EDISON

# Cereno Scientific

## Entering a value inflection phase

Company outlook

Healthcare

12 January 2026

**We refresh our investment case for Cereno Scientific as the company enters a key value inflection period, with its lead asset CS1 expected to initiate a Phase IIb study in Q226, followed by Phase II development of CS014 shortly thereafter. With the FDA green light for the CS1 Phase IIb study design in December 2025 and financing in place with the recent raise of up to SEK665m, we see a clear path to timely initiation of the CS1 trial, although we now expect a modest delay to CS014 timelines. We believe the CS1 Phase IIb trial represents the most significant upcoming catalyst for Cereno, with potential to drive a material re-rating. Winrevair/sotatercept's sales trajectory since launch in March 2024 underscores the unmet need in PAH and the market's receptiveness to potentially disease-modifying treatments, such as CS1. Reflecting adjustments to our peak sales and success probability assumptions for CS1, our valuation for Cereno resets to SEK6.6bn or SEK21.1/share (from SEK5.4bn or SEK17.5/ share previously).**

| Year end | Revenue (SEKm) | PBT (SEKm) | EPS (SEK) | DPS (SEK) | P/E (x) | Yield (%) |
|---|---|---|---|---|---|---|
| 12/23 | 0.0 | (46.4) | (0.20) | 0.00 | N/A | N/A |
| 12/24 | 0.0 | (98.1) | (0.35) | 0.00 | N/A | N/A |
| 12/25e | 0.0 | (95.4) | (0.33) | 0.00 | N/A | N/A |
| 12/26e | 0.0 | (73.5) | (0.25) | 0.00 | N/A | N/A |

Note: PBT and EPS are normalised, excluding amortisation of acquired intangibles, exceptional items and share-based payments.

## A crucial period ahead for CS1

We expect 2026 to be a strategically important year for lead asset CS1 (targeting pulmonary arterial hypertension (PAH)). The commencement of the Phase IIb dose-finding study will mark the first focused assessment of CS1's optimal dosing, efficacy and disease-modifying potential in a larger, randomised study. The choice of a 36-week initial treatment duration (versus the traditional 24 weeks) looks strategic, with the aim to maximise treatment efficacy and patient exposure. Top-line results in Q428 will be the most important business milestone for the company to date.

## CS014 progressing towards Phase II, with slight lag

While CS014 was expected to commence Phase II in idiopathic pulmonary fibrosis (IPF) in H126, we believe there has been a slight shift in timelines, not unexpected given the clinical focus on CS1. We estimate the CS014 Phase II study will now commence in H127. The approval of Jascayd (nerandomilast) in October 2025 (the first novel IPF therapy approved since 2014 per our understanding) has intensified competition, and meaningful differentiation for CS014 will come from showcasing its favourable safety and tolerability profile and potential disease reversal signals, beyond incremental slowing of disease decline.

## Valuation: SEK6.6bn or SEK21.1 per share

We raise the PoS for CS1 to 50% (from 45%) following the FDA Phase IIb clearance and peak sales estimate to $2.8bn (from $2bn), reflecting positive readacross from Winrevair's market uptake and CS1's more favourable tolerability profile. This is partially offset by adjusted launch timelines for CS1 and CS014 (2032 and 2033, respectively). The recent raise of up to SEK665m has secured the runway to Q427.

| | |
|---|---|
| **Price** | **SEK6.91** |
| **Market cap** | **SEK2,154m** |
| | SEK9.23/$ |
| Estimated pro-forma net cash/ (debt) at 31 December 2025 | SEK(126.1)m |
| Shares in issue | 311.7m |
| Free float | 93.0% |
| Code | CRNO B |
| Primary exchange | NGM |
| Secondary exchange | N/A |

### Share price performance


| % | 1m | 3m | 12m |
|---|---|---|---|
| Abs | (16.5) | (23.2) | 36.2 |
| 52-week high/low | | SEK11.9 | SEK4.7 |

### Business description

Cereno Scientific is a clinical-stage biotech based in Sweden, focused on the development of innovative, effective and safe treatments for indications with high unmet needs. Lead asset CS1 is an HDAC inhibitor that acts as an epigenetic modulator. Cereno reported positive top-line results from the Phase IIa study in pulmonary arterial hypertension in September 2024 and FDA clearance for the Phase IIb trial in December 2025. Second asset CS014, a proprietary NCE and HDACi, is being developed for idiopathic pulmonary fibrosis (Phase II-ready), and preclinical asset CS585 is likely to target rare thrombosis-related indications.

### Next events

| | |
|---|---|
| Capital markets day | February 2026 |
| CS1 Phase IIb trial launch | Q226 |
| CS014 Phase II trial launch | H127 |

### Analysts


Jyoti Prakash, CFA +44 (0)20 3077 5700
Arron Aatkar, PhD +44 (0)20 3077 5700

healthcare@edisongroup.com
Edison profile page



***Cereno Scientific is a research client of Edison Investment Research Limited***

# Investment summary

## Company description: Addressing rare diseases with high unmet needs

Cereno Scientific is clinical-stage biotech focused on developing novel and effective treatment options for patients with rare cardiovascular and pulmonary diseases; it has been listed on the Nasdaq First North Growth Market since June 2023. The company's portfolio comprises two clinical and one preclinical programme. The candidate is a patent-protected delayed immediate release formulation of VPA, acting as an epigenetic modulator through histone deacetylase inhibition (HDACi). Preclinical and clinical data of CS1 indicate a potential disease-modifying capability in PAH. CS1 is a well-tolerated oral therapy with a favourable safety profile that has shown encouraging efficacy signals in a Phase IIa trial in patients with PAH, including improvements in right heart function and patient quality of life, consistent with reverse vascular remodelling. It holds Orphan Drug Designation in the US and EU and the FDA Fast Track designation. Following FDA clearance in December 2025, Cereno is gearing up to launch a subsequent Phase IIb trial from Q226. The company's second asset, CS014, is a next-generation precision-engineered HDACi and new chemical entity. It recently successfully completed a Phase I safety study in healthy volunteers (showing a favourable safety and tolerability profile), laying a robust foundation for further development efforts. Next steps will be a Phase II trial in IPF patients, which we expect will commence in H127. Cereno's third asset, CS585, is a prostacyclin (IP) agonist in the preclinical stages of development. While a precise indication is yet to be decided for the candidate, preclinical research has demonstrated its potential to prevent thrombosis without the increased risk of bleeding, showing promise in overcoming a key challenge associated with currently available anti-thrombotic medicines. CS585 is expected to enter the clinic from 2027.

## Financials: Well capitalised with recent SEK665m financing

Being clinical-stage, Cereno's operations continue to be supported by external capital. In December 2025, the company completed a financing package of up to SEK665m to support its upcoming clinical plans, comprising SEK100m from a directed share issue, SEK175m in convertible debt, up to SEK175m in loan facilities and up to SEK215m from potential warrant conversions. The financing was completed on premium terms (eg convertibles at SEK10/share, a 30% premium to last close) but introduces an element of execution and market risk, as access to the loan tranche is partially contingent on share price performance (SEK10/share). That said, we see scope for a share price re-rating as CS1 enters Phase IIb, with the proceeds extending Cereno's cash runway into Q427.

## Valuation: Material upside to be unlocked

We value Cereno at SEK6.6bn or SEK21.1/share (from SEK5.4bn or SEK17.5/share previously) using a risk-adjusted NPV (rNPV) approach derived from its two clinical-stage assets, CS1 and CS014. We exclude CS585, which remains preclinical, but note the potential upside upon clinical progression. Following FDA clearance of the CS1 Phase IIb study design, we raise our probability of success (PoS) for CS1 to 50%, from 45% previously. We also increase our peak penetration assumption to 25% (from 20%), reflecting the expanding opportunity for disease-modifying therapies in PAH, supported by the strong commercial uptake of Winrevair. Note that Winrevair is administered subcutaneously and can be associated with major side effects such as serious bleeding risk (4% of patients on Winrevair in the Phase III STELLAR trial, versus 1% on placebo). CS1's more convenient oral administration and a relatively benign safety profile provide a competitive advantage in this regard. Offsetting these upgrades, we push out our assumed launch timelines by one year for CS1 (to 2032) and CS014 (to 2033). CS1 and CS014 account for 84% and 16% of our implied enterprise value for Cereno, respectively.

## Sensitivities: Favourable safety profiles somewhat de-risk development

Cereno is subject to the usual clinical, regulatory and financing risks associated with biopharma companies. A key sensitivity, in our view, is the reliance on a single clinical asset (CS1) to drive the valuation (84% in our model), though we acknowledge that as CS014 progresses, it will increasingly contribute to the value of the company. For CS1, proof-of-concept has been established with encouraging Phase IIa outcomes, however, the upcoming Phase IIb trial will be the first robust test of efficacy. PAH, the target indication for CS1. The PAH treatment landscape is rapidly evolving, with increased focus on treatments targeting underlying disease biology. We believe that key differentiators of CS1 could be its favourable safety profile, its convenient once-daily oral dosing (which also enhances its combination potential with

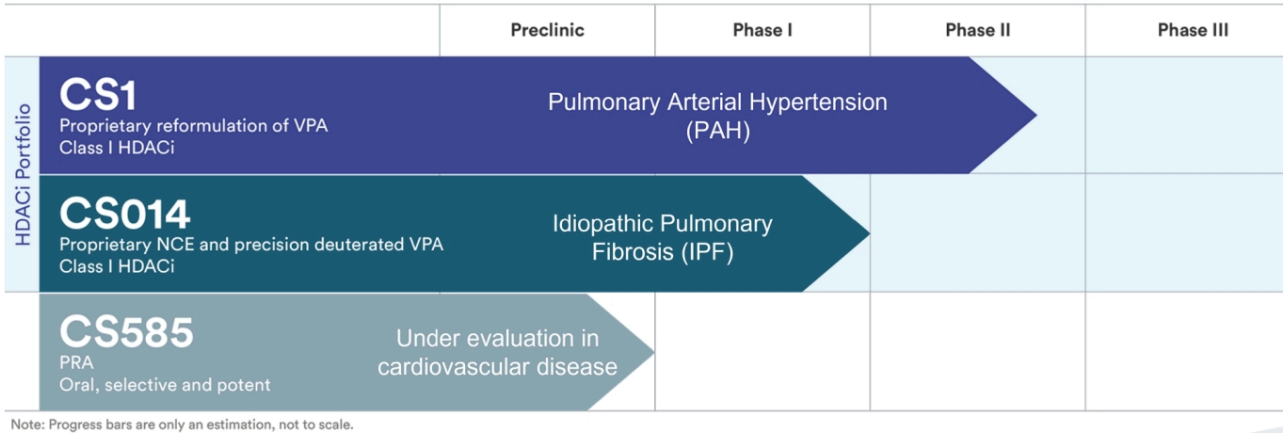
currently approved treatments), alongside its positioning as a potentially disease-modifying therapy for the condition. Access to funding remains another key risk despite the recent sizeable financing package. Pivotal trials in PAH will likely be expensive and difficult to recruit (given the need for frequent follow-ups and testing), requiring external funding, either through capital raises or in the form of an out-licensing partnership. Should financings be achieved through equity issuances, the pricing may not be favourable, with the potential to be dilutive for shareholders.

## Pipeline to address rare indications

Cereno's clinical pipeline is positioned to address indications with high unmet medical needs, with a focus on rare indications, through the development of new, safe and effective therapies (Exhibit 1). In our view, the strategy of targeting rare diseases is logical for Cereno, as it provides various R&D benefits and commercial advantages, including smaller clinical trials, lower-risk capital and potentially accelerated routes to market with extended periods of exclusivity.

Lead asset CS1 is a patent-protected delayed immediate release formulation of VPA, being developed as a potential disease-modifier for PAH. Second asset CS014 is a novel deuterated analogue of VPA, which holds promise in IPF and other diseases involving vascular remodeling and fibrosis. Cereno's third asset is CS585, which is in the preclinical stages of development. It has been designed as an oral, selective and potent agonist of the IP receptor. Management is yet to confirm the precise indication that will be targeted by CS585, but it has shown promise in preventing thrombosis without increased bleeding risk.

**Exhibit 1: Cereno's clinical development pipeline**

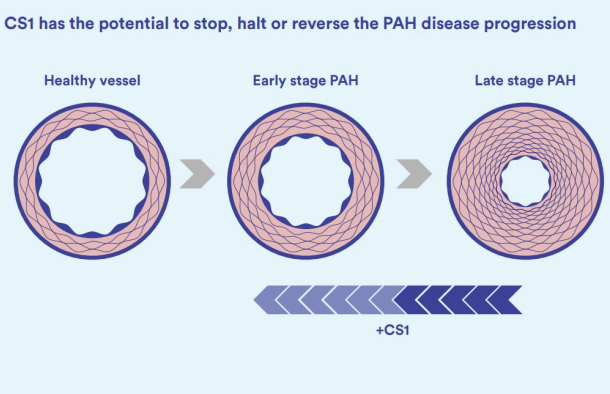



Source: Company resources

## Clinical-stage assets leverage principles of epigenetic modulation

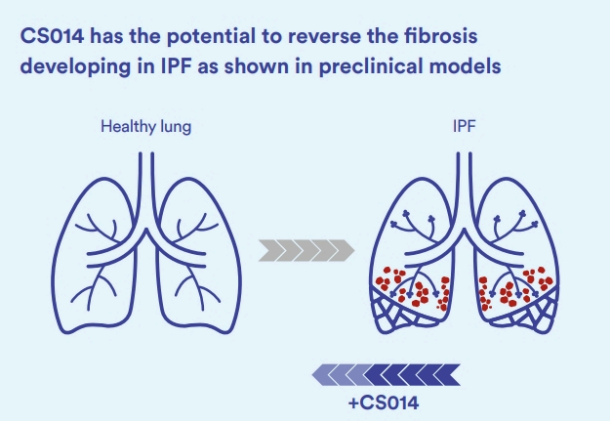
The clinical-stage assets CS1 and CS014 form Cereno's HDACi portfolio. These have been designed to leverage the principles of epigenetic modulation to achieve disease modification and reverse pathological remodelling. PAH is characterised by thickening and tightening of arteries in the lung, restricting the flow of blood from the right side of the heart to the lungs. These effects increase the resistance of blood flow, referred to as vascular remodelling, leading to high blood pressure and impaired circulation. As an HDACi, CS1 aims to prevent disease progression (Exhibit 2). With IPF, the lungs become scarred, leaving them stiff and less able to efficiently transfer oxygen, making it difficult to breathe. CS014 aims to reverse these effects (Exhibit 3).

**Exhibit 2: CS1 is designed as a potentially disease-modifier for PAH**




Source: Company resources

**Exhibit 3: CS014 holds promise in addressing unmet needs in IPF**




Source: Company resources

# CS1: Set to enter Phase IIb in Q226

## Encouraging results in Phase IIa support further development

In September 2024, encouraging top-line results were reported from the Phase IIa CS1-003 trial, which investigated the lead candidate in PAH patients on top of standard of care. The trial was a randomised, open-label, blinded endpoint, multi-centre study, primarily designed to evaluate safety and tolerability (primary endpoint), with exploratory measures of pharmacokinetics and efficacy. An important component of the trial was the collaboration with Abbott, providing access to its CardioMEMS Heart Failure (HF) System, which was implanted in patients and allowed for continuous measurements of pulmonary pressure alongside other measures of cardiopulmonary function. The innovative design of this trial was recognised with a publication in *Pulmonary Circulation*.

The enrolled patients (n=25) were randomised to receive either 480mg, 960mg or 1,920mg of CS1. Of the nine patients in the 480mg arm, two were not evaluable, while in the 960mg and 1,920mg arms, one patient from each group was not evaluable. As a result, there were seven evaluable patients in each arm, all of whom were included in the efficacy analyses, and these patients were treated as a pooled group (n=21), since pharmacokinetic observations showed that therapeutic drug exposure was achieved even in the lowest dose group.

Importantly, in terms of safety, there were no CS1-related serious adverse events at any of the tested doses across all patients, and only two patients discontinued treatment due to treatment-emergent adverse events, which were deemed unrelated to CS1. In our view, these safety and tolerability results compare positively to currently available PAH treatments, which often come with undesirable side effects. For example, prostanoids/IP analogues from a class of drugs called vasodilators (used in combination background therapy for PAH) come with side effects such as nausea, vomiting, diarrhoea, dizziness and headache. Sotatercept (brand name: Winrevair), the treatment for PAH approved in March 2024, is also associated with serious side effects such as (but not limited to) bleeding, epistaxis (nosebleeds), telangiectasia (skin redness/dots), erythema, diarrhoea and dizziness and increased haemoglobin/platelet counts. We note that in addition to safety and tolerability, CS1 may offer differentiation through its convenient oral dosing, compared to the injectable or infusion routes required for the aforementioned treatment options.

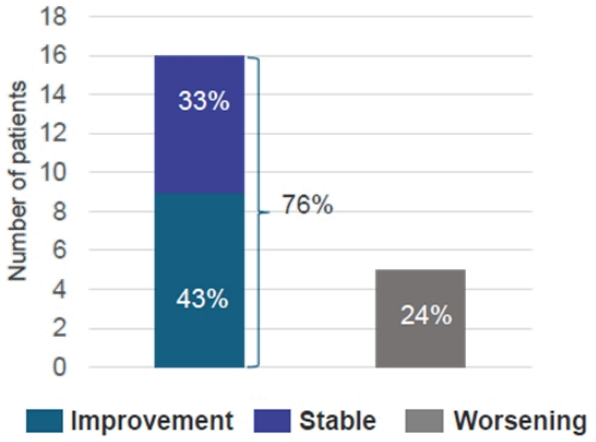
We note that while the trial was not powered for statistical significance measures of efficacy, analyses of the various exploratory efficacy endpoints do provide encouragement. In particular, Cereno shared results for three standard PAH efficacy measures:

- Registry to Evaluate Early and Long-Term PAH Disease Management (REVEAL) risk scores.
- Functional class (FC) changes: PAH is classified under four FCs by the New York Heart Association (NYHA) and World Health Organization (WHO), ranging from I (no symptoms) to IV (debilitating symptoms).
- Haemodynamics: mean pulmonary arterial pressure (mPAP), measured as area under curve (AUC).

For the first of these efficacy measures, the data showed that 43% (9/21 patients) achieved at least a one-point improvement based on the REVEAL risk scores, with 71% (15/21 patients) reporting an improved or stable risk score. Cereno released additional data in February 2025 (discussed below), which demonstrated a further improvement in scores, with 76% (16/21 patients) reporting an improved or stable risk score (Exhibit 4). We believe this is particularly noteworthy, as a one-point reduction over 12 weeks is associated with a 23% reduction in relative risk of death after 12 months (note that scores range from 0 to 22, with the latter representing the highest risk). We also highlight that 33% (7/21 patients) having stable scores may be considered meaningful, since PAH is a fast-progressing condition that is rarely associated with rapid improvements.

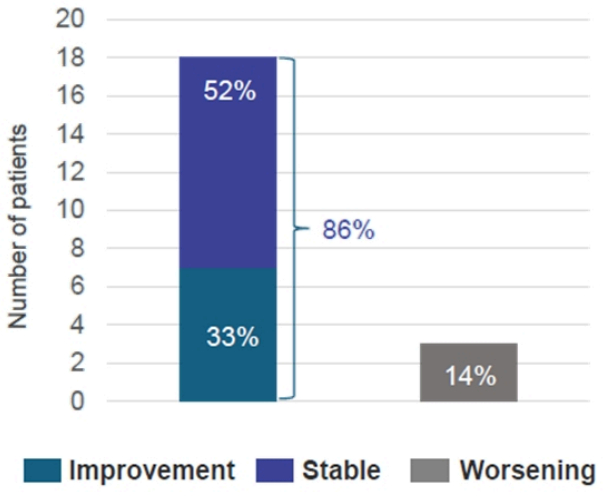
The second efficacy measure was change in the NYHA FC from baseline, an indicator of movement and physical activity levels. The 25 randomised patients were categorised as FC II (slight limitation of activity; 10 patients) and III (marked limitation of activity; 15 patients). Of the 21 evaluable patients, the data showed that 33% (7/21 patients) reported an improvement in FC, while 86% (18/21 patients) achieved an improved or stable FC (Exhibit 5).

**Exhibit 4: CS1-003 data: REVEAL risk score changes from baseline**




Source: Company resources

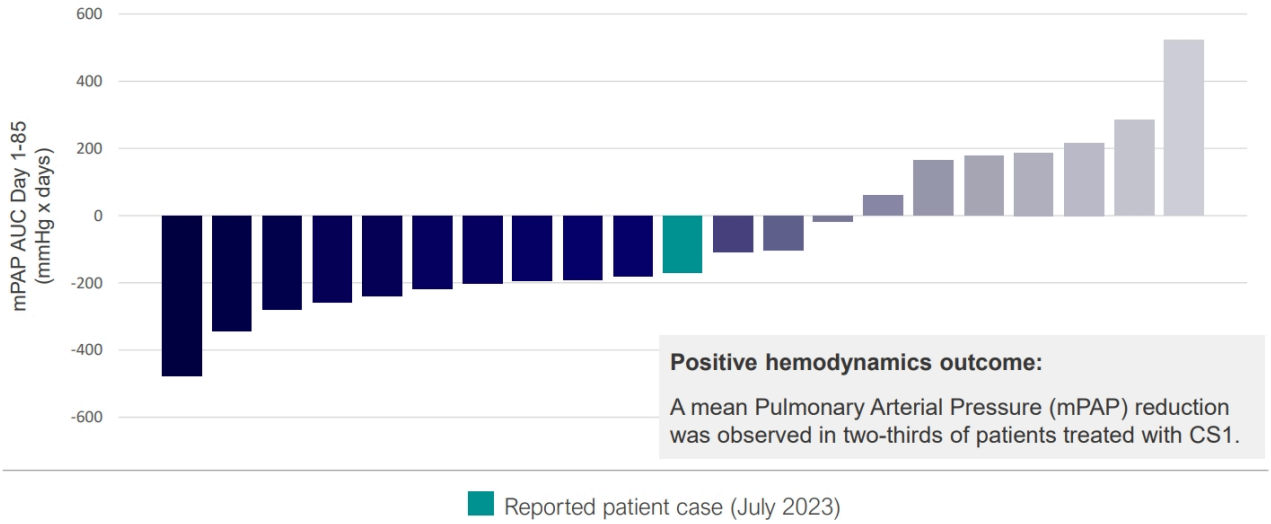
**Exhibit 5: CS1-003 data: NYHA FC changes from baseline**




Source: Company resources

The third efficacy parameter was changes in mPAP. As mentioned above, Cereno worked in collaboration with Abbott, using its CardioMEMS HF System to take daily readings of pulmonary pressure (85 readings, as an average over 20 seconds, per patient over the 12-week study duration), providing a robust and objective basis for analyses. Normal pulmonary artery pressure is 11– 20mmHg at rest, and a reading of >20mmHg is associated with PAH. The CS1-003 data indicated that 67% (14/21 patients) had durable reductions in mPAP. Furthermore, one patient experienced a reduction in mPAP as high as 5mmHg, while reductions ranged from 0.3–4.3mmHg for the other patients who reported improvement with the CS1 treatment (Exhibit 6).

**Exhibit 6: CS1-003 data: mPAP changes measured by CardioMEMS (AUC, day 1–85)**




Source: Company resources

For reference, management highlighted the strong correlation between reduction in estimated pulmonary artery diastolic pressure (ePAD, similar to PAP) and mortality rates, with some research indicating a 5mmHg reduction in ePAD from baseline over six months reduced mortality risk by 30%. We also highlight that the case shown in green in the exhibit above related to a patient case study, from the first patient to complete the trial. This patient reported significant improvements on all PAH measures, including a 30% reduction in mPAP, and improvement from FC II to FC I. The full study results highlight that 10 patients exhibited greater reductions in mPAP than this case study, highlighting the potential of CS1 to induce clinically meaningful benefits in PAH, in our view.

## CS1's potential further bolstered by incremental data

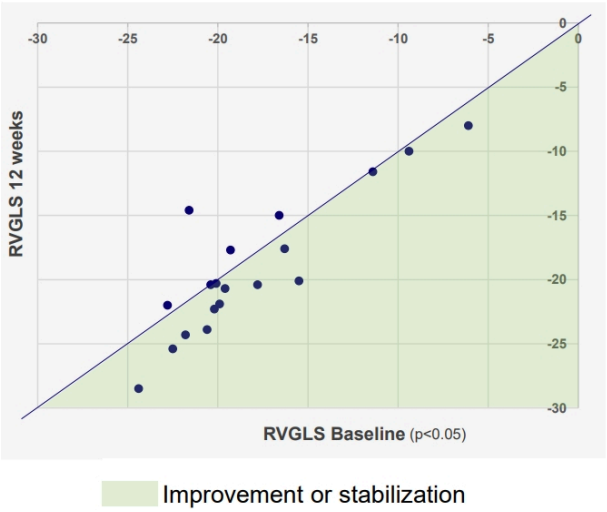
In February 2025, Cereno presented further data from CS1-003, which became available after completion of the clinical study report, supporting CS1's potential to reverse vascular remodelling, backed by its potentially disease-modifying mechanism as an HDACi. The data (further details disclosed in March 2025) showed CS1's positive impact on right-ventricular global longitudinal strain (RVGLS; a highly predictive indicator of right-ventricular remodelling at early stages of disease and future mortality), a gradual improvement over time (in the 12-week treatment period) on the REVEAL 2.0 risk score and the NYHA FC, as well as improvement in pulmonary vascular resistance (PVR) for a subset of early-stage patients.

Disease-modification is the key goal for many treatments in development, particularly for progressive conditions such as PAH. In this context, the incremental data from CS1-003 was particularly encouraging, in our view. While Cereno had previously discussed achieving positive signals in exploratory efficacy parameters, this data provided more context on specific parameters related to vascular remodelling and right heart functioning (a key predictor of mortality in PAH), and CS1's ability to reverse or stabilise these.

**RVGLS**, a measure of right heart function and treatment response. Since PAH is characterised by high blood pressure in the pulmonary arteries, leading to right ventricular dysfunction, RVGLS is a strong predictor of right heart functioning and clinical worsening, including mortality. Normal RVGLS values range from -24.5 ± 3.8%, with lower values indicating impaired RV function. Further analysis of the Phase IIa data showed that CS1 was able to provide improvements and/or stabilisation in RVGLS for the majority of patients in the trial (Exhibit 7). Studies have shown that a 1% improvement in RVGLS is associated with a 5% decreased risk of mortality.

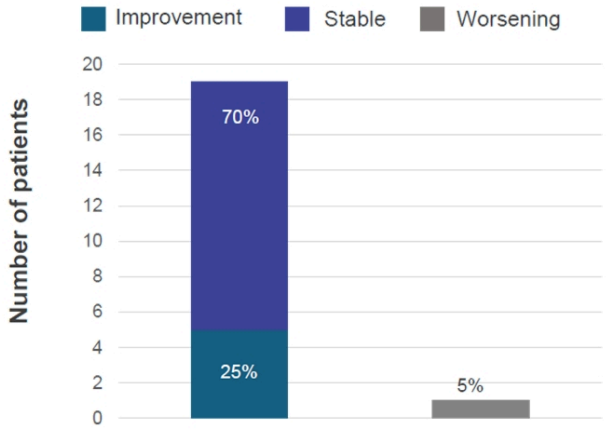
**Tricuspid regurgitation (TR)** is a common complication of PAH, stemming from increased pressure and volume overload in the right ventricle, leading to the tricuspid valve not closing properly and blood flowing back to the right atrium. This is indicative of poor prognosis in PAH. CS1 was able to show an improvement and/or stabilisation in this parameter in 95% of patients (Exhibit 8).

**Exhibit 7: CS1-003 additional data: RVGLS improvements from baseline**




Source: Company resources

**Exhibit 8: CS1-003 additional data: TR improvements from baseline**

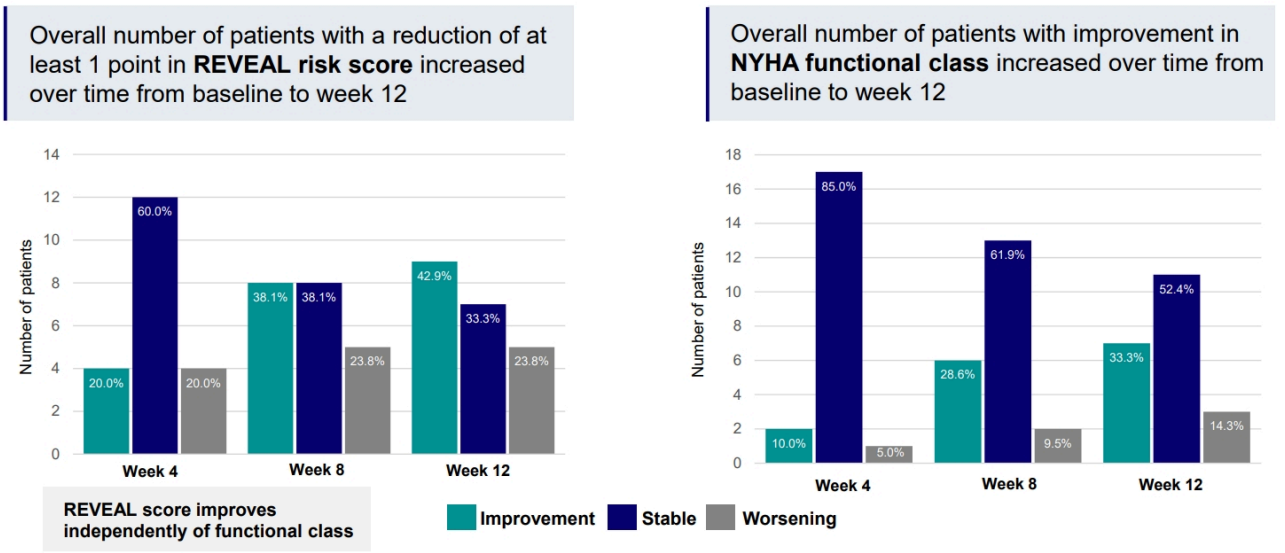



Source: Company resources

**PVR**, characterised by increased vascular resistance due to narrowing or blockage of blood vessels in the lungs, forcing the heart to work harder to pump blood through them. CS1 demonstrated marked improvement in a sub-set of early-stage patients. Management had previously communicated that 10 of the 21 patients who completed the Phase IIa study showed an improvement in PVR, with five showing significant improvement.

**REVEAL 2.0 risk score and the NYHA FC**, standard parameters used to assess disease severity in PAH. Management noted that CS1 showed a gradual improvement over time on these parameters during the 12-week study period. On the REVEAL risk score, while 20% of the patients reported an improvement (of at least one point on the 22-point scale) at week four, this proportion increased to 42.9% by week 12. Similarly, while 10% of the patients showed improvement in their NYHA FC at week four, 33.3% had noted an improvement by week 12 (Exhibit 9).

**Exhibit 9: Improvements in REVEAL risk scores and NYHA FCs**




Source: Company resources

**Quality of life.** CS1 was able to demonstrate improvement in this parameter as measured by PAH-SYMPACT and the Minnesota Living with Heart Failure Questionnaire (MLHFQ) (Exhibit 10). While PAH-SYMPACT is highly sensitive and specific to PAH, the MLFHQ is a self-administered questionnaire for patients and relates to heart failure.

Collectively, we view the trial results as highly encouraging for Cereno's HDACi programme, though we note that the results relate to only a small patient population, and therefore cannot be termed conclusive. However, it is our opinion that the observations provide early evidence of CS1's potentially disease-modifying properties, supporting the further development activities, testing efficacy in a larger population.

**Exhibit 10: CS1-003 quality of life outcomes**

| Minnesota Living With Heart Failure Questionnaire | PAH-SYMPACT Cognitive/Emotional Impacts | PAH-SYMPACT Physical Impacts | PAH-SYMPACT Cardiopulmonary Symptoms | PAH-SYMPACT Cardiovascular Symptoms |
|---|---|---|---|---|
| **71%** of the patients **improved** QoL (15/21) | **35%** of the patients **improved** C/E impacts (7/20) | **45%** of the patients **improved** physical impacts (9/20) | **50%** of the patients **improved** CP symptoms (9/18) | **50%** of the patients **improved** CV symptoms (9/18) |
| **76%** of the patients **improved** or had **stable** QoL (16/21) | **75%** of the patients **improved** or had **stable** C/E impacts (15/20) | **65%** of the patients **improved** or had **stable** physical impacts (13/20) | **61%** of the patients **improved** or had **stable** CP symptoms (11/18) | **83%** of the patients **improved** or had **stable** CV symptoms (15/18) |

Source: Company resources

## Regulatory green light for Phase IIb received

In December 2025, Cereno announced that it had been granted FDA clearance for a global Phase IIb trial, further testing CS1 in PAH. This will be a double-blind, randomised, placebo-controlled, dose-finding study (expected n=126) involving approximately 65 sites across the US, Europe and South America. It has been designed to compare the efficacy, safety and tolerability of CS1 to determine an optimal dose for Phase III (testing two doses, in combination with the standard of care) and to placebo. A notable feature of the trial design is the 36-week core treatment period, which we note is longer than the c 24-week duration typically employed in mid-stage PAH studies (eg sotatercept, seralutinib; CS1-003 was 12 weeks). The total trial duration will be 60 weeks, including re-randomisation at 36 weeks, where the placebo group will then receive CS1, with the CS1-treated patients either continuing on CS1 or switching to placebo. We view this design to be strategically driven, permitting all participants to receive CS1 at some point during the trial and allowing Cereno to evaluate previously observed disease-modifying signals (eg reverse vascular remodelling and improved right heart function) in a larger, controlled population.

The endpoints for the trial include change in PVR and six-minute walk distance(6MWD) through a withdrawal period at week 36, which are well-validated regulatory benchmarks in PAH. It will also assess biomarker changes, other measures of heart function, pharmacokinetics and patient-reported outcomes.

The trial is expected to commence in Q226, representing an important near-term milestone for the lead programme. Top-line results have been guided for late-2028 and will represent a significant inflection point for Cereno.

## Additional data from the expanded access programme in H126

In addition to the Phase IIb plans, CS1 is also running an expanded access programme (EAP) for patients who completed the 12-week treatment as part of the previous Phase IIa clinical study. The EAP, which includes 10 eligible patients from the 21-patient Phase IIa study, is being conducted under an FDA-approved protocol and is designed to provide insights on the long-term use of CS1 over a 12-month period. The first patient under the EAP was dosed in August 2024, and we understand that additional insights obtained may be used to support discussions with regulators and potential partners. In June 2025, Cereno presented an encouraging four-month follow-up update from the EAP. While precise details were not directly disclosed, management noted that the data (from a 10-patient cohort) aligned with the safety, tolerability and signals of efficacy observed in the Phase IIa trial. Additional details corresponding to 12 months of follow-up are anticipated in H126.

Note that the company is also undertaking a sub-study (with five to seven patients from the EAP) under its agreement with Fluidda, which will utilise the company's novel imaging technology to visualise the effect of CS1 on inducing long-term reverse remodelling. We expect insights from the 12-month follow-up under the EAP programme along with data from the Fluidda sub-study to help build understanding of long-term usage of CS1.

## PAH background and landscape

PAH is a rare form of a not so rare disease, and has a significant unmet need for safe, effective and disease-modifying treatment options. It is a complex and aggressive condition characterised by high blood pressure in the lungs, caused by increasing vasoconstriction (narrowing) and vasculature remodelling (structural changes to the blood vessel wall) of the distal pulmonary arteries, which makes the disease progressive in nature. Common symptoms include shortness

of breath, fatigue, dizziness and chest pain, which are associated with poor quality of life and significant morbidity and mortality as the disease progresses.

PAH has an estimated prevalence of 10 people per 100,000 in the worldwide population. Available data indicate that there are c 100,000 PAH cases in the US and Europe. The condition has been classified across four FCs by the NYHA and the WHO, based on limitations to physical activity, which is a key element in assessing patients with PAH and an important driver of treatment choice. The two classification systems are similar and used interchangeably (Exhibit 11).

**Exhibit 11: Functional assessment of PAH**

| WHO classification | | NYHA classification | |
|---|---|---|---|
| Class I | Patients with pulmonary hypertension but without resulting limitation of physical activity. Ordinary physical activity does not cause undue dyspnea (shortness of breath) or fatigue, chest pain, or near syncope (fainting). | Class I | No symptoms with ordinary physical activity. |
| Class II | Patients with pulmonary hypertension resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity causes undue dyspnea or fatigue, chest pain or near syncope. | Class II | Symptoms with ordinary activity. Slight limitation of activity. |
| Class III | Patients with pulmonary hypertension resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary activity causes undue dyspnea or fatigue, chest pain or near syncope | Class III | Symptoms with less than ordinary activity. Marked limitation of activity. |
| Class IV | Patients with pulmonary hypertension with inability to carry out any physical activity without symptoms. These patients manifest signs of right-heart failure. Dyspnea and/or fatigue may even be present at rest. Discomfort is increased by any physical activity. | Class IV | Symptoms with any activity or even at rest. |

Source: Medscape, Edison Investment Research

The current standard of care is a category of drugs termed vasodilators, which provide symptomatic relief by widening blood vessels, without modifying the underlying pulmonary vasculature. These drugs target one of the three pathways contributing to the pathogenesis of PAH (endothelin, nitric oxide and IP) and are classified under four distinct categories: endothelin receptor antagonists, phosphodiesterase type 5 inhibitors, soluble guanylate cyclase stimulators and IP analogues or IP receptor agonists. Despite these available treatments, mortality rates remain high (median survival of seven years versus three years prior to the availability of these treatments). Clinicians have highlighted an increasing need for triple-combination treatment, in the absence of which more than 50% of patients remain high risk, post-treatment (Exhibit 12). However, even with upfront triple therapy, the three-year mortality rate remains >20%. Moreover, c 50% of patients experience significant adverse events from the treatment, resulting in high discontinuation rates.

While several PAH-specific drugs have entered the market in the last two decades, they all target the same three pathways (endothelial, nitric oxide and IP). With growing interest on targeting the underlying cause of PAH, research has intensified towards developing potentially disease-modifying options. Some of the targeted mechanisms include bone morphogenetic protein signalling, tyrosine kinase receptors, epigenetic approaches, serotonin metabolism, oestrogen metabolism, extracellular matrices and angiogenesis.

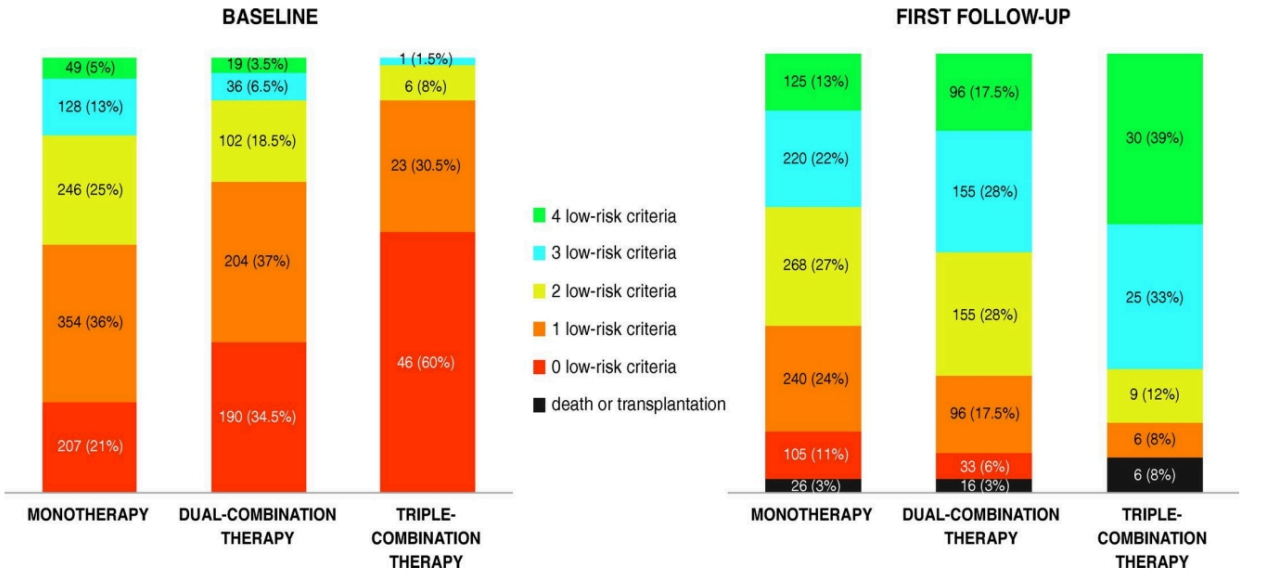
We highlight that a significant development in the field was the approval of Merck's Winrevair in March 2024, based on its pivotal Phase III STELLAR data involving 324 FC II and FC III patients, meeting the primary endpoint of improvement in 6MWD from baseline at 24 weeks. The drug works by decreasing the proliferation of cells in the pulmonary artery walls, targeting the underlying arterial stiffening that causes PAH, and is classified as a disease-modifier. Merck has also presented encouraging data from the ZENITH trial in 172 FC III and FC IV PAH patients, achieving its primary endpoint of time to clinical worsening to first morbidity or mortality event, suggesting it may be on track for a label expansion to include a broader patient population. We note, however, that Winrevair's tolerability concerns (especially around bleeding and haemoglobin/platelet changes) and price tag (annual list price of $240k) have received pushback from payors, with the company facing challenges in securing reimbursement in major geographies (with the exception of the US and Germany). For example, in August 2025, NHS England's appraisal body, NICE, issued provisional guidance recommending against reimbursing for PAH due to cost-effectiveness.

Another key candidate in development is Gossamer Bio's seralutinib, an inhaled platelet-derived growth factor receptor (PDGFRα and PDGFRβ) inhibitor/tyrosine kinase inhibitor. In December 2022, results from the Phase II TORREY trial were reported from 86 patients, whereby the primary endpoint was met, reporting a 14.3% improvement in PVR from baseline to week 24. However, it did not meet the secondary endpoint, the 6MWD, where the improvement from baseline was not statistically significant. This was attributed to the trial not being sufficiently powered and balanced for disease severity. In December 2023, the company initiated the Phase III PROSERA trial with a modified design (influenced by the Phase II results); results are expected in February 2026.

Given the current phase of clinical development and strength of data, we expect seralutinib to enter the market before CS1, if approved. However, we believe that CS1's favourable safety profile to date and its convenient dosing as an oral medication could be a key differentiator, compared to both sotatercept and seralutinib, if disease-modification is

proved. Moreover, given the heterogeneity of PAH and the different mechanism of actions of all three drugs, they may complement, rather than compete with one another. The benefit of combination treatments is already evident in PAH, and may hold true for other mechanisms of actions as well, in our opinion.

**Exhibit 12: Patient stratification on combination treatment in PAH**




Source: Company resources. Note: As presented by Raymond Benza, system director of pulmonary hypertension at Mount Sinai Icahn School of Medicine, at Cereno's October 2024 CMD.

# CS014: Phase I results lay robust foundation for Phase II

In July 2025, Cereno announced positive top-line results from its Phase I trial for its second clinical-stage asset, CS014. The study was conducted in two parts (single- and multiple-ascending oral doses), investigating safety, tolerability, pharmacokinetics and pharmacodynamics of the candidate in 48 healthy volunteers. The single-ascending dose (n=30) portion of the study was completed in February 2025, while the multiple-ascending dose portion was concluded in April 2025. Importantly, the results showed favourable outcomes in terms of safety and tolerability, with no participants withdrawing and only mild side effects observed, and no cases of serious adverse events.

Notably, CS014 also achieved levels in the bloodstream exceeding the projected threshold required to achieve maximal effects on the reversal of pulmonary vascular remodelling and fibrosis (based on non-clinical data), validating Cereno's approach to develop CS014 as a potential disease-modifier in IPF. Details of the Phase I results were presented at Pharmacology 2025 in December 2025. During the same period, results from a preclinical study testing CS014's HDAC inhibitory properties against VPA (in both in-vivo and in-vitro models) were published in the *Journal of Thrombosis and Hemostatis*. Study results noted that CS014 worked just as well as VPA at managing thrombosis after blood vessel injury and, more importantly, did so without increased hepatotoxicity and increased risk of bleeding, re-enforcing the drug's favourable safety profile.

The results from Phase I, in combination with additional data demonstrating a favourable impact on plexiform lesions in a preclinical research model, are expected to provide insights to support dose selection for the next stages of clinical development.

Overall, we believe this outcome lays a robust foundation for CS014, as management prepares for the subsequent stages of clinical development. We expect it to enter Phase II from 2027, and do not rule out the possibility of potential partnering discussions in the interim. According to the company, while the initial target will be IPF, the non-clinical and clinical data to date also suggest a broader potential in disease areas involving pulmonary vascular remodelling and fibrosis.

## IPF background and landscape

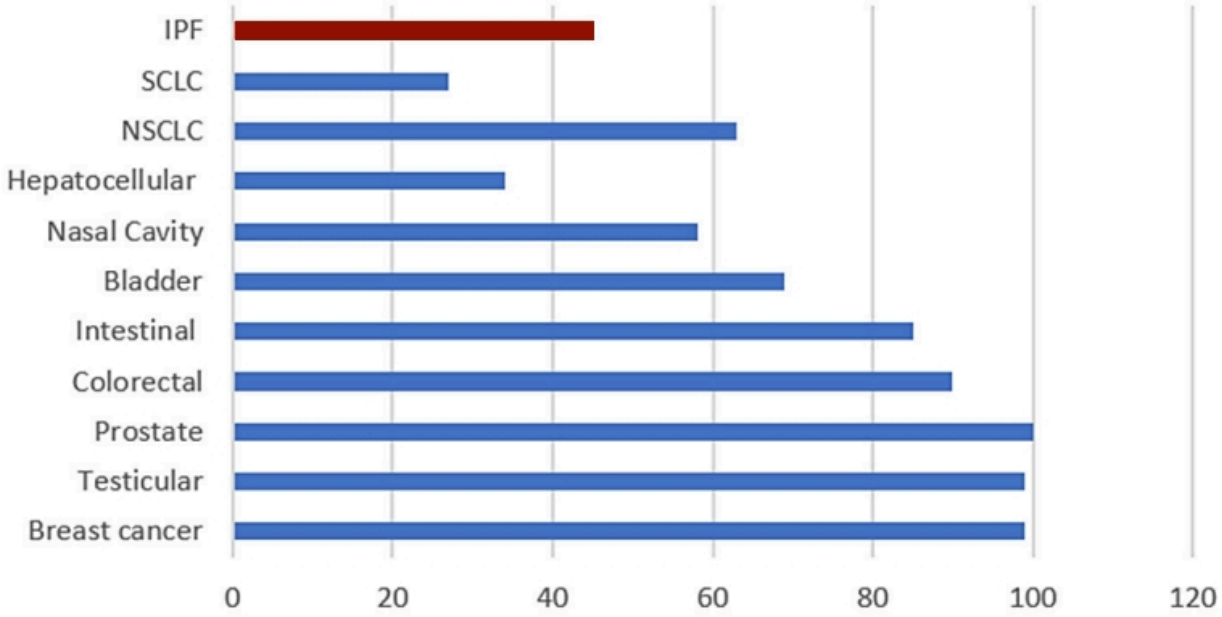
IPF is a serious chronic lung disease, characterised by the thickening of the lung tissue through the excessive production and deposition of extracellular matrix components (without any underlying reason, hence the term idiopathic), which worsens over time, leading to fibrosis (scarring). This causes difficulties in breathing, which often progress to life-

threatening conditions such as respiratory failure. IPF is the most common form of interstitial lung disease (ILD), a group of more than 200 conditions defined by progressive inflammation and fibrosis in the lungs, primarily around the alveoli. IPF accounts for c 20–50% of all ILDs, with a prevalence of c 300,000 across the US and Europe (EU4 plus the UK). The disease burden remains high with 30,000 to 50,000 new cases diagnosed in the US each year. Despite available treatments, life expectancy is generally limited to three to five years, with a five-year survival rate of c 45%, lower than several types of cancers, highlighting the significant unmet need in the space (Exhibit 13).

While idiopathic in nature, IPF generally affects men over the age of 50, with smokers at a higher risk. Viral infections, genetics, air pollution and certain workplace exposures also increase risk factors. Common symptoms include shortness of breath, dry cough, tiredness, weight loss, muscle aches and clubbing (widening and rounding) of the tips of the fingers or toes (a sign of poor oxygenation). These symptoms are similar to other lung conditions, making the diagnosis of IPF challenging, requiring a battery of tests, including imaging tests (eg X-rays, CT scans, high-resolution computed tomography), pulmonary function tests, cardiopulmonary exercise tests, lung diffusion tests and 6MWD tests.

IPF generally worsens over time, though progression can vary significantly across patients given the heterogenous nature of the condition. IPF patients also tend to present with co-morbidities such as pulmonary hypertension, gastroesophageal reflux disease (GERD), obstructive sleep apnoea and even lung cancer. Notably, pulmonary hypertension affects up to 50% of all IPF cases, and these patients tend to have even poorer prognoses.

**Exhibit 13: Five-year survival rate of common cancers compared to IPF**




Source: Company resources

Despite the progressive nature of IPF, treatments targeting underlying disease pathology are limited, with most options aimed at managing symptoms such as inflammation, cough and GERD. Until recently, only two anti-fibrotic drugs were approved (pirfenidone (brand name: Esbriet) and nintedanib (brand name: Ofev)), and c 70% of IPF patients have been treated with one of them. Ofev is a tyrosine kinase inhibitor that targets growth factors implicated in the proliferation of fibroblasts in pulmonary fibrosis. The drug was developed by Boehringer Ingelheim and approved for IPF in 2014 in the US, followed by Europe, and is patent protected to 2029. Ofev was also approved for the treatment of progressive pulmonary fibrosis in 2022 in patients who have failed standard management for fibrotic ILD, other than IPF. Esbriet is a synthetic pyridone drug that works by inhibiting the production and activity of fibroblasts by regulating the transforming growth factor-beta (TGFβ) and other growth factors. The drug was developed by InterMune, which was subsequently acquired by Roche in 2015 for $8.3bn. Esbriet was approved for the treatment of IPF in Europe in 2011 and in the US in 2014. We note that these drugs are approved for patients with mild, moderate and severe IPF and, while effective in slowing down the pace of scarring and deterioration in lung functioning, they are unable to halt or reverse disease progression. Further, the drugs are not associated with improvement in certain physical outcomes like day-to-day functioning, fatigue or the six-minute walk test. In addition, both drugs are associated with significant gastrointestinal side effects. While one-third of patients on Esbriet are affected by nausea, as many as two-third of patients on Ofev experience diarrhoea. According to published data, discontinuation rates within one year for Ofev and Esbriet were as high as 50% and 48.5%, respectively, and average survival rates remain low at three to five years.

In a key development, in October 2025, the FDA approved a new drug, nerandomilast (brand name: Jascayd), for

IPF, representing the first new therapy for the condition in over 10 years. Nerandomilast reported positive top-line data from its registrational Phase III FIBRONEER trial in September 2024, meeting its primary endpoint of absolute change from baseline in forced vital capacity at week 52 compared to placebo. While this is beneficial for IPF patients and encouraging for the field as a disease-modifier, we note that the drug has not demonstrated an ability to fully halt or reverse progression of the disease.

Despite the various ongoing trials in IPF and recent approval, we believe that novel treatment options offering the potential to halt or reverse disease progression will be of particular interest to the market. While CS014 is still in the early stages of development, should it demonstrate such signals in clinical trials, it could offer significant development, partnering and/or commercial potential. We add the caveat that IPF drug development is inherently risky, exemplified with numerous trial failures in the late stages of development in recent periods.

## CS585: Promise shown in preclinical research

CS585 was in-licensed from the University of Michigan in 2023, and the research collaboration is ongoing with the goal of transitioning the candidate into the clinic. It is an oral, selective and potent inhibitor of the IP receptor, currently undergoing preclinical research. We note that while a precise indication has not yet been determined for CS585, it has demonstrated potential as a treatment to prevent thrombosis without the increased risk of bleeding, offering promise in overcoming a key challenge with currently available anti-thrombotic medicines. Furthermore, it has also shown potential in addressing pulmonary hypertension, and management has communicated that rare diseases with high unmet medical needs, such as antiphospholipid syndrome (an autoimmune condition associated with the formation of thromboses), are being considered, in line with other drug candidates being developed by Cereno. Overall, the data from preclinical research for the candidate have been encouraging, and recognised with a publication in the journal *Blood*. More recently, additional updates have demonstrated that CS585 can inhibit platelet activation and the formation of clots for up to 24 hours after being administered, and shown that it is highly selective for its target, supporting its application in the sustained prevention of thrombosis.

We understand that preclinical research is set to continue throughout 2026, with plans for it to commence first-in-human clinical studies from 2027.

## Management team

**CEO: Sten R Sörensen.** Sten has been CEO of Cereno Scientific since 2015 and has extensive experience in pharma, biotech and finance industries. Prior to Cereno Scientific, he held senior positions in major pharma companies, including head of international marketing operations for the SEK10bn pharma portfolio at Monsanto and global marketing director for the SEK4bn portfolio of Secondary Prevention Products, Cardiovasculars at AstraZeneca. At Monsanto and AstraZeneca, he initiated two groundbreaking preventive survival studies in heart failure, RALES and MERIT-HF, both establishing a paradigm shift for mineralocorticoid receptor antagonism and beta-blocker drug therapies in heart failure, significantly improving quality of life and life expectancy. He was also a board member of Cereno Scientific between 2014 and 2016 and since 2024. Sten holds a bachelor's degree in chemistry from Lund University. He is the chair of SARomics Biostructure and a board member of SynAct Pharma.

In 2025, Sten was shortlisted for the CEO of the Year award in the prestigious European Lifestars Awards. See below the Edison executive interview we recently conducted with Sten.

**CFO: Eva Jagenheim.** Eva has broad experience across various roles within finance. This includes working as an accountant at PWC, a consultant at Arthur Andersen, and at companies of varying sizes across several industries. Most recently she was CFO at RLS Global, a medtech company listed on Nasdaq First North Growth Market. Eva has an MSc in business and economics from Växjö University and an MBA from Gothenburg Business School.

**CMO and head of R&D: Dr Rahul Agrawal.** Rahul is an experienced senior executive leader with a diverse background spanning big pharma and biotech. His expertise encompasses the entire value chain, including R&D and medical affairs, as well as commercial and strategy experience across various therapeutic areas such as cardiovascular, renal, respiratory and rare/orphan drugs, and he has launched seven drugs globally. His previous roles include CMO at Cardior, VP and global medicines leader at AstraZeneca, and global director of medical affairs and clinical development at Bayer HealthCare. He has an MD degree from the Free University of Berlin, Germany, and Cornell University, New York, US, and is board-certified in cardiology, internal medicine and emergency medicine. Additionally, he holds an MBA from Buckinghamshire New University, UK.

**CSO: Dr Björn Dahlöf.** Björn has over 35 years of clinical experience, complemented by extensive experience in

cardiovascular research, pharmacology, drug development and clinical trials (all phases), and he has lectured in these areas internationally. He is an adviser to small and large pharmaceutical companies regarding drug development in all phases from preclinical development to larger lifecycle management studies after registration. He has initiated and led several major national and multinational mortality and morbidity studies that have had significance for guidelines in cardiovascular prevention and authored over 400 scientific publications. He is a medical doctor from the University of Gothenburg, internal medicine physician and associate professor at Sahlgrenska University Hospital, University of Gothenburg.

**Head of preclinical development: Nicholas Oakes.** Nicholas has more than 20 years of experience in the pharmaceutical industry in both efficacy and safety-related aspects of preclinical research to discover and develop new effective and safe medicines in metabolic, cardiovascular and renal disease areas. He holds a PhD in cardiovascular and metabolic research from the University of New South Wales, Sydney, Australia.

**Head of IR and communications: Tove Bergenholt.** Tove is a skilled professional in communications, investor relations and integrated marketing within life sciences and healthcare, specialised in public biotech companies headquartered in the Nordics. She has broad experience in milestone communications at various points of the business and product life cycle, from disease awareness and patient adherence to brand building and crisis and issues management in global, regional and local markets. Her previous experience in global healthcare includes PR for AstraZeneca, Merck and Bayer. Tove holds an MSc in digital business management from Manchester Metropolitan University, UK, and a dual BSc in business administration with specialisation in business development and accounting from the University of Borås.

**Cereno Scientific – executive interview**


Source: Edison Investment Research

## Sensitivities

With its strategic focus on rare diseases and the potential to deliver disease-modifying therapies, Cereno provides exposure to structurally attractive yet underserved markets, including PAH and IPF, each offering meaningful long-term commercial opportunities. While recent financing and regulatory progress have reduced near-term execution risk, the investment thesis remains highly sensitive to clinical differentiation, competitive dynamics and capital market conditions. Key risks and sensitivities are outlined below.

**Binary risks related to a concentrated portfolio.** Cereno's valuation is highly concentrated, with CS1 accounting for approximately 84% of group rNPV, rendering our valuation particularly sensitive to clinical trial outcomes. Although proof-of-concept has been established through encouraging Phase IIa data, the upcoming Phase IIb study (expected to

commence in Q226) represents the first robust test of efficacy in a larger, controlled setting. Failure to reproduce early efficacy signals, demonstrate clinically meaningful benefit, or substantiate a disease-modifying effect could materially impair the company's overall valuation.

**Evolving therapeutic landscape and competitive intensity.** The PAH landscape is evolving rapidly with development focus increasing shifting to treatments targeting the underlying disease biology, most notably Winrevair and late-stage assets such as seralutinib. We view CS1's favourable safety profile to date, oral dosing convenience and disease-modifying positioning as important potential differentiators. However, to realise its full commercial potential, CS1 will need to demonstrate incremental efficacy beyond current standards of care. In the event of regulatory approval, CS1 is likely to be positioned initially as an add-on combination therapy, requiring clear evidence of compatibility with existing treatments, including the absence of clinically meaningful drug-drug interactions, to secure regulatory and payor support.

The second focus indication, IPF, remains one of the most challenging disease areas to target, given multiple overlapping pathogenic pathways and biological heterogeneity between patients. Only three therapies are currently approved, with all three focusing on slowing disease progression rather than reversal. To achieve meaningful differentiation, CS014 will need to demonstrate benefit beyond incremental slowing of disease decline. While preclinical data suggest CS014 can reverse fibrosis and promote reverse remodelling of the pulmonary vasculature, including plexiform lesions, these findings must be replicated in well-controlled clinical studies to support both regulatory approval and commercial relevance.

**Financing and liquidity risk.** The recent SEK665m financing de-risks Cereno's near-term development plan and extends the cash runway into Q427. Nevertheless, we expect additional capital will be required ahead of a potential out-licensing deal. Furthermore, access to certain loan tranches and warrant conversions is partially contingent on share price performance, introducing an element of execution and market risk. Any future equity-based financing would add to dilution risk.

**Partnering and deal execution risk.** Our investment case for Cereno assumes out-licensing deals for CS1 and CS014 in 2029 ahead of the initiation of pivotal Phase III studies. While these deals can range from smaller regional deals to a single global licensing deal, for simplicity we currently model large global out-licensing deals for both CS1 and CS014. However, the timing, scope and economics of such deals remain uncertain, particularly in the context of increasing competitive intensity. Successful execution will depend on both programmes demonstrating clear clinical differentiation and compelling benefit-risk profiles relative to existing and emerging therapies.

# Valuation

We continue to value Cereno using a rNPV methodology for its two clinical-stage programmes, CS1 and CS014, while updating our assumptions to reflect:

- the expected pace of clinical progression,
- recent regulatory and clinical newsflow, and
- evolving dynamics within the competitive landscape.

A flat discount rate of 12.5% is applied across both assets, consistent with Edison's standard approach for clinical-stage development programmes.

## CS1: Lead value driver approaching a key inflection point

Lead asset CS1, targeting PAH, continues to be the core driver of our valuation for the company. The programme is entering a pivotal phase in its development trajectory, with the first patient in the Phase IIb study expected to be recruited in Q226. While detailed patient inclusion criteria have yet to be disclosed, we currently assume the trial will enrol WHO FC II–III PAH patients receiving background therapy (including standard-of-care vasodilator therapy with a combination of endothelin receptor antagonists, PDE-5 inhibitors and prostacyclin pathway agents). This positioning broadly mirrors the commercial label for Winrevair (sotatercept) and the target population in the Phase III PROSERA study of seralutinib, supporting the relevance of peer benchmarking.

**Pricing.** For the US market, we assume a list price of $250k per patient per year, and apply a 30% payor discount, resulting in an effective net price of US$175k. This is closely benchmarked to Winrevair's estimated annual treatment cost of c $240k. In Europe, we assume a more conservative and competitive pricing environment, modelling an annual

treatment cost of US$62,500.

**Target population and market penetration.** Based on prevalence estimates across the US and the EU, adjusted for life expectancy and incidence, we estimate a target PAH population of approximately 115,000 patients across these geographies. We assume that c 90% of patients are diagnosed, of whom 85–90% seek treatment. We further assume that 77% of all patients seeking treatment are classified as FC II and FC III, which we expect will be the initial target patient sub-set for CS1.

Guided by the strong early market uptake of Winrevair following its March 2024 launch, we increase our peak second-line penetration assumption for CS1 to 25%, from 20% previously. This upward revision is made despite the probability that seralutinib reaches the market ahead of CS1, with PROSERA Phase III top-line data expected in February 2026. Our assumption reflects the significant and persistent unmet need in PAH, particularly for therapies that modify underlying disease biology rather than provide incremental symptomatic benefit. This results in our peak sales estimate for CS1 increasing to $2.8bn, from $2.0bn previously. While this is more conservative than the $5–8bn peak sales expectations for Winrevair, the latter includes upside from label expansion opportunities (in FC III–IV patients and earlier treatment lines in PAH as well as other related conditions) following supportive Phase III findings from the ZENITH and HYPERION trials. Should CS1's scope be expanded to include broader patient sub-sets, the commercial potential could rise commensurately.

Note that CS1 benefits from orphan drug designation in both the US and Europe, conferring seven and 10 years of market exclusivity, respectively, post-approval, an important consideration for long-term value capture.

**PoS and development timeline.** Following FDA clearance of the Phase IIb study design, we raise our PoS for CS1 to 50%, from 45%. This is modestly above the typical 40% PoS for Phase II cardiovascular assets, which we believe is justified by the positive Phase IIa clinical readout and early signals suggestive of disease modification, a key differentiator versus existing therapies. However, we push back our assumed commercial launch by one year to 2032 (from 2031), reflecting the 36-week primary treatment duration in Phase IIb (versus the 24 weeks we had assumed previously), which may be replicated in Phase III and extend overall development timelines.

**Commercial assumptions.** We assume Cereno will self-fund and sponsor the Phase IIb study for CS1 (top-line results expected in Q428) before entering into an out-licensing agreement in 2029, at which point the partner would assume responsibility for late-stage clinical development and commercialisation. As noted above, while regional licensing deals are a possibility, we currently assume a single global out-licensing deal. We estimate the Phase IIb programme will cost approximately $30m (c SEK275m), with expenditure spread across 2026–28. Note that, given the ongoing partnering discussions, we do not rule out the possibility of a deal being signed prior to Phase II completion.

We continue to model a total deal value of $2.0bn for CS1, but increase the assumed upfront payment to $150m, from $100m previously, reflecting reduced development risk following Phase IIb initiation and increasing strategic interest in disease-modifying PAH assets. We assume a flat 15% royalty on net sales, alongside a 30:70 split between development and regulatory and commercial milestones. By way of comparison, Chiesi Pharma acquired ex-US commercial rights to seralutinib in May 2024 in a transaction valued at $486m, with royalties in the mid-to-high teens on sales outside the US.

Overall, we assign an rNPV of SEK5.6bn (SEK18 per share) to CS1, representing c 84% of our current valuation for Cereno, underscoring the asset's centrality to the investment case.

## CS014: Approaching Phase II, albeit with a slight delay

Cereno' second clinical-stage asset, CS014, is targeting IPF, which despite available treatments remains a serious chronic lung disease with a five-year survival rate lower than 50%. Given management's prioritisation of CS1 in 2026, we now assume a modest shift in CS014's development timeline, estimating the Phase II protocol submission in mid-2026 and trial initiation in H127 (from H126 previously). All other core assumptions remain broadly unchanged and are outlined below.

**Target population and market penetration.** Based on prevalence estimates across the US and the EU, adjusted for life expectancy and incidence, we estimate a target IPF population of approximately 300,000 patients across these geographies. We assume that c 90% of patients are diagnosed, of whom 75% seek treatment. Drawing on real-world utilisation data for approved anti-fibrotic therapies (Ofev and Esbriet), we further assume that c 70% of treated patients would be eligible for CS014.

We maintain a conservative peak penetration assumption of 15% across both geographies. Following the approval of Boehringer Ingelheim's PDE-4B inhibitor, Jascayd, in October 2025, we will continue to monitor how the treatment landscape evolves and reassess our assumptions accordingly. As noted previously, Jascayd is positioned as an anti-fibrotic and anti-inflammatory agent aimed at slowing disease progression and preserving lung function. Should CS014

demonstrate disease reversal or a sustained halt in disease progression, it could command meaningfully higher market share, reflecting its differentiated positioning.

**Pricing.** We continue to assume a list price of $150k annually for CS1 but now reduce the payor discount to 50% from 70% previously (effective annual treatment price of $105k) to reflect the $194k annual treatment cost for Jascayd, which is materially higher than the c $110k annual treatment cost for Ofev and Esbriet. For the EU, we assume an effective treatment price of $52,500 per year. We assume a 2% year-on-year growth in treatment price.

**Clinical timeline and peak sales.** As noted above, we now expect the Phase II study to commence in H127, with completion in 2029. We assume that further development and eventual commercialisation will be undertaken under a partnership or licensing agreement. Our model assumes a market launch in 2033, with peak sales of c $1.7bn achieved by 2040. Given IPF's classification as a rare disease, we assume orphan drug designation for CS014, conferring seven years of market exclusivity in the US and 10 years in Europe.

**R&D costs.** We estimate Phase II R&D expenditure of c $15m, based on a trial size of c 100 patients and an assumed per-patient cost of $150k. For context, Jascayd's Phase II IPF trial enrolled 147 patients.

**Commercial assumptions**. We assume that CS014 will be out-licensed following the completion of Phase II studies and model a partnering agreement in 2029, with a total deal value of $1.5bn, including an upfront payment of $150m. We also assume a 15% royalty rate on sales.

**PoS.** We keep the PoS unchanged at 20% ahead of the Phase II trial initiation, reflecting the high clinical risk inherent to IPF drug development.

## CS585: Preparing for clinical entry

Cereno's remaining pipeline asset, CS585, is an oral and selective prostacyclin receptor agonist currently in the preclinical stage of development. While it has not yet been assigned a specific target indication, it has shown promise in thrombosis prevention without increased risk of bleeding in preclinical studies. According to the last available information, the asset is expected to enter the clinic in 2027. Pending clinical progression and clarity on the target indication, we continue to exclude CS585 from our valuation but note the potential for incremental upside on inclusion.

In addition to the aforementioned adjustments, our valuation also incorporates Cereno's estimated end-FY25 net debt position of SEK126.1m. This includes the SEK5m cash inflow from warrant conversions in January 2026 (discussed in more detail in the Financials section below). Reflecting the above, our overall valuation for Cereno adjusts to SEK6.6bn or SEK21.1 per share (from SEK5.4bn or SEK17.5 per share previously). This represents a c 3x upside from current trading, indicating material upside potential to be unlocked. Exhibit 14 presents the breakdown of our rNPV valuation for the company.

**Exhibit 14: Cereno's rNPV valuation**

| Asset | Indication | Development phase | Launch | Peak sales ($m) | Peak sales year | NPV (SEKm) | Probability | rNPV (SEKm) | rNPV/share (SEK) |
|---|---|---|---|---|---|---|---|---|---|
| CS1 | PAH | Phase IIb-ready | 2032 | 2,765 | 2039 | 11,286.8 | 50% | 5,643.4 | 18.1 |
| CS014 | IPF | Phase II-ready | 2033 | 1,733 | 2040 | 5,287.8 | 20% | 1,057.6 | 3.4 |
| **Total** | | | | | | 16,574.6 | | 6,700.9 | 21.5 |
| Estimated pro-forma net cash/(debt) at 31 December 2025 | | | | | | | | (126.1) | (0.4) |
| **Valuation** | | | | | | | | **6,574.8** | **21.1** |

Source: Edison Investment Research. Note: The per-share valuation is based on outstanding shares of 311.7m.

# Financials

## SEK665m financing package supports upcoming clinical milestones

In December 2025, Cereno announced a comprehensive financing package of up to SEK665m, designed to fund the company through its next major clinical inflection points. The structure combines equity, convertible debt, loan facilities and warrants, and comprises:

- SEK100m via a directed share issue,

- SEK175m in convertible debt (conversion price: SEK10/unit),
- up to SEK175m in loan facilities, and
- up to SEK215m from warrant exercises (conversion prices: SEK10/unit and SEK12/unit, respectively).

This latest financing package was arranged with Fenja Capital and its affiliated entity (Fenja: 71.4%, affiliate: 28.6%), reinforcing continued institutional support. As part of the agreement, Cereno will refinance its existing SEK180m loan, consisting of SEK25m owed to Venusat and SAJ Finans and SEK155m to Fenja Capital and Arena Investors.

We note that while proceeds from the directed equity issue and convertible debt are immediately available, access to the SEK175m loan facility is staged and will be available between 1 April 2026 and 30 June 2027. Drawdowns are contingent on predefined financial conditions and are linked to the conversion and divestment of the convertible debt, with issuance capped at up to 5m shares per quarter through Q127 (maximum 17.5m shares). This structure implicitly requires the share price to be at or above SEK10 at the point of conversion, a threshold we view as achievable, particularly as CS1 advances into Phase IIb. While the financing includes attached warrants, we view the premium conversion terms as providing a favourable risk-reward trade-off and as evidence of constructive investor sentiment toward Cereno's medium-term outlook.

## Recent warrant conversions add incremental liquidity

In January 2026, Cereno received approximately SEK5m from the conversion of 728,957 warrants by Arena Investors at SEK6.82 per share, following an earlier conversion of 600,000 warrants in November 2025, which generated SEK4m. We remind readers that a total of 5.75m warrants were issued to Fenja Capital and Arena Investors under the November 2024 financing (split 45:55, respectively), of which 4.4m warrants remain outstanding. These can be exercised until 30 April 2029, representing potential additional proceeds of up to SEK30m.

## Runway extended into Q427

Despite the inherently dilutive nature of these raises and warrants, we view them as necessary investments to underpin long-term value creation, with the enhanced liquidity and balance-sheet flexibility outweighing near-term dilution concerns. Management has guided that the current financing extends the cash runway into Q427. Assuming clinical execution remains on track, we believe Cereno may have scope to refinance or restructure elements of the facility, potentially extending funding coverage through CS1 Phase IIb top-line readouts in Q428.

Under the assumption that the full SEK665m is utilised by 2027/28 (including full conversion of the convertible debt and exercise of all warrants), we estimate Cereno will require an additional c SEK100m raise in 2028, ahead of a potential out-licensing transaction for CS1 in 2029, which would then provide a meaningful upfront cash inflow.

## Exhibit 15: Financial summary

| Accounts: K3, Yr end: December 31, SEK:000s | 2022 | 2023 | 2024 | 2025e | 2026e |
|---|---|---|---|---|---|
| **PROFIT & LOSS** | | | | | |
| **Net sales** | **0** | **0** | **0** | **0** | **0** |
| Capitalised work for own account | 57,538 | 49,277 | 80,903 | 50,000 | 170,000 |
| Total revenues | 57,538 | 49,277 | 80,903 | 50,000 | 170,000 |
| Total operating expenses | (85,037) | (93,927) | (156,739) | (113,118) | (234,165) |
| R&D and other expenses | (76,620) | (71,152) | (128,675) | (83,717) | (204,054) |
| *Of which - R&D expenses* | *(57,538)* | *(49,277)* | *(80,903)* | *(50,000)* | *(170,000)* |
| *Of which - other expenses* | *(18,899)* | *(21,658)* | *(46,880)* | *(32,816)* | *(33,144)* |
| Personnel costs | (7,514) | (18,763) | (26,108) | (29,122) | (30,111) |
| Other operating items | (903) | (4,012) | (1,956) | (279) | 0 |
| Operating income (reported) | (27,499) | (44,650) | (75,836) | (63,118) | (64,165) |
| EBITDA (normalized) | (27,485) | (44,636) | (75,549) | (62,398) | (63,877) |
| Finance income/(expense) | (149) | (3,456) | (23,690) | (32,300) | (9,369) |
| Profit before tax (reported) | (27,649) | (48,106) | (99,526) | (95,418) | (73,534) |
| Profit before tax (normalised) | (27,649) | (46,436) | (98,106) | (95,418) | (73,534) |
| Income tax expense (includes exceptionals) | (6) | 0 | 0 | 0 | 0 |
| Net income (reported) | (27,654) | (48,106) | (99,526) | (95,418) | (73,534) |
| Net income (normalised) | (27,654) | (46,436) | (98,106) | (95,418) | (73,534) |
| End of period number of shares, '000 | 137,515 | 233,775 | 281,702 | 295,917 | 295,917 |
| Basic EPS (SEK) | (0.20) | (0.21) | (0.35) | (0.32) | (0.25) |
| Adjusted EPS (SEK) | (0.20) | (0.20) | (0.35) | (0.32) | (0.25) |
| | | | | | |
| **BALANCE SHEET** | | | | | |
| Intangible Assets | 146,987 | 196,264 | 277,167 | 327,167 | 497,167 |
| Fixtures, tools and installation | 29 | 14 | 3,599 | 2,879 | 2,591 |
| Other long-term receivables | 10 | 9 | 10 | 10 | 10 |
| Total non-current assets | 147,025 | 196,287 | 280,775 | 330,056 | 499,768 |
| Other receivables | 1,248 | 1,124 | 2,880 | 1,601 | 2,465 |
| Prepaid expenses and accrued income | 335 | 407 | 2,540 | 2,540 | 2,540 |
| Cash and bank balance | 67,046 | 87,169 | 127,578 | 134,275 | 180,940 |
| Total current assets | 68,629 | 88,699 | 132,997 | 138,416 | 185,944 |
| Accounts Payable | 9,411 | 6,930 | 13,951 | 10,068 | 20,842 |
| Other Current Liabilities | 4,331 | 16,231 | 17,495 | 17,495 | 17,495 |
| Short-term Debt | 0 | 0 | 0 | 0 | 0 |
| Total current liabilities | 13,742 | 23,162 | 31,446 | 27,563 | 38,337 |
| Long-term Debt | 0 | 45,000 | 190,000 | 265,000 | 540,000 |
| Other debt | 400 | 400 | 400 | 400 | 400 |
| Total non-current liabilities | 400 | 45,400 | 190,400 | 265,400 | 540,400 |
| Equity attributable to company | 201,511 | 216,424 | 191,926 | 175,508 | 106,975 |
| | | | | | |
| **CASH FLOW STATEMENT** | | | | | |
| Net profit | (27,654) | (48,106) | (99,526) | (95,418) | (73,534) |
| Depreciation | 14 | 14 | 287 | 720 | 288 |
| Translation difference | (90) | 34 | 0 | 0 | 0 |
| Accrued costs | 450 | 777 | 6 | 0 | 0 |
| Share based payments | 0 | 1,671 | 1,420 | 0 | 0 |
| Taxes paid | (4) | 0 | 0 | 0 | 0 |
| Movements in working capital | 8,669 | 8,695 | (5,609) | (2,604) | 9,911 |
| Cash from operations (CFO) | (18,615) | (36,915) | (103,422) | (97,303) | (63,335) |
| Purchase of intangible assets | (57,538) | (49,277) | (80,903) | (50,000) | (170,000) |
| Purchase of PPE | 0 | 0 | (3,871) | 0 | 0 |
| Cash used in investing activities (CFIA) | (57,538) | (49,277) | (84,774) | (50,000) | (170,000) |
| Loans received | 0 | 45,000 | 245,000 | 350,000 | 275,000 |
| Loan repayments | (5,000) | 0 | (90,000) | (200,000) | 0 |
| Equity issued | 58,791 | 61,315 | 73,605 | 0 | 0 |
| Other Financing Cash Flows | (226) | 0 | 0 | 4,000 | 5,000 |
| Cash from financing activities (CFF) | 53,564 | 106,315 | 228,605 | 154,000 | 280,000 |
| Cash and equivalents at beginning of period | 89,635 | 67,046 | 87,169 | 127,578 | 134,275 |
| Increase/(decrease) in cash and equivalents | (22,589) | 20,123 | 40,409 | 6,697 | 46,665 |
| Cash and equivalents at end of period | 67,046 | 87,169 | 127,578 | 134,275 | 180,940 |
| Net (debt)/cash | 66,646 | 41,769 | (62,822) | (131,125) | (359,460) |

Source: Company documents, Edison Investment Research

## Contact details

Cereno Scientific AB
BioVentureHub
Pepparedsleden 1
431 83 Mölndal, Sverige
Gothenburg, Sweden
https://cerenoscientific.com/

## Revenue by geography

N/A

## Management team

### CEO: Sten R Sörensen

Sten R Sörensen has been CEO of Cereno Scientific since 2015 and has extensive experience in the pharma, biotech and finance industries. Prior to Cereno Scientific, he held senior positions in major pharma, including head of international marketing operations for the SEK10bn pharma portfolio at Monsanto and global marketing director for the SEK4bn portfolio of Secondary Prevention Products, Cardiovasculars at AstraZeneca. At Monsanto and AstraZeneca, he initiated two groundbreaking preventive survival studies in heart failure, RALES and MERIT-HF, both establishing a paradigm shift for mineralocorticoid receptor antagonism and beta-blocker drug therapies in heart failure, significantly improving quality of life and life expectancy. He was also a board member of Cereno Scientific between 2014 and 2016 and since 2024. Sten holds a bachelor's degree in chemistry from Lund University. He is the chair of SARomics Biostructure and a board member of SynAct Pharma.

### CFO: Eva Jagenheim

Eva Jagenheim has broad experience of various roles within finance, including as an accountant at PWC, a consultant at Arthur Andersen, and at companies of varying sizes across several industries. She most recently worked as CFO at RLS Global, a medtech company listed on Nasdaq First North Growth Market. Eva has an MSc in business and economics from Växjö University and an MBA from Gothenburg Business School.

### CMO and head of R&D: Dr Rahul Agrawal

Dr Rahul Agrawal is an experienced senior executive leader with a diverse background spanning big pharma and biotech. His expertise encompasses experience across various therapeutic areas such as cardiovascular, renal, respiratory and rare/orphan drugs, and he has launched seven drugs globally. Previous roles include CMO at Cardior, VP and global medicines leader at AstraZeneca, and global director of medical affairs and clinical development at Bayer HealthCare. He has an MD degree from the Free University of Berlin, Germany and Cornell University, New York, US, and is board-certified in cardiology, internal medicine and emergency medicine. Additionally, he holds an MBA from Buckinghamshire New University, UK.

### CSO: Dr Björn Dahlöf

Björn Dahlöf has over 35 years of clinical experience, complemented by extensive experience in cardiovascular research, pharmacology, drug development and clinical trials (all phases), and has lectured in these areas internationally. He is an adviser to small and large pharmaceutical companies regarding drug development in all phases from preclinical development to larger lifecycle management studies after registration. Björn has initiated and led several major national and multinational mortality and morbidity studies that have had significance for guidelines in cardiovascular prevention and has authored over 400 scientific publications. He is a medical doctor from the University of Gothenburg, internal medicine physician and associate professor at Sahlgrenska University Hospital, University of Gothenburg.

### Head of preclinical development: Nicholas Oakes

Nicholas Oakes has more than 20 years of experience in the pharmaceutical industry in both efficacy and safety-related aspects of preclinical research to discover and develop new effective and safe medicines in metabolic, cardiovascular and renal disease areas. He holds a PhD in cardiovascular and metabolic research from the University of New South Wales, Sydney, Australia.

### Head of IR and communications: Tove Bergenholt

Tove Bergenholt is a skilled professional in communications, investor relations and integrated marketing within life sciences and healthcare, specialised in public biotech companies headquartered in the Nordics. She has broad experience in milestone communications at various points of the business and product life cycle, from disease awareness and patient adherence to brand building and crisis and issues management in global, regional and local markets. Her previous experience in global healthcare includes PR for AstraZeneca, Merck and Bayer. Tove holds an MSc in digital business management from Manchester Metropolitan University, UK, and a dual BSc in business administration with specialisation in business development and accounting from the University of Borås.

## Principal shareholders

| | % |
|---|---|
| Försäkringsaktiebolaget Avanza Pension | 15.1 |
| Myrlid As | 5.4 |
| Ejlegård Andreas | 1.3 |
| HANDELSBANKEN LIV FÖRSÄKRINGSAKTIEBOLAG | 1.1 |
| Butt Jan | 1.1 |
| FRANK FREDRIK | 1.1 |
| Swedbank Försäkring AB | 0.9 |
| GEVRYIE DORY | 0.8 |
| DNB Bank ASA | 0.8 |
| Jern Claes Sverker | 0.6 |

Notes:

1. Shareholdings as of 28 November 2025.
2. CEO Sten R Sörensen has holdings both personally and via wholly owned companies. These holdings are not combined in the formal owner lists by Euroclear. Several members of management and the board also have a substantial number of warrants.

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**London | New York | Frankfurt**
20 Red Lion Street
London, WC1R 4PS
United Kingdom