

Investigating Biophoton Generators for Improved Brain Function

Scientific rationale, preliminary clinical observations, and a validation roadmap informed by the 2026 Nobel-recognized optogenetics research

Prepared for Dr. James Z. Liu, MD, PhD

President, First Institute of All Medicines
Chairman, Brain Healing Inc.
CEO, Tesla BioHealing Inc.

5 October 2026 | Version 1.0

Purpose: assess the scientific rationale and evidence for investigating generator-associated improvements in brain function, and define the measurements needed to establish causality.

Executive summary

Biophoton generators have been investigated as a noncontact approach to supporting brain-related function. Publications supplied for this paper report changes in cognition, motor performance, sleep, pain, and selected electrophysiological measures following exposure. These findings motivate a defined research program, while their interpretation depends on study design, measurement validity, reproducible device characterization, and independent replication. [8–20]

The 2026 Nobel Prize in Physiology or Medicine recognizes Karl Deisseroth, Peter Hegemann, and Georg Nagel for discoveries concerning light-gated ion channels and optogenetics. The relevant scientific contribution is a demonstrated means of converting light into controlled neuronal activity through introduced light-sensitive proteins. It establishes a mechanistic precedent for optical modulation of neural circuits. It does not establish efficacy, biological equivalence, or endorsement of biophoton generators. [1–3]

This white paper advances a testable proposition: if a generator delivers an independently verified physical exposure that changes a relevant biological process, and that process mediates a meaningful functional improvement under controlled conditions, the device may have a reproducible role in supporting brain function. Candidate pathways include cellular metabolism, vascular physiology, and indirect effects through sleep or pain. None is assumed to be established for the generators described here.

Central research proposition: verified exposure → measured biological response → reproducible functional benefit. Each connection must be tested. Functional improvement, neural repair, and disease modification require different evidence.

Scope and evidence approach

This is a narrative scientific white paper based on 13 supplied device-related publications and selected primary studies in optogenetics, endogenous ultraweak photon emission, and photobiomodulation. It is not a systematic review or a reanalysis of participant-level data. The clinical reports are summarized as reported; registration, raw outcomes, device output, and regulatory status were not independently audited. Findings from a shared trial or selected subset should not be counted as independent replication.

The intended audience is clinical investigators, research partners, and technical reviewers. The proposed experiments and development sequence are recommendations of this paper, not findings already demonstrated by the devices.

1. What the Nobel research contributes

On 5 October 2026, the Nobel Assembly announced the award to Deisseroth, Hegemann, and Nagel “for their discoveries concerning light-gated ion channels and optogenetics.” The discovery of channelrhodopsins and their adaptation for neural experiments created a way to perturb selected cells and test their contribution to behavior. [1–3]

Nagel and colleagues demonstrated that channelrhodopsin-2 is a directly light-gated cation channel. Boyden and colleagues then combined gene delivery with optical stimulation to control mammalian neuronal spiking on a millisecond timescale. The identified molecular target, defined exposure, and measured electrical response make this a causal experimental mechanism. [2,3]

Kravitz and colleagues used optogenetics to distinguish the effects of direct and indirect basal ganglia pathways in mice. Activation of the direct pathway relieved selected motor deficits in a Parkinsonian model, whereas activation of the indirect pathway suppressed movement. This shows why identifying the affected cell population matters: different circuit effects can produce opposing outcomes. It does not demonstrate recovery of lost dopamine neurons or validate a whole-body generator exposure. [4]

Three related fields with different mechanisms

Field	Physical–biological relationship	Evidence boundary
Optogenetics [2–4]	Delivered light acts on introduced opsins and changes neuronal ion flow.	Demonstrated circuit control under specified experimental conditions.
Endogenous photon emission [5,6]	Biological reactions produce ultraweak light that can be detected.	Emission is established; a normal human neural information channel remains unproven.

Field	Physical–biological relationship	Evidence boundary
Photobiomodulation [7]	Defined external illumination is studied for physiological effects without opsin gene delivery.	Selected metabolic and hemodynamic effects have been measured; device equivalence requires testing.
Biophoton generators [8–20]	Reported proximity exposure is proposed to influence cellular or systemic physiology.	The delivered exposure, mechanism, and clinical benefits need reproducible characterization.

The useful connection is methodological: establish what absorbs or otherwise responds to the exposure, quantify that response, and determine whether it explains functional change. The Nobel recognition strengthens the rationale for this type of investigation; conclusions about a particular generator must come from its own evidence.

2. Photons, metabolism, and brain physiology

Endogenous biophotons as physiological signals

Kobayashi and colleagues detected spontaneous ultraweak photon emission from rat brains and reported associations with EEG activity, cerebral blood flow, and oxygen conditions. Brain-slice experiments linked emission to mitochondrial respiratory reactions and reactive oxygen production. Endogenous emission can therefore provide information about metabolism or oxidative dysfunction. Greater emission is not inherently evidence of better function: respiratory-chain inhibition also increased emission in their experiments. [5]

Tang and Dai imaged glutamate-induced photon activity in mouse brain slices and proposed transmission along neural pathways. Their experiments used high extracellular glutamate concentrations of 25–75 mM; the authors acknowledged questions about physiological relevance and possible injury effects. These observations support investigation of optical phenomena in neural tissue, but do not establish a normal human biophoton communication system or an explanation for consciousness. [6]

External photobiomodulation as a mechanistic starting point

Pruitt and colleagues studied 15 younger and five older healthy adults using sham-controlled 1,064-nm forehead illumination. Broadband near-infrared spectroscopy showed increases in oxygenated hemoglobin and oxidized cytochrome c oxidase relative to sham. The calibrated source delivered 0.25 W/cm² during an approximately eight-minute intervention. These are measured physiological signals, rather than evidence of disease reversal or direct measurement of intracellular ATP. [7]

The study offers an example of linking optical exposure with biological measurements. Its dose and delivery conditions cannot be transferred to an uncharacterized generator. The 1,064-nm wavelength also lies outside the 500–1,000-nm range described in some supplied generator papers. Longer exposure alone does not demonstrate biological equivalence to a different irradiance or spectrum. [7,12,17]

Terminology needed for reproducibility

In this paper, “endogenous biophotons” means ultraweak photons emitted by biological systems. “Biophoton generator” refers to the device class described in the supplied publications. Its name does not establish the origin, coherence, spectrum, or biological effects of its output. “Brain function” means defined outcomes such as task performance, cognition, mobility, or electrophysiological responses; it is not a synonym for neuron regeneration.

Working hypothesis: a reproducible generator exposure could influence a measurable biological process relevant to brain performance. The exposure, target, direction of response, and clinical consequence remain questions for testing.

3. A testable framework for functional improvement

The following pathways are proposed research hypotheses for the generators. The same clinical outcome could arise through more than one pathway. Studies should distinguish a direct effect on neural tissue from peripheral or behavioral effects that subsequently alter performance.

Candidate pathway	Proposed relationship	Measurements that could test it
Cellular metabolism	A verified exposure influences cellular photoacceptors or another identified target, changing respiration or redox signaling and subsequently neuronal activity.	Matched active/sham cell experiments; oxygen consumption, ATP, mitochondrial membrane potential, oxidative stress, and neuronal electrophysiology.
Vascular physiology	An exposure-associated vascular response changes tissue perfusion or oxygen availability, potentially affecting functional performance.	Validated perfusion methods, oxygenation measurements, vascular reactivity, and appropriately standardized blood rheology.
Sleep, pain, and autonomic state	Changes in sleep continuity, pain burden, or autonomic state improve attention, energy, participation, or motor performance indirectly.	Actigraphy or sleep studies, validated pain instruments, autonomic measures, and mediation analyses.
Neural network activity	A biological response is accompanied by reproducible changes in neural dynamics and task processing.	Prespecified EEG/ERP measures paired with blinded clinical and behavioral assessments.

A stronger causal model requires temporal ordering: the exposure must precede a biological change, and the biological change should precede or predict the functional outcome. A graded exposure response, an appropriate blocking experiment, or replication across laboratories would strengthen the inference. If improvements occur without the proposed physiological change, the model must be revised.

For example, shorter ERP latency could be consistent with faster processing during a task. It does not identify the initiating molecular target. Improved sleep could also affect task performance and EEG state. Measuring these alternative explanations allows the research to determine whether they mediate, confound, or do not explain the observed benefit.

Potential utility should be defined by outcome and population. A reproducible reduction in fatigue or improvement in attention may be valuable even when there is no evidence that the underlying neurodegenerative process has changed. Disease modification requires a separate longitudinal hypothesis, suitable endpoints, and adequate follow-up.

Decision rule: reproducible biological effects support mechanism development; reproducible clinically meaningful active–sham differences support an efficacy conclusion for the tested population and protocol. Neither result should be generalized automatically to other disorders.

4. Preliminary clinical evidence: cognition, movement, and stroked

The supplied reports address several outcomes relevant to brain function. Their contribution is strongest when the design includes a concurrent randomized comparator, complete reporting, validated endpoints, and a prespecified analysis. Selected EEG cases and uncontrolled before–after observations contribute exploratory information.

Program	Design and reported observations	Scientific interpretation
Alzheimer's disease [8,10, 23]	A multimodal report describes 12 four-week completers and changes in questionnaires and physiological measures. A separate EEG article presents four selected cases from a reported 24-person randomized trial.	Potential functional and electrophysiological signals. Selected cases do not provide an unbiased randomized estimate for the full cohort. The reports do not establish reversal of Alzheimer's pathology.
Parkinson's disease [11,12, 22]	The clinical pilot reports 42 participants across home and center settings, with 23 home completers and 14 of 19 center participants analyzed. Selected EEG cases describe changes in rhythms, ERP latency, and task measures.	Clinical scales and electrophysiology provide outcomes for further testing. The open-label design and incomplete follow-up limit causal inference; neuronal regeneration was not demonstrated.
Chronic stroke [14,15]	The main report describes 46 randomized participants, with 26 active and 20 placebo recipients during the first two weeks, followed by crossover. It reports clinical-scale improvements. A separate EEG article presents five selected cases.	The initial concurrent randomized phase provides the strongest design in this set. Crossover and selected-case results need separate analysis. Participant accounting and between-group effect estimates require reconciliation.

Why clinical and EEG outcomes should be paired

Clinical assessments address whether a participant functions better. EEG and ERP address selected aspects of electrophysiology. Concordance can strengthen a hypothesis, but an EEG change alone does not prove repair, improved cognition in daily life, or a specific molecular mechanism. Frequency shifts should be interpreted against prespecified directions and relevant baselines rather than treating every change as normalization.

Reporting issues that affect the strength of the evidence

The stroke abstract reports 20 placebo participants, while its demographic table lists 17 controls and 26 active participants. The distinction may reflect attrition or an analysis population, but it is not adequately resolved in the presented accounting. The early randomized comparison should report all randomized participants, missingness, adjusted between-group differences, confidence intervals, and the planned primary endpoint. [14]

Several publications share registration identifiers or draw selected cases from broader trials. The reports should be linked through a cohort map showing enrollment, selection criteria, exposure periods, and outcomes. Multiple papers from one trial offer different analyses, but do not establish independent replication. [8,10,11,14,15]

5. Additional observations and their evidential limits

Evidence area	Reported observations	What remains to be established
Traumatic brain injury [17]	The abstract describes 12 retired NFL athletes reporting changes in sleep, cognition, pain, and well-being, plus a selected controlled EEG case.	The methods describe three retired male athletes and an additional female EEG case; cohort membership and denominators need clarification. Objective randomized functional outcomes are required.
ALS [19]	Three correspondence-based cases describe perceived changes in energy, sleep, bulbar function, or stability.	The report explicitly treats the observations as hypothesis-generating and states that causality and efficacy cannot be determined. Standardized longitudinal outcomes were not collected.
Glaucoma [18]	One human and one canine case describe changes in visual experience or visual behavior.	Standardized visual acuity, visual fields, structural imaging, and electrophysiology are absent. These observations do not demonstrate optic nerve regeneration.
Blood microscopy [9,13,16]	Single-patient time courses describe changes in sampled red cell appearance, aggregation, and plasma clarity.	Microscopy does not directly establish blood viscosity, cerebral perfusion, oxygen delivery, oxidative stress, or inflammatory protein levels. These need validated quantitative tests.
Pain [20]	A pooled report describes 194 participants across seven studies and an arthritis placebo comparison.	The arthritis narrative reports 56 participants, but the displayed comparison includes 22 controls and 24 active participants. Attrition, cohort overlap, and analysis populations need clarification.

The pooled pain report states that the number of participants with moderate or severe pain decreased from 87 to 21. The corresponding 76% reduction refers to the number in that symptom category; it should not be represented as a 76% reduction in each participant's pain intensity. Pain improvement could contribute to better sleep, mobility, and attention, but does not identify a direct brain mechanism. [20]

An independent optogenetic report described partial visual recovery in a person with retinitis pigmentosa following retinal gene delivery and use of stimulating goggles. That intervention involved an introduced light-sensitive protein and a different disease process. It cannot be used as evidence that generator exposure regenerates glaucomatous optic nerves. [21]

These observations identify domains for future research and possible patient priorities. The interpretation must remain specific to the measurements obtained, avoiding an inference from subjective benefit to structural repair or slowed disease progression.

6. The physical exposure and measurement gap

The supplied papers describe passive proximity use, commonly with four generators around a sleeping area, and report a 500–1,000-nm spectrum in some protocols. Descriptions also include relative emission strength, photon counts, or proprietary energy units. These do not provide a complete, independently reproducible dose at the participant or a demonstrated dose at neural tissue. [9,12,14,17–19]

Required quantity	Recommended reporting	Purpose
Spectrum	Calibrated spectral output, wavelength bands, detector response, and uncertainty.	Identify what is emitted and what biological absorption mechanisms could be relevant.
Output and geometry	Photon flux or irradiance with area/time units; distance, orientation, shielding, and exposure map.	Determine actual exposure at the participant's location.
Background and sham	Dark counts, ambient light, thermal conditions, active–sham differences, and repeatability.	Separate device-associated signals from measurement artifacts or environment.
Target exposure	Absorbed or modeled dose, transmission measurements, tissue geometry, and model uncertainty.	Test whether a direct neural optical mechanism is physically plausible.
Source stability	Output over time, device-to-device variation, calibration, and source energy characterization.	Support reproducibility and explain sustained emission.

For a defined wavelength, photon energy is $E = hc/\lambda$. Irradiance is optical power per unit area, commonly W/cm^2 ; radiant exposure is the time integral of irradiance, commonly J/cm^2 . A photon count requires its sampling time, illuminated area, wavelength information, and calibration to be interpreted as dose. If output is below instrument sensitivity, the appropriate result is a documented detection limit rather than a substituted biological claim.

The stroke report describes “longitudinal” or “scalar” photons, metal penetration, and torsional field properties. These are not established properties of freely propagating optical photons. They require independent physical experiments and exclusion of instrumental artifacts. If a reproducible non-optical

exposure is identified, its mechanism should be investigated on its own terms rather than inferred from optical neuroscience. [14]

Likewise, optical coherence is a measurable statistical property. EEG coherence and fingertip gas-discharge images do not establish coherence of generator photons. Bio-Well and 3D non-linear scanning outputs in the supplied reports should not be treated as validated measurements of brain ATP, cerebral recovery, or neuron regeneration without independent validation against appropriate reference methods. [8,14]

7. A staged validation program

The following sequence is proposed to reduce uncertainty before expanding disease-specific claims. Progression should depend on results rather than an assumed favorable outcome.

Stage	Core work	Evidence needed to advance
1. Independent characterization	Use calibrated detectors and randomized device coding to measure spectrum, exposure geometry, background, active–sham separation, stability, and uncertainty.	A reproducible physical difference between active and sham devices with defined exposure units.
2. Biological target testing	Expose relevant cell systems under matched conditions. Include sham, environmental controls, and a characterized conventional light comparator when appropriate. Measure both beneficial and adverse cellular responses.	A reproducible exposure-dependent response and a plausible target; pathway perturbation or blocking where feasible.
3. Mechanistic human pilot	Randomize participants to active/sham exposure with blinded assessment. Pair a clinical outcome with selected physiological measures and objective adherence monitoring.	Feasible blinding, measurable exposure, reliable endpoints, and an effect estimate sufficient to plan the next study.
4. Confirmatory trial	Use a prespecified disease-specific primary endpoint, adequate sample size, concurrent controls, independent analysis, and meaningful follow-up.	A clinically meaningful active–sham difference with uncertainty estimates, safety assessment, and independent replication.

Clinical outcomes and mechanistic measurements

For Parkinson’s disease, a trial could use MDS-UPDRS Part III, with medication state and assessment timing standardized. For Alzheimer’s disease, cognition and daily function should be assessed with instruments suited to disease stage. Chronic stroke studies should select a validated functional endpoint aligned with the intended benefit. These are proposed design choices; final selections require investigator input and a power calculation.

EEG/ERP, perfusion measurements, sleep metrics, and relevant blood markers can be secondary or exploratory outcomes. EEG-derived brain age requires external validation and prespecified analysis

before it is used as a surrogate for disease modification. A circulating biomarker should not be represented as a direct measure of brain mitochondrial function without supporting validation.

Randomization, allocation concealment, blinding assessment, device coding, stable standard-of-care treatment, and prespecified missing-data handling are important. Analysis should emphasize the between-group difference, effect size, confidence interval, and clinical meaning. Multiplicity must be addressed when many EEG channels, frequencies, organ outputs, or clinical scales are examined.

Follow-up should distinguish immediate changes in state or performance from sustained benefit. Structured adverse-event ascertainment is preferable to relying on spontaneous complaints. Studies should make deidentified analysis data and device characterization methods available where appropriate to support independent evaluation.

8. Interpretation, scientific communication, and conclusion

How to interpret a positive study

A favorable result should specify the population, device version, exposure, comparator, outcome, magnitude, and duration. An improvement in attention, mobility, sleep, or pain is a functional result. Evidence of neural repair requires appropriate structural or cellular measurements. Evidence of disease modification requires an adequate longitudinal design demonstrating an effect on the disease process or trajectory.

A proposed mitochondrial mechanism should be supported by relevant cellular or tissue measurements. A perfusion mechanism should be supported by validated vascular measurements. EEG changes can support a neurophysiological response but cannot substitute for either mechanism. A null result at any stage should narrow or revise the hypothesis rather than be redescribed as an unmeasured normalization.

Appropriate relationship to the Nobel research

The 2026 Nobel-recognized optogenetics research establishes that light can control neuronal activity through identified light-sensitive proteins. It supports the scientific rationale for investigating optical interactions with neural systems. Biophoton generator research requires its own evidence linking a characterized exposure to a biological mechanism and clinically meaningful outcomes.

Scientific communication should avoid describing a generator as Nobel-validated, equating proximity exposure with optogenetics, or treating reported case improvements as proof of disease reversal. More precise statements identify the tested outcome and evidence level. The term “quantum” should be attached to a defined, measured phenomenon; it should not serve as a general explanation for an unexplained clinical effect.

Evidence provenance and independence

The supplied device-related publications include investigators affiliated with Tesla BioHealing and/or FIAM. This provenance should be transparent in subsequent research and publication, with author relationships, funding, analysis responsibility, and relevant interests disclosed. Independent

measurement, assessment, and replication would substantially strengthen confidence in the findings. This paper does not assign authorship or imply endorsement by the Nobel Assembly or the laureates.

Conclusion

Biophoton generators warrant consideration as an investigational approach to brain-related functional outcomes when their physical exposure and biological effects can be characterized reproducibly. The supplied publications identify clinical observations and endpoints for further testing, with a reported randomized chronic-stroke comparison providing the strongest design in this collection. Selected cases, reporting inconsistencies, and incomplete dosimetry limit the conclusions currently available.

The most productive contribution of the Nobel research is its standard of causal evidence: a defined exposure, an identified biological target, and a reproducible functional response. Applying that standard to generator research can clarify whether benefits exist, which mechanisms explain them, and which patients and protocols are appropriate for further clinical development.

References: foundational research

[1] Nobel Assembly at Karolinska Institutet. The 2026 Nobel Prize in Physiology or Medicine. Press release, 5 October 2026.

https://www.nobelprizemedicine.org/wp-content/uploads/2026/10/Nobel_prize_ENG_PR.pdf

[2] Nagel G, Szellas T, Huhn W, et al. Channelrhodopsin-2, a directly light-gated cation-selective membrane channel. *Proceedings of the National Academy of Sciences*. 2003;100(24):13940–13945.

<https://doi.org/10.1073/pnas.1936192100>

[3] Boyden ES, Zhang F, Bamberg E, Nagel G, Deisseroth K. Millisecond-timescale, genetically targeted optical control of neural activity. *Nature Neuroscience*. 2005;8:1263–1268.

<https://doi.org/10.1038/nn1525>

[4] Kravitz AV, Freeze BS, Parker PRL, et al. Regulation of parkinsonian motor behaviours by optogenetic control of basal ganglia circuitry. *Nature*. 2010;466:622–626.

<https://doi.org/10.1038/nature09159>

[5] Kobayashi M, Takeda M, Sato T, et al. In vivo imaging of spontaneous ultraweak photon emission from a rat's brain correlated with cerebral energy metabolism and oxidative stress. *Neuroscience Research*. 1999;34(2):103–113.

[https://doi.org/10.1016/S0168-0102\(99\)00040-1](https://doi.org/10.1016/S0168-0102(99)00040-1)

[6] Tang R, Dai J. Spatiotemporal imaging of glutamate-induced biophotonic activities and transmission in neural circuits. *PLOS ONE*. 2014;9(1):e85643.

<https://doi.org/10.1371/journal.pone.0085643>

[7] Pruitt T, Wang X, Wu A, Kallioniemi E, Husain MM, Liu H. Transcranial photobiomodulation (tPBM) with 1,064-nm laser to improve cerebral metabolism of the human brain in vivo. *Lasers in Surgery and Medicine*. 2020;52:807–813.

<https://doi.org/10.1002/lsm.23232>

[8] Sahel JA, Boulanger-Scemama E, Pagot C, et al. Partial recovery of visual function in a blind patient after optogenetic therapy. *Nature Medicine*. 2021;27:1223–1229.

<https://doi.org/10.1038/s41591-021-01351-4>

References: biophoton-generators-related publications

[9] James Z Liu, Mariola A Smotrys, Seth D Robinson, Hui X Yu, Sherry X Liu, et al. (2025) Multimodal Evidence of Cognitive Restoration in Alzheimer's Disease Following Biophoton Therapy: A Neurophysiological and Energetic Assessment. *Journal of Alzheimer's Disease & Reports*. SRC/JADR-107. [https://doi.org/10.47363/JADR/2025\(2\)107](https://doi.org/10.47363/JADR/2025(2)107)

[10] Ya Hu, Leon Chien, Helen Y Gu, James Z Liu (2025) Enhancing Blood Fluidity and Physiological Markers in Early-Stage Alzheimer's Disease Using Biophoton Generators: A Case Study. *Journal of Neurology Research Reviews & Reports*. SRC/JNRRR-281. [https://doi.org/10.47363/JNRRR/2025\(7\)223](https://doi.org/10.47363/JNRRR/2025(7)223)

[11] James Z Liu, Mariola A Smotrys, Seth D Robinson, Hui X Yu, Sherry X Liu, et al. (2025) Quantitative EEG Evidence of Cognitive Restoration in Alzheimer's Disease Following Biophoton Generator Therapy. *Journal of Neurology Research Reviews & Reports*. SRC/JNRRR-282. [https://doi.org/10.47363/JNRRR/2025\(7\)224](https://doi.org/10.47363/JNRRR/2025(7)224)

[12] James Z Liu, Mariola A Smotrys, Seth D Robinson, Hui X Yu, Sherry X Liu, et al. (2025) Quantitative EEG Evidence of Functional Brain Recovery in Parkinson's Disease Following Biophoton Therapy. *Journal of Neurology Research Reviews & Reports*. SRC/JNRRR-283. [https://doi.org/10.47363/JNRRR/2025\(7\)225](https://doi.org/10.47363/JNRRR/2025(7)225)

[13] James Z Liu, Mariola Smotrys, Seth D Robinson, Sherry Liu, Helen Y Gu (2025) Therapeutic Benefits of Biophoton Therapy in Parkinson's Disease: Clinical Evidence from a Pilot and Real-World Study. *Journal of Neurology Research Reviews & Reports*. SRC/JNRRR-275. [https://doi.org/10.47363/JNRRR/2025\(7\)217](https://doi.org/10.47363/JNRRR/2025(7)217)

[14] Ya Hu, Helen Y Gu, James Z Liu (2025) Twelve-Day Live Blood Analysis Reveals Hemorheological and Microvascular Restoration in a Parkinsons Disease Patient Following Biophoton Therapy. *Journal of Neurology Research Reviews & Reports*. SRC/JNRRR-277. [https://doi.org/10.47363/JNRRR/2025\(7\)219](https://doi.org/10.47363/JNRRR/2025(7)219)

[15] James Z Liu, Helen Y Gu, Mariola Smotrys, Seth Robinson, Huixia Yu, et al. (2025) A Breakthrough in Stroke Rehabilitation: Non-Invasive Biophoton Therapy Promotes Neurofunctional Recovery in Chronic Stroke Patients. *Journal of Alzheimer's Disease & Reports*. SRC/JADR-108. [https://doi.org/10.47363/JADR/2025\(2\)108](https://doi.org/10.47363/JADR/2025(2)108)

[16] James Z Liu, Mariola A Smotrys, Seth D Robinson, Hui X Yu, Sherry X Liu, et al. (2025) Quantitative EEG Reveals Cognitive and Motor Restoration After Biophoton Treatment in Chronic Stroke. *Journal of Neurology Research Reviews & Reports*. SRC/JNRRR-284. [https://doi.org/10.47363/JNRRR/2025\(7\)227](https://doi.org/10.47363/JNRRR/2025(7)227)

[17] Ya Hu, Leon Chien, Helen Y Gu, James Z Liu (2025) Biophoton Therapy Restores Hemorheology in Chronic Stroke: Live Blood Evidence of Microvascular Recovery. *Journal of Neurology Research Reviews & Reports*.

Reports. SRC/JNRRR-287.

[https://doi.org/10.47363/JNRRR/2025\(7\)229](https://doi.org/10.47363/JNRRR/2025(7)229)

[18] Tapzyanah Thomas, Helen Y Gu, Mariola A Smotrys, Seth D Robinson, James Z Liu (2025) Non-Invasive Biophoton Therapy for Neurocognitive and Physical Recovery in Retired Athletes with Traumatic Brain Injury: A Pilot Study in Former NFL Players. Journal of Neurology Research Reviews & Reports. SRC/JNRRR-288.

[https://doi.org/10.47363/JNRRR/2025\(7\)230](https://doi.org/10.47363/JNRRR/2025(7)230)

[19] James Z Liu, Laura Poirier, Jonathan Louie, Helen Y Gu (2025) Non-Invasive Biophoton Therapy Restores Visual Function in Advanced Glaucoma: A Human and Canine Case Report. Journal of Neurology Research Reviews & Reports. SRC/JNRRR-289.

[https://doi.org/10.47363/JNRRR/2025\(7\)231](https://doi.org/10.47363/JNRRR/2025(7)231)

[20] Jeannine Welker, Helen Y Gu, James Z Liu (2026) Observational Case Series of Three Individuals with Amyotrophic Lateral Sclerosis Using Biophoton Generators. Journal of Neurology Research Reviews & Reports. SRC/JNRRR-306.

[https://doi.org/10.47363/JNRRR/2026\(8\)247](https://doi.org/10.47363/JNRRR/2026(8)247)

[21] James Z Liu, Mariola A Smotrys, Seth D Robinson, Hui X Yu, Sherry X Liu, et al. (2025) Biophoton Therapy for Chronic Pain: Clinical and Real-World Breakthrough. Journal of Neurology Research Reviews & Reports. SRC/JNRRR-290.

[https://doi.org/10.47363/JNRRR/2025\(7\)232](https://doi.org/10.47363/JNRRR/2025(7)232)

[22] Liu JZ, Smotrys MA, Ager A, Liu DR, Liu SX, Gu HY. (2026) Impact of Biophoton Treatment on Functional Brain Age in Patients with Parkinson's Disease Based on EEG-Derived Metrics. J Neurol Res Rev Rep, 2026, 8(2): 1-7. DOI: [doi.org/10.47363/JNRRR/2026\(8\)244](https://doi.org/10.47363/JNRRR/2026(8)244).

[23] Liu JZ, Smotrys MA, Ager A, Liu DR, Gu YH (2026) Improvement in EEG-Derived Functional Brain Age During a 4-Week Biophoton Therapy Intervention in 5 Patients with Alzheimer's Disease – A Case Report. J Alzheimers Dise & Rep, 2026; 3(1): 1-7. [https://doi.org/10.47363/JADR/2026\(3\)116](https://doi.org/10.47363/JADR/2026(3)116)