Herantis Pharma

Momentum continues into FY26

Herantis Pharma's FY25 results reflect positive momentum for HER-096, which is being developed as a potential disease-modifier for Parkinson's disease (PD). Notably, the candidate successfully completed the Phase Ib trial, meeting primary and secondary endpoints. The associated biomarker programme also added to a robust foundation for Phase II, in our view. With a year-end gross cash position of €2.6m, plus a directed share issue of €4.2m (gross) in February 2026, Herantis has a cash runway into Q127 (according to management guidance). While this is not sufficient to execute the Phase II trial, it should cover the remaining preparations. Management is actively seeking partners to continue development activities, but is also exploring both equity and grant financing options.

Phase Ib results highlight HER-096 potential

The top-line results for the Phase Ib trial were announced in October 2025, representing a key milestone for the reporting period. The key takeaways were the favourable safety profile in PD patients and confirmation of blood-brain barrier (BBB) penetration. In January 2026, Herantis presented biomarker outcomes, showing that HER-096 exposure was associated with key biological changes across multiple PD-related pathways. While these biomarker results do not represent efficacy signals, they do add confidence in the translation of preclinical research to clinical outcomes, de-risking the subsequent stages of development, in our view. The next inflection point for the programme will be efficacy measures in Phase II.

Recent funding supports next steps

A directed share issue was announced in February 2026, raising €4.2m in gross proceeds, strengthening the company's balance sheet. The proceeds will financially support preparations for a subsequent Phase II trial of HER-096 in PD, partnership discussions and general corporate activities. Herantis is actively seeking potential pharma partners as it aims to identify the optimal route forward for the candidate. Herantis recently announced that it has been selected for a grant from the Horizon Europe 2025 Research and Innovation programme of up to €8.0m, providing additional operational flexibility and serving as external validation of Herantis's approach towards developing a potential disease-modifier for PD.

Funded to early 2027 based on historical cash burn

At end-2025, Herantis had a gross cash position of €2.6m, bolstered in February 2026 by a €4.2m (gross) equity raise and, subject to final negotiations, a grant of up to €8.0m. Assuming that cash burn rates remain similar, these funds would be projected to provide a runway to early 2027.

Historical financials

Year end	Revenue (€m)	PBT (€m)	EPS (€)	DPS (€)	P/E (x)	Yield (%)
12/23	0.0	0.3	0.02	0.00	124.7	N/A
12/24	0.0	(4.9)	(0.24)	0.00	N/A	N/A
12/25	0.0	(6.6)	(0.28)	0.00	N/A	N/A

Source: Herantis Pharma

Healthcare

9 March 2026

Price	€2.03
Market cap	€54m

Share price performance

Share details

Code	HRTIS
Listing	HEL
Shares in issue	26.5m
Pro forma gross cash/ equivalents at 31 December 2025 (including the February 2026 directed share issue)	€6.8m

Business description

Herantis Pharma is a clinical-stage biotechnology company based in Finland. It is focused on developing disease-modifying therapies to stop or reverse the progression of neurodegenerative diseases. Lead candidate HER-096 is a peptide mimic of CDNF protein and has successfully completed Phase Ib for Parkinson's disease.

Bull points

- Lead candidate has a novel mechanism of action and has shown promising early pharmacokinetics data in humans.
- Sizeable commercial opportunity for an effective PD treatment with disease-modifying properties.
- External validation received via funding from recognised organisations, including the European Innovation Council, the MJFF and Parkinson's UK.

Bear points

- Extended time to market and reliant on external funding to progress the development of HER-096.
- Typical regulatory, development and funding risks associated with the early stages of drug development.
- With its reliance on a single programme, Herantis is exposed to binary event risks.

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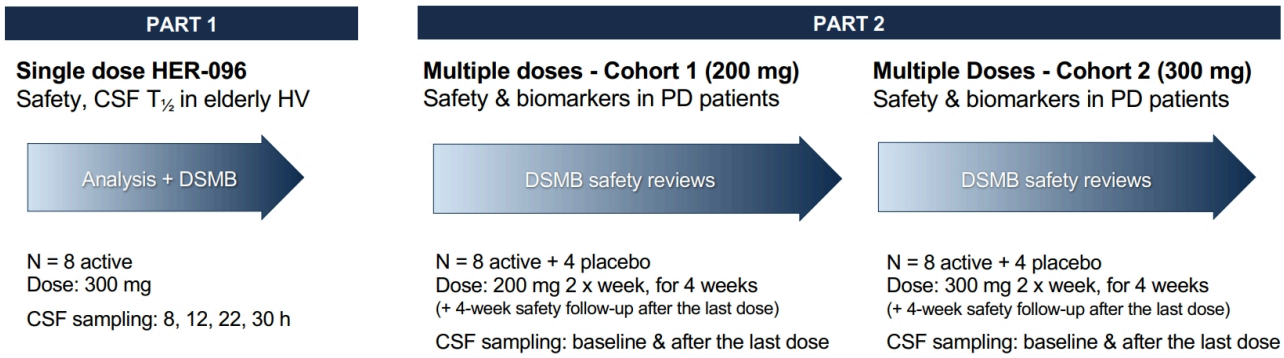
HER-096 in Phase Ib: A robust foundation for further development

Herantis's Phase Ib trial for HER-096 was based across two clinical sites in Finland and involved two parts (Exhibit 1). Part 1 was completed within H224, having tested single doses of HER-096 in elderly, healthy volunteers (n=8; dose: 300mg), showing favourable safety, tolerability and pharmacokinetic (PK) data, consistent with prior reported Phase Ia data. Part 2 was the randomised, double-blind portion of the trial, designed to assess safety and tolerability following repeated doses of HER-096 (as the primary endpoint), alongside PK (as a secondary endpoint) and exploratory biomarker analyses providing an evaluation of biological response to treatment. Part 2 was the first test of HER-096 in PD patients (eight at 200mg plus four on placebo in cohort 1; eight at 300mg plus four on placebo in cohort 2; all patients were dosed twice-weekly over a four-week period). The concentration of HER-096 in cerebrospinal fluid (CSF) was also measured throughout the study, as an indicator of BBB penetration, and to provide insights on an optimal dosing regimen to be tested in Phase II. We note that continued use of symptomatic treatment was allowed throughout the study, with patients taking between one and three medications in addition to the study drug/placebo. The trial included an exploratory biomarker programme, which was completed following the conclusion of the main part of the study.

Exhibit 1: Design of the Phase Ib study for HER-096



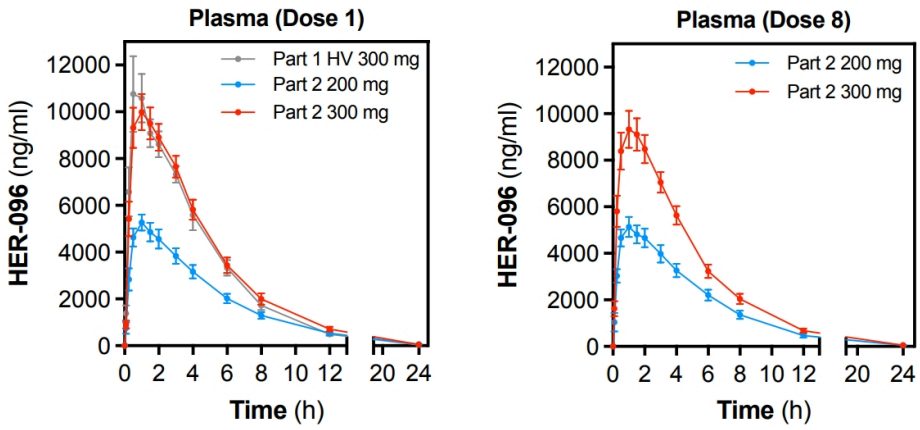
Source: Herantis Pharma

Top-line results

The top-line results for the Phase Ib trial were announced in October 2025, with the primary and secondary endpoints met. Importantly, in terms of safety, HER-096 demonstrated a favourable profile following repeated doses, suggesting the candidate is safe and well-tolerated in PD patients. There was only one serious adverse event, corresponding to a patient in a placebo group, and hence completely unrelated to HER-096. Other adverse events were spread evenly across those in the placebo group and those taking two doses of the drug candidate, and were mainly related to injection site reactions, which were considered mild and transient. Importantly, there were no systemic adverse events, suggesting no concerns for this type of treatment. Brain magnetic resonance imaging was also conducted, but did not identity any concerns in relation to the study drug. Furthermore, the study checked for anti-drug antibodies, a key test for peptide-based therapeutics. Encouragingly, there was no evidence of anti-drug antibody formation in participants. In addition, the Unified Parkinson's Disease Rating Scale (Part III, relating to motor symptoms) was used to measure for safety, with the results showing stable symptoms throughout the study. We note that this result should not be interpreted as an efficacy signal, as the study was not powered to measure efficacy, with the data limited by the small sample size and short treatment duration. Collectively, the safety results were favourable, in our view, supporting advancement of the candidate to the subsequent stages of clinical development.

The plasma PK profiles from the Phase Ib study were found to align with the single doses in healthy volunteers in Phase Ia and the preclinical PK data, showing no accumulation after repeated dosing (Exhibit 2). The profile was considered well-matched with the planned intermittent dosing regimen based on the 'hit-and-run' mechanism of action of HER-096. We note that in this context, a hit-and-run mechanism of action refers to HER-096 as a peptidomimetic drug candidate designed to deliver a meaningful biological effect after only brief target engagement, without needing to stay bound or remain in the body for long. In practice, the candidate hits the target pathway to trigger downstream signalling, and the benefit may persist even after the candidate has cleared.

Exhibit 2: Plasma PK profile of HER-096 in the Phase Ib study

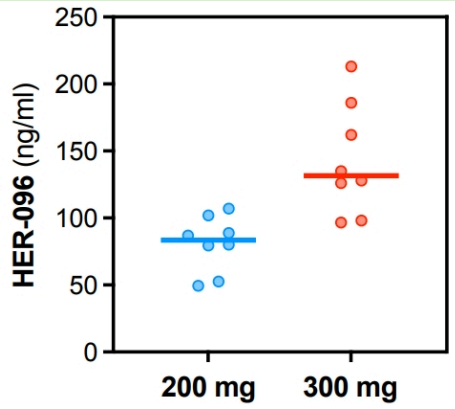


Source: Herantis Pharma

In Part 2, HER-096 levels were also measured through CSF sampling eight hours after patients received their final dose of the drug candidate. The data here showed that both the 200mg and 300mg doses of HER-096 achieved CSF levels in PD patients comparable to pharmacologically active levels identified by preclinical research (Exhibit 3; management noted that the wider dispersion in the 300mg group was likely due to a greater dispersion in patient weight). The CSF levels showed a good correlation with the plasma levels, with the higher doses corresponding to higher concentrations as expected. To provide a cross-sectional view of the CSF PK profiles, data from Phase Ia (at 200mg) and Part 2 of Phase Ib (at 300mg) were combined, normalised to dose (Exhibit 4). The clinical data show the elimination of HER-096 from the central nervous system by c 48 hours, supporting a potential dosing regimen of every two to three days.

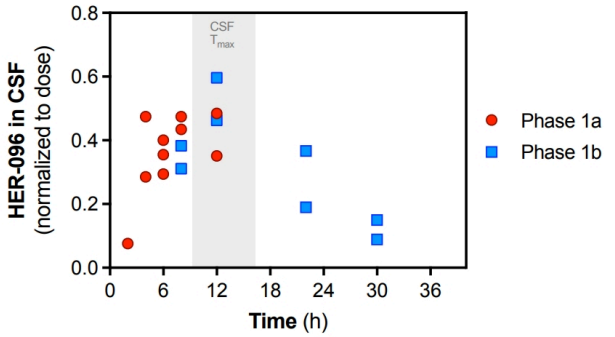
Collectively, these outcomes support the intended dose levels and frequency of HER-096 in PD. The results will inform the design of the subsequent planned Phase II trial, alongside the biomarker programme (financially supported by the Michael J Fox Foundation (MJFF) and Parkinson's UK Virtual Biotech, as well as grant funding from the European Innovation Council (EIC)), which we discuss in more detail below.

Exhibit 3: CSF PK profile of HER-096 in Phase Ib (Part 2)



Source: Herantis Pharma

Exhibit 4: Combined single dose CSF PK data for HER-096 in healthy volunteers (Phase Ia and Part 1 of Phase Ib)



Source: Herantis Pharma

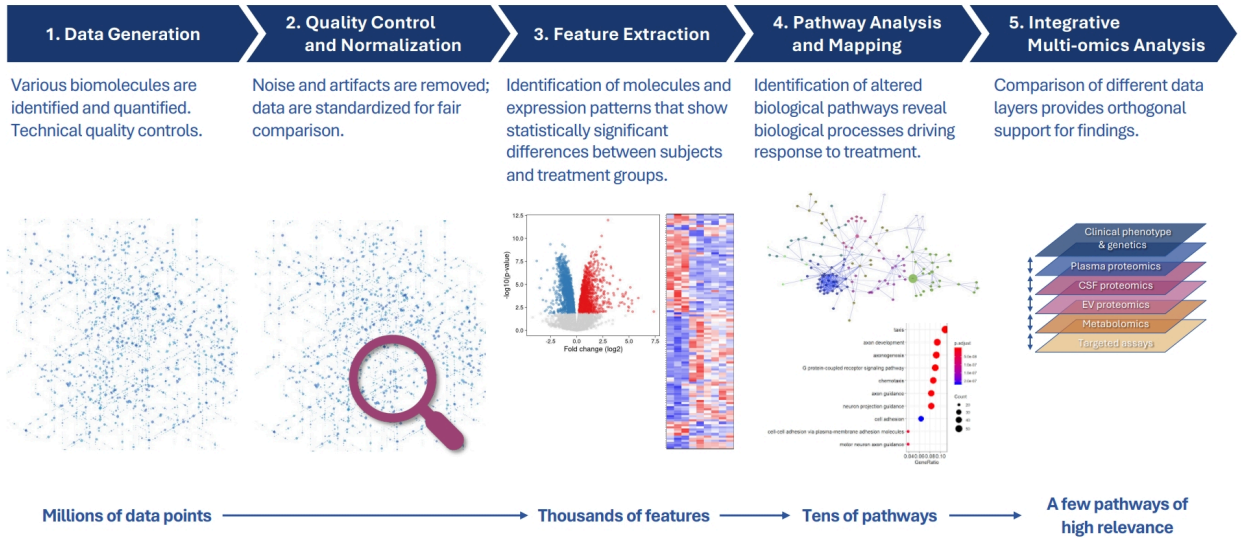
Biomarker results

A biomarker is a measurable biological signal, such as a protein level, genetic change or imaging readout, that indicates what is happening in a disease, or how a patient is responding to a treatment. Biomarker analyses are important in drug development as they can show early signs that a study drug is hitting its intended target and/or affecting the underlying disease biology, the outcomes of which can support dose selection and refinement of endpoints and potentially help identify which patients are most likely to benefit. Ultimately, this can reduce clinical risk, cost and timelines before entering late-stage drug development. Specifically for Herantis, the biomarker programme was designed to gain evidence of a biological response to HER-096 in patients with PD, an indication for which there is no gold standard, with multiple underlying disease drivers at play and currently no approved disease-modifying therapies. A key goal was to demonstrate preclinical-to-clinical translatability, and hence proof-of-biology. In our view, the outcomes (presented in

January 2026) achieved this, however, we note that the biomarker results should not be confused for efficacy signals; the first test of efficacy will come in the subsequent stages of clinical development.

The biomarker programme collected over 2.5m data points, based on the identification and quantification of various biomolecules from the 16 patients receiving HER-096 across the duration of Part 2 of the Phase Ib study, compared to the eight patients receiving placebo. Parallel untargeted and targeted analyses were conducted to provide complementary evidence of the biological effects of the candidate (Exhibit 5). The comparison of different data layers was an important part of the process, as it added confidence in observations that were consistent across levels.

Exhibit 5: Biomarker analysis process



Source: Herantis Pharma

As a reminder, HER-096 targets the unfolded protein response (UPR) pathway as the pathogenesis of PD, with the goal of restoring proteostasis (ie maintaining a stable and functional pool of proteins within the central nervous system). Preclinical evidence has supported this approach, where proteostasis modulation was found to have broad effects on neuronal function. The following parameters were investigated to demonstrate biological response to HER-096:

Proteostasis: Untargeted plasma proteomics was used to see whether HER-096 treatment had an effect on protein expression. The results showed that HER-096 dosing caused systemic changes in protein expression after the four-week treatment duration, in a dose-dependent manner. Broad and coordinated modulation of multiple proteostasis domains followed dosing with HER-096, supporting meaningful engagement of pathways consistent with its mechanism.

Mitochondrial function: A glutathione assay was used as a targeted approach to explore the effect of HER-096 on mitochondrial function. It was found that HER-096 treatment led to enhanced glutathione recycling and improved mitochondrial efficiency, addressing the neuroinflammation and neurodegenerative processes associated with PD. These effects were also found to be dose-dependent, including a statistically significant difference between the 300mg group and placebo, in line with preclinical findings.

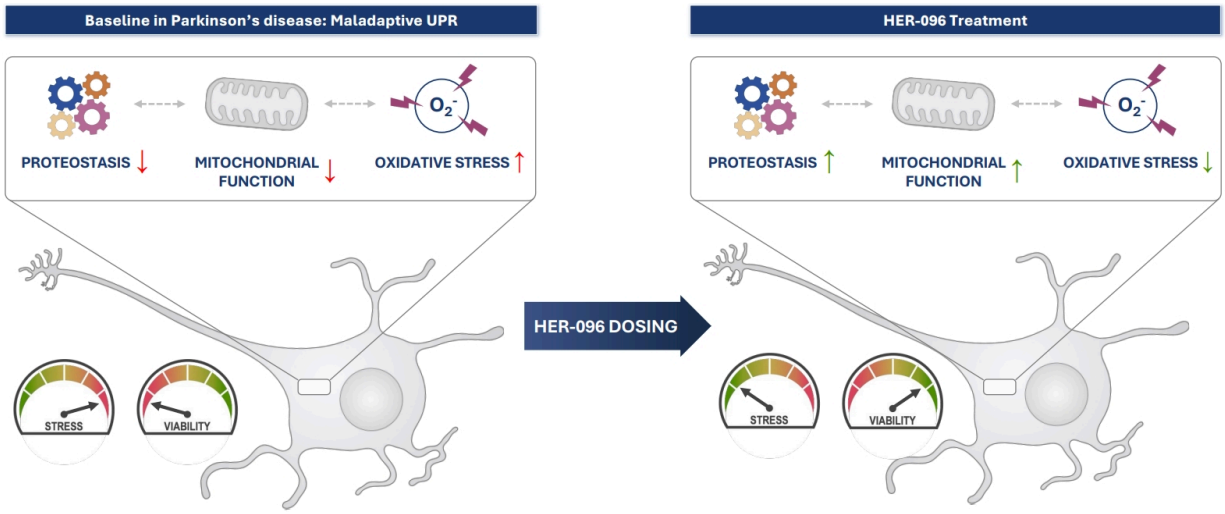
Oxidative stress: A targeted assay was also utilised to assess mitochondrial DNA lesions in peripheral blood mononuclear cells. The results here demonstrated that reduced mitochondrial DNA damage in the blood, mediated by HER-096, was linked to enhanced oxidative stress defence.

Collectively, these clinical biomarker observations were deemed to align with established unfolded protein response (UPR) biology, and the coordinated biological changes were consistent with the normalisation of maladaptive UPR signalling (Exhibit 6). Importantly, these outcomes were in line with preclinical research, supporting HER-096's potential as a disease-modifier for PD and its continued clinical development. The data are likely to support plans for Phase II in terms of dose selection, refinement of endpoints, and possibly also with patient selection and stratification. According to the latest update from management, Phase II will likely be a double-blind, placebo-controlled, safety and efficacy study in early-stage PD patients. However, we await confirmation on the final design and size of the trial.

The next steps involve Herantis actively seeking potential partners to continue the clinical development of HER-096, with a preference for securing such a partnership ahead of Phase II, as discussions with pharma companies are ongoing. We expect that the recent presentation of the biomarker programme outcomes may facilitate these discussions, as they somewhat de-risk the subsequent stages of development. However, Herantis's management is already preparing

for Phase II, and should a suitable partner not be secured, it is also considering an equity raise and non-dilutive grant financing options. We expect an update within H126, ahead of a Phase II launch in H226, according to management guidance.

Exhibit 6: HER-096 potential to address maladaptive UPR signalling in PD



Source: Herantis Pharma

Disease-modifying potential in PD represents a major opportunity

PD is the second most common neurodegenerative disease (behind Alzheimer's disease/dementia), and is estimated to affect over 10 million people worldwide. Despite its prevalence, current treatment options are limited to addressing just the symptoms of the condition. Levodopa is one of the most commonly used PD drugs to improve motor symptoms, but it comes with myriad side effects, and long-term use can lead to dyskinesia. Many patients also experience diminishing results over time with continued use of the drug. The global market for PD therapeutics was estimated to be worth $7.0bn in 2025 and is projected to reach $13.3bn by 2034, corresponding to a compound annual growth rate of 7.4%. We believe that this growth rate will stem mainly from developments in disease-modifying treatment options for PD, hence, there could be a sizeable opportunity for Herantis in this space, should the clinical data for HER-096 continue to be positive.

HER-096 is a peptidomimetic drug candidate, based on the cerebral dopamine neurotrophic factor (CDNF) protein. CDNF was previously being developed as a therapeutic protein drug candidate by Herantis, having shown promise in preclinical and early clinical research. However, as it did not cross the BBB, patients could only receive it via intracranial administration, hindering its potential accessibility. HER-096 was designed to mimic the therapeutic properties of CDNF, but, importantly, was optimised to cross the BBB, allowing administration by subcutaneous injection.

As mentioned above, HER-096 works by targeting the UPR pathway as the pathogenesis of PD. This is the cellular signalling pathway triggered by endoplasmic reticulum stress that leads to the aggregation of misfolded α-synuclein in the substantia nigra of the brain, leading to neuroinflammation and dopamine neuron loss. HER-096 has a multipronged mechanism of action, modulating the UPR pathway to restore homeostatic levels of this cell stress, to ultimately slow down or stop neurodegeneration. Therefore, rather than working as a dopamine replacement agent (as with the current standards of care), HER-096 has been designed as a potential disease-modifier, targeting the root cause of the disease. To our knowledge, this is a novel approach to addressing PD, with Herantis being the only clinical-stage company targeting this mechanism.

Financials

Herantis reported its FY25 results in March 2026. As an early-stage biotechnology company, it continues to operate without recurring revenue, funding its operations primarily through equity issuances, grants and other forms of non-dilutive funding. In FY25, other operating income amounted to €0.2m, notably lower than the FY24 figure of €1.6m. The prior year figure was largely related to the European Innovation Council (EIC) Accelerator project, ReTreatPD (to

develop biomarkers for monitoring target engagement and treatment responses), which was finalised in April 2025.

Alongside grant funding, Herantis continues to receive research funding from the MJFF and Parkinson's UK Virtual Biotech. Announced in July 2024, the funding (total of €3.6m; €1.8m from each) provided financial support for the Phase Ib trial of HER-096 in PD patients, alongside the associated biomarker project. The funding is paid in instalments over two years, upon completion of pre-agreed milestones. Repayment is only required if Herantis enters into a partnership agreement, if HER-096 generates product sales or if there is a change of control of the company or intellectual property rights associated with the asset. In such a scenario, no more than 10% of the cash or non-cash consideration received by the company would be repaid to the funding partners until a maximum of four times the original research funding has been returned. Consistent with FY24, these proceeds are recognised on the balance sheet as long-term debt rather than income.

Operating expenses for FY25 amounted to €6.6m, broadly in line with FY24 (also €6.6m). Within this, R&D expenditure totalled €4.3m (up from €3.6m in the previous year), reflecting the execution of the Phase Ib clinical study of HER-096 and biomarker work, intended to support the subsequent planned Phase II trial. Payroll and related expenses increased to €1.9m (compared to €1.5m in FY24), driven by a higher employee count and bonus payments in Q125. These increases were partly offset by a reduction in other operating expenses, which declined to €4.7m (from €5.1m in FY24), largely reflecting lower spending associated with the completion of the EIC project. Overall, Herantis reported a net loss of €6.6m for FY25 (compared with a loss of €4.9m in FY24).

At end-FY25, Herantis reported a gross cash position of €2.6m, comprising €0.75m in cash and cash equivalents, plus €1.85m held in short-term fixed income securities. Long-term debt stood at €3.4m, largely reflecting the MJFF and Parkinson's UK Virtual Biotech research funding. As a result, the company reported negative equity of €1.7m at the end of the year. The board noted that the fair value of the company's intellectual property related to HER-096 is significantly higher than its book value, and therefore it did not deem it necessary to file a register notification regarding the negative equity position.

Post-period end, the company's financial position has been bolstered. In February 2026, Herantis completed a directed share issue raising €4.2m in gross proceeds (2.41m new shares issued at €1.75 per share, representing a 15.7% discount to the recent volume-weighted average price). In addition, the company announced that it is leading a consortium that has been selected for a grant, pending final negotiations, of €8.0m from the Horizon Europe 2025 Research and Innovation programme.

Looking ahead, management has indicated that the current cash resources, including the February 2026 financing, should be sufficient to support chemistry, manufacturing and controls (CMC) work, as well as clinical preparations for the Phase II trial of HER-096. However, additional capital, estimated by management at c €20–25m, will be required to initiate and execute the Phase II trial itself (alongside other operations, such as other R&D, CMC, and general corporate activities, during the trial period). The company is therefore evaluating several potential financing routes, including further equity issuance, non-dilutive funding and development partnerships. Herantis also remains eligible to receive up to €15m in direct equity investments from the EIC Fund, which may participate in up to one-third of future capital raises. As of end-February 2026, €4.2m of this facility had already been invested.

Based on the company's historical operating cash outflow of approximately €6.1m in FY25, broadly consistent with prior years, we estimate that the current funding position provides operational headroom into early 2027, consistent with management guidance of a cash runway to Q127. This time frame should allow Herantis to complete the remaining preparatory work for its planned Phase II proof-of-concept trial of HER-096, and continue discussions about securing the additional capital required to advance the programme into the next stage of clinical development.

Exhibit 7: Financial summary

€'000s	2023	2024	2025
Year end 31 Dec	FAS	FAS	FAS
Income statement			
Revenue	**0.0**	**0.0**	**0.0**
Profit before tax (as reported)	279.8	(4,939.3)	(6,620.0)
Net income (as reported)	**279.8**	**(4,939.3)**	**(6,620.0)**
EPS (as reported) (€)	0.02	(0.24)	(0.28)
Dividend per share (€)	0.00	0.00	0.00
Balance sheet			
Total non current assets	0.0	0.0	0.0
Total current assets	6,745.6	2,571.5	2,668.0
Total assets	**6,745.6**	**2,571.5**	**2,668.0**
Total non current liabilities	29.8	2,180.4	3,443.0
Total current liabilities	1,989.4	633.9	912.0
Total liabilities	**2,019.3**	**2,814.4**	**4,356.0**
Net assets	4,726.4	(242.9)	(1,688.0)
Shareholder equity	**4,726.4**	**(242.9)**	**(1,688.0)**
Cashflow			
Net cash from operating activities	(4,636.0)	(6,545.0)	(6,095.0)
Net cash from investing activities	607.1	(474.1)	(244.0)
Net cash from financing activities	4,496.2	2,150.6	6,456.0
Net cash flow	**467.2**	**(4,868.5)**	**116.0**
Cash & cash equivalents at year end	**5,503.0**	**634.5**	**750.5**

Source: Company accounts

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