HERANTIS PHARMA

Neurorestorative therapeutics for Parkinson's disease

April 2026

Herantis Pharma Oyj (HEL: HRTIS)

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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.

Herantis Pharma

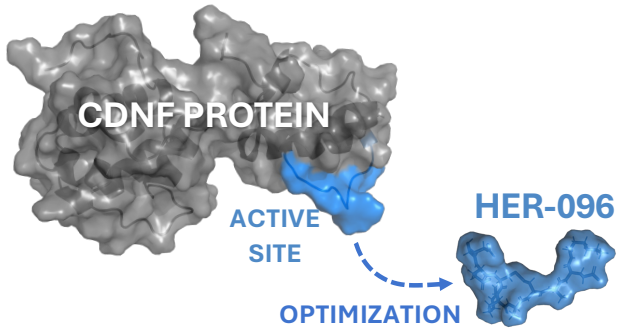
Nasdaq First North Growth Market Finland - HRTIS

- Clinical-stage public company developing neurorestorative therapies to stop Parkinson's disease progression
- Lead asset HER-096
  - Protect dopamine neurons from degeneration and support their functional restoration
  - Efficient brain penetration enabling administration under the skin
- Phase 1 program completed
  - Solid safety data: patients, healthy volunteers
  - Efficient brain penetration: patients, healthy volunteers
  - Biomarker data: biological response in Parkinson's patients
  - Phase 2 design nearing final confirmation
- Next
  - Phase 2 efficacy trial

HER-096

First-in-class peptide targeting the key drivers of Parkinson's disease

Mechanism of Action



- Small peptide based on the active site of CDNF
- Modulates Unfolded Protein Response (UPR)
- Restores **proteostasis**
- Reduces **neuroinflammation** and **a-syn aggregation**

➔**Protects dopamine neurons and supports their functional recovery**

Clinical and therapeutic profile

Disease-modifying and symptomatic potential
Slowing or halting mid-brain neuron degeneration

Biological validation
Biomarker response consistent with mechanism

Confirmed brain penetration
Demonstrated in Phase 1b

Convenient dosing
Subcutaneous administration twice weekly

A differentiated, brain penetrant therapy designed to stop the progression of Parkinson's disease

Parkinson’s Disease: High Unmet Need Driving Market Growth

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

$ >250 billion

Estimated economic impact of Parkinson’s globally today

$ 13 billion

Forecasted therapeutic market in PD by 2034

10 → 25 million patients

Estimated increase until 2050

HER-096: De-Risked and Ready for Phase 2

Reduced development risk

Established safety profile in humans

Confirmed brain penetration

Biomarker-confirmed biological activity

Human data reduce translational risk

Ready for Phase 2

Signal-seeking proof-of-concept trial

~100 early-stage Parkinson's patients

Multi-center European study

Study design nearing final confirmation

Differentiated asset

Designed to modify disease progression

First-in-class UPR-modulating mechanism

Targets core drivers of Parkinson's pathology

Distinct within the current clinical landscape

Multiple routes to Phase 2 execution

Capital strategy aligned to support Phase 2

Strategic partnering options under evaluation

Multiple financing pathways available (equity & non-dilutive)

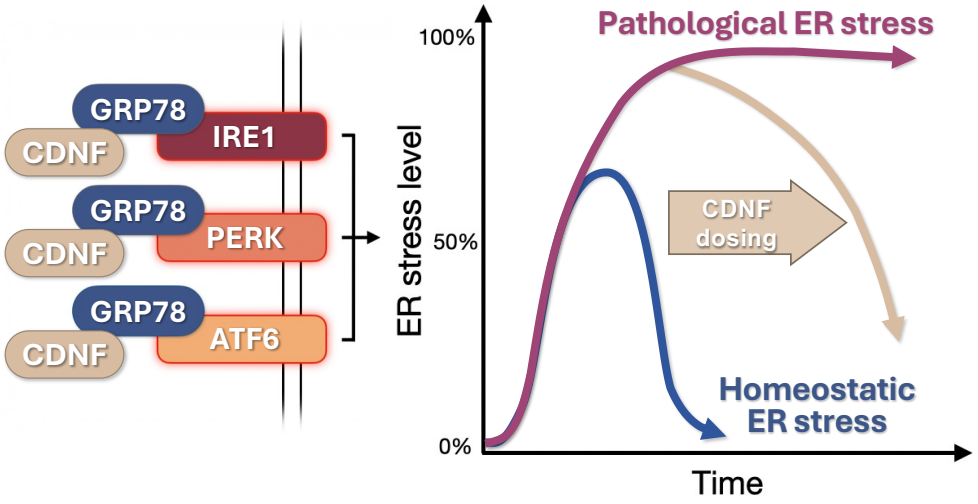
Selcted for €8M EU Horizon grant

Unique Mechanism of Action and Robust Preclinical Data

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

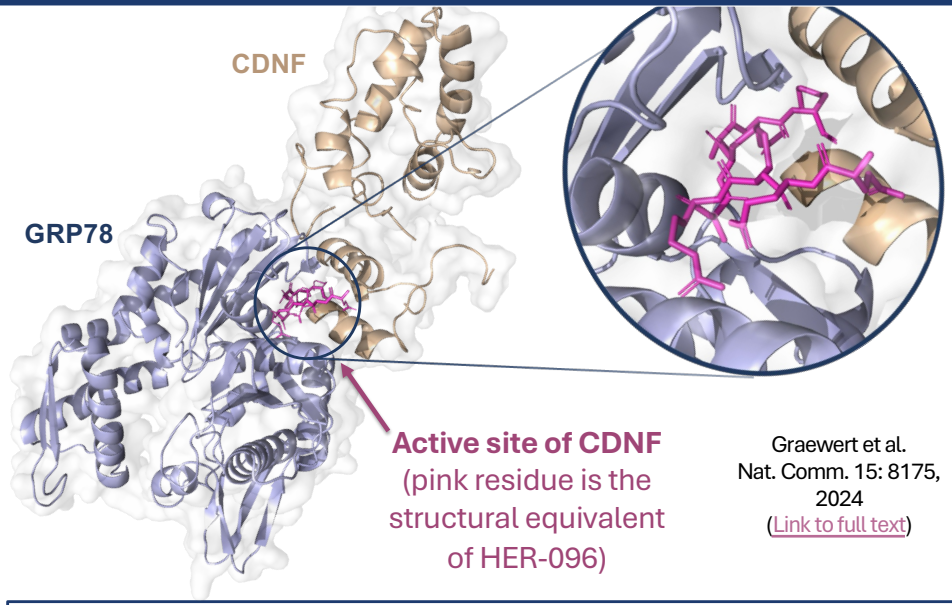
GRP78 Serves as the Master Regulator of the Unfolded Protein Response (UPR) Pathway

Unfolded Protein Response (UPR) Pathway Signaling and CDNF



The Unfolded Protein Response (UPR) is a central regulatory mechanism within the proteostasis network that senses endoplasmic reticulum (ER) protein misfolding and orchestrates adaptive responses to restore protein homeostasis. In normally functioning cells, the **UPR acts as a homeostatic mechanism**: it is activated in response to the accumulation of misfolded proteins in the ER and is downregulated once the imbalance is resolved. In contrast, **pathological ER stress—such as that observed in Parkinson's disease—is harmful and can ultimately lead to cell death. CDNF's physiological role is to modulate the UPR to prevent pathological (maladaptive) ER stress and restore the UPR to its normal homeostatic function.**

HER-096 is designed based on the CDNF / GRP78 binding interface



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

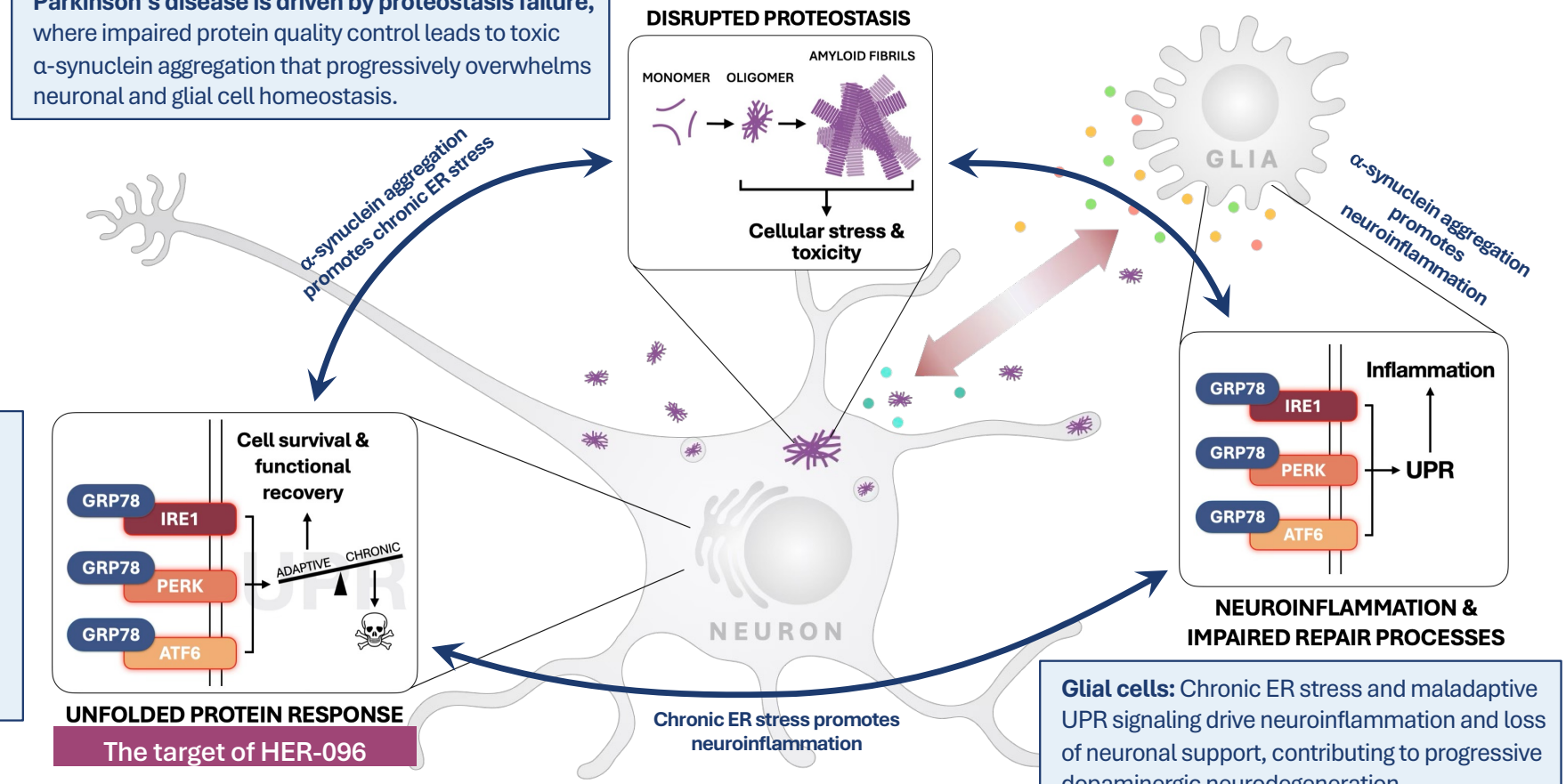
HER-096 mimics CDNF's effects on the UPR system but has significant additional benefits:

- Blood-brain barrier penetration allows subcutaneous administration
- Fully synthetic molecule; enhanced 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.

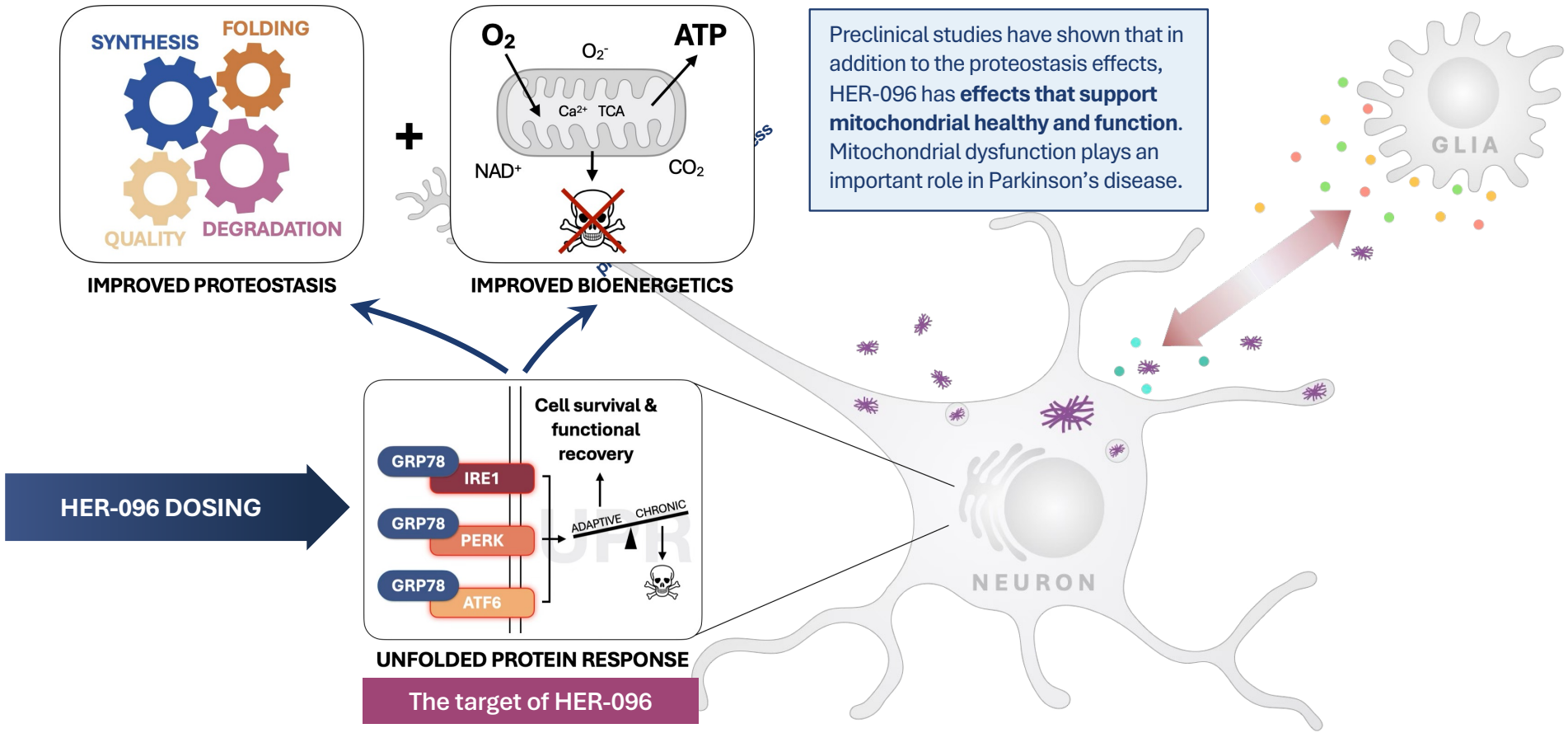


In Parkinson's disease, **unresolved ER stress locks the UPR into a chronic, maladaptive state** that suppresses neuronal maintenance, drives inflammation, and **accelerates dysfunction and degeneration of dopamine neurons**.

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

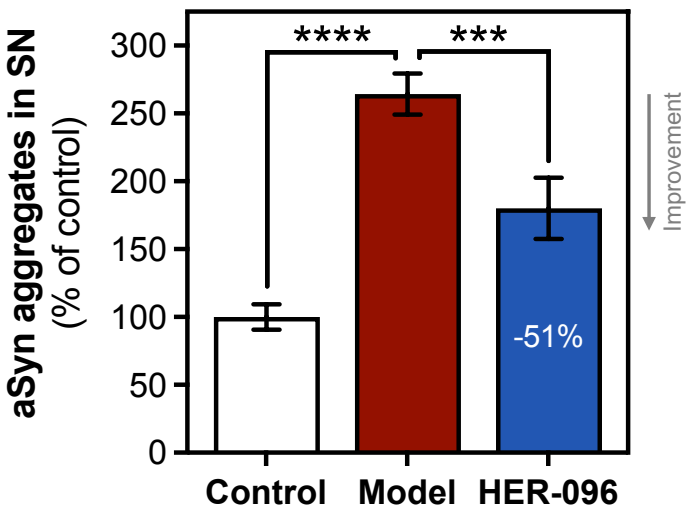
Restorative Effects on Neuronal Proteostasis and Mitochondrial Function



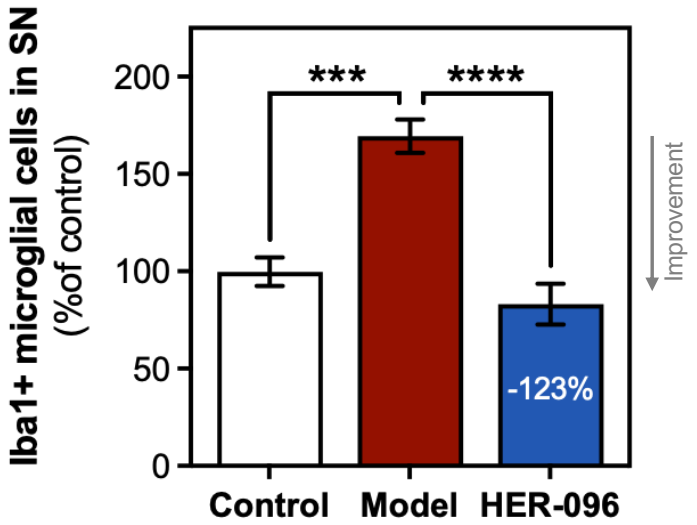
Disease modification – Effect on α-synuclein and Neuroinflammation

In vivo Preclinical Studies (Parkinson's mouse model)

α-SYNUCLEIN AGGREGATES



NEUROINFLAMMATION

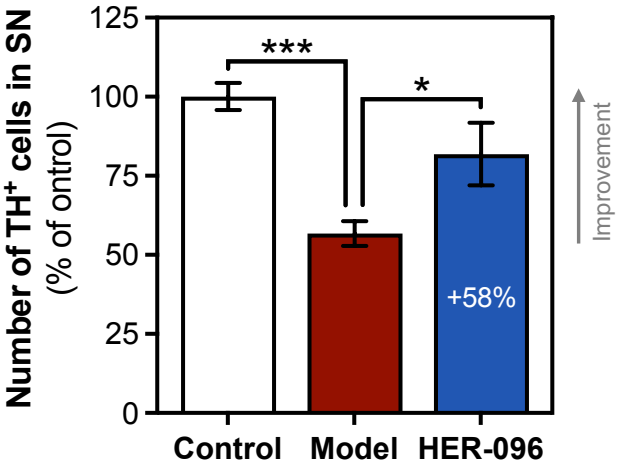


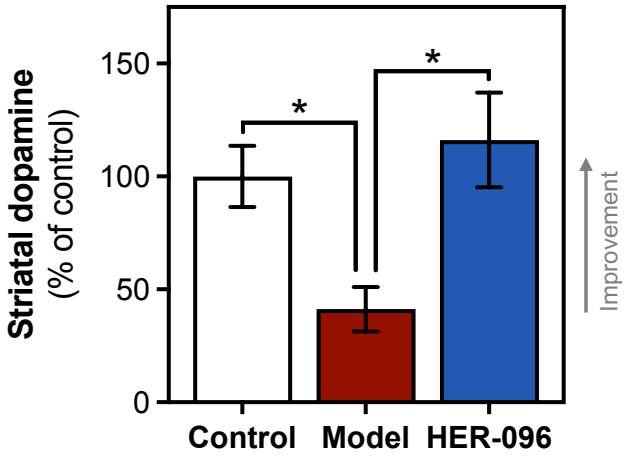
Mice were administered IP a human-equivalent dose of 200 mg HER-096 3 times per week for 4 weeks. Control = normal aged mouse without drug (vehicle only). PD model = PD model aged mouse without drug (vehicle only). HER-096 = PD model aged mouse with HER-096 drug. For substantia nigra (SN; graphs 1, 2, 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

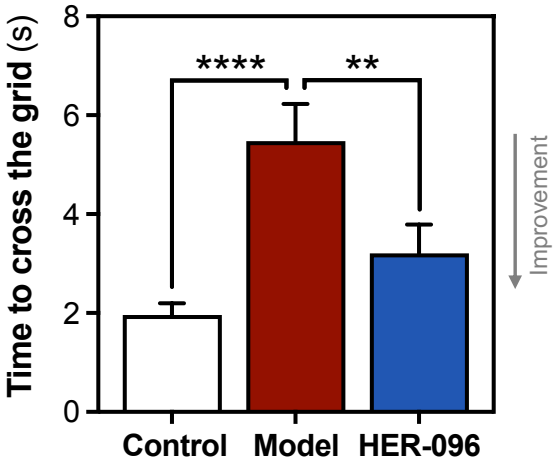
Disease modification – effect on dopamine system

In vivo Preclinical Studies (Parkinson's mouse model)

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







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HER-096 demonstrate robust neuroprotection

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

Phase 1 Program is Completed With Very Encouraging Data

HER-096 Phase 1 Program is completed

PHASE 1A STUDY

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

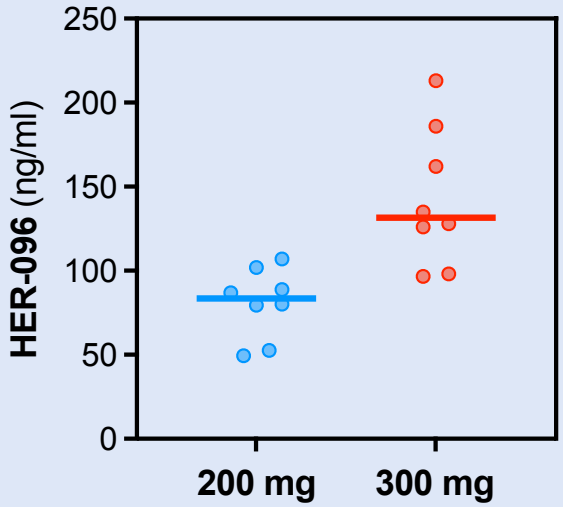
Clean safety profile

- 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

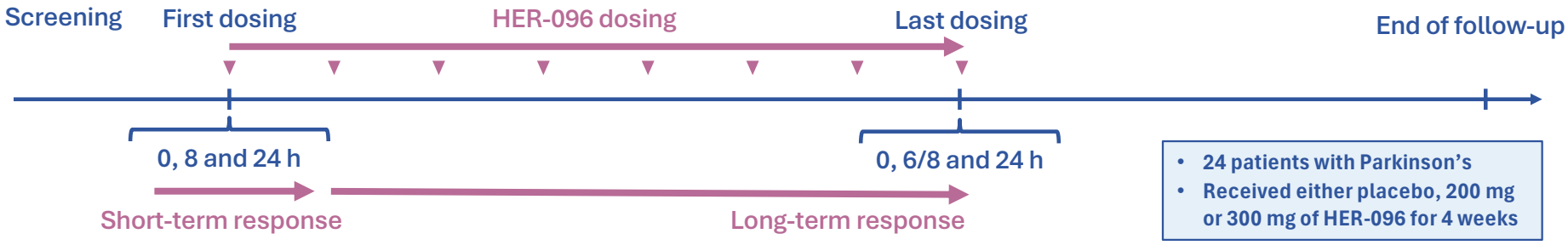
HER-096 in the CSF of Parkinson's patients



- 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

Biomarker Profiling with over 2.5 Million Data Points Generated

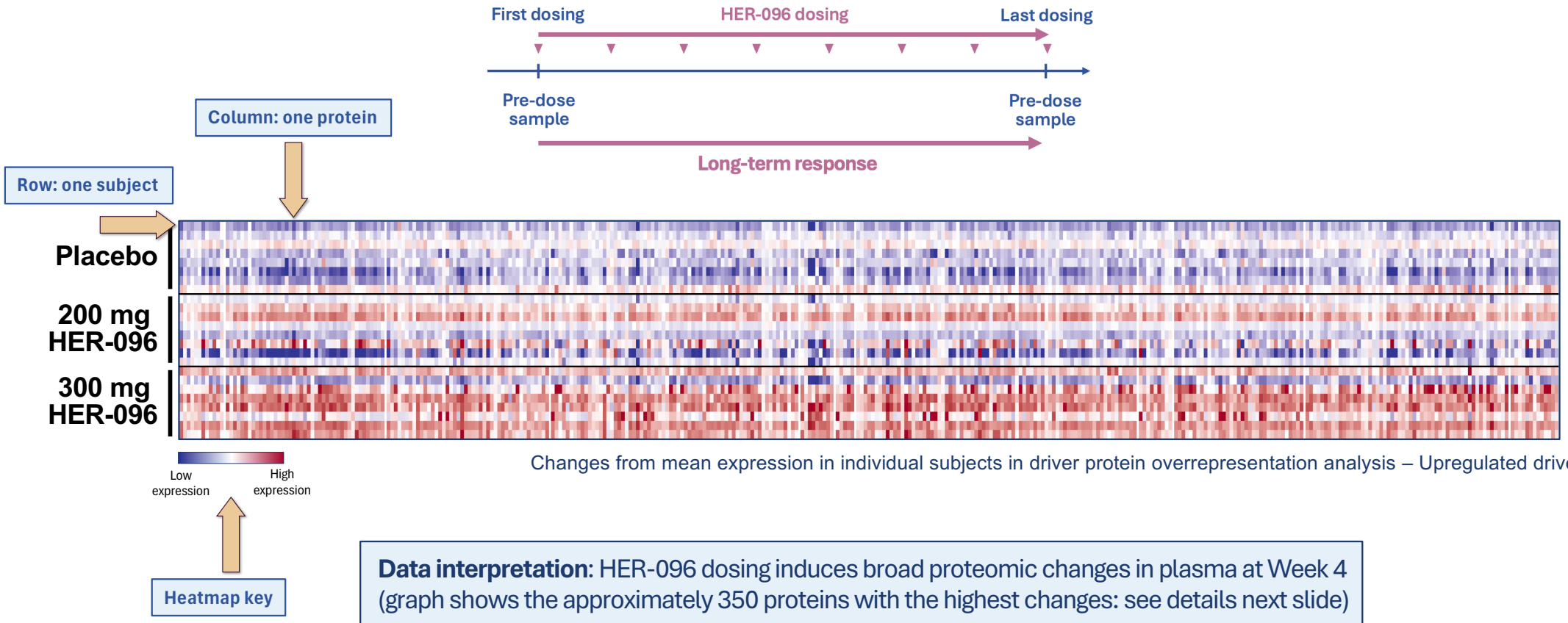
Exploratory Fluid Biomarker Analyses in the Phase 1b Clinical Study



		Analytes	Matrices	Timepoints	Subjects	Total
TARGETED	**Cytokines**	5	2	6 | 2	24	1 440
TARGETED	**NULISA**	100	2	5 | 2	24	9 600
TARGETED	**MitoDNA lesions**	1	1	2	24	96
TARGETED	**Mitochondrial metabolites**	6	1	2	24	288
UNTARGETED	**SomaLogic proteomics**	11 000	2	6 | 2	24	2 112 000
UNTARGETED	**Mass spec proteomics**	2 114	1	2	24	101 472
UNTARGETED	**NeuroSPARC (neuronal EVs)**	1	1	3	24	72
UNTARGETED	**EV mass spec proteomics**	2700	1	3	24	194 400
UNTARGETED	**Metabolomics**	1 400	2	2 | 2	24	134 400
	TOTAL					**2 553 768**

Example of Biomarker data: Plasma Proteomics – Systemic Biological Response to HER-096

Aptamer-based Proteomics Used for Longitudinal Monitoring of 11 000 Plasma Proteins



Changes from mean expression in individual subjects in driver protein overrepresentation analysis – Upregulated driver pr

Data interpretation: HER-096 dosing induces broad proteomic changes in plasma at Week 4 (graph shows the approximately 350 proteins with the highest changes: see details next slide)

somalogic

HER-096 Broadly Modulates Proteostasis-Regulating Proteins in Plasma

Aptamer-based Proteomics: Overrepresentation Analysis of HER-096 Effect vs Placebo Over 28 days

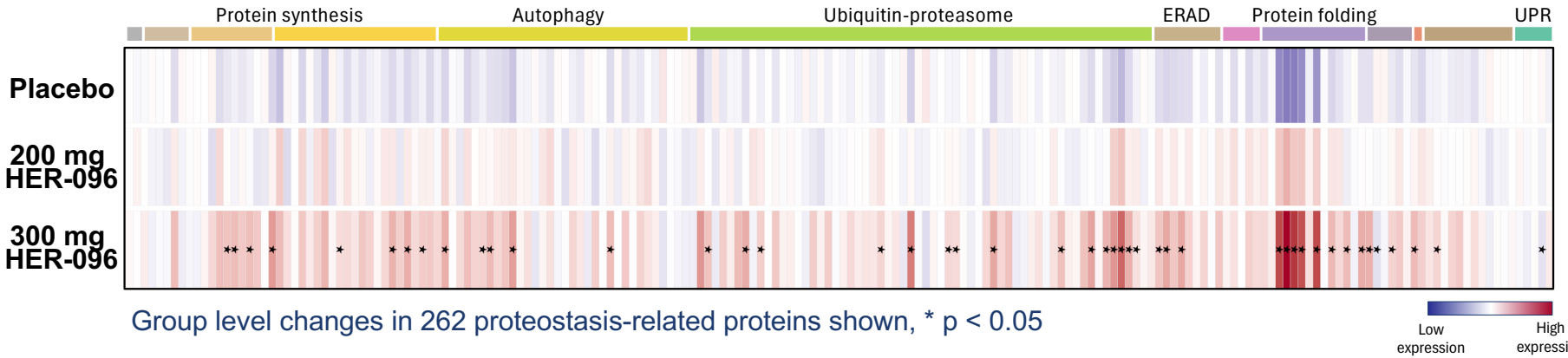
Overrepresentation Analysis (ORA): All HER-096 vs Placebo

Category	Number of Gene Ontology terms	Adjusted p-value
Proteostasis	18	0.0002
Endolysosomal trafficking	12	0.0000004
RNA processing	8	0.004
Autophagy	5	0.0004

Last dose vs first dose (pre-dose samples), comparison all subjects receiving HER-096 to placebo group, overrepresentation analysis → semantic Gene Ontology term clustering

Data interpretation: Plasma proteomics reveals broad and coordinated **modulation of multiple proteostasis domains** following HER-096 treatment, supporting biologically meaningful engagement of proteostasis pathways in line with the drug's proposed mechanism-of-action

Proteostasis: Group Level Comparison

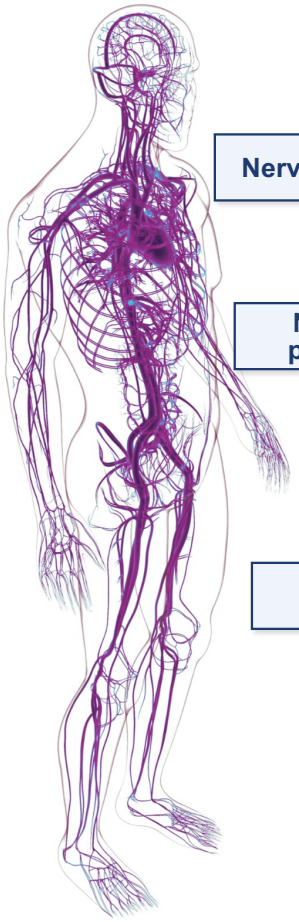


Group level changes in 262 proteostasis-related proteins shown, * p < 0.05

somalogic

Clear Biomarker Responses to HER-096 Dosing in Humans

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



Nervous system markers

Neuronal-enriched plasma EV markers

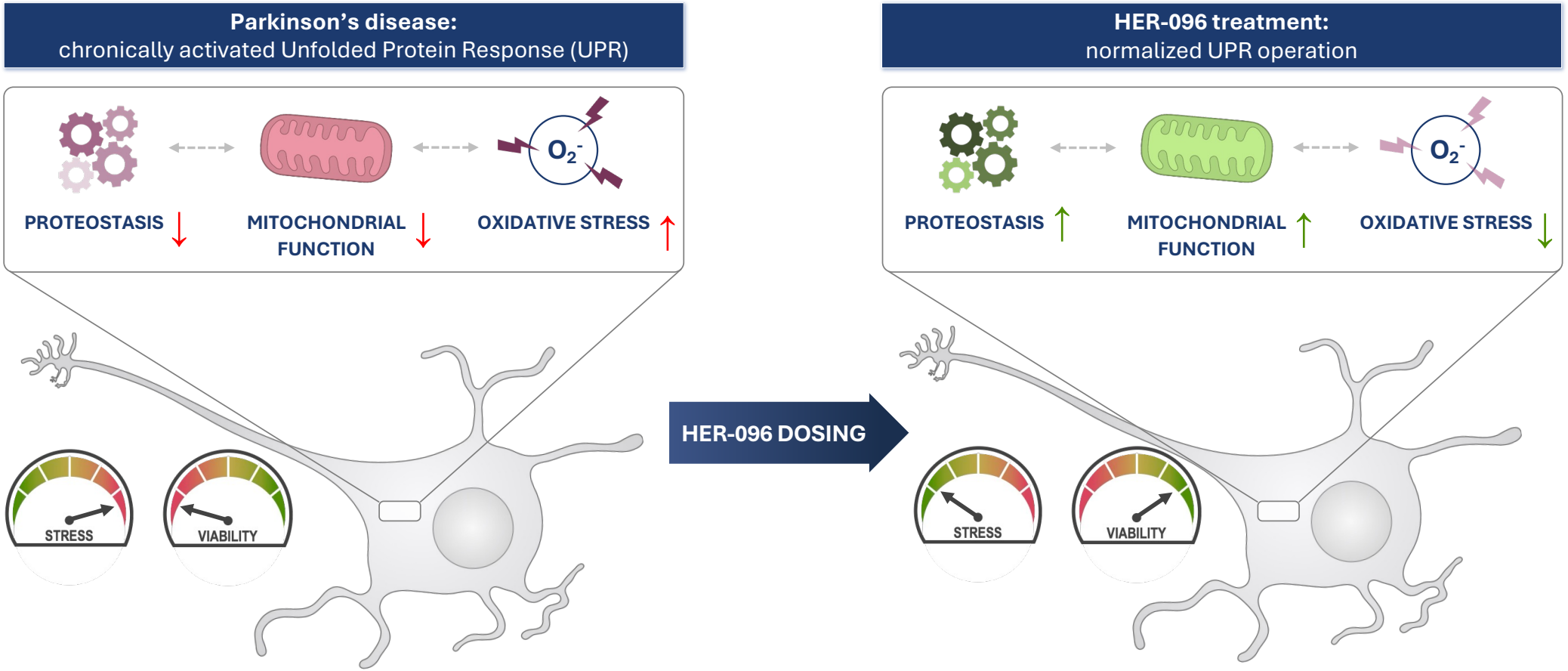
Systemic markers

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

- Proteostasis
- Inflammation
- Mitochondrial function & oxidative stress defense

HER-096 dosing: Clinical Biomarker Changes Aligned with Restoration of UPR Function

Consistent biomarker changes support normalization of UPR signaling in line with established biology



Phase 1b Biomarker Data: De-Risking the Path to Clinical Efficacy

- Proof of biology: clear evidence of biological response to HER-096 in Parkinson's patients
- A key development milestone demonstrating preclinical-to-clinical translatability
- A key catalyst for strategic partnering and investment discussions

	Preclinical data	Phase 1b (Parkinson's patients)
Safety and tolerability	✅ Demonstrated	✅ Demonstrated
Biological response (biomarkers)	✅ Demonstrated	✅ Demonstrated
Effects on target pathways	✅ Demonstrated	✅ Demonstrated
Effect on symptoms	✅ Demonstrated	To be evaluated in Phase 2

Next Step: A Phase 2a Signal-Finding Study

Digital Motor Score-driven Study Design for Maximal Sensitivity

- A **proof-of-concept study** aiming to demonstrate symptomatic improvement in early-stage Parkinson's patients using a sensitive *digital motor score* (DMS) as an endpoint
  - Smartphone-based active testing/monitoring
- Randomized, placebo-controlled study of 6 months, followed by a 6-month open-label extension
  - One dose level of HER-096, 1:1 randomization ratio
  - Two weekly s.c. injections
- Patients with early-stage idiopathic Parkinson's disease
- Study planned to start in early 2027 (pending financing), expected run-through time ~2 years
- EU Horizon 2025 funding: 8 million € grant-based funding to support the Phase 2a study (announced in Feb 2026)
  - Leading European clinical sites committed
- Total additional funding need ~25 million €

Strong External Validation and Financial Support for HER-096

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

+

THE MICHAEL J. FOX FOUNDATION
FOR PARKINSON'S RESEARCH

=

€3.6M research financing

Phase 1b clinical trial and biomarker development completed

Co-funded by the European Union

€ 2.5 million for biomarker development (completed)

€ 15 million investment commitment from EIC Fund
€4.2 million utilized (can invest maximum of 1/3 of total equity)

€ 8 million for the Phase 2 trial conduct
Herantis leading the consortium including leading clinical sites

- HER-096 is a potentially game-changing therapy that could become the first disease-modifying treatment for Parkinson’s disease
- Huge market opportunity: the PD therapeutic market is expected to grow to up to $13bn by 2034, with no disease-modifying therapies currently available and few in development
- Herantis is backed by 15+ years of research, with robust external validation and funding from the Michael J. Fox Foundation, Parkinson's UK, and the European Innovation Council
- Broad functionality of HER-096 opens wide therapeutic opportunities into other neurodegenerative disorders and beyond
- Next step:
  - Phase 2 efficacy study in PD

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