Webcast presenting Phase IIa trial results for CS1 in PAH

March 4, 2025

Cereno Scientific

Today's webcast

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Agenda

- Cereno Scientific in short
- The HDACi platform
- PAH and CS1
- Phase IIa trial results and next steps
- Future outlook
- Q&A session
  - Analysts: Rx Securities & Edison
  - Written questions from audience

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Cereno Scientific is enhancing and extending lives of people living with rare cardiovascular and pulmonary diseases

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Cereno pioneers epigenetic modulating HDACis to transform the treatment of rare cardiovascular and pulmonary diseases

PIPELINE

CS1

Class I HDACi in Pulmonary Arterial Hypertension (PAH)

- ODD in PAH in US & EU
- Phase IIa met primary endpoint and demonstrated ability of reverse remodeling effects:
  - Ongoing expanded access program (EAP) and sub-study utilizing imaging technology by Fluidda

CS014

Class I HDACi in Idiopathic Pulmonary Fibrosis (IPF)

- Phase I study: Single Ascending Dose (SAD) part 1 of 2 concluded without safety concerns; Multiple Ascending Dose (MAD) part ongoing; top-line in mid-2025

CS585

Preclinical prostacyclin receptor agonist (PRA)

Safe

Well-tolerated

Disease-modifying

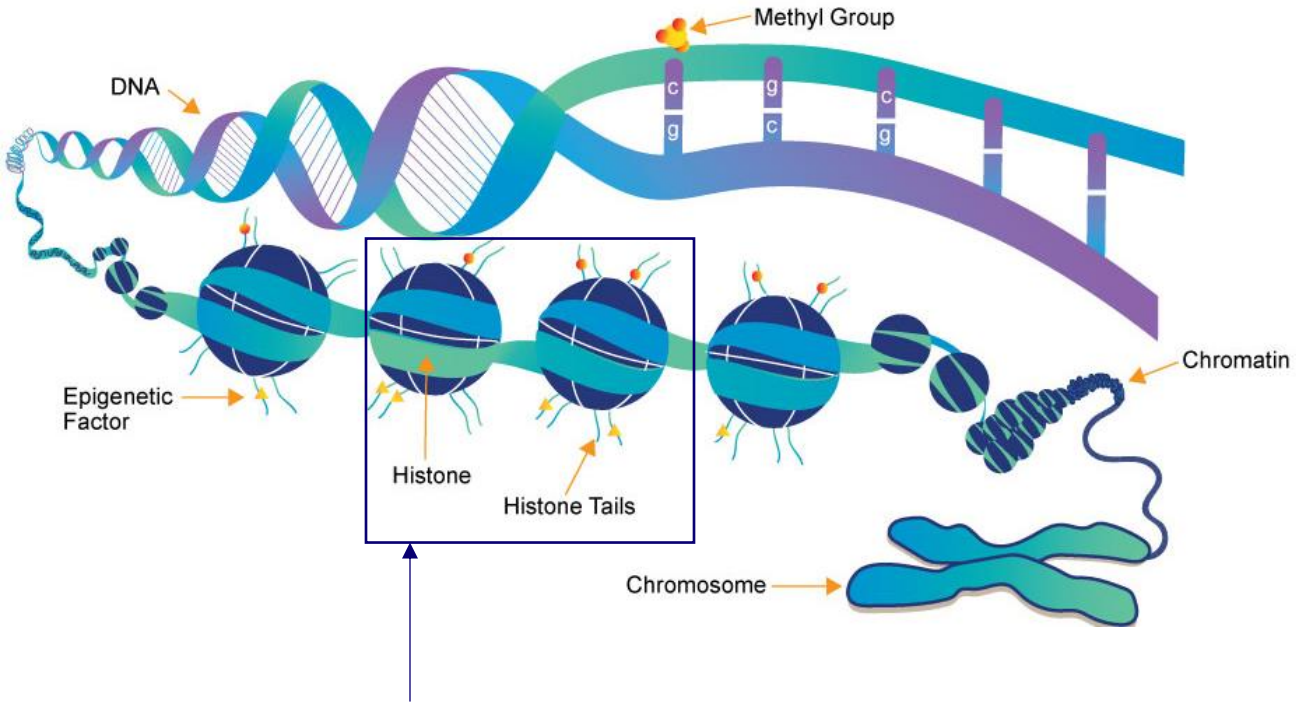
CVD – Cardiovascular Diseases, HDAC- Histone Deacetylases, ODD- Orphan Drug Designation, PAH – Pulmonary Arterial Hypertension, IPF – Idiopathic pulmonary fibrosis


What if…

Epigenetic modulation via HDAC inhibition (HDACi)

HDACs via epigenetic mechanisms play an important role in cardiovascular and pulmonary diseases

- Epigenetic modulation - alteration of gene expression without altering genetic material[1,2]
- Epigenetic modulation plays key role in aging, cardiovascular and pulmonary diseases
- **Histone deacetylase (HDACs) contributes to cardiovascular (CVD) and pulmonary diseases via epigenetic modulation[1-14]**



Histone modification by acetylation : binding of acetyl groups to histone tails alters the extent to which DNA is wrapped around histones and availability of genes in DNA to be transcribed

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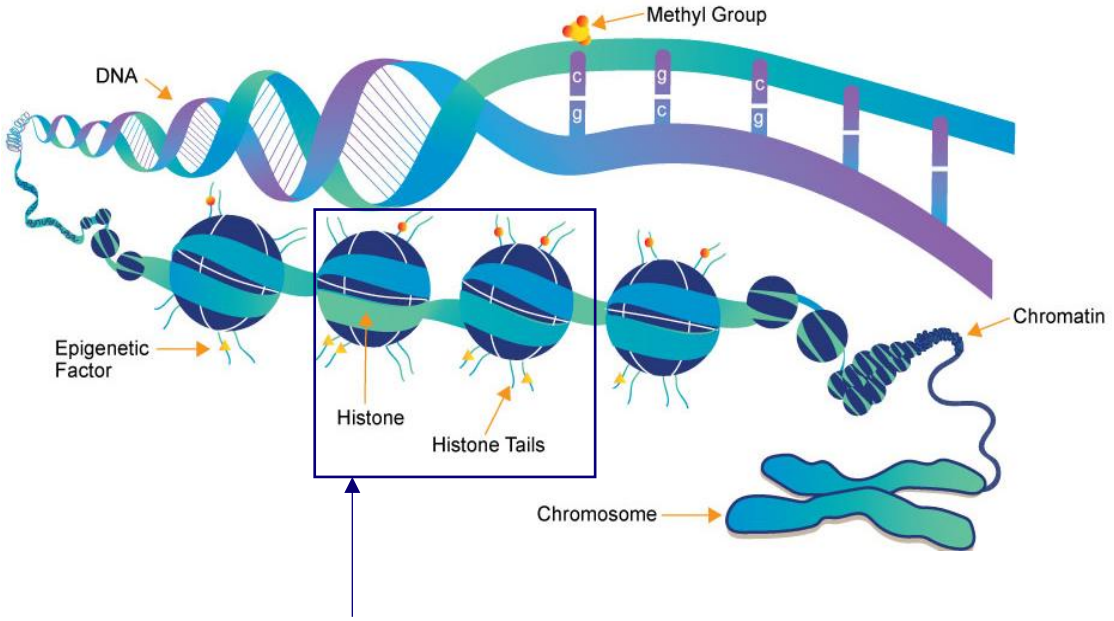
Source:1. Ferreira JP, Pitt B, and Zannad F, Lancet Healthy Longev 2021;2,e371-379; 2. Bisserier M. et.al. Vasc Biol. 2020 Jan;2(1):R17-R34. 3. Duenas-Gonzales, A., et al, 2008, Link; 4. Han, W., et al, 2021, Link; 5. Kabel, A., et al, 2016, Link; 6. Lan, B., et al, 2015, Link; 7. Zhao, L., et al, 2012, 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.
Picture source: https://www.whatisepigenetics.com/fundamentals/

Cereno's HDAC inhibition portfolio untaps the potential of epigenetic modulation in cardiovascular and pulmonary diseases



Histone modification by acetylation

Disease-modifying elements of cardiovascular and pulmonary diseases addressed by HDACi

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

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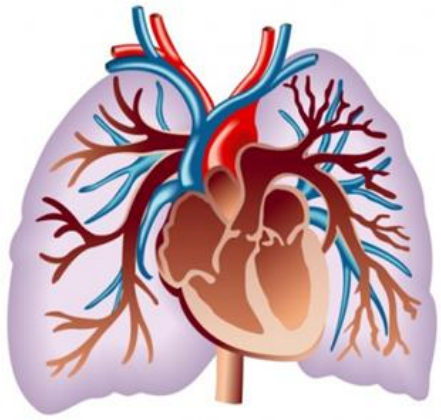
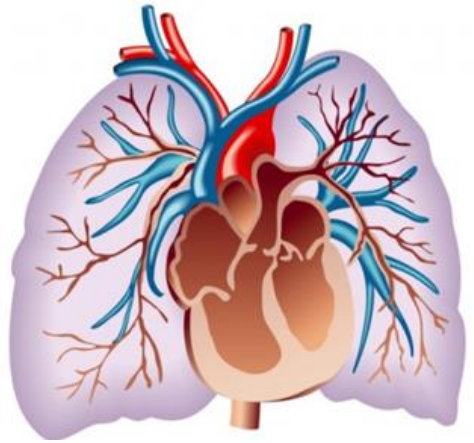
Pulmonary arterial hypertension (PAH) is a fatal disease with no spontaneous improvement

- Progressive narrowing and pathological remodeling of the pulmonary vessels, ultimately leading to right heart failure and death
- Median age at diagnosis is 62 years
- Majority are females
- Life expectancy is 2.5 years without therapy, 7.5 years with current therapy
- No cure for PAH except for lung transplantation





PAH DISEASE PROGRESSION

Healthy heart and lungs

↓

Pathological vascular remodeling

↓

Pulmonary arterial hypertension

↓

Right heart failure

Illustration adapted from: chahalcardiovascularcentre.com/

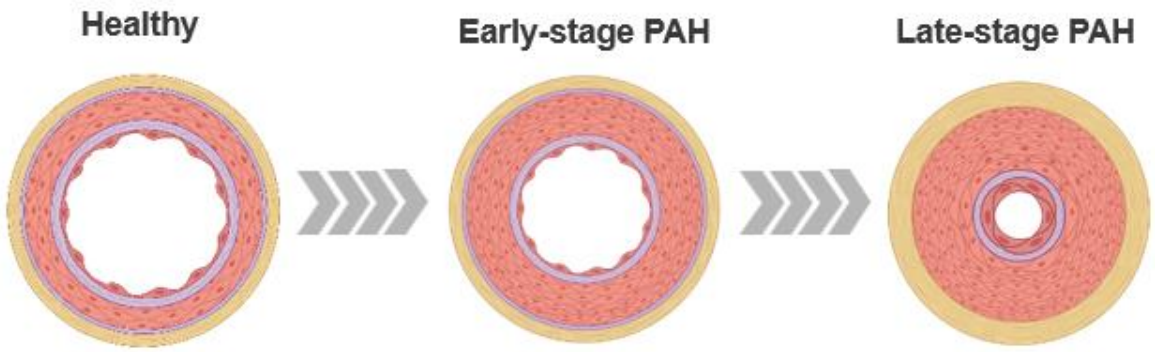
HDACs via epigenetic changes linked to PAH pathophysiology and progression

HDAC class I (Histones 1,2,3 & 8) are of high relevance in PAH pathophysiology

PAH pathophysiology

- Pathological vascular remodeling
- Endothelial dysfunction
- Plexiform lesions
- Inflammation
- Fibrosis
- Vasoconstriction
- Right ventricular (RV) hypertrophy

Pathological remodeling of pulmonary vasculature



1. K. Jankowski et.al. Biomedicines 2024, 12(1), 10; 2. M. Bisserier et.al. Rev. Cardiovasc. Med. 2020, 21(2), 163–179

CS1

First-in-class HDACi with reverse remodeling potential in PAH

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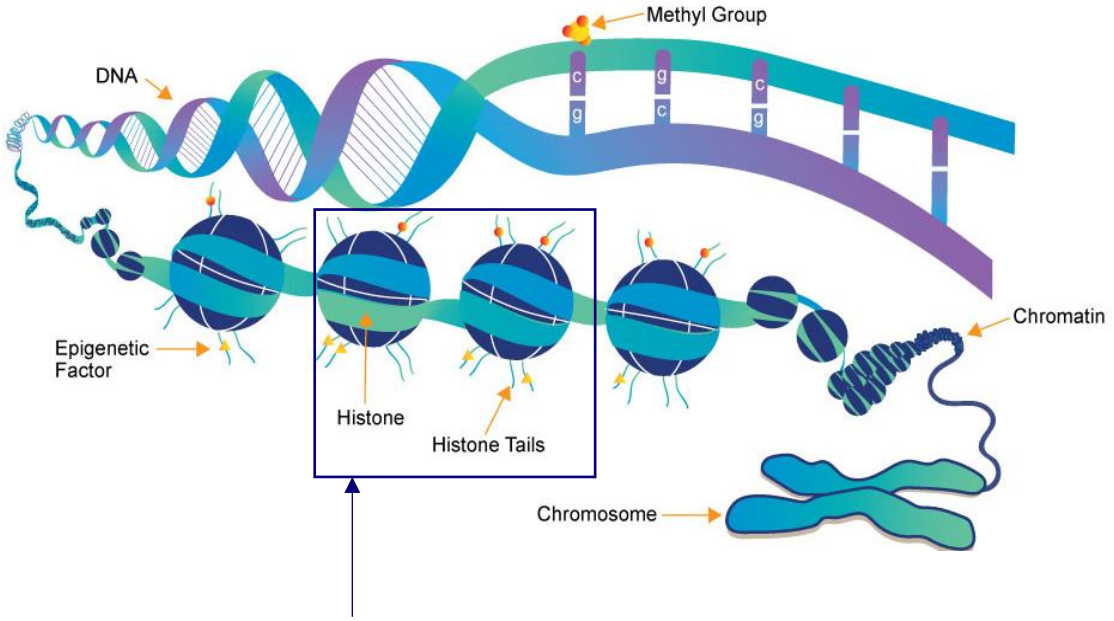
Lead drug candidate CS1

- Proprietary reformulation of valproic acid (VPA)
- Class I HDAC inhibitor that acts via epigenetic modulation

Cereno's HDAC inhibition portfolio untaps the potential of epigenetic modulation in cardiovascular and pulmonary diseases



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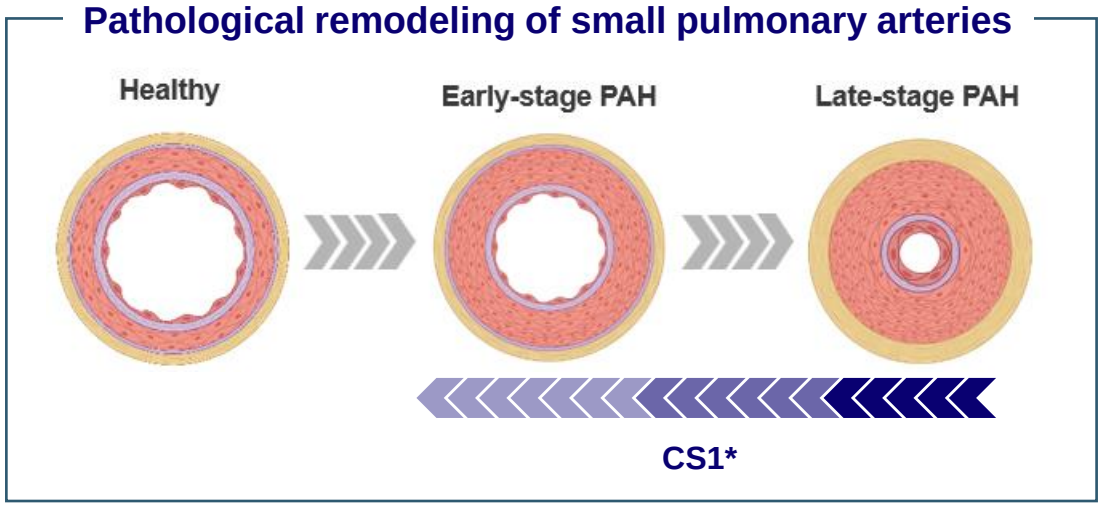
PAH Preclinical data: CS1 has documented multifold reverse remodeling properties



Inhibition of cell proliferation & migration[1]- Pulmonary artery endothelial cells (PAEC) & pulmonary artery smooth muscle cells (PASMC)

Anti-inflammatory effect[1-6]- **Reduction of proinflammatory cytokines, e.g., TGF-β, IL-6 MCP-1** and TNF-α

Anti-fibrotic effect (reduction of profibrotic factors such as TGF-β, fibronectin and and Smad3)

Apoptosis induction[1,5]**-** Reduction of anti-apoptotic Bcl2, Bcl-xl, and increase of apoptotic Caspase3 gene expression

Antiangiogenic effect[3,9]- VEGF (angiogenic factor) regulation

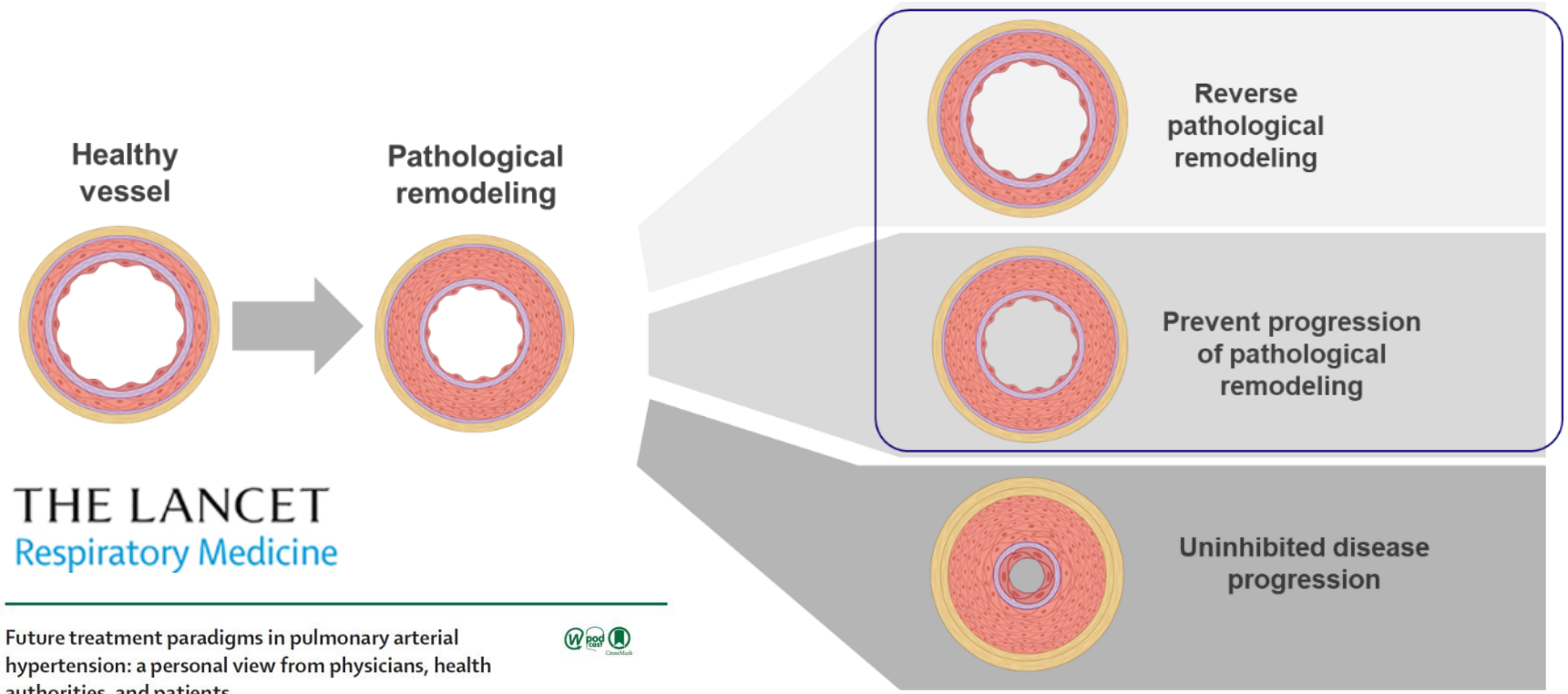
Anti-thrombotic effect[6-8]- Increases vascular t-PA and decreases circulating PAI-1 antigen

*CS1 active substance VPA

1 – In PAH models; 2 – In lung fibrosis models; 3 – In peritoneal fibrosis models; 4 – In hypertension models; 5 – In models of lung injury; 6 – In atherosclerotic men; 7 – In models of thromboembolic conditions; 8 – In healthy men; 9 – In conjunctival scarring models.

Bisserier, M., et al., 2020, Link; Duenas-Gonzales, A., et al, 2008, Link; Han, W., et al, 2021, Link; Kabel, A., et al, 2016, Link; Lan, B., et al, 2015, Link; Zhao, L., et al, 2012, Link; Cardinale, J., et al, 2010, Link; Costalonga, E., et al, 2017, Link; Seet, L., et al, 2019, Link; Wu, S., et al, 2015, Link; Larsson, P., et al, 2016, Link; Saluveer, O., et al, 2014, Link; Svennerholm, K., et al, 2015, Link.

Goal of disease modification in PAH: Prevent progression and/or reverse pathological vascular remodeling



THE LANCET
Respiratory Medicine

Future treatment paradigms in pulmonary arterial hypertension: a personal view from physicians, health authorities, and patients

Franck F Rahaghi, Marc Humbert, Marius M Hoeper, R James White, Robert P Frantz, Paul M Hassoun, Anna R Hemnes, Steven M Kawut, Vallerie V McLaughlin, Gergely Meszaros, Peter G M Mol, Steven D Nathan, Mitchel A Psotka, Farbod N Rahaghi, Olivier Sitbon, Norman Stockbridge, Jason Weatherald, Faiez Zannad, Sandeep Sahay

Main goals of PAH therapy

- Improve symptoms
- Enhance functional capacity
- Slow disease progression
- Reduce hospitalizations
- Prolong survival

PAH= Pulmonary arterial hypertension

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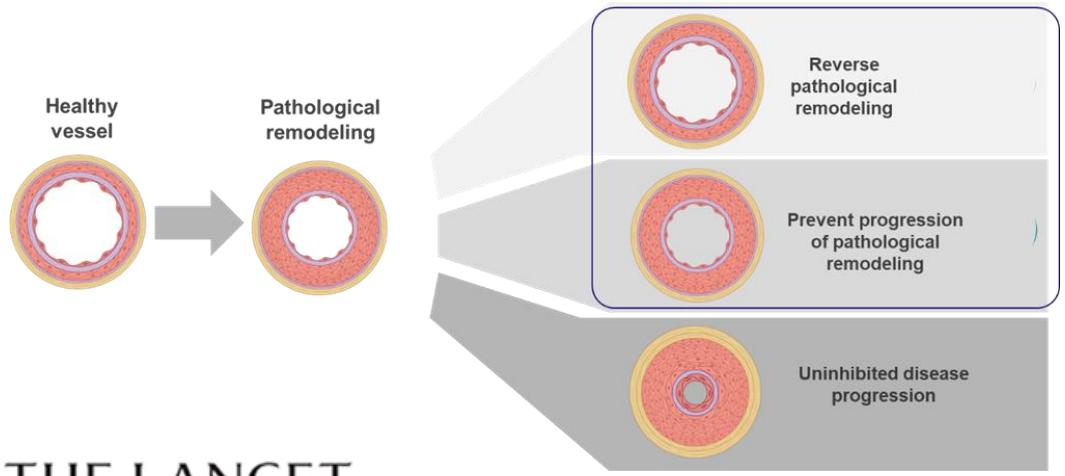

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Main goals of PAH therapy	**Clinical read out**
Improve symptoms	Quality of life (QoL)
Enhance functional capacity	NYHA Functional class
Slow disease progression	REVEAL 2.0 risk score; NYHA Functional class; Right ventricular function
Reduce hospitalizations	Hospitalizations
Prolong survival	REVEAL 2.0 risk score; Right ventricular function

PAH= Pulmonary arterial hypertension

Phase IIa trial of CS1 in PAH

Lead candidate CS1 was evaluated for safety, tolerability and exploration of efficacy in patients with the rare disease PAH.

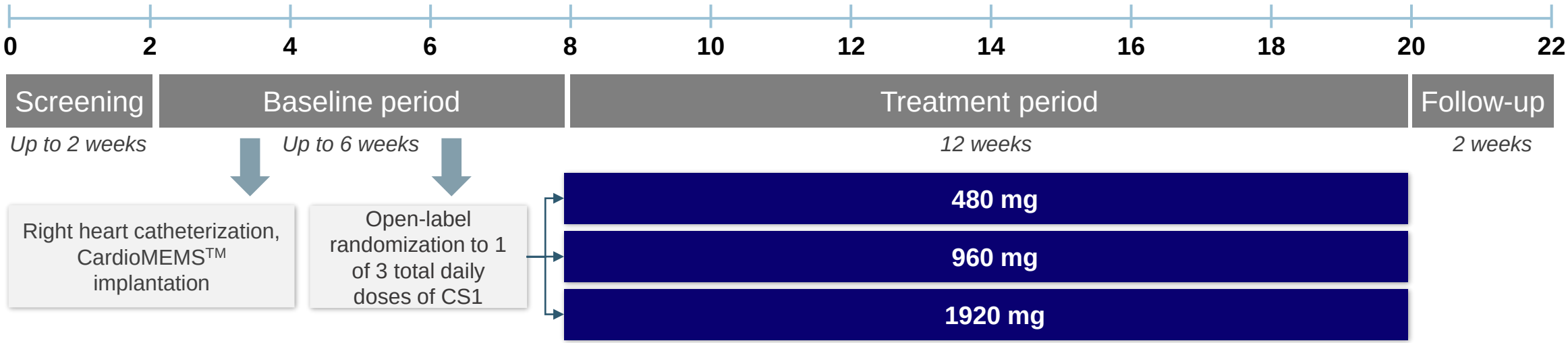
CS1 Phase IIa trial to evaluate safety, tolerability and explore efficacy

Primary endpoint: **Safety and tolerability**

Exploratory endpoints: All standard efficacy endpoints for PAH, validated risk score, PK, and dose-finding

Abbott's CardioMEMS™ System for monitoring pulmonary pressure and pulmonary/RH hemodynamics

Study size: 25 (ITT population) randomized patients at 10 US clinical sites



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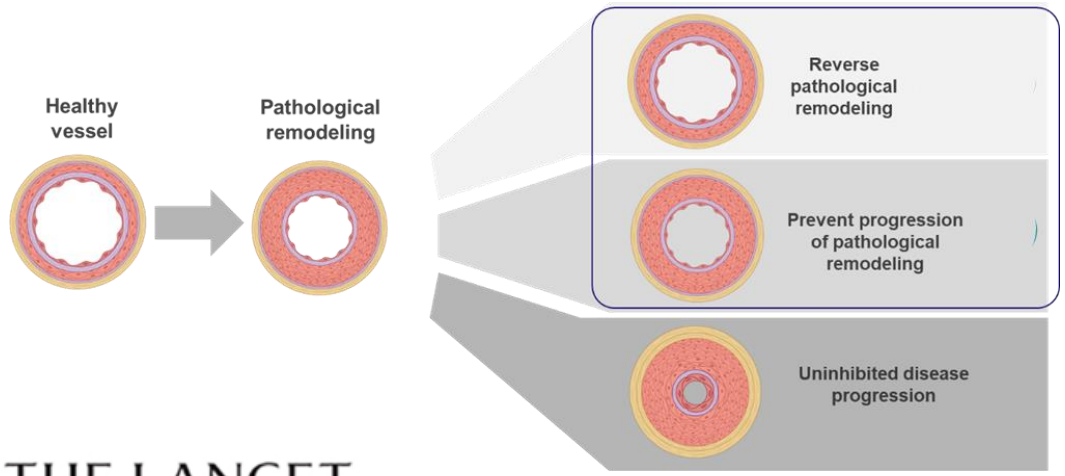

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PAH= Pulmonary arterial hypertension

CS1 - Primary endpoint of safety and tolerability met
No serious adverse events related to CS1

- **No hospitalizations or deaths**
- **No clinically significant abnormalities** in hematology, chemistry, urinalysis, or coagulation parameters
- **No abnormal clinically significant ECG findings, including QT changes**
- Vital signs generally remained stable over time.
- Dose-dependent non-serious treatment-related TEAEs
- Non-serious adverse events in line with the well-known VPA profile

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 (%)	Events n	Subjects n (%)	Events n	Subjects n (%)	Events n	Subjects n (%)	Events n
Any TEAEs	6 (66.7%)	21	5 (62.5%)	21	8 (100.0%)	34	19 (76.0%)	76
Serious TEAEs	2 (22.2%)	2	0 (0.0%)	0	0 (0.0%)	0	2 (8.0%)	2
Treatment-related TEAEs	2 (22.2%)	3	3 (37.5%)	16	6 (75.0%)	28	11 (44.0%)	47
Serious Treatment-related TEAEs	**0 (0.0%)**	**0**	**0 (0.0%)**	**0**	**0 (0.0%)**	**0**	**0 (0.0%)**	**0**
TEAE Leading to Study Drug Discontinuation	1 (11.1%)	1	1 (12.5%)	7	0 (0.0%)	0	2 (8.0%)	8
TEAE Leading to Study Early Withdrawal	1 (11.1%)	1	1 (12.5%)	1	0 (0.0%)	0	2 (8.0%)	2
TEAEs of Special Interest	0 (0.0%)	0	1 (12.5%)	1	3 (37.5%)	3	4 (16.0%)	4
Death	0 (0.0%)	0	0 (0.0%)	0	0 (0.0%)	0	0 (0.0%)	0

CS1 - Improved REVEAL 2.0 risk score, NYHA functional class and mPAP (AUC) indicate better patient outcomes

43% of the patients improved REVEAL 2.0 risk score:

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

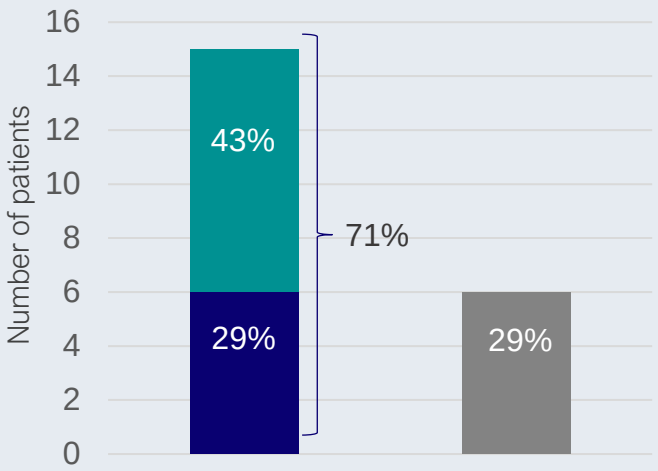
Functional Class: 86% improved or had stable functional class (FC):

Improvement in FC associated with improved survival[2,3] .
Two patients achieved FC I, No patients deteriorated to FC IV

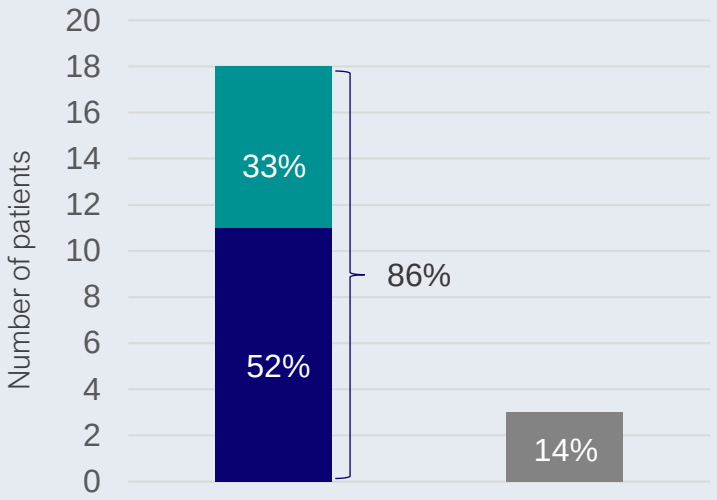
Sustained reduction of mPAP AUC in 67% of patients:

Small change of ePAD of 3, 4, or 5 mmHg from baseline to 6 months is associated with decreased mortality risk[4]

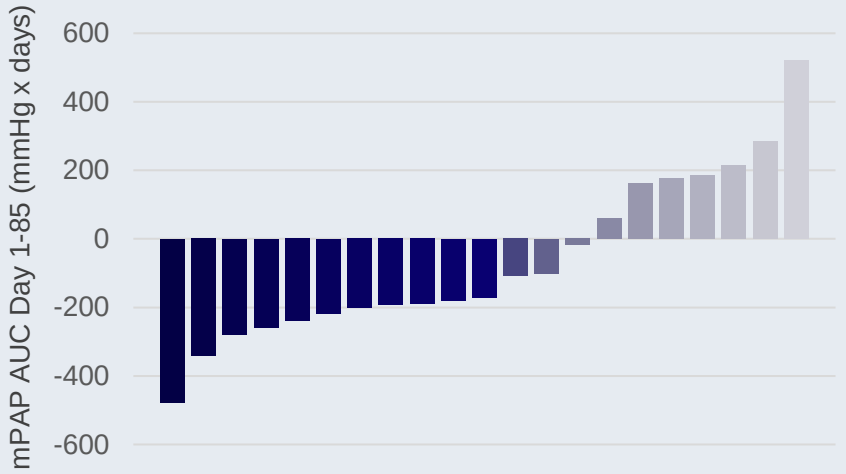
REVEAL risk score change from baseline



NYHA Functional Class change from baseline



Changes in mPAP from CardioMEMS (AUC Day 1-85)



Improvement **Stable** **Worsening**

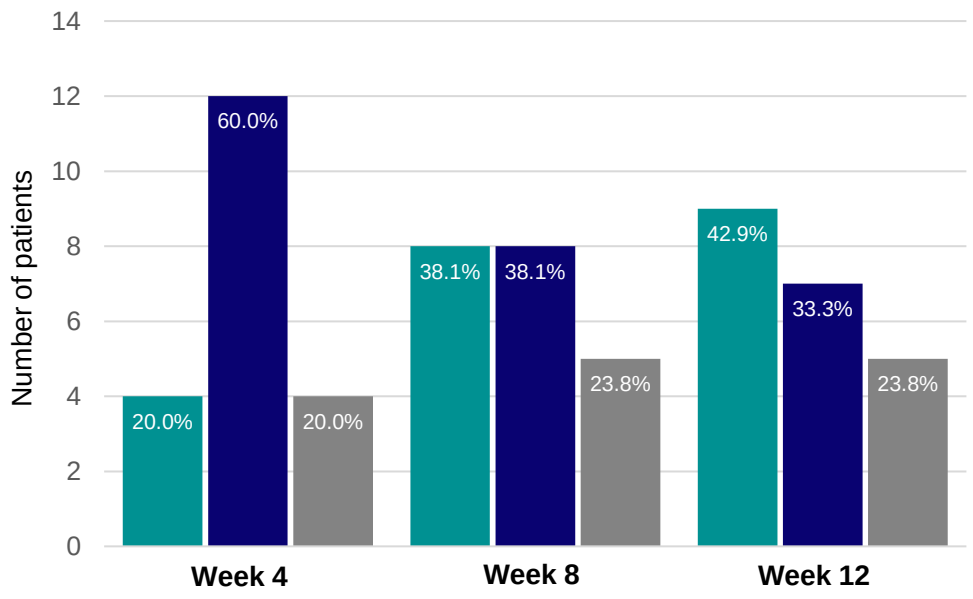
Improvement: At least 1 point reduction in REVEAL risk score. Worsening: At least 1 point increase in risk score.

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; 2. Hoeper, Marius M. et al. The Journal of Heart and Lung Transplantation, Volume 41, Issue 7, 971 – 981; 3. Barst, Robyn J. et al.CHEST, Volume 144, Issue 1, 160 – 168; 4. Zile MR, Bennett TD, El Hajj S, et al. Circ Heart Fail. 2017

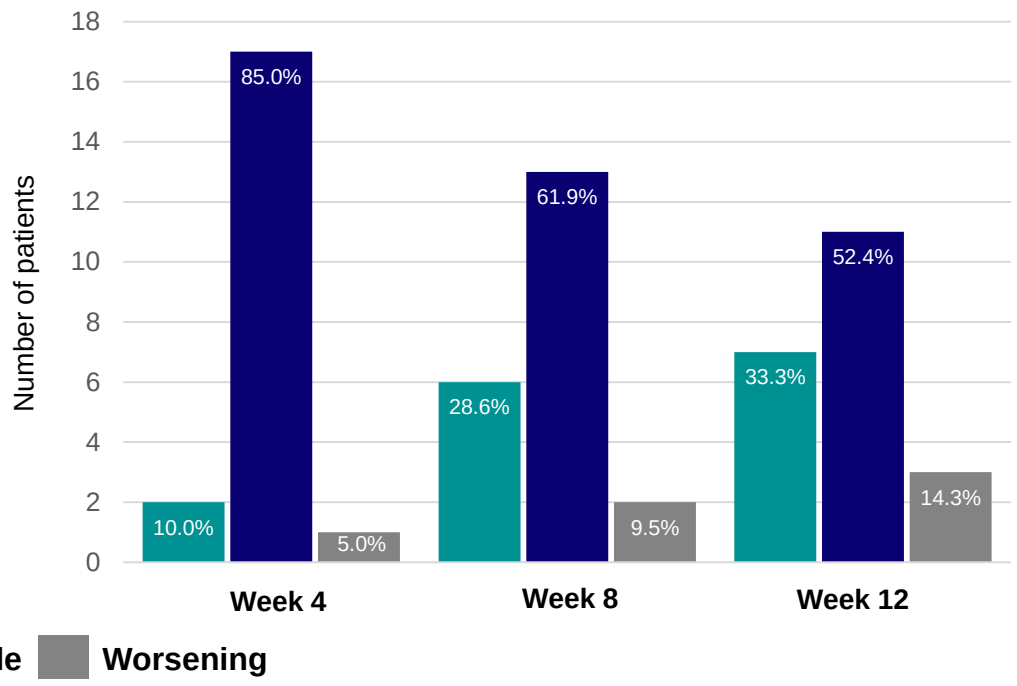
CS1 - Gradual improvement over time on REVEAL 2.0 risk score & NYHA functional class indicating impact on disease progression

Overall number of patients with a reduction of at least 1 point in **REVEAL risk score** increased over time from baseline to week 12



REVEAL score improves independently of functional class

Overall number of patients with improvement in **NYHA functional class** increased over time from baseline to week 12



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PAH leads to right ventricular (RV) dysfunction due to pressure overload resulting in RV failure – fatal outcome for patients

Pathological remodeling of the pulmonary vessels

↓

Lead to pressure overload in RV

↓

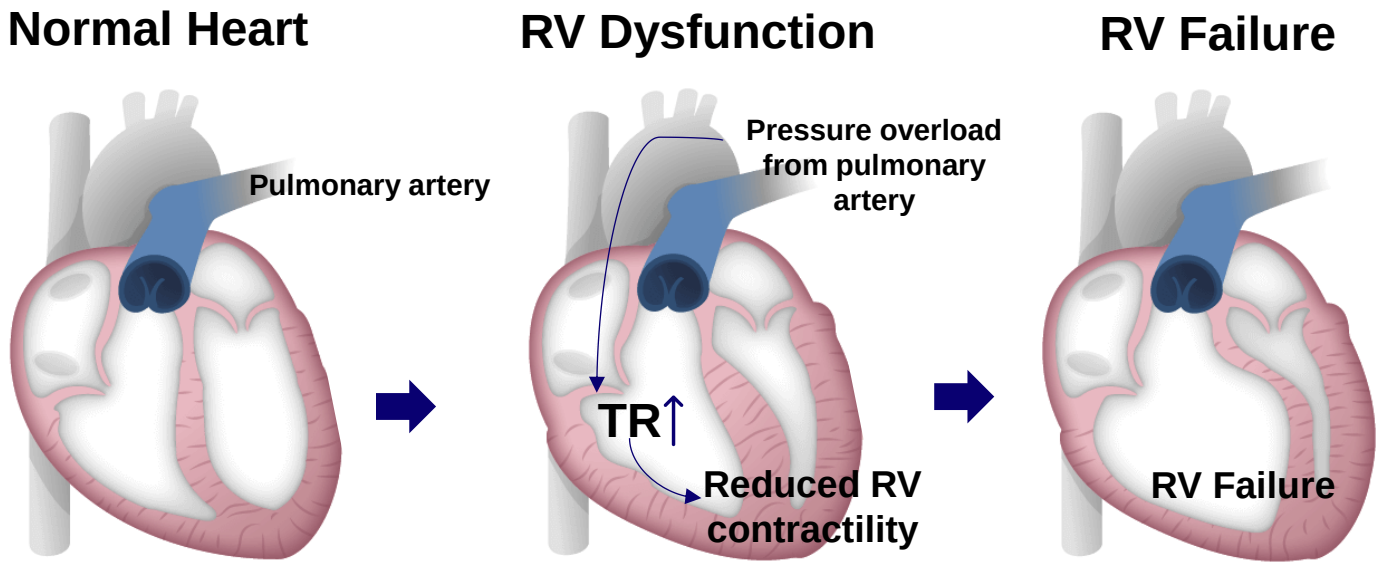
Increased Tricuspid Regurgitation (TR) exacerbates right-sided volume overload

↓

RV dilation and reduced RV contractility

↓

Right ventricular (RV) failure



1. Picture adapted from PAH initiative, LINK: 2. Ryan JJ, Archer SL.Circ Res. 2014 Jun 20;115(1):176-88. 3. Noordegraaf AV et.al., Journal of the American College of Cardiology, Volume 69, Issue 2, 2017,Pages 236-243; 4. Sharifi DK et.al. Front. Physiol. , 28 May 2021. Sec. Clinical and Translational Physiology, Volume 12 – 2021

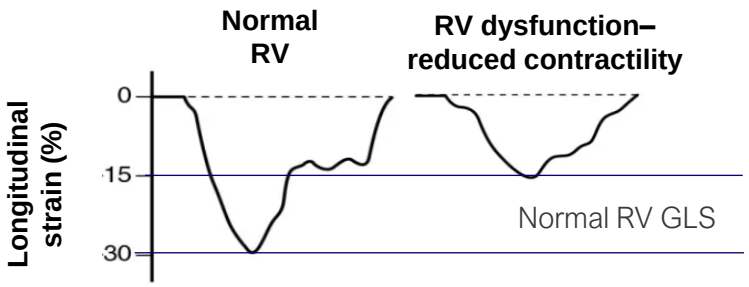
Reverse remodeling of right ventricle improves patient function and prognosis

Key parameters to measure right ventricular function

Right ventricular global longitudinal strain (RV GLS) – a 5% reduction in GLS leads to 7-fold lower mortality rates within four years

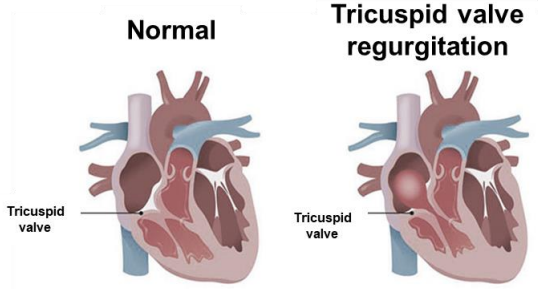
GLS is a highly sensitive indicator for:

1. Identifying subclinical RV dysfunction at early stages of PAH
2. Indicating treatment-induced improvements
3. Predicting clinical worsening, and mortality



Tricuspid Regurgitation (TR) reduction - closely tied to disease severity and prognosis, including mortality

- Common and clinically significant complication of PAH, reflecting right ventricular dysfunction
- Increased tricuspid regurgitation - strongly associated with poor prognosis in PAH patient
- Reduction in TR reflects positive impact of therapies



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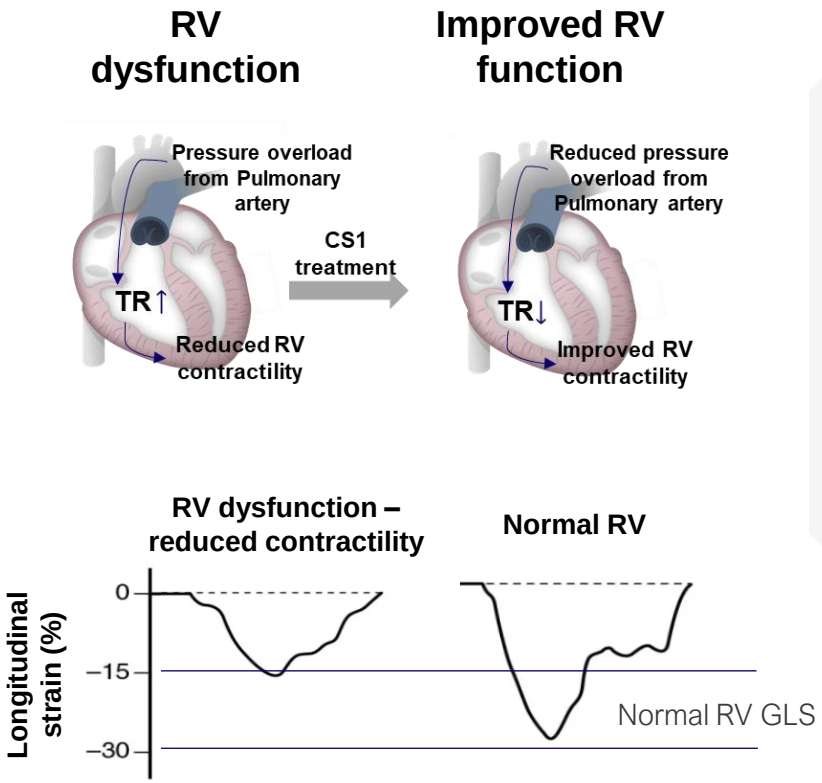
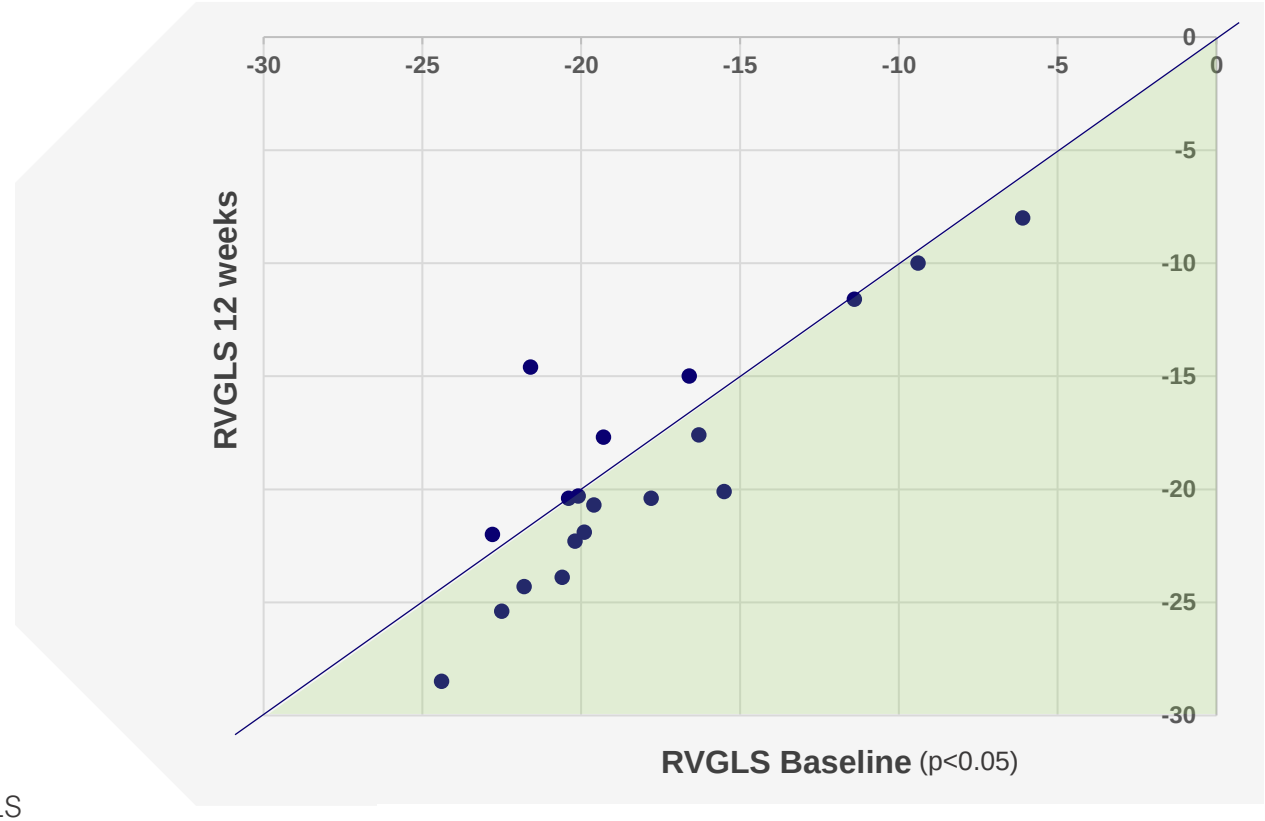
1. Hardegree, Evan L., et al. *The American journal of cardiology* 111.1 (2013): 143-148. 2. Puwanant, Sarinya, et al. *Circulation* 121.2 (2010): 259-266; 3. Van Kessel, Marco, et al. The international journal of cardiovascular imaging 32 (2016): 905-912; 4. Tadic, Marijana, et al.*Frontiers in Cardiovascular Medicine* 8 (2021): 698158.; 5. Evaluation of right ventricular function and pulmonary hypertension (Link); 6. Vos, J.L.et al. Int J Cardiovasc Imaging 38, 1699–1710 (2022). 7. Yoshida, K., et al. European Heart Journal 44.Supplement_2 (2023): ehad655-2000.

CS1 - Improved right ventricular Global Longitudinal Strain (RV GLS) from baseline indicating better RV function





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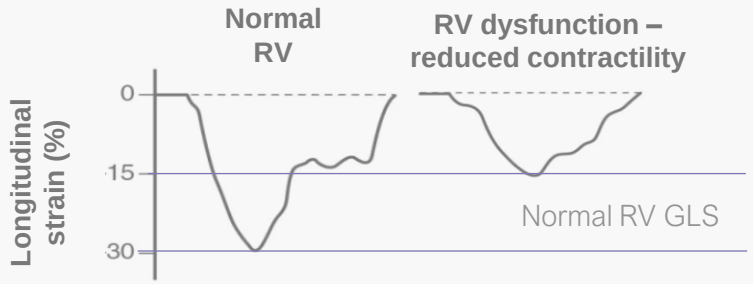
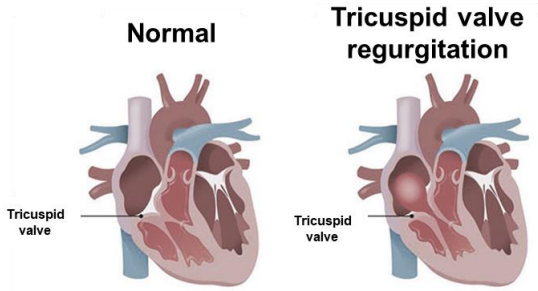
2. Indicating treatment-induced improvements
3. Predicting clinical worsening, and mortality



Tricuspid Regurgitation (TR) reduction - closely tied to disease severity and prognosis, including mortality

- Common and clinically significant complication of PAH, reflecting right ventricular dysfunction
- Increased tricuspid regurgitation - strongly associated with poor prognosis in PAH patient
- Reduction in TR reflects positive impact of therapies



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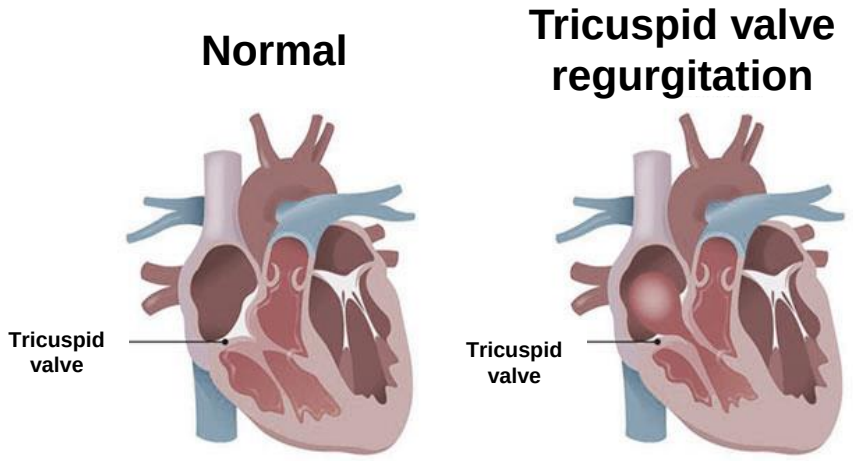
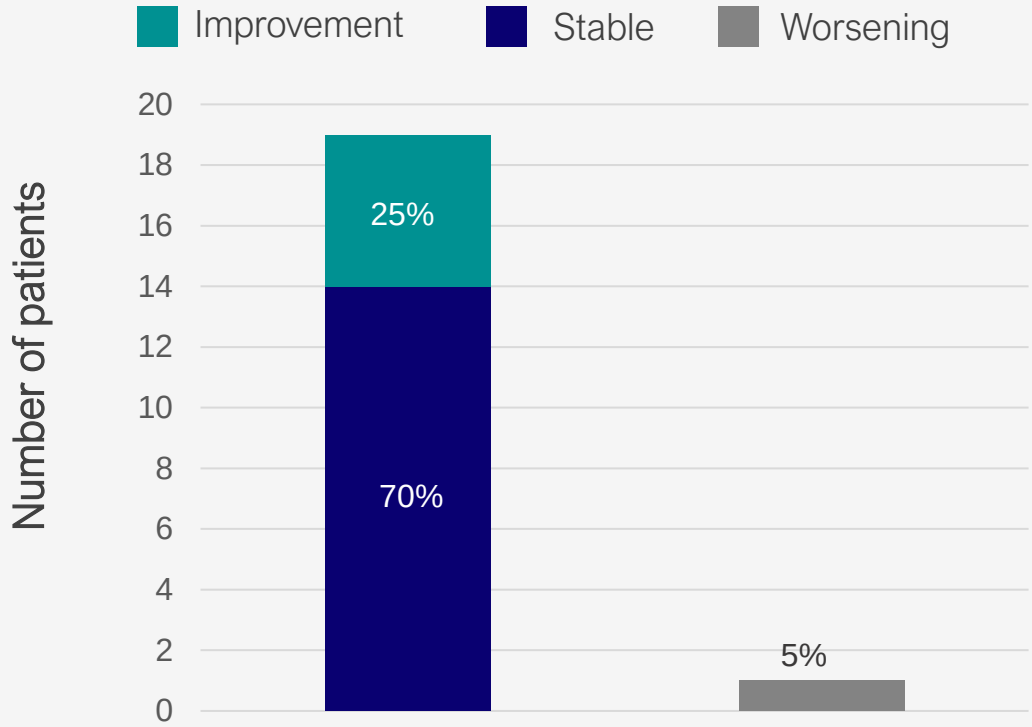
1. Hardegree, Evan L., et al. *The American journal of cardiology* 111.1 (2013): 143-148. 2. Puwanant, Sarinya, et al. *Circulation* 121.2 (2010): 259-266; 3. Van Kessel, Marco, et al. The international journal of cardiovascular imaging 32 (2016): 905-912; 4. Tadic, Marijana, et al.*Frontiers in Cardiovascular Medicine* 8 (2021): 698158.; 5. Evaluation of right ventricular function and pulmonary hypertension (Link); 6. Vos, J.L.et al. Int J Cardiovasc Imaging 38, 1699–1710 (2022). 7. Yoshida, K., et al. European Heart Journal 44.Supplement_2 (2023): ehad655-2000.

CS1 - Reduced tricuspid regurgitation from baseline indicating positive impact on right ventricular function





Picture adapted from Singapore Heart Foundation. Link

CS1 - Positive impact on quality of life (QoL) in patients with PAH

Minnesota Living With Heart Failure Questionnaire

71% of the patients **improved** QoL (15/21)

76% of the patients **improved** or had **stable** QoL (16/21)

PAH-SYMPACT

Cognitive/Emotional Impacts

35% of the patients **improved** C/E impacts (7/20)

75% of the patients **improved** or had **stable** C/E impacts (15/20)

PAH-SYMPACT

Physical Impacts

45% of the patients **improved** physical impacts (9/20)

65% of the patients **improved** or had **stable** physical impacts (13/20)

PAH-SYMPACT

Cardiopulmonary Symptoms

50% of the patients **improved** CP symptoms (9/18)

61% of the patients **improved** or had **stable** CP symptoms (11/18)

PAH-SYMPACT

Cardiovascular Symptoms

50% of the patients **improved** CV symptoms (9/18)

83% of the patients **improved** or had **stable** CV symptoms (15/18)

Change in Minnesota Living with heart Failure (MLHF) questionnaire and PAH-SYMPACT from baseline to week 12 for PP (n=21, 21, 20, and 18 assessable data points).

CS1 - Key results of the Phase IIa trial

- **Primary endpoint of safety & tolerability** was successfully met
- **Encouraging signs of reverse vascular remodeling** observed, which are accompanied by
  - **Gradual impact over time on REVEAL 2.0 risk score and NYHA functional class**
  - Measures of improved right ventricular function of the heart – **improved RV GLS and reduced TR**
  - **Improved quality of life** as seen in both PAH-SYMPACT and Minnesota Living With Heart Failure

Path forward for CS1 in PAH

- Expanded Use Program (EAP) / long-term data
  - 10 patients enrolled, first dosed in August 2024
  - **Interim data readout - Q2 2025**
- EAP sub-study
  - Imaging by Fluidda, patient enrollment started
  - **Three CT scans over 12 months, results in Q1 2026**
- Regulatory interactions
  - **Type C meeting**
- Phase IIb PAH program in PAH
  - **FDA acceptance of trial – H1 2025**
  - Start of trial – H1 2026

CS1 has shown potential to

- **Reverse pathological vascular remodeling**
- **Improve quality of life**
- **Extend life expectancy**

of PAH patients

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CS1 – Transforming treatments for PAH

- PAH – a rare, fatal, and progressive disease with high unmet need
- A market void of safe and well-tolerated treatments addressing underlying pathophysiology of PAH

- Combined preclinical and Phase IIa data for CS1 is consistent with a reverse vascular remodeling potential
- CS1 is an oral, safe, and well-tolerated treatment with reverse vascular remodeling effect for rare disease PAH

- CS1 has patent protection plus ODD market exclusivity of 7 (US) and 10 (EU) years
- Market size of $12Bn

Cereno Scientific is enhancing and extending lives of people living with rare cardiovascular and pulmonary diseases

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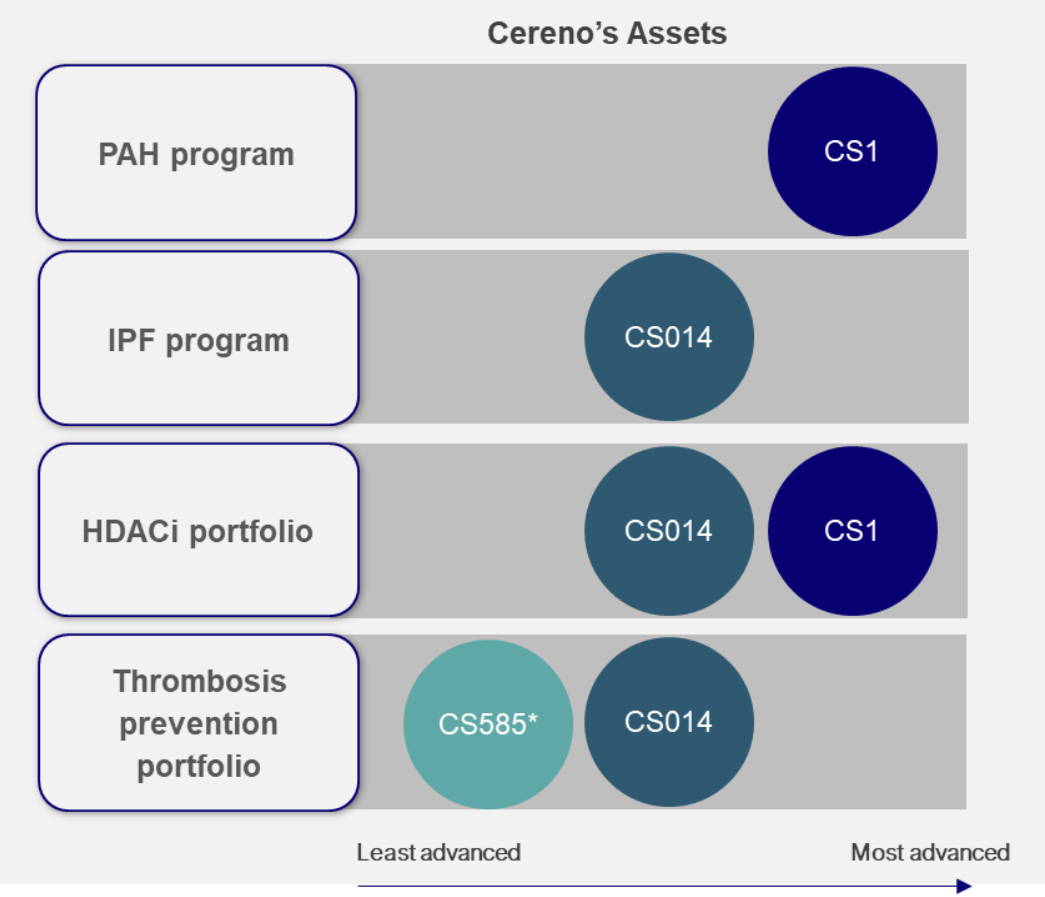
Attractive opportunity for strategic partnerships or exit via M&A given near-term inflection points

Cereno's assets/portfolio for:

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



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

Cereno Scientific is pioneering treatments to enhance and extend life. Our innovative pipeline offers disease-modifying drug candidates to empower people suffering from rare cardiovascular and pulmonary diseases to live life to the full.

Lead candidate CS1 is an HDACi that works through epigenetic modulation, being developed as an oral, safe, well tolerated and effective disease modifying treatment for rare disease Pulmonary Arterial Hypertension (PAH). A Phase IIa trial evaluating CS1's safety, tolerability, and exploratory efficacy in patients with PAH demonstrated that CS1 was safe, well-tolerated and showed a positive impact on exploratory clinical efficacy parameters. An Expanded Access Program enables patients that have completed the Phase IIa trial to gain access to CS1. HDACi CS014, in Phase I development, is a new chemical entity with disease-modifying potential. CS014 employs a multi-modal mechanism of action as an epigenetic modulator, targeting key unmet needs in patients with rare disease Idiopathic Pulmonary Fibrosis (IPF). Cereno Scientific is also pursuing a preclinical program with CS585, an oral, highly potent and selective prostacyclin (IP) receptor agonist that has demonstrated the potential to significantly improve disease mechanisms relevant to cardiovascular diseases. 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 Thrombosis prevention without increased risk of bleeding and Pulmonary Hypertension.

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 Scientific is listed on the Nasdaq First North (CRNO B).

Pioneering treatments to enhance and extend life.