

# Cereno Scientific

FY25 results

## Inflection points coming into view

Cereno Scientific's **FY25 results** mark a strategically productive period, with multiple value inflection points coming into focus in FY26. With the Phase IIb start for lead asset CS1 expected in Q226, we anticipate a step-up in clinical activity and partnering momentum. We view CS014's pivot to **PH-ILD** from IPF as a rational move given the increasingly crowded IPF landscape and the more attractive, underpenetrated PH-ILD market, where treatment options remain limited. Importantly, CS014's anti-fibrotic and reverse-remodelling profile is well aligned with PH-ILD pathobiology and could offer meaningful differentiation. Recent regulatory clearance for a Phase I PK study, circumvents the need for additional safety and Phase IIa studies and expedites progression to Phase IIb, planned for Q127. Assuming full utilisation of the up to SEK665m November 2025 financing (SEK270m received to date), we forecast a cash runway into Q427, in line with guidance. Reflecting the revised CS014 strategy and updated net debt, our valuation moves to SEK21.3/share, from SEK21.1/share previously.

Year end	Revenue (SEKm)	PBT (SEKm)	EPS (SEK)	DPS (SEK)	P/E (x)	Yield (%)
12/24	0.0	(98.1)	(0.35)	0.00	N/A	N/A
12/25	0.0	(117.8)	(0.38)	0.00	N/A	N/A
12/26e	0.0	(92.3)	(0.30)	0.00	N/A	N/A
12/27e	0.0	(138.5)	(0.45)	0.00	N/A	N/A

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

## CS1: Next value trigger in sight

With Phase IIb initiation expected in Q226, backed by an optimised trial design, CS1 is approaching a key clinical and strategic inflection point. The recent Phase III failure of seralutinib underscores the high efficacy bar in PAH and the limited pool of disease-modifying assets, which in our view strengthens CS1's positioning. As the most advanced HDAC inhibitor and potentially disease modifying treatment in the clinic now, successful execution of Phase IIb would be a major value catalyst (top-line results in Q428), with scope to reframe the competitive landscape.

## CS014: Pivot to PH-ILD a rational move

We see the merits of pivoting to pulmonary hypertension associated with interstitial lung disease (PH-ILD), which has a clear unmet need, high morbidity and limited therapeutic options beyond inhaled vasodilators. We see a strong mechanistic fit for CS014 with its dual anti-fibrotic and reverse-remodelling properties targeting both the vascular and interstitial components of PH-ILD. We note however that the development pathway is complex, given patient heterogeneity and disease biology (>200 types of ILDs) and severity. Phase IIb initiation in Q127 will be a key catalyst, with study design details and early signals critical to establishing differentiation.

## Valuation: SEK6.6bn or SEK21.3 per share

We update our valuation for the FY25 results, latest net debt and CS014's pivot to PH-ILD. We keep peak penetration rates at 20% for CS014 but reduce PoS to 15% (from 20% for IPF), given the heterogeneity of ILD conditions. Our valuation adjusts to SEK6.6bn or SEK21.3/share (from SEK6.6bn or SEK21.1/share).

Healthcare

23 March 2026

**Price** **SEK6.30**  
**Market cap** **SEK1,868m**

Pro forma net cash/(debt) at 31 December 2025	SEK(45.4)m
Shares in issue	311.4m
Free float	93.0%
Code	CRNO B
Primary exchange	NGM
Secondary exchange	N/A

### Share price performance



%	1m	3m	12m
Abs	(8.7)	(17.3)	14.3
52-week high/low	SEK11.9	SEK5.1	

### 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. Phase IIb is expected to commence in Q226. Second asset CS014, a proprietary NCE and HDACi, is being developed for PH-ILD (Phase II-ready), and preclinical asset CS585 is likely to target rare thrombosis-related indications.

### Next events

CS1 EAP initial data	Q126
CS1 Phase IIb trial launch	Q226

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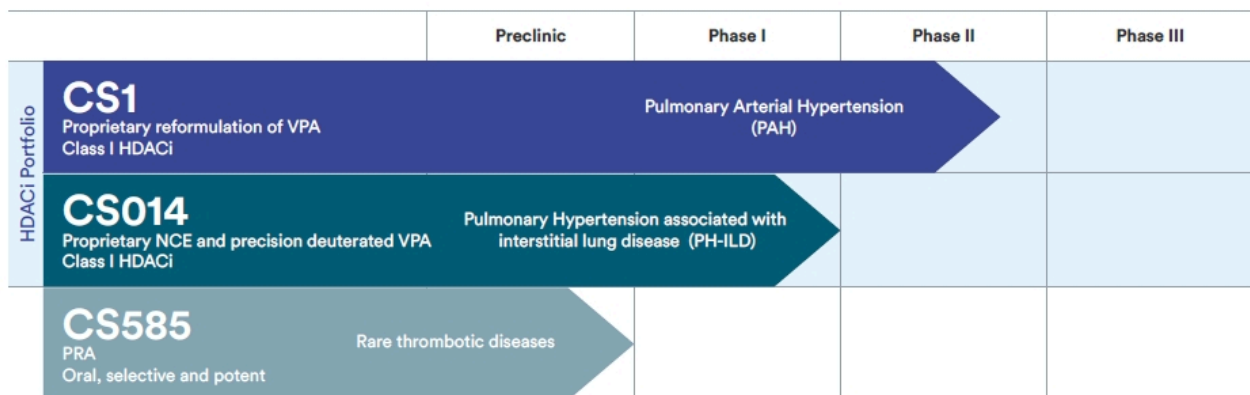
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## Advancing towards mid-stage clinical validation

Cereno Scientific's investment case is based on its differentiated pipeline centred on epigenetic modulation through histone deacetylase (HDAC) inhibition, targeting the underlying pathophysiology of rare cardiopulmonary diseases. With two clinical-stage assets, CS1 and CS014, targeting rare vascular and fibrotic pulmonary indications, the company's platform approach is aimed at disease modification rather than symptomatic treatment. FY25 was a year of tangible clinical and regulatory progress across the portfolio. Following positive Phase II data in late 2024, lead asset CS1 continued to advance the expanded access programme during FY25, with the year capped by the FDA clearance for its global Phase IIb trial in pulmonary arterial hypertension (PAH), expected to start in Q226. In parallel, next-generation HDAC inhibitor (HDACi) CS014 also progressed following successful completion of its Phase I safety study, with Phase II plans initially focused on idiopathic pulmonary fibrosis (IPF) before a recent strategic realignment to broaden the scope to PH-ILD, a group of conditions with an even higher unmet need. In addition to the two clinical programmes, Cereno's development pipeline also includes CS585, a prostacyclin (IP) receptor agonist targeting rare thrombotic diseases, without increased bleeding risk. The asset is currently undergoing preclinical development with work focused on advancing the asset toward first-in-human studies. We believe that CS585's clinical development may also focus on rare conditions, such as antiphospholipid syndrome (APS), as previously highlighted by the company. Exhibit 1 presents a schematic of Cereno's development pipeline.

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



Source: Cereno Scientific

With the transition from establishing safety and early proof of concept towards broader mid-stage clinical validation, we expect the company's efforts in FY26 to be centred on the CS1 Phase IIb trial (expected to commence patient recruitment in Q226) and preparing CS014 for Phase IIb with its revised PH-ILD focus. Management expects CS014 to commence the Phase IIb study in Q127, with efforts in FY26 focused on completing the [Phase I pharmacokinetic \(PK\) study](#) of CS014 for which the company recently received clearance from Swedish Medical Products Agency. The study will be an open-label, randomized, two-period crossover trial, evaluating steady-state PK following seven days of repeat oral dosing of CS014 compared to valproic acid (VPA). Notably the study design has been finalised based on feedback from a pre-IND meeting with the FDA and will remove the requirement for additional non-clinical safety and Phase IIa studies, should comparative bioavailability to VPA be demonstrated in the trial. The study is expected to complete by mid-2026, which should provide sufficient lead time to prepare for the initiation of a Phase IIb efficacy study within the targeted Q127 timeline.

We also expect partnering discussions for CS1 to intensify during the year, with management not ruling out a deal (either local, regional or global) potentially within FY26. Strategic interest in the PAH landscape remains strong, as reflected in GSK's recent \$950m acquisition of [35Pharma](#) for its Phase II-ready PAH asset HS235.

Financially, Cereno enters FY26 with a strengthened balance sheet following the financing package secured in November 2025. The company reported year-end gross cash of SEK74.6m, with access to additional capital through a SEK175m loan facility and up to SEK215m in potential warrant proceeds, alongside SEK45m of convertible debt already drawn in January 2026. Assuming utilisation of the available facilities across FY26 and FY27, we estimate the company has an operational runway into Q427, providing funding visibility to progress the CS1 Phase IIb study and advance the broader pipeline.

## CS1 Phase IIb initiation represents a key inflection

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### Clinical data to date supports further development

We view the initiation of the Phase IIb study for CS1 as the most significant upcoming catalyst for Cereno. CS1 is a patent-protected delayed immediate release formulation of valproic acid (VPA), being developed as a potential disease-modifier - reverse remodelling agent for PAH. The Phase IIb development plans for the asset are backed by encouraging top-line results from the prior Phase IIa CS1-003 trial (announced in [September 2024](#)), which investigated the lead candidate in PAH patients already on standard of care treatment with vasodilators.

The study, which enrolled 25 patients (21 evaluable for efficacy analysis), demonstrated a favourable safety profile for CS1, with no treatment-related serious adverse events reported and only two discontinuations due to adverse events unrelated to the drug. In our view, this safety profile provides material competitive differentiation to current PAH therapies, which are often associated with significant side effects and complex administration (for example sotatercept administration is associated with serious bleeding events, telangiectasia and thrombocytopenia as well as some cases of pericardial effusion). CS1's oral dosing could also represent a practical advantage relative to injectable or infusion-based therapies.

Although not powered for statistical efficacy, Phase IIa exploratory endpoints showed encouraging reverse remodelling signals. Improvements in the REVEAL risk score were observed in 43% of patients, with 71% reporting improved or stable risk status, increasing to 76% following additional analysis in early 2025. Similarly, 33% of patients demonstrated improvement in New York Heart Association (NYHA) functional class, with 86% reporting improvement or stability overall. Haemodynamic data collected via CardioMEMS showed sustained reductions in mean pulmonary arterial pressure (mPAP) in 67% of patients, with reductions of up to 5mmHg observed. Additional analyses released in early 2025 further supported CS1's disease-modifying potential, including improvements in right-ventricular global longitudinal strain (RVGLS), pulmonary vascular resistance (PVR) and tricuspid regurgitation. Improvements were also observed in quality-of-life measures. While we acknowledge that these results have been derived from a relatively small cohort, they nonetheless provide early evidence that CS1 may influence the underlying pathophysiology of PAH, a hypothesis that the Phase IIb study aims to validate (for further details on the CS1-003 trial results, we direct readers to our recent [outlook note](#)).

### Differentiated Phase IIb study design

Following a successful Type C meeting with the FDA in April 2025, Cereno received regulatory clearance for the Phase IIb global trial in December 2025. This will be the company's largest study to date and will be a double-blind, placebo-controlled trial evaluating two dose levels of CS1 to determine the optimal Phase III dose. The study will recruit 126 patients across 65 sites in the US, Europe and South America and will evaluate CS1's efficacy in combination with standard of care background therapy (versus placebo). The key study endpoints will be change in PVR tested via right-heart catheterisation, change in the six-minute walk distance (6MWD), biomarker changes, cardiac function measures, pharmacokinetics and patient-reported outcomes.

A notable feature of the study is the 36-week core treatment period, which is materially longer than the c 24-week duration typically employed in mid-stage PAH studies. The total trial duration will be 60 weeks, including re-randomisation at 36 weeks with the placebo group receiving CS1 and the treated patients either continuing on CS1 or switching to placebo. In our view, this design reflects a deliberate strategic choice, enabling all trial participants to receive active treatment at some point while also allowing the company to better capture potential disease-modifying effects such as reverse vascular remodelling and improvements in right-heart function in a larger controlled cohort. We also believe that the re-randomisation at 36 weeks (with some patients on the treatment arm moving to placebo) aligns with the latest regulatory and industry expectations that a withdrawal study may be required to demonstrate evidence of sustained treatment effects, a key requirement for establishing disease modification. This perspective is supported by a [February 2025 article](#) in The Lancet Respiratory Medicine. In the context of a potentially disease-modifying therapy in a complex indication such as PAH, we believe this measured approach is justified.

Patient recruitment is expected to begin in Q226, with top-line results anticipated in late 2028. While management has indicated that it may seek a partnering agreement prior to completion of the Phase IIb study, for modelling purposes we currently assume a licensing transaction in 2029, with a partner responsible for Phase III development and subsequent commercialisation.

## Upcoming EAP data to provide additional insights

Alongside the anticipated initiation of the Phase IIb trial, we also look forward to the forthcoming readouts from the Expanded Access Programme (EAP), which has now been completed. Data from the programme are expected to be reported in Q126 and Q226. As a reminder, the EAP enrolled 10 eligible patients from the 21-patient Phase IIa study and was designed to generate insights on the longer-term use of CS1 over a 12-month treatment period. While the relatively small cohort size limits the potential for statistically robust conclusions, we believe the programme may nonetheless provide valuable qualitative insights to support the ongoing development of CS1, particularly with respect to longer-term safety, tolerability and the durability of previously observed efficacy signals.

## PAH remains an area of unmet need with strong pharma interest

Despite available treatments, PAH remains an area of significant unmet need. Being a progressive disease (average life expectancy of 7.5 years even after treatment with the standard of care vasodilators) the overarching goal of new treatments is disease-modification or 'remission' as noted by Professor Marc Humbert, a key opinion leader, during Cereno's recent capital markets day. Given the progressive nature of the disease, a treatment that can reverse the underlying disease pathophysiology (particularly reverse remodelling of the pulmonary vasculature) or even halt progression will be highly coveted.

Strategic interest from large pharmaceutical companies in such potentially disease-modifying approaches remains strong. This is illustrated by the recent acquisition of 35Pharma by GSK for \$950m, centred on its Phase-II ready lead candidate HS235, targeting PAH. 35Pharma is a Canadian-based, private clinical-stage biopharma company focused on novel protein-based therapeutics. HS235 targets the activin receptor signalling pathway, the same biological axis validated by Sotatercept (Winrevair) in PAH, although HS235 has been designed to be more selective, which could potentially translate into a better safety profile. However, this needs to be demonstrated in larger patient studies as well to establish superior safety. We note that Merck's acquisition of Acceleron Pharma, the developer of sotatercept, for \$11.5bn in September 2021 represents the largest transaction in the PAH space to date and underscores the strategic value attributed to potential disease-modifying PAH therapies.

While the demand for innovative PAH treatments remains robust, it is also prudent to highlight that drug development remains inherently risky, as evidenced by the recent [Phase III failure](#) of Gossamer Bio's inhaled PDGFR/c-KIT kinase inhibitor, seralutinib. In the Phase III PROSERA study (n=390) seralutinib demonstrated a 13.3 metre improvement over placebo in the 6MWD test, but missed the 25 metre improvement threshold for statistical significance. This follows the news of Keros Therapeutics discontinuing its [Phase II TROPOS trial](#) evaluating its candidate ciboterecept (also an activin signalling inhibitor) in PAH in early 2025, following safety concerns, further illustrating the complexity of developing therapies targeting pulmonary vascular remodelling.

That said, we believe that CS1's favourable safety profile to date and its convenient dosing as an oral medication could be a key differentiator against competition if disease-modification is achieved. Following seralutinib's setback, CS1 now represents one of the most advanced, potentially disease-modifying therapies for PAH currently in clinical development. Successful execution of the upcoming Phase IIb trial could therefore represent a significant inflection point for Cereno and has the potential to materially reshape the competitive landscape in PAH.

## CS014's PH-ILD pivot a strategically sound decision

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Cereno's second clinical-stage asset, CS014, like its lead programme CS1, is an HDACi. However, CS014 represents a novel precision deuterated analogue of VPA and is classified as a new chemical entity. Based on preclinical and early clinical data generated to date, the compound shows potential activity in disease areas characterised by pulmonary vascular remodelling and fibrosis, supporting its evaluation in conditions such as PH-ILD.

CS014 successfully completed its Phase I trial in July 2025, with positive top-line results. The study enrolled 48 healthy volunteers across single-ascending dose (n=30) and multiple-ascending dose cohorts, assessing safety, tolerability, pharmacokinetics and pharmacodynamics. The programme demonstrated a favourable safety and tolerability profile, with no serious adverse events, no withdrawals and only mild side effects reported, that resolved fully on their own. Importantly, systemic drug exposure levels exceeded the projected pharmacological threshold required to influence pulmonary vascular remodelling and fibrotic pathways, as suggested by preclinical models.

## PH-ILD: A severe PH subtype with limited treatment options

Cereno had previously chosen IPF as the target indication for Phase II, but in February 2026 the company announced CS014's pivot towards PH-ILD, broadening its opportunity set. PH-ILD is not a singular condition but a group of more than 200 conditions defined by progressive inflammation and fibrosis in the lungs, primarily around the alveoli (tiny air sacs located at the end of the respiratory tract in the lungs, which allow oxygen to enter the bloodstream). It is classified as WHO Group 3 pulmonary hypertension, which includes PH that develops as a secondary consequence of lung damage due to chronic lung disorders or long-standing low oxygen levels. PH-ILD starts as an interstitial lung disease, which is characterised by inflammation, scarring and fibrosis of the lung tissues. As a result, lung tissue cannot efficiently move oxygen into the bloodstream, resulting in thickening and narrowing of pulmonary blood vessel walls. This increases PVR and forces the right ventricle to pump harder to maintain blood flow, leading to high blood pressure in the arteries of the lungs or pulmonary hypertension, which may eventually result in right-heart failure. Clinically, PH-ILD is generally defined by an mPAP >20 mmHg and PVR  $\geq 2$  Wood units, confirmed through right-heart catheterisation.

IPF represents the largest ILD subtype, accounting for roughly 20–50% of ILD cases, with pulmonary hypertension developing in 30–60% of patients with advanced disease. Other important PH-ILD subtypes include connective tissue disease-associated ILD, chronic hypersensitivity pneumonitis and combined pulmonary fibrosis and emphysema. Epidemiological data suggest that PH-ILD affects c 180,000 patients across the seven major markets (US, EU4+UK and Japan), with the US accounting for roughly 45% of cases (c 80,000).

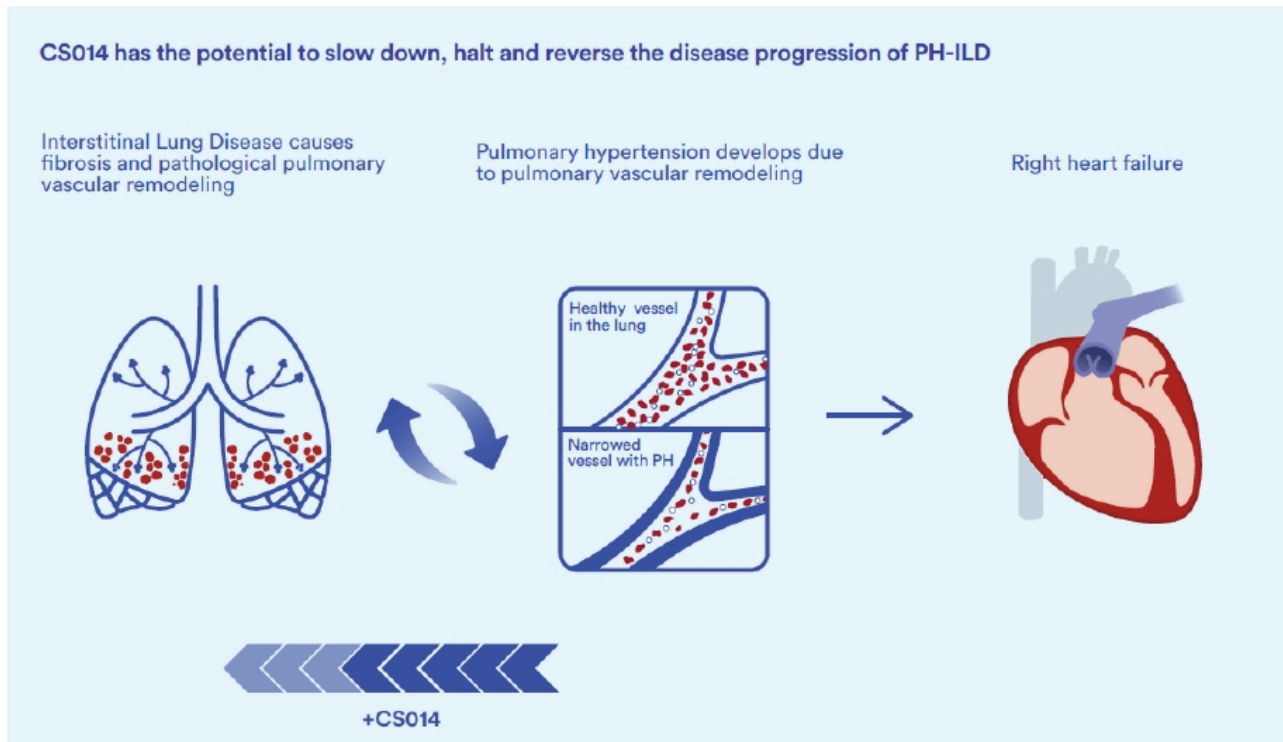
PH-ILD is a particularly severe phenotype of ILD, associated with accelerated disease progression and poor survival, with median survival estimated at 1.5–2.0 years following diagnosis, with a three-year survival rate of around 40%. Lung transplantation remains the only potentially curative intervention. Despite the clear unmet need, the therapeutic landscape remains limited. Traditional vasodilator therapies developed for PAH have generally shown limited benefit in PH-ILD due to fundamental differences in disease biology. While PAH is primarily driven by vascular narrowing, PH-ILD arises from fibrotic lung destruction, meaning vasodilators can inadvertently worsen ventilation–perfusion mismatch by increasing blood flow to poorly ventilated regions of the lung. As a result, treatments traditionally have largely focused on managing the underlying interstitial lung disease and providing supportive care.

## Treatment landscape underdeveloped despite recent progress...

A notable advance occurred with the approval of inhaled treprostinil (Tyvaso) in April 2021, following the INCREASE trial, which demonstrated improvements in exercise capacity and established the therapy as the first drug specifically approved for PH-ILD. The rationale for an inhaled version was lower systemic exposure coupled with the ability to deliver the drug directly to the airways, therefore reaching better-ventilated areas of the lung. Subsequent formulation improvements followed, including Tyvaso dry powder inhalation (DPI) in 2022 and Liquidia's treprostinil inhalation powder YUTREPIA in 2025. However, these therapies primarily provide haemodynamic and symptomatic benefits rather than addressing the underlying fibrotic and vascular remodelling processes driving disease progression. Consequently, a significant unmet need remains for therapies capable of modifying disease biology through effects on inflammation, fibrosis and vascular remodelling.

Against this backdrop, we believe that Cereno's decision to broaden CS014's development into PH-ILD appears strategically sound. The mechanism of HDAC inhibition is believed to influence several of the key pathophysiological drivers implicated in PH-ILD, including vascular remodelling and inflammatory signalling (Exhibit 2). The pivot potentially also reflects the challenging drug development environment in IPF, where multiple late-stage programmes have failed. The approval of nerandomilast (Jascayd) in October 2025 may have been another driver for the decision, given that it is the third antifibrotic to be approved for IPF after Ofev (nintedanib) and Esbriet (pirfenidone) and is believed to have a superior safety and tolerability profile to the previously approved antifibrotic treatments with fewer gastrointestinal side effects.

## Exhibit 2: CS014's disease modifying potential in PH-ILD



Source: Cereno Scientific

## ...Driving the shift toward potentially disease-modifying approaches

With current treatments limited to inhaled treprostinil, the PH-ILD development pipeline is beginning to evolve, with several programmes targeting disease mechanisms beyond vasodilation. These include approaches aimed at vascular remodelling, inflammatory signalling and fibrotic pathways, suggesting a gradual shift toward potentially disease-modifying strategies. Several mid-stage clinical trials are currently underway (Exhibit 3), indicating that the competitive landscape could expand meaningfully over the coming years as these programmes mature. Should forthcoming clinical data support its proposed mechanism and demonstrate meaningful disease modification, CS014 could emerge as a differentiated therapeutic candidate within this developing treatment landscape.

## Exhibit 3: PH-ILD clinical development landscape

Candidate	Company	Mechanism of action	PH-ILD development stage	Primary endpoint	Disease-modifying potential
Seralutinib (inhaled DPI)	Gossamer Bio / Chiesi	Inhaled tyrosine kinase inhibitor (targets pathways implicated in vascular remodelling/inflammation)	Phase III (development on hold following the failure of the Phase III PROSERA trial in PAH in February 2026)	6MWD (change vs baseline, week 24)	Yes
L606 (treprostinil liposome inhalation suspension)	Liquidia	Sustained-release inhaled prostacyclin (treprostinil) formulation	Phase III	6MWD (change vs baseline, week 16)	No
Treprostinil Palmitil Inhalation Powder (TIPIP)	Insmed	Inhaled treprostinil (sustained lung exposure)	Phase III	6MWD (change vs baseline, week 24)	No
Mosliciguat (inhaled)	Pulmovant (Roivant)	Inhaled soluble guanylate cyclase activator (nitric oxide-cyclic guanosine monophosphate pathway)	Phase II	6MWD (change vs baseline, week 16)	No
Mirivadelgat (FP-045; oral)	Foresee Pharma	Aldehyde dehydrogenase 2 activator	Phase II (first patient dosed in May 2025)	PVR change (to week 12)	Yes
ROC-101 (oral)	AllRock Bio	ROCK inhibitor	Phase IIa	PVR change (to week 24)	Yes
<b>CS014 (oral)</b>	<b>Cereno Scientific</b>	<b>HDAC inhibitor</b>	<b>Phase II-ready</b>	<b>N/A</b>	<b>Yes</b>
HB-1614 (oral)	Halo Biosciences	Reformulation of 4-methylumbelliferone (4-IND-enabling MU)		N/A	Yes

Source: Edison Investment Research

## Recent regulatory discussions have paved the path for Phase IIb in Q127

On 17 March 2026, Cereno announced that it has received regulatory approval from the Swedish Medical Products Agency to initiate a Phase I PK study of CS014, with the study design and planning in alignment with the FDA. Importantly, the agreed study design has the potential to remove the requirement for additional non-clinical safety work and a Phase IIa study, effectively enabling a direct transition to a Phase IIb trial in PH-ILD, targeted for Q127.

The trial is a randomised, open-label, crossover study in 14 healthy volunteers, comparing steady-state PK of CS014 versus VPA. By generating comparative bioavailability data, Cereno aims to leverage the well-established safety profile of VPA to strengthen CS014's regulatory package. Positive results, expected in mid-2026, would support advancement directly to a more advanced stage Phase IIb efficacy study.

Overall, we view this as a strategically important development milestone, with the early regulatory-alignment meaningfully de-risking the clinical pathway and accelerating development timelines, while optimising capital outlays required for additional studies.

## Financials

### Operating performance: No real surprises

In Q424, Cereno reported operating expenses of SEK36.8m, broadly in line with the Q424 figure of SEK38.8m and up 41.6% q-o-q (SEK26.0m in Q424). This included external costs of SEK26.4m (down 12.3% y-o-y and up 34.2% q-o-q) and personnel expenses of SEK10.2m (up 30.3% y-o-y and 68.4% q-o-q). While not explicitly stated by the company, we believe that the quarter-on-quarter increase was driven by an acceleration in preparatory activities related to the upcoming Phase IIb trial for CS1, including clinical trial material preparation and site selection and training. Of the total external expenses in the quarter, SEK9.7m have been capitalised by the company, which is reflected as income (capitalised work for own account) in the P&L. This does not have an impact on operating profitability or loss, with the company reporting an operating loss of SEK27.1m in Q425 versus losses of SEK13.5m in Q325 and SEK27.2m in Q424. Interest and financial expenses rose materially during the quarter (SEK19.0m vs SEK7.8m in Q325) and we believe this increase was largely driven by the up to SEK665m financing arrangement announced by the company in November 2025, including SEK175m in convertible debt and another SEK175m in a loan facility. This translated to a net loss for the period of SEK44.7m, versus SEK21.3m in Q325 and SEK40.3m in Q424. Free cash outflow for the period was SEK47.7m, reflecting the operating results. This compares to a free cash outflow of SEK28.3m in Q325 and SEK56.4m in Q424.

### Cash runway potentially into Q427 with November 2025 financing

Cereno ended Q425 with a gross cash balance of SEK74.6m, supported by the receipt of SEK4m from warrant exercises by Arena Investors in November (600k shares at SEK6.67/share), SEK100m from the November 2025 directed share issue and another SEK125m from the convertible debt facility (conversion price of SEK10/unit). These proceeds were partially utilised to repay the SEK180m in outstanding debt under the previous November 2024 agreement (SEK25m owed to Venusat and SAJ Finans and SEK155m to Fenja Capital and Arena Investors). Net debt at the end of the year was SEK50.4m.

In January 2026, Arena Investors converted another SEK5m worth of warrants (729k shares) and the company received a further SEK45m tranche under the convertible loan facility. With this drawdown, we believe that the SEK175m convertible debt facility has been fully utilised. This leaves up to SEK390m in capital potentially available to the company. This includes:

- up to SEK175m in loan facilities, and
- up to SEK215m from warrant exercises. While the SEK100m warrants issued alongside the directed issue are exercisable between October and December 2026 (at a conversion price of SEK10/unit), the other SEK115m relates to the debt facility and is exercisable up to November 2030 at a conversion price of SEK12/unit.

We note that access to the SEK175m loan facility will be available between 1 April 2026 and 30 June 2027 with drawdowns contingent on predefined financial conditions and 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, which is a c 59% premium to the last closing price of SEK6.30/share. With upcoming data from the EAP and the planned initiation of the CS1 Phase IIb study, we believe that there are several re-rating events in the calendar that could warrant an uplift in the share price to the required threshold.

Should the outstanding November 2025 facilities be fully utilised and assuming 100% conversion of the attached warrants (although currently out-of-money), management estimates the company will have a cash runway into Q427. We also note the possibility of Cereno refinancing or restructuring elements of the facility in FY27, potentially extending funding coverage through CS1 Phase IIb top-line readouts in Q428. If we stress test this assumption and assume that the SEK10/share conversion threshold is not achieved in 2026, we estimate that Cereno will need to raise SEK120m in incremental capital in H226.

## Estimate revisions

Based on the Q425 results and currently visibility on Cereno's clinical plans, we have made slight adjustments to our FY26 estimates and also introduce FY27 estimates as we roll our model forward. Given the plans for CS1 and pending further details on CS014's Phase II design and plans in PH-ILD, we modestly trim our R&D expense estimate for FY26 to SEK150m, from SEK170m, pushing out part of the expenses into FY27. On the other hand, we raise our estimates for other external expenses and personnel costs to SEK40.8m and SEK34.0m, from SEK33.1m and SEK30.1m, respectively, reflecting the FY25 trend. Overall, we now estimate an operating loss of SEK75.8m in FY26 versus an operating loss of SEK64.2m previously. Note that since Cereno capitalises its R&D expenses, the impact on operating profitability is limited. For FY27 we estimate an operating loss of SEK77.9m and free cash outflow of SEK294.8m. Note that our estimates assume Cereno self-sponsors the CS1 Phase IIb and CS014 Phase II trials before a partner takes over subsequent development. Any changes in this assumption would require a reassessment of our estimates.

## Valuation

We value Cereno using a risk-adjusted net present value (rNPV) framework, applying phase-appropriate probabilities of success (with slight adjustments based on the target indication, competitive landscape and drug positioning) and a flat discount rate of 12.5%. Our valuation reflects contributions from the company's two clinical-stage programmes: CS1, which is expected to initiate a Phase IIb study in PAH in Q226, and CS014, planned to enter Phase II development in PH-ILD in Q127. We continue to exclude CS585 from our valuation given its preclinical status, although we note potential upside once the programme enters the clinic.

## CS1 assumptions unchanged

With development plans for CS1 progressing as expected, we maintain our long-term assumptions for the asset. For a detailed discussion of these assumptions, we refer readers to our recent [outlook note](#) on the company. CS1 remains the primary driver of our valuation, contributing c 86% of the total implied value, corresponding to an rNPV of SEK5.7bn or SEK18.5 per share.

## CS014 expectations refreshed following PH-ILD pivot

The main update to our valuation relates to CS014, where we replace IPF with PH-ILD as the primary Phase II target indication. While management has not ruled out revisiting IPF in the future, for simplicity our model currently focuses solely on PH-ILD.

**Target population and market penetration:** Based on available epidemiological data, we estimate a target population of c 80,000 patients in the US and c 100,000 across the EU4, the UK and Japan. Given the severity of PH-ILD, we assume 95% diagnosis rates and 90% treatment uptake. We define the addressable population as patients stable on antifibrotic therapy, and estimate that c 70% of treated patients would be eligible for CS014.

While vasodilators are well established in PAH, the PH-ILD treatment landscape remains relatively nascent, with inhaled treprostinil currently the only approved therapy. Adoption has been gradual: according to Liquidia, around 6,000 PH-ILD patients were treated with inhaled treprostinil between 2021 and 2025, although prescription trends appear to be accelerating following the approval of YUTREPIA in May 2025. At this stage it remains unclear whether CS014 will ultimately be positioned in combination with vasodilators, and we will revisit our assumptions as the treatment paradigm evolves.

Given the limited therapeutic options targeting the PH component of PH-ILD, we assume peak market penetration of 20%, higher than the 15% penetration previously assumed for IPF.

**Pricing:** For the US market, we assume a list price of \$250k per patient per year, applying a 50% payer discount, resulting in a net annual price of \$125k. This represents a premium to the \$150k annual price assumed for IPF, which we believe is justified given the more severe disease course in PH-ILD. In addition, the assumed pricing appears reasonable in the context of Tyvaso's wholesale acquisition cost of c \$220k per patient per year. For European and Japanese markets, we assume a more conservative net annual price of \$62,500, reflecting higher pricing sensitivity.

**Clinical timeline and peak sales:** Assuming the Phase IIb study initiates in Q127, we estimate completion in 2029. We assume that subsequent development and commercialisation will occur under a licensing or partnership agreement, which we model as occurring in 2029. Our base case assumes market launch in 2033, with peak sales of c \$2.4bn reached by 2040. We model a total deal value of \$1.5bn, including an upfront payment of \$150m, alongside a 15% royalty on future sales.

**R&D costs:** Pending further details on the Phase II design, we model trial-related costs of c \$12m, based on an estimated enrolment of c 75 patients and an assumed per-patient cost of \$150k. For reference, Pulmovant's Phase II study of moslicigat DPI in PH-ILD enrolled 120 patients.

**Probability of success (PoS):** We reduce our probability of success assumption to 15% ahead of Phase II initiation (20% previously assumed for IPF) given the heterogeneity of ILD conditions covered under the PH-ILD banner and potential for varied efficacy among them.

Based on the aforementioned assumptions, we estimate an rNPV valuation of SEK0.9bn or SEK3.0/share to CS014.

Overall, our updated valuation for Cereno stands at SEK6.6bn or SEK21.3/share (SEK6.6bn or SEK21.1/share previously). The revised valuation also reflects the latest pro forma net debt of SEK45.4m (including SEK5m in warrant proceeds received in January 2026). Exhibit 4 presents a breakdown of our valuation for Cereno.

#### Exhibit 4: Cereno's rNPV valuation

Asset	Indication	Development phase	Launch	Peak sales		NPV (SEKm)	Probability	rNPV	rNPV/share
				(\$m)	Peak sales year			(SEKm)	(SEK)
CS1	PAH	Phase IIb-ready	2032	2,765	2039	11,494.6	50%	5,747.3	18.5
CS014	PH-ILD	Phase II-ready	2033	2,435	2040	6,161.6	15%	924.2	3.0
<b>Total</b>						17,656.2		6,671.5	21.4
Pro forma net cash/(debt) at 31 December 2025								(45.4)	(0.1)
<b>Valuation</b>								<b>6,626.2</b>	<b>21.3</b>

Source: Edison Investment Research. Note: The per-share valuation is based on outstanding shares of 311.4m.

**Exhibit 5: Financial summary**

Accounts: K3; year end 31 December; SEK000s	2023	2024	2025	2026e	2027e
<b>PROFIT &amp; LOSS</b>					
Net sales	0	0	0	0	0
Capitalised work for own account	49,277	80,903	44,273	150,000	157,500
Total revenues	49,277	80,903	44,273	150,000	157,500
Total operating expenses	(93,927)	(156,739)	(118,875)	(225,782)	(235,358)
R&D and other expenses	(71,152)	(128,675)	(85,593)	(191,733)	(199,651)
<i>Of which - R&amp;D expenses</i>	<i>(49,277)</i>	<i>(80,903)</i>	<i>(44,273)</i>	<i>(150,000)</i>	<i>(157,500)</i>
<i>Of which - other expenses</i>	<i>(21,658)</i>	<i>(46,880)</i>	<i>(40,419)</i>	<i>(40,823)</i>	<i>(41,231)</i>
Personnel costs	(18,763)	(26,108)	(32,935)	(34,049)	(35,707)
Other operating items	(4,012)	(1,956)	(347)	0	0
Operating income (reported)	(44,650)	(75,836)	(74,602)	(75,782)	(77,858)
EBITDA (normalised)	(44,636)	(75,549)	(73,814)	(75,487)	(77,593)
Finance income/(expense)	(3,456)	(23,690)	(43,153)	(16,468)	(60,635)
Profit before tax (reported)	(48,106)	(99,526)	(117,755)	(92,251)	(138,493)
Profit before tax (normalised)	(46,436)	(98,106)	(117,755)	(92,251)	(138,493)
Income tax expense (includes exceptionals)	0	0	0	0	0
Net income (reported)	(48,106)	(99,526)	(117,755)	(92,251)	(138,493)
Net income (normalised)	(46,436)	(98,106)	(117,755)	(92,251)	(138,493)
End of period number of shares, 000s	233,775	281,702	310,492	310,492	310,492
Basic EPS (SEK)	(0.21)	(0.35)	(0.38)	(0.30)	(0.45)
Adjusted EPS (SEK)	(0.20)	(0.35)	(0.38)	(0.30)	(0.45)
<b>BALANCE SHEET</b>					
Intangible Assets	196,264	277,167	321,440	471,440	628,940
Fixtures, tools and installation	14	3,599	2,880	2,585	2,319
Other long-term receivables	9	10	5	5	5
Total non-current assets	196,287	280,775	324,324	474,029	631,264
Other receivables	1,124	2,880	1,988	2,677	2,566
Prepaid expenses and accrued income	407	2,540	1,723	1,723	1,723
Cash and bank balance	87,169	127,578	74,639	171,073	41,269
Total current assets	88,699	132,997	78,350	175,473	45,558
Accounts Payable	6,930	13,951	10,094	19,173	19,986
Other Current Liabilities	16,231	17,495	15,109	15,109	15,109
Short-term Debt	0	0	0	0	0
Total current liabilities	23,162	31,446	25,203	34,281	35,095
Long-term Debt	45,000	190,000	125,000	450,000	615,000
Other debt	400	400	0	0	0
Total non-current liabilities	45,400	190,400	125,000	450,000	615,000
Equity attributable to company	216,424	191,926	252,472	165,221	26,728
<b>CASH FLOW STATEMENT</b>					
Net profit	(48,106)	(99,526)	(117,755)	(92,251)	(138,493)
Depreciation	14	287	788	295	265
Translation difference	34	0	(32)	0	0
Accrued costs	777	6	1,308	0	0
Share based payments	1,671	1,420	0	0	0
Taxes paid	0	0	0	0	0
Movements in working capital	8,695	(5,609)	3,826	8,389	924
Cash from operations (CFO)	(36,915)	(103,422)	(111,865)	(83,567)	(137,303)
Purchase of intangible assets	(49,277)	(80,903)	(44,273)	(150,000)	(157,500)
Purchase of PPE	0	(3,871)	(138)	0	0
Cash used in investing activities (CFIA)	(49,277)	(84,774)	(44,411)	(150,000)	(157,500)
Loans received	45,000	245,000	200,000	325,000	515,000
Loan repayments	0	(90,000)	(200,000)	0	(350,000)
Equity issued	61,315	73,605	103,179	5,000	0
Other Financing Cash Flows	0	0	159	0	0
Cash from financing activities (CFF)	106,315	228,605	103,337	330,000	165,000
Cash and equivalents at beginning of period	67,046	87,169	127,578	74,639	171,073
Increase/(decrease) in cash and equivalents	20,123	40,409	(52,938)	96,433	(129,803)
Cash and equivalents at end of period	87,169	127,578	74,639	171,073	41,269
Net (debt)/cash	41,769	(62,822)	(50,361)	(278,927)	(573,731)

Source: Company documents, Edison Investment Research

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