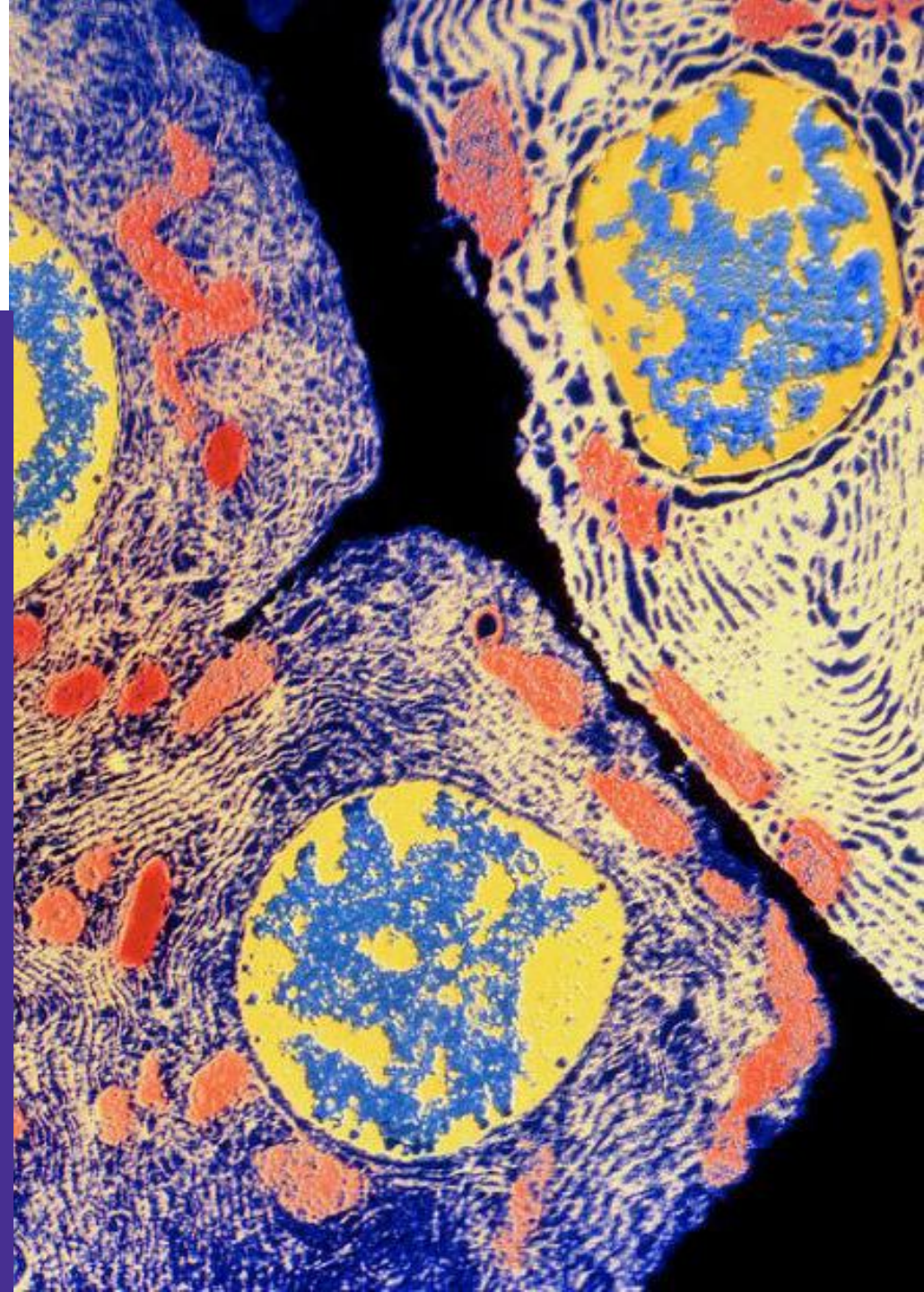


Corporate Presentation

October 2026



Forward-looking statements

This presentation contains forward-looking statements which include, but are not limited to, statements regarding CalciMedica's business strategy and clinical development plans; the design and potential benefits of CalciMedica's product candidates; CalciMedica's ongoing and planned clinical trials; expected Intellectual Property (IP) protections; the timing for CalciMedica's receipt and announcement of data from its clinical trials and other clinical and regulatory milestones, including expectations regarding the timing for regulatory approvals; the estimated patient populations and addressable market for CalciMedica's product candidates; and expectations regarding CalciMedica's financial position, capital requirements, and anticipated cash runway. These forward-looking statements are subject to the safe harbor provisions under the Private Securities Litigation Reform Act of 1995. CalciMedica's expectations and beliefs regarding these matters may not materialize. Actual outcomes and results may differ materially from those contemplated by these forward-looking statements as a result of uncertainties, risks, and changes in circumstances, including but not limited to risks and uncertainties related to: the impact of fluctuations in global financial markets on CalciMedica's business and the actions it may take in response thereto; CalciMedica's ability to execute its plans and strategies; the ability to obtain and maintain regulatory approval for CalciMedica's product candidates; results from clinical trials may not be indicative of results that may be observed in the future; potential safety and other complications from CalciMedica's product candidates; economic, business, competitive, and/or regulatory factors affecting the business of CalciMedica generally; CalciMedica's ability to protect its intellectual property position; expected length of IP protection for CalciMedica's product candidates; the impact of government laws and regulations; and CalciMedica's cash runway and need for additional capital. Additional risks and uncertainties that could cause actual outcomes and results to differ materially from those contemplated by the forward-looking statements are included under the caption "Risk Factors" in CalciMedica's most recently filed periodic report, and subsequent periodic reports filed by CalciMedica, under the Securities Exchange Act of 1934, as amended, from time to time and available at www.sec.gov. These documents can be accessed on CalciMedica's web page at calcimedica.com.

These forward-looking statements are based on information available to, and expectations of, CalciMedica as of the date of this presentation. CalciMedica disclaims any obligation to update these forward-looking statements, except as may be required by law.

CalciMedica: Leaders in CRAC channel inhibition

Inhibiting CRAC channels to control excessive calcium signaling and protect organ tissues

Two selective
drug candidates

Auxora (IV)
Acute hospital treatment

CM5480 (oral)
Chronic treatment

Pulmonary hypertension

Advancing with recent financing

New Frontier in PH treatment:

**Direct right-heart protection and improved function
along with pulmonary pressure reduction**

Supported by preclinical evidence across five models

Acute pancreatitis

Potential upside

Phase 2b Trial complete:

**Reduction in severe organ failure,
particularly respiratory failure**

FDA-aligned next trial in pivotal program

Catalysts
Mid-2027

- Auxora PAH proof-of-concept Phase 1b data
- CM5480 IND submission

Cash runway
Into 2H 2027

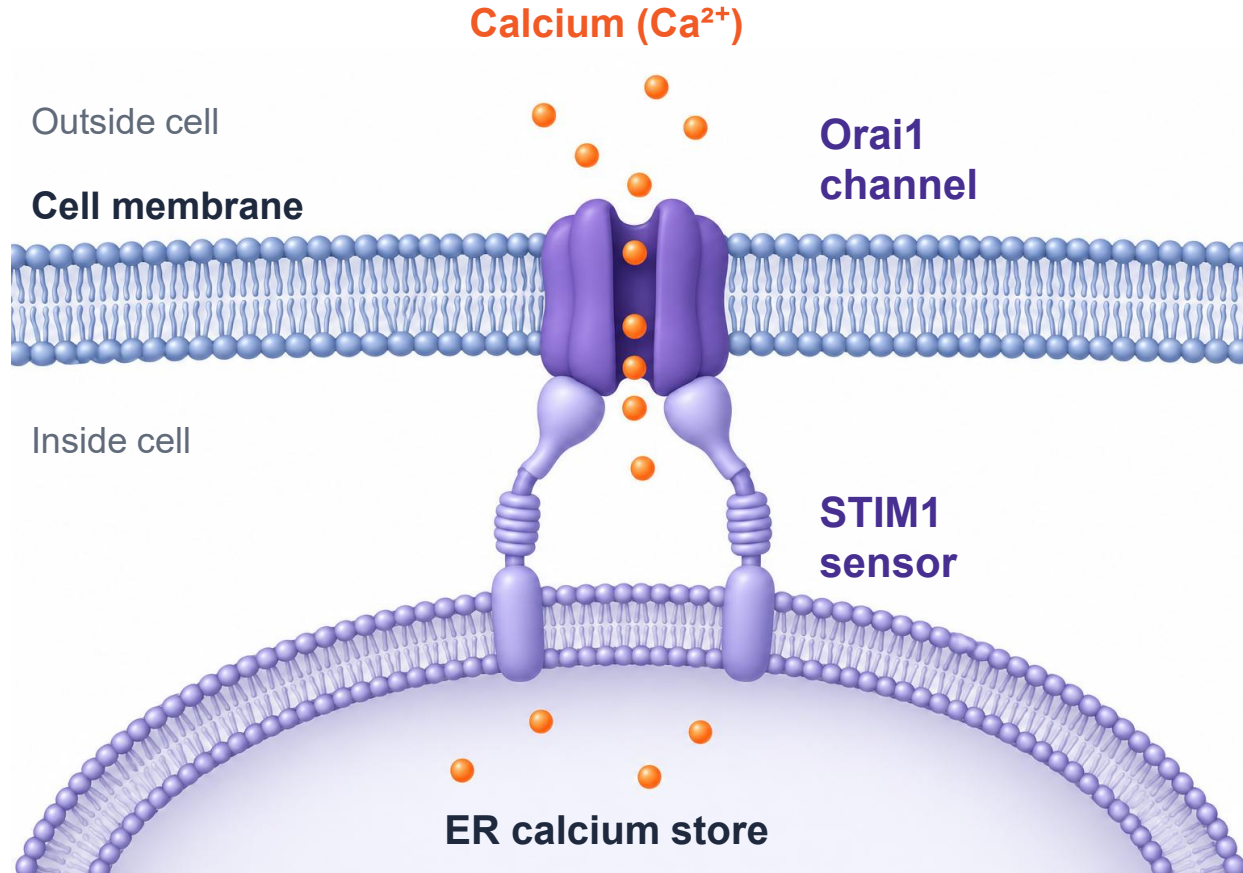
Pipeline & Candidates



PH-focused pipeline with human proof-of-concept data expected in mid-2027

Program	Purpose	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated milestones
Pulmonary hypertension						
Auxora – IV¹ PAH	Establish proof of concept in PAH and inform CM5480 development					IND submission — 2H 2026 Ph1b data — mid-2027
CM5480 – Oral PAH and CpcPH-HFpEF	Oral candidate for chronic dosing					IND submission — mid-2027
Acute critical disease						
Auxora – IV Acute pancreatitis	Acute hospital treatment					✓ FDA-aligned planned Phase 2b design (subject to funding)

CRAC channels: tightly regulated in health, overactivated and harmful in disease



In health

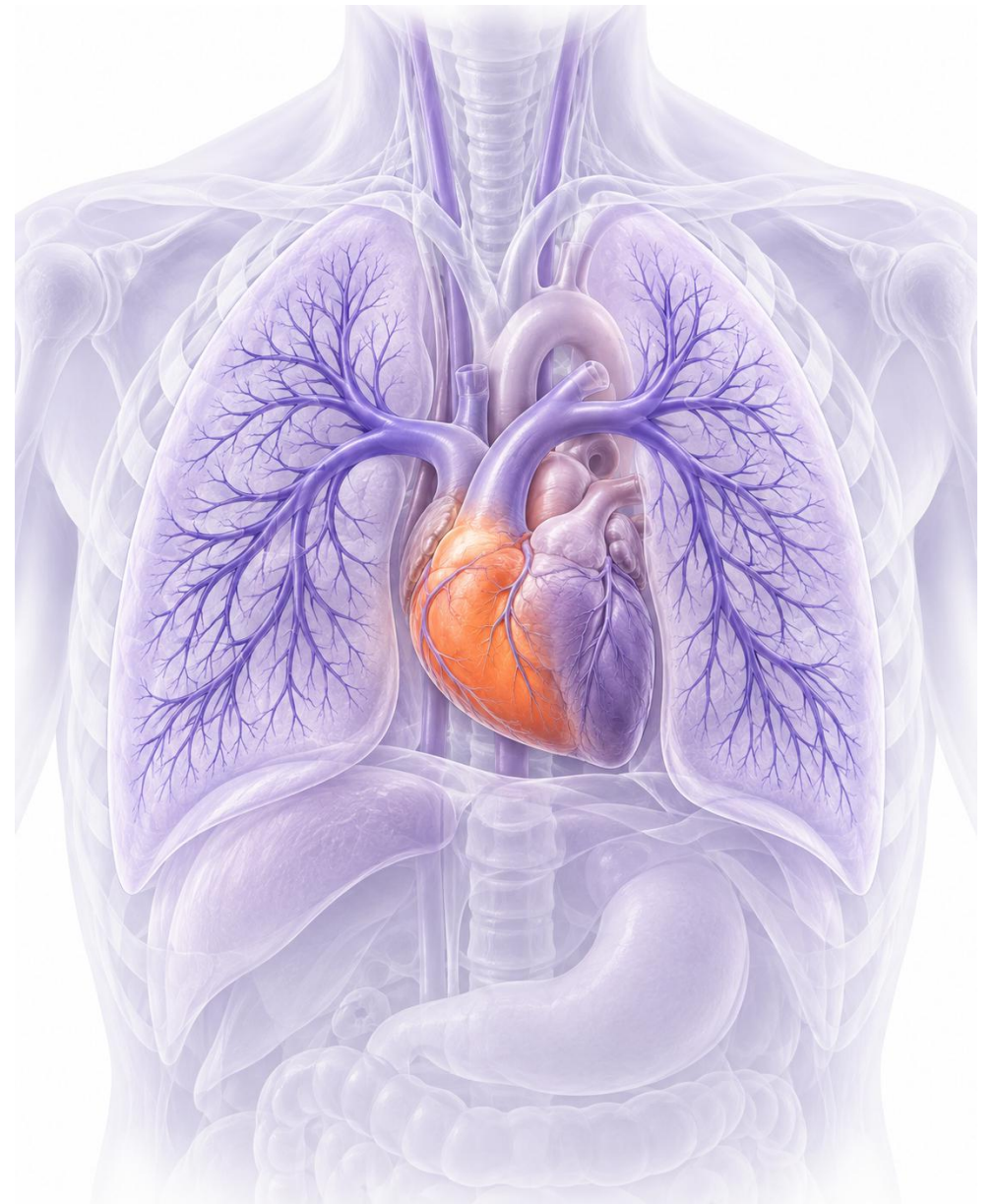
Calcium enters when internal stores run low. Feedback controls limit further entry.

In disease

Persistent activation can sustain inflammation, tissue damage, vascular leakage, and adverse remodeling of the heart and blood vessels.

CRAC channel inhibition aims to limit excessive, pathological calcium signaling

CRAC channel inhibition for pulmonary hypertension (PH)



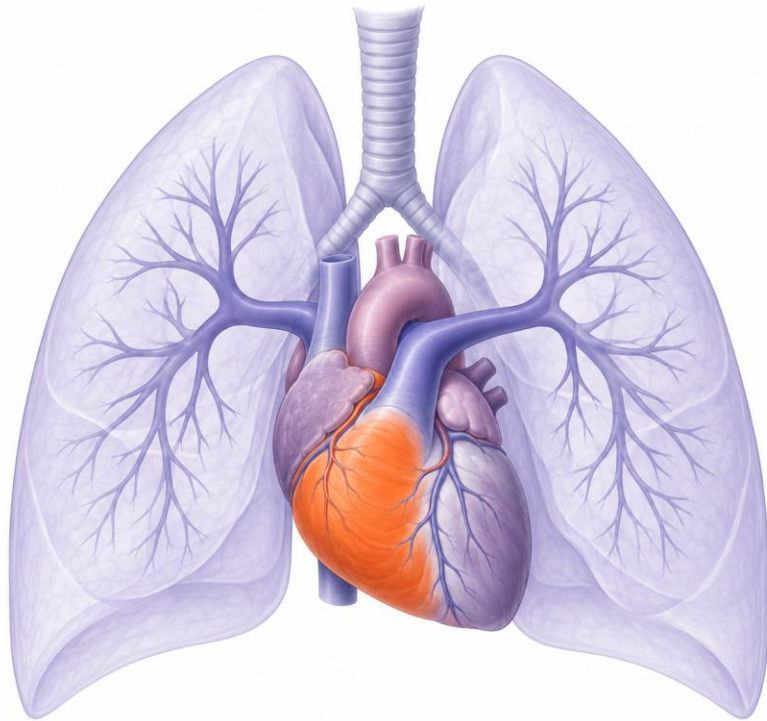
PH is a large opportunity with unmet need across five patient groups

Group 1	Group 2	Group 3	Group 4	Group 5
Pulmonary Arterial Hypertension (PAH)	Left Heart Disease	Lung Disease / Hypoxia	Pulmonary artery obstruction (including CTEPH)	Unclear / Multifactorial Mechanisms
~\$5.7B	~500K	~80K	~10–17K	Varied causes
2025 worldwide sales ¹ Selected PAH products	Est. U.S. patients ² CpcPH-HFpEF	Est. U.S. patients ³ PH-ILD	Est. U.S. patients ⁴ CTEPH	Treatment depends on the underlying disease
Residual disease despite treatment	No specifically approved therapy	Limited approved treatment options	Surgery, angioplasty and medical therapy	

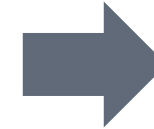
CpcPH-HFpEF = combined pre- and post-capillary pulmonary hypertension associated with heart failure with preserved ejection fraction; **PH-ILD** = pulmonary hypertension associated with interstitial lung disease; **CTEPH** = chronic thromboembolic pulmonary hypertension

1. J&J and Merck, FY2025 annual reports. 2. Modeled estimate using ~3.5M U.S. HFpEF patients × 60% with PH × 25% with CpcPH. Johns Hopkins Medicine 2024; 2022 ESC/ERS PH guidelines. 3. DelveInsight, Epidemiology Forecast 2025. 4. Delcroix M et al., Eur Respir J 2021: 30–50 cases/million × 2025 U.S. Census population

Across PH groups, elevated pressure threatens right ventricular function



**Elevated pulmonary
vascular pressure**



**Right ventricular
failure**

Focus of existing therapies

Not directly targeted
by approved PH therapies

**High pressure persists
despite advances**

- Even with combination therapy, arterial pressure remains ~2x ULN^{1,2}

**Unmet need to protect
RV for any given afterload**

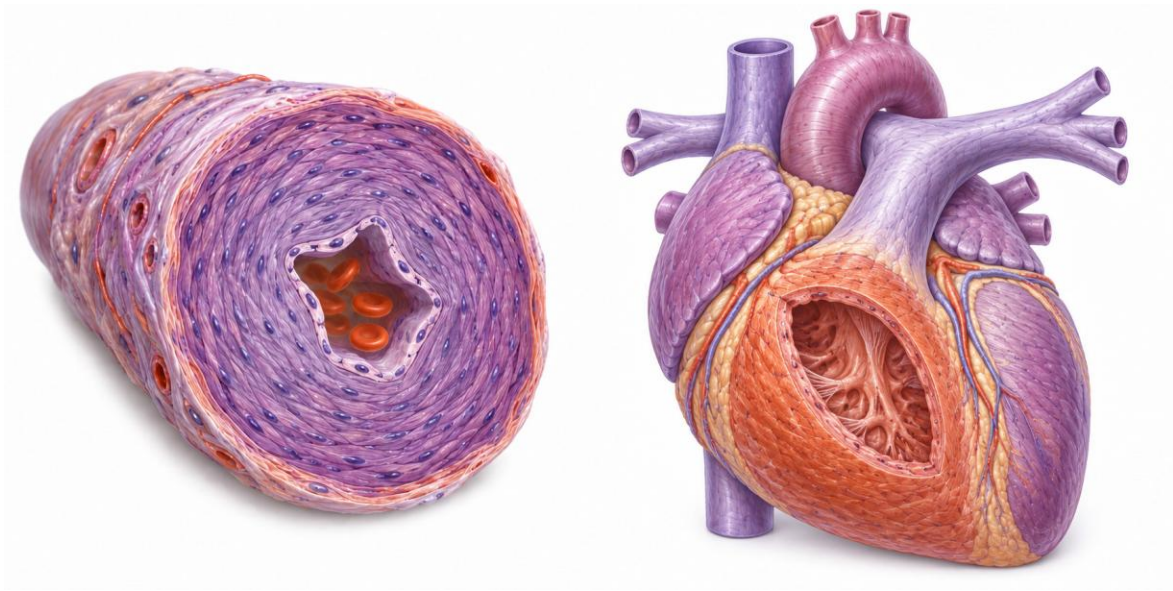
- RV dysfunction can persist despite improved hemodynamics³

RV function is a major determinant of prognosis and mortality³

CRAC channel inhibition targets both pulmonary pressure and direct protection of RV function

Pulmonary vasculature

Right ventricle



Vasoconstriction and remodeling

RV dysfunction and remodeling

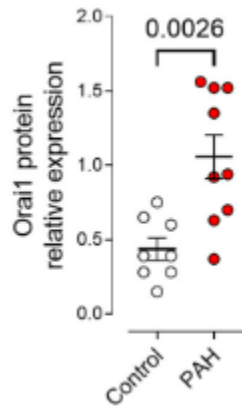
Therapeutic approach	Reduce pulmonary vascular pressure		Protect RV function
	Vasodilation	Vascular remodeling	Direct RV protection
Vasodilators	✓	—	(indirect)
Activin pathway / TKIs	—	✓	(indirect)
CRAC channel inhibition	✓	✓	✓

Pathway studies support distinct signaling of CRAC channel inhibition and potential additive benefit with existing therapies (see appendix)¹

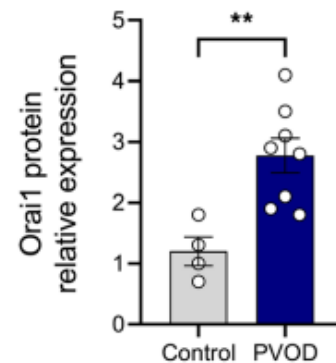
CRAC channels are upregulated in human pulmonary vascular and cardiac disease

Pulmonary Vasculature

Pulmonary Arteries (PAH)

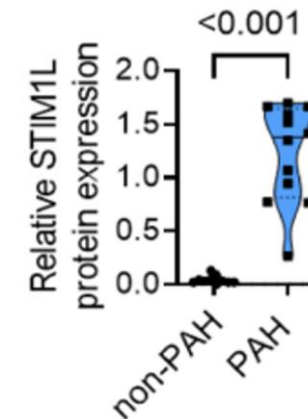


Pulmonary Veins (PVOD)

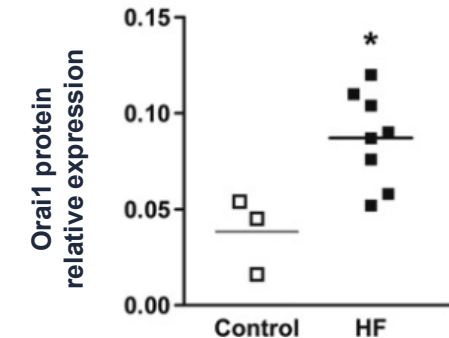


Heart

Right Ventricle (PAH)



Left Ventricle (Heart Failure)



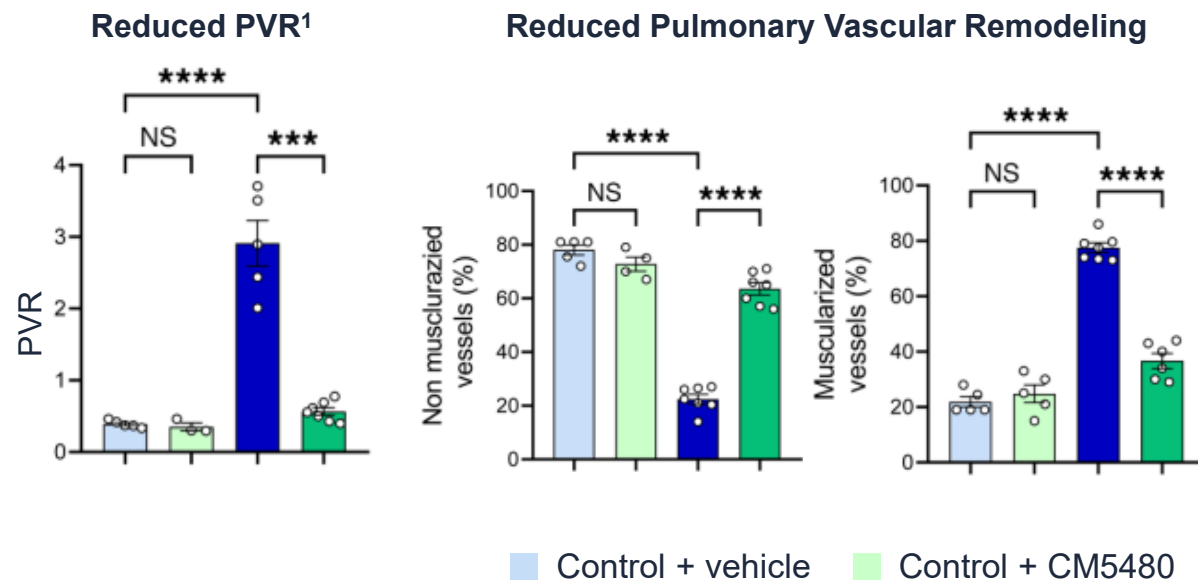
Further, CRAC channels appear to be linked to the remodeling and hypertrophy associated with PH

CRAC channel inhibition improves lung and heart outcomes across complementary preclinical models

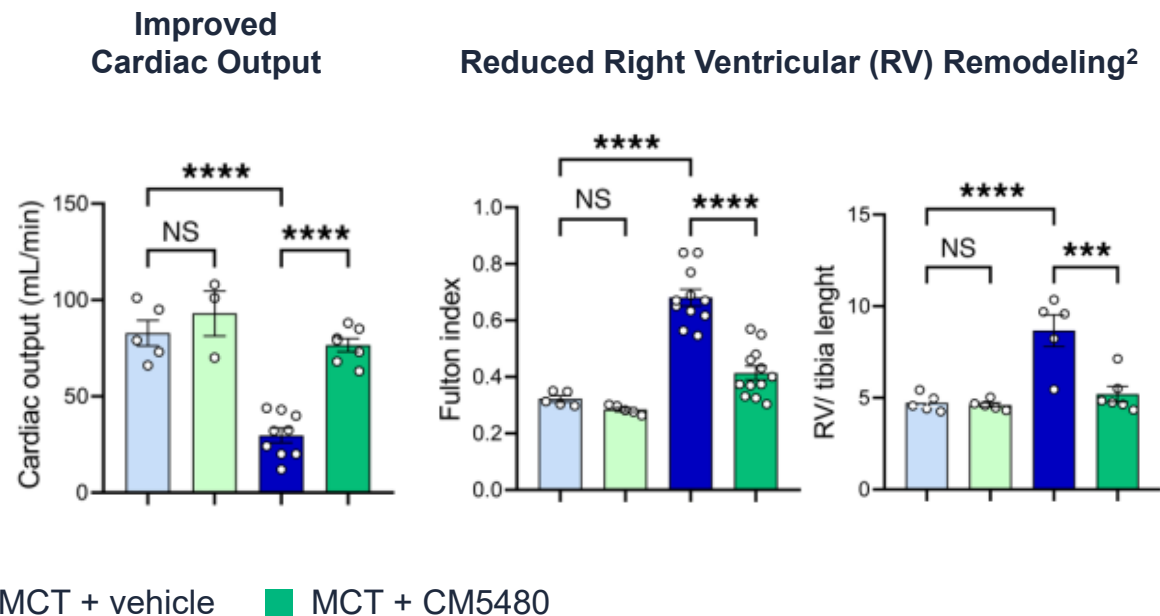
Model and compound	Pulmonary vasculature		Heart	
	Pulmonary pressure ↓	Vascular remodeling ↓	Ventricular function ↑	Ventricular remodeling ↓
MCT rat¹ CM5480	✓	✓	✓	✓
Sugen/hypoxia rat² Tool compound	✓	✓	✓	✓
Chronic hypoxia rat² Tool compound	✓	✓	✓*	✓
Pulmonary artery banding rat³ CM5480	Cardiac model Pulmonary endpoints not assessed		✓	✓
Transverse aortic constriction mouse⁴ Tool compound	Cardiac model Pulmonary endpoints not assessed		✓	✓

CM5480 improved function and reduced remodeling in the vasculature and heart in the MCT rat PAH model

Pulmonary Vasculature



Heart



CM5480 showed additive benefit in combination with vasodilators (sildenafil or ambrisentan)

CM5480 reversed a broad RV gene-expression signature in the MCT rat PAH model

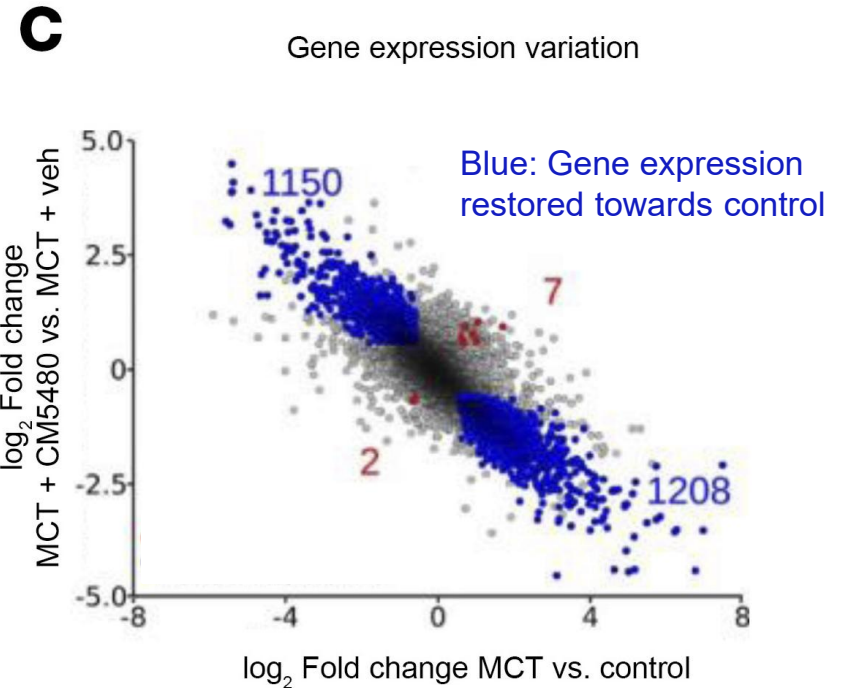
Right ventricle RNA sequencing in the MCT rat model

2,358

RV genes with expression changes restored towards control

Vs. only 9 genes showed amplified changes

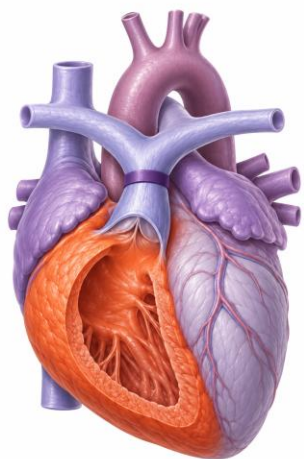
Pathways included heart contraction, mitochondrial metabolism and inflammation



Improved gene expression was also observed in the lung

Direct RV protection under pressure overload differentiates CM5480 from other therapies

Pulmonary artery
banding model (rat)



Fixed RV afterload isolates
direct cardiac effects from
pulmonary vascular effects

Metric	PAB untreated	CM5480 ¹	Sotatercept ²	Imatinib ³	Tadalafil ⁴ (vasodilator)
RV systolic function	↓	↑	↔ / ↓	↓	↔
RV diastolic function	↓	↑	↔	↓	↔
RV hypertrophy	↑	↓	↓	↑	↔
RV fibrosis	↑	↓	↓	↑	↔

PAB findings support potential direct RV-protective activity of CM5480

Cardiac and pulmonary venous findings support a Group 2 PH rationale for CRAC channel inhibition

Transverse Aortic Constriction Model (TAC) Results¹

Mice: 5 weeks of LV pressure overload, then 3 weeks of a tool Orai1 inhibitor or vehicle

EJECTION FRACTION

56.9% → 71.2%

+14.3 points

p = 0.001

END-SYSTOLIC VOLUME

0.089 mL → 0.044 mL

-51%

p = 0.003

LV-ARTERIAL COUPLING

1.36 → 2.86

2.1× higher

SV / ESV

FIBROTIC GENE EXPRESSION

Col1a1 · Col3a1

Prevented

p < 0.05 vs. vehicle

Rationale for Group 2 PH

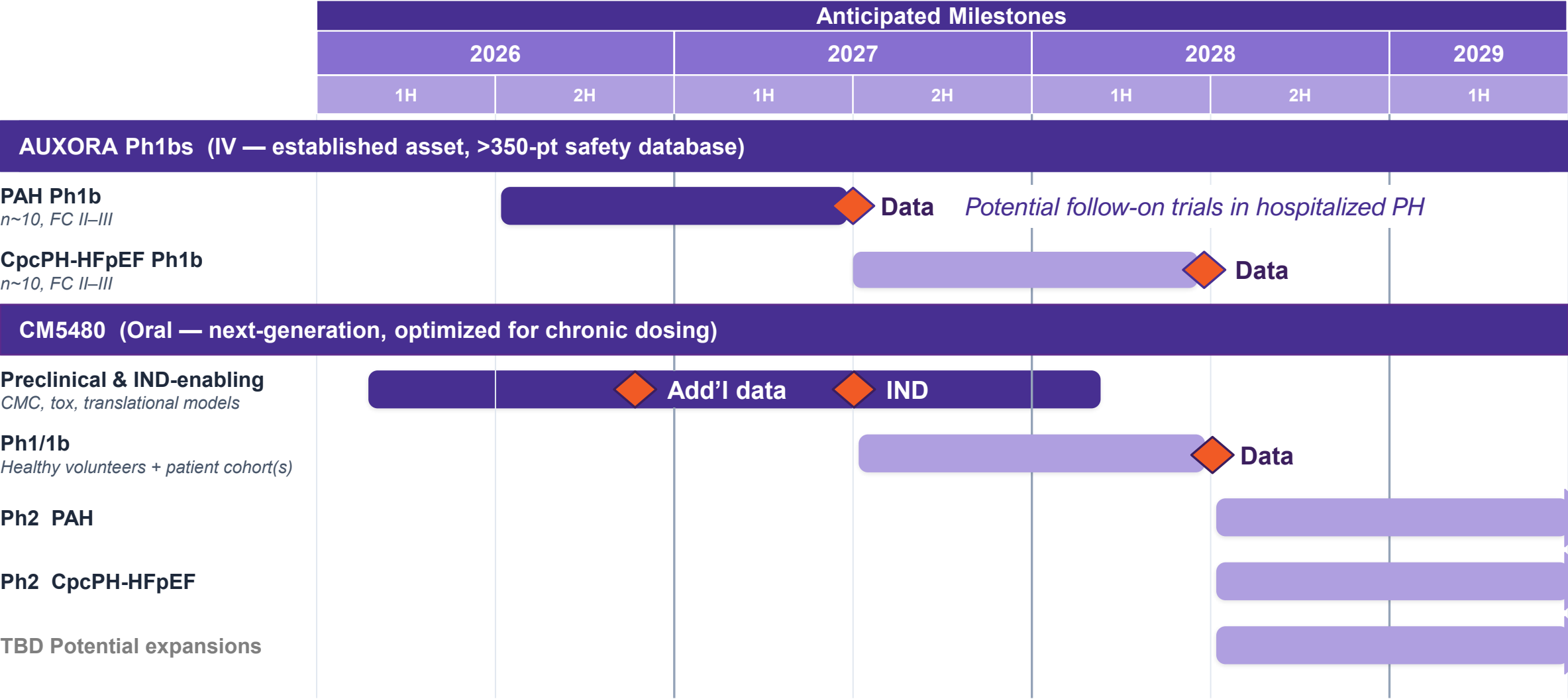
- **Protect the stressed LV:** Orai1 inhibition improved LV systolic function in TAC mice
- **Target pulmonary venous disease:** Increased Orai1 expression and calcium entry in human PVOD vein cells support a rationale in Group 2 PH, where venous remodeling is prominent^{2,3}
- **Limit cardiac fibrosis:** Orai1 inhibition prevented collagen-gene induction in TAC mice and reduced collagen secretion in human failing-LV fibroblasts⁴

LV = left ventricle; SV / ESV = stroke volume / end-systolic volume

Note: Study used JP111, a small-molecule Orai1 inhibitor used as tool compound for target validation; LV-arterial coupling shown here is calculated as SV/ESV; the authors do not report a coupling ratio.

Sources: 1. Bartoli F et al., Circulation 2020. 2. Saint-Martin Willer et al., JCI Insight 2025. 3. Fayyaz AU et al., Circulation 2018. 4. Ross GR et al., Biol Open 2017.

Auxora PAH data and CM5480 IND submission are expected in mid-2027



Auxora Phase 1b: PAH data expected in mid-2027

ANTICIPATED TIMELINE:		
2H 2026 — IND Submission		
Mid-2027 — Data		
POPULATION	TREATMENT	ENDPOINTS
PAH Functional class II and III • ~10 patients • Baseline PVR $\geq$5 Wood units • Stable on SOC incl. sotatercept	5-day IV course Single treatment course • IV infusion daily for 5 consecutive days	RV and LV function and hemodynamics RHC / ECHO / CMR + biomarkers • LV & RV function: contractility, strain, TAPSE, mass • Hemodynamics: PVR, mPAP, PCWP, CI • EmPHasis-10 questionnaire • NT-proBNP, Cystatin-C • Assessed at Day 5, weekly, and Day 35¹
STRATEGIC OBJECTIVES		
De-risk CM5480 in PAH and other PH Groups		Establish Auxora as a potential IV therapy in hospitalized PH

Auxora for acute pancreatitis



Acute pancreatitis: substantial unmet need and a potential \$2 billion U.S. market

High unmet need

~255K U.S. AP admissions annually¹

0 approved therapies in U.S. —
care is supportive only

~\$3.0B annual U.S. hospital costs¹
>1.1M hospital days

Potential market opportunity

~\$1.6–2.0B

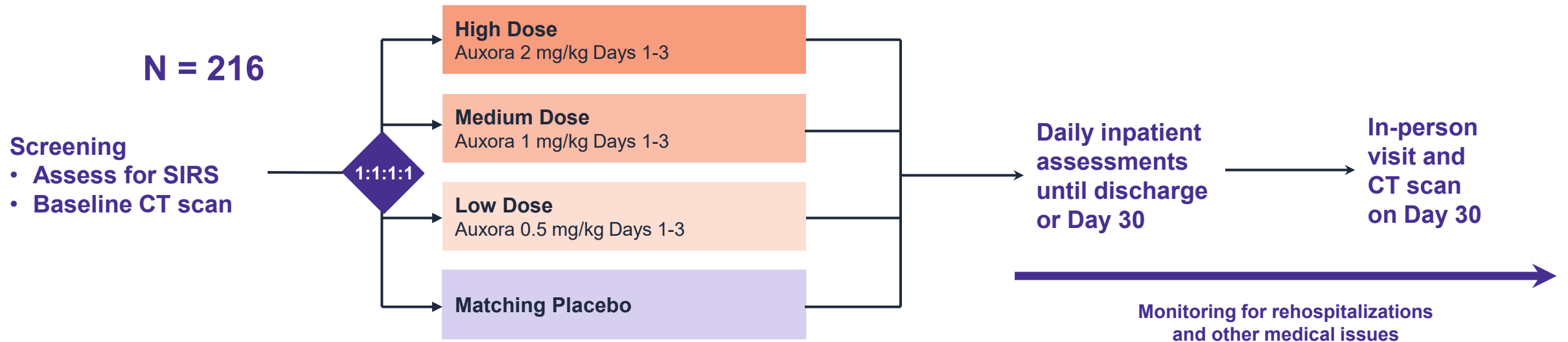
Potential annual U.S. market

~79,000 episodes × \$20,000–25,000/course

255K admissions × 62% with SIRS
× 50% with LDH >240 U/L (enrichment approach)

CARPO: completed Phase 2b trial in AP with SIRS

Dose-ranging study to establish efficacious dose, safety, target patient population and regulatory endpoints

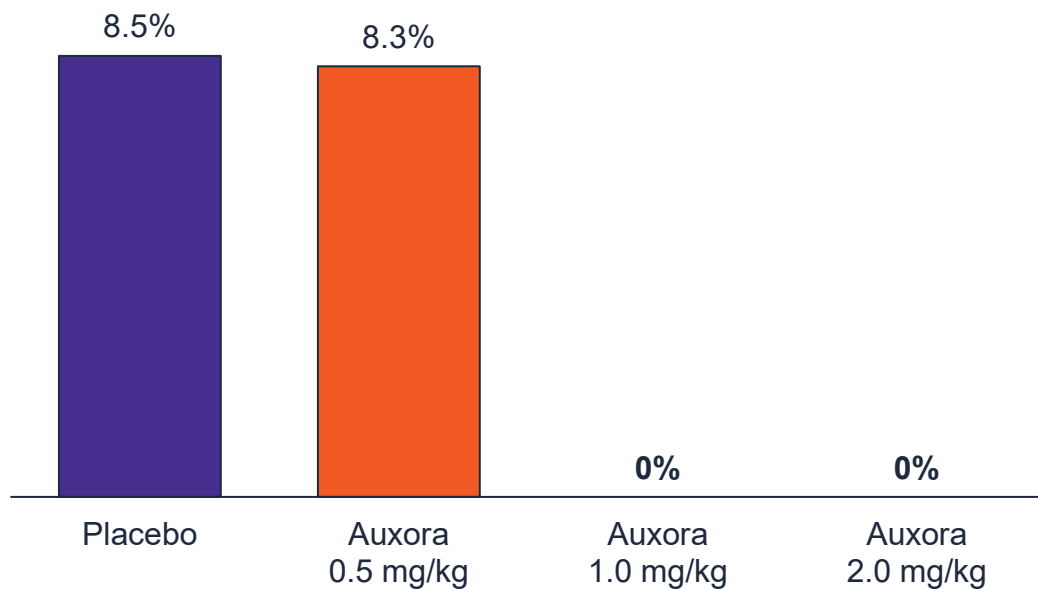


Endpoints

- Time to solid food tolerance (primary endpoint)
- Severe organ failure
- Respiratory failure
- Length of hospital stay
- Time to medically indicated discharge
- Necrosis

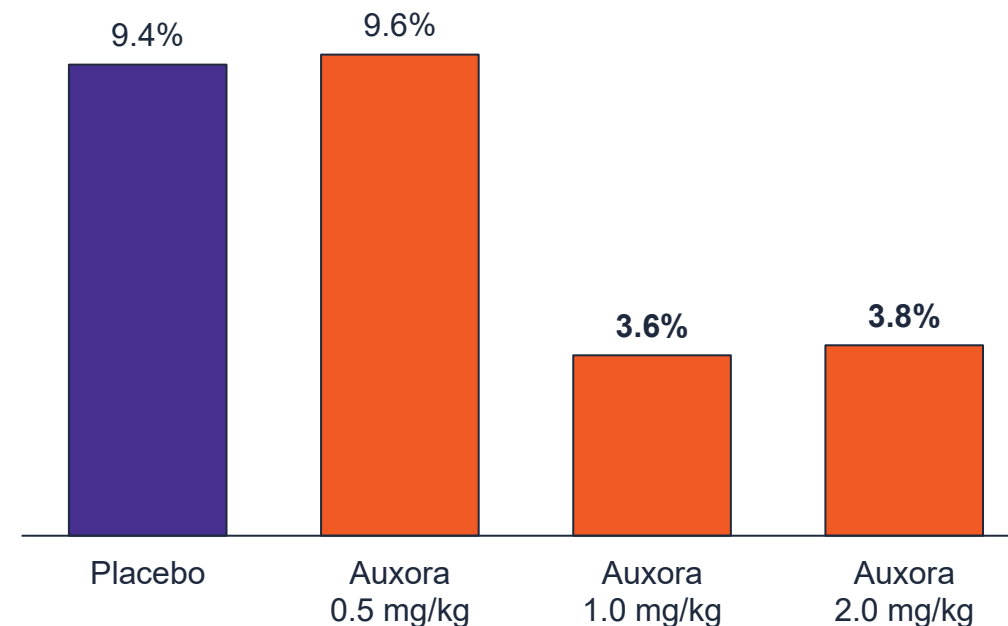
Auxora prevented new severe respiratory failure

100% reduction in new severe respiratory failure¹



Each $p < 0.05$ vs. placebo

60% reduction in severe organ failure²



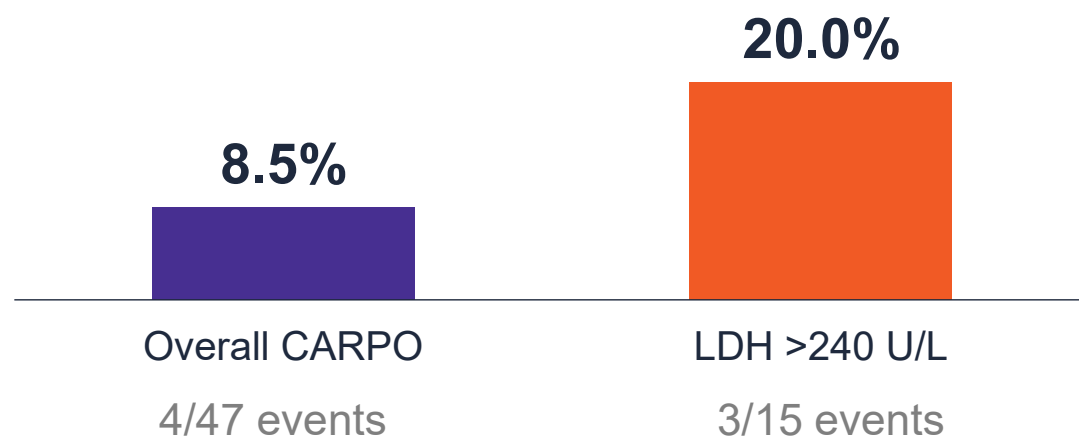
Stratified Win Ratio Composite including mortality, respiratory failure, pancreatic necrosis and recovery:

1.64 (95% CI 1.03-2.61, $p=0.037$)³

CARPO suggests a ~20% placebo event rate with LDH enrichment

CARPO analysis

New-onset severe respiratory failure on placebo



6 of 8 new severe respiratory-failure cases across all arms had LDH >240 U/L¹

Independent publication

LDH was the study's strongest individual predictor of ARDS²

111 ICU patients with hypertriglyceridemic acute pancreatitis

Closely aligned LDH cutoffs

CalciMedica

Independent study

>240 U/L

249.5 U/L

Published after CalciMedica selected its cutoff

A routine marker to enrich for patients at greater risk of severe outcomes

FDA-aligned on the planned Phase 2b design expected to inform a potential future registrational study in AP

FDA-aligned design

Following a Type C Meeting

First potential registrational endpoints in AP

Establishes the first clinical-trial endpoint framework in AP

Primary Endpoint:

New-onset Severe Respiratory Failure

Key Secondary Endpoints:

Multi-organ Failure

Time to Medically Indicated Discharge

LDH enrichment marker

Evaluating elevated LDH to identify patients at higher risk of severe respiratory failure

CARPO analysis suggests

~20%

New-onset severe respiratory failure in placebo patients with elevated LDH

Preparations for the trial are underway; initiation is subject to additional funding

Catalysts



Two differentiated candidates, with PH milestones expected in mid-2027

Pulmonary hypertension

Advancing with recent financing

Pulmonary vascular and direct cardiac protection observed preclinically

Acute pancreatitis

Clinical efficacy signal identified

Reduced new-onset severe respiratory failure & severe organ failure; FDA-aligned Phase 2b design to enrich the signal

Auxora PAH data

Mid-2027

CM5480 IND Submission

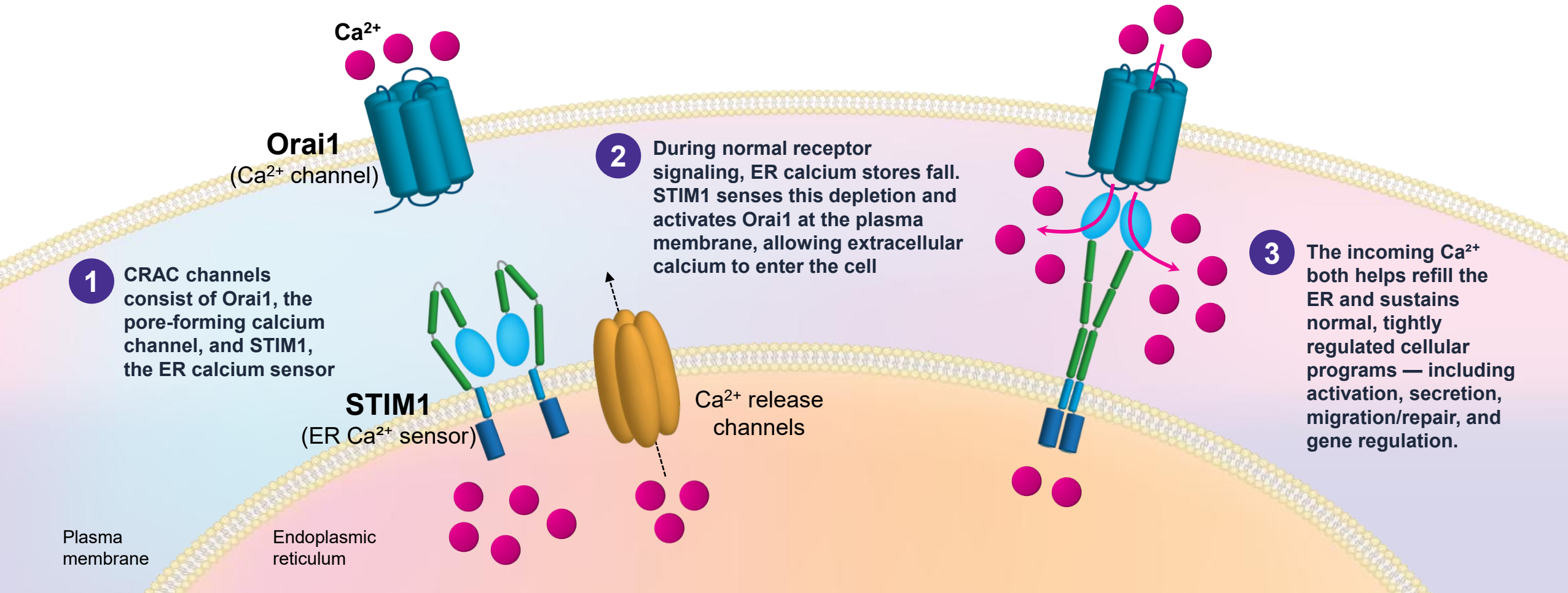
Mid-2027

Cash runway

Into 2H 2027

Appendix

CRAC channels: A store-operated calcium signaling system



In healthy cells, CRAC signaling is tightly regulated, localized, and self-limited

Mechanisms linking excess CRAC channel signaling to disease

Persistent inflammation

Repeated inflammatory signals sustain calcium release and CRAC activation

Disrupted calcium homeostasis

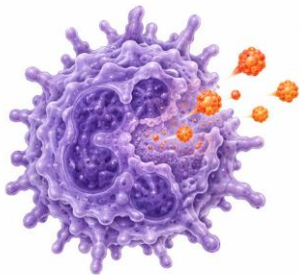
Cell injury depletes calcium stores and impairs recovery, prolonging CRAC activation

Increased channel production

Inflammatory and growth pathways increase Orai1 expression and calcium-entry capacity

Sustained Orai1-mediated Ca^{2+} entry

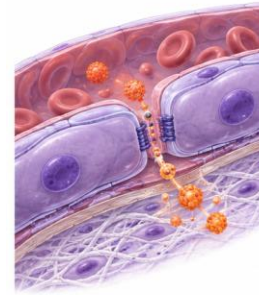
Excess calcium activates calcineurin/NFAT and NF- κ B signaling, promotes mitochondrial stress, and alters pathways controlling vascular integrity and tissue remodeling



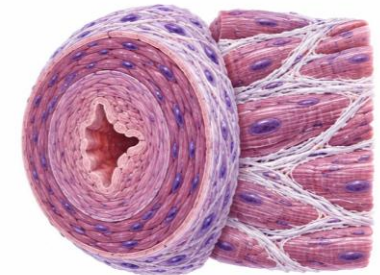
Persistent inflammation



Direct tissue injury



Vascular leakage and dysfunction



Vascular and cardiac remodeling

CRAC channel inhibition targets a pathway distinct from existing PAH therapies

Human pulmonary artery smooth muscle cells from patients with PAH

PAH mechanism	Orai1 knockdown	PAH pathway drugs		
	Pathway readout	Result	Drug / probe	Orai1 protein
Endothelin	EDNRA, EDNRB mRNA	No change	Ambrisentan Endothelin receptor antagonist	No change
NO–cGMP	PDE5 mRNA	No change	Sildenafil PDE5 inhibitor	Increased
Activin / BMP / TGF-β	BMPR2, BMPR1A, TGFB2, TGFB3 mRNA	No change	Bimagrumab Activin receptor antibody	No change
	TGFB1 mRNA	Decreased		
	SMAD1/5/9 and SMAD2/3 phosphorylation	No change		
PDGFR	Not assessed	—	Imatinib Tyrosine kinase inhibitor	No change

Supports a combination thesis with existing PAH therapies