CerebroLab™

CerebroLab is an AI-driven research platform and patient resource dedicated to advancing the treatment of neurodegenerative diseases.

Clarity,
Not Confusion.

Alzheimer's, Amyotrophic Lateral Sclerosis (ALS), Multiple Sclerosis, and Parkinson's are considered diagnoses of exclusion. No single test, blood marker, or imaging scan exists to definitively diagnose them. There are no cures for any of them.

The disease pathologies are linked to misfolded protein aggregates. The etiology of protein aggregates are frustratingly opaque, often described as toxic invaders or the result of sporadic mutations. It creates more questions than answers.

Clinical treatment of neurodegenerative diseases has not kept pace with advances in basic and translational research. This is largely because therapeutic development has remained predominantly brain-centric. Pharmaceutical strategies have traditionally emphasized reductionist approaches aimed at discrete molecular targets such as receptors, enzymes, or ion channels to achieve specific pharmacodynamic effects.

The 2012 discovery of the glymphatic system has provided a new framework to understand brain health. It shows that neurodegenerative disease is rarely just a neuron problem, but rather a failure of a larger, neurovascular-glymphatic unit.

A New Strategy —
The Biomechanical Model

The human brain can be modeled as a self-regulating, biomechanical system analogous to a graphics processing unit (GPU). In this framework, the brain functions as the central processing architecture, while the vascular system provides the essential power supply, delivering oxygen and glucose required for sustained neuronal metabolism. The glymphatic system operates as the thermal regulation and waste-clearance network, maintaining ionic balance and preventing the accumulation of neurotoxic metabolites.

When vascular perfusion is compromised, neuronal energy metabolism declines, leading to reduced synaptic efficiency and impaired cognitive output.

Likewise, dysfunction of the glymphatic system diminishes metabolic clearance, resulting in neuroinflammation and cellular stress which is comparable to thermal overload in an electronic processor.

Collectively, these failures reduce the brain's computational throughput, manifesting as cognitive impairment and disrupted neuronal signaling or innervation. These symptoms are the hallmarks of neurodegeneration in Alzheimer's, ALS, Multiple Sclerosis, Parkinson's, and Dementia.

Conversely, when the neurovascular-glymphatic system functions properly, the brain maintains homeostasis.

In this model, neurodegeneration can be defined as the delta between optimized, homeostatic brain health and dysregulation.

CerebroLab is a forum to showcase the research talent leading advancement in neurodegenerative disease.

By generating a real time feedback loop between patients, researchers, and clinicians, innovation can accelerate in a dynamic ecosystem.

→ **Innovative research connecting with patients.**

→ **Refined through iterative testing.**

→ **Translating into medical breakthroughs.**

The Science & Methodology Behind the Platform

The 5 Pillar Framework is CerebroLab's integrative model for understanding how interconnected dysfunction across anatomical, vascular, immune, and metabolic systems drives neurodegeneration, and help determine where intervention can make a difference.

Homeostatic Brain Health —
5 Pillars of Neurodegeneration Centered on Pathophysiological Dysfunction

Dysregulation of the brain's biomechanical system is a complex, multifactorial pathogenesis. Homeostatic modification and optimization necessitates an integrative therapeutic paradigm. Effective interventions require coordinated modulation across molecular, cellular, and musculoskeletal (MSK) levels.

This framework identifies five core, interconnected pathophysiological pillars through which homeostatic dysfunction contributes to neurodegenerative pathogenesis. It integrates anatomical, mechanical, vascular, immune, and metabolic consequences.

Clinical Relevance —
The CerebroLab Framework is a Model for:

- **UNDERSTANDING IDIOPATHIC OR ATYPICAL NEURODEGENERATION WITH BRAINSTEM FEATURES**
- **EVALUATING CHRONIC SUBCLINICAL MUSCULOSKELETAL DYSFUNCTION IN EARLY-STAGE NEURODEGENERATION**
- **INFORMING THERAPEUTIC STRATEGIES TARGETING ALIGNMENT, DRAINAGE, AND INFLAMMATION**

Research Links —
The Academic Foundation for the Framework

01 GLYMPHATIC / DETOXIFICATION FAILURE

Impaired cerebrospinal fluid (CSF)–interstitial fluid exchange leads to accumulation of neurotoxic proteins and metabolites.

- Loss of astrocytic aquaporin-4 (AQP4) polarization disrupts directional glymphatic flow and accelerates amyloid-β accumulation (Simon et al., 2022).
- Meningeal lymphatic–glymphatic coupling is essential for CNS waste clearance; disruption worsens protein aggregation (Louveau et al., 2017).
- Glymphatic dysfunction precedes tau pathology, indicating clearance failure is an early event in Alzheimer's disease progression (Yuying Jiao et al.2025,).
- Arterial stiffness, reduced pulsatility, and venous outflow obstruction mechanically impair glymphatic influx and efflux (Benveniste & Nedergaard, 2021).

02 CEREBROVASCULAR DYSFUNCTION (NVU, BBB, GLYCOCALYX)

Breakdown of the neurovascular unit (NVU) impairs cerebral perfusion, blood–brain barrier (BBB) integrity, and nutrient delivery.

- NVU dysfunction and BBB breakdown occur prior to cognitive decline in Alzheimer's disease, suggesting vascular injury precedes amyloid deposition (Sweeney et al., 2019).
- Endothelial dysfunction and loss of the endothelial glycocalyx reduce nitric oxide bioavailability, increase permeability, and promote neuroinflammation (Zlokovic, 2011).
- Vertebrobasilar insufficiency and jugular venous congestion lead to chronic cortical–brainstem hypoxia and impaired waste clearance. (Benjamin et al., 2025)
- Endothelial senescence and inflammatory SASP signaling have been demonstrated in ALS vasculature (Jingyuan Ya et al, 2024).

03 BIOMECHANICAL DYSFUNCTION

Craniocervical instability and mechanical strain disrupt brainstem signaling, CSF dynamics, venous drainage, and autonomic regulation.

- Craniocervical junction instability and brainstem compression alter CSF pulsatility and neural conduction (Michael F. Flanagan, 2015).
- Mechanical compromise affects cranial nerves IX–XII, contributing to dysautonomia, dysphagia, respiratory instability, and postural fatigue (Ross Hauser, et al., 2025)
- Loss of vagal tone impairs anti-inflammatory signaling and gut–brain communication (Mlaak Rob, et al., 2025).
- Functional neuroimaging demonstrates early brainstem connectivity abnormalities in Parkinson's and Alzheimer's disease (Arianna Sala, et al., 2017).

04 NEUROINFLAMMATION / IMMUNE DYSFUNCTION

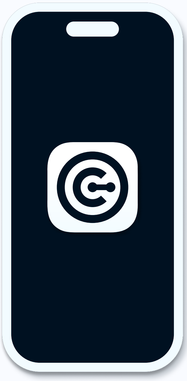
Chronic innate immune activation drives synaptic toxicity, neuronal loss, and propagation of neurodegeneration.

- Mechanical and vascular microtrauma prime microglia toward exaggerated inflammatory responses. (Norden et al., 2014)
- BBB disruption exposes CNS antigens to peripheral immunity, sustaining chronic inflammation. (Huang et al., 2020)
- Disease-associated microglia (DAM), MHC-II+, and interferon-reactive phenotypes correlate with distinct neurodegenerative stages (Yi-Hsuan Cheng, et al., 2025).
- Persistent TLR4 and NLRP3 inflammasome activation promotes neurotoxicity and disease progression (Junling Yang, et al., 2020).
- Viral immune priming, including HERV-K activation, accelerates inflammatory cascades in ALS and related disorders (Paul Dembny, et al., 2019).

05 BIOENERGETIC / METABOLIC COLLAPSE

Mitochondrial failure and oxidative stress reduce neuronal resilience and amplify vulnerability to upstream insults.

- Impaired glucose and lipid metabolism leads to synaptic energy failure and neuronal dysfunction (Pratishtha Chatterjee et al., 2020)
- Deficient NFE2L2 (NRF2) nuclear translocation weakens antioxidant defenses across AD, PD, and ALS (Emilia Zgorzynska et al., 2021; Elisa Navarro et al., 2024).
- Excess reactive oxygen species (ROS) damage mitochondrial DNA, proteins, and membranes (Eui-Hwan Choi et al., 2024).
- GLP-1 receptor agonists reduce amyloid processing, enhance antioxidant enzymes (MnSOD), and improve mitochondrial function in preclinical models (Ayush Gandhi et al.,2025.: Niklas Reich et al., 2022).



CerebroLab™

The CerebroLab App is a research search engine and clinical management tool for neurodegenerative Disease patients, caregivers, and clinicians.

PLEASE VISIT:
WWW.CEREBROLAB.COM
TO GET STARTED

App Features —
Integrative Tools for Neurodegenerative Care

Comprehensive resources for research and clinical management.

→ **AI-DRIVEN RESEARCH ENGINE**	An AI-driven research engine that is sourced, cited, and grounded in peer-reviewed medical literature.
→ **PATIENT TRACKER**	The App provides a comprehensive Patient Tracker with management tools for clinicians and patients.
→ **PROTOCOL MANAGEMENT**	Manage medications, supplements, peptides, and dietary protocols with ease.
→ **CARE TEAM COORDINATION**	Stay connected with your physicians and specialists in one place.

How the App Works —
From Tracking to Transformation

A simple three-step process to help optimize your brain health:

01
Track →

Easily log your daily activities, sleep patterns, and cognitive exercises.

02
Analyze →

The app analyzes your data to identify patterns and personalized insights.

03
Optimize →

Receive tailored recommendations to help enhance your brain health journey.

How the App Works —
Five Neurophysiologic Domains

The CerebroLab app organizes neurophysiologic domains into five interacting axes. Each domain is framed as a measurable physiologic system known to interact with neurodegenerative vulnerability pathways.

01	**SLEEP** →	**RESTORATION**
02	**CEREBROVASCULAR** →	**METABOLISM**
03	**BIOMECHANICS** →	**STRUCTURE**
04	**NEUROINFLAMMATION** →	**IMMUNE**
05	**BIOENERGETICS** →	**INNERVATION**

Privacy & Data Policy —
Your Data Stays with You

Your health data is deeply personal. We built Cerebrolab with privacy at its core, ensuring your information remains anonymous, secure and under your control.

No data sold to third parties.

Anonymized data stored securely.

Full data export available.

About CerebroLab

Neurodegenerative disease represents a vast, urgent, and inefficiently served translational challenge — one that is far more addressable than current clinical pathways reflect.

Though it affects millions, there is currently a wide chasm between research and clinical implementation.

CerebroLab can close this gap by creating an efficient ecosystem where innovative research can connect with patients who seek them, be refined through iterative testing, and ultimately translate into medical breakthroughs.

Our Story —
Why We Created CerebroLab

Eight years ago, I was diagnosed with ALS and given three to five years to live. I discovered that there had been no meaningful clinical advances in ALS treatment since Lou Gehrig's death in 1941. My options were palliative care or experimental trials. Within a year I was in a wheelchair.

Facing a disease with more questions than answers, I had to decide how to spend the time I had left.

I chose to learn. I rejected experimental therapies and instead spent years meeting with specialists across neurology, vascular biology, immunology, endocrinology, orthopedics, and regenerative medicine.

What emerged from those conversations was a striking disconnect: the basic science had advanced enormously, but clinical treatment hadn't kept pace. Therapeutic development remained stubbornly narrow - focused on single molecular targets rather than the complex, multisystem nature of neurodegenerative disease.

The foundational step on this educational journey was meeting Dr. Rudy Tanzi who directs Alzheimer's research at Harvard/Massachusetts General Hospital. Dr. Tanzi first shared the concept of brain biomechanics. He introduced me to the pioneering research of Dr. Jonathan Kipnis and Dr. Maiken Nedergaard. Their collective work showed that neurodegenerative disease is rarely just a neuron problem, but rather a failure of a larger, neurovascular-glymphatic unit.

CerebroLab is what we built from that realization. It's a platform designed to bring that fragmented research together - and to give patients, clinicians, and researchers the integrative tools the field has been missing.

If progress is to be made in the treatment of neurodegenerative disease, it will be built on what Dr. Tanzi, Dr. Kipnis and Dr. Nedergaard have started. We hope that CerebroLab can serve as a forum for that advancement.

The Founding Team —

→ **Jon Steingart** ALS Patient	Jon Steingart was diagnosed with ALS in 2018. Prior to that, he worked in entertainment. Theater: Ars Nova (Co-Founder), Film: Black Dynamite (Producer), Animation: Ultra Super Pictures (Partner).
→ **Kristi Clay** Clinical Researcher	Kristi Clay is a Clinical Researcher specializing in complex diagnoses.
→ **Caroline Helm** Research Assistant	Caroline Helm is a student at The Brearley School. In 2025 she worked as a research intern for the Harvard/Massachusetts General Hospital Institute for Neurodegenerative Disease.

If you are a Neurodegenerative Disease Patient, Clinician, or Researcher and would like to learn more about CerebroLab, please contact us or download the Mobile App to get started.

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www.cerebrolab.com