: 1. Holbrook JA, et al. Fron Pharmacol 2021;12:643254; 2. Sutterwala FS, et al. Ann N Y Acad Sci 2014;1319:82-95; 3. Jung ES, et al. Aging Cell 2022;21:e13623; 4. Pagano G, et al. AD/PD 2026; 5. F. Hoffmann-La Roche Ltd. Data on file; Aβ, amyloid beta; AD: Alzheimer's disease; CSF: Cerebral spinal fluid; PD: Parkinson's Disease

Prasinezumab: Trend towards delaying motor progression

Signal of efficacy demonstrated beyond effective symptomatic therapy

Anti-α-synuclein mAb



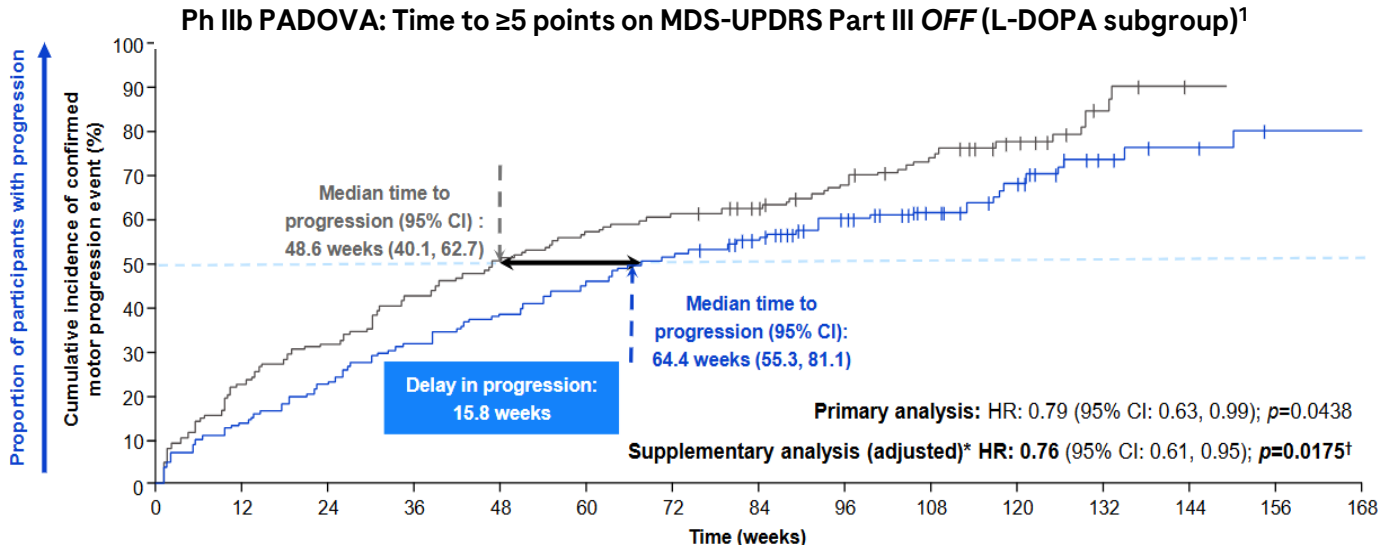
Proposed effects:

- Reducing neuronal toxicity
- Preventing cell-to-cell transfer of pathogenic α-synuclein aggregates
- Slowing disease progression

- Prasinezumab has the potential to be the first disease modifying therapy in PD
- Ph III (PARAISO) enrolling in L-DOPA patients (n=900)

Ph IIb PADOVA results

AD/PD™ 2026 ADVANCES IN SCIENCE & THERAPY



- Signal of efficacy in pre-specified L-DOPA subgroup (~74% of total study population)
- First biomarker evidence of prasinezumab impacting disease pathology: stabilization of neuromelanin and reduced iron accumulation in key brain regions
- Favorable safety profile
- Over 900 PD study participants, with more than 500 treated for 2.5 to 5 years

Prasinezumab in collaboration with Prothena; Sources: 1. Nikolcheva et al. Presented at ADPD 2026; L-DOPA: levodopa; mAb: Monoclonal antibody; MDS-UPDRS: Movement disorder society-sponsored revision of the unified Parkinson's disease rating scale; OFF: practically defined OFF state; PD: Parkinson's diseas

Immunology

Larry Tsai
SVP and Global Head of Product Development
Immunology

Immunology R&D focus areas

Critical Capabilities		Examples
Optimize pathways	*Build on known mechanisms and identify novel pathways/targets for transformational benefit*	Various approaches for enhanced B-cell depletion, TL1a pathway, and complement pathway
Combinations	*Target multiple pathways to achieve improved efficacy and deeper remission*	p40xTL1A bispecific combines orthogonal targets to raise efficacy in IBD TSLPxIL33 bispecific targets key epithelial "alarmin" cytokines that trigger airway inflammation in COPD
Endotypes	*Identify patients' subsets to improve efficacy and guide therapy*	Afimkibart IBD trials explore biomarkers to predict better response to treatment; COPD pipeline focused on patient identification on areas of high unmet need, e.g. muco-obstructive and non-Th2 disease
Cure	*Aim for curative treatment to achieve long-term remission*	Lunsumio and next-gen CD19/CD20 TCBs (in preclinical development)
E2E investment	*Invest end-to-end in select disease areas from discovery, R&D, to commercialization*	Inflammatory bowel disease (IBD) and chronic obstructive pulmonary disease (COPD)

IBD: Inflammatory bowel disease; CAR-T: Chimeric antigen receptor T cell

Immunology pipeline

Broad development portfolio across E2E and breakthrough innovation disease areas

Phase I

RG6767	**undisclosed** immunology
RG6160	**cevostamab** SLE/LN
RG6981	**IL33:TSLP** COPD/asthma
RG6421	**TMEM16A potentiator** COPD
RG6498	**undisclosed** IBD
CHU	**RAY121 (anti-C1s recycling Ab)** Immunology
RG6527	**undisclosed*** COPD
RG6644	**undisclosed*** COPD

Phase II

RG7828	**Lunsumio** SLE/LN
RG6631	**afimkibart** Rheumatoid arthritis
RG6730	**p40 x TL1A** Ulcerative colitis
CHU	**anti-HLA-DQ2.5 x gluten peptides** Celiac disease

Phase III

RG6299	**sefaxersen** IgAN
RG6631	**afimkibart** Ulcerative colitis
RG6631	**afimkibart** Crohn's disease

Registration

RG7159	**Gazyva** SLE
RG7159	**Gazyva** Membranous nephropathy
RG7159	**Gazyva** Childhood onset INS

Approved

RG7159	**Gazyva** LN
RG7159	**Gazyva** Childhood onset INS

Small molecule
Antibody
RNA-targeting therapeutics
Bispecifics

Immune-mediated kidney & rheumatological diseases
Respiratory & allergy
Gastroenterology
Others/undisclosed

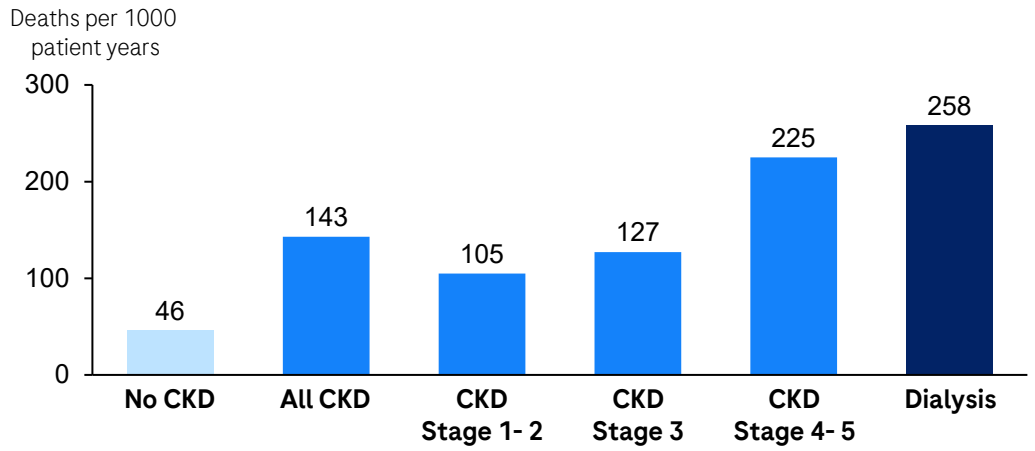
* Program to be initiated in Ph I; E2E: end to end: Ab; Antibody; COPD: Chronic obstructive pulmonary disease; IBD: Inflammatory bowel disease; IgAN: IgA nephropathy; INS: Idiopathic nephrotic syndrome; LN: Lupus nephritis; SLE: Systemic lupus erythematosus

Immune-mediated diseases are a leading cause of CKD

Aiming to prevent progression to end-stage kidney disease and subsequent need for dialysis/transplantation

CKD to be 5th leading cause of death globally by 2040[1]

Mortality significantly increases with increasing loss of renal function[2]



- Immune-mediated kidney diseases are associated with high morbidity and mortality, ranking as the 3rd most common cause of ESKD
- Up to 25% of lupus, 50% of IgAN, and 30% of MN patients develop ESKD despite treatment with current available therapies[3,4,5]

Development program in diseases driving kidney disfunction

Molecule	Indication	Ph I	Ph II	Ph III	Status
Gazyva	LN			REGENCY	✓ Approved
	SLE			ALLEGORY	✓ PDUFA Dec 4
	MN			MAJESTY	✓ PDUFA Nov 15
	INS			INShore	✓ Approved
PiaSky	aHUS			COMMUTE	Filing 2026
sefaxersen	IgAN			IMAgINATION	✓ 1 EP met*
Lunsumio	SLE/LN		SOLUNA		Ph II
RG6767	undisclosed				Ph I initiated
cevostamab	SLE/LN				Ph I initiated

✓ *Approved or primary endpoint met*

**Based on interim analysis from ph III IMAgINATION study*

1. J. Jager, C. Kovesdy, R. Langham, et al. Kidney Int, 96 (2019), pp. 1048-1050; 2. Adapted United States Renal Data System. 2024 USRDS Annual Data Report: Epidemiology of kidney disease in the United States. NIH, 2024; 3. Arthritis Rheumatol. 2023 April ; 75(4): 567–573. doi:10.1002/art.42375; 4. Fervenza et al. 2019 N Engl J Med. 2019 Jul 4;381(1):36-46; 5. Pitcher D, Long-Term Outcomes in IgA Nephropathy. Clin J Am Soc Nephrol. 2023 Jun 1;18(6):727-738. doi: 10.2215/CJN.0000000000000135 ; 6. Roche Pharma Day 2025 Appendix-Epidemiology Overview7. US Dept of Health 2020; 8. A systematic EU approach to chronic kidney disease - European Parliament Question Feb 2022; aHUS: Atypical hemolytic uremic syndrome; CKD: chronic kidney disease; ESKD: End stage kidney disease; IgAN: IgA nephropathy; INS: Idiopathic nephrotic syndrome (Childhood onset INS also known as PNS: Pediatric nephrotic syndrome); LN: Lupus nephritis; MN: Membranous nephropathy; SLE: Systemic lupus erythematosus

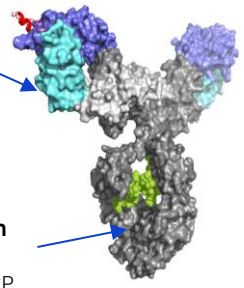
Gazyva: Deeper B-cell depletion key to improved clinical response

B cells are central to immune mediated kidney disease pathogenesis

Type II anti-CD20 mAbs binding capabilities result in superior direct cell death

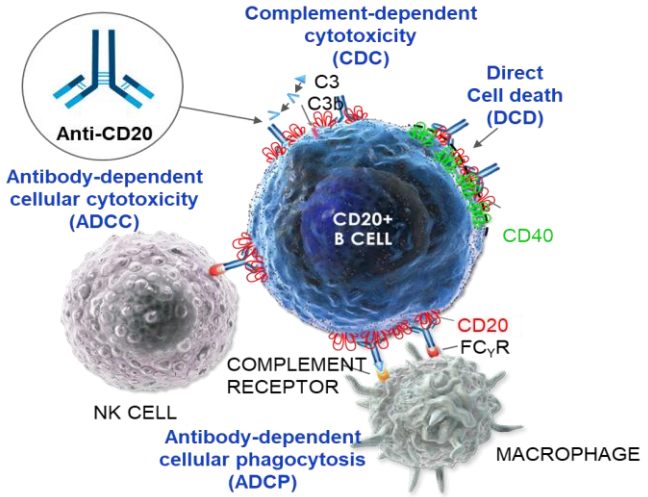
Gazyva (obinutuzumab): Type II anti-CD20 mAb

Type II anti-CD20 region
- Increased direct cell death
- Decreased CDC
- Reduced internalization



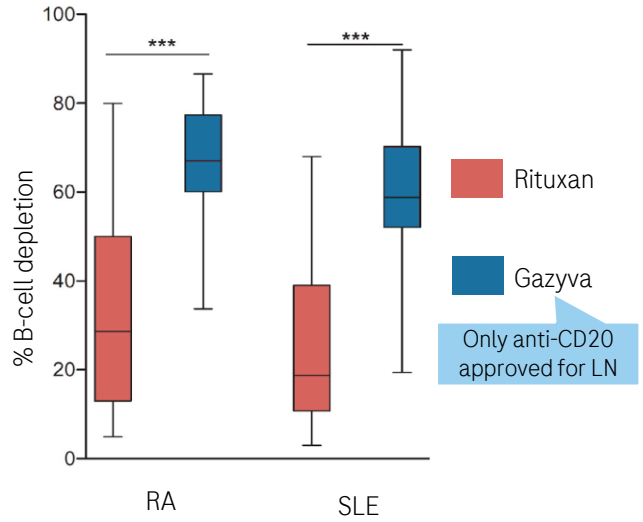
Glycoengineered Fc region
- Higher FcγR affinity
- Increased ADCC/ADCP

B cell depletion by anti-CD20 mAbs



- First glycoengineered type II aCD20 for LN, SLE, MN, INS to directly target B cells, an underlying cause of disease[1]
- Type II anti-CD20 region with increased direct cell death, decreased CDC and reduced internalization
- Glycoengineered Fc region with higher FcγR affinity and increased ADCC/ADCP[1,2]

B cell depletion vs Rituxan[3]



- Greater potency than Rituxan in depleting B-cells[3]
- Tested in multiple immune mediated kidney diseases, including disease in which earlier generation anti-CD20s failed to demonstrate benefit[4]

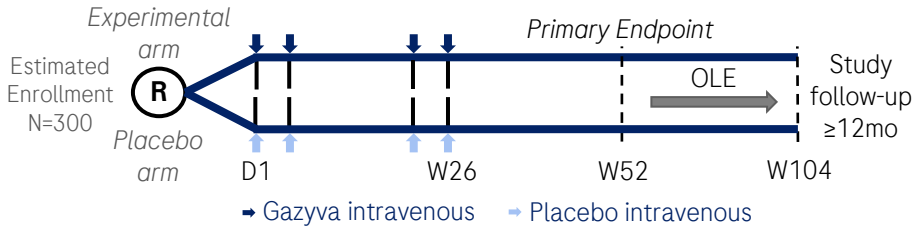
1. Rahman A and Isenberg DA. N Engl J Med. 2008;358:929-938; 2. Gomez Mendez LM, et al. Clin J Am Soc Nephrol. 2018;13:1502-1509; 3. Adapted from Reddy V, et al. Rheumatology (Oxford).2017;56:1227-1237; 4. Rovin BH, et al. WCN 2025; ADCC: antibody-dependent cell-mediated cytotoxicity; FcγR: Fc gamma receptor; ADCP: antibody-dependent cellular phagocytosis; CDC: complement-dependent cytotoxicity; DCD: direct cell death; INS: Idiopathic nephrotic syndrome (Childhood onset INS also known as PNS: Pediatric nephrotic syndrome); LN: Lupus nephritis; MN: Membranous nephropathy; RA: Rheumatoid arthritis; SLE: Systemic lupus erythematosus

Gazyva: Type II aCD20 mAb transforming standard of care in SLE

Filed in SLE in US/EU with PDUFA date Dec 4

Gazyva: First B-cell depleting agent to reduce SLE disease activity in a global Ph III study

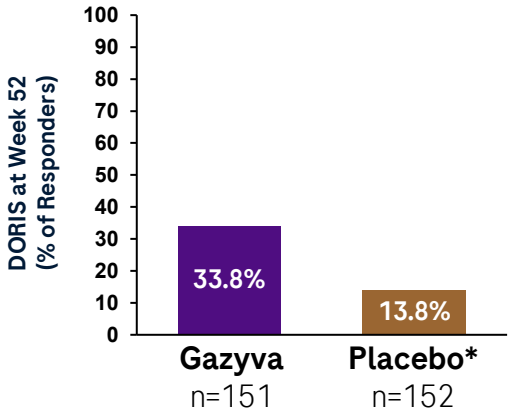
PH III ALLEGORY trial design



Primary Endpoint: % of patients achieving SRI-4 at week 52
Additional Endpoints: % of patients achieving sustained response (week 40 to week52), % of patients achieving sustained corticosteroid control

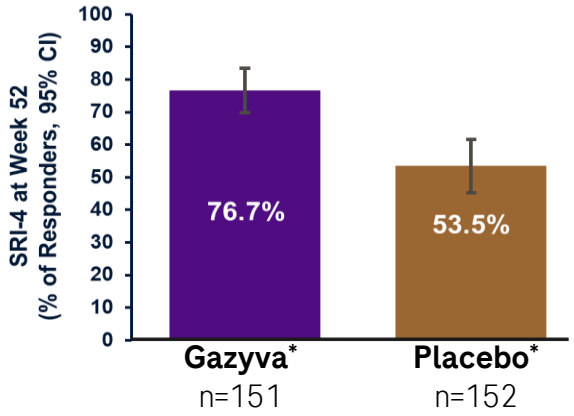
DORIS remission score: Adjusted treatment difference[1]

19.9% (95% CI, 10.6 to 29.2)



SLE disease activity: Adjusted treatment difference[1]

23.1% (95% CI, 12.5 to 33.6); P<0.001



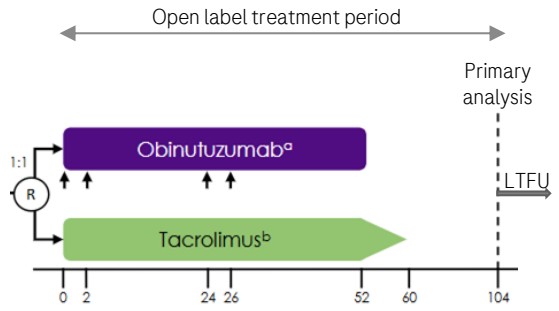
- SLE affects 3.4M people worldwide, 50% develop LN which is associated with >6-fold increased mortality
- Primary endpoint of minimum four-point improvement in SLE Responder Index 4 (SRI-4) at 52 weeks was met with a statistically significant and clinically meaningful treatment difference of 23.1%
- Additionally, Gazyva demonstrated statistically significant and robust efficacy over placebo across all secondary endpoints, including a ~20% treatment difference on the DORIS remission score
- ACR update 2025 recommends earlier introduction of conventional and/or biologic immunosuppressive therapies

Furie R et al. SLEuro 2026; ACR: American College of Rheumatology; SLE: systemic lupus erythematosus; SoC: standard of care; SRI-4: Systemic Lupus Erythematosus Responder Index 4; US: United States; W: week; *Plus standard therapy of antimalarial and/or immunosuppressant and/or prednisone (or equivalent)

Gazyva: Best in disease potential for MN and childhood onset INS

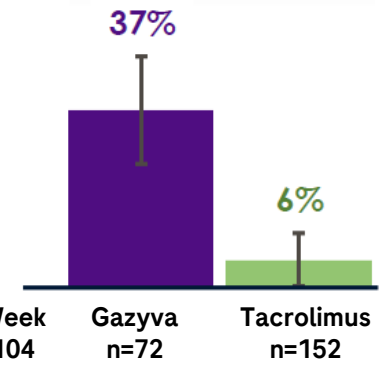
INS FDA approval achieved in Sep 2026; MN under FDA priority review with approval expected in Q4

First targeted therapy in MN to demonstrate efficacy in Ph III



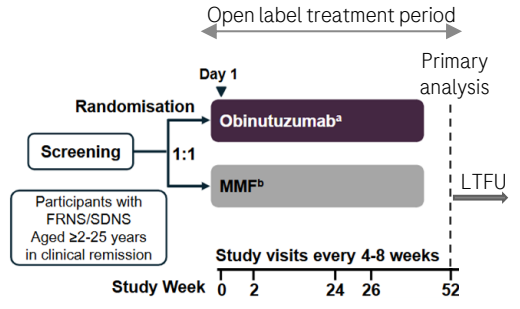
a) Obinutuzumab dose: 1,000 mg infusion
b) Tacrolimus dose: initially 0.05 mg/kg dry weight, titrated to maintain trough concentration of 5-7 ng/mL for up to 52 weeks, then tapered over 8 weeks. Participants who achieve complete proteinuric response (UPCR ≤0.3 g/g) may be considered for early taper starting at 9 months. Dose tapering must be complete by Week 60
c) LTFU, where patients will be observed and re-treated with obinutuzumab per clinical evaluation

Adjusted treatment difference 31% (95% CI, 18 to 44); P<0.001



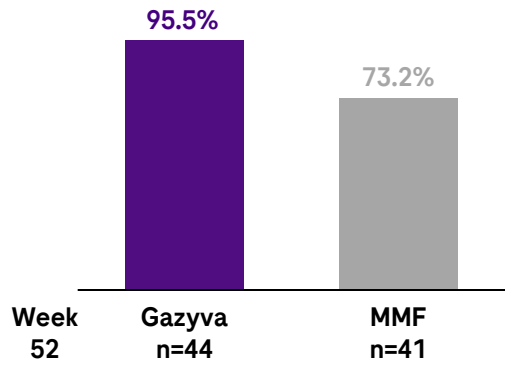
- Up to 30% of patients with MN progress to kidney failure over 10 years despite current treatment approaches[1,2]
- Primary endpoint of complete remission at week 104 met: Statistically significant and clinically meaningful treatment difference of 31.1% compared with tacrolimus
- Gazyva was superior to tacrolimus in achieving overall remission (complete or partial) at week 104 and complete remission at week 76

First approved targeted therapy in INS



a) Obinutuzumab infusion of 1000 mg or 20 mg/kg if <45 kg on Week 0 (Day 1), Week 2 (Day 15), Week 24 (Day 168) and Week 26 (Day 182)
b) MMF oral dose titrated by Week 4 to 1200 mg/m2/day is given orally twice daily until Week 52, after which administration must be discontinued or tapered over the subsequent 3 months

Adjusted treatment difference 23.36% (95% CI, 6.85 to 38.9); P=0.0031



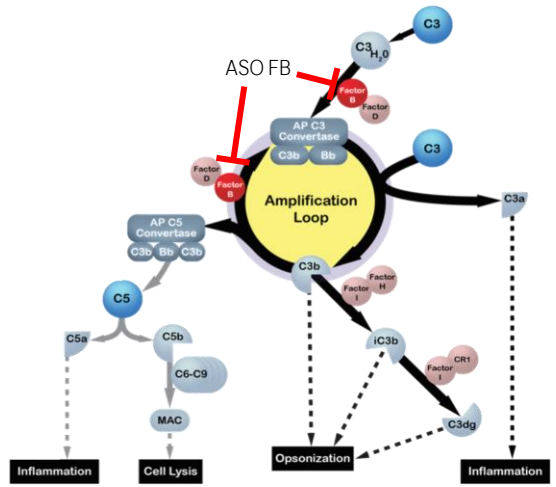
- Childhood onset INS is the most common primary kidney disease in children[3]; 70% of patients cannot successfully taper steroids[4]
- Primary endpoint met of complete remission at week 52 met : Statistically significant and clinically meaningful treatment difference of 23.4% compared with mycophenolate mofetil
- Gazyva could help children and young adults sustain remission, potentially with a reduced need for steroids

1. Keri KC, et al. Postgrad Med J. 2019;95(1119):23-31; 2. Kanigicherla DAK, et al. Nephrol Dial Transplant. 2016;31(12):2108-2114; 3. Veltkamp F, et al. Pediatrics. 2021;148(1):e2020029249; 4. Ehren R, et al. BMJ Open. 2018;8(10):e024882; 5. Complete remission is defined as achievement of UPCR ≤0.3 g/g and eGFR <15% below baseline. eGFR is calculated using the Chronic Kidney Disease Epidemiology Collaboration equation. Patients experiencing intercurrent events of escape therapy, treatment failure or early study discontinuation were considered to be non-responders under the composite strategy; BTD: Breakthrough Therapy Designation; ESRD: End-stage renal disease; FRNS: Frequently relapsing nephrotic syndrome; INS: Idiopathic nephrotic syndrome (childhood onset INS also known as PNS:Pediatric nephrotic syndrome); LTFU: Long-term follow-up; MMF: Mycophenolate mofetil; MN: Membranous nephropathy; SDNS: Steroid-dependent nephrotic syndrome

Sefaxersen: First ASO with positive Ph III interim (IMAgINATION) in IgAN

Clinically meaningful reduction in 24-hour proteinuria; BIC potential for selective complement suppression in IgAN

Sefaxersen (ASO Factor B)

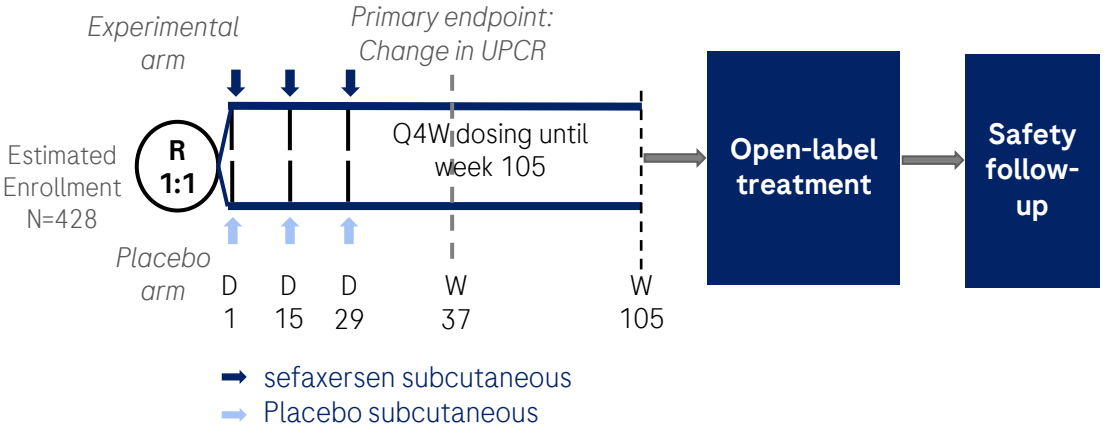


- IgAN is driven by an autoimmune response to circulating abnormal IgA, leading to complement system activation and inflammation in the kidneys
- High levels of CFB are associated with IgAN[1,2]
- Sefaxersen downregulates CFB production by inhibiting mRNA translation

Ph III (IMAgINATION) trial design

Key eligibility criteria

- Primary IgAN confirmed by kidney biopsy
- UPCR ≥1 g/g or urine protein excretion ≥1 g/day (with UPCR ≥0.8g/g) from a 24-hour collection
- eGFR ≥20ml/min/1.73m^2
- Age min. 18 years



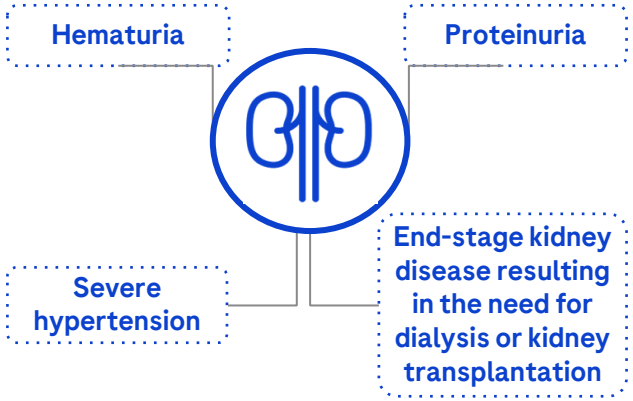
- Primary endpoint met at interim with sefaxersen achieving statistically significant and clinically meaningful improvements in proteinuria reduction vs. placebo at 37wk as measured by UPCR
- UPCR signals early response; study is continuing for long-term functional eGFR confirming slower kidney-function decline (key secondary endpoint) at week 105
- Data to be discussed with global health authorities and presented at upcoming conference

Sefaxersen in partnership with IONIS Pharmaceuticals; Figure adapted from: Barbour et al. ASN Annual Meeting 2024; 1. Chiu et al. Front Immunol. 2021; 12: 638309; 2. Yeo et al. Pediatr Nephrol 2018: 33:763-777; ASO: Antisense oligonucleotide; CFB: Complement factor B; D: Day; GN: Glomerulonephritis; IgAN: IgA nephropathy; MoA: Mechanism of action; mRNA: Messenger RNA; OLE: Open label extension; Q4W: Every 4 weeks; SC: Subcutaneous; UPCR: Urine protein/creatinine ratio; UPE: Urinary protein excretion; W: Week

IgAN: Major cause of chronic kidney disease and kidney failure

Up to 50–60% of IgAN patients remain uncontrolled even on advanced therapies

Clinical characteristics of IgAN



- IgAN is the most common primary glomerulonephritis, globally affecting ~25 adults per million every year[1,2]
- IgAN is one of the leading causes of chronic kidney disease and end-stage kidney disease in adults[3]

20-40[4]
Age of onset (in years)

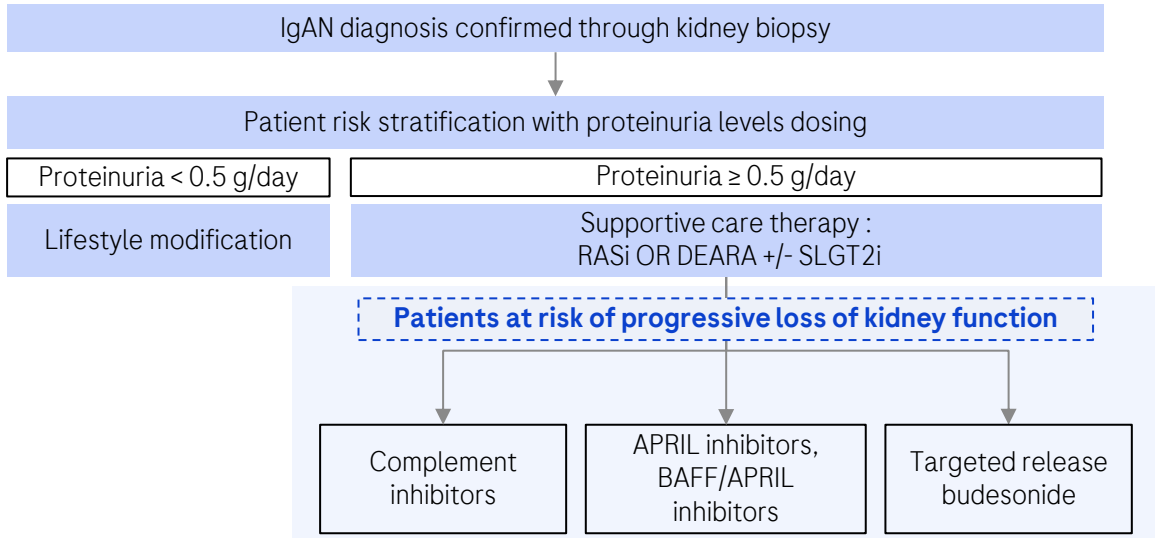
11.4 yrs[5]
Mean kidney survival after biopsy

48 yrs[6]
Mean age at kidney failure or death



Highest reported incidence in **Asia**

Current IgAN treatment paradigm[7]



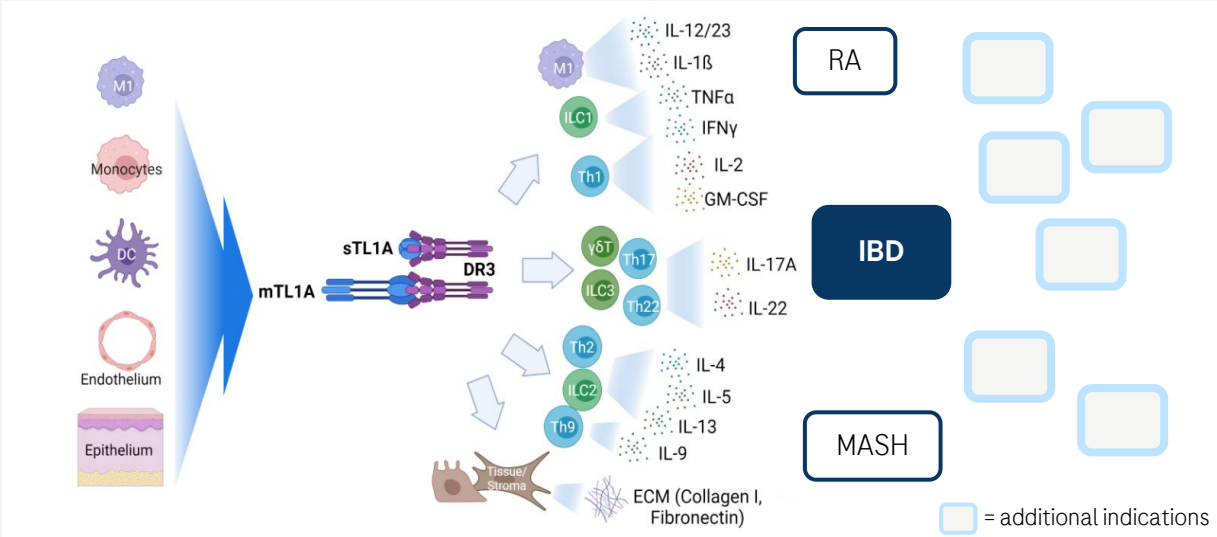
- Sefaxersen has BIC potential within complement inhibitors
- Focusing on high-risk 1L patients presenting persistent active inflammation / hematuria and 2L pts inadequately controlled by current frontline therapies
- Convenient SC monthly dosing provides potential for better patient adherence and improved QoL

1. McGrogan A, et al. Nephrol Dial Transplant. 2011;26:414–30; 2. Rodrigues JC, et al. Clin J Am Soc Nephrol. 2017;12:677–86; 3. Zaidi O, et al. BMC Nephrol. 2024;25(1):136; 4. Caster DJ, et al. Kidney Int Rep. 2023;8(9):1792-1800; 5. Pitcher D, et al. Clin J Am Soc Nephrol. 2023;18(6):727-738; 6. Jarrick S, et al. J Am Soc Nephrol. 2019;30(5):866-876; 7. Kidney International IgAN guidelines (2025); APRIL: A proliferation-inducing ligand; BAFF: B-cell activating factor; BIC: Best-in-class; DEARA: Dual endothelin and angiotensin receptor antagonist; ESKD: End-stage kidney disease; IgAN: IgA nephropathy; QoL: Quality of life; RASi: Renin-angiotensin system inhibitor; SC: Subcutaneous; SGLT2i: Sodium-glucose cotransporter-2 inhibitor

Afimkibart: Broad anti-TL1A development program

TL1A is linked to multiple immunological diseases: IBD, MASH, RA trials ongoing

TL1A is upstream of multiple immunological cytokine & cellular pathways, presenting pan-immunological potential



- TL1A/DR3 binding acts as a key amplifier of inflammatory pathways and tissue remodeling in immune-mediated diseases[1,2,3]
- TL1A- and DR3-expressing cells are known drivers of different immune-mediated and fibrotic diseases[1,2,3]
- Non-clinical and translational studies demonstrated its involvement in pathogenesis of fibrotic conditions

Clinical development program

Indication	Ph I	Ph II	Ph III	Status
Ulcerative colitis	*AMETRINE 1 & 2*			Data 2027
Crohn's disease	*SIBERITE 1 & 2*			
Atopic dermatitis	*VELARITE*			✕
Rheumatoid arthritis	*dRAvite*			Data 2027
MASH				

- Ph III UC studies completed enrolment Q3 2026
- Pediatric UC and CD registrational studies ongoing
- Ph II RA data expected in 2027
- Ph Ib FIBROLITE in MASH completed enrolment Q3 2025

1. Solitano V, et al. Med. 2024;5(5):386-400; 2. Hassan-Zahraee M, et al. Inflamm Bowel Dis. 2022;28(3):434-446; 3. Bamias G, et al. Gut. 2025;74(4):652-668; 4. Xu WD, et al. Front Immunol. 2022;13:891328; ; CD: Crohn's disease; DR3: death receptor 3; FPI: First patient in; IBD: Inflammatory bowel disease; MASH: Metabolic dysfunction-associated steatohepatitis; RA: Rheumatoid arthritis; TL1A: Tumor necrosis factor-like cytokine 1A; TNFα: Tumor necrosis factor alpha; UC: Ulcerative colitis

IBD: Chronic relapsing/remitting disease with high unmet need

Despite options targeting different pathways, ~70-80% of patients fail to achieve remission within one year

IBD pathophysiology

2 common forms

Ulcerative Colitis[1]



Continuous inflammation limited to the mucosal layer of the colon and rectum

Crohn's Disease[2]



Inflammation in patches across the GI tract, commonly the terminal ileum & colon

- Dysregulated immune response to intestinal microbiota resulting in chronic inflammation and tissue injury[3-5]
- High disease burden severely impacting quality of life

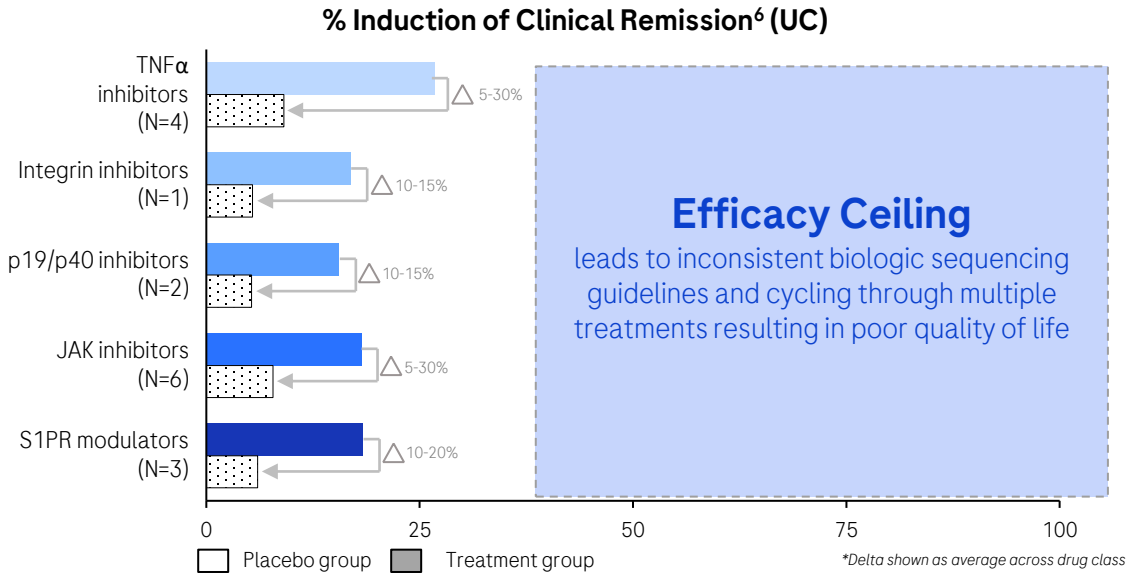
15-30
Age of onset (in years)

63%
of patients are 19-65 years old

50/50
Males and females
Highest incidence in non-Hispanic White

65%
moderate/severe, similar across countries and UC vs. CD

UC induction clinical remission rates of current therapies



Efficacy Ceiling
leads to inconsistent biologic sequencing guidelines and cycling through multiple treatments resulting in poor quality of life

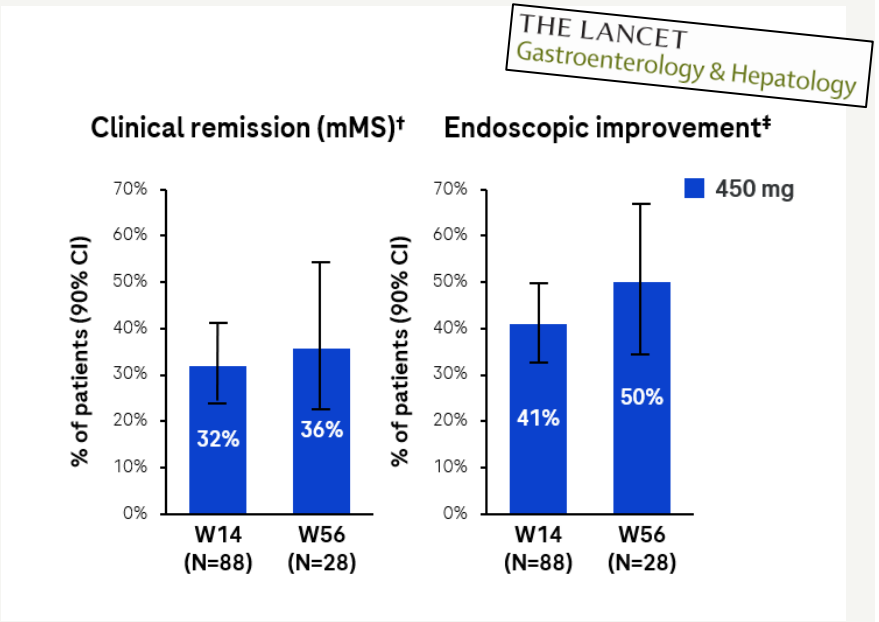
- All therapies exhibit an efficacy ceiling; initial response limited to 20-50%
- 20-30% of responders achieve remission within 1 year, durable remission is a long-term goal
- TL1a emerging as a novel MoA and treatment option
- Development combining multiple pathways, including TL1a, underway

1. CDC. https://www.cdc.gov/inflammatory-bowel-disease/about/ulcerative-colitis-basics.html; 2. CDC. https://www.cdc.gov/inflammatory-bowel-disease/about/crohns-disease-basics.html; 3. Ungaro R, et al. Lancet. 2017;389:1756-1770; 4. Feuerstein JD, et al. Mayo Clin Proc. 2014;89:1553-1563; 5. Storch I, et al. Inflamm Bowel Dis. 2003;9:104-115; 6. Adapted from Alsoud et al., 2021 [unable to verify full citation details, requires author input]; CD: Crohn's disease; CDC: Centers for Disease Control and Prevention; GI: Gastrointestinal; IBD: Inflammatory bowel disease; JAK: Janus kinase; N: Number; S1PR: Sphingosine-1-phosphate receptor; SoC: Standard of care; TNFα: Tumor necrosis factor alpha; UC: Ulcerative colitis; *Delta shown as average across drug class

Afimkibart in UC: AMETRINE-1&2 enrollment complete

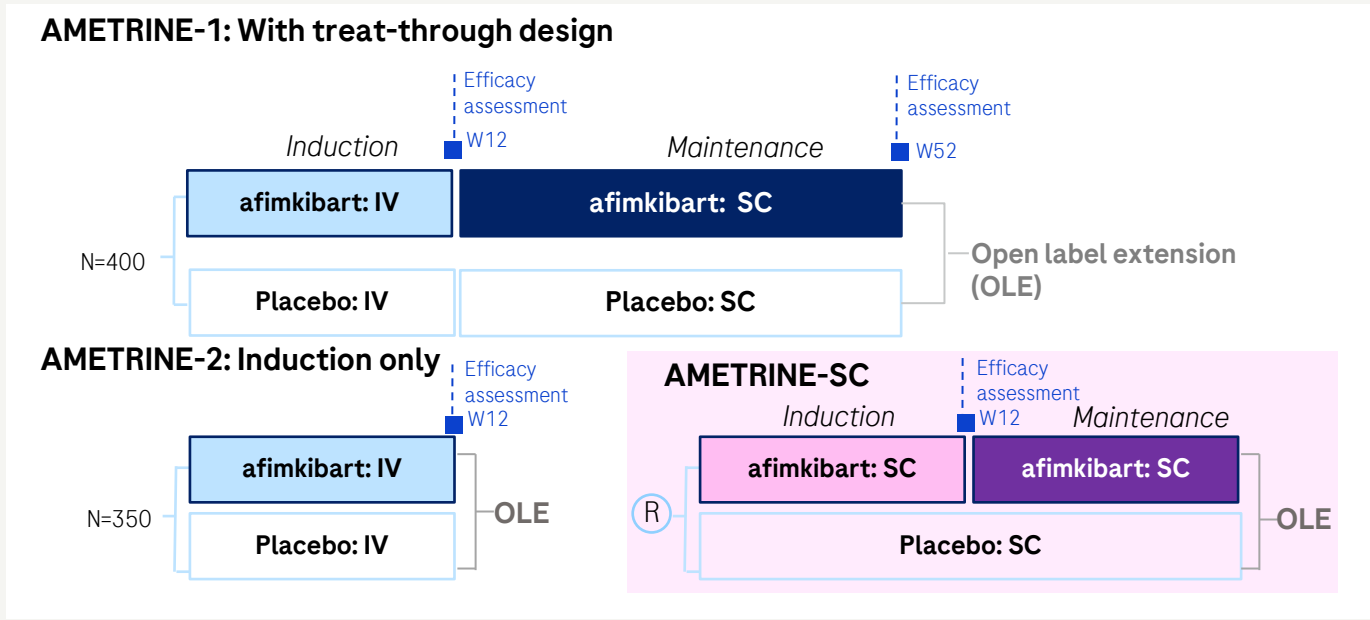
Potential for subcutaneous administration in induction and maintenance

Ph IIb (TUSCANY-2) in UC[1]



- Ph IIb (TUSCANY-2) in UC demonstrated strong efficacy and safety in a large group of pts (n=245)
- Sustained clinical remission and endoscopic improvement from induction to chronic phase

Ph III AMETRINE-1,2 and new AMETRINE–SC study design in UC



- Ph III (AMETRINE-1&2) completed enrolment Q3 2026 ; Ph III results expected 2027
- Ph III UC SC study to assess the efficacy and safety of subcutaneous induction therapy to be initiated 2026
- Exploring biomarker test which may predict better response to treatment

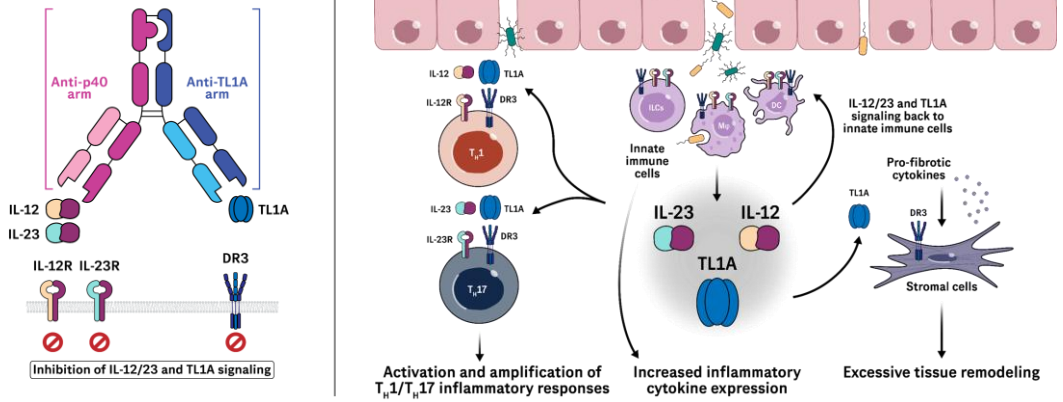
1. Danese S, et al. Lancet Gastroenterol Hepatol. 2025; ISSN 2468-1253; †Defined per FDA definition with an mMS 0–2 (endoscopic subscore = 0 or 1, and rectal bleeding subscore = 0); ‡Defined as endoscopic subscore = 0 or 1;
aTL1A: Anti-tumor necrosis factor-like cytokine 1A; CD: Crohn's disease; CI: Confidence interval; FDA: Food and Drug Administration; FPI: First-patient-in; IV: Intravenous; MASH: Metabolic dysfunction-associated steatohepatitis;
mMS: modified Mayo score; OLE: Open-label extension; Ph: Phase; pts: atients; Q: Quarter; R: Randomization; SC: Subcutaneous; UC: Ulcerative colitis; W: week

Leveraging our multispecifics pipeline for success in IBD

Establishing IBD portfolio with afimkibart, complemented by novel MoAs

p40xTL1A inhibits two key targets central to IBD pathology: IL-12/ IL-23 and TL1A

p40xTL1A bispecific*[1,2]



- IL-12 and IL-23 are proinflammatory cytokines containing the p40 subunit, promote intestinal inflammation, and are strongly associated with IBD pathology[3-7]
- The TL1A cytokine is an amplifier of immune responses that plays a key role in chronic inflammation and tissue damage
- Due to the diverse mechanisms driving IBD, simultaneously inhibiting multiple targets may overcome the therapeutic efficacy ceiling[8-10]

IBD pipeline

Molecule	Pre-clinical	Ph I	Ph II	Ph III	Status
p40xTL1A bispecific	SUNCREST				2027
Bispecific					
Emerging combinatorial					

- Ph IIb (SUNCREST) in UC ongoing, data expected in 2027
- Other multispecific antibodies and oral therapies in development, targeting validated and novel driver pathways in IBD

*Global collaboration with Pfizer; 1. Solin KA, et al. J Crohns Colitis. 2021;15(11):1959-1973; 2. Schmitt H, et al. Gut. 2019;68(5):814-828; 3. Valatas V, et al. Front Immunol. 2019;10:583; 4. Strober W, Fuss IJ. Gastroenterology. 2011;140(6):1756-1767; 5. Li L, et al. Arch Dermatol Res. 2014;306(10):927-932; 6. Verstockt B, et al. Nat Rev Gastroenterol Hepatol. 2023;20(7):433-446; 7. Xu WD, et al. Front Immunol. 2022;13:891328; 8. Bamias G, et al. J Immunol. 2003;171(9):4868-4874; 9. ClinicalTrials.gov Identifier: NCT06536440; 10. ClinicalTrials.gov Identifier: NCT06979336; FPI: First patient in; IBD: Inflammatory bowel disease; IL: Interleukin; MOA: Mechanism of action; p40: Interleukin-12/23 subunit p40; Ph: Phase; TH: T helper; TL1A: Tumor necrosis factor-like ligand 1A; UC: Ulcerative colitis

Ophthalmology

Christopher Brittain
SVP and Global Head of Product Development
Ophthalmology

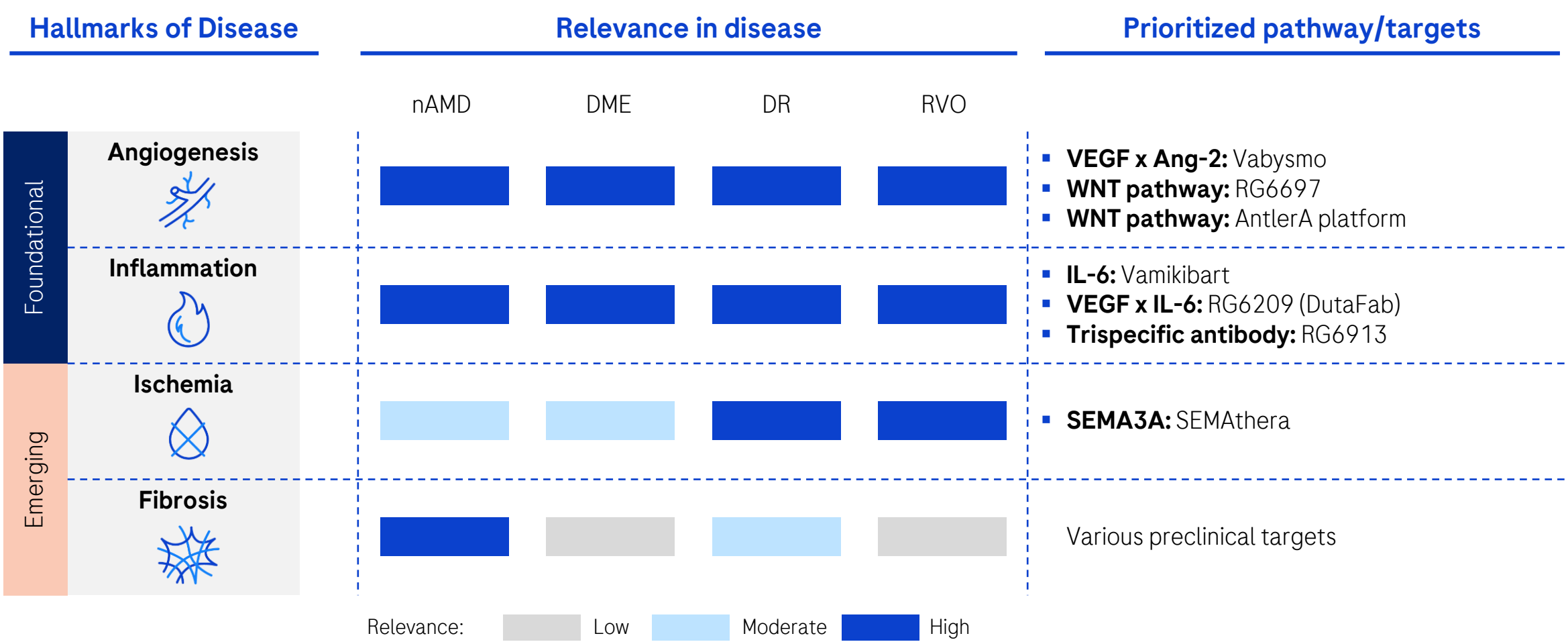
Ophthalmology R&D focus areas

Critical Capabilities	Examples
Novel MoAs & new indications, addressing multiple disease pathways	New MoAs to target a broader range of disease pathways and address additional indications
Extended durability & future technologies	Susvimo VELODROME trial exploring 9-month dosing expected in 2027
Digital capabilities	Expanded capabilities that apply biomarkers and data analytics, remote vision monitoring, and AI-supported clinical decision-making
E2E investment	Invest end-to-end in retinal vascular disease (RVD), geographic atrophy (GA) and iAMD from discovery, R&D, to commercialization

MoA: Mechanism of action; iAMD: intermediate age-related macular degeneration

Hallmarks of retinal disease and prioritized pathways/targets

Investing in alternative pathways in pursuit of better outcomes beyond anti-VEGF alone

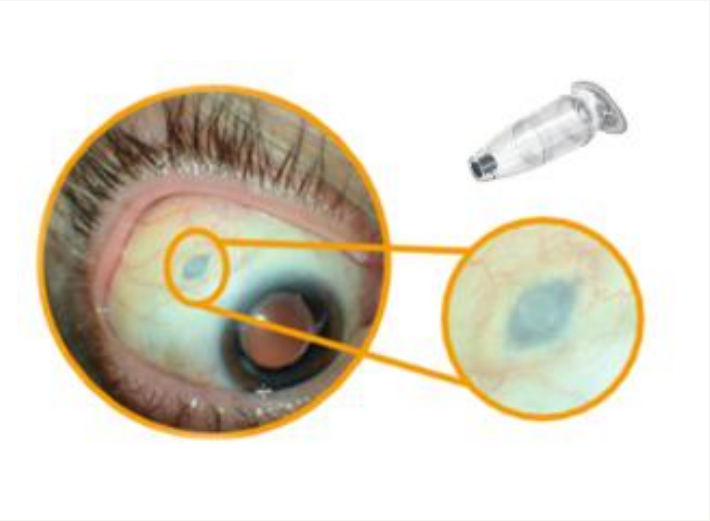


DME: Diabetic macular edema; DR: Diabetic retinopathy; nAMD: neovascular age-related macular degeneration; RVO: Retinal vein occlusion

Port Delivery Platform (PDP)

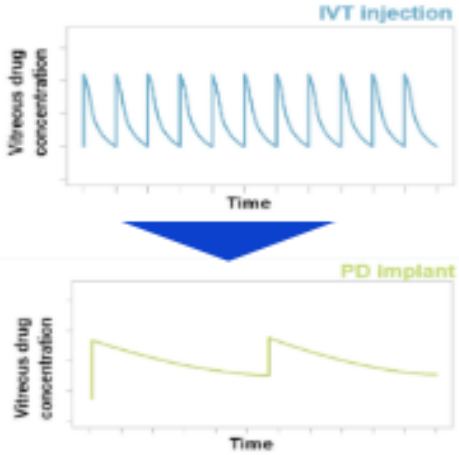
Addressing key challenges in ophthalmology, such as the need for frequent IVT injections

Innovative platforms



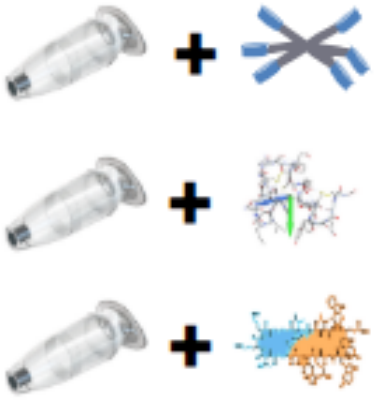
- Unique platform; first and only approved surgically-implanted long-acting treatment for RVD

Dosing confidence



- Unparalleled durability for almost all patients, with dosing confidence via continuous delivery
- Sustained, optimal drug levels in tissue over time with potential to lower safety risks
- Susvimo 9 mo dosing data (VELODROME) expected in 2027

Potential for new assets



- Optionality for future assets, customizing novel molecules
- Open for external innovation

IVT: Intravitreal; PDP: Port delivery platform: RVD: Retinal vascular disease

Ophthalmology pipeline

VEGFxIL6 DutaFab moving to Ph III; Susvimo approved in EU for nAMD

Phase I		Phase II		Phase III		Registration	
RG6327	**Undisclosed** GA	RG6501	**OpRegen® cell therapy**[1] GA	RG6321	**Susvimo** nAMD (36-week)	RG6168	**Enspryng** TED
RG6441	**Undisclosed** DR	RG6858	**CFB siRNA**[2] GA	RG7716	**Vabysmo** mCNV	RG6179	**vamikibart** UME
RG6697	**Undisclosed** DME			RG6209	**VEGFxIL6 DutaFab** DME		
RG6913	**Undisclosed** Retinal diseases						

Antibody
RNA-targeting therapeutics
Bispecifics
Cell therapy
Port Delivery Platform
DutaFab

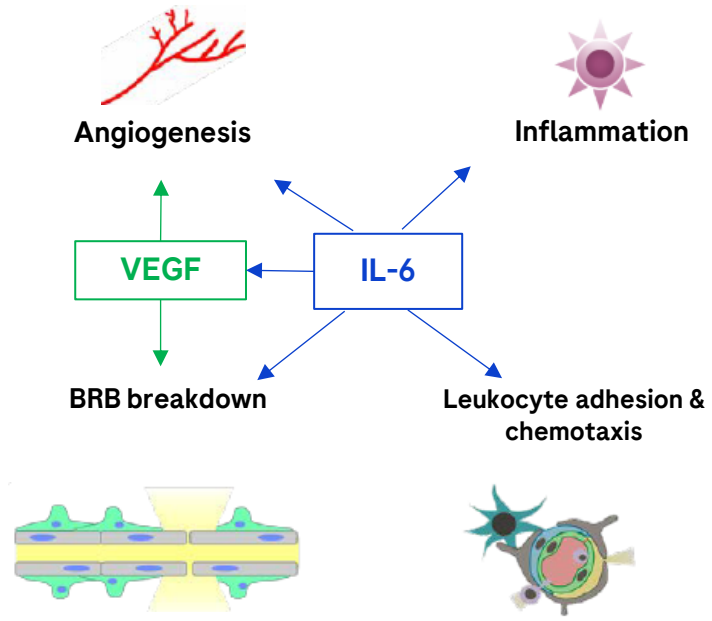
nAMD
DME/UME
GA
DR
CNV
TED

1. In collaboration with Lineage Cell Therapeutics (LCTX) 2. Licensed from SanegeneBio; CFB: Complement Factor B; CNV: Corneal neovascularization; DME: Diabetic macular edema; DR: diabetic retinopathy; DutaFab: Dual targeting fragment antigen-binding; GA: Geographic atrophy; nAMD: Neovascular age-related macular degeneration; NME: New molecular entity; TED: Thyroid eye disease; UME: Uveitic macular edema

Targeting IL-6 to achieve better visual outcomes

Enspryng filed in TED, vamikibart filed in UME, VEGFxIL6 DutaFab advancing to Ph III in DME

Inflammation is a currently sub-optimally treated pathway in a number of ocular diseases



- Il-6 is a key pro-inflammatory cytokine in the pathogenesis of uveitis, retinal diseases, and thyroid eye disease

IL-6 targeted development

Molecule	Indication	Ph I	Ph II	Ph III	Status
Enspryng	TED			SatraGO-1 & 2	PDUFA Oct 15
Vamikibart	UME			SANDCAT/MEERKAT	Filed in US/EU
VEGFxIL6 DutaFab	DME				Ph III to be initiated
Trispecific					

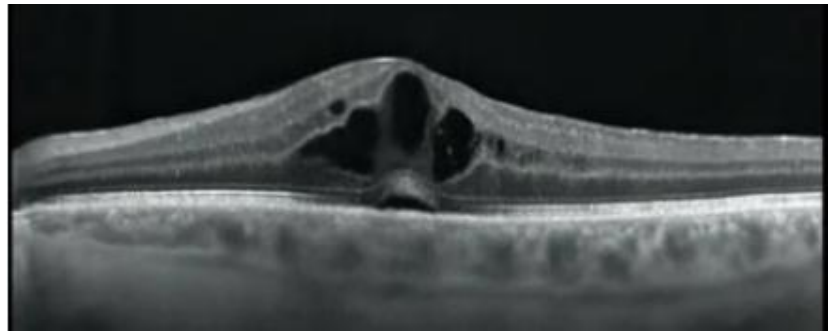
- Enspryng in TED US filing accepted with Priority Review (PDUFA Oct 15)
- Vamikibart in UME filed in the US and EU; Ph III (SANDCAT/MEERKAT) data presented at ASRS 2026
- VEGF x IL6 DutaFab advancing to Ph III
- Trispecific antibody in development

DME: Diabetic macular edema; DutaFab: Dual targeting fragment antigen-binding; IL-6: Interleukin-6; TED: Thyroid eye disease; UME: Uveitic macular edema; VEGF: Vascular endothelial growth factor

Vamikibart: Potential first non-steroid IVT treatment in UME

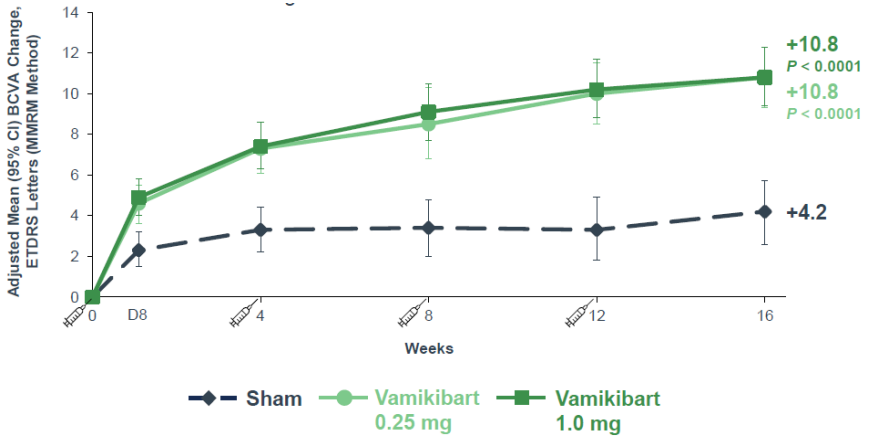
Filed in US and EU in Q3

High unmet need in UME



- UME is the most frequent cause of vision loss in uveitis[1], affecting around one-third of patients, primarily people of working age[2]
- Despite immunomodulatory therapies, UME persists in 40% of eyes[3,4] and corticosteroids, the mainstay of treatment, are associated with undesirable ocular and systemic side effects[5,6]
- Intraocular, periocular, and systemic corticosteroids are the mainstay of treatment, but have recognized side effects and efficacy limitations

Ph III (SANDCAT/MEERKAT) in UME[7]



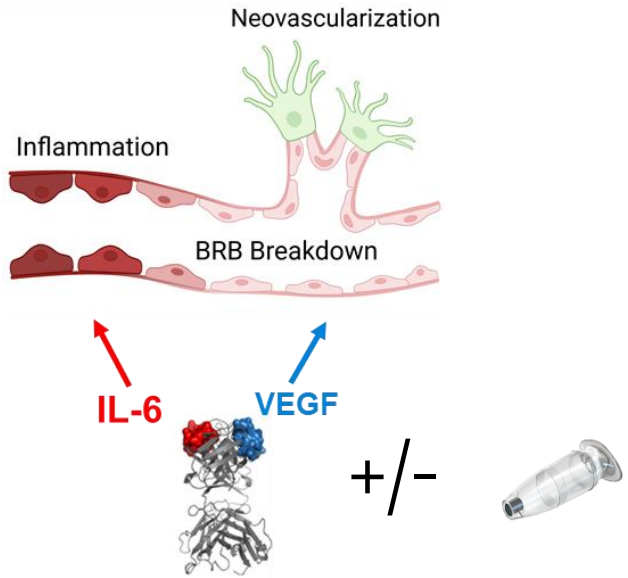
- Rapid improvements in vision and anatomical outcomes that were highly consistent across dose arms
- Well tolerated, with low incidence of treatment-related events of IOI, raised IOP, and cataract
- 52-week data to be presented at AAO

1. Massa H et al. Clin Ophthalmol. 2019;13:1761-1777. 2. Lardenoye CWTA et al. Ophthalmology. 2006;113:1446-1449. 3. Kempen JH et al. Ophthalmology. 2011;118:1916-1926. 4. Tomkins-Netzer O et al. Ophthalmology. 2015;122:2351-2359. 5. Pleyer U et al. Ophthalmol Ther. 2013;2:55-72. 6. Jobling AI and Augusteyn RC. Clin Exp Optom. 2002;85:61-75; 7. Glenn J Jaffe et al. Presented at ASRS; Jul 15-18; IOI: Intraocular inflammation; IOP: intraocular pressure; IVT: Intravitreal; UME: Uveitic macular edima;

VEGFxIL6 DutaFab: Two Ph III in DME H2H vs. Vabysmo to be initiated

Ph II with proof of concept for combining IL6 and VEGF pathways

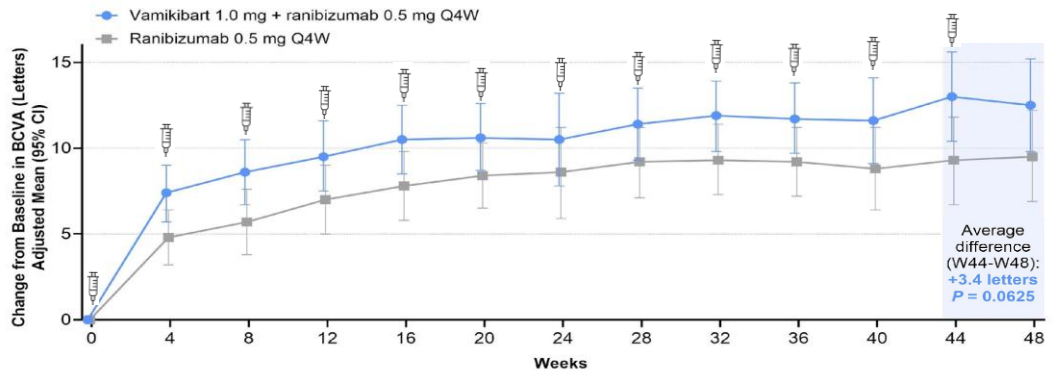
VEGFxIL6 DutaFab: Next generation bispecific



- Intended to inhibit angiogenesis, vascular permeability and inflammation by binding and blocking VEGF and IL-6
- Compatible with Port Delivery Platform

Ph II data of anti-IL-6 + anti-VEGF shows superior efficacy to VEGF monotherapy in DME*

Ph II (BARDENAS): Change from baseline in BCVA for treatment-naïve patients[1]



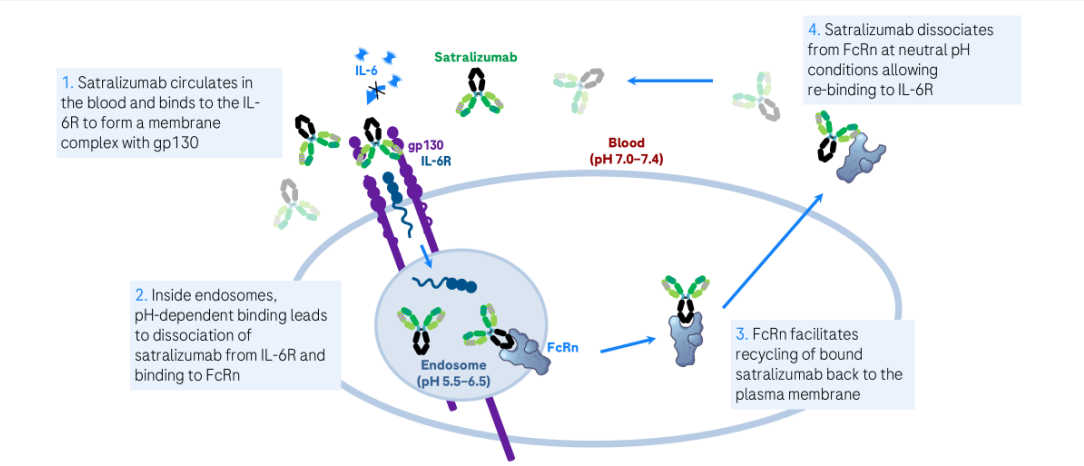
- In a treatment-naïve population, results show 44.7% of patients receiving anti-IL-6 + anti-VEGF gained ≥15 BCVA letters vs. 28.6% with anti-VEGF alone

*Primary endpoint: Change from baseline in BCVA in the BARDENAS treatment-naïve population; Sources: 1. Goldberg et al. ARVO 2026; BCVA: Best-corrected visual acuity; DME: Diabetic macular edema; DutaFab: Dual targeting fragment antigen-binding; IL-6: Interleukin-6; VEGF: Vascular endothelial growth factor

Enspryng in TED: Subcutaneous delivery with at-home autoinjector

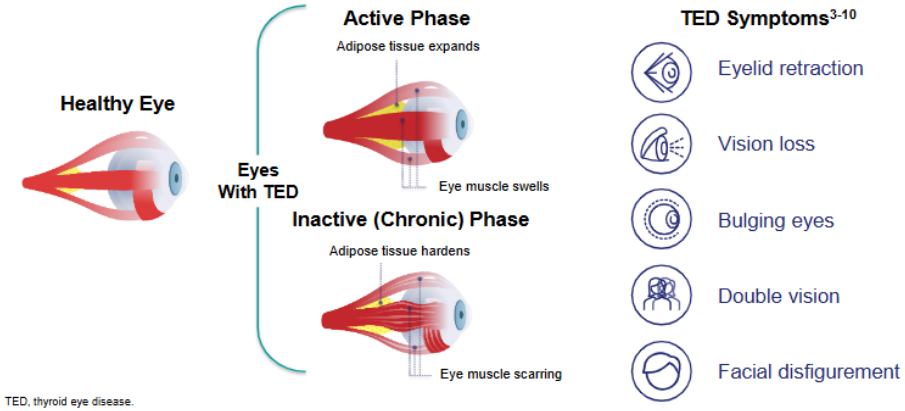
US filing accepted with Priority Review (PDUFA Oct 15)

Enspryng is designed to enable maximal sustained IL-6 suppression and allow practical SC dosing



- Anti-IL-6 designed using novel antibody recycling technology, enabling extended subcutaneous delivery (autoinjector approved)
- Inflammation is central in the pathogenesis of TED (serum IL-6 elevated in patients with TED, and correlated with inflammatory disease activity
- Enspryng is approved for NMOSD treatment, with a well-established and highly favorable safety profile in >10,000 patients for up to 7 years

High unmet need in TED for disease modifying therapies that minimize long term complications



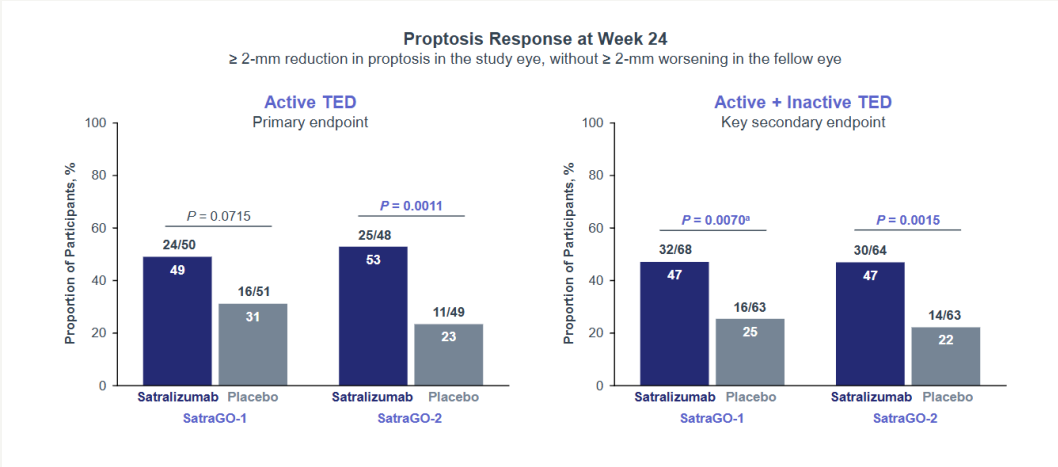
- Thyroid Eye Disease (TED) is a complex inflammatory autoimmune disease that causes facial disfigurement and visual disabilities impacting QoL, psychological wellbeing, and social interactions
- 80% of TED diagnoses are in women, with the median age of diagnosis being 43 (prevalence: 155/100,000)[1]
- Current treatments are limited and associated with serious AEs such as muscle spasms, alopecia, hyperglycemia, menstrual disorders, hearing loss, and teratogenicity

1 Bartalena L, et al. Epidemiology, Natural History, Risk Factors, and Prevention of Graves' Orbitopathy. Front Endocrinol. 2020;11:615993; AE: Adverse event; IL-6: Interleukin-6; IL-6R: Interlukin-6 receptor; NMOSD: Neuromyelitis optica spectrum disorder; QoL: Quality of life; SC: Subcutaneous; TED: Thyroid eye disease

Enspryng was efficacious and well tolerated in TED patients

Ph III SatraGo-1 and SatraGO-2 results published in Ophthalmology

Ph III SatraGO-1 and SatraGO-2[1]



- Clinical benefits observed in both active and inactive TED population
- Showed clinically meaningful improvements across key efficacy endpoints, including proptosis, diplopia, and CAS in active TED

Safety profile was highly favorable and distinct from other treatments

Most common adverse events: active + inactive TED

Participants Experiencing AEs Through Week 24, n (%)	SatraGO-1		SatraGO-2	
	Satralizumab n = 67	Placebo n = 62	Satralizumab n = 64	Placebo n = 62
Nasopharyngitis	7 (10.4)	4 (6.5)	4 (6.2)	7 (11.3)
Headache	6 (9.0)	5 (8.1)	6 (9.4)	7 (11.3)
Influenza	4 (6.0)	4 (6.5)	1 (1.6)	4 (6.5)

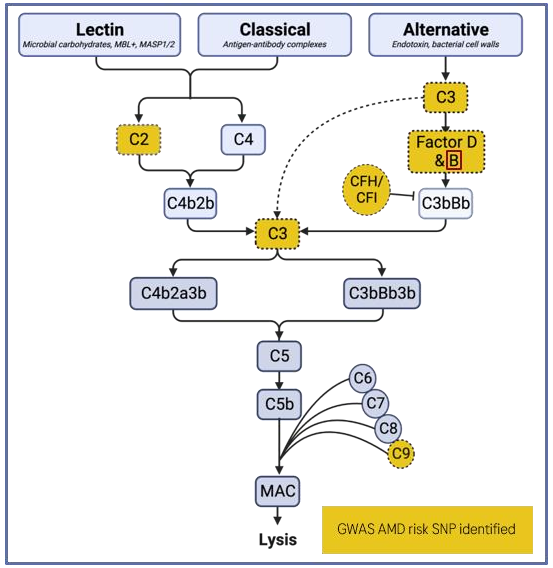
- There was a low incidence of serious adverse events and treatment discontinuation, and no serious infections
- No events of hearing loss, hyperglycemia and low rate (<3%) of neutropenia

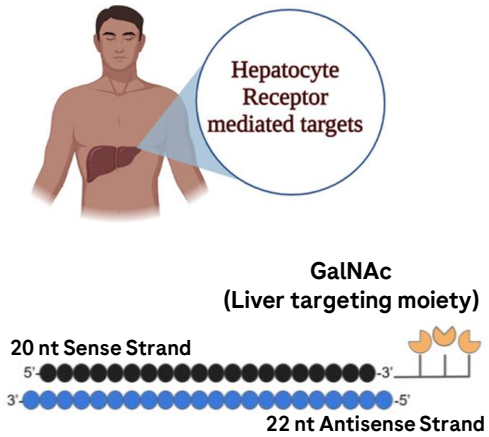
1. Sources: Subramanian P, et al. NASAOS 2026; Nominal *P* value, not adjusted for multiplicity; AE: Adverse event; CAS: Clinical activity score; TED: Thyroid eye disease

RG6858 (Complement Factor B siRNA) advancing to Ph II in GA

Systemic therapy with extended durability for Geographic Atrophy

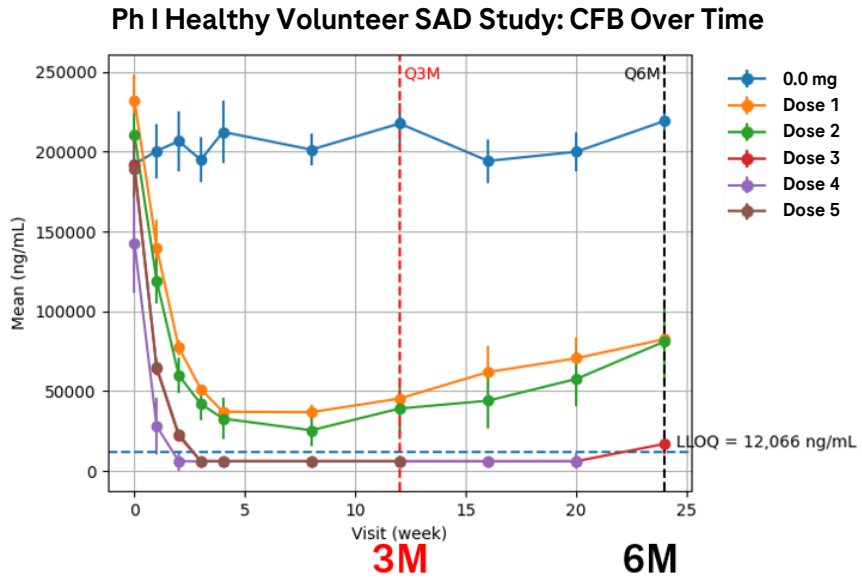
CFB siRNA with GalNAc liver targeting





- On-market complement inhibitors in GA require frequent IVT injections[1]
- CFB is produced primarily in the liver, with minimal expression in the eye; GalNAc conjugation in RG6858 enables targeted hepatocyte delivery for systemic CFB KD[2]

RG6858 demonstrates superior CFB inhibition[3]



- For Dose levels 3 and above, RG6858 achieved near 100% inhibition of CFB and AP activity at 3 mo follow-up
- Prior experience from the Ph II GOLDEN study, with CFB-ASO showed ~70% CFB knockdown and ~35% AP activity inhibition, resulting in small, but non-stat sig effect on GA lesion growth[4]

RG6858 in partnership with SanegeneBio; Sources: 1. GlobalData 2024 – Geographic Atrophy and Intermediate AMD Market & Epidemiology Reports; 2. Orozco et al; AMD; Age-related macular degeneration; 3. Data on file; 4. Jaffe et. Al AAO 2026; AP: Alternative pathway; ASO: Antisense oligonucleotide; CFB-ASO: Complement Factor B; GA: Geographic atrophy; KD: Knockdown; SAD: Single ascending dose

Cardiovascular, Renal and Metabolism

Manu Chakravarthy
SVP and Global Head of Cardiovascular, Renal and Metabolism (CVRM) Product Development

Unlocking the potential of our pipeline to address global CVRM disease burden

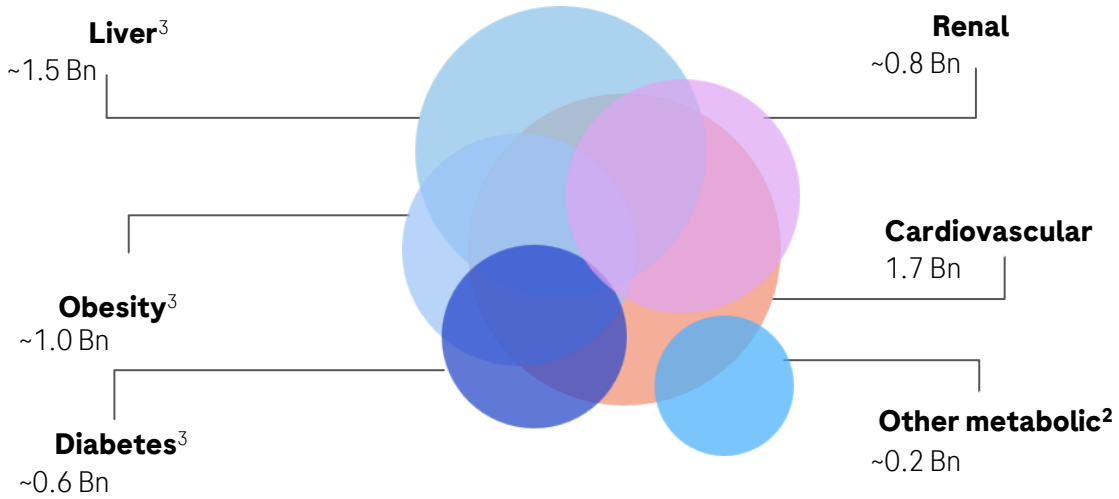
Critical Capabilities

Next gen NMEs with broad MoAs	*Address the breadth of diseases across CVRM by tackling adiposity dependent and independent drivers*
Depth of research and technologies	*Understand the interconnected science, utilize diverse modalities*
Combinations	*Establish a portfolio of combination therapies within CVRM and adjacent TAs*
Holistic patient solutions	*Synergies with Roche Pharma + Diagnostics/Digital Health solutions to prevent and treat CVRM diseases*

CVRM: Cardiovascular, renal, metabolism; NME: New molecular entity; MoA: Mechanism of action; TA: Therapeutic area

Growing prevalence of CVRM diseases is driven by aging, obesity and multimorbidity

Global prevalence in 2025 (Bn patients)[1]



Patient populations for these disease areas are overlapping due to comorbidities

Total CVRM burden: 4+ billion

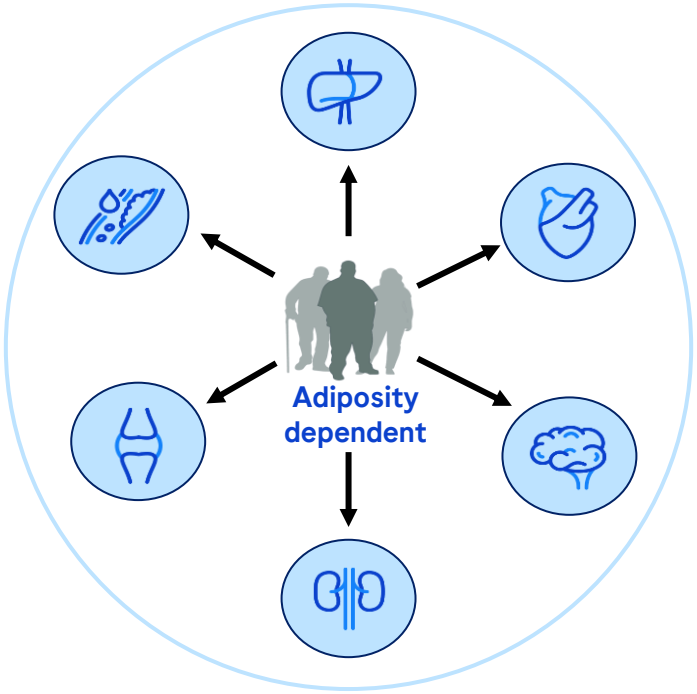
CVRM disease will remain significant in the future

60-65%
of the global adult population expected to have a **CVRM disease by 2050**

1. Numbers are rounded to the nearest 0.1 Bn; prevalence estimates based on scientific literature 2. Other metabolic includes e.g., polyendocrine metabolic ovarian syndrome, gout 3. Liver includes mainly MASH, MASLD 4. Est. growth: SEA +15 pp, Western Pacific +13 pp, Eastern Mediterranean +12 pp, Africa +8 pp, Americas +9 pp, Europe +2 pp; 3. Clarivate DRG Epidemiology Dashboard 2025; Sources: Hu SS et al., Chin Med J, 2025; Wang L et al., JAMA Intern Med, 2023; Zhou J et al., Clin Gastroenterol Hepatol, 2025; Chen Y et al., J Clin Transl Hepatol, 2024; Estes C et al., J Hepatol, 2018; IDF Diabetes Atlas, 11th ed., 2025; Zhou B et al., Lancet, 2024; Phelps NH et al., Lancet, 2024; Wang L et al., Lancet, 2021; Dandona L et al., Lancet Glob Health, 2018; Kalra A et al., Int J Environ Res Public Health, 2025; GBD 2023 CKD Collaborators, Lancet, 2025; Duseja A et al., J Hepatol, 2024; Kalra S et al., J Gastroenterol Hepatol, 2024; Misra A et al., Diabetes Ther, 2025; Anjana RM et al., Lancet Diabetes Endocrinol, 2023; Srivastava S et al., BMJ Open, 2025; Yusuf S et al., Lancet, 2004; Satoh M et al., Hypertension Res, 2026; WHO Hypertension Key Facts, 2025

Adiposity is a key driver of multi-organ disease; however, adiposity-independent drivers also contribute

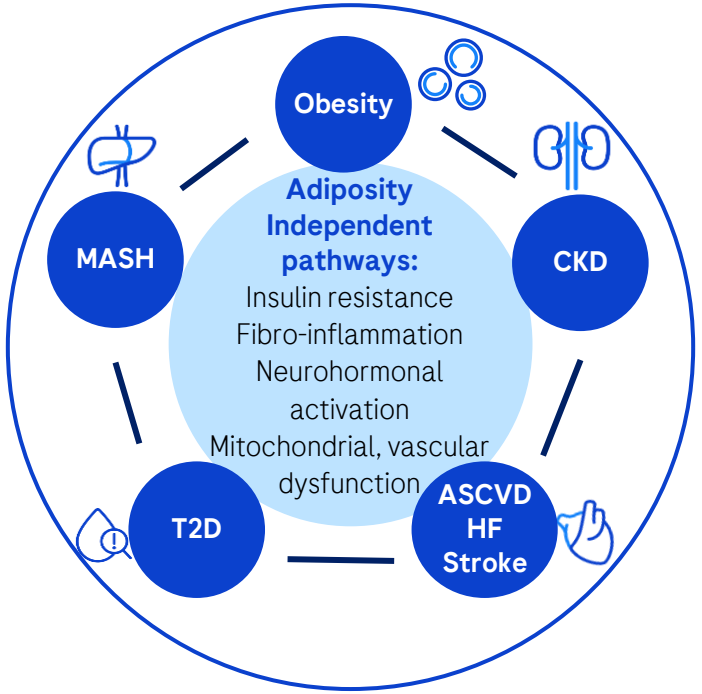
Reducing body weight with incretin-based therapy improves obesity associated comorbidities



Each disease treated in isolation

20% MACE recurrence within 5 years
40% Residual elevated inflammation
6-8% Residual cardiorenal event

CVRM diseases are interconnected with both adiposity and non-adiposity drivers impacting organ function and mortality



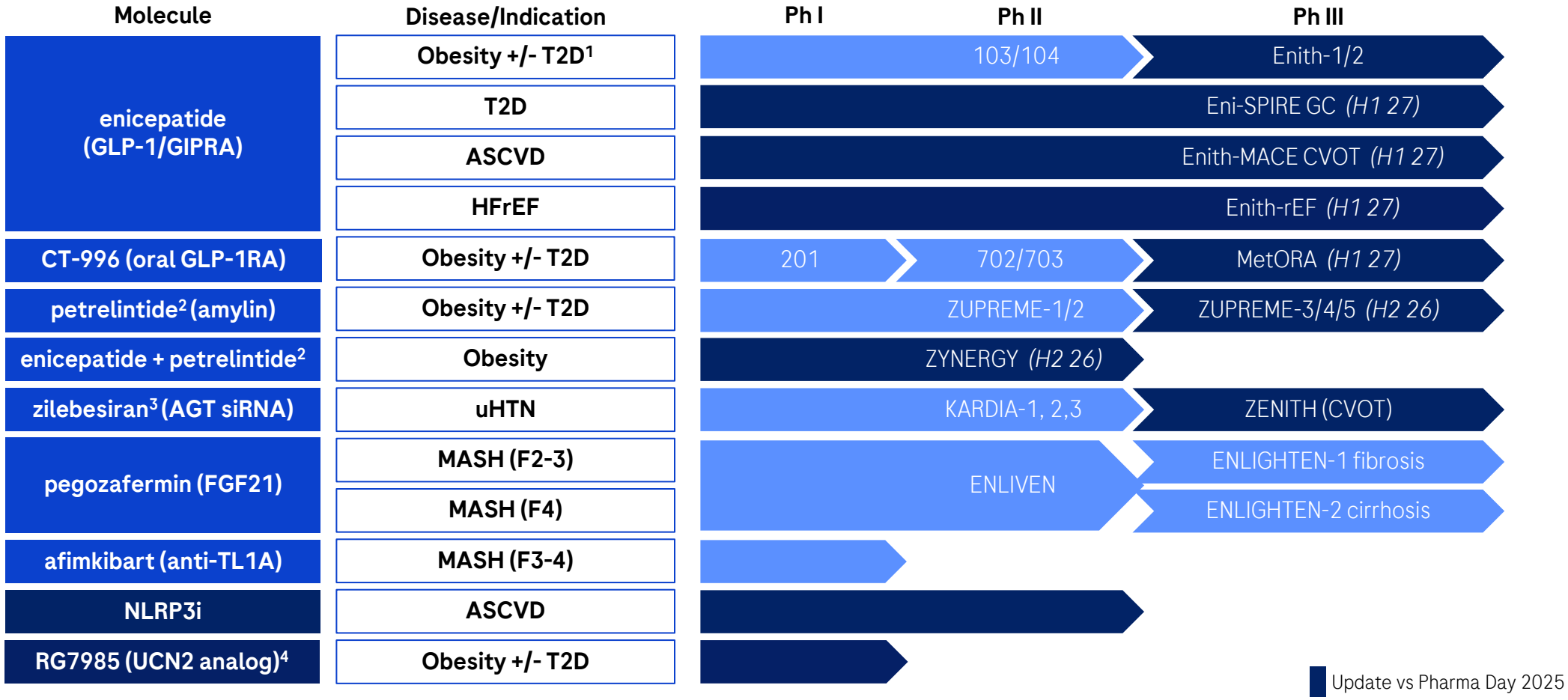
Pathways/Pathophysiology

Interconnected CVRM diseases

LEADER (NEJM 2016); SUSTAIN-6 (NEJM 2016); SELECT (NEJM 2023). NEJM: New England Journal of Medicine

A pipeline designed for multi-organ protection

Addressing both adiposity dependent and independent drivers across the interconnected CVRM spectrum

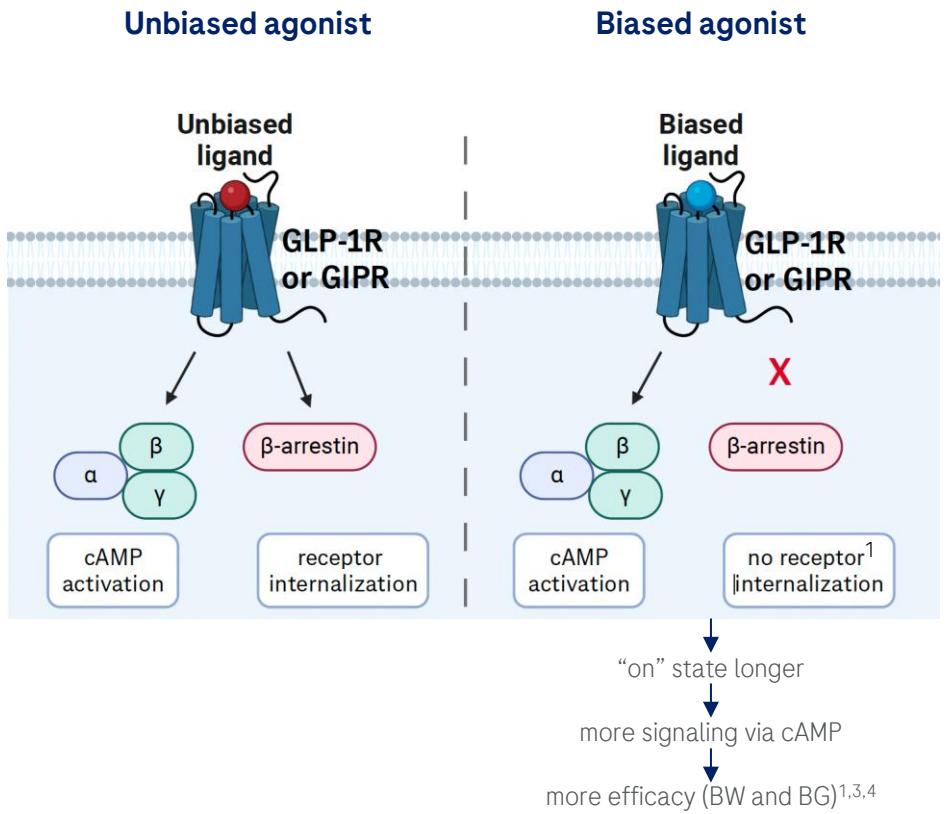


1. Patients with obesity or overweight with at least one weight-related comorbidity including type 2 diabetes; 2. Petrelintide in partnership with Zealand Pharma; 3. Zilebesiran in partnership with Alnylam; 4. RG7985, HM17321 in collaboration with Hanmi Pharma, UCN2: Urocortin-2 (UCN2) analog, pending deal closure; ASCVD: Atherosclerotic cardiovascular disease; GC: Glycemic control; ; CAD: Coronary arterial disease; CVOT: Cardiovascular outcomes trial; HFrEF: Heart Failure with reduced Ejection Fraction; MASH: Metabolic dysfunction-associated steatohepatitis; OW/OB: Overweight/Obesity; T2D: Type-2 diabetes; uHTN: uncontrolled hypertension

Enicepatide is intentionally designed to be signal-biased

Leveraging biased GLP-1/GIP agonism for potentially greater efficacy and tolerability

Enicepatide is a fully biased agonist



Minimal to no ß-arrestin recruitment results in prolonged glucose lowering and weight loss

- Native hormones and approved GLP-1R agonists signal through both cAMP and β-arrestin – "unbiased" agonism[1]
- Enicepatide is G protein-biased limiting β-arrestin recruitment but retaining cAMP signaling potency[2-4]
- Elimination of β-arrestin coupling minimizes receptor degradation potentially leading to prolonged pharmacological activity[5]

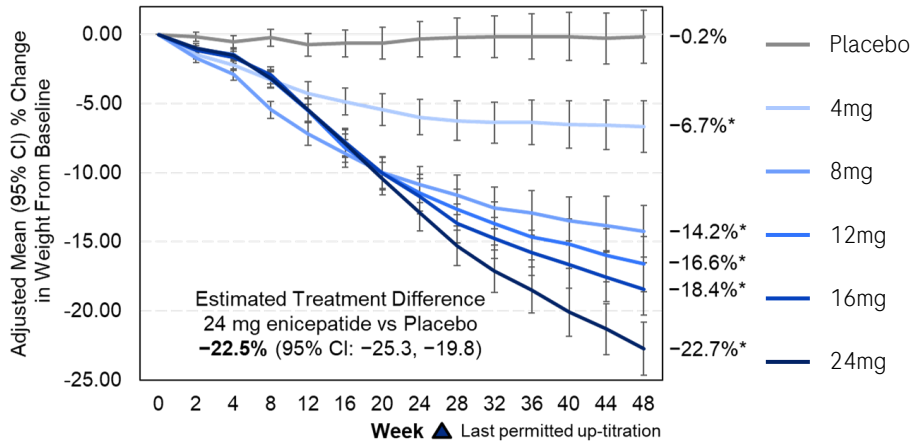
1. Jones B et al. Nature Communications. 2018;9:160 2. Rodriguez R et al. Presented at the American Diabetes Association 83rd Scientific Session, 23-26 June 2023; San Diego, CA. 3. Chakravarthy MV et al. Presented at the European Association for the Study of Diabetes, 59th Annual Meeting, 2-6 October 2023; Hamburg, Germany. 4. Luo J et al. Presented at the American Diabetes Association 84th Scientific Sessions; June 21-24, 2024; Orlando, FL.; 5. https://carmot.us/incretin-receptor-signaling-bias/; BG: blood glucose; BW: body weight; cAMP: cyclic adenosine monophosphate; GIPR: glucose-dependent insulinotropic polypeptide receptor; GLP-1R: Glucagon-like peptide 1 receptor

Enicepatide Ph II (103) in OW/OB: Robust weight loss with no plateau

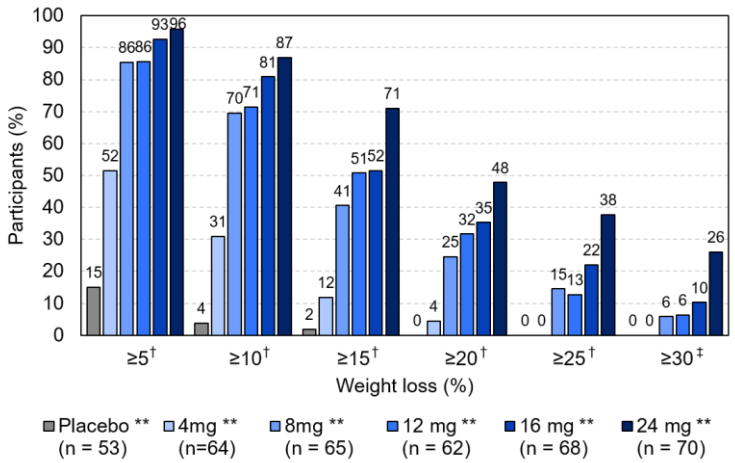
More than a quarter of treated participants achieved ≥ 30% weight loss by 48 weeks

Ph II (103) results in overweight/obesity[1]

Weight loss (efficacy estimand)



Weight reduction thresholds (at 48 weeks)



EASD 2026 data presentations

- Ph II (103) study results in OW/OB pts at 48-weeks (ORAL)
- Enicepatide reduced vascular, haemodynamic and body fluid parameters in OW/OB people, Ph II (103) study (short oral)
- Enicepatide reduced cv prognostic markers of central and visceral adiposity in OW/OB adults with ≥ 1 comorbidity, Ph II (103) study (short oral)
- Enicepatide reduced cardiovascular risk factors in OW/OB people, Ph II (103) study (short oral)

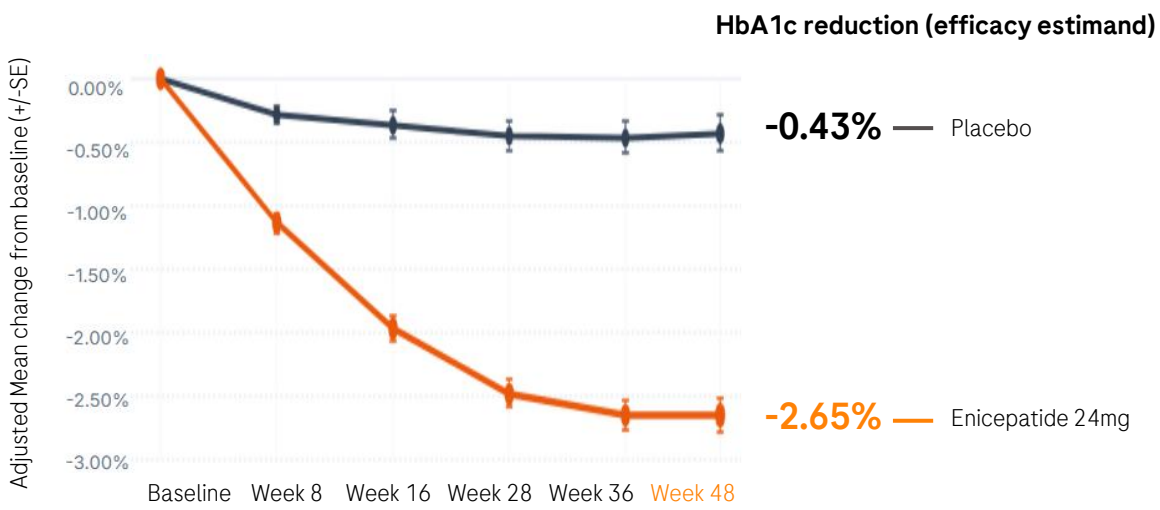
- Dose-dependent WL with up to 22.7% change in BW without a plateau at 48 weeks → potential to achieve additional WL with longer treatment
- At 48 weeks, 48% of participants achieved ≥ 20% WL, 38% ≥ 25% WL, and 26% ≥ 30% WL at the highest dose
- Tolerability profile generally consistent with other incretin-based therapies at similar development stage
- @EASD 2026: changes in BP, lipids, CRP and body composition

1. Lingvay I et al., ADA 2026, Poster-2813-LBA; *p ≤ 0.001 vs placebo; **The denominator to calculate the proportion is based on the number of participants who have reached week 48. †Prespecified endpoint; BIC: Best in class; BP: blood pressure; BW: body weight; CI: Confidence interval; CRP: C-reactive protein; CVOT: Cardiovascular outcomes trial; T2D: Type 2 diabetes; WL: weight loss

Enicepatide Ph II (104) in OW/OB + T2D: Potential for BID glycemic control

> 60% of treated pts achieved normoglycemia, those with baseline HbA1c >8.5 dropped HbA1c >4% by 48 wks

Ph II (104) Enicepatide in T2D pts with overweight/obesity



HbA1c reduction by current therapies

Molecule	HbA1c reduction*	
	All comers	BL A1c > 8.5%
Enicepatide	-2.65%	-4.13%
semaglutide	-1.3 to -2.2%[1]	-2.8%[5]
tirzepatide	-1.93 to -2.37%[2]	-3.24%[6]
cagrisema	-1.91 to -2.1%[3]	-2.58%[7]
retatrutide	-1.7 to -2.0%[4]	

**Values are non-pbo adjusted HbA1c reductions using the efficacy estimand; studies with similar baseline HbA1c to study104. Standard cross-trial comparison caveats apply, incl. variations in background medications, body weight, treatment duration, etc.*

- Clinically meaningful and statistically significant HbA1c reduction from baseline to W48 of 2.65% (baseline HbA1c 8.1%)
- HbA1c reduction of 4.13% in patients with poor baseline glycemic control (baseline A1c >8.5%)
- Proportion of patients at W48 achieving target HbA1c ≤6.5% = 90% and <5.7% = 62%
- WL at W48: -15.5% with no plateau
- Discontinuation due to AEs: 2.0% in enicepatide arms vs 0% in placebo; no level 2 hypoglycemia; safety & tolerability in line with incretin class

1. Semaglutide: -1.3% (SUSTAIN 2, semaglutide 0.5 mg) to -2.2% (SUSTAIN FORTE, semaglutide 2 mg); 2. tirzepatide: -1.93% (SURPASS 3, tirzepatide 5 mg) to -2.37% (SURPASS 3, tirzepatide 15 mg); 3. cagrisema: -1.9% (REIMAGINE 2, cagrisema 2.4/2.4 mg) to -2.1% (REDEFINE 2, cagrisema 2.4/2.4 mg); 4. retatrutide: -1.7% (TRANSCEND T2D-1, retatrutide 4 mg) to -2% (TRANSCEND T2D-1, retatrutide 9 mg): only published trial in T2DM population; 5. semaglutide: -2.8% (SUSTAIN 1 trial); 6. tirzepatide: -3.24% (SURPASS 3); 7. cagrisema: -2.58% (REIMAGINE-2); AEs: adverse events; BID: best-in-disease; BL: baseline; BMI: body mass index; BW: body weight; Co-1 EP: co-primary endpoint; CV: cardiovascular; FU: follow-up; HbA1c: glycated hemoglobin; OW/OB: overweight/obesity; pbo: placebo; Ph: phase; reta: retatrutide; SE: standard error; sema: semaglutide; SGLT2i: sodium-glucose cotransporter-2 inhibitor; T2D/T2DM: type 2 diabetes / type 2 diabetes mellitus; WL: weight loss

Enicepatide: Broadening Ph III clinical development program

Ph III in Glycemic control, HFrEF and CVOT will be initiated in H1 2027

Clinical development program

Molecule	Disease/Indication	Ph I	Ph II	Ph III	Status
Enicepatide GLP-1/GIP RA (QW)	OW/OB	CT-388-101			✓
	OW/OB	CT-388-103			✓
	OW/OB + T2D	CT-388-104			✓
	OW/OB	Enith-1 (CWM)			Ongoing
	OW/OB + T2D	Enith-2 (CWM)			Ongoing
	T2D	Eni-SPIRE (GC)			To be initiated H1 27
	ASCVD ± T2D	Enith-MACE (CVOT, including T1D subgroup)			To be initiated H1 27
	HFrEF ± T2D	Enith-rEF (CV death/HF event reduction)			To be initiated H1 27

✓ Primary endpoint met

- Ph III CWM studies (Enith-1/2) in people with overweight/obesity is ongoing with recruitment on track
- Ph III GC study in people with T2D, including a head-to-head study against tirzepatide – H1 2027
- Ph III CVOT-MACE and CVOT-HFrEF in people with ASCVD/heart failure – H1 2027

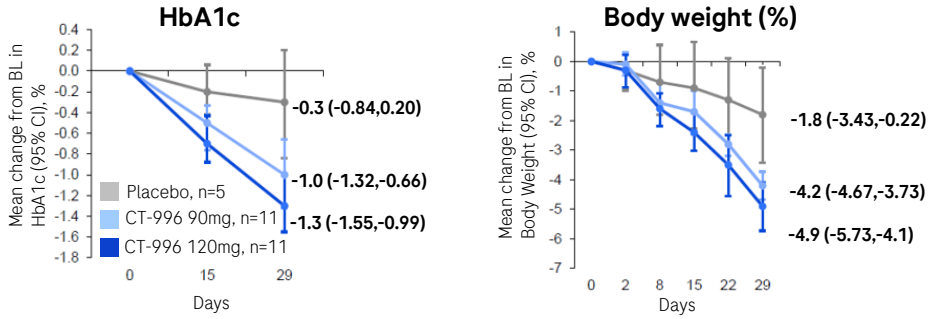
ASCVD: Atherosclerotic cardiovascular disease; CWM: Chronic weight management; CV: Cardiovascular; CVOT: Cardiovascular outcomes trial; GC: Glycemic control; GIP: Glucose-dependent insulinotropic polypeptide; GLP-1: Glucagon-like peptide-1; HF: Heart failure; HFrEF: Heart failure with reduced ejection fraction; MACE: Major adverse cardiovascular events; RA: Receptor agonist; QW: Once weekly; OW/OB: Overweight/Obesity; T2D: Type 2 diabetes

CT-996: Oral small molecule with positive Ph II, moving into Ph III

Additional Ph I data in T2D to be presented Oct 1 at EASD, demonstrating strong reduction of HbA1c

Ph I 4wk data in OW/OB +T2D

CT-996 Ph I (201, arm 3) OW/OB + T2D, 4 wks



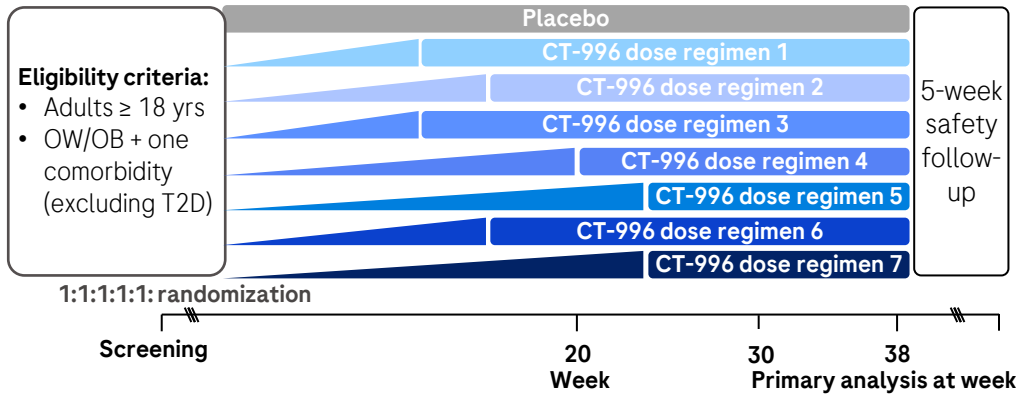
HbA1c reduction up to 1.3%

Body weight reduction up to 4.9%

- HbA1c reduction of up to 1.3% within 4 weeks
- Strong FPG lowering with no glycemic excursions during MMTT
- WL up to 4.9% and no plateau
- PK supports once-daily dosing without food/water restrictions
- Data to be presented at EASD 2026 on Oct 1

CT-996: Ph II in overweight/obesity

CT-996 Ph II obesity trial without T2D participants[1]



- Wide dose ranging and various titration regimens explored
- Overall data support advancing the program to Phase 3

1. NCT07081958; BL: Baseline; CI: Confidence interval; EASD: European Association for the Study of Diabetes; EoP2: end of Phase 2; GI: Gastrointestinal; HbA1c: Glycated hemoglobin; OW/OB: Overweight/Obesity; QD: Once daily; T2D: Type 2 diabetes; TEAE: Treatment-emergent adverse event; WL: weight loss

CT-996: Ph III initiation for chronic weight management

Oral small molecule combining convenience with efficacy to optimize real-world adherence

Clinical development program

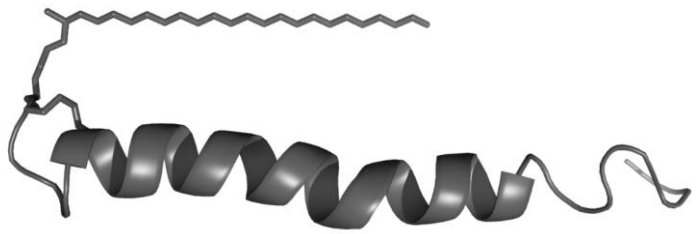
Molecule	Disease/Indication	Ph I	Ph II	Ph III	Status
CT-996 GLP-1 RA (QD)	OW/OB	CT-996-201, Arm 1			✓
	Obesity	CT-996-201, Arm 2			✓
	OW/OB + T2D	CT-996-201, Arm 3			✓
	OW/OB	CT-996 (-702 study)			✓
	OW/OB + T2D	CT-996 (-703 study)			Ongoing
	OW/OB + T2D	MetORA CMW Program			To be initiated H1 27

✓ Primary endpoint met

GLP-1: Glucagon-like peptide-1; RA: Receptor agonist; QD: Once daily; OW/OB: Overweight/Obesity; T2D: Type 2 diabetes; CWM: Chronic weight management

Petrelintide: Potential to redefine weight management experience with double-digit weight loss and improved tolerability

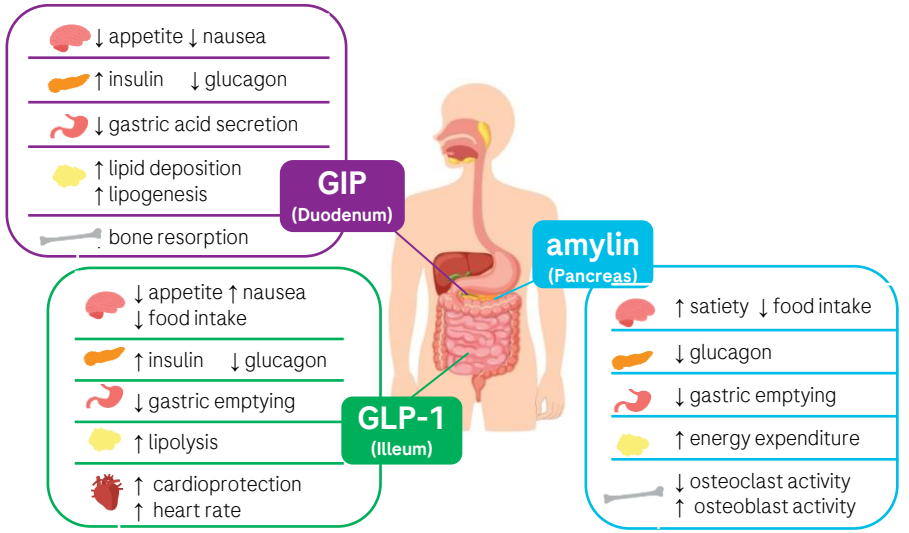
Petrelintide: Long-acting amylin analog suitable for Q1W dosing[1-3]



36 amino-acid acylated peptide

- Potent balanced agonist effect on amylin and calcitonin receptors[2,4]
- Restores leptin sensitivity to increase satiety (vs. suppressing appetite)[5] along with improved insulin sensitivity

Enicepatide + petrelintide: Complementary MoA with potential for BID weight loss and glycemic control



- Amylin and GLP-1/GIP signaling may support satiety through distinct pathways, potentially offering BID weight loss while maintaining a favourable tolerability profile
- Ph II (ZYNERGY) will evaluate the efficacy and safety of petrelintide + enicepatide combination in pts with OW/OB6; FPI expected Q4 2026

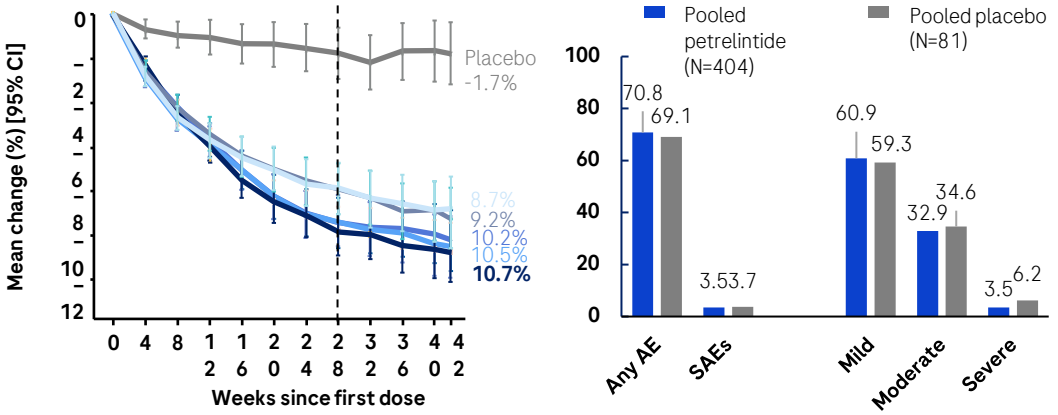
1. Developed in collaboration with Zealand Pharma; 2. Data on file; 3. Brændholt Olsen et al. Poster 92-LB. Presented at ADA 83rd Scientific Sessions, June 23–26, 2023, San Diego, CA; 4. Eriksson et al. Presentation at ObesityWeek, November 1–4, 2022, San Diego, CA; 5. Hayes et al. Annu Rev Nutr 2014;34:237–260; 6. Weight-related comorbidities include prediabetes, hypertension, dyslipidemia, obstructive sleep apnea, or cardiovascular disease; BID: Twice daily; FDC: Fixed-dose combination; FPI: First patient in; GIP: Glucose-dependent insulinotropic polypeptide; GLP-1: Glucagon-like peptide-1; MoA: Mechanism of action; OW/OB: Overweight/Obesity; Q1W/QW: Once weekly

Petrelintide: Ph III monotherapy program in OW/OB +/- T2D initiated

Ph II data in OB/OW achieved double digit WL with improved tolerability

Ph II ZUPREME-1 results

Baseline characteristics: 53% female, mean BMI 37



- Statistically significant and clinically meaningful weight loss from baseline after 28 weeks in all five treatment arms vs. placebo
- Weight loss of up to 10.7% sustained through week 42 vs 1.7% on placebo
- AE rates comparable to placebo

Clinical development program

Molecule	Disease/Indication	Ph I	Ph II	Ph III	Status
Petrelintide amylin analog (QW)	OW/OB				✓
	OW/OB		ZUPREME-1		✓
	OW/OB + T2D		ZUPREME-2		Data 2026
	OW/OB			ZUPREME-3	FPI Q4 2026 (for Z-3)
	OW/OB + T2D			ZUPREME-4	
	OW/OB			ZUPREME-5	

✓ Primary endpoint met

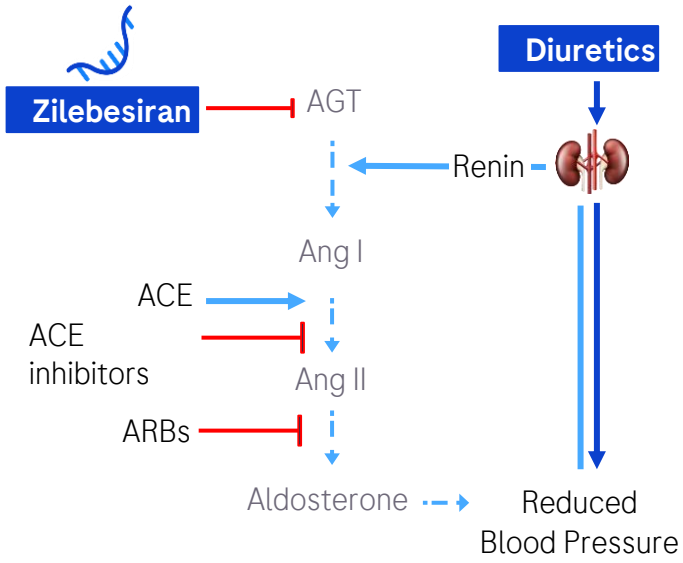
- Ph II (ZUPREME-1) in obesity met primary endpoint in H1 2026; data presented at ADA, additional data at EASD 2026
- Ph II (ZUPREME-2) in obesity with T2D data expected H2 2026
- Ph III studies in OW/OB (ZUPREME-3) FPI expected Q4 2026
- Evaluating initiation of additional Ph III trials

Petrelintide developed in collaboration with Zealand Pharma; ADA: American Diabetes Association; AE: Adverse event; BMI: Body mass index; CI: Confidence interval; EASD: European Association for the Study of Diabetes; FPI: First patient in; H2: Second half; OW/OB: Overweight/Obesity; QW: Once weekly; SAE: Serious adverse event; T2D: Type 2 diabetes

Zilebesiran: Novel MoA with potential to transform HTN management

Continuous control of blood pressure with 2x yearly dosing aiming to reduce cardiovascular risk

Zilebesiran targets most upstream precursor to RAAS pathway[1]

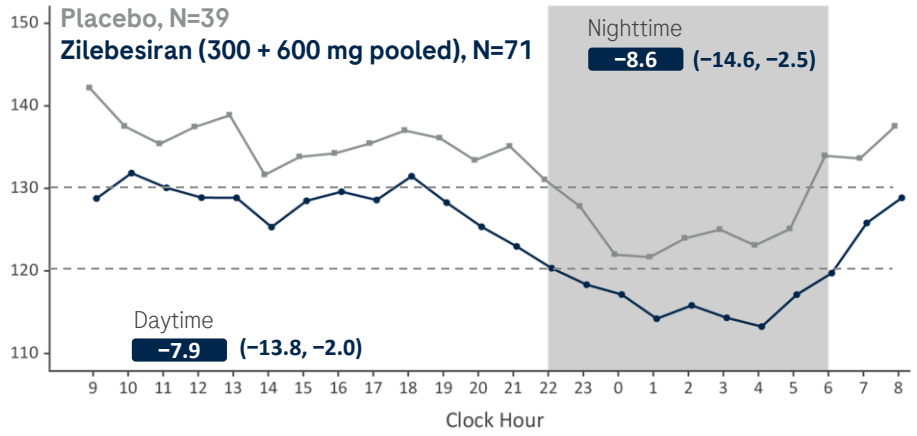


- Despite existing oral therapies, less than 50% of patients achieve BP control due to poor adherence
- Zilebesiran offers potential for tightest and continuous control of BP thereby improving CV outcomes

Ph II Kardia-3 posthoc analysis in pts on baseline diuretic with office SBP ≥140mm Hg at month 6[2]

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Change from baseline (pbo-adj) in Daytime, Nighttime, and 24-hour SBP at mo 6



Phase III CVOT (ZENITH)

- 11,000 patients with CVD/risk, SBP >140 mmHg on stable treatment including a diuretic)
- **Primary Outcome:** Composite endpoint of CV death, nonfatal MI, nonfatal stroke, or HF event
- Recruitment on track

- K-3 post hoc analysis: −8.1 mm Hg (−13.9, −2.4) reduction in 24h SBP at month 6 in pts receiving diuretic with office systolic BP ≥140 mmHg → informed design for ZENITH
- Significant reduction in nighttime SBP → single strongest predictor of CV mortality (JACC Advances 2024)

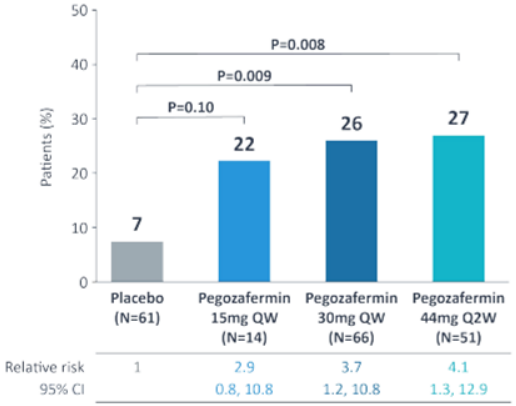
1. Figure adapted from Kario K. Prog Cardiovasc Dis. 2016;59(3):262–281; 2. Pagidipati et al. ESC 2026; Zilebesiran in partnership with Alnylam Pharmaceuticals; ACE: Angiotensin-converting enzyme; AGT: Angiotensinogen; Ang I/II: Angiotensin I/II; ARB: Angiotensin receptor blocker; BP: Blood pressure; CV: Cardiovascular; CVD: Cardiovascular disease; CVOT: Cardiovascular outcomes trial; HF: Heart failure; HTN: Hypertension; K-3: Kardia-3; MI: Myocardial infarction; RAAS: Renin-angiotensin-aldosterone system; SBP: Systolic blood pressure

Pegozafermin: Potential best-in-disease therapy in MASH

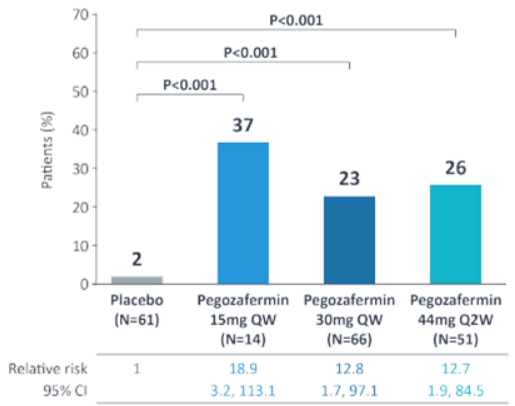
Further strengthens CVRM portfolio and offers optionality for future combination development

Ph II (ENLIVEN) week 24 results in pts with MASH and fibrosis (F2-F4)[1]

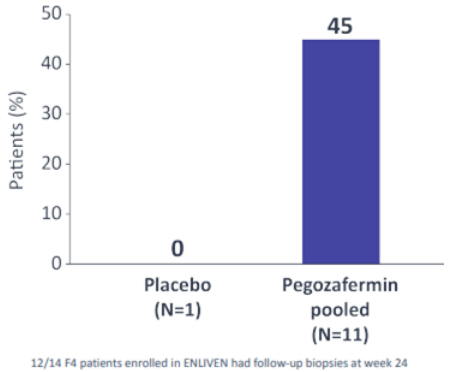
Fibrosis improvement ≥ 1stage without worsening of MASH



MASH resolution without worsening of fibrosis



Pts with F4 at baseline: Fibrosis improvement ≥1 stage without worsening of MASH



- Within 24 weeks, improvements in both MASH resolution and fibrosis observed, including fibrosis improvement in those with compensated cirrhosis (F4c) at baseline*
- Sustained benefits on fibrosis markers were observed vs. placebo in patients on background GLP-1 therapy at week 48 (ENLIVEN 48-week extension data)
- Pegozafermin was well tolerated across patients

Clinical development program

Indication	Ph I	Ph II	Ph III	Status
MASH F2-F3		ENLIGHTEN-Fibrosis		Ongoing
MASH F4		ENLIGHTEN-Cirrhosis		Ongoing

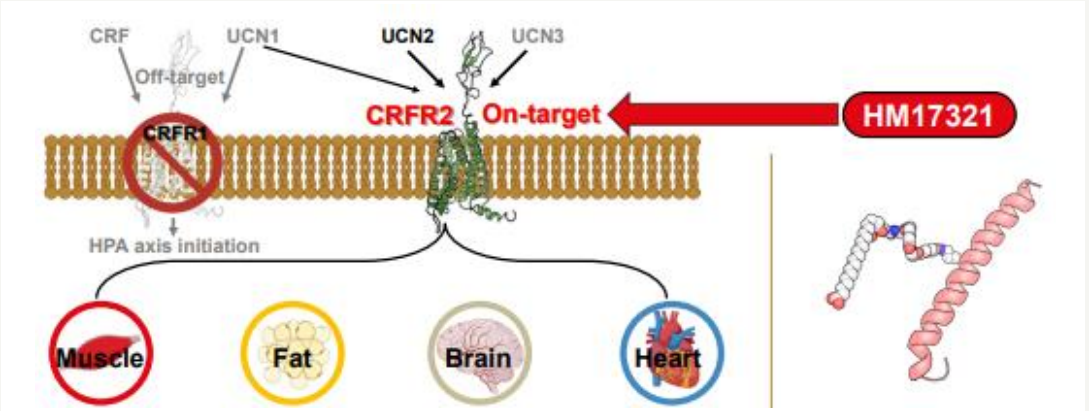
- Ph III ENLIGHTEN-Fibrosis (F2-F3): Topline histology data exp. in H1 2028
- Ph III ENLIGHTEN-Cirrhosis (F4): Topline histology data exp. in 2028+
- MASH diagnosis: Elecsys PRO-C3 test combined with the ADAPT algorithm

1. Loomba et al. EASL 2023; *Fourteen ENLIVEN F2/F3 subjects were reclassified as F4 by 3-panel read; CVRM: Cardiovascular, renal and metabolism; GLP-1: Glucagon-like peptide-1; MASH: Metabolic dysfunction-associated steatohepatitis; Q2W: Every two weeks; QW: Once weekly

RG7985[1]: UCN2 analog in obesity with FIC potential

Pleiotropic effects for weight loss, improved insulin sensitivity, muscle preservation, and cardiac protection

CRFR2-selective biased urocortin 2 (UCN2) analog



- Hypertrophy ↑
- Insulin sensitivity ↑
- Energy expenditure ↑
- Lipolysis ↑
- Appetite ↓
- Cardiac protection (under investigation)
- High selectivity to on-target
- Long-acting suitable for QW

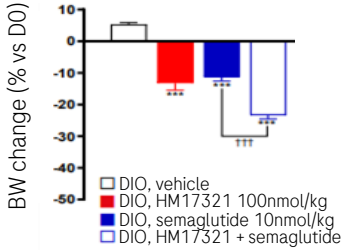
Figure adapted from Kim J et al, Hanmi Pharmaceutical, EASD Sep15-19, 2025

- Long-acting UCN2 selectively targeting the corticotropin-releasing hormone receptor 2 (CRFR2) in brain, skeletal muscle and heart
- The UCN2 pathway has profound pleiotropic potential, enabling a single molecule to simultaneously drive fat breakdown, improve ins sensitivity, preserve muscle, and support cardiovascular function

Preclinical data in DIO mice

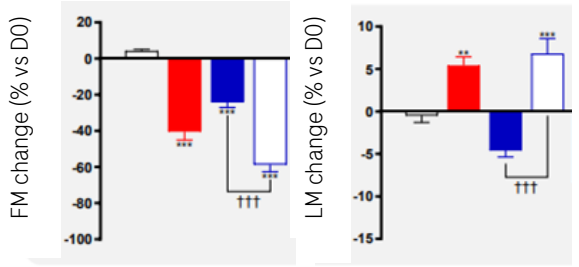
Effects of HM17321 (RG7985) in combination with incretin-based drugs on body weight and body composition

Body weight (BW) change at EoT



HM17321 showed similar WL to semaglutide alone with **additive WL effects in combination**

Fat (FM) and lean mass (LM) change at EoT

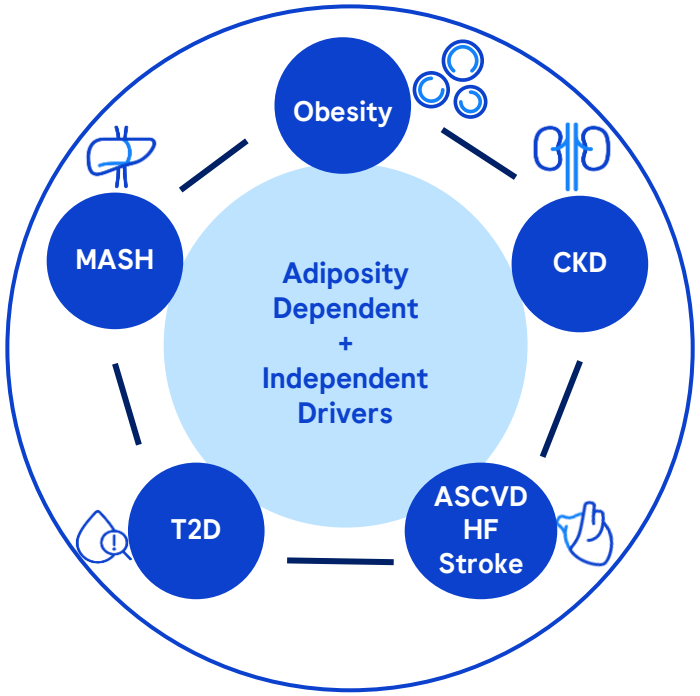


HM17321 + Sema resulted in greater fat mass loss while maintaining/enhancing lean mass

Such a profile suggests enhanced muscle insulin sensitization, and could contribute to durable WL maintenance

1. RG7985 (HM17321) in collaboration with Hanmi Pharma, pending deal closure; BW: Body weight; BWL: Body weight loss; CRF: Corticotropin-releasing factor; CRFR2: Corticotropin-releasing factor receptor 2; CRFR1: Corticotropin-releasing factor receptor 1; DIO: Diet-induced obese; EASD: European Association for the Study of Diabetes; EoT: End of treatment; FIC: First-in-class; FM: Fat mass; HPA: Hypothalamic-pituitary-adrenal; LM: Lean mass; QW: Once weekly; Sema: Semaglutide; UCN: Urocortin;

Treating and preventing CVRM diseases holistically for those living with or without obesity



Treat organ dysfunction → **Prevent organ damage** → **Reduce Global Health burden**

Our focus: Address both adiposity-dependent and –independent drivers using both external + internal R&D to develop transformative medicines to reduce the burden of CVRM diseases

Doing now what patients need next

Translating operational rigor and R&D excellence into sustainable, long-term growth



R&D excellence

- All major solutions embedded as business-as-usual
- The Bar applied across all-inhouse and partnered assets
- Total pipeline value (aNPV) at an all-time high



Operational excellence

- Augmented manufacturing productivity & network optimization
- Disciplined capital allocation to maximize long-term value creation
- AI integration across the full value chain



Commercial excellence

- Maximizing key on-market assets, incl. via line extensions and devices
- Targeted execution across entrench, expand and establish archetypes
- Scaling capabilities for upcoming NME launches across TAs



Launching the next wave of innovations

Near-term catalysts
Giredestrant & fenebrutinib leading the first wave, plus up top 4 additional NME launches by YE 2027

20 NMEs by 2030
Total wave of up to 20 NME launches projected by 2030

6 new Phase III transitions
Refueling the pipeline with 6 NMEs advancing to Ph III since Pharma Day 2025

aNPV: adjusted net present value; NME: New molecular entity; TA: Therapeutic areas

Appendix

Changes to the development pipeline

Pharma Day update

New to phase I

3 NMEs:
RG7985[1] UCN-2 analog - obesity +/- T2D
CHU allosteric ALK inhibitor (AQUA07) – NSCLC
RG6767 NME - immunology

Phase I initiation* (3 NMEs + 1 AI) :

RG6375 CEA12Pb PRIT - mCRC (exp Q3 26)
RG6527 NME - COPD (exp Q3 26)
RG6644 NME - COPD (exp Q4 26)
RG6160 cevostamab - SLE +/- LN (exp Q3 26)

New to phase II

1 NME:
RG6794 CDK4/2i - HR+ HER2- BC

Phase II initiation* (2 NMEs) :

RG6434 NRLP3i – AD, PD (exp Q4 26)
RG6035 Brainshuttle™ CD20 - multiple sclerosis (exp H1 27)

New to phase III

Phase III initiation* (5 NMEs) :

RG6849[2] petrelintide - obesity +/- T2D (H2 26)
RG6209 VEGF x IL6 DutaFab - DME (exp H1 27)
RG6652 CT-996 - obesity +/- T2D (exp H1 27)
RG6640 enicepatide (CT-388) - obesity +/- T2D, ASCVD, HFrEF (exp H1 27)
RG6919 tambotatug pelitecan – 2L+ SCLC (Q4 26)

New to registration

1 AI (US):
RG6168 Enspryng - MOGAD

2 NMEs (US):
RG7845 fenebrutinib - RMS, PPMS
RG6179 vamikibart - UME

Removed from phase I

1 NME:

RG6795 CDK4i - HR+ HER2- BC

Removed from phase II

1 NME:

RG6237 emugrobart (GYM 329) – obesity

1 AI:
RG6631 afimkibart (anti-TL1A) - atopic dermatitis

Removed from phase III

Approvals

1 NME (EU):
RG6321 Susvimo – wAMD

1 AI (US):
RG7159 Gazyva – childhood onset idiopathic nephrotic syndrome

Status as of September 28, 2026

*FPI pending, not yet accounted for in the pipeline/count ;1. in collaboration with Hanmi Pharma, pending deal closure; 2. Zealand Pharma managed

Roche Group development pipeline

Phase I (38 NMEs + 5 AIs)

RG6026	Columvi monotherapy + combos	heme tumors
RG6171	giredestrant monotherapy + combos	solid tumors
RG6330	divarasib monotherapy + combos	solid tumors
RG6411	enzelkitug (anti-CCR8)	solid tumors
RG6505	PanRAS inhibitor	solid tumors
RG6537	AR degrader	mCRPC
RG6538	P-BCMA-ALLO1	r/r multiple myeloma
RG6540	P-CD19 x CD20 - ALLO1	heme tumors
RG6620	KRAS G12D inhibitor	solid tumors
RG6648[1]	cMET ADC	solid tumors
RG6733	-	MM
RG6735	-	heme tumors
RG6810[2]	DLL3 ADC	SCLC
RG6852	-	mCRPC
RG6919[1]	tambotatug pelitecan (B7-H3 ADC)	solid tumors
RG6975[3]	CDH17 ADC	CRC
RG7828	Lunsumio monotherapy + combos	heme tumors
CHU	clesitamig (DLL3 trispecific)	solid tumors
CHU	codrituzumab	HCC
CHU	MINT91	solid tumors
CHU	anti-CTLA-4 switch antibody	solid tumors
CHU	pan-KRAS inhibitor (AUBE00)	solid tumors
CHU	allosteric ALK inhibitor (AQUA07)	NSCLC
RG6421	TMEM16A potentiator	Muco-obstructive respiratory disease
RG6498	-	IBD
RG6767	-	immunology
RG6981[4]	Il33:TSLP	COPD/Asthma
CHU	anti-C1s recycling antibody	immunology
RG6622	-	coronary artery disease
RG6631	afimkibart (anti-TL1A)	MASH
RG7985[9]	UCN-2 analog	obesity +/- T2D
RG6035	Brainshuttle CD20	multiple sclerosis
RG6434	NLRP3i	neurodegenerative disorders
RG6627[5]	APOE ASO	Alzheimer's disease
RG6662	HTT miRNA GT	Huntington's Disease
RG6209	VEGF x IL6 DutaFab	DME
RG6327	-	geographic atrophy
RG6441	-	DR
RG6697	-	DME
RG6858	-	retinal diseases
RG6913	-	DME
RG6006	zosurabalpin	bacterial infections
CHU	REVN24	acute diseases

Phase II (12 NMEs + 5 AIs)

RG6114	Itovebi + enzalutamide	CRPC
RG6114	Itovebi + ribociclib + fulvestrant	1L HR+ Chr8P-loss PIK3CA-wt mBC
RG6180	autogene cevumeran	solid tumors
RG6596	HER2 TKI (ZN-1041)	HER2+ mBC
RG6794	CDK4/2i	HR+ HER2- BC
RG6797	dirloctocogene samoparvovec (SPK-8011QQ)	hemophilia A
RG6631	afimkibart (anti-TL1A)	rheumatoid arthritis
RG6730[6]	p40xTL1A bispecific antibody	UC
RG7828	Lunsumio	SLE
CHU	anti-HLA-DQ2.5 x gluten peptides antibody	celiac disease
RG6418	selnoflast (NLRP3i)	coronary artery disease
RG6652	GLP-1 RA (CT-996)	obesity/ T2D glycemic control
RG6849[7]	petrelintide	obesity +/- T2D
RG6182	MAGL inhibitor	multiple sclerosis
RG6289	nivegacetor (γ-secretase modulator)	Alzheimer's
RG6356[8]	Elevidys	DMD, <4 years
RG6501	OpRegen	geographic atrophy

New Molecular Entity (NME)
Additional Indication (AI)
Oncology / Hematology
Immunology
Cardiovascular, Renal & Metabolism
Neurology
Ophthalmology
Other

Status as of September 28, 2026

RG-No - Roche/Genentech; CHU - Chugai managed; 1. MediLink managed; 2. Innovent managed; 3. In collaboration with Hansoh Pharma; 4. Qyuns Therapeutics managed; 5. In collaboration with IONIS; 6. In collaboration with Pfizer; 7. Zealand Pharma managed; 8. US rights with Sarepta; 9. In collaboration with Hanmi Pharma, pending deal closure

Roche Group development pipeline

Phase III (12 NMEs + 19 AIs)

RG3502	Kadcyla + T	HER-2+ eBC high-risk
RG6013	Hemlibra	Type 3 VWD
RG6026	Columvi + Polivy + R-CHP	1L DLBCL
	Columvi	r/r MCL
RG6107	PiaSky	aHUS
RG6114	Itovebi + fulvestrant	post CDKi HR+ PIK3CA-mut. BC
	Itovebi + Phesgo	1L HER2+ PIK3CA-mut. mBC
	Itovebi + CDK4/6i + letrozole	1L ES PIK3CA-mut. HR+ HER2- advanced BC
RG6160	Cevostamab	r/r MM
RG6171	giredestrant + Phesgo	1L ER+/HER2+ BC
	giredestrant + CDK4/6i	1L ET resistant ER+/HER2- BC
RG6330	divarasib	2L NSCLC
	divarasib + pembrolizumab	1L NSCLC
RG6512	zemocimig (NXT007)	hemophilia A
RG7473	bexobrutideg	r/r CLL
RG7828	Lunsumio + lenalidomide	2L+ FL
RG7853	Alecensa	LA stage III NSCLC
RG6149	astegolimab	COPD
RG6299[2]	sefaxersen (ASO factor B)	IgA nephropathy
RG6631	afimkibart (anti-TL1A)	ulcerative colitis
	afimkibart (anti-TL1A)	Crohn's disease
RG6615[1]	zilebesiran	Hypertension
RG6640	enicepatide (CT-388)	obesity +/- T2D
RG6881	pegozafermin	MASH with fibrosis stage F2 or F3
	pegozafermin	MASH with fibrosis stage F4
RG6102	trontinemab	early Alzheimer's
RG6168	Enspryng	autoimmune encephalitis
RG6356[3]	Elevidys	DMD, 8 years and older
RG7935	prasinezumab	Parkinson´s
RG6321	Susvimo	wAMD, 36-week
RG7716	Vabysmo	CNV

Registration US & EU (3 NMEs + 9 AI)

RG6171	giredestrant + everolimus†	post-CDK4/6 ER+/HER2- BC
	giredestrant†	ER+ BC adj
RG7446	Tecentriq	stage III dMMR/MSI-H colon cancer
RG7828	Lunsumio + Polivy†	2L+ DLBCL
RG7159	Gazyva	systemic lupus erythematosus
	Gazyva††	childhood onset idiopathic nephrotic syndrome*
	Gazyva	membranous nephropathy
RG6168	Enspryng†	TED
RG6179	vamikibart	UME
RG6168	Enspryng	MOGAD
RG7845	fenebrutinib†	RMS
	fenebrutinib†	PPMS

†Filed in US
††Filed in EU, approved in US

New Molecular Entity (NME)
Additional Indication (AI)
Oncology / Hematology
Immunology
Cardiovascular, Renal & Metabolism
Neurology
Ophthalmology
Other

Status as of September 28, 2026

1. Alnylam Pharmaceuticals managed; 2. In collaboration with IONIS; 3. US rights with Sarepta; T:Tecentriq, *also known as pediatric nephrotic syndrome (PNS)

Doing now what patients need next