Sten R. Sörensen
Chief Executive Officer

Webcast CS1 phase II topline results

September 30th, 2024 @2PM CET

Cereno Scientific

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Cereno is led by a group of seasoned professionals with extensive experience

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Chief Executive Officer

Dr. Björn Dahlöf
Chief Scientific Officer

Dr. Rahul Agrawal
Chief Medical Officer and Head of R&D

Eva Jagenheim
Chief Financial Officer

Nicholas Oakes
Head of Preclinical Development

Julia Fransson
Director Business Development

Source: Cereno Website

Cereno develops novel drugs to transform treatment landscape in PAH and other cardiovascular diseases

Pioneering epigenetic modulation through HDAC inhibition (HDACi) with disease-modifying potential in CVD

Pipeline portfolio:

CS1: Phase II HDACi completed, ODD in PAH

CS014: Phase I HDACi ongoing

CS585: Preclinical prostacyclin receptor agonist (PRA)

Lead program CS1 completed Phase II (USA) in PAH with positive topline data

FDA-approved CS1 Expanded Access Program initiated – Q1 2024

Cereno's global presence

HQ: Gothenburg, Sweden

US subsidiary: Boston, MA

CS1 Clinical Phase II: 10 centers in the US in collaboration with Abbott

Preclinical R&D collaboration: University of Michigan, MI

Listed on Nasdaq First North Growth Market (CRNO B)

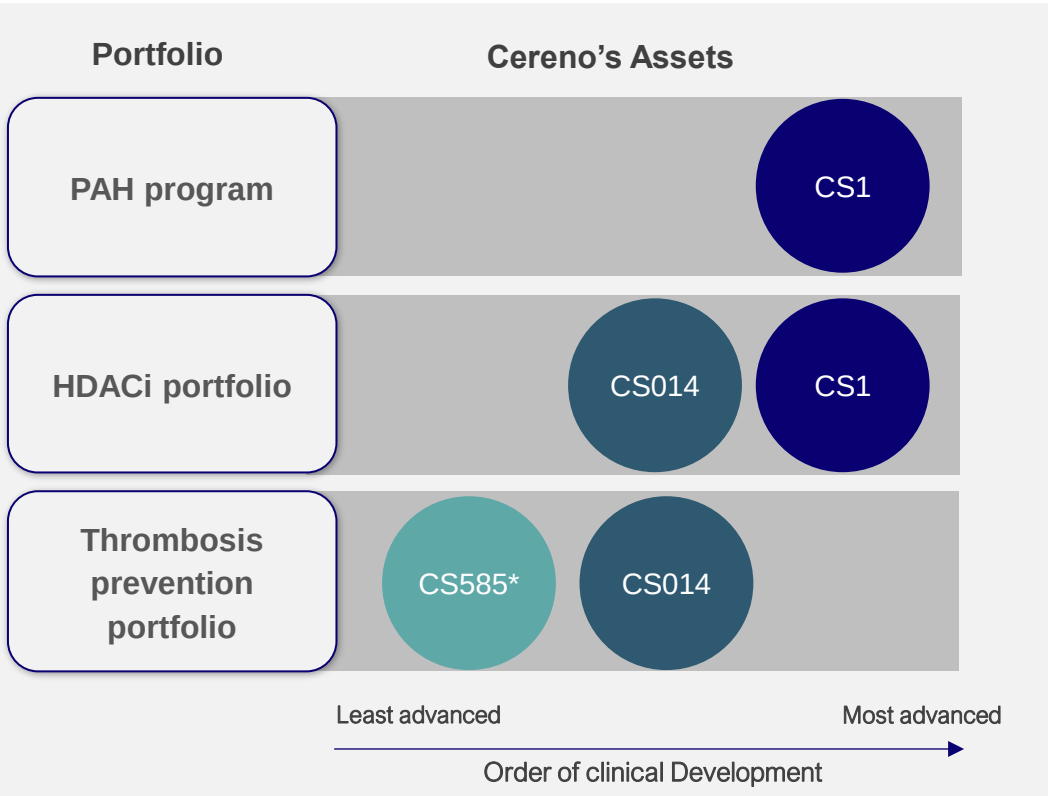
CVD – Cardiovascular Diseases, HDAC- Histone Deacetylases, ODD- Orphan Drug Designation, PAH – Pulmonary Arterial Hypertension

Cereno develops its pipeline to become attractive for development with strategic financial/pharma partners or exit through M&A

Cereno's assets can be packaged in three different portfolios for

- Co-development
- Out-licensing,
- Asset trade sale and
- M&A
- Commercialization

Program	Target	Indication	Preclinical	Phase I	Phase II	Phase III	Status
CS1	HDACi with disease-modifying potential	PAH					Phase II top-line results in Q3 2024 Expanded Access Program initiated in Q1 2024
CS014	HDACi with disease-modifying potential + Anti-thrombotic without increased risk of bleeding	Thrombosis prevention					Phase I initiated in Q2 2024
CS585	PRA oral, selective and potent	CVD					Ongoing preclinical development during 2024/25



*No Indication has been chosen for CS585. It reflects its potential.

Cereno Scientific – Latest news!

- Compassionate use: First patient dosed
- CS014 dose dependent remodeling
- Positive CS1 topline results
- Fluidda partnership to document the impact of CS1 on reverse remodeling of pulmonary arteries
- Capital Markets Day – October 17th @13.30, Posthuset – Sthlm & webcast

CS1-003: A phase IIa, prospective, randomized, multicenter trial to investigate the safety, tolerability and explore efficacy of CS1 in Pulmonary Arterial Hypertension (PAH)

NCT05224531

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Webcast – September 30th, 2024 @2PM CET

CS1 PAH phase IIa trial – Summary results

- **Primary endpoint of safety & tolerability met successfully**
- Positive impact on exploratory clinical efficacy parameters:
  - **REVEAL risk score**: 43% improved; 71% improved or stable
  - **Functional class**: 33% improved; 86% improved or stable
  - **mPAP**: 67% had sustained pressure reduction
- CS1 study data, together with preclinical information, is **consistent with reversing pathological remodeling**

→ Clear path forward - Engaging with regulatory authorities for pivotal trial

CS1 PAH phase IIa trial - Agenda

- Executive summary
- Pulmonary arterial hypertension
- PAH management goals
- Preclinical data in PAH
- Phase IIa clinical trial design and goals
- Phase IIa safety data
- Compelling signs of efficacy
- Summary of findings
- In-depth analysis
- Conclusions

Phase IIa CS1-003 trial recruitment closed July 2024

- *“On June 28, 2024, Cereno announced the decision to close patient recruitment for the Phase IIa CS1-003 trial by July 1, 2024, after the Clinical Steering Committee recommended sufficient data had been gathered to evaluate the next development steps.”*

→ 25 patients randomized and 21 patients analyzed for efficacy.

CS1 PAH trial results (1)
Primary endpoint of safety & tolerability met successfully

CS1 showed good safety & tolerability profile

Safety

- No CS1-related serious adverse events including hospitalizations/mortality
- No CS1-related changes in liver lab values
- No CS1-related clinically significant platelets decrease or bleedings

Tolerability

- CS1 was well tolerated

CS1 PAH trial results (2)
Positive impact on exploratory clinical efficacy parameters

Results:

- **REVEAL risk score:**
  - **43%** (9/21) of the patients **improved** risk score
  - **71%** (15/21) of the patients **improved** or had **stable** risk score
- **Functional Class:**
  - **33%** (7/21) of the patients **improved** functional class
  - **86%** (18/21) of the patients **improved** or had **stable** functional class
- **Mean pulmonary arterial pressure (mPAP, AUC):**
  - **67%** (14/21) of the patients had **sustained pressure reduction**

*Efficacy data from the three doses were pooled together due to therapeutic drug exposure (i.e. plasma concentration) also in the low-dose group.

Pulmonary Arterial Hypertension

- Progressive, debilitating & fatal disease
- No spontaneous improvement
- No cure, except for lung transplantation

Pulmonary Arterial Hypertension is a debilitating disease with no spontaneous improvement

- Progressive narrowing of the pulmonary vessels, ultimately leading to right heart failure and death
- Life expectancy is 2.5 years without therapy, 7.5 years with current therapy
- No cure for PAH except for lung transplantation

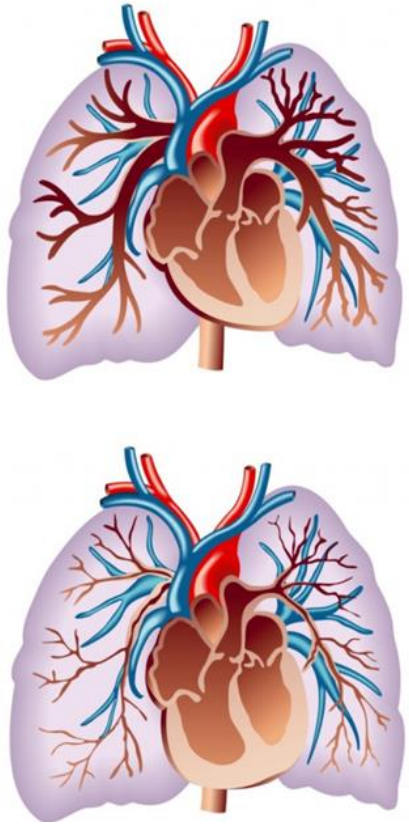
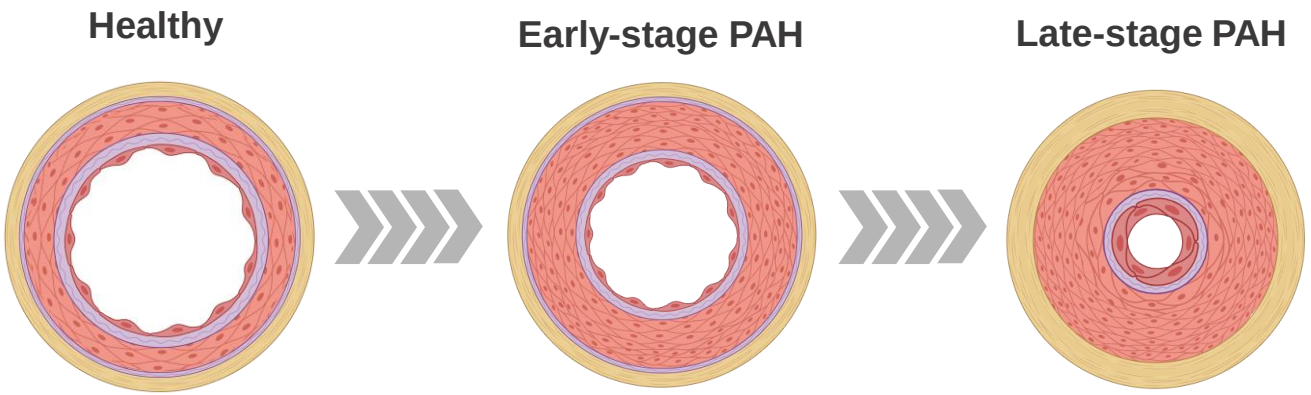


Illustration adapted from: chahalcardiovascularcentre.com/

Epigenetic mechanism through HDAC drives PAH progression

Pathological remodeling of small pulmonary arteries



PAH pathophysiology

- Endothelial dysfunction
- Inflammation
- Fibrosis
- Plexiform lesions
- Vasoconstriction
- Vascular & RV hypertrophy

Clinical consequence → Risk score and functional class deterioration

HDAC: Histone deacetylase. RV: Right ventricular.

PAH management goals

- Improvement of risk score – Measured by REVEAL
- Improvement of symptoms and physical capacity – Measured by functional class
- Improvement of hemodynamics – Measured by pulmonary pressure

→ CS1 aims to reach the management goals by reversing pathological remodeling

Goals of PAH treatment and management

- **PAH treatment goals are to improve:**
  - Risk score status (e.g., REVEAL)
  - Functional class
  - Hemodynamics (e.g., PVR, mPAP)*
  - Right ventricular function
  - Survival

PVR: Pulmonary Vascular Resistance; mPAP: mean Pulmonary Artery Pressure.

Existing treatment options are insufficient in PAH

Current therapies do not address the root cause of the disease

Key unmet needs in PAH:

- Therapies that have disease-modifying capacity
- Safer and more tolerable treatments

CS1 aims to address the unmet medical needs

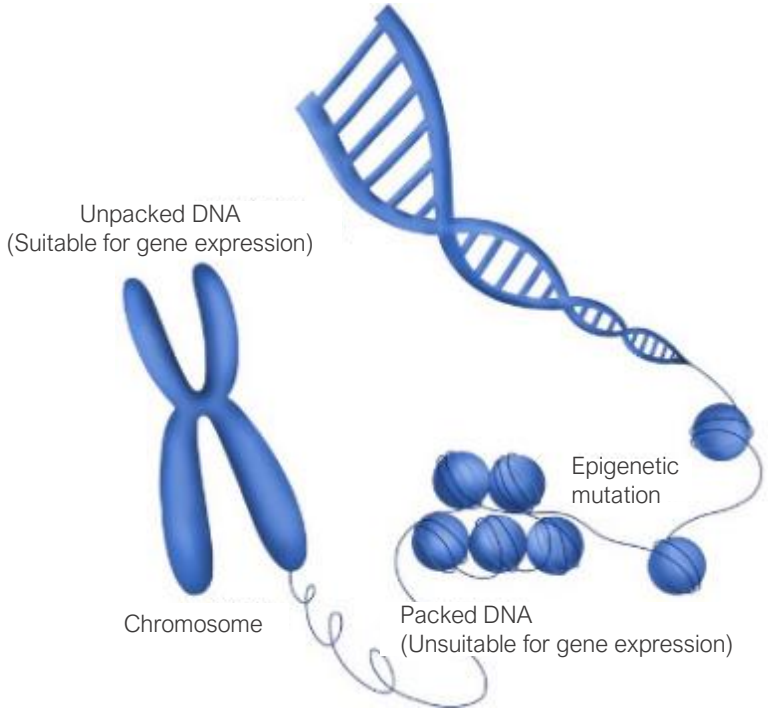
Scientific rationale for conducting CS1 phase IIa trial:

Preclinical data in PAH

- Epigenetic modulation through HDAC inhibition provides attractive proposition to address root cause of PAH disease
- Documentation of prevention and reversal of pathological remodeling
- Documentation of mPAP reduction

Cereno's HDACi portfolio is untapping the potential of epigenetic modulation in CVD

- Histone deacetylase inhibition (HDACi) plays important role in epigenetic modulation.[1-14]
- Epigenetic modulation - alteration of gene expression without altering genetic material.[1,2]



Disease-modifying elements of CVD addressed by HDACi

1. Reverse pathological remodeling
2. Anti-fibrotic
3. Anti-inflammatory
4. Pulmonary pressure reduction
5. Anti-thrombotic (fibrinolytic, anti-platelet)

THE LANCET
Healthy Longevity
Histone deacetylase inhibitors for cardiovascular conditions and healthy longevity

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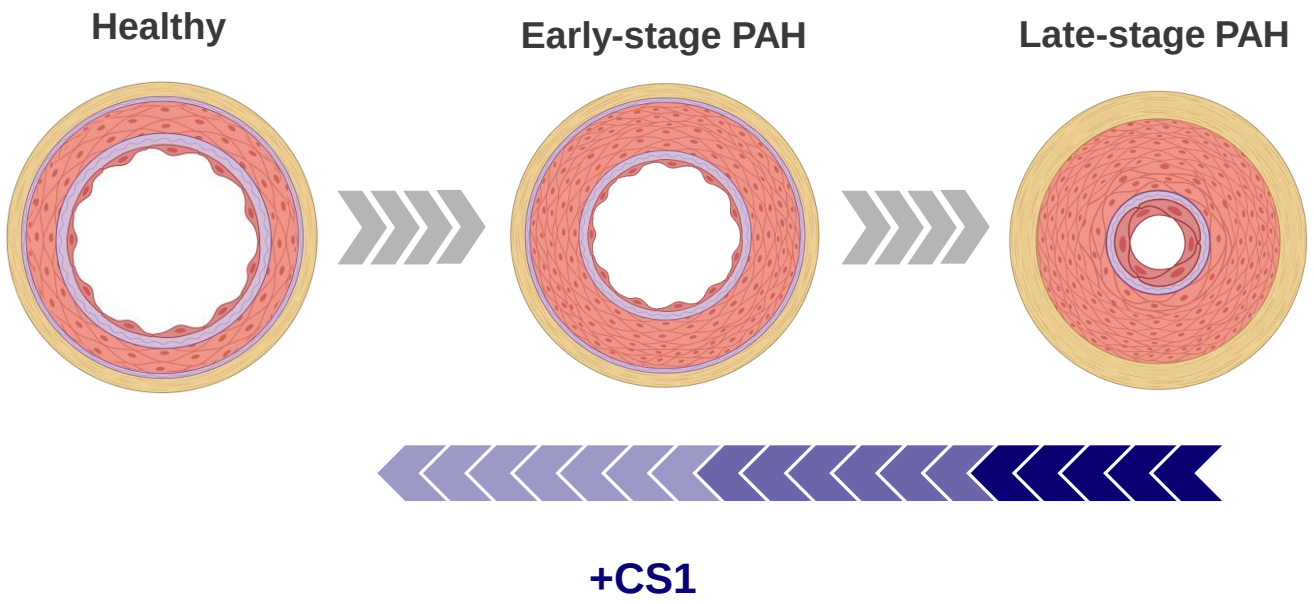
Source:1. Ferreira JP, Pitt B, and Zannad F, Lancet Healthy Longev 2021;2,e371-379; 2. Bisserier M. et.al. Vasc Biol. 2020 Jan;2(1):R17-R34. 3. Duenas-Gonzales, A., et al, 2008, Link; 4. Han, W., et al, 2021, Link; 5. Kabel, A., et al, 2016, Link; 6. Lan, B., et al, 2015, Link; 7. Zhao, L., et al, 2012, Link; 8. Cardinale, J., et al, 2010, Link; 9. Costalonga, E., et al, 2017, Link; 10. Seet, L., et al, 2019, Link; 11. Wu, S., et al, 2015, Link; 12. Larsson, P., et al, 2016, Link; 13. Saluveer, O., et al, 2014, Link; 14. Svennerholm, K., et al, 2015, Link.

CS1 tackles PAH root cause through reverse remodeling

CS1 is developed as a disease-modifying therapy



CS1 characteristics in preclinical models

- Reverse pathological remodeling
- Anti-fibrotic
- Anti-inflammatory
- Pulmonary pressure reduction
- Anti-thrombotic (fibrinolytic, anti-platelet)

Objective of reverse remodeling → Risk score and functional class improvement

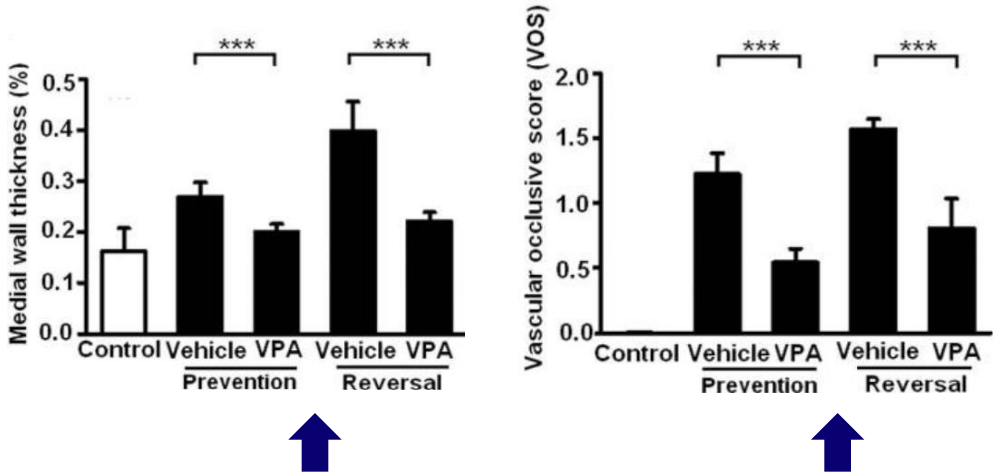
PAH preclinical data – Prevention and reversal of pathological remodeling and reduced mPAP

RESEARCH ARTICLE

Therapeutic Efficacy of Valproic Acid in a Combined Monocrotaline and Chronic Hypoxia Rat Model of Severe Pulmonary Hypertension

Beidi Lan, Emiko Hayama, Nanako Kawaguchi, Yoshiyuki Furutani, Toshio Nakanishi*

Department of Pediatric Cardiology, Tokyo Women's Medical University, Tokyo, Japan

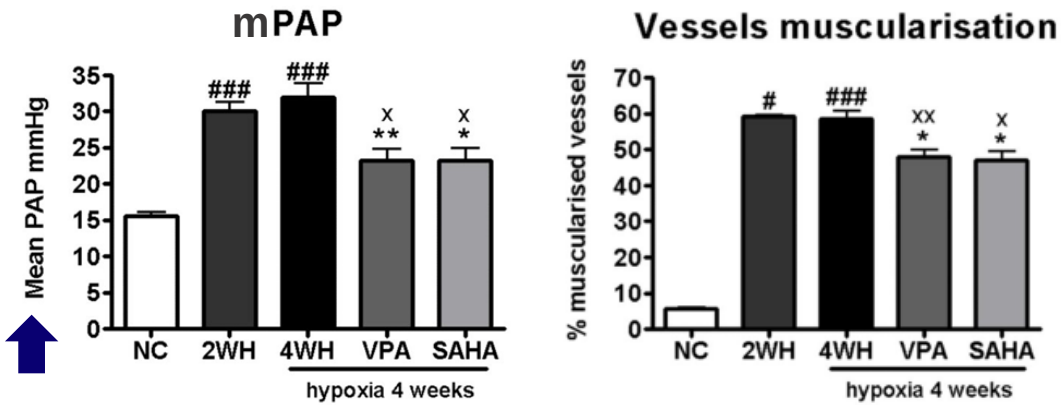


Histone deacetylation inhibition in pulmonary hypertension: therapeutic potential of valproic acid (VPA) and suberoylanilide hydroxamic acid (SAHA)

Lan Zhao, M.D PhD[1,*], Chien-Nien Chen, M.D[1], Nabil Hajji, PhD[1], Eduardo Oliver, PhD[1], Emanuele Cotroneo, PhD[1], John Wharton, PhD[1], Daren Wang, PhD[2], Min Li, PhD[2], Timothy A. McKinsey, PhD[2], Kurt R. Stenmark, M.D[2], and **Martin R. Wilkins, M.D[1]**

[1]Centre for Pharmacology and Therapeutics, Experimental Medicine, Imperial College London, Hammersmith Hospital, Du Cane Road, London W12 ONN, UK

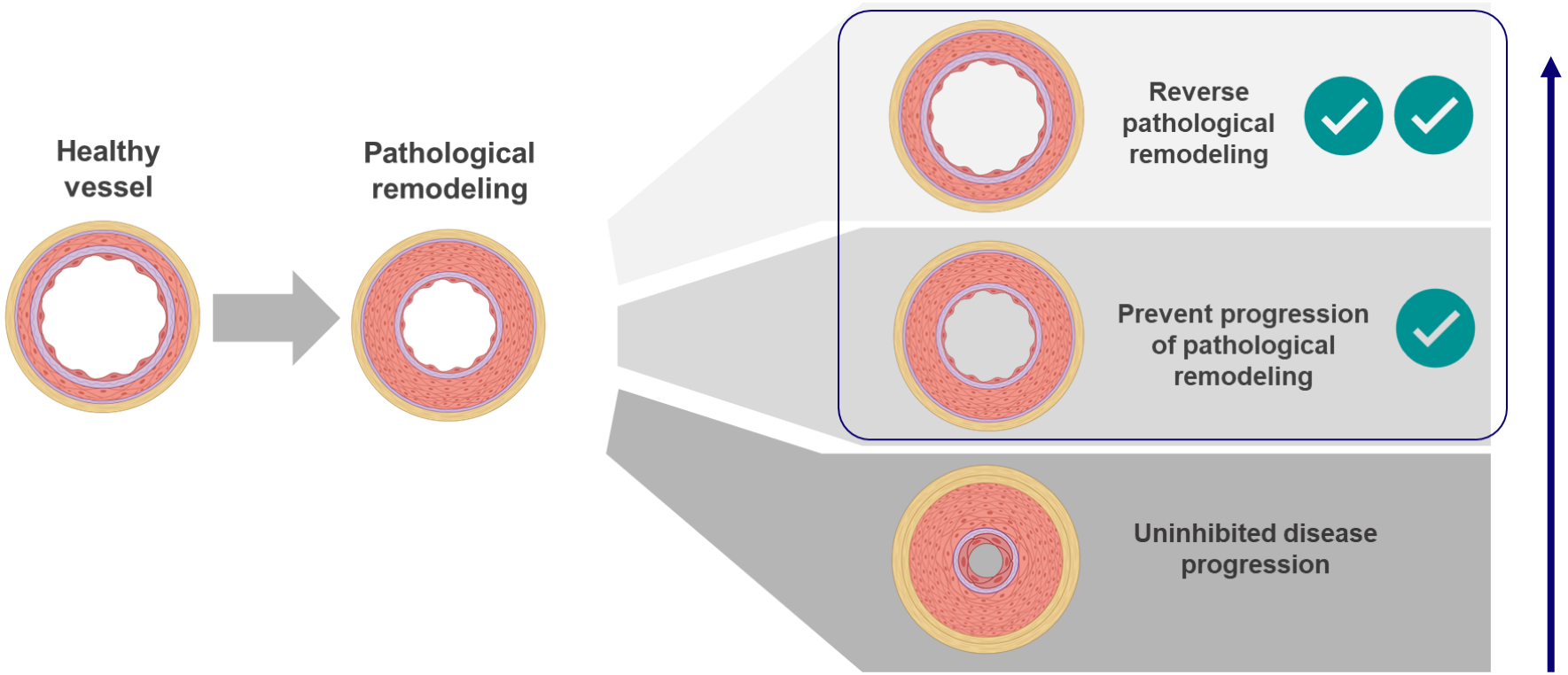
[2]Department of Pediatrics, Division of Critical Care Medicine, University of Colorado Denver, USA



PAP: Pulmonary artery pressure.

Source: Lan et al PLoS ONE, 2015; Zhao, L. et al., Circulation, 2012.

Goal of PAH therapy – Prevent and reverse pathological remodeling



Clinical goal of therapy:

- **Improvement of risk score**
- **Improvement of functional class**

CS1 PAH phase IIa clinical trial design and goals

- Primary endpoint of safety & tolerability
- Explore efficacy parameters and signals of reverse remodeling
- Prepare to pursue pivotal trial

Dr. Rahul Agrawal
Chief Medical Officer and Head of R&D

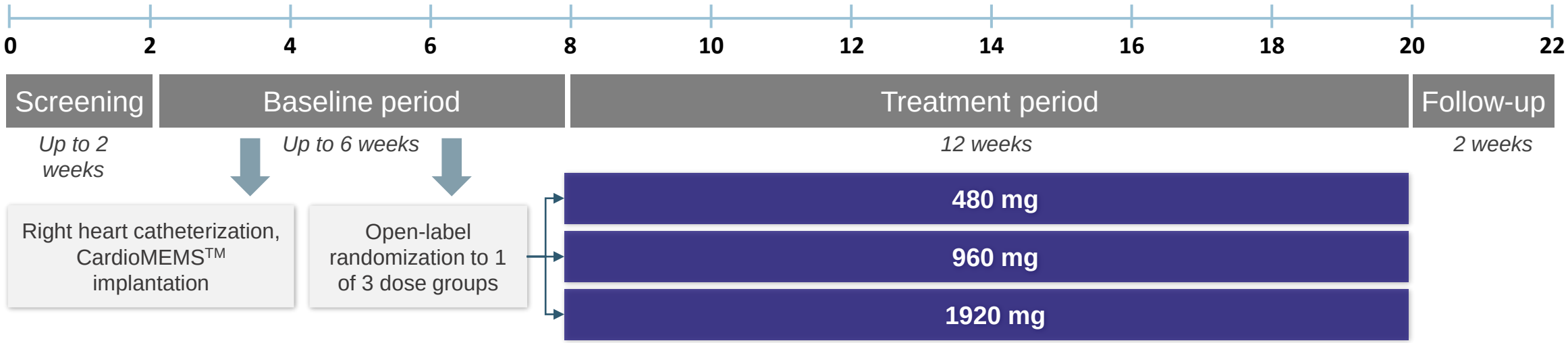
CS1 PAH phase IIa trial design

Primary endpoint: **Safety and tolerability**

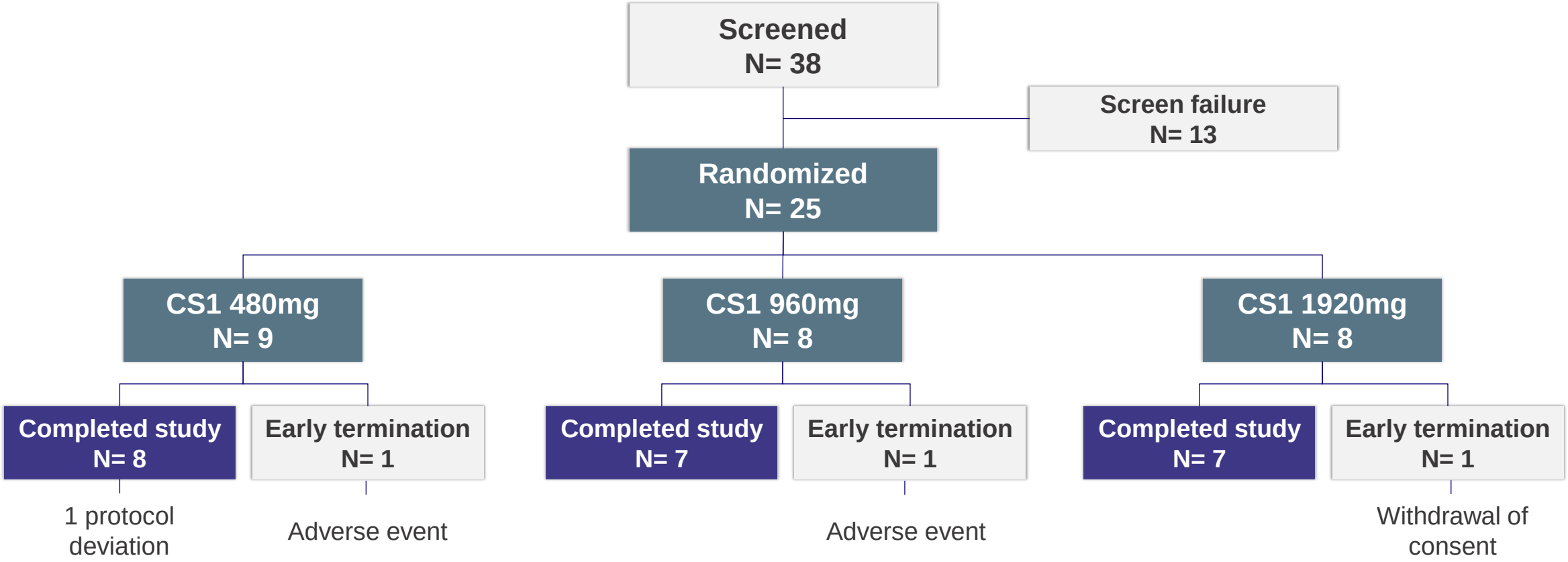
Exploratory endpoints: Including validated risk score, functional class

Implanted device for pulmonary pressures

Trial size: 25 patients 10 US clinical sites



CS1 Phase IIa trial – 25 patients randomized for safety analysis, 21 patients per protocol



Per protocol: 21 patients who completed the treatment without protocol deviation

CS1 Phase IIa trial – Demographic and clinical characteristics at baseline show representative PAH population

Variable	Overall (N=25)
Female Sex	19 (76.0%)
Race	
White	16 (64.0%)
Black or african American	4 (16.0%)
Asian	1 (4.0%)
Native american or alaska native	1 (4.0%)
Other	3 (12.0%)
NICE Clinical Classification of PAH category	
Idiopathic PAH	20 (80.0%)
Heritable PAH	1 (4.0%)
Drug or toxin-induced	3 (12.0%)
PAH associated with connective tissue disease	1 (4.0%)
NYHA/WHO Functional Class Assessment	
Class II	10 (40.0%)
Class III	15 (60.0%)
Pulmonary vascular resistance (Wood unit)	8.0±2.3
Mean Pulmonary Arterial Pressure (mmHg)	47.8±9.2

CS1 – Phase IIa safety data

- Primary endpoint successfully met
- Good safety and tolerability profile

Primary Endpoint met – No serious adverse events related to CS1

Treatment-Emergent Adverse Events (TEAEs)	CS1 480 mg QD (N=9)	CS1 960 mg QD (N=8)	CS1 1920 mg QD (N=8)	Overall (N=25)
	Subjects n (%)	Subjects n (%)	Subjects n (%)	Subjects n (%)
Any TEAEs	6 (66.7%)	5 (62.5%)	8 (100.0%)	19 (76.0%)
Serious TEAEs	2 (22.2%)	0 (0.0%)	0 (0.0%)	2 (8.0%)
Treatment-related TEAEs	2 (22.2%)	3 (37.5%)	6 (75.0%)	11 (44.0%)
Serious Treatment-related TEAEs	**0 (0.0%)**	**0 (0.0%)**	**0 (0.0%)**	**0 (0.0%)**
TEAE Leading to Study Drug Discontinuation	1 (11.1%)	1 (12.5%)	0 (0.0%)	2 (8.0%)
TEAE leading to dose reduction	0 (0.0%)	2 (25.0%)	2 (25.0%)	4 (16.0%)

Primary endpoint of safety & tolerability successfully met

CS1 showed good safety & tolerability profile

Safety

- No CS1-related serious adverse events including hospitalizations/mortality
- No CS1-related changes in liver lab values
- No CS1-related clinically significant platelets decrease or bleedings

Tolerability

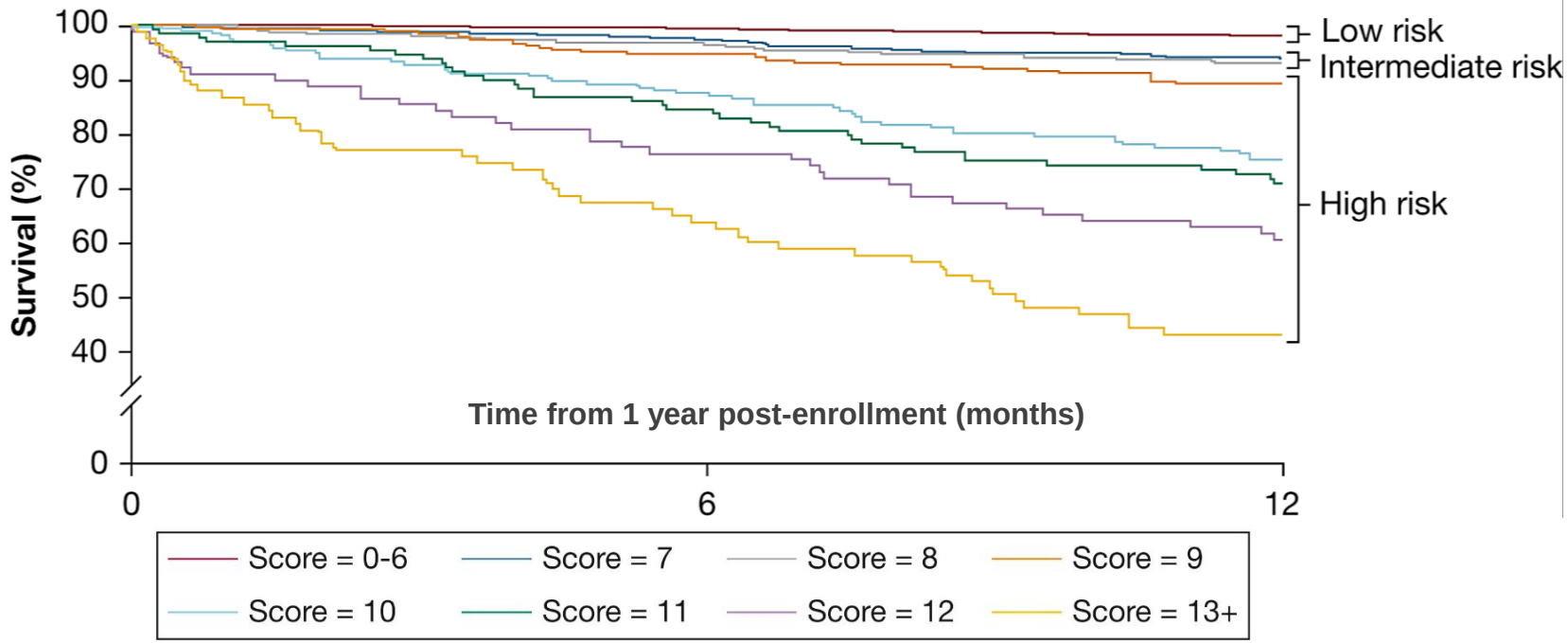
- CS1 was well tolerated

CS1 phase IIa – Compelling positive signs of efficacy

- Reduction in REVEAL risk score
- Improvement in functional class
- Reduction in mean pulmonary arterial pressure (mPAP, AUC)

REVEAL risk score predicts survival

Published data: REVEAL score has the highest prognostic power



Published data (Benza et al., 2022):

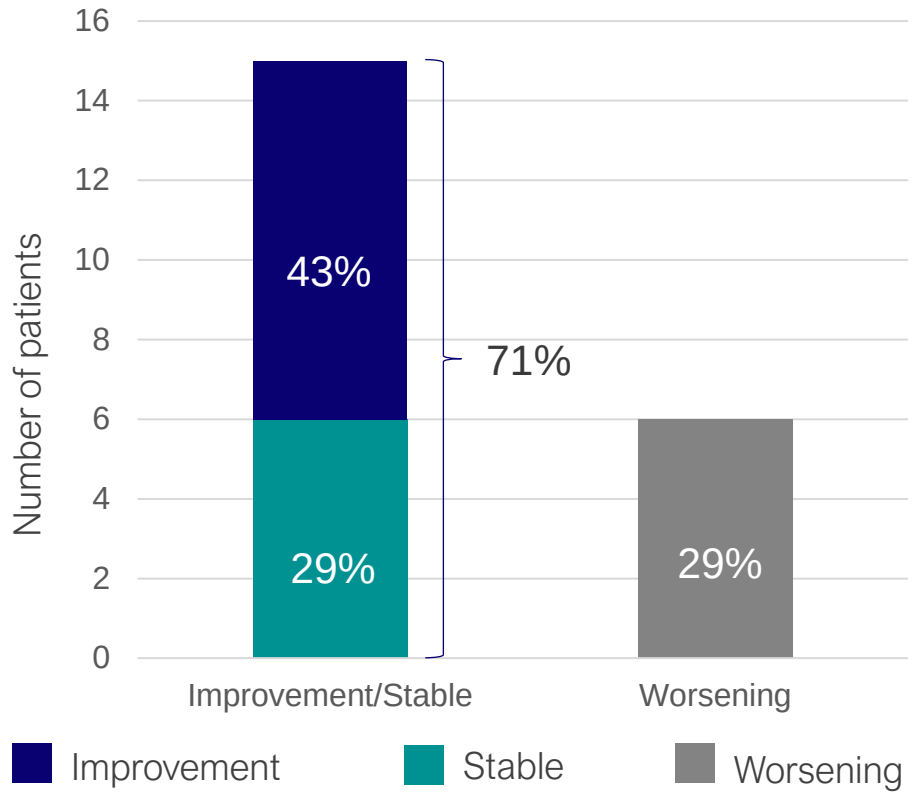
1-point reduction in risk score in 12 weeks associated with 23% reduction in relative risk of death at 12 months[1]

REVEAL risk score parameters: WHO subgroup, demographics & comorbidities, functional class, vital signs, 6MWT, BNP/NT-proBNP, echocardiogram, pulmonary function test, right heart catheterization.

Benza R., et al Chest 2019; Benza RL et al J Heart Lung Transplant 2022

CS1 phase IIa – Compelling signs of efficacy (1)
REVEAL risk score: 43% of the patients improved

REVEAL risk score change from baseline



- **43%** of the patients (9/21) improved by at least 1 point **reduction** in REVEAL risk score
- **71%** of the patients (15/21) **improved** or had **stable** REVEAL risk score

Published data (Benza et al., 2022):

1-point reduction in risk score in 12 weeks associated with 23% reduction in relative risk of death at 12 months[1]

Improvement: At least 1 point reduction in REVEAL risk score. Worsening: At least 1 point increase in risk score.

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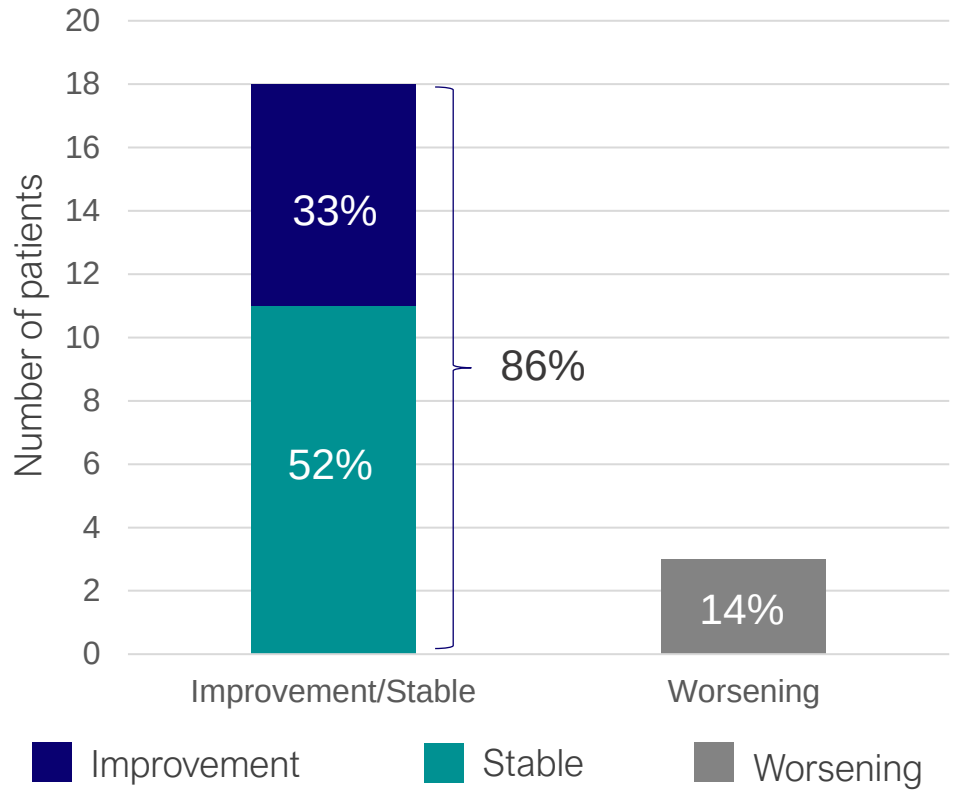
Percentages are rounded; as a result, the sum of the individual numbers does not always add up to 100%.

1. Benza RL et al J Heart Lung Transplant 2022

CS1 phase IIa – Compelling signs of efficacy (2)
Functional Class: 86% improved or had stable functional class

NYHA Functional Class change from baseline



- **33%** (7/21) of patients **improved** functional class
- **86%** (18/21) **improved** or had **stable** functional class

Functional class: Describes how severe a patient's symptoms and limitations are

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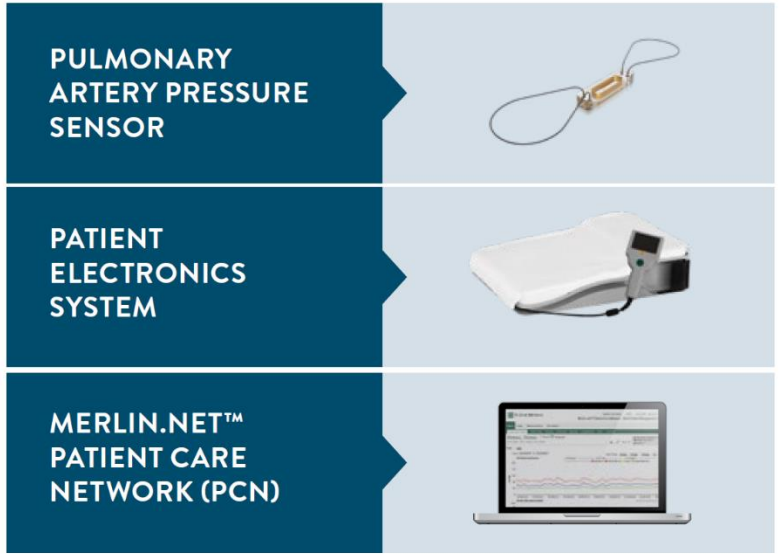
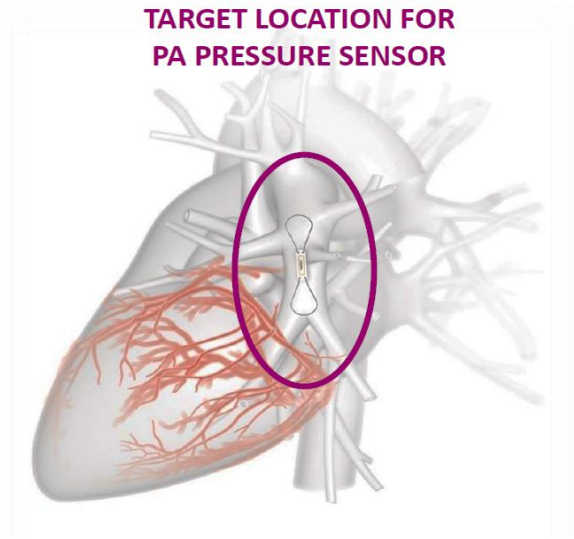
Percentages are rounded; as a result, the sum of the individual numbers does not always add up to 100%.

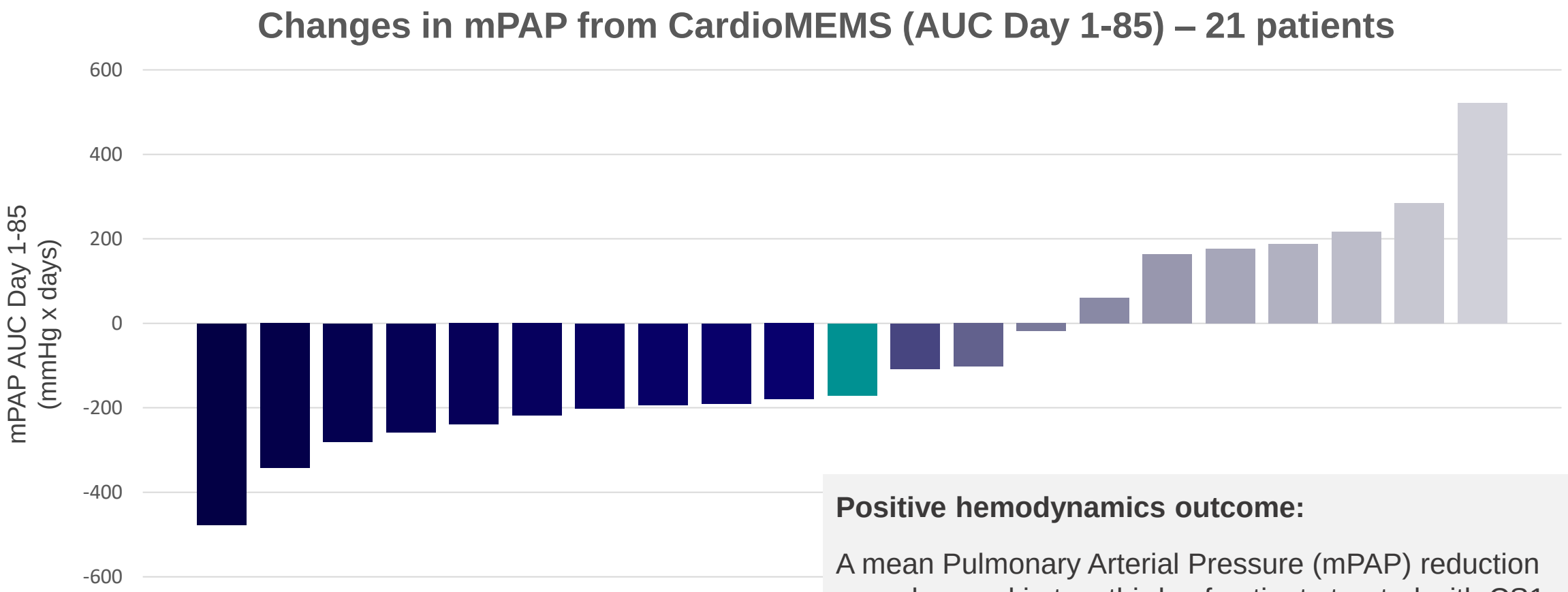
CS1 phase IIa trial – CardioMEMS permits daily non-invasive monitoring of pulmonary arterial pressure



Mean Pulmonary Arterial Pressure (mPAP) area under the curve (AUC) is a measure of the pressure burden of pulmonary pressure on the right ventricle of the heart



CS1 phase IIa trial – Compelling signs of efficacy (3)
CardioMEMS: Sustained reduction of mPAP AUC in 67% (14/21) of patients

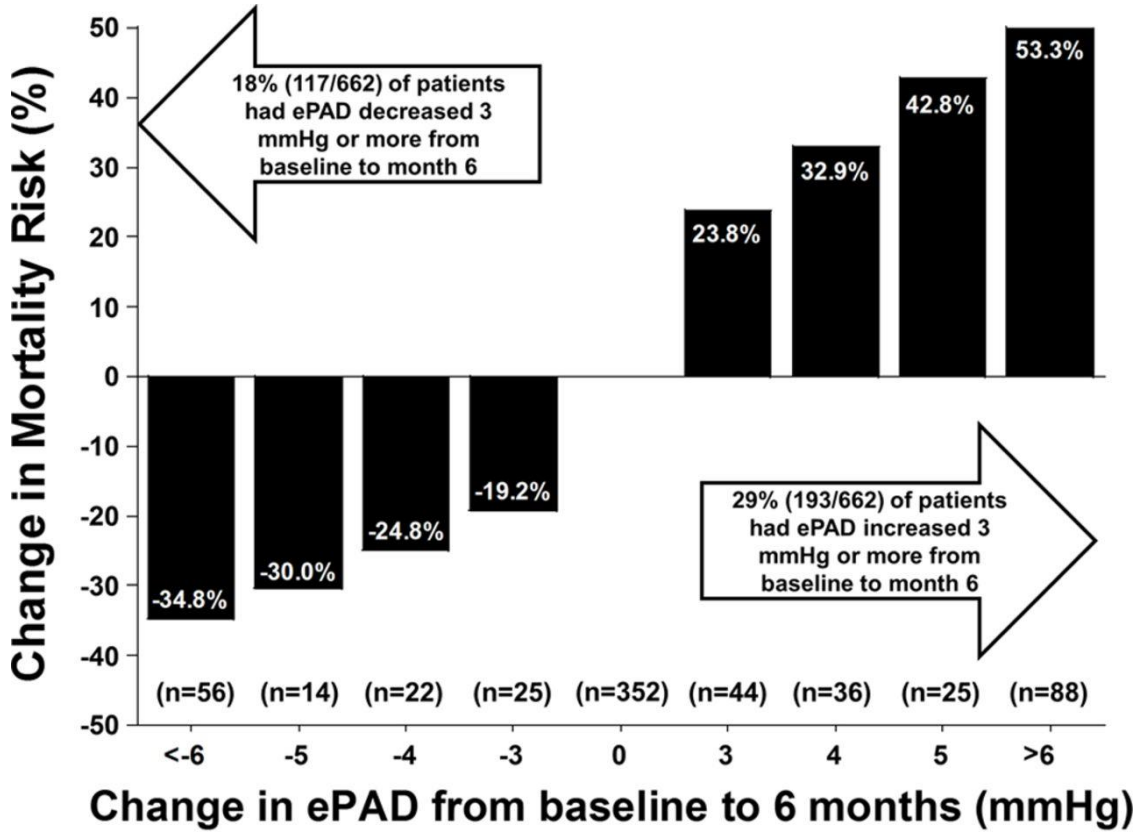


Positive hemodynamics outcome:

A mean Pulmonary Arterial Pressure (mPAP) reduction was observed in two-thirds of patients treated with CS1.

Even a small change (3-5mmHg) in pulmonary artery diastolic pressure (ePAD) is an independent predictor of mortality

Relationship between change in ePAD and mortality



Published data:

Decreased ePAD of 3, 4, or 5 mmHg from baseline to 6 months was **associated with decreased mortality risk**

Source: Zile MR, Bennett TD, El Hajj S, et al. Circ Heart Fail. 2017

CS1 PAH phase IIa trial – Summary of results

- **Primary endpoint of safety & tolerability met successfully**
- **Compelling positive impact** on exploratory clinical parameters **already over 12-week treatment**:
  - **REVEAL risk score:**
    - **43%** (9/21) of the patients **improved** risk score
    - **71%** (15/21) of the patients **improved** or had **stable** risk score
  - **Functional Class:**
    - **33%** (7/21) of the patients **improved** functional class
    - **86%** (18/21) of the patients **improved** or had **stable** functional class
  - **Mean pulmonary arterial pressure (mPAP, AUC):**
    - **67%** (14/21) of the patients had **sustained pressure reduction**

CS1 PAH phase IIa trial investigator Dr. Jason Guichard:

"I am very pleased with the positive outcomes we are seeing in our patients following treatment with CS1.

Their improvements in health and well-being are encouraging and reflect the potential effectiveness of the therapy.

This progress reaffirms my commitment to advancing treatments that can make a meaningful difference in patients' lives with PAH"

Dr. Jason Guichard, Advanced Heart Failure and Transplant Cardiology, Department of Medicine, Division of Cardiology, Prisma Health-Upstate, Assistant Professor of Medicine, University of South Carolina School of Medicine Greenville and investigator in the Phase IIa trial of CS1 in PAH.

In-depth analysis

- Recent inhouse evidence of reverse remodeling from preclinical data
- Pulmonary Vascular Resistance (PVR) in the CS1 Phase IIa trial
- Remarkable responders: evidence consistent with reverse remodeling and improved RV function in the CS1 Phase IIa trial
- Overall findings suggest lower dose range (480-960mg) optimal

Nicholas Oakes
Head of Preclinical Development

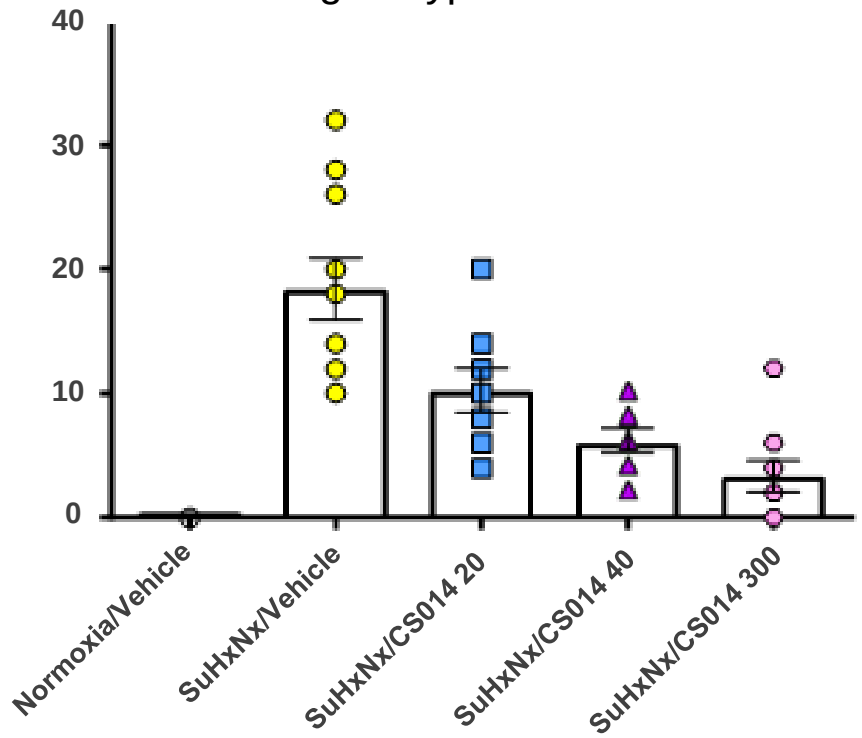
Cereno Scientific

Preclinical data – Dose-dependent reduction of plexiform lesions

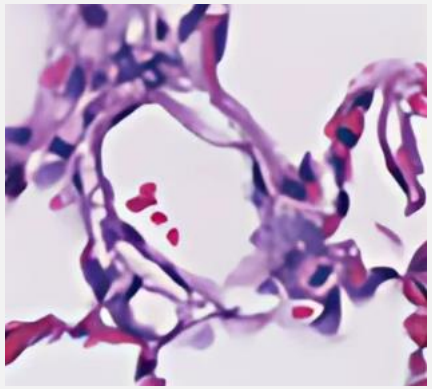
Hallmark of PAH vascular remodeling

Reduced incidence of plexiform lesions in small pulmonary arteries (<100µm) (%)

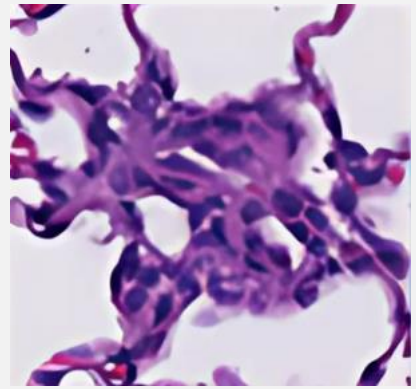
Sugen/hypoxia rat model



Healthy



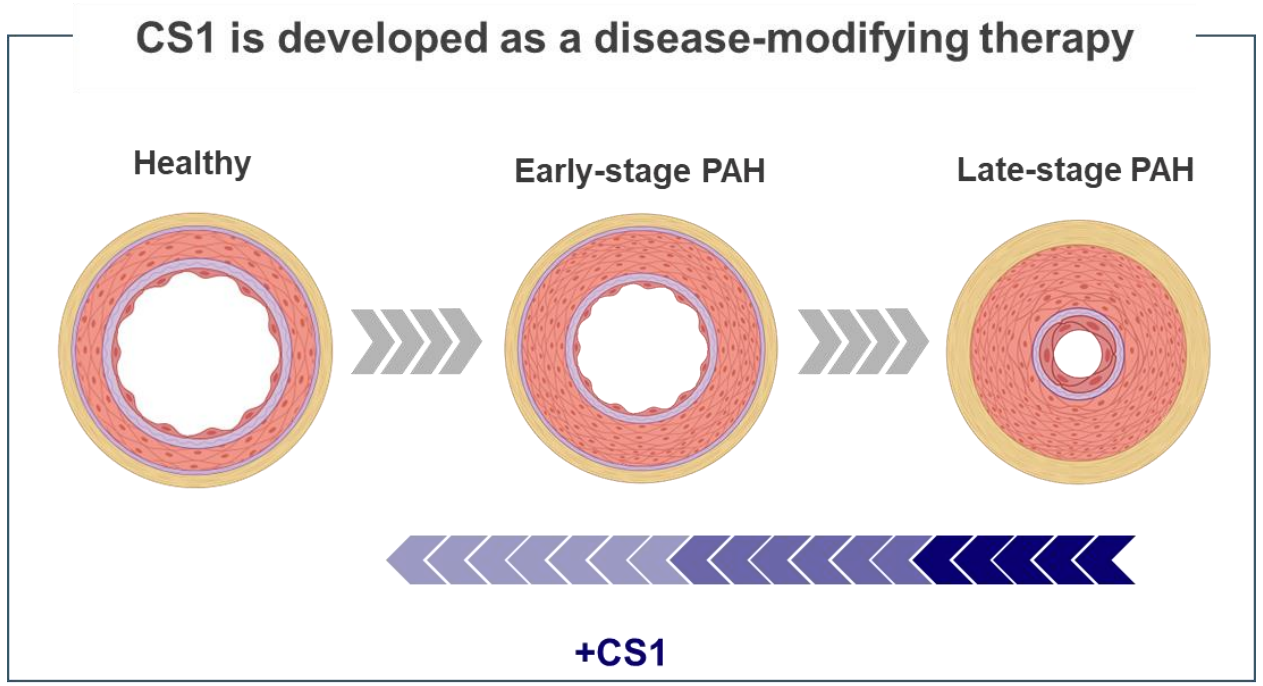
Plexiform Lesions



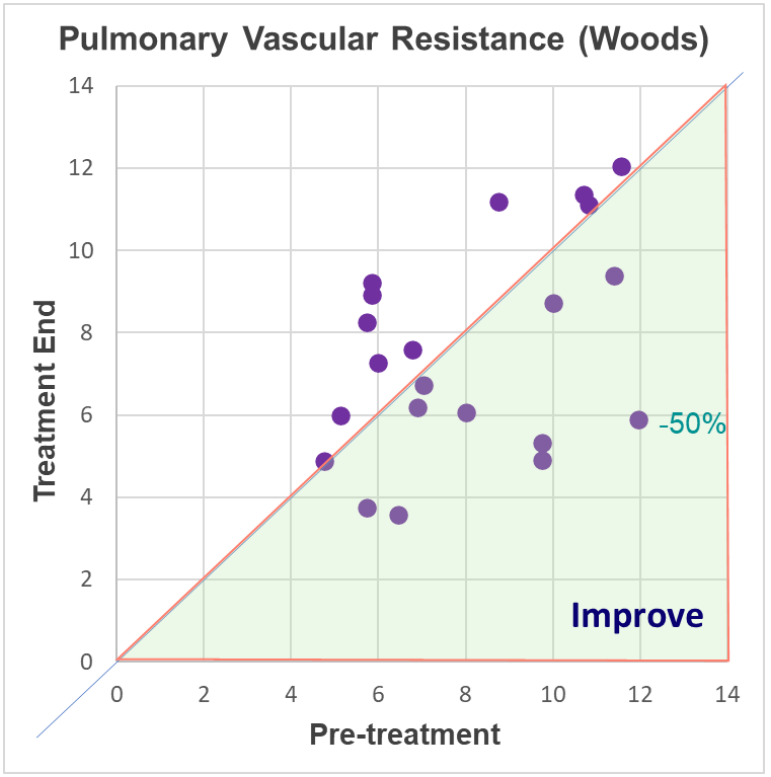
Cross sections of arteries (<100 µm)

CS014 also reduced small artery-associated fibrosis

CS1 phase IIa trial – Reversal of vascular remodeling is expected to result in a reduction of PVR



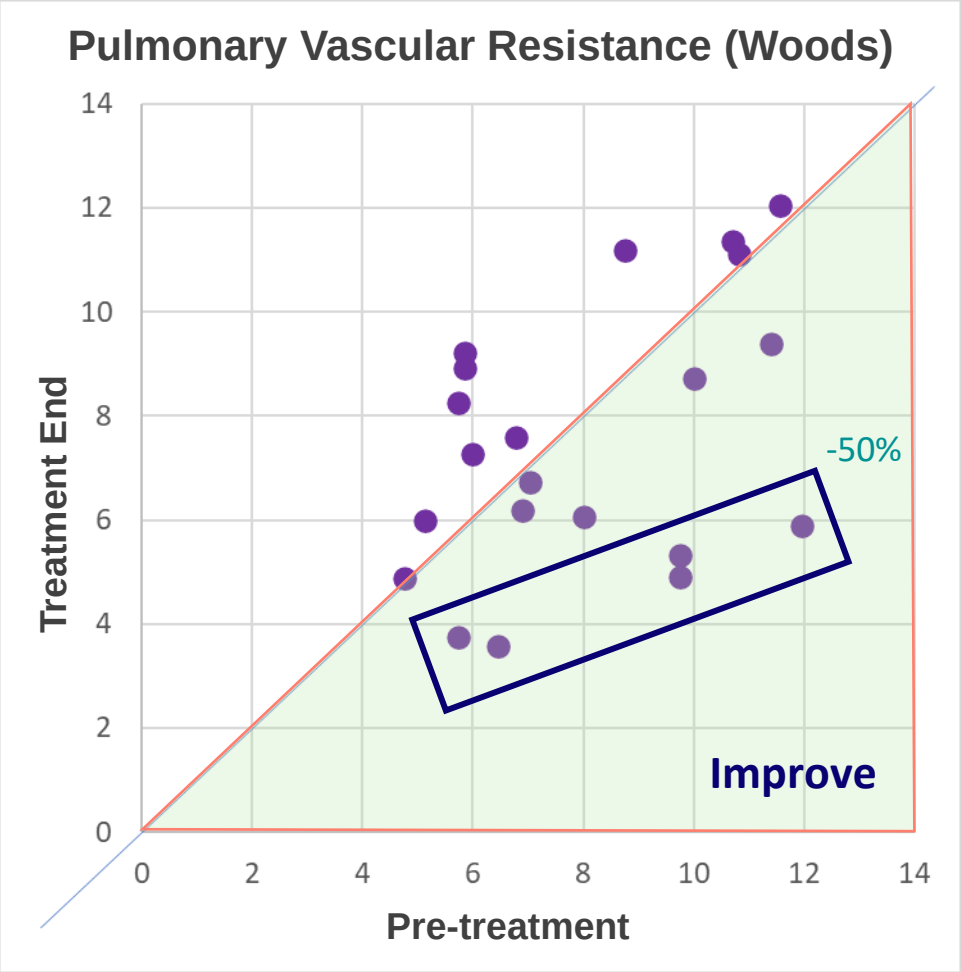
PVR is the hemodynamic parameter that best reflects reverse remodeling



CS1 lowers PVR in several patients from baseline to end of treatment

Patients with improved PVR after 12 weeks therapy

CS1 phase IIa trial – Remarkable responders in PVR mostly in low dose group



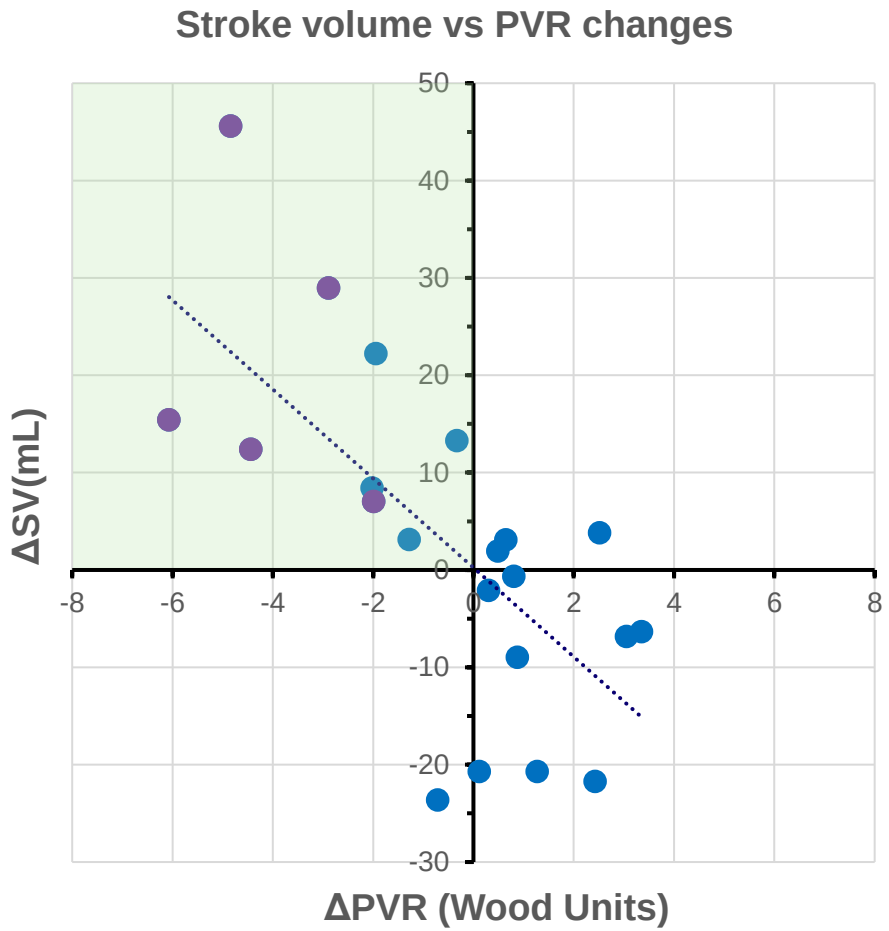
PVR remarkable responders:

- **Reduction in PVR of >30%**
- 5 patients identified, range 35-51% reduction, mean 45%
- 4/5 of the PVR-responders are in low-dose group

PVR reductions of this magnitude have an extremely low probability of occurring by chance*

*Analysis based on STELLAR study placebo data

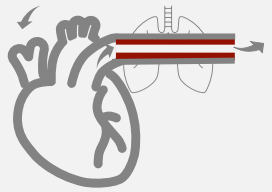
CS1 phase IIa trial – Increased stroke volume associated with reduced PVR in the remarkable responder group



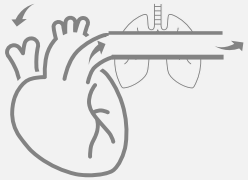
PVR remarkable responders:

- Reduction in PVR and increase in stroke volume
- Clinically meaningful increase in SV: > 10 mL[1]

Impact of PVR on heart function:



Remodeling results in increased PVR and worsened right heart function



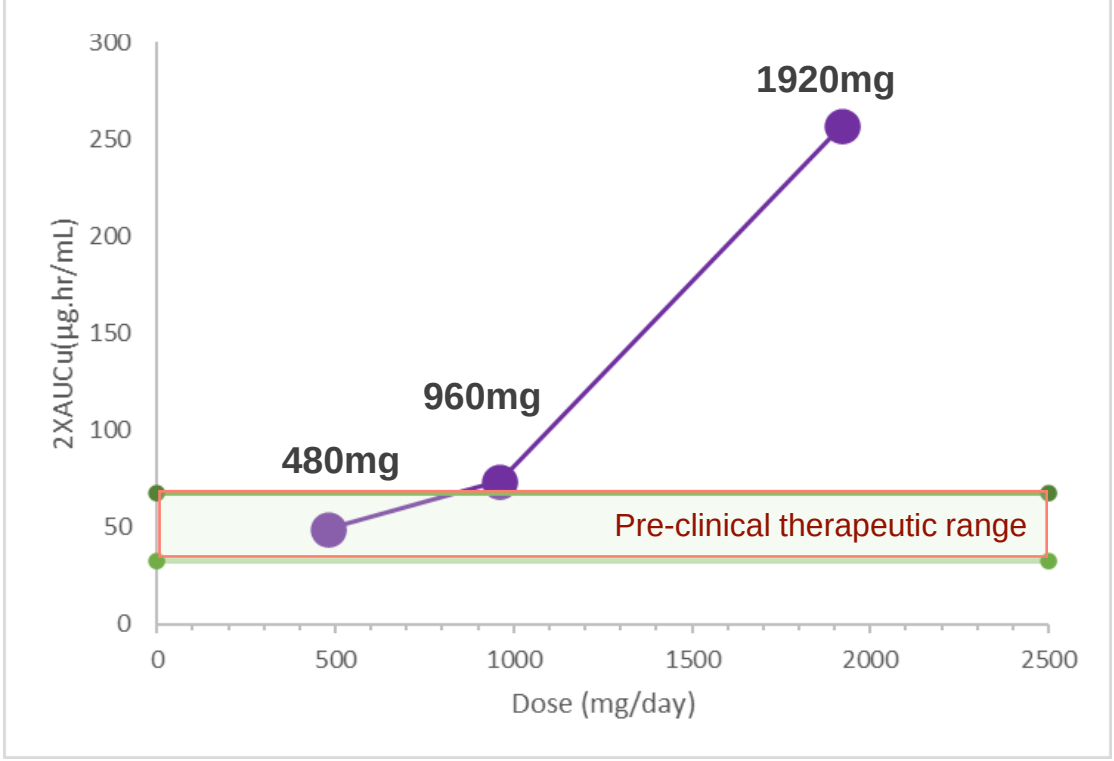
CS1 reduces PVR and improves right heart function

Note: This slide has been updated following the webinar on the 30th of September in order to correct an error.

1. Wolferen, Chest 139(5), 2011

Overall findings suggest lower dose range (480-960mg) optimal

VPA in CS1 patients vs CS014 in preclinical model estimated unbound exposures



Preclinical therapeutic range of CS014 is consistent with phase IIa clinical response

- CS014 is an equipotent analog of the active ingredient of CS1
- Maximally effective preclinical unbound exposures correspond to low-mid dose levels in our phase IIa clinical trial
- Majority of remarkable PVR-responders are in low-dose group

Summary of in-depth analysis – Evidence of vascular remodeling

- Recently obtained preclinical data with CS014 demonstrates:
  - **Dose-dependent reversal of remodeling** of lung resistance arteries in a PAH model
  - **Dose-dependent reduction of plexiform lesions**
  - **Reduction of fibrosis** associated with pulmonary arteries
  - Maximal efficacy at equivalent exposures to CS1 Ph IIa trial lower-dose range
- **24% (5/21)** of the patients **responded to CS1** with **remarkably large reductions in PVR** consistent with the proposed reversal of pathological vascular remodeling
- These **reductions in PVR (35-51%, mean 45%)** were strongly associated with **robust increases in right ventricular stroke volume**

Conclusions

- Positive topline results of Phase IIa trial with lead candidate CS1 in PAH

Sten R. Sörensen
Chief Executive Officer

Cereno Scientific

CS1 PAH phase IIa trial – Summary results

- **Primary endpoint of safety & tolerability met successfully**
- Positive impact on exploratory clinical efficacy parameters:
  - **REVEAL risk score**: 43% improved; 71% improved or stable
  - **Functional class**: 33% improved; 86% improved or stable
  - **mPAP**: 67% had sustained pressure reduction
- CS1 study data, together with preclinical information, is **consistent with reversing pathological remodeling**

→ Clear path forward - Engaging with regulatory authorities for pivotal trial

CS1 PAH – Path forward

- Completing the analysis of the trial
- Fluidda study
- Compassionate use and long-term data
- Regulatory path

Cereno Scientific

CS1 – Clear path forward to develop asset as a disease-modifying therapy for PAH

- Complete analysis of the PAH trial
- Regulatory path
  - Engaging with regulatory authorities for pivotal trial
- Compassionate use
  - Expanded access program (compassionate use) ongoing and will provide long-term data
- Fluidda partnership to document the impact of CS1 on reverse remodeling of pulmonary arteries

Cereno signs agreement with Fluidda to evaluate the impact of CS1 on reverse remodeling in a clinical setting

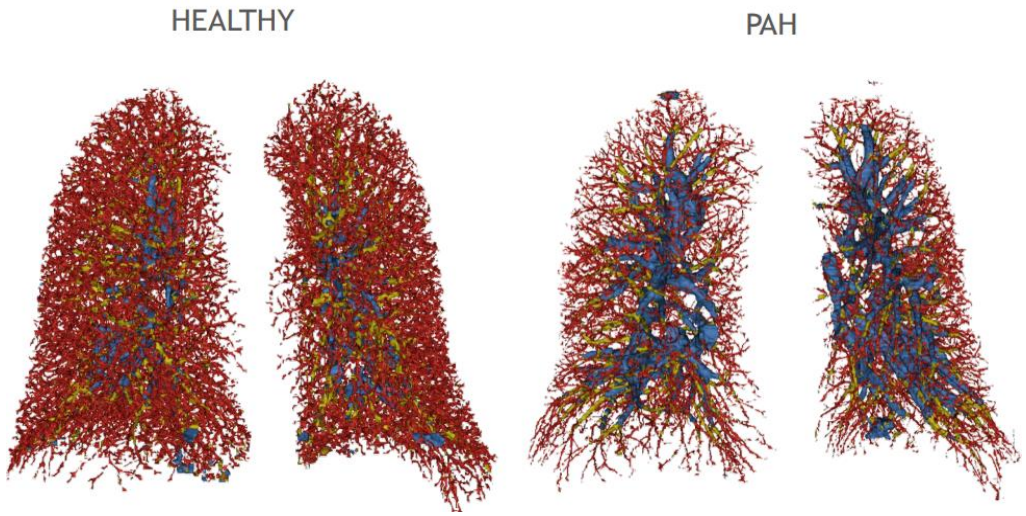
Press release
September 30, 2024

Cereno Scientific

Cereno Scientific signs agreement with Fluidda, to evaluate the impact of its HDAC inhibitor CS1 on reverse remodeling of pulmonary vessels in patients with PAH

Cereno Scientific (Nasdaq First North: CRNO B), a pioneering biotech developing innovative treatments for rare and common cardiovascular disease, today announced that the Company has signed an agreement with medical technology company Fluidda on Respiratory Imaging solutions, with the aim to visualize signs of reverse remodeling of lead drug candidate CS1 in Pulmonary Arterial Hypertension (PAH) in a clinical setting.

"We have a vision to develop new therapies which address the root cause of cardiovascular disease as we believe this will provide high value to patients with respect to improvement of quality of life and survival. On the heels of our increasing knowledge of impact of reverse remodeling capacity of HDACi from our preclinical program CS014, together with our top line results from our Phase IIa study with our lead HDACi program CS1, I am excited to announce our partnership with Fluidda. This collaboration will allow Cereno to use Fluidda's cutting-edge technology to visualize CS1's ability for long-term reverse remodeling in PAH patients," **said Sten R. Sörensen, CEO, Cereno Scientific**



Source: FLUIDDA company presentation

Cereno Scientific – Path forward

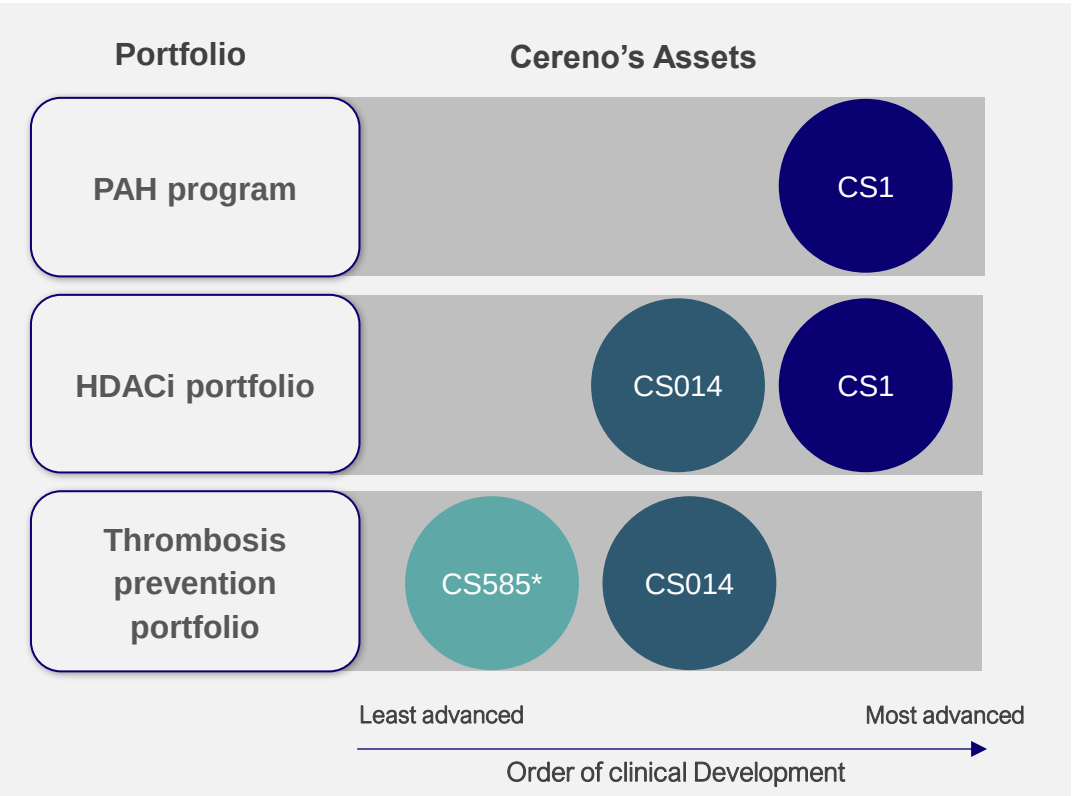
- Portfolio development
- Key upcoming milestones

Cereno develops its pipeline to become attractive for development with strategic financial/pharma partners or exit through M&A

Cereno's assets can be packaged in three different portfolios for

- Co-development
- Out-licensing,
- Asset trade sale and
- M&A
- Commercialization

Program	Target	Indication	Preclinical	Phase I	Phase II	Phase III	Status
CS1	HDACi with disease-modifying potential	PAH					Phase II top-line results in Q3 2024 Expanded Access Program initiated in Q1 2024
CS014	HDACi with disease-modifying potential + Anti-thrombotic without increased risk of bleeding	Thrombosis prevention					Phase I initiated in Q2 2024
CS585	PRA oral, selective and potent	CVD					Ongoing preclinical development during 2024/25



*No Indication has been chosen for CS585. It reflects its potential.

Cereno Scientific – Key upcoming milestones 2025

- CS1 compassionate use program long term (H1)
- CS1 FDA pivotal study approval (H1)
- Reverse remodeling Fluidda (H1)
- CS014 phase I completion (H1)
- CS014 phase II approval (H2)
- Continued partnering activities

Webcast CS1 phase II topline results – Q&A

Q&A sessions:

- Webcast CS1 phase II topline results – Q&A
- Send questions to info@cerenoscientific.com
- Capital Markets Day – October 17th @13.30

Cereno Scientific

Cereno Scientific develops innovative treatments for rare and common cardiovascular disease. The lead drug candidate, CS1, is an HDAC (histone deacetylase) inhibitor that acts as an epigenetic modulator with pressure-reducing, reverse-remodeling, anti-inflammatory, anti-fibrotic and anti-thrombotic properties. A Phase IIa trial evaluating CS1's safety, tolerability, and exploratory efficacy in patients with the rare disease pulmonary arterial hypertension (PAH) demonstrated that CS1 was safe, well-tolerated and showed a positive impact on exploratory clinical efficacy parameters. CS1 study data, together with preclinical information, is consistent with reversing pathological remodeling. A collaboration agreement with global healthcare company Abbott allowed Cereno to use their cutting-edge technology CardioMEMS HF System in the trial. Since January 2024, we are delighted that the FDA´s Expanded Access Program will enable patients with PAH, a serious life-threatening disease condition, to gain access to CS1 where no comparable alternative therapy options are available. Cereno's pipeline comprises two additional programs in development through research collaborations with the University of Michigan CS014 is a new chemical entity with a multi-fold mechanism of action as an epigenetic modulator – regulating platelet activity, fibrinolysis, and clot stability for the prevention of thrombosis without an increased risk of bleeding as documented in preclinical trials. The drug candidate has also demonstrated a favorable profile in preclinical models of other cardiovascular diseases, such as PAH, with reverse remodeling of pulmonary arterial vessels and effects on vascular fibrosis. On 28th of June, 2024, Cereno initiated a first-in-human Phase I trial of CS014. Preclinical candidate CS585 is an oral, highly potent and selective prostacyclin (IP) receptor agonist that has demonstrated the potential to significantly improve disease mechanisms relevant to cardiovascular disease. While CS585 has not yet been assigned a specific indication for clinical development, preclinical data indicates that it could potentially be used in indications like Pulmonary Hypertension and thrombosis prevention without increased risk of bleeding. CS585 was in-licensed from the University of Michigan in 2023. The Company is headquartered in GoCo Health Innovation City, in Gothenburg, Sweden, and has a US subsidiary; Cereno Scientific Inc. Based in Kendall Square, Boston, Massachusetts, US. Cereno is listed on the Nasdaq First North (CRNO B). The Certified Adviser is Carnegie Investment Bank AB, certifiedadviser@carnegie.se. More information is on www.cerenoscientific.com.