HER-096: Advancing Toward the First Disease-Modifying Treatment for Parkinson's Disease

September 2026

Herantis Pharma Oyj (HEL: HRTIS)

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Herantis Pharma

Nasdaq First North
HRTIS

Clinical-stage Company Developing Disease-Modifying Therapies for Parkinson's Disease (PD)

Parkinson's remains a major unmet clinical need

- Current treatments improve symptoms but do not slow disease progression
- No disease-modifying treatments available

HER-096: differentiated disease-modifying approach

- Brain-penetrant peptide for subcutaneous administration
- Designed for neuroprotection and neurorestoration

Next: Phase 2 built to deliver proof-of-concept

- The field still lacks convincing proof of disease modification
- Successful HER-096 Phase 2 data could represent a major breakthrough in Parkinson's treatment

Aim: slow or stop Parkinson's disease progression

Executive Team with Deep Expertise in Drug Development

Complementary expertise spanning clinical development, translational science, drug discovery, partnering, corporate strategy, and finance

Dr Antti Vuolanto, DSc (Tech)
Chief Executinve Officer
Experienced biotech executive with a strong track record in financing, partnering, and advancing biological drug programs

Joined **Herantis Pharma** in 2018 as COO

Previously served as COO at **Valo Therapeutics,** Executive Vice President at **Targovax ASA** and COO and co-founder at **Oncos Therapeutics**

DSc in Technology at **Aalto University** in bioprocess engineering

Dr Juha Savola, MD, PhD
Chief Medical Officer
Over 25 years of experience in drug development, regulatory affairs and executive leadership in neurology, gene therapy and rare diseases.

Previously Vice President, Clinical Development at **Spark Therapeutics,** senior leadership positions at **Teva Pharmaceuticals, F. Hoffmann-La Roche, Santhera Pharmaceuticals** and **Juventia Pharma.**

Juha is a physician-scientist with an MD and PhD from the University of Oulu, Finland.

Dr Henri Huttunen, PhD
Chief Scientific Officer
Co-founder of Herantis, a leading expert in neurodegenerative disease biology with 25+ years of neuroscience research experience

Co-founded **Herantis Pharma** in 2008 and served as the company's founding CEO for 2 years

PhD in biochemistry from **University of Helsinki** and adjunct professor at **University of Helsinki**.

Tone Kvåle
Chief Financial Officer
Over 25 years of financial leadership experience in biotech and life sciences, including CFO roles in public companies

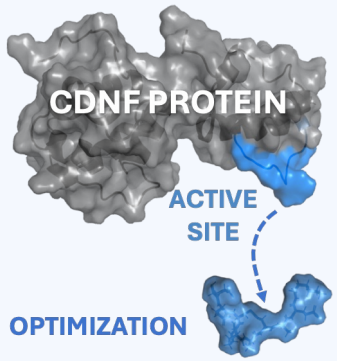
Previously as CFO at **Nordic Nanovector**, **NorDiag**, **Kavli Holding** and **Dynal Biotech**

Held senior management positions at **Invitrogen/Life Technologies** (now part of **ThermoFisher**)

Board member of **Medincell SA** (MEDCL), and previously board member of **LifeCare ASA** and **Bonesupport AB** (BONEX)

HER-096: a strong foundation for Phase 2 development

Validated Biology to Phase 2



Earlier validation of the biology

- CDNF biology supporting **neuronal survival & function**
- Extensive **clinical and preclinical evidence**

Strong biological foundation

HER-096 retains key CDNF biology

- Brain-penetrant peptide for **subcutaneous administration**
- Preclinical evidence of CDNF-like **neuroprotection and neurorestoration**

HER-096: CDNF biology designed for practical delivery

Highly encouraging Phase 1 data

- Safe and well tolerated with **efficient brain penetration**
- Biomarker changes in Parkinson's patients consistent with HER-096 biology

Clinical translation demonstrated

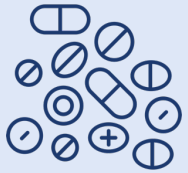
Next: Phase 2 proof-of-concept

- ~100 early-stage Parkinson's patients
- 15-month study designed to demonstrate meaningful clinical benefit

Built to deliver clear proof-of-concept data

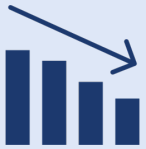
Parkinson’s Disease: Opportunity Continues to Expand

Unmet clinical need



Current therapies only treat the symptoms, not the disease itself

Many patients have no symptomatic benefit or may have significant side effects



The effectiveness of current treatments decline over time as the disease progresses

Market is growing rapidly

10 → 25 million patients

Estimated increase until 2050

$ >250 billion

Estimated economic impact of Parkinson’s globally today

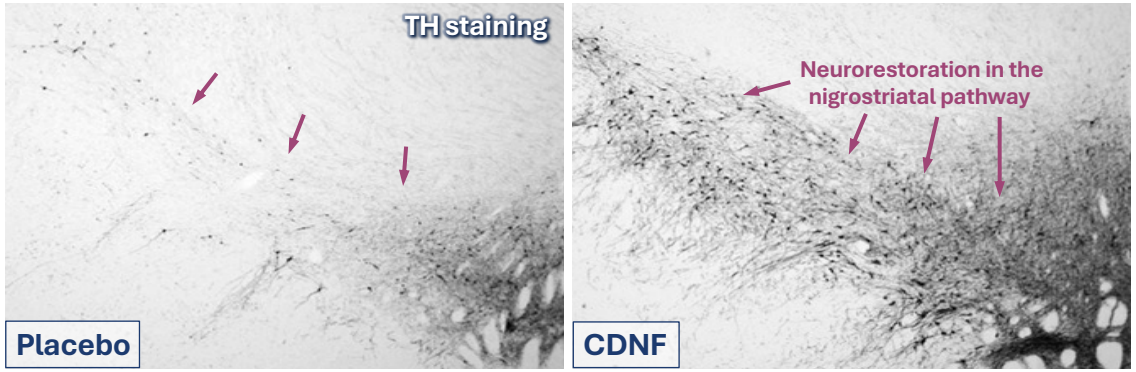
$ 13 billion

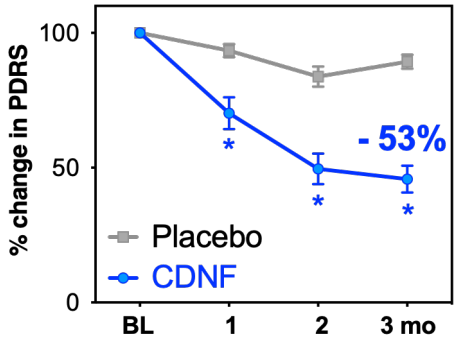
Forecasted therapeutic market in PD by 2034 ($6.6b in 2024)

Strong pharma interest in disease-modifying PD, e.g., Novartis licensed a-synuclein targeted preclinical asset from Arrowhead in 2025: $200m upfront + up to $2bn milestones

CDNF Demonstrated Restorative Potential in Parkinson's Disease

Neurorestorative Effect of CDNF in Aged Rhesus Monkey PD Model

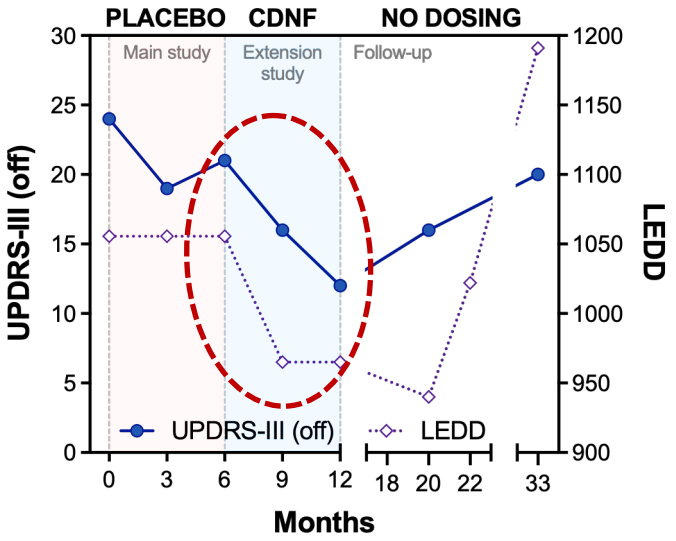


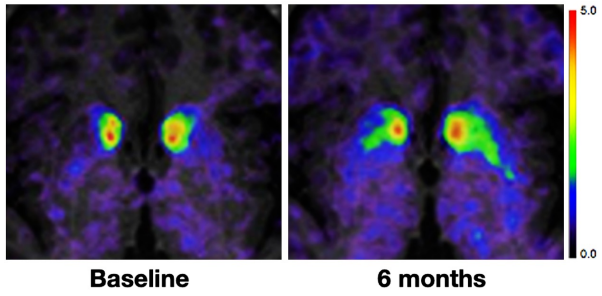


- Intraputamenal CDNF infusions promoted nigrostriatal **neurorestoration** in aged Rhesus monkey MPTP model (treatment started 6 weeks post-MPTP lesion)
- Improvement of both motor (PDRS-scale, above) and non-motor effects (observations by animal care takers)

Preliminary Evidence of Biological Response in PD Patients

- Phase 1 study with intraputamenal CDNF in moderately advanced Parkinson's patients (>10 years from diagnosis)(NCT03295786)
- Intraputamenally infused CDNF was safe and well tolerated
- Although primarily a safety study with PD patients, several individual subjects showed signs of biological response or clinical improvement
  https://movementdisorders.onlinelibrary.wiley.com/doi/10.1002/mds.29426





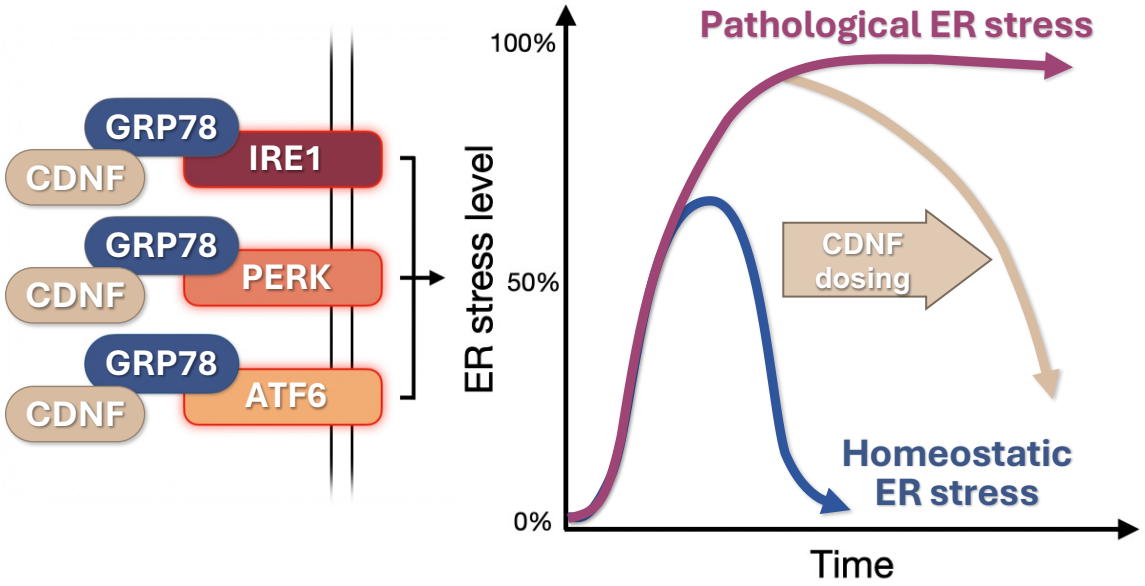
Imaging data from individual patients: increased dopamine transporter (DAT) availability in the putamen and substantia nigra following CDNF infusions

Clinical data from individual patients: 10-point improvement in UPDRS Part III (off) following initiation of once-monthly intraputamenal CDNF infusions, accompanied by a reduction in levodopa equivalent daily dose (LEDD) due to the development of dyskinesia, which indicates increased dopaminergic activity.

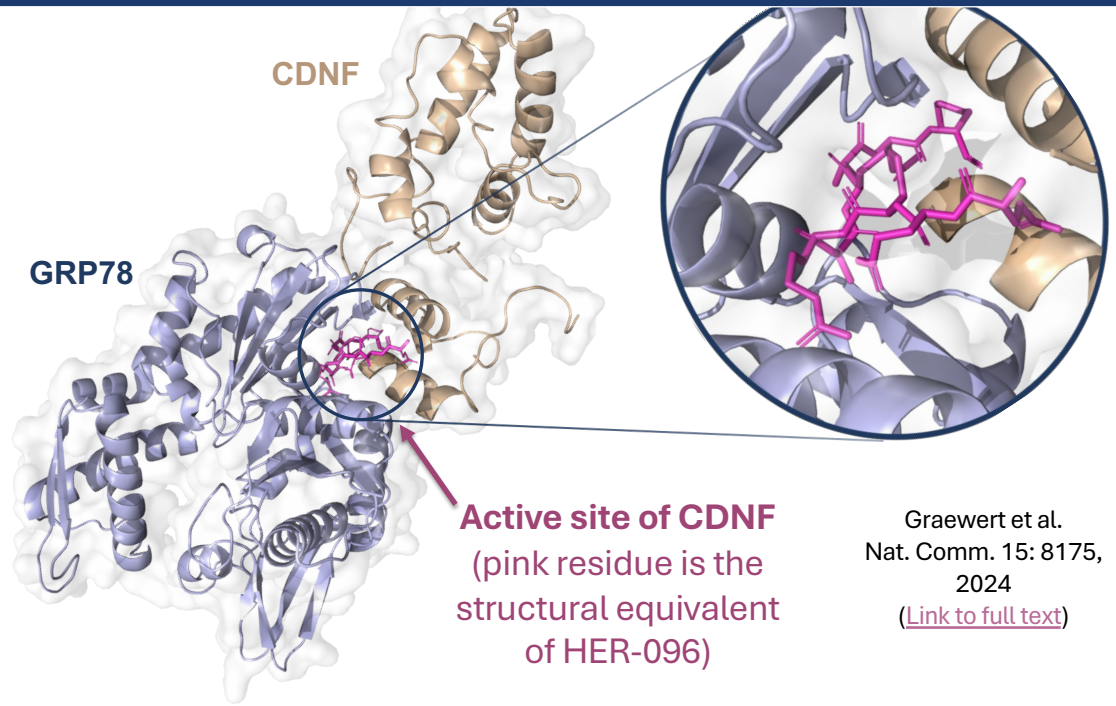
HER-096 Mimics CDNF's Neuroprotective Activity via Interaction with GRP78

GRP78 Serves as the Master Regulator of the Unfolded Protein Response (UPR) Pathway

CDNF modulates the UPR to restore ER homeostasis



HER-096 is derived from the CDNF / GRP78 binding interface



Graewert et al. Nat. Comm. 15: 8175, 2024 (Link to full text)

UPR maintains cellular protein homeostasis

- UPR responds to ER stress, including the accumulation of misfolded proteins, and activates adaptive responses to restore proteostasis
- Under normal conditions, the response is transient and returns to homeostasis
- In Parkinson's disease, persistent ER stress can drive maladaptive UPR signaling, neuronal dysfunction, and cell death
- CDNF modulates the UPR, helping restore a balanced, homeostatic stress response

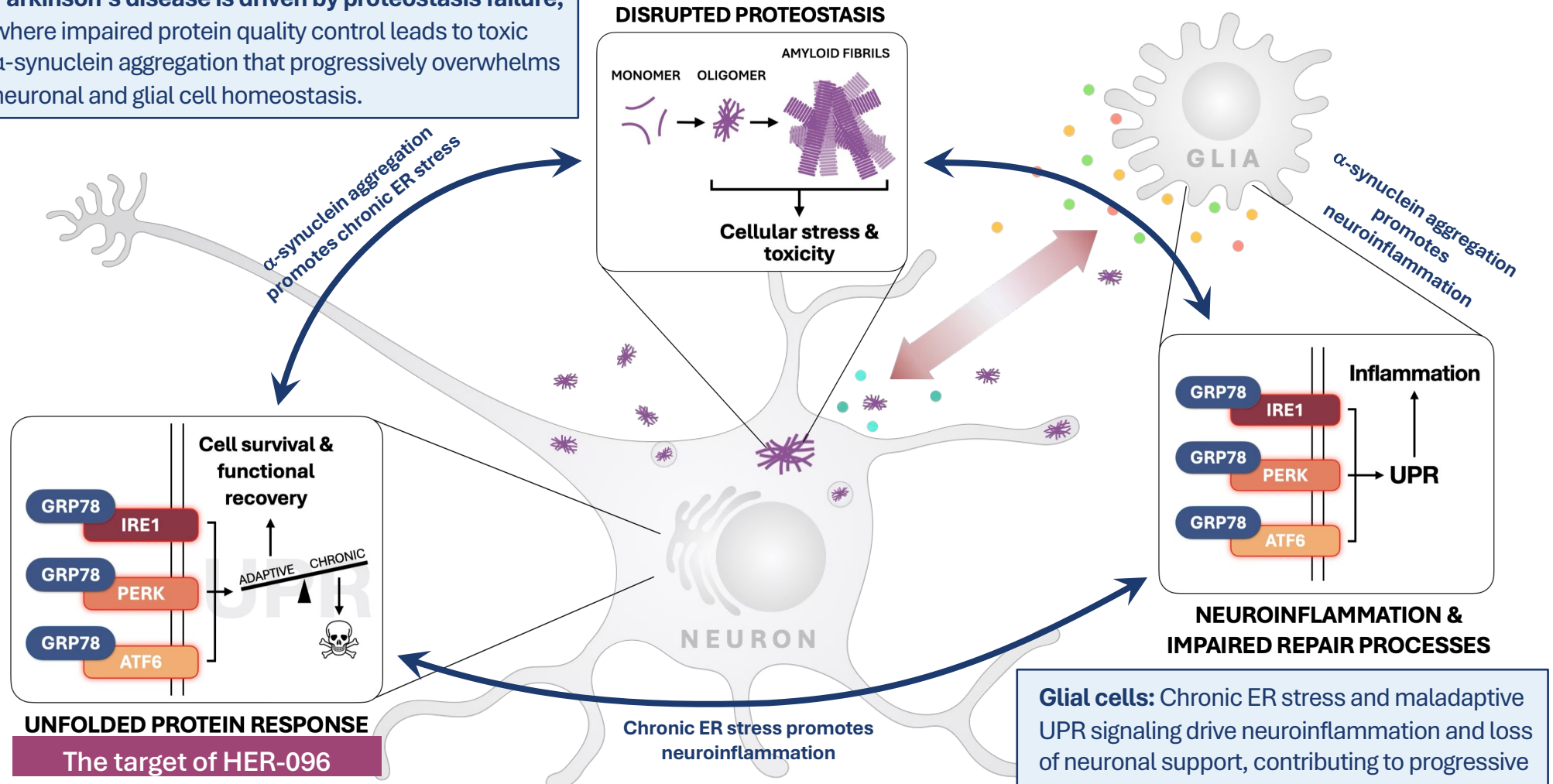
HER-096 is designed to retain CDNF's UPR-modulating activity with a drug-like profile

- Brain penetration enabling subcutaneous administration
- Fully synthetic with improved metabolic stability

Parkinson's disease: A Vicious Cycle Drives the Disease Pathology

Accumulation of Toxic α-Synuclein Aggregates, Chronic ER Stress and Neuroinflammation

Parkinson's disease is driven by proteostasis failure, where impaired protein quality control leads to toxic α-synuclein aggregation that progressively overwhelms neuronal and glial cell homeostasis.

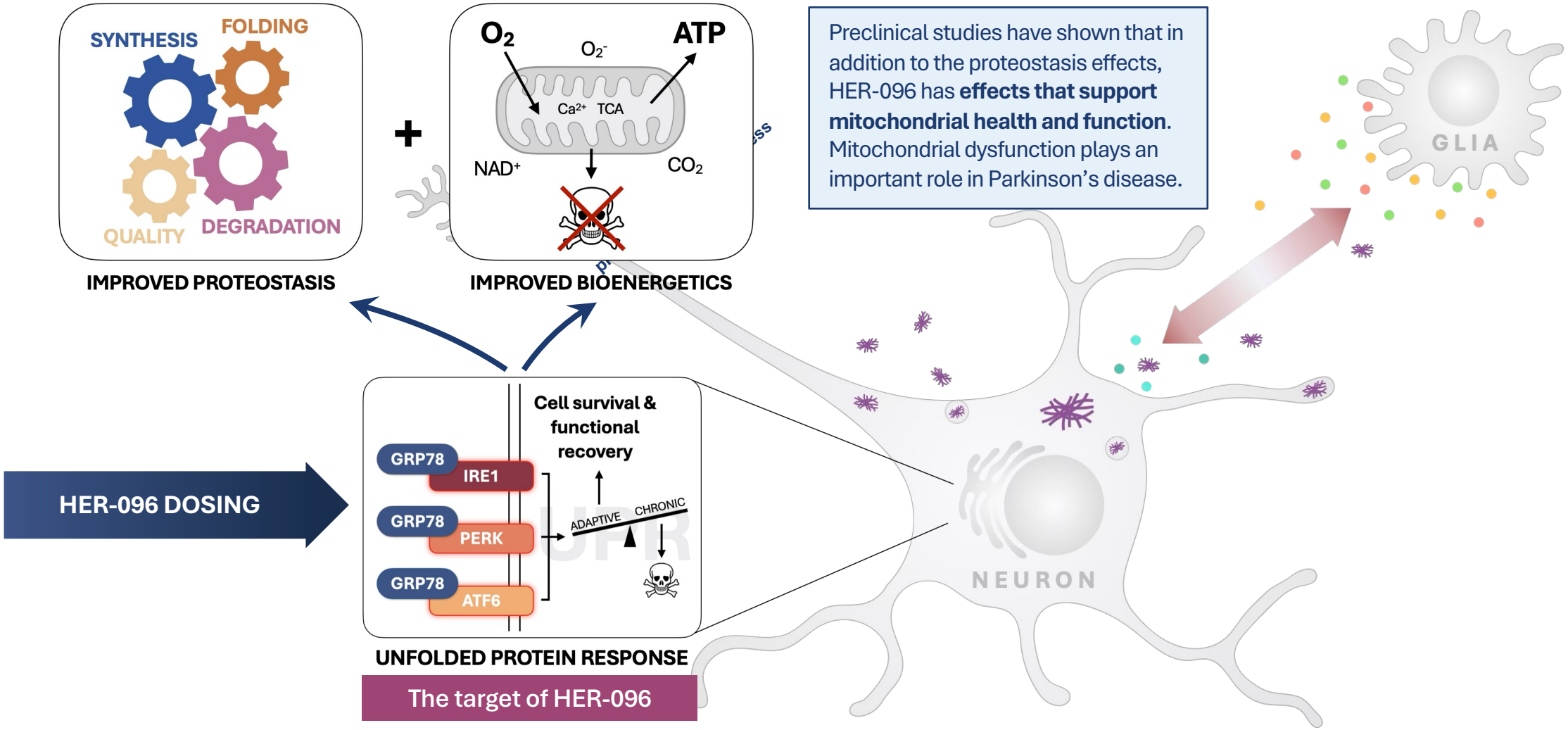


In Parkinson's disease, **unresolved ER stress locks the UPR into a chronic, maladaptive state** that suppresses neuronal maintenance, drives inflammation, and **accelerates dysfunction and degeneration of dopamine neurons.**

Glial cells: Chronic ER stress and maladaptive UPR signaling drive neuroinflammation and loss of neuronal support, contributing to progressive dopaminergic neurodegeneration.

Preclinical Studies: HER-096 Modulation of UPR Showed Broad Effects on Neuronal Functionality

Restorative Effects on Neuronal Proteostasis and Mitochondrial Function

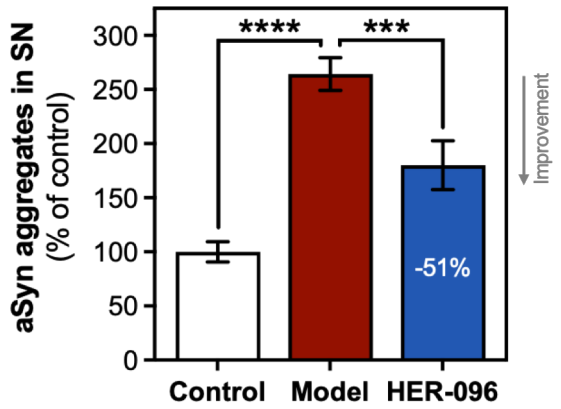


HER-096 Demonstrates Broad Neurorestorative Effects in a PD Mouse Model

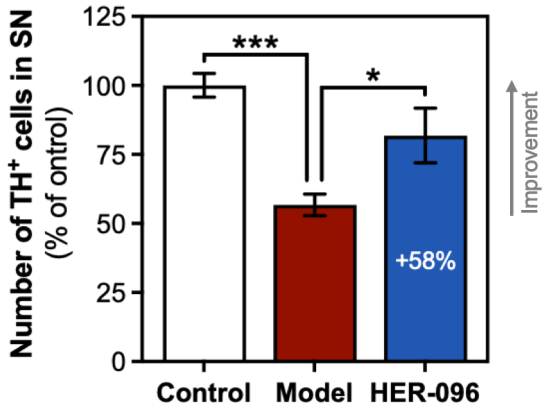
Reduced PD pathology is accompanied by preservation of the dopamine system and improved motor function

Disease Pathology → Dopamine System → Functional Outcome

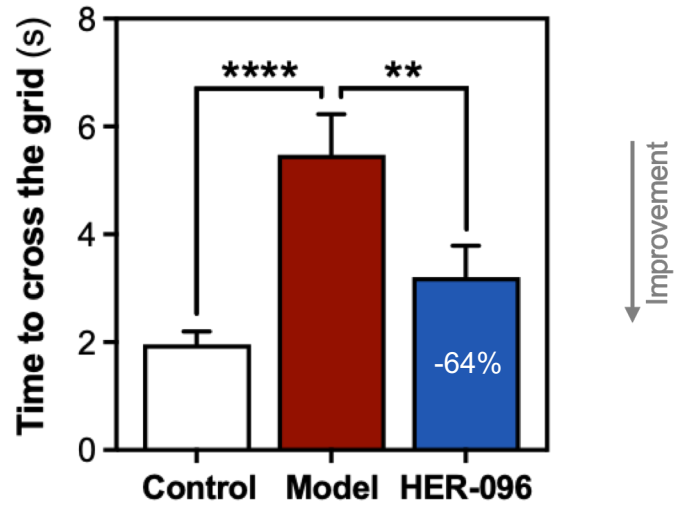
α-SYNUCLEIN AGGREGATES



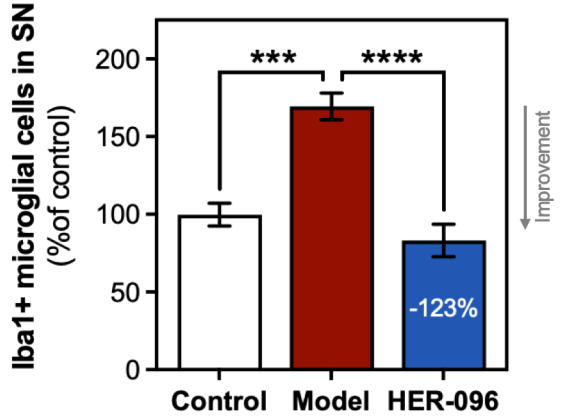
PROTECTION OF DOPAMINE NEURONS



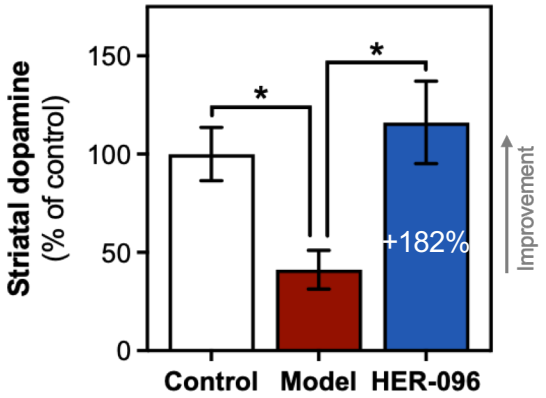
IMPROVEMENT OF MOTOR SYMPTOMS



NEUROINFLAMMATION



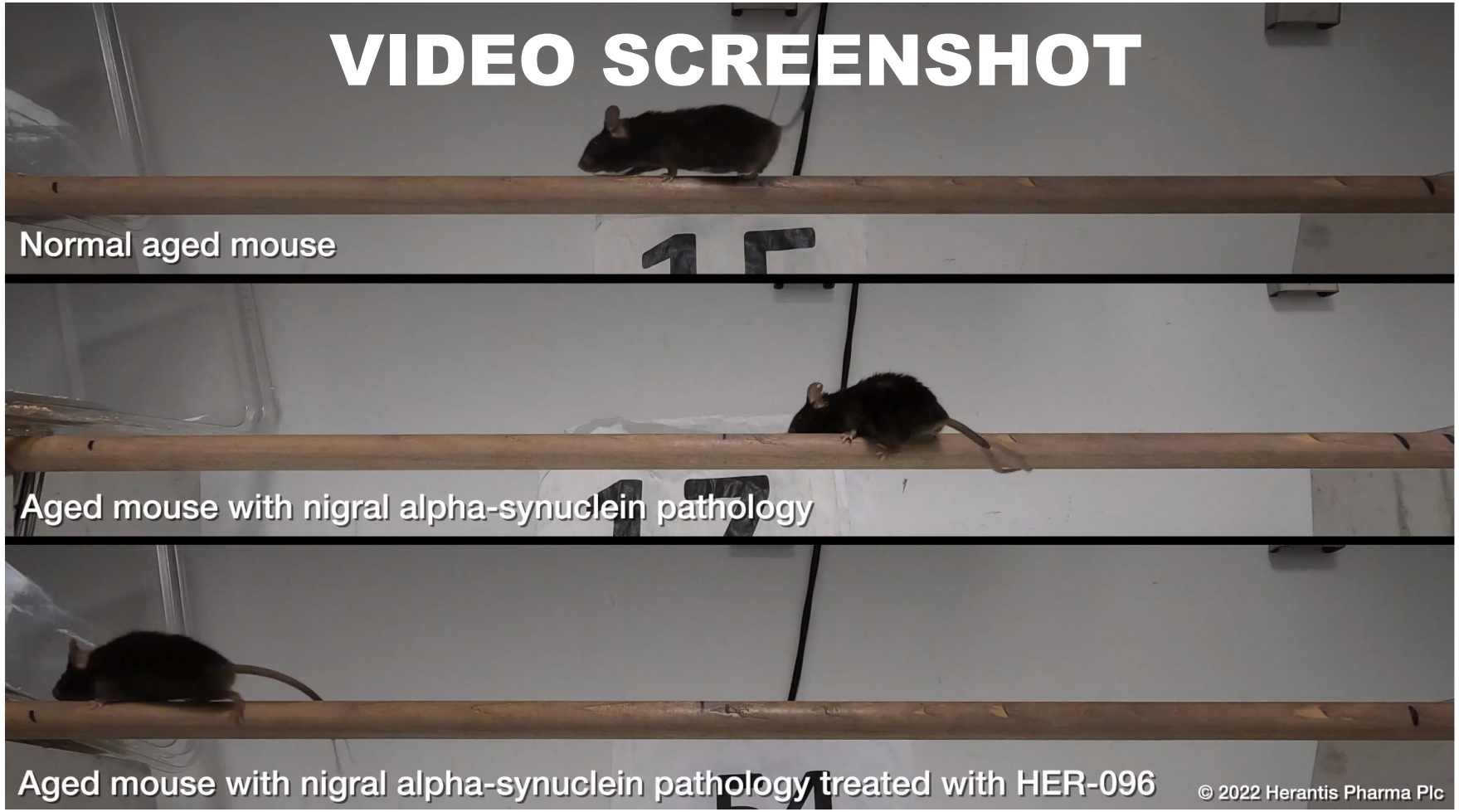
DOPAMINE LEVEL IN STRIATUM



* p<0.05
** p<0.01
*** p<0.001
**** p<0.0001

Aged α-syn/CBE PD mouse model; HER-096 10 mg/kg (human-equivalent dose: 200 mg); administered 3× weekly for 4 weeks.
Kulesskaya et al 2024, Cell Chem. Biol., doi: 10.1016/j.chembiol.2023.11.005

HER-096 Restores Motor Function in a Parkinson's Disease Mouse Model



CLICK THE BELOW LINK TO SEE THE VIDEO:

https://youtu.be/L3WmkhP2Opw

HER-096 Phase 1 Program Suggested Biological Response Aligned with MoA

PHASE 1A STUDY

Single Ascending Dose HER-096 (10 – 300 mg)
Safety and PK, including CSF $T_{½}$ in elderly healthy volunteers (HV)

N = 60

N = 48 young healthy individuals (2/6 placebo/active per dose level)
Dose levels: 10 – 300 mg (6 dose levels)

N = 12 elderly HVs
Dose: 200 mg
CSF sampling: 2 – 12 h (one sample per subject)

Main findings:

- Good safety and tolerability profile of single dose in healthy subjects
- Efficient brain penetration in elderly healthy individuals
- Favourable pharmacokinetic profile in young and elderly healthy subjects

ClinicalTrials.gov: NCT05915247

PHASE 1B STUDY

Single Dose HER-096
CSF $T_{½}$ in elderly HV

N = 8

N = 8 elderly
Dose: 300 mg
CSF sampling: 8 – 30 h
(one sample per subject)

Multiple Doses (200 or 300 mg)
Safety, PK & biomarkers in PD patients

N = 24

N = 16 active + 8 placebo
Doses: 200 or 300 mg 2 x week, for 4 weeks
(+ 4-week safety follow-up after the last dose)

CSF sampling: baseline & after the last dose

Main findings:

- Good safety and tolerability profile of repeated doses of HER-096 in PD patients (main findings are related to the injection site as expected)
- Pharmacokinetics in CSF: twice weekly dosing of 200 or 300 mg HER-096 are feasible for Phase 2
- Biomarker analysis: Biological response to HER-096 dosing aligned with mechanism

ClinicalTrials.gov: NCT06659562; EUCT: 2024-512532-30-00

Phase 1 summary: Safety and Brain Penetration in Humans

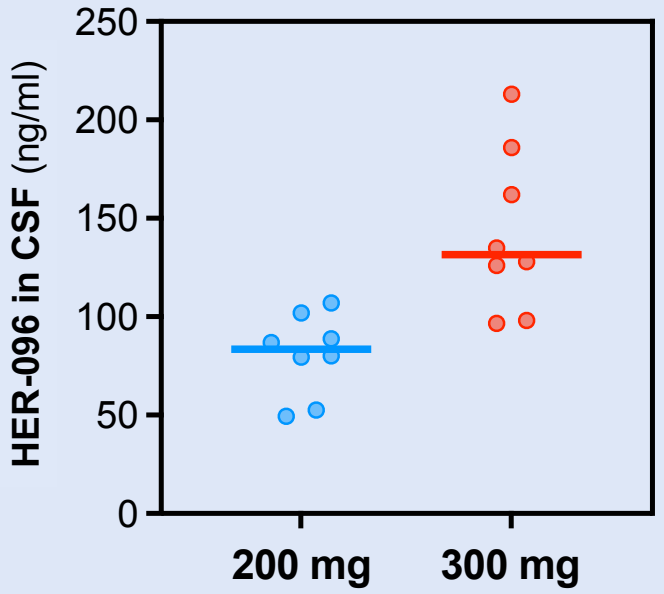
Clean safety profile

- Favorable safety profile observed across all tested dose levels, including highest planned clinical exposure
- Low incidence of systemic adverse events
- Most reported adverse events were related to the injection site, and were mild and transient

HER-096 dosings	Phase 1a	Phase 1b
Severe TEAEs*	0	0
Serious Adverse Events	0	0
Dose Limiting Toxicities	0	0
Maximum Tolerated Dose	Not reached	Not reached

*TEAE = Treatment Emergent Adverse Event

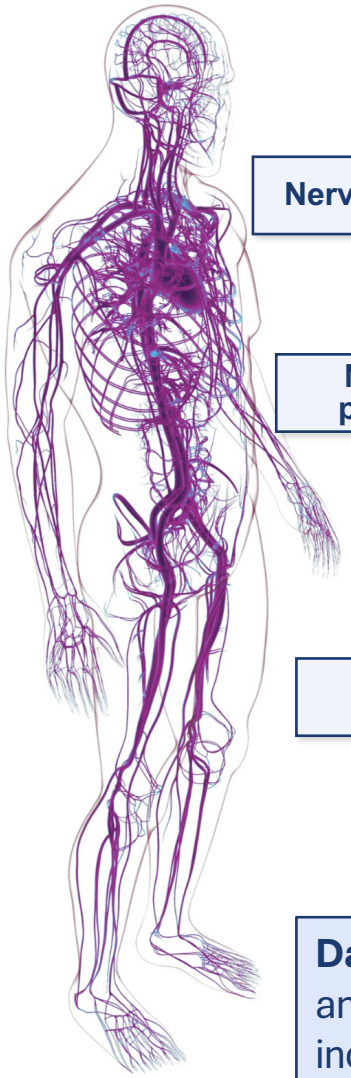
Dose-dependent CSF exposure of HER-096 demonstrated in Parkinson's patients



- CSF sampling at 8 h after last dose
- CSF levels well-correlated with plasma exposure
- CSF levels reached by both 200 and 300 mg doses match to the CSF levels reached by pharmacologically active dose levels in preclinical models

Summary: Clear Biomarker Responses to HER-096 Dosing in PD Patients

Multiple Data Layers Show Concordant Shifts in Biology Aligned with Mechanism of Action



Nervous system markers

Neuronal-enriched plasma EV markers

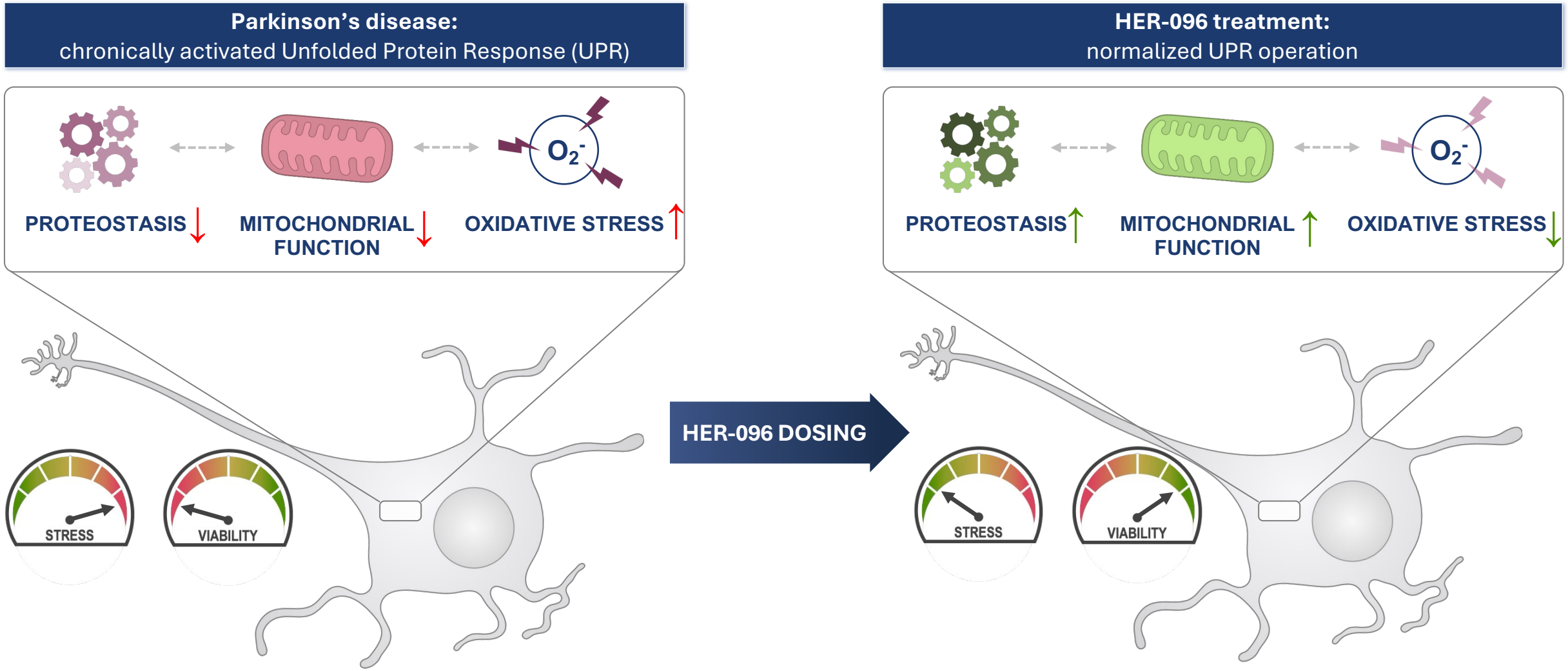
Systemic markers

Sample type	Cells / Source	System	Pathways affected	Interpretation
CSF	Neurons	Proteostasis and oxidative stress	Proteostasis and redox proteins ↑	Proteostasis modulation and improved oxidative stress defense
CSF	Immune and glial cells	Inflammation	Microglial polarization ↑↓	Neuroinflammation polarized toward resolution and repair
NeuroSPARC EV	CNS/ Peripheral	Mitochondria	Oxidative phosphorylation-related proteins ↓	Improved mitochondrial health results in reduced secretion of mitochondria-derived vesicles
NeuroSPARC EV	CNS/ Peripheral	Innate immunity	Type I interferon response ↓	Downregulation of innate immune signaling
Plasma	Peripheral tissue	Proteostasis	Multiple proteostasis systems ↑	Improved cellular proteostasis
Plasma	Peripheral tissue	Vesicle trafficking and autophagy	Multivesicular body, autophagy regulators, ESCRT complex ↑	Improved function of the endolysosomal pathway, enhanced autophagy/mitophagy
Whole blood	PBMC	Mitochondria	mtDNA lesions ↓	Improved mitochondrial health results in better mtDNA integrity
Whole blood	Blood cells	Mitochondria	Glutathione redox balance improved	Improved mitochondrial efficiency reduces oxidative stress

Data interpretation: Consistent biomarker changes across sample types and analytical approaches, affecting disease-relevant pathways, including proteostasis and mitochondrial function, and aligning with the proposed mechanism of action.

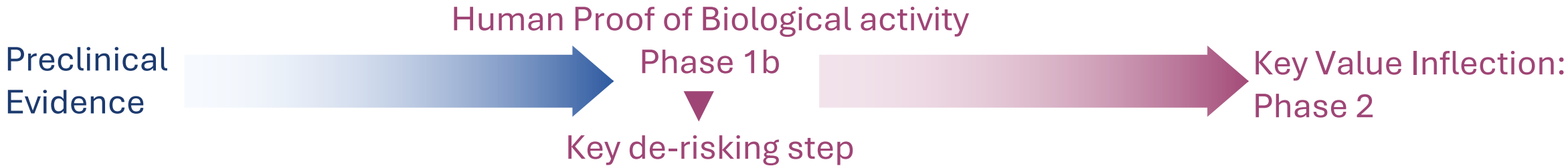
- **Proteostasis**
- **Inflammation**
- **Mitochondrial function & oxidative stress defense**

HER-096 dosing: Biomarker Changes Aligned with Restoration of UPR Function in PD patients



Phase 1 Provides Human Proof of Biological Activity and De-Risks Phase 2

HER-096 demonstrates translation from preclinical evidence to biological activity in Parkinson's patients



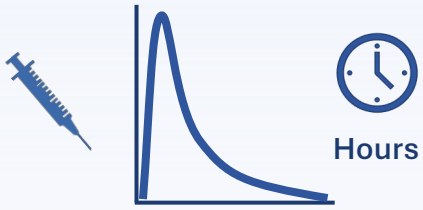
	Preclinical data	Human evidence – Phase 1b
Safety and tolerability	Supportive	Demonstrated in PD patients
Biological response	Demonstrated	Demonstrated in PD patients
MoA-related pathway effect	Demonstrated	Biomarker changes aligned with MoA
Functional benefit	Demonstrated in PD models	Phase 2 objective →

HER-096 Phase 1b demonstrates translation of CDNF biology into humans; Phase 2 tests translation into clinical benefit

Sustained Biological Effects Support Twice-Weekly HER-096 Dosing

Translating Sustained Biological Activity into Phase 2 Design

Drug Exposure



- Plasma half-life ~2 hours
- Transient exposure initiates downstream cellular responses

Biological Response

Biological effects persist beyond drug exposure

- UPR/proteostasis pathway modulation
- Downstream cellular responses

Sustained Effect

Sustained biological activity demonstrated

- Phase 1b: concordant changes across mechanistically linked biomarkers
- Preclinical HER-096 and CDNF studies: neuroprotection, reduced pathology and functional recovery

Phase 2 Translation

- 300 mg HER-096 twice weekly s.c.
- Digital Motor Score as Phase 2 proof-of-concept primary endpoint

Transient exposure drives sustained biology, supports intermittent dosing and longitudinal efficacy assessment

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Phase 2 for Efficacy Signal in Early-Stage Parkinson's Disease Patients

- A proof-of-concept study aiming to show improvement in motor symptoms
  - Continous objective monitoring of motor symptoms using a sensitive digital motor score (DMS)
  - Clinical symptom assessment (MDS-UPDRS Part II & III)
  - Brain imaging (neuromelanin-MRI, iron accumulation MRI, DaT-SPECT/PET, FDG-PET)
  - Selected fluid biomarkers based on Phase 1b data

- Randomized, placebo-controlled, double-blind multicenter trial

PART 1, 9 months:
- 100 subjects with early stage Parkinson's
- Randomized 1:1 – 300 mg HER-096 or placebo twice weekly s.c.

Interim efficacy readout

PART 2, 6 months:
All subjects 300 mg HER-096 twice weekly s.c.

Phase 2 data

- Multi-center study, sites in Europe
  - Finland, Sweden, Netherlands, Luxembourg, possibly UK, Spain, Italy
- Timelines: regulatory submission Q4/2026, first patient in 1H/2027, interim efficacy data 1H/2029, all data 2H/2029

Feedback supports planned Phase 2 development strategy
- FDA considered the Phase 2 trial design appropriate for the current stage of development
- No concerns raised regarding CMC or preclinical packages
- Feedback supports submitting IND

HER-096: Strong Position Within PD Disease-Modifying Landscape

Uniquely broad mechanism of action addressing core pathology with potential to protect and restore neuronal function

- Late-stage pipeline of disease-modifying Parkinson's therapies remains limited
- Most competing programs target specific disease mechanisms or patient subpopulations
  - α-synuclein immunotherapies
  - LRRK2 inhibition
  - GBA/lysosomal pathways
  - GLP-1/metabolic approaches
- Cell replacement therapies
  - Broad commercial adoption constrained by invasive surgery, manufacturing complexity, limited scalability and high treatment costs

Phase 3 / Pivotal

- Prasinezumab (Roche/Prothena): α-synuclein antibody
- Bemdaneprocel (BlueRock/Bayer): cell replacement

Phase 2 / Proof-of-concept

- BIIB122 (Denali): LRRK2 inhibitor, Phase 2b failed in idiopathic PD, development continues in genetically selected LRRK2-PD
- Bezisterim (BioVie): Phase 2 exploratory signals with oral small molecule targeting neuroinflammation (ERK/NF-κB/TNF-α pathways)
- TB006 (TrueBinding): anti-galectin-3 antibody targeting neuroinflammation and α-synuclein aggregation (Phase 2a)

Strong External Validation and Significant Funding for Phase 2

€8M grant secured with multiple funding routes for the remaining need

External Validation

Parkinson's UK and MJ Fox Foundation

- Funded the HER-096 Phase 1b clinical trial in Parkinson's disease (€3.6M)

Parkinson's UK

THE MICHAEL J. FOX FOUNDATION FOR PARKINSON'S RESEARCH

European Union

- €2.5M grant for biomarker development
- €8M grant for Phase 2 conduct
- Equity investment commitment through European Innovation Council Fund (EIC Fund)

Co-funded by the European Union

Phase 2 Financing

€8M secured

€25-30M remaining need

Non-dilutive EU Horizon Grant for Phase 2 conduct

EIC FUND

- Total commitment €15M
- €4.2M invested / €10.8M remaining
- Up to 1/3 of an equity round

Financing routes

- EIC Fund
- Strategic alternatives
- Parkinson's organizations
- New equity

Funding HER-096 Through Phase 2 Value Inflection

- **€25–30M to reach Phase 2 efficacy data and establish disease-modifying potential in early Parkinson's disease**
- **Strong position**
  - Phase 1 completed: clean safety and confirmed brain penetration
  - Phase 1 biomarker response in PD patients aligned with mechanism of action
  - €8M EU grant secured to support Phase 2
- **Major value inflection**
  - **Phase 2 efficacy readout** across clinical, digital, imaging and biomarker endpoints
  - Data package supporting partnering and late-stage development

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