



# Partnering to Halt Parkinson's Disease

InnoMedica, Q1 2026

# InnoMedica is offering a unique Partnering Opportunity



Together, we change Parkinson's Disease therapy by making TALINEUREN the new standard-of-care

## 12 Million

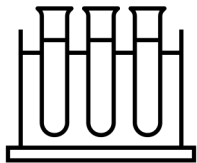
Patients

## 0

Approved disease-modifying  
therapies

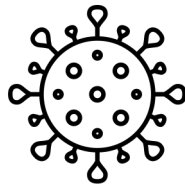
## 45 Billion

Yearly market potential in US alone



Together,  
we deliver **scientific  
breakthrough** to address  
unmet need in Parkinsons

Novel treatment approach  
with GM1



Together,  
we **engineer**  
the business case

Proprietary know-how and  
Strong IP with InnoMedicas  
liposomal Plattform



Together,  
we **co-develop**  
TALINEUREN in the  
clinics

PHASE II ahead



Together,  
we **commercialize**  
TALINEUREN worldwide

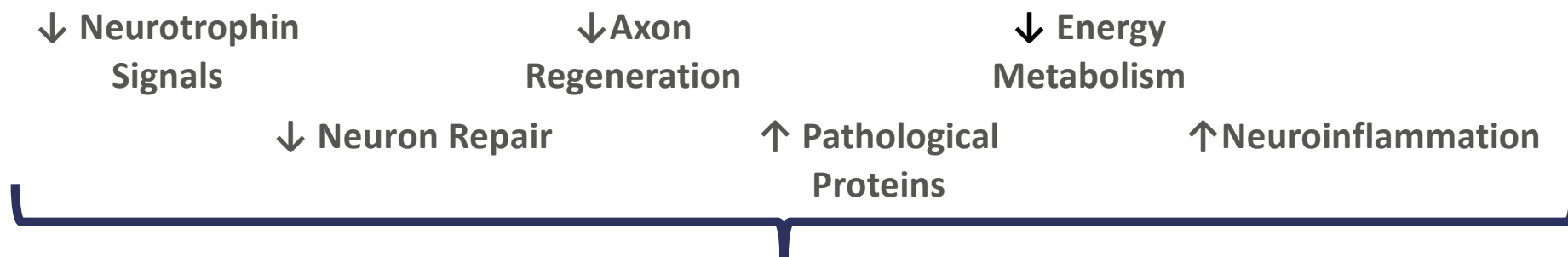
Huge potential for first  
in class therapy

# Parkinson's Disease and InnoMedica's USP



TALINEUREN is the first lipid only drug acting on all aspects of Parkinson's Disease at same time.

**The Challenge:** PD is a very broad disease associated with GM1 deficiency



**The solution** through InnoMedica's TLN:

A pleiotropic drug reinstalling a functional neuronal membrane biology through external supplementation of GM1

# TALINEUREN – Overview

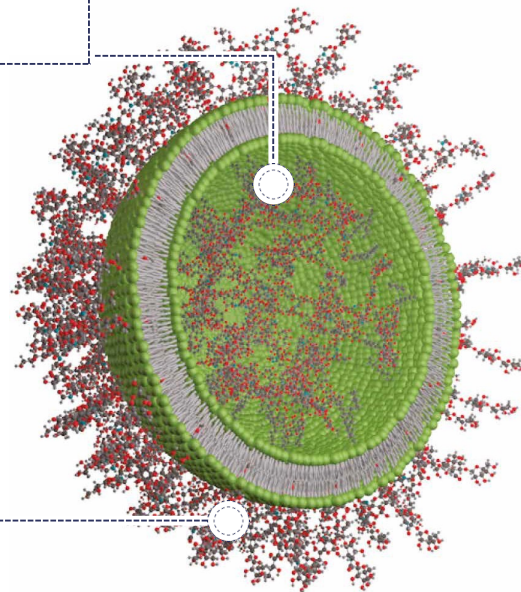


TALINEUREN is a biological nanoparticle and consists of 100 % body-own molecular building blocks.

## Nanostructure of Talineuren

### Transport Shell

The liposomal nanocarrier has a particle diameter of 35 nm.



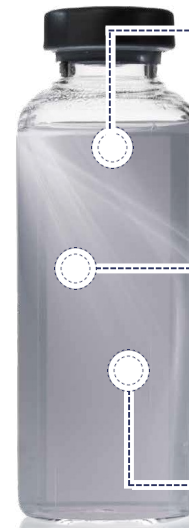
### Active Ingredient

The glycolipid GM1 is an integral part of the lipid envelope of the nanocarrier.

## Unique features of Talineuren

### Crosses the Blood-Brain Barrier

Talineuren transports GM1 to the affected nerve cells.



### High Safety

Long-known endogenous active ingredient with good tolerability

### Wide Potential

Application in various neurodegenerative diseases (e.g. ALS)

# Phase I/IIa Trial NEON



## Safety Evaluation of Intravenous TLN

### Trial Design

- Open-label, single arm, and single center interventional trial with TLN as add-on therapy to Levodopa.
- Weekly infusions of 720 mg GM1 over 180 weeks (11 patients) and 48 weeks (additional 10 patients)

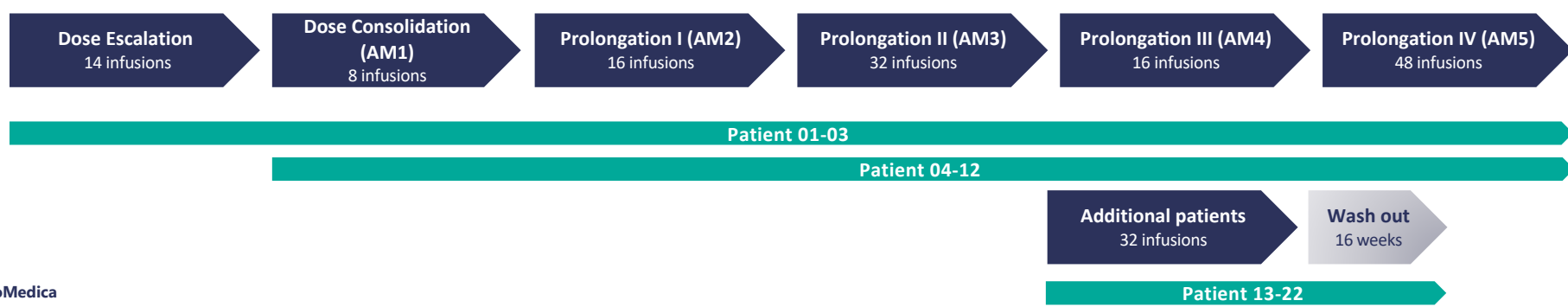
### Safety Evaluation

- Generally, well tolerated
- Mild (83%) to severe (1%) adverse reactions to infusions (fatigue and dizziness) and transient pseudo neutropenia in early infusions (habituation effect). Infusion reactions can be managed with slow administration speed as demonstrated in additional patients 13 to 22.

### Efficacy

- Over 1,600 infusions and collectively over 40 patient treatment years.
- 9 out of 11 patients showed improvement over baseline, of which 6 patients improved beyond what is considered a minimal clinically important difference. On average, the patients scored -7.5 points in total MDS-UPDRS "off".
- Subsequent treatment prevented disease progression over 3.5 years.

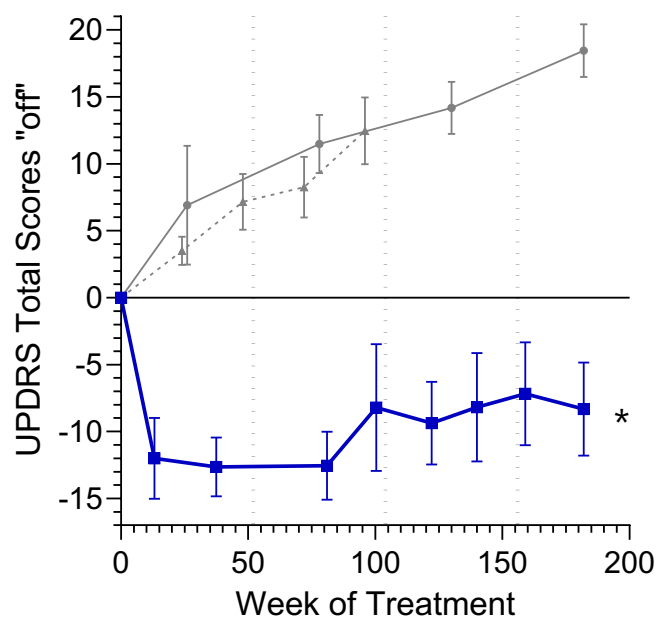
### Trial Design



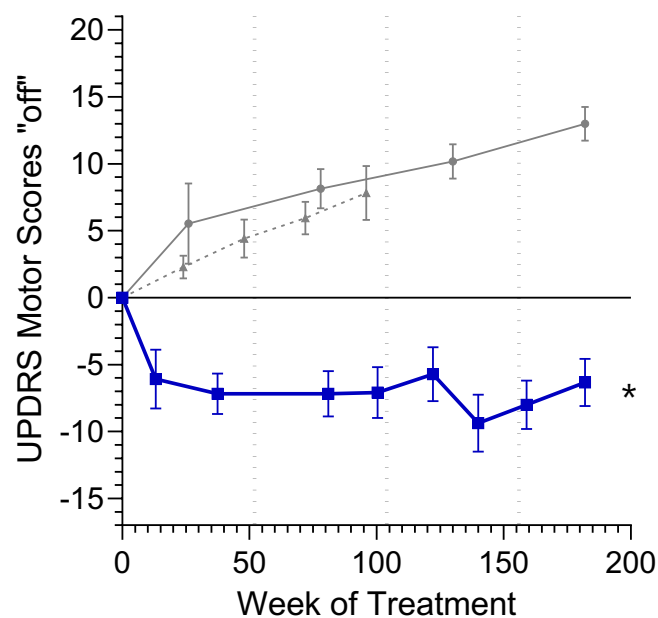
# NEON Phase I/IIa Clinical Data



UPDRS Total Score Change from Baseline



UPDRS Motor Score Change from Baseline (Part III)



## MDS-UPDRS Scores

- ❖ I – Non-motor experiences of daily living
- ❖ II – motor experiences of daily living
- ❖ **III – motor examination**
  - ❖ III “off” – off standard L- dopa treatment 12 h prior to motor examination
- ❖ IV – motor complications
- ❖ **Total Score:** sum of I, II “best”, III “off”, IV
- ❖ Error bars = SEM

\* 3 patients only for this point

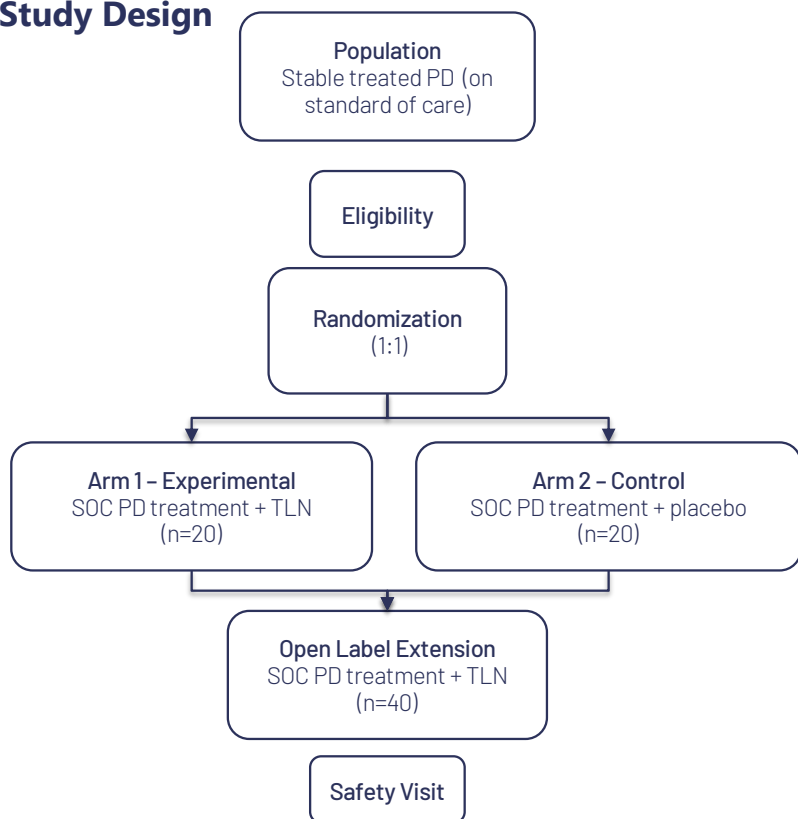
- Talineuren
- Early-stage PD PPMI cohort (Simuni et al. 2018)
- ▲ Control patients (Schneider et al. 2013)

# Phase I/IIa Clinical Trial LIBRA



Randomized, placebo-controlled, double-blinded, multi-center pilot trial

## Study Design



## Aim:

- Estimates of the short-term Efficacy of Talineuren and Placebo in patients with Parkinson's Disease

## Primary objective:

- MDS-UPDRS part III score in the defined "off" medication state

## Secondary objective:

- MDS-UPDRS (other)
- Parkinson medication (LEDD)
- Assessments of quality of life and mental condition
- Safety
- Biomarkers

## Treatment per patient:

- 24 infusion double blind + 24 infusions open label

## Study Timelines:

- Expected start in first Q4 of 2026
- Study duration 2 years

# Biomarkers in LIBRA



A total of 17 disease- and treatment-relevant biomarkers will be investigated in the LIBRA trial and provide deep biological insight into how Talineuren affects Parkinson's at its core.

Family of biomarkers in the context of PD	Biomarker	Sample	Changes in this biomarker have been clinically associated with:
<b><math>\alpha</math>-synuclein species</b>	total $\alpha$ -synuclein	plasma	cognitive decline (Lin, Yang et al. 2017)
<b>Inflammatory mediators</b>	Selected cytokines (TNF- $\alpha$ , IL1- $\beta$ , IL-2, IL-6, IL-10, IFN- $\gamma$ )	serum	motor progression and lower cognitive status (Williams-Gray, Wijeyekoon et al. 2016, Rathnayake, Chang and Udagama 2019)
	C-reactive protein (CRP)	serum	reduced life prognosis (Sawada, Oeda et al. 2015)
	Neutrophil-to-lymphocyte ratio	whole blood	indicative of overall inflammatory status (Munoz-Delgado, Macias-Garcia et al. 2021)
<b>Mitochondrial dysfunction, iron homeostasis and oxidative stress</b>	Ferritin	serum	low antioxidants alter ROS/RNS production and dysregulate iron homeostasis, contribute to alterations observed in the pathophysiology of PD (neurodegeneration) (Medeiros, Schumacher-Schuh et al. 2016)
	Iron	serum	
	Transferrin	serum	
	NOx	serum	
	Thiobarbituric acid reactive substances (TBARS)	serum	
<b>Axonal damage</b>	Advanced oxidation protein products (AOPP)	plasma	motor impairment and cognitive decline (Ye, Locascio et al. 2021) (Niemann, Lezius et al. 2021) (Ygland Rodstrom, Mattsson-Carlgren et al. 2022)
	Neurofilament (NfL)	serum	
<b>Neurotrophins</b>	Brain-derived neuronal factor (BDNF)	serum	cognitive impairment, depression, and restless legs syndrome (Azman and Zakaria 2022)



## **Mode of Action**

TALINEUREN: A pleiotropic drug against neurodegenerative diseases

# The Effects of GM1 Homeostasis on the Human Body



TALINEUREN enhances the positive pleiotropic effects of GM1 through augmented Pharmacokinetics in GM1 deficient PD

	Neurotrophin Signaling Pathways	Neuronal Repair and Axonal Regeneration	Protein Aggregation	Energy Metabolism	Neuroinflammation
Normal Physiology	Neurotrophins NGF, BDNF, GDNF → Trk receptors functional → neuron survival, differentiation, plasticity	Intact receptor activation, signal amplification, and cytoskeletal coordination → repair damaged neurites, extend new axonal branches, maintain synaptic connectivity	α-Synuclein associated to membrane modulates neurotransmitter trafficking and maintenance of synaptic function. Protein aggregation is efficiently controlled.	The high energy need of neurons is supported by a well-coordinated energy production in mitochondria	Balanced signaling between neurons, astrocytes, and microglia maintains immune homeostasis → low levels of pro-inflammatory mediators (TNF-α, IL-1β, IL-6) → Efficient clearance of cellular debris without chronic inflammation
PD Pathology	Reduced GM1 levels → Destabilized Trk receptors organization → dysfunctional signaling → neuronal degeneration	Reduced GM1 levels → Loss of capacity for functional repair → reduction in neurite outgrowth, and axonal sprouting; progressive synaptic loss → dopaminergic circuit failure	Reduced GM1 levels → α-Synuclein self-aggregates into fibrils. → aggregates accumulate → synaptic dysfunction, axonal degeneration, neuronal death	Reduced GM1 levels → lowered mitochondrial biogenesis and impaired mitochondrial maintenance. Also dead mitochondria accumulation due to lysosomal dysfunction. Also impaired electron transport chain, decreased Ca <sup>2+</sup> signaling, increased ROS generation and ATP depletion through α-Synuclein aggregates	Reduced GM1 levels → chronic microglial activation triggered by α-Synuclein aggregates → Release of pro-inflammatory cytokines and chemokines → Increased reactive oxygen and nitrogen species damaging neurons → Activated astrocytes amplify inflammation and sustain neurodegenerative feedback loops
TLN: Externally administered GM1	Potentially restored Trk organization → potentially restored neurotrophin signaling → <b>Potential neuroprotection</b>	Potentially stabilized lipid microdomains, restored protein-lipid interactions, improved receptor clustering → Potential partial restoration of repair capacities → <b>Potential dopaminergic circuit recovery</b>	Potentially restored membrane environment → potential stabilization of membrane-bound, soluble α-Synuclein form → <b>potentially improved mitochondria function, reduced oxidative stress, improved autophagic activity</b>	Potentially restored cellular energy homeostasis → <b>improved mitochondrial performance and resilience against stress supports neuronal maintenance, signaling, and repair</b>	Potentially reduced microglial activation → reduced inflammation and restored immune homeostasis in the central nervous system → <b>potential neuroprotection</b>

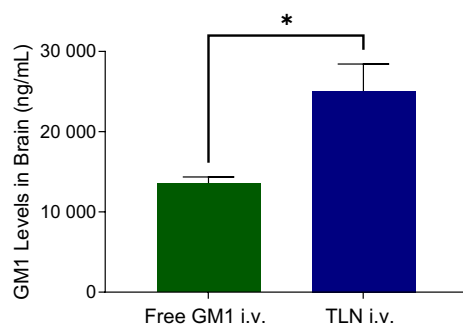
# TALINEUREN – Transport of GM1 across the BBB



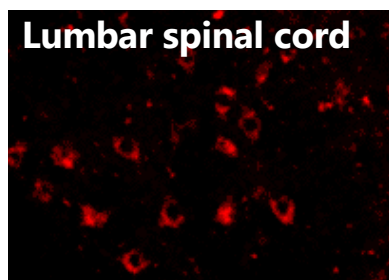
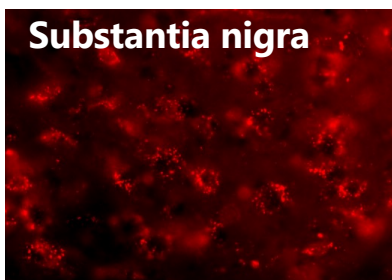
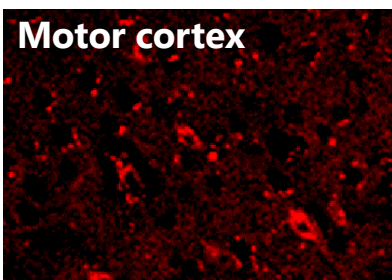
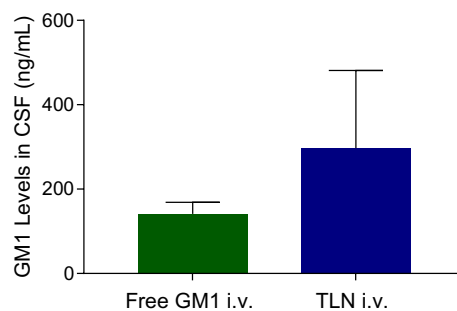
TALINEUREN significantly enhances the transport of GM1 across the blood-brain barrier after oral and intravenous application

## TLN applied intravenously

### GM1 in Brain (Mouse)



### GM1 in Cerebrospinal Fluid (Mouse)

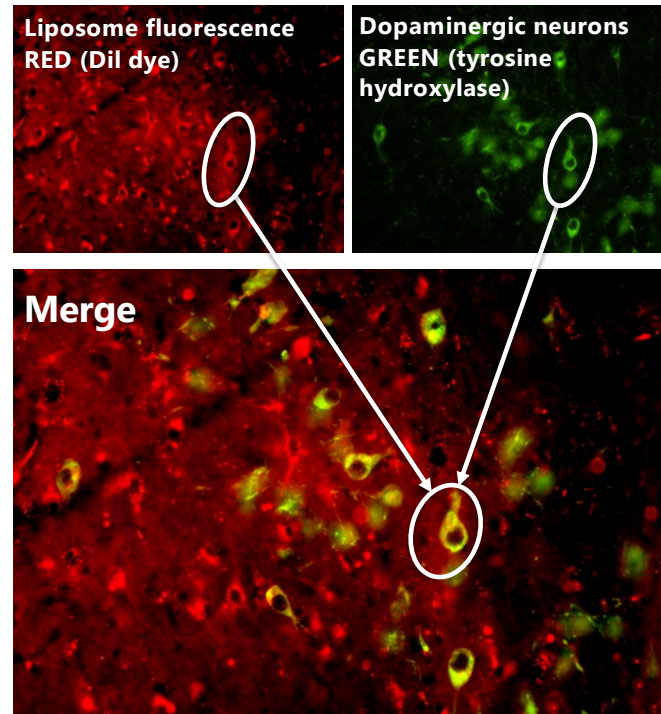


## TLN applied orally

Liposome fluorescence  
RED (Dil dye)

Dopaminergic neurons  
GREEN (tyrosine  
hydroxylase)

Merge



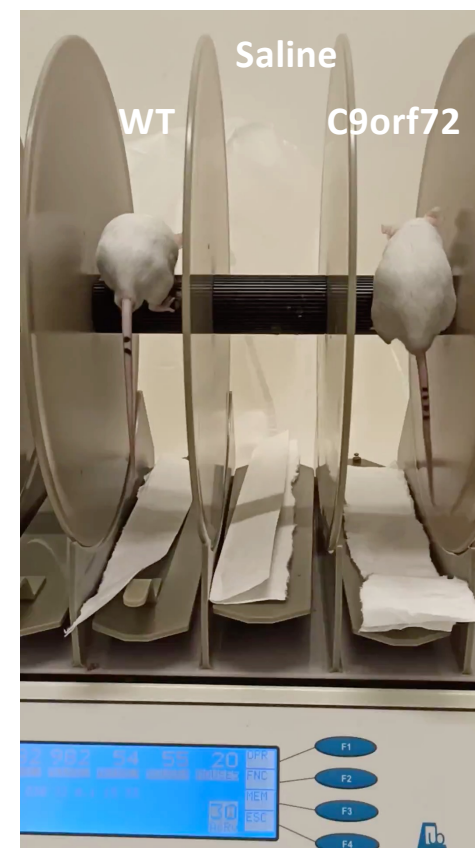
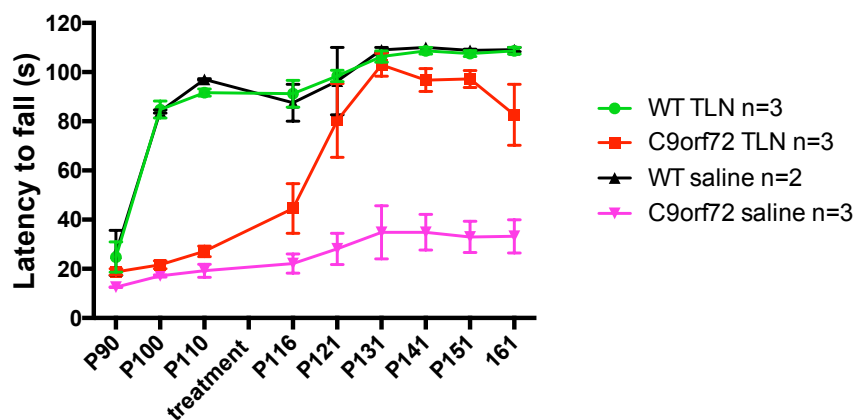


# Treating Amyotrophic Lateral Sclerosis

TALINEUREN with Orphan Drug Designation EMA/FDA

# TALINEUREN: Promise as Broad Drug – ALS

Preclinical data show positive effects of TLN in ALS models





# IP, Pipeline, Management

Talineuren: First-in-Class, Clinical-Stage

# TALINEUREN is Covered by 2 Utility Patents



**Liposomes Comprising Sphingomyelin (Priority December 2017 -> Running until December 2038)**

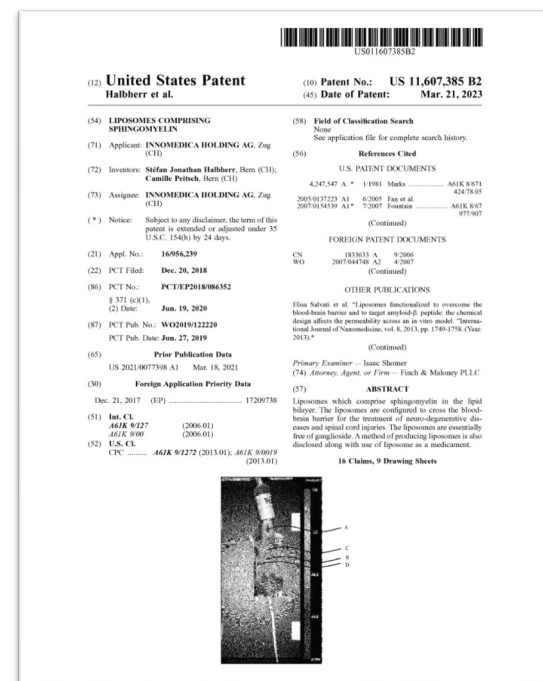
Approved in Europe, Japan, Switzerland, United Kingdom, U.S.A. Pending in other geographies.

The invention relates to liposomes. The liposomes comprise sphingomyelin in the lipid bilayer. The liposomes are configured to cross the blood-brain barrier for the treatment of **neurodegenerative diseases and spinal cord injuries**. The liposomes are essentially free of ganglioside. The invention also relates to a method of producing liposomes and to the use of liposome as medicament.

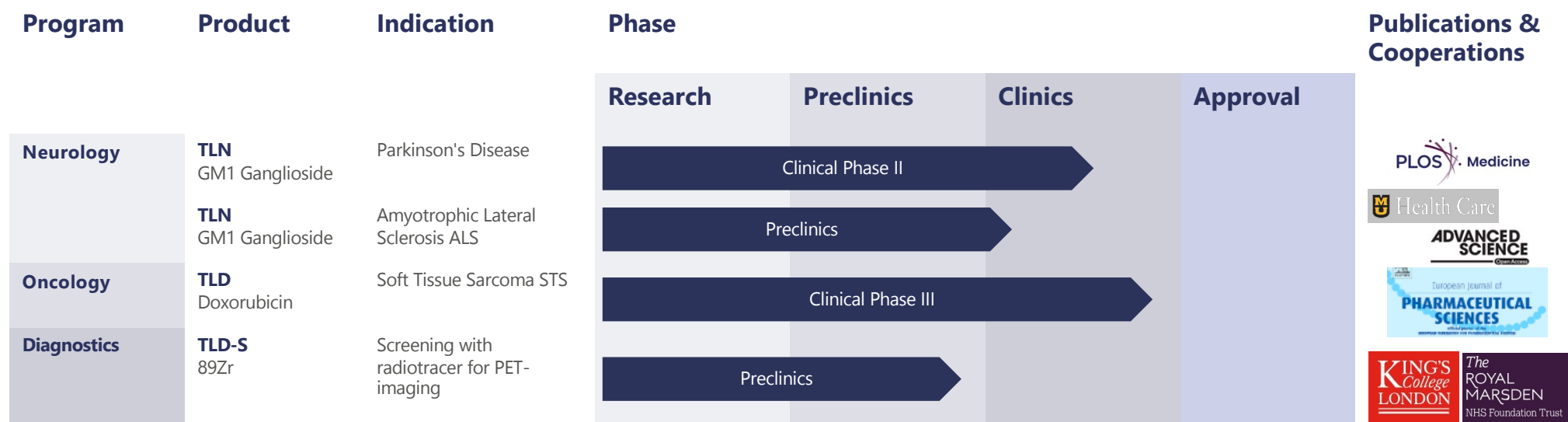
**Talineuren: Liposomal composition for use in a method of treating Parkinson's disease (Priority January 2023 -> Running until January 2044)**

Patent pending.

The present invention relates to a liposomal composition for use in a method of treating **Parkinson's disease**. The liposomal composition comprises sphingomyelin in a lipid bilayer and a therapeutically effective amount of monosialotetrahexosyl-ganglioside (GM1), wherein a therapeutically effective dose of said liposomal composition is administered at most every 4 days in a primary mode of administration with at least 3 days between each administration; preferably at most every 6 days in a primary mode of administration with at least 5 days between each administration; most preferably at most every 7 days in a primary mode of administration with at least 6 days between each administration.



# InnoMedica offers a Highly Attractive Pipeline Portfolio



# InnoMedica – GMP-Grade NanoFactory in Switzerland



TALIDOX is produced in the NanoFactory — InnoMedica's proprietary manufacturing platform for growth and quality

- **Independence, flexibility, scalability** through own GMP production in Marly
- **Proprietary** lipid nanoparticle production technology
- **Full control** over quality, costs, and timelines
- For **clinical and commercial quantities**; exponentially scalable
- **Strategic advantage** for approvals and partnerships

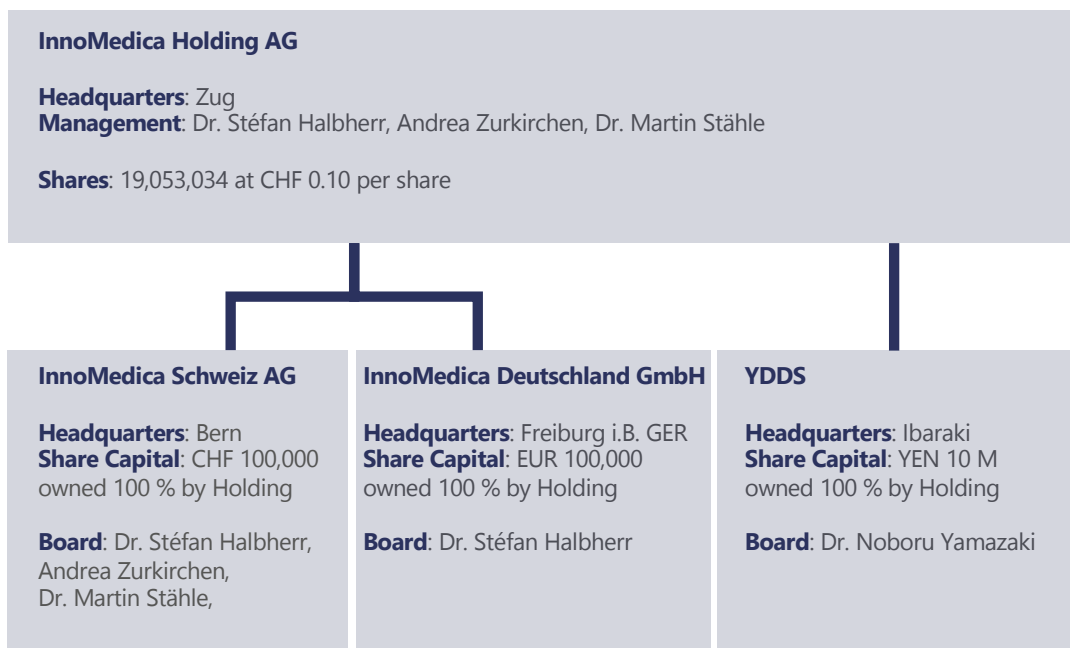


# InnoMedica – Corporate Structure



Supported by a broad shareholder base with a strong shareholder pool: CHF 87 million raised to date

## Corporate Structure



## Executive Board of InnoMedica Holding AG

**CEO**



Co-Founder  
**Stéfan Halbherr**  
 Dr. rer. nat.  
 Universität Bern

**Head Investor Relations, Legal, Staff**



Co-Founder  
**Andrea Zurkirchen**  
 lic. phil.  
 Universität Zürich

**CFO**



With InnoMedica for 7 years  
**Martin Stähle**  
 Dr. oec.  
 Universität Hohenheim

### Well-coordinated team

- Clear division of roles and strong mutual trust
- Leadership continuity through long-standing experience within InnoMedica

# InnoMedica – Board of Directors



	<b>Christian Mauriand</b>	<b>Dr. Leila Nobs</b>	<b>Dr. Denis Bron</b>
<b>Prof. Dr. Patrick Hunziker</b>	<b>Prof. Dr. Urs Wälchli</b>	<b>Pascal Brenneisen</b>	
Deputy Head of Intensive Care Medicine, University Hospital Basel	Global Head Corporate Development Roivant	<b>President</b> Professor of Corporate Finance (Simon Business School, London Business School, Université de Neuchâtel, Rochester-Bern Executive Programs)	<b>Vice-President</b> Chief Physician Swiss Air Force
President of the International Society for Nanomedicine	Sale of assets worth over USD 11 billion (incl. acquisition of Telavant by Roche)	Scientific Director at TRB Chemedica International SA, Geneva Pharmacist	Neurological research (Harvard Medical School, Boston)
	Entrepreneur, board member, advisor	Serial entrepreneur in pharma and med-tech industry	

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