HERANTIS PHARMA

Neuroregenerative therapeutics for Parkinson’s disease

January 2025

Herantis Pharma Oyj (HEL: HRTIS)

Antti Vuolanto, D.Sc. CEO antti.vuolanto@herantis.com

Forward-looking statements

This company presentation includes forward-looking statements which are not historical facts but statements regarding future expectations instead. These forward-looking statements include without limitation, those regarding Herantis' future financial position and results of operations, the company's strategy, objectives, future developments in the markets in which the company participates or is seeking to participate or anticipated regulatory changes in the markets in which the company operates or intends to operate. In some cases, forward-looking statements can be identified by terminology such as "aim," "anticipate," "believe," "continue," "could," "estimate," "expect," "forecast," "guidance," "intend," "may," "plan," "potential," "predict," "projected," "should" or "will" or the negative of such terms or other comparable terminology. By their nature, forward-looking statements involve known and unknown risks, uncertainties and other factors because they relate to events and depend on circumstances that may or may not occurin the future.

Forward-looking statements are not guarantees of future performance and are based on numerous assumptions. The company's actual results of operations, including the company's financial condition and liquidity and the development of the industry in which the company operates, may differ materially from (and be more negative than) those made in, or suggested by, the forward-looking statements contained in this company release. Factors, including risks and uncertainties that could cause these differences include, but are not limited to risks associated with implementation of Herantis' strategy, risks and uncertainties associated with the development and/or approval of Herantis' drug candidates, ongoing and future Clinical trials and expected trial results, the ability to commercialize drug candidates, technology changes and new products in Herantis' potential market and industry, Herantis' freedom to operate in respect of the products it develops (which freedom may be limited, e.g., by competitors' patents), the ability to develop new products and enhance existing products, the impact of competition, changes in general economy and industry conditions, and legislative, regulatory and political factors. In addition, even if Herantis' historical results of operations, including the company's financial condition and liquidity and the development of the industry in which the company operates, are consistent with the forward-looking statements contained in this company release, those results or developments may not be indicative of results or developments in subsequent periods.

Executive Summary

- Clinical-stage public company developing therapeutics for Parkinson's disease (PD) and other CNS disorders
- HER-096: a disease-modifying peptide mimic of CDNF protein, shown to reverse damage and break the cycle of PD pathogenesis
- Phase 1a with subcutaneous administration:
  - Efficient brain penetration
  - Good safety profile
- Efficacy data:
  - Phase 1 trial of CDNF, which HER-096 mimics, showed signs of biological response in advanced PD patients
  - Strong preclinical data of HER-096
- Significant unmet need:
  - 328,000+ patients diagnosed yearly in target geographies
  - $10B+ global market in 2030
  - No existing disease-modifying or neurorestorative therapeutics

- Phase 1b trial ongoing
- Preparing for Phase 2 trials of HER-096 for Parkinson's disease

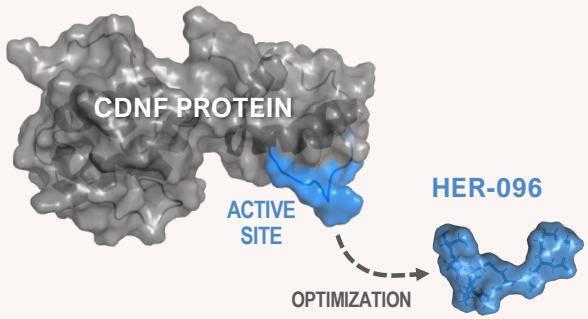
HER-096 is an Ideal Drug Candidate for Parkinson's Disease

HER-096

- Synthetic peptidomimetic molecule
- Designed based on the active site of CDNF protein
- Unique and broad Mechanism of Action: Modulation of Unfolded Protein Response (UPR) pathway to reduce cell stress to slow down or stop neurodegeneration
- Penetrates blood-brain barrier in humans

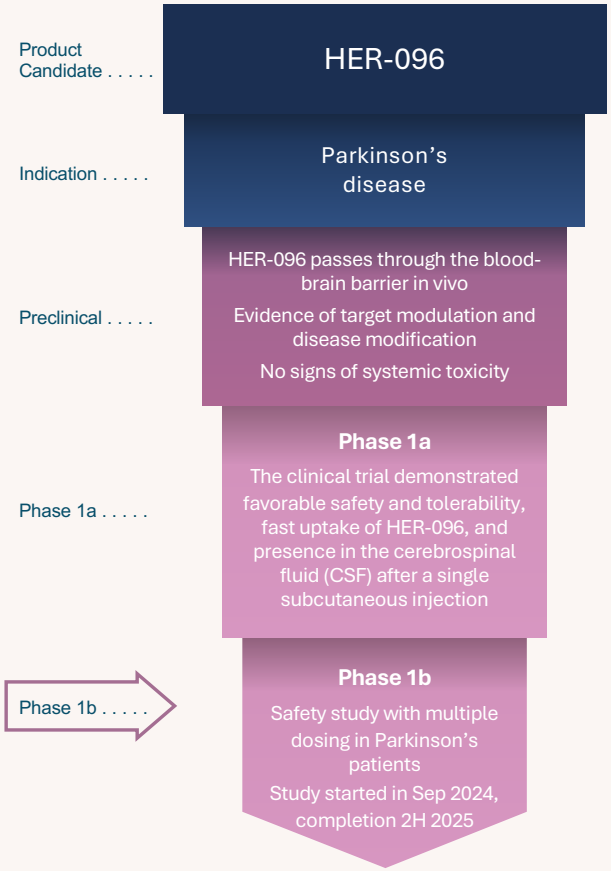
HER-096 & Parkinson's disease treatment

- **Symptomatic improvement**
- **Long-term effect with disease modification**: slow down or stop the process of midbrain neuron degeneration at the early stage of the disease
- Subcutaneous administration 1 – 3 times per week
- Differentiated mechanism with potential opportunities in combination with current assets in development



Multi-billion market opportunity

- 10 million patients globally
- Current Parkinson's disease pharmaceuticals market size $5B
- Market 2029: $11B, growth driven by disease-modifying treatments
  (source: GlobalData)

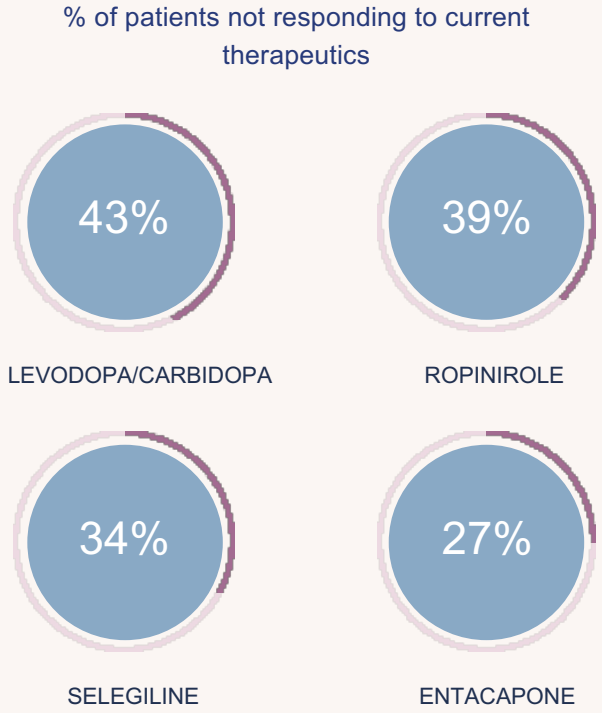


Innovation is urgently needed for PD treatment

Current therapies only treat symptoms and have significant side effects

Many patients have no symptomatic benefit, and none improve

No disease-modifying or regenerative therapeutics are available



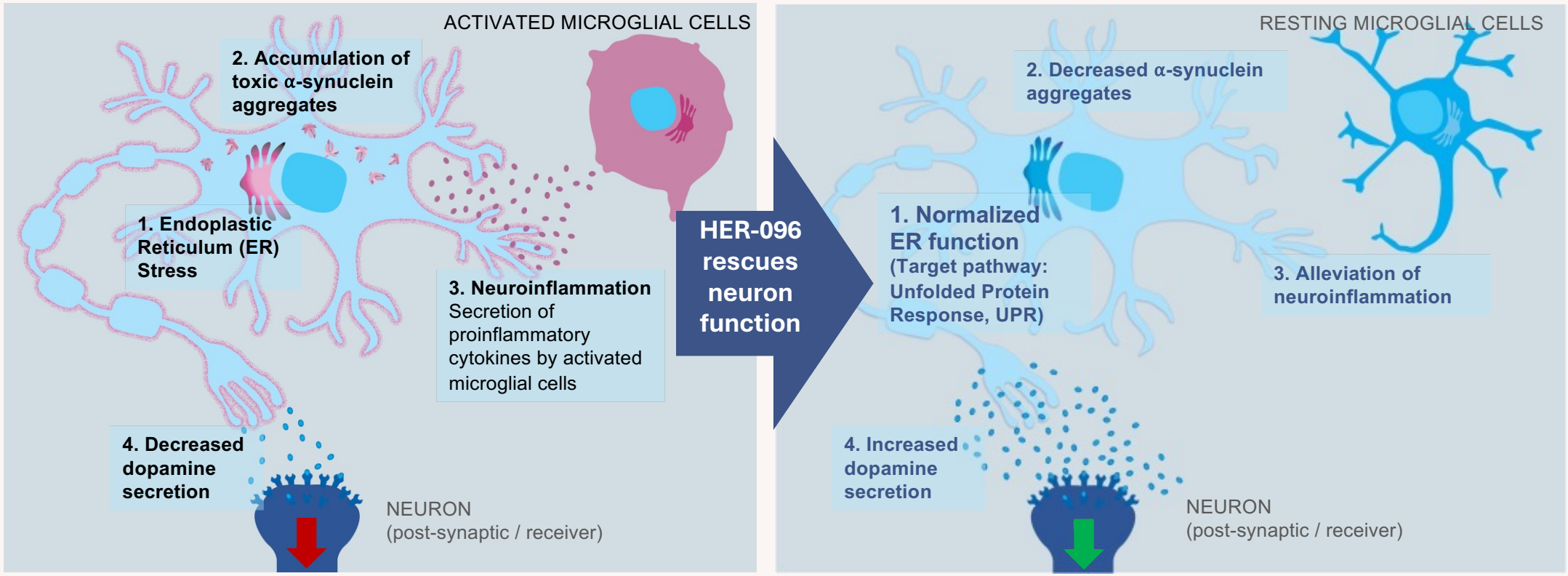
1. Levodopa/carbidopa: 100% minus response rate (patients attaining 25% improvement at 30 weeks); Ropinirole: patients not significantly improved at week 48 in clinical trial;
2. Selegiline: patients with significant wearing off effect + patients removed from trial due to insufficient response; Entacapone: 100% minus patients reporting clinical improvement

ref: Pahwa et al 2013 Parkinsonism and Related Disorders, Sethi et al 1998 JAMA Neurology, Pålhagen et al 2006 Neurology, Brooks et al 2005 European Neurology

HER-096 has a neurorestorative, disease-modifying mechanism of action

DEGENERATING DOPAMINERGIC NEURON
(pre-synaptic / transmittor)

FUNCTIONAL DOPAMINERGIC NEURON
(pre-synaptic / transmittor)



MOVEMENT DISORDER

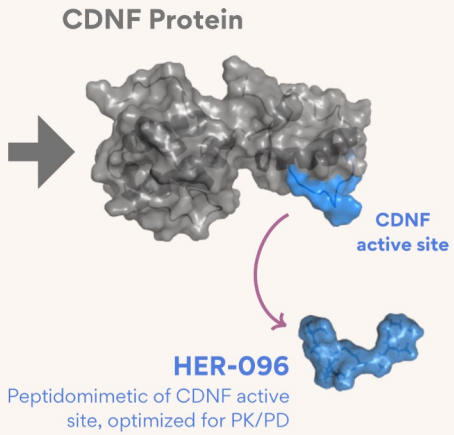
NORMALIZED MOTOR FUNCTION

Data & Clinical Trial Design

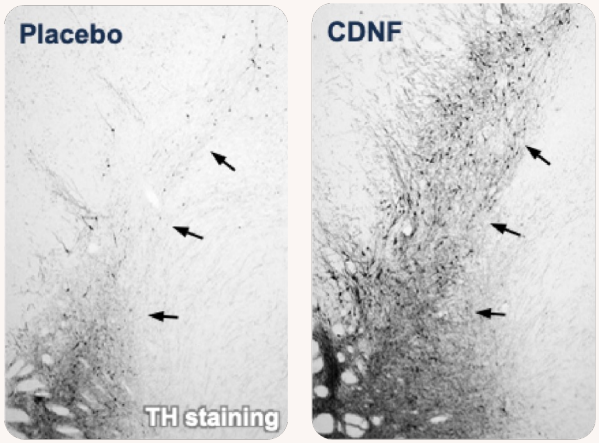
Preclinical, Phase 1a, and Phase 1b

PRECLINICAL PROOF-OF-CONCEPT

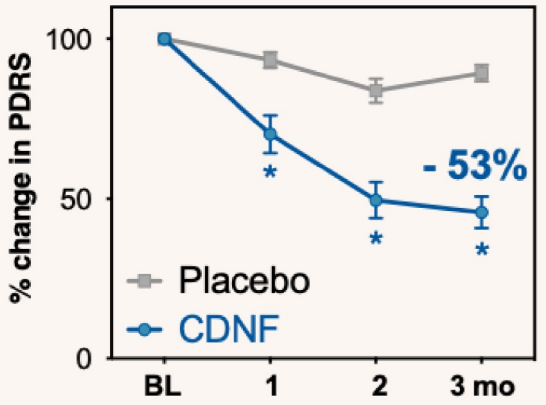
Neurorestoration by intraputamenal CDNF protein in a Rhesus model of PD



CDNF protein used as proof-of-concept for optimized therapeutic HER-096



2x increase in nigrostriatal dopamine neuron numbers with significantly enhanced axonal sprouting



>50% improvement in gross motor function on Rhesus PD rating scale (PDRS)

Three monthly doses of intraputamenal CDNF* were administered in an aged Rhesus monkey MPTP model starting six weeks after MPTP lesion. Clinical improvement was measured by PDRS and histological improvement was measured by TH staining at study endpoint.

*Intraputamenal CDNF was the proof of concept compound for HER-096. CDNF does not cross the blood-brain-barrier while HER-096 does. PK = pharmacokinetics; PD = pharmacodynamics.

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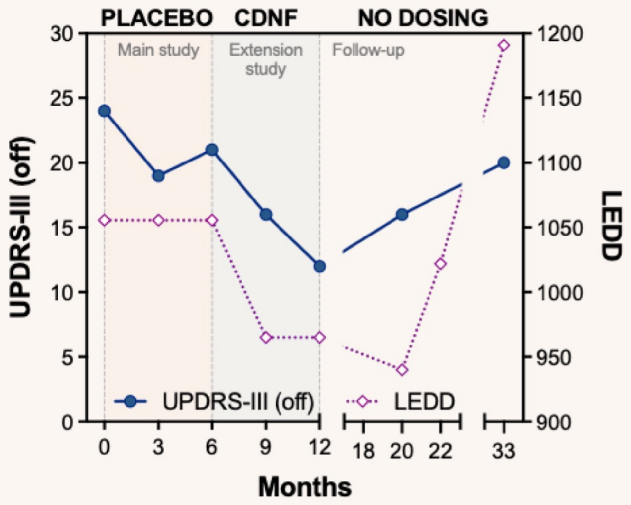
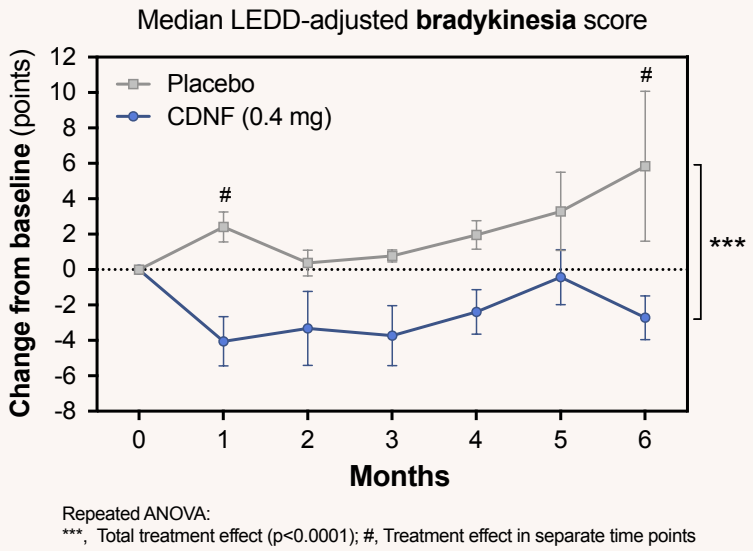
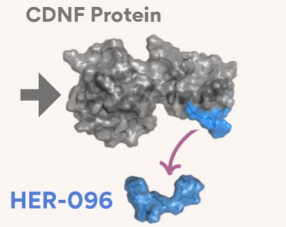
Phase 1 proof-of-concept study of intraputamenal CDNF shows signs of clinical and biological responses in advanced PD patients



Although primarily a safety study with advanced patients, clinical improvement was observed in several patients



Statistically significant improvement in bradykinesia (slowness of movement) seen already 1 month after starting CDNF administrations



Unlike HER-096, requires delivery via brain implant

An intraputamenal device was implanted for administration of CDNF. Patients were at least 10 years post-diagnosis and all showed advanced symptoms.

*Intraputamenal CDNF was the proof of concept compound for HER-096. CDNF does not cross the blood-brain-barrier while HER-096 does. Huttunen et al 2023, Movement Disorders, doi: 10.1002/mds.29426

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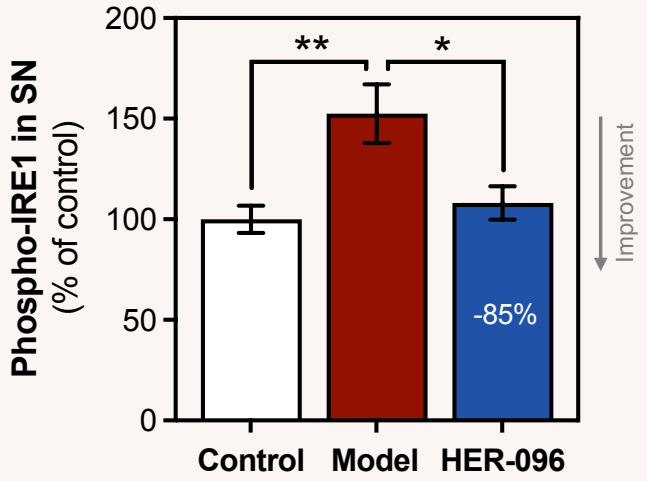
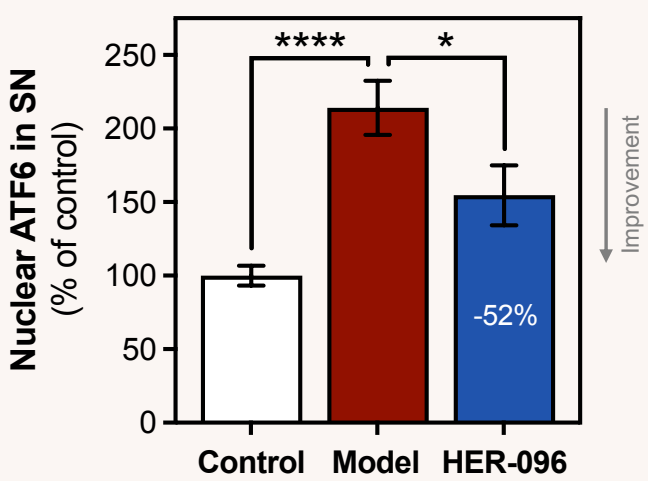
In vivo preclinical studies:

Subcutaneous HER-096 modulates the target UPR pathway in brain

UPR MARKER 1 (IRE1)



UPR MARKER 2 (ATF6)

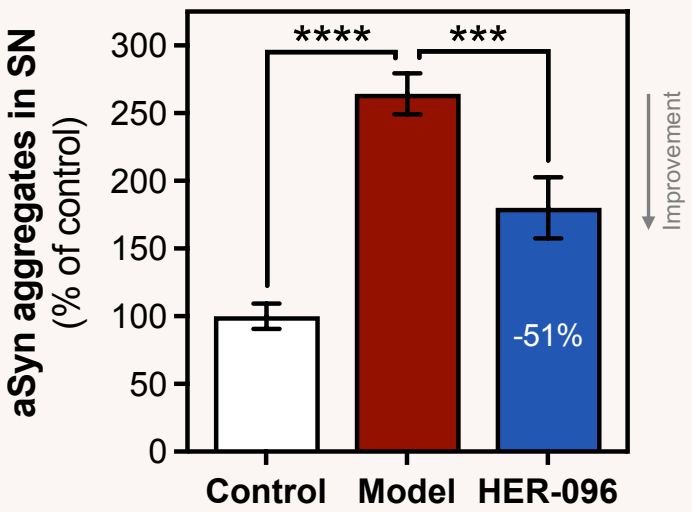


Mice were administered IP a human-equivalent dose of 200 mg HER-096 3 times per week for 4 weeks. Control = normal aged mouse without drug (vehicle only). PD model = PD model aged mouse without drug (vehicle only). HER-096 = PD model aged mouse with HER-096 drug. For substantia nigra (SN; graphs 1, 2, 4): Whole brains were prepared for immunohistochemistry of TH, Iba1, and α-Syn and the SN analyzed. For striata (graph 3): Striata were dissected from whole brain and lysed. Dopamine levels were determined by HPLC. Kulesskaya et al 2024, Cell Chem. Biol., doi: 10.1016/j.chembiol.2023.11.005

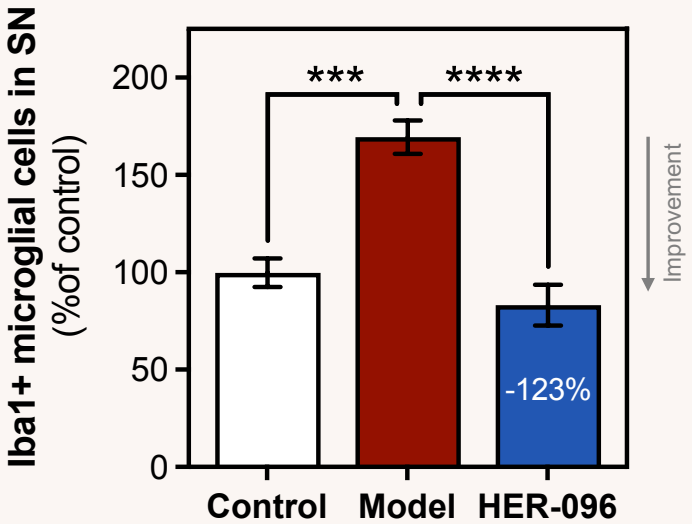
In vivo preclinical studies:

Disease modification – effect on α-synuclein and neuroinflammation

α-SYNUCLEIN AGGREGATES



NEUROINFLAMMATION



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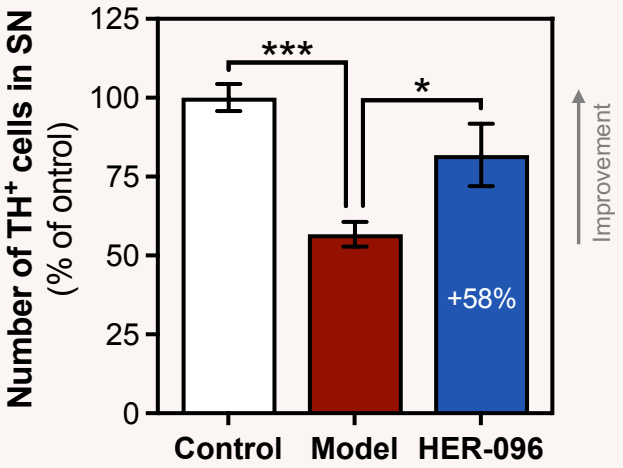
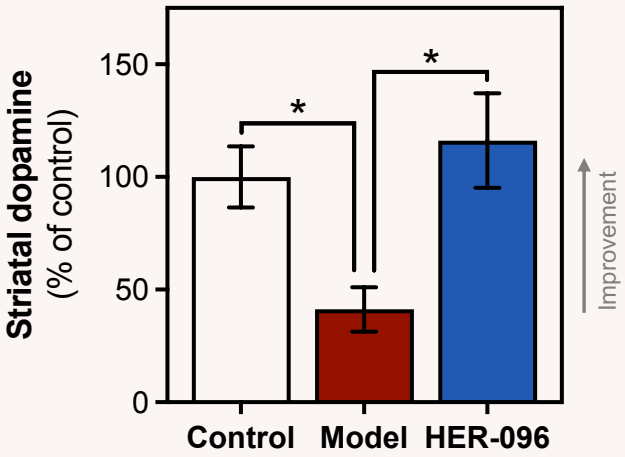
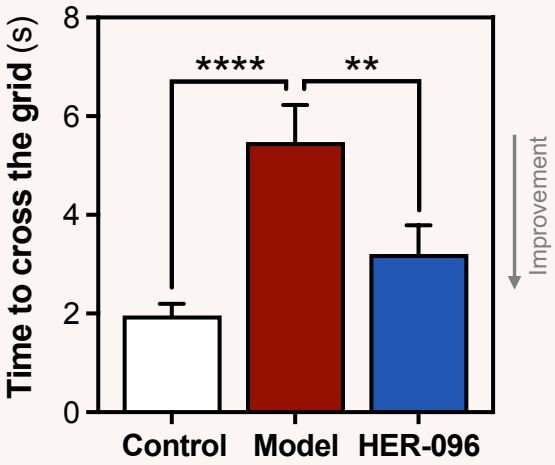
In vivo preclinical studies:

Disease modification – effect on dopamine system

PROTECTION OF DOPAMINE NEURONS → **DOPAMINE LEVEL IN STRIATUM** → **IMPROVEMENT OF MOTOR SYMPTOMS**







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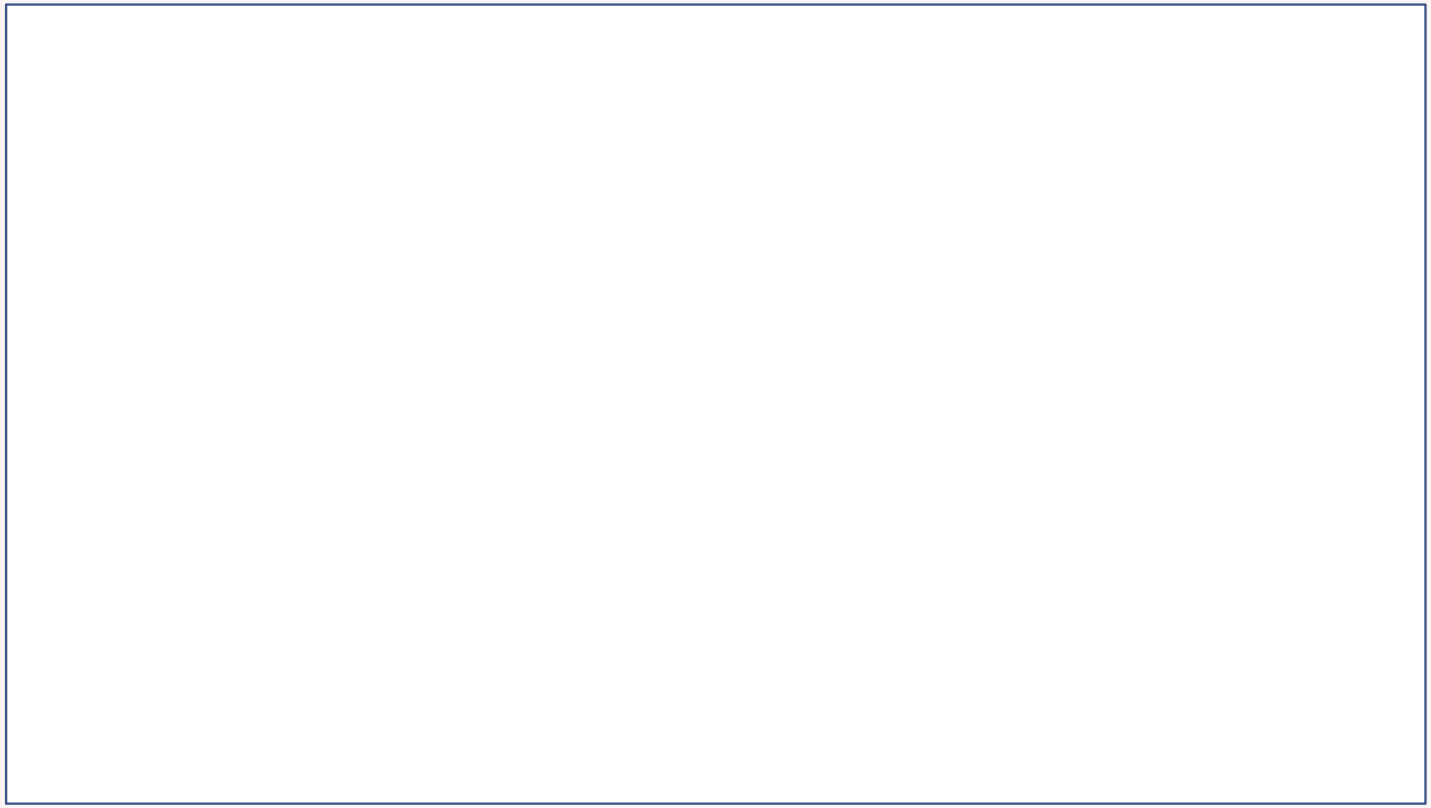
In vivo preclinical studies:

HER-096 demonstrate robust neuroprotection



PD mouse model administered HER-096 has superior beam walking speed to both untreated PD mouse and normal aged mouse

Completed HER-096 Phase 1a First-in-Human Trial

Conducted in 2023

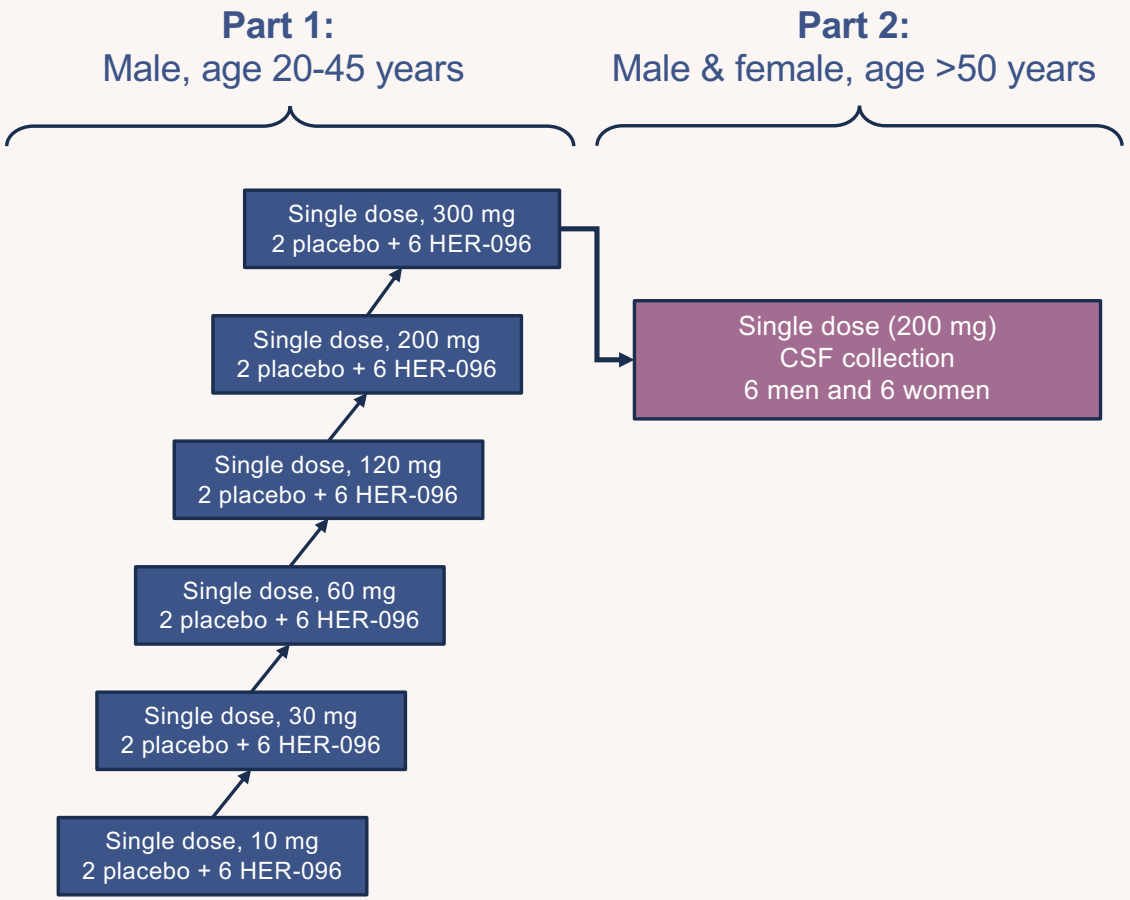
Study design:

- Double-blind, placebo-controlled clinical trial
- Subcutaneously administered single ascending dose of HER-096 to healthy subjects
- Total of 60 healthy subjects participated in the trial (48 in Part 1 and 12 in Part 2)

Main results:

- ✓ Single dose of HER-096 was found to be safe and well-tolerated
- ✓ Pharmacokinetics:
  - Pharmacokinetic profile was as expected
  - HER-096 penetrates the blood-brain barrier with pharmacologically active levels
- ✓ Identification of potential treatment response / pharmacodynamic biomarkers

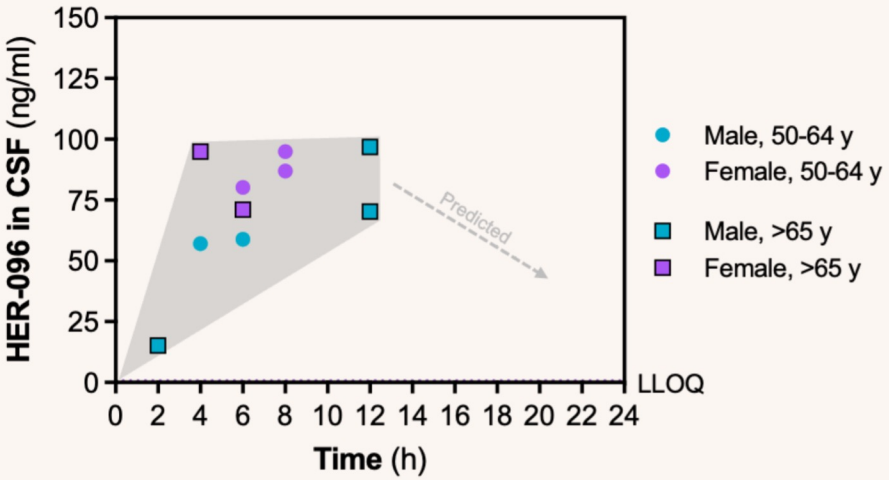
ClinicalTrials.gov ID: NCT05915247



Phase 1a Clinical Trial of Subcutaneous HER-096:

HER-096 penetrates efficiently the brain

Levels of HER-096 in CSF after subcutaneous administration



HER-096 (200 mg dose) effectively penetrates BBB to reach CSF concentration predicted to be within therapeutic range

Phase 1b Clinical Trial Design

Study started Sep 2024 / Part A completed in Nov 2024 / expected topline data Q3/2025

PART A (completed)

Single Dose (300 mg)
Safety, CSF T½ in **elderly HV**

Analysis + DSMB

N = **12 active**
Dose: **300 mg**

Completed in Nov 2024:

- PK profile in CSF ➔ data aligned with Phase 1a and enabling Phase 2 planning

PART B (ongoing)

Multiple doses
Cohort 1 (200 mg)
Safety & biomarkers in **PD patients**

DSMB safety review

N = **8 active + 4 placebo**
Dose: **200 mg** 2 x week, for 4 weeks
(+ 4-week follow-up after the last dose)

Multiple Doses
Cohort 2 (300 mg)
Safety & biomarkers in **PD patients**

DSMB safety review

N = **8 active + 4 placebo**
Dose: **300 mg** 2 x week, for 4 weeks
(+ 4-week follow-up after the last dose)

Outcome measures:

- Safety and tolerability of repeated doses of HER-096 in PD patients
- Pharmacokinetics of repeated dosing
- Exploratory biomarker analyses with focus on assessment of biological response to treatment
- Symptom assessment by UPDRS (clinical scale) and a wearable device

Key Advantages of HER-096

Neurorestorative

Disease-modifying MOA shows evidence of not just slowing but reversing striatal damage for symptomatic improvement

Subcutaneous

Patient-friendly administration 1-3 times weekly, while effectively crossing the blood-brain-barrier for therapeutic effect

Safe

No systemic toxicity or serious adverse events noted, including those common with other PD treatments (dyskinesias, neuropsychiatric issues, hypo/hypertension)

Business & Funding

The unmet medical need in PD is matched by a large global market for PD therapeutics

$9B

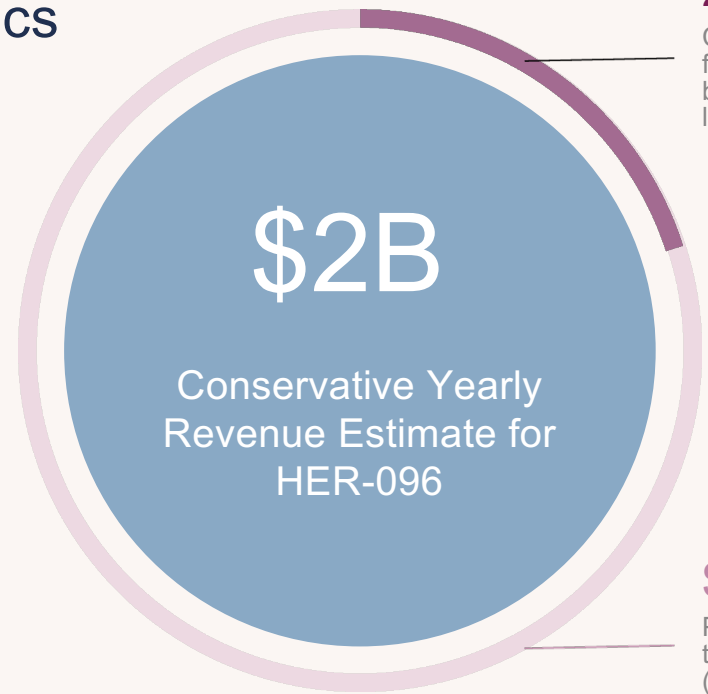
Addressable market in US and EU5 by 2030

1.1M

Total PD patients in US and EU

3.2%

Estimated CAGR of PD therapeutics market through 2030



20%

Conservative estimate for market capture based on competitive landscape

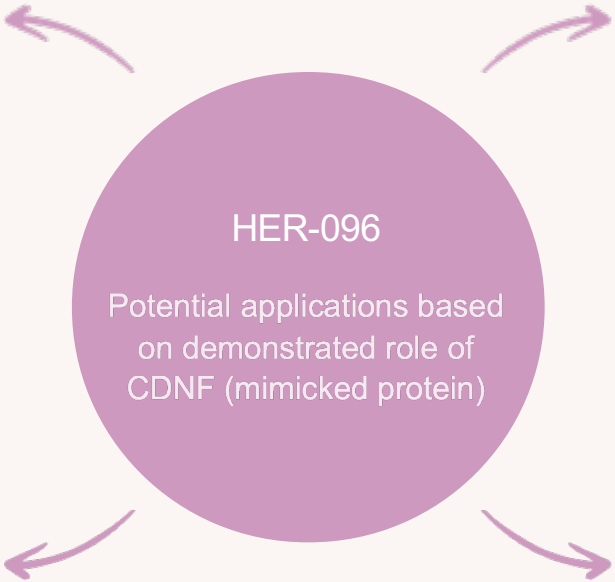
$10B

Projected market in target geographies (number of patients x[1] projected cost)

1) Based on estimated drug costs at 20% premium to Duodopa price from bioscience valuation for Herantis (up to $45K/patient), variability across geographies and with different payees (e.g., Medicare), and number of patients across target geographies (US and EU)

BEYOND PD:

HER-096 has potential utility in several neurodegenerative and ischemic diseases



ALS

CDNF has demonstrated therapeutic effect in multiple preclinical models of amyotrophic lateral sclerosis, including protection of motor neurons and improvement of motor function

Alzheimer's disease

CDNF has been shown to improve long-term memory in a preclinical model of Alzheimer's disease

Stroke

CDNF has improved functional recovery in preclinical models of ischemic and hemorrhagic stroke owing to its role in neurogenesis previously ischemic neurons

Neuroregeneration

CDNF is broadly involved in neuronal regeneration and has many theoretical implications across diverse modes of injury, ischemia, aging, and neurodegeneration

HIGHLIGHTED PRESS RELEASE

Secured research funding for Phase 1b Clinical Trial of HER-096 for PD

THE MICHAEL J. FOX FOUNDATION
FOR PARKINSON'S RESEARCH

PARKINSON'S UK
CHANGE ATTITUDES.
FIND A CURE.
JOIN US.

€3.6M cash

In 3 tranches over 2 years, contingent on milestone completion

Repayment triggered by out-licensing or sales revenue

Projects supported

1. Fund Phase 1b Clinical Trial of HER-096 for PD
(repeated subcutaneous dosing safety in PD patients)

2. Support ongoing biomarker identification project
(exploratory biomarker data collection during Phase 1b trial, and ongoing research on predictive outcomes)

Herantis has secured €6.1M non-dilutive research funding since April 2023
European Innovation Council (EIC) Accelerator grant of €2.5M in April 2023 for advancing Phase 2 preparation

Co-funded by the European Union

Herantis has secured €15M investment commitment from EIB
EIB committed direct equity investments through EIC Fund. First tranche of €1.5M raised in December 2023.

Preparing for Phase 2 trial of HER-096 for PD

Milestone	Program	2H 2024	1H 2025	2H 2025	1H 2026	2H 2026+
Phase 1b Trial	HER-096 for PD	●	●	●		
Phase 2 Trial	HER-096 for PD				●	●
Seek Out-lic./Collab.	HER-096 for PD	●	●	●	●	●

Partnering status

Summary of partnering interactions 2023 - 2025

- ✓ 75 meetings held
- ✓ 55 Pharma targets met

What can trigger a partnering agreement

- ✓ Phase 1b clinical trial data read-out (expected 2H/2025)
- ✓ More biomarker data linking HER-096 to PD, both preclinical and Phase 1b clinical (continuously accumulating)
- ✓ More data on pharmacokinetics / pharmacodynamics, both preclinical and clinical Phase 1b (2H/2025)
- ✓ Readiness to launch a Phase 2 program that can provide clinical proof of concept and/or has meaningful biomarker endpoints (expected to start Phase 2 in 2026)

Executive Team

Antti Vuolanto, DSc

CEO

20+ years experience in financing, partnering, R&D, and biologics manufacturing; D.Sc. in Bioprocess Engineering

Tone Kvåle

CFO

25+ years experience in biotech, medtech, and life science, including multiple prior roles as CFO of public biotech companies

Henri Huttunen, PhD

CSO, Co-Founder

25+ years experience in neuroscience and therapeutics development; served as CEO prior to transitioning to CSO

BOARD OF DIRECTORS

Timo Veromaa, MD, PhD, EMBA

Chairman of the Board

Current: Professor of Practice of Drug Development, University of Turku, Finland; Chairman of Teneboron Ltd.
Former: Exec. Chairman DomainEx Ltd; CEO & Pres. of BioTie Therapies (acq. Accord Tx); Chairman Finnish Bio Banks; Chairman Finnish Bioindustries

Frans Wuite, MD, MBA

Vice Chairman of the Board

Current: Board Director Healthcap VII GPSA; co-founder Rigi Tpx AG
Former: CEO Acesion Pharm ApS; CEO & President Oncos Tpx; COO Warren Pharma; Co-founder & Board Director Araim Pharma; Amgen senior management

Aki Prihti

Board Member

Current: CEO Aplagon Oy; Board member Rokote Lab Finland; Founding partner Inveni Capital
Former: Board member & CFO HVR Cardio; Corporate Finance at Salomon Brothers

Mats Thorén

Board Member

Current: CEO Vixco Capital; Board member Arcoma AB, Xbrane Biopharma, and FluoGuide
Former: Founding parter Catella Healthcare

Hilde Furberg, MSc

Board Member

Current: Industrial advisor Investinor; Board member Pluvia Biotech, Bio-Me, PCI Biotech, Calliditas, Sedana Medical
Former: European Head of Rare Disease Europe/General Manager Diseases EMEA at Genzyme/Sanofi

SCIENTIFIC ADVISORS

Anders Gersel Pedersen, MD

Chairman of the Scientific Advisory Board

Current: Board Member and Scientific Committee Chair at Hansa Biopharma; Board Member at Genmab; Deputy Chairman of the Board at Bavarian Nordic; Chairman of the Board at Aelis Farma
Former: 19 years at Lundbeck, including as EVP of R&D; Director of oncology clinical research at Eli Lilly; Board Member at TopoTarget and ALK-Abelio

Alberto Espay, MD, MSc

Scientific Advisory Board Member

Current: Director, Professor, and endowed chair of the University of Cincinnati Gardner Family Center for Parkinson's Disease and Movement Disorders (OH, USA)
Former: Chair of the Movement Disorders Section of the American Academy of Neurology; associate editor of Movement Disorders (journal); 300+ academic papers

David Dexter, PhD

Scientific Advisory Board Member

Current: Associate Research Director of Parkinson's UK; Visiting Professor of Neuro-pharmacology at Imperial College London; biology lead for Parkinson's virtual biotech
Former: Deputy Head of the Division of Brain Sciences at Imperial College London; founder of Parkinson's UK Brain Bank; identified 3 of the 6 mechanisms linked to PD

Daniele Bravi, MD

Scientific Advisory Board Member

Current: Associate Prof. at the Movement Disorder research center, S. Raffaele Inst.
Former: Vice President of PD Strategy at Lundbeck R&D; CMO and VP of Drug Development at Lundbeck USA; EFPIA clinical development group member; Speaker of the European School for Scientific and Regulatory Affairs

Highlights

- First-in-class disease-modifying, neuro-regenerative therapeutic for Parkinson's Disease
- Phase 1 trial of key compound CDNF demonstrated clinical response in several patients, even with advanced-stage disease
- Successfully completed Phase 1a clinical trial of lead candidate HER-096
- Phase 1b trial ongoing - readout in Q3/2025
- Preparing for Phase 2 trials of HER-096 for PD

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