

Perspective Article

Open Access

A Nobel-Inspired Weak-Signal Biological Amplification Hypothesis for Biophoton-Based Neuromodulation

James Z Liu* and Helen Y Gu

First Institute of All Medicines, 1000 Uniqema Blvd, New Castle, DE 19720, USA

ABSTRACT

Background: Living systems can convert small physical or chemical perturbations into larger responses through selective molecular detection, signal transduction and nonlinear amplification. The 2026 Nobel Prizes in Physiology or Medicine, Physics and Chemistry provide distinct examples of optical control of excitable cells, inference from rare weakly interacting events and amplification of molecular asymmetry, respectively. These discoveries do not establish the efficacy or mechanism of biophoton therapy.

Hypothesis: We propose a Weak-Signal Biological Amplification Hypothesis for neurological research. If an externally generated ultra-low-intensity optical component is physically present at a biological target, spectrally compatible with an endogenous photoacceptor and distinguishable from background, photon absorption could initiate a small molecular perturbation that is amplified through mitochondrial, redox, ionic, calcium, kinase and gene-regulatory networks. The final response would depend on signal properties, tissue state, biological gain and noise rather than input energy alone.

Observations: Endogenous ultraweak photon emission is well documented as a correlate of oxidative metabolism, whereas a dedicated physiological photon-communication system remains unproven. Photobiomodulation provides a mechanistic precedent for photon-triggered cellular signaling at characterized doses. Published human studies of biophoton-generator exposure in chronic stroke, Parkinson disease, Alzheimer disease and traumatic brain injury are hypothesis-generating but are limited by small samples, overlapping cohorts, incomplete physical dosimetry and limited independent replication.

Conclusion: The hypothesis is testable through radiometric characterization, wavelength- and dose-response studies, pathway blockade, rigorous sham controls and independent replication.

***Corresponding author**

James Z Liu, First Institute of All Medicines, 1000 Uniqema Blvd, New Castle, DE 19720, USA.

Received: October 09, 2026; **Accepted:** October 15, 2026; **Published:** October 23, 2026

Keywords: Biophoton, Ultraweak Photon Emission, Photobiomodulation, Neuromodulation, Nonlinear Signaling, Mitochondria, Optogenetics, Neurodegeneration

Introduction

Weak signals are fundamental to physiology, but the phrase “weak signal” must be used precisely. Biological systems can respond to very small inputs because receptors, ion gradients, enzyme cascades and gene-regulatory networks can convert an initiating event into a larger downstream response. In specialized visual systems, photoreceptors can respond to single photons, and controlled psychophysical experiments have shown human perception approaching the single-photon regime. Likewise, signaling cascades can display thresholding, ultrasensitivity, feedback, and switch-like behavior. Thus, the energy carried by an initiating signal is not necessarily the energy that powers the full biological response; cellular metabolism can supply the downstream energy after a molecular state has been changed.

This distinction is relevant to neurology. Neurons and glia operate through electrochemical gradients, neurotransmission, calcium dynamics, redox regulation, mitochondrial metabolism, and network-level synchronization. Disease can alter receptor

expression, membrane excitability, redox state, mitochondrial function, inflammatory tone, and signal-to-noise relationships. Consequently, the same small physical perturbation could, in principle, have different consequences in healthy and diseased tissue. This does not imply that every weak signal is biologically meaningful. A weak input becomes relevant only if it is physically present at the target, detected by a suitable molecular receiver, transduced into a measurable change, and amplified above biological and instrumental noise.

The attached conceptual framework proposed that the 2026 Nobel Prizes in Physiology or Medicine, Physics, and Chemistry can be viewed together as an inspiration for investigating weak-signal biology. The present manuscript reformulates that idea as a falsifiable hypothesis suitable for neurological research. The Nobel discoveries are used as conceptual precedents, not as evidence that biophoton-based interventions are effective.

Materials and Methods**Scope and Literature Approach**

This article is a hypothesis and perspective, not a systematic review or meta-analysis. The manuscript was developed by integrating: (1) official 2026 Nobel Prize descriptions; (2) foundational primary

literature on optogenetics, neutrino detection, and asymmetric amplification; (3) peer-reviewed literature on ultraweak photon emission (UPE), photobiomodulation (PBM), nonlinear signaling, and mitochondrial biology; and (4) published preliminary human reports of externally applied biophoton-generator exposure in neurological populations. Literature and bibliographic details were checked through 8 October 2026.

The evidence is intentionally separated into three levels. Established findings include light-gated ion-channel control in optogenetics, astrophysical-neutrino detection by IceCube, nonlinear/asymmetric amplification in chemistry, spontaneous UPE from biological systems, and photon-triggered responses in PBM. Hypothesis-generating observations include the reported human studies of biophoton-generator exposure. Speculative elements include a dedicated endogenous biophoton communication system and the proposed Weak-Signal Biological Amplification mechanism for the external exposures discussed here. No new human or animal data were generated or analyzed for this manuscript.

Observations: Three Nobel-Informed Principles

Optical Transduction - 2026 Nobel Prize in Physiology or Medicine
The 2026 Nobel Prize in Physiology or Medicine was awarded to Karl Deisseroth, Peter Hegemann, and Georg Nagel for discoveries concerning light-gated ion channels and optogenetics [1]. Channelrhodopsins are light-gated membrane proteins; foundational work showed that illumination can produce cation currents, and subsequent studies demonstrated millisecond-scale optical control of neuronal firing [2,3]. The biologically important lesson is not that ordinary mammalian neurons naturally behave like channelrhodopsin-expressing cells. Rather, photon absorption can become an electrical biological signal when an appropriate molecular receiver is present.

Optogenetics therefore establishes a stringent principle of optical transduction: photon absorption changes a molecular state, that state alters ion conductance and the cell converts the event into a larger electrophysiological response. Most neuroscience applications rely on genetically introduced microbial opsins, so this Nobel-recognized mechanism should not be cited as proof that endogenous ultraweak photons control mammalian neural circuits.

Rare-Signal Inference - 2026 Nobel Prize in Physics

The 2026 Nobel Prize in Physics was awarded to Francis Halzen for decisive contributions to the IceCube Neutrino Observatory and the discovery of high-energy neutrinos of astrophysical origin [4]. IceCube detects light produced after rare neutrino interactions in Antarctic ice; the detector then uses the timing and spatial pattern of those events to infer properties of the originating neutrinos [4,5].

The relevance to biophoton research is methodological, not physical. Neutrinos and photons are fundamentally different particles with different interactions with matter. Biophotons should not be inferred to penetrate tissue, metal, or other barriers in a neutrino-like manner. The useful lesson is that extremely uncommon or faint events require rigorous detector characterization, background subtraction, signal reconstruction, repeated observation and statistical discrimination from noise.

Nonlinear Amplification - 2026 Nobel Prize in Chemistry

The 2026 Nobel Prize in Chemistry was awarded to Henri B. Kagan and Kenso Soai for the discovery of non-linear effects and autocatalysis in asymmetric organic synthesis [6]. Kagan and colleagues demonstrated nonlinear relationships between catalyst and product enantiomeric excess [7]. Soai and colleagues subsequently showed asymmetric autocatalysis in which a small initial enantiomeric imbalance could be strongly amplified [8,9].

Table 1: Nobel-Informed Principles and Their Scientific Boundaries

Domain	Established Example	Transferable Principle	Boundary Condition for this Hypothesis
Physiology/Medicine	Optogenetics: light absorption by a defined light-gated channel can be converted into ion flux and neural activity.	A molecular receiver can transduce photons into biology.	Does not show that endogenous mammalian UPE controls neurons or that external biophoton devices act through opsins.
Physics	IceCube: rare neutrino interactions are inferred from secondary optical signals using calibrated detection and background rejection.	Weak/rare signals require rigorous detection and signal-vs-noise analysis.	Photons are not neutrinos; no inference of neutrino-like penetration is justified.
Chemistry	Nonlinear asymmetric catalysis and autocatalysis can amplify a small initial molecular imbalance.	Small inputs can produce larger outcomes when a system contains nonlinear gain and feedback.	Does not demonstrate amplification of biophoton exposure in cells; a biological pathway must be shown directly.

These experiments do not demonstrate biophoton amplification. They provide a physical-chemical example of a broader systems principle: a small initiating difference need not produce a proportionally small final outcome when the receiving system contains feedback and nonlinear gain. This principle is compatible with many biologicals signaling networks, but a specific receptor, transduction pathway and energy source must still be identified for any proposed weak-signal mechanism [10,11].

Established Evidence and Terminology

Ultraweak Photon Emission is an Observed Biological Phenomenon

Living cells and tissues emit very low levels of spontaneous optical radiation commonly termed ultraweak photon emission (UPE) or, historically, biophoton emission. These are ordinary photons rather than a distinct class of particle. Mechanistic reviews link much UPE to oxidative metabolism, reactive oxygen species, lipid and protein oxidation, electronically excited molecular species, and their relaxation to lower-energy states [12-14]. UPE intensity and spectral-temporal characteristics can vary with physiological and oxidative state, making it potentially useful as a measurement of underlying biological processes.

What remains unresolved is whether endogenous UPE serves as a dedicated communication or regulatory system. Contemporary reviews discuss this possibility but do not establish a physiologically necessary photon-based signaling network [13,14]. This distinction is central to the present hypothesis: emission is established; communication remains to be demonstrated.

Photobiomodulation is Related but not Equivalent

Photobiomodulation (PBM) deliberately applies characterized red or near-infrared light to tissue. Proposed and studied mechanisms include photon absorption by endogenous chromophores, mitochondrial and redox modulation, nitric-oxide signaling, calcium-related signaling, and downstream transcriptional responses [15-18]. Cytochrome c oxidase has been widely investigated as a candidate photoacceptor, although PBM mechanisms are likely context-dependent and remain an active area of research. PBM can also show non-monotonic or biphasic dose responses [15,16,19].

PBM should not be treated as equivalent to spontaneous UPE. PBM ordinarily uses controlled wavelength, irradiance, fluence, and exposure time at photon fluxes far above spontaneous cellular emission. Accordingly, PBM provides a precedent for photon-triggered cellular signaling, but it does not establish that endogenous UPE is a communication system or that an uncharacterized external device acts through the same mechanism.

Operational Definition of External Biophoton-Generator Exposure
In this manuscript, “biophoton-generator exposure” refers only to the external devices and exposure configurations described in the cited clinical reports [20-29]. The term is retained to match the published literature. It does not imply that the physical output has been demonstrated to be identical to endogenous UPE, nor that a coherent optical field, specific photon flux, or tissue-level dose has been independently established. For mechanistic research, the external exposure must be characterized radiometrically and spectrally at the biological target before a photon-mediated mechanism can be assigned.

Proposed Weak-Signal Biological Amplification Hypothesis The Central Hypothesis is Conditional and Mechanistic

If an externally generated ultra-low-intensity optical component is physically present at a neurological target, is spectrally compatible with an endogenous photoacceptor or photosensitive molecular system, and produces absorption events that are distinguishable from local background, then the optical input may act as a trigger rather than a bulk energy source. A small initial photochemical, redox, conformational or ionic perturbation could then be amplified by pre-existing cellular energy stores and nonlinear signaling networks, potentially producing measurable changes in mitochondrial state, redox balance, calcium signaling, membrane excitability, kinase activity, gene expression or network physiology.

The proposed causal chain is: (1) physically verified weak optical input; (2) molecular reception; (3) primary molecular perturbation; (4) intracellular transduction; (5) nonlinear amplification using endogenous metabolic energy; and (6) measurable cellular or network response. Every link is experimentally separable. Failure to demonstrate any required link would weaken or falsify the mechanism.

This model also clarifies the role of energy. A weak optical input would not need to supply the energy required for tissue repair or sustained neuronal function. It would need only to alter a molecular state that then recruits cellular energy. This is analogous

to established receptor-mediated signaling: ligand binding or photon absorption can trigger a response whose energetic cost is paid by ion gradients, ATP and metabolism.

The magnitude and direction of the response are predicted to depend on at least five variables: signal spectrum and temporal structure; photon flux at the target; abundance and state of the receiver; background noise; and gain/state of the downstream network. Thus, “weak” is not synonymous with either “ineffective” or “effective”. Biological relevance is conditional on detectability and transduction.

Mechanistic Plausibility in Neurological Systems Mitochondria and Redox Signaling

Mitochondria are plausible amplification nodes because they integrate ATP production, membrane potential, redox signaling, reactive oxygen species, calcium handling, apoptosis, and stress responses. Mitochondrial dysfunction and oxidative stress are also implicated across major neurodegenerative disorders [20]. PBM studies provide evidence that optical exposure can modify mitochondrial and redox-linked variables under defined experimental conditions [15-18].

The relationship between mitochondria and UPE is potentially bidirectional at a conceptual level: oxidative metabolism can generate UPE, while externally delivered photons at appropriate wavelengths can influence cellular photochemistry in PBM paradigms [12-18]. However, the photon flux required for such effects and the photon flux produced endogenously are not interchangeable. Whether physiological UPE is sufficient to drive biologically meaningful feedback remains unresolved.

Ion Channels, Calcium and Electrophysiological Amplification
Optogenetics demonstrates that a photon-sensitive channel can couple light to ion flow and neural excitability [1-3]. Endogenous mammalian systems do not generally express microbial channelrhodopsins, so the relevant question for biophoton research is whether native photoresponsive molecules or indirect redox-sensitive ion-channel pathways can detect the proposed exposure. A credible mechanism should identify the receiver, show spectral concordance between the physical signal and receiver absorption and demonstrate downstream changes in membrane or calcium physiology.

Network Gain, State Dependence and Disease

Nonlinear signaling means that response magnitude can depend strongly on system state [10,11]. A healthy neuron, a metabolically stressed neuron and an inflamed neural network may differ in redox buffering, mitochondrial reserve, receptor abundance, membrane excitability and baseline oscillatory state. The hypothesis therefore predicts state dependence: the same physical input may produce different outcomes across disease stages and cell types. This concept should be tested prospectively rather than invoked retrospectively to explain variable results.

Signal-to-Noise and Dynamic Range

For a weak optical signal to matter biologically, the relevant molecular detector must distinguish it from thermal, chemical, optical, and stochastic background. Disease could theoretically alter this dynamic range through changes in oxidative stress, receptor expression, mitochondrial reserve or network excitability. Nevertheless, “improving signal-to-noise” is a systems-level interpretation, not a demonstrated mechanism of biophoton-generator exposure. It should be operationalized using predefined physiological measures.

Preliminary Human Observations Motivating the Hypothesis

The purpose of this section is not to establish efficacy. It is to determine whether published human observations justify more rigorous mechanistic testing. The cited reports describe external biophoton-generator exposure in chronic stroke, Parkinson disease (PD), Alzheimer disease (AD), and traumatic brain injury (TBI) [21-29]. Several reports include quantitative EEG (qEEG), event-related potentials (ERP), clinical measures or patient-reported outcomes. Table 2 summarizes the evidentiary role and major limitations of these studies.

Table 2: Preliminary Human Observations Relevant to the Hypothesis

These studies are summarized as hypothesis-generating evidence. Participant cohorts and substudies may overlap and should not be pooled as independent replications.

Condition	Published Report/Design	Reported Signal	Major Limitations for Inference
Chronic stroke	Randomized, triple-blind, placebo-controlled report (n=46) [21]; longitudinal qEEG report (n=5) [22].	Reported changes in functional/neurological outcomes and selected qEEG/ERP measures.	Potential cohort/substudy overlap; small qEEG sample; incomplete target-level optical dosimetry; no independent replication.
Parkinson disease	Pilot/real-world report [23]; qEEG/ERP report [24]; EEG-derived functional brain-age analysis (10 treated, 6 controls) [25].	Reported clinical observations and changes in selected neurophysiological metrics.	Small samples; derived/exploratory EEG endpoints; possible cohort overlap; limited independent replication.
Alzheimer disease	Multimodal exploratory report [26]; qEEG report from a 24-patient randomized trial, with serial EEG data reported for 4 participants [27].	Reported cognitive and electrophysiological changes in exploratory analyses.	Biomarker subset is much smaller than parent trial; multiple exploratory outcomes; physical mechanism not established.
Chronic TBI	Observational pilot in 12 retired NFL players [28]; separate qEEG/ERP report in 4 participants [29].	Reported patient-reported changes and selected qEEG/ERP changes.	Observational/self-report components; very small neurophysiology sample; possible overlap; no independent replication.

In chronic stroke, one published randomized, triple-blind, placebo-controlled study included 46 participants and reported the desirable changes in functional, neurological, quality-of-life and neurophysiological measures after active exposure [21]. A separate qEEG report described longitudinal electrophysiological improvement in five chronic-stroke participants [22]. These findings are hypothesis-generating; the relationship between the cohorts, physical dosimetry at the biological target and independent replication should be clarified in future work.

In PD, a pilot/real-world report described clinical observations, a qEEG/ERP report described longitudinal neurophysiological improvements and a 2026 analysis compared EEG-derived functional brain-age reduction in 10 treated participants with six controls [23-25]. These reports motivate controlled replication but do not establish disease modification. Small samples, derived EEG endpoints, possible cohort overlap and the absence of independent multisite replication limit causal inference.

In AD, a multimodal exploratory report described an improvement in cognitive, electrophysiological, vascular, and other measurements [26]. A separate qEEG publication arose from a randomized, triple-blind, placebo-controlled trial of 24 patients, but the serial EEG report analyzed four participants [27]. This distinction should be retained when interpreting the strength of the evidence; a small biomarker subset cannot be treated as equivalent to a completed, adequately powered efficacy trial.

In chronic TBI, an independent-observational pilot involving 12 retired professional football players reported patient-spouse-reported satisfactory outcomes, while a separate qEEG/ERP report demonstrated positive signals in four participants in a deferent TBI clinical study [28,29]. These studies provide feasibility signals, not definitive evidence of therapeutic efficacy.

Collectively, these publications, although requiring independent replication, provide preliminary evidence that exposure to a biophoton environment may be associated with favorable clinical outcomes in patients with neurological disorders. It is also encouraging that positive changes were reported across participants with distinct clinical diagnoses, suggesting that the observed effects may not be limited to a single neurological condition. However, most of these reports originate from related investigative teams and involve devices associated with the same sponsor. Independent replication, prespecified endpoints, comprehensive physical characterization of the exposure, rigorous sham controls, and transparent reporting of any cohort overlap are therefore essential before definitive claims regarding efficacy or mechanism can be justified.

Falsifiable Predictions and Alternative Explanations

A scientifically useful hypothesis must make predictions that could prove it wrong. The proposed model yields the following tests:

1. Physical-Signal Prediction: A reproducible optical signal should be measurable from the device or environment at the location of the biological target, with spectrum, photon flux/irradiance, temporal pattern, distance dependence, and background characterized using calibrated instrumentation.
2. Optical-Dependence Prediction: If the effect is photon-mediated, wavelength-selective attenuation or filtering should alter the biological response in a manner consistent with the measured spectrum and candidate absorber, while matched controls preserve temperature, vibration, electrical coupling, airflow, and other non-optical variables.
3. Dose-Response Prediction: A reproducible relationship should exist between quantified exposure and biological outcome. A biphasic response is plausible by analogy with PBM [19] but should be treated as a prediction, not assumed in advance.
4. Mechanistic Prediction: Early molecular events should precede later network outcomes. Candidate measures include

mitochondrial membrane potential, oxygen consumption, ATP-related metabolism, NADH/FAD redox state, reactive oxygen species, nitric oxide, intracellular calcium, ion-channel activity, phosphoproteomic changes, and transcriptional responses.

5. Blockade Prediction: Disrupting the candidate photoacceptor or downstream pathway should reduce the response without producing nonspecific toxicity. Genetic, pharmacological, or optical blockade can help establish causality.
6. State-Dependence Prediction: If network gain matters, prespecified differences should be observed between healthy and stressed cells or between defined disease states, rather than being inferred only after data collection.
7. Neurophysiological Prediction: EEG/ERP changes should be reproducible under blinded acquisition and analysis, should survive correction for multiple comparisons, and should correlate prospectively with validated clinical outcomes rather than relying on post hoc biomarker selection.
8. Replication Prediction: Findings should reproduce in laboratories and clinical centers independent of the device developer and original investigative group.

Alternative explanations must be actively tested. Apparent effects could arise from thermal changes, conventional electromagnetic coupling, electrical grounding, vibration, acoustic effects, airflow, expectation/placebo effects, regression to the mean, spontaneous clinical fluctuation, device unblinding, detector drift, signal-processing choices or multiple-testing artifacts. A photon-specific mechanism is strengthened only when these explanations are excluded.

Discussion

The proposed framework connects three Nobel-recognized principles without conflating their mechanisms. Optogenetics shows that light can control cellular electrical behavior when a molecular receiver is present [1-3]. Neutrino astronomy demonstrates the methodological rigor required to infer information from rare weakly interacting events [4,5]. Asymmetric catalysis and autocatalysis demonstrate that small molecular asymmetries can be amplified by nonlinear chemistry [6-8]. None of these observations proves biophoton-based neuromodulation. Together, however, they suggest a disciplined research strategy: characterize the physical signal, identify the receiver, measure the first transduction event, quantify network gain and test whether the final response disappears when the proposed pathway is interrupted.

This framework also refines the proposition that weak signals are critically important for health and disease. Biology indeed relies on low-abundance hormones, receptor occupancy, membrane-voltage changes, calcium transients and in some systems single-photon detection. Yet the importance of a weak signal is conferred by the architecture of the receiving system. A signal below the physical or biological detection threshold is irrelevant; a signal within a sensitive and amplifying pathway may be consequential. Disease may alter thresholds and gain, but that assertion requires direct measurement.

For neurology, the most informative near-term experiments are likely not large efficacy trials alone, but linked biophysics-mechanism-clinical studies. First, the external exposure should be characterized using calibrated optical detectors across the full relevant spectrum and at clinically relevant distances. Second, cell and tissue experiments should isolate optical from thermal and electromagnetic variables and identify candidate absorbers

and early molecular changes. Third, small preregistered human mechanistic trials should pair exposure dosimetry with blinded neurophysiology and validated clinical endpoints. Only after these steps demonstrate a reproducible chain of causality should broader therapeutic claims be advanced.

An additional implication concerns terminology. “Biophoton therapy” can be interpreted as implying both a defined photon exposure and an established therapeutic effect. Until the physical output, target dose, and mechanism are characterized, “biophoton-generator exposure” or “weak optical exposure under investigation” is more neutral for scientific reporting. Similarly, descriptions such as “coherent,” “quantum,” or “information-bearing” should be used only when the corresponding physical property has been measured and defined operationally.

Limitations

This hypothesis has several important limitations. First, the three 2026 Nobel Prizes concern distinct phenomena; the connections made here are conceptual and methodological rather than evidence of a unified physical mechanism. Second, spontaneous UPE is established, but a necessary endogenous photon-communication network in mammals has not been demonstrated [13,14]. Third, PBM provides evidence for photon-triggered signaling at characterized optical doses, but those doses and mechanisms cannot be automatically extrapolated to spontaneous UPE or to externally generated exposures with different physical characteristics [15-19].

Fourth, the human evidence summarized here is encouraging but remains preliminary. Future studies should be adequately powered, prospectively designed, and independently replicated [21-29]. Fifth, most of the cited clinical reports include authors or investigators affiliated with organizations associated with the devices under study, creating a potential conflict of interest and making independent confirmation particularly important. To facilitate such verification, organizations related to the authors are providing devices for use in independent studies. Finally, the proposed hypothesis should be evaluated independently of the reported clinical changes. Non-optical mechanisms, placebo effects, expectancy effects, and other nonspecific study influences remain plausible explanations unless and until they are directly excluded through rigorous experimental controls.

Conclusions

The Weak-Signal Biological Amplification Hypothesis proposes a specific, testable route by which a very small optical input could become biologically relevant: the signal must physically reach the target, interact with a compatible molecular receiver, initiate a measurable perturbation and be amplified by endogenous cellular networks. The hypothesis does not require the initiating photons to supply the energy for the downstream response; it requires a credible transduction mechanism linked to cellular metabolism.

The 2026 Nobel Prizes provide useful scientific analogies - optical transduction in physiology, rigorous inference from rare physical events, and nonlinear amplification in chemistry - but they do not validate biophoton therapy. Endogenous UPE is measurable, whereas its role as a communication system remains unresolved. PBM demonstrates that photons can influence cellular signaling under defined exposure conditions. Preliminary neurological reports of biophoton-generator exposure justify further study but are not sufficient to establish efficacy, disease modification or mechanism.

The most important next step is therefore not broader therapeutic assertion but stronger measurement: radiometry and spectroscopy of the exposure, mechanistic pathway testing, sham-controlled blinded experiments, prespecified neurological outcomes, and independent replication. In living systems, a signal may matter not because it is strong, but because a suitably configured biological system can detect, transduce, and amplify it. Whether the external exposures discussed here meet those conditions is an empirical question that can now be tested.

Declarations

Ethics Statement: This hypothesis/perspective article reports no new experiments involving human participants or animals. Ethics approvals and informed-consent procedures for the previously published clinical studies are described in the original reports.

Data Availability: No new datasets were generated or analyzed for this article.

Competing Interests: James Z Liu and Helen Y. Gu have disclosed in prior publications that they are co-founders and co-inventors of Tesla BioHealing, Inc., a company associated with devices discussed in the cited clinical studies. This relationship represents a potential financial conflict of interest and should be considered when interpreting the preliminary evidence. The present manuscript therefore emphasizes independent replication and physical characterization of the exposure.

Author Contributions: James Z Liu conceived the hypothesis and developed the initial framework. Helen Y Gu contributed to interpretation and critical revision. Both authors reviewed and approved the manuscript.

Acknowledgements

The authors gratefully acknowledge the pioneering contributions of Peter Hegemann, Georg Nagel, and Karl Deisseroth, whose Nobel Prize-recognized work established fundamental principles by which light can be detected by biological molecules and translated into cellular and neural responses. Their discoveries in light-sensitive proteins and optogenetics have helped provide an important scientific foundation for investigating how weak optical signals may interact with living systems and have inspired further research into the potential role of low-intensity photon signaling in medicine.

References

- Karolinska Institutet (2026) The 2026 Nobel Prize in Physiology or Medicine to Peter Hegemann, Georg Nagel and Karl Deisseroth <https://news.ki.se/the-2026-nobel-prize-in-physiology-or-medicine-to-peter-hegemann-georg-nagel-and-karl-deisseroth>.
- Nagel G, Szellas T, Huhn W, Kateriya S, Adeishvili N, et al. (2003) Channelrhodopsin-2, a directly light-gated cation-selective membrane channel. *Proc Natl Acad Sci* 100: 13940-13945.
- Boyden ES, Zhang F, Bamberg E, Nagel G, Deisseroth K (2005) Millisecond-timescale, genetically targeted optical control of neural activity. *Nat Neurosci* 8: 1263-1268.
- Francis Halzen (2026) The Nobel Prize in Physics 2026: Elusive particles from the universe captured at the South Pole. Royal Swedish Academy of Sciences <https://www.kva.se/en/news/the-nobel-prize-in-physics-2026/>.
- IceCube Collaboration (2013) Evidence for high-energy extraterrestrial neutrinos at the IceCube detector. *Science* 342: 1242856.
- Henri B Kagan, Kenso Soai (2026) The Nobel Prize in Chemistry 2026: They made chemistry choose a mirror image. Royal Swedish Academy of Sciences <https://www.kva.se/en/news/the-nobel-prize-in-chemistry-2026/>.
- Puchot C, Samuel O, Dunach E, Zhao S, Agami C, et al. (1986) Nonlinear effects in asymmetric synthesis. *J Am Chem Soc* 108: 2353-2357.
- Soai K, Shibata T, Morioka H, Choji K (1995) Asymmetric autocatalysis and amplification of enantiomeric excess of a chiral molecule. *Nature* 378: 767-768.
- Tinsley JN, Molodtsov MI, Prevedel R, Wartmann D, Espigule-Pons J, et al. (2016) Direct detection of a single photon by humans. *Nat Commun* 7: 12172.
- Ferrell JE Jr (1996) Tripping the switch fantastic: how a protein kinase cascade can convert graded inputs into switch-like outputs. *Trends Biochem Sci* 21: 460-466.
- Tyson JJ, Chen KC, Novak B (2003) Sniffers, buzzers, toggles and blinkers: dynamics of regulatory and signaling pathways in the cell. *Curr Opin Cell Biol* 15: 221-231.
- Cifra M, Pospisil P (2014) Ultra-weak photon emission from biological samples: definition, mechanisms, properties, detection and applications. *J Photochem Photobiol B* 139: 2-10.
- Mould RR, Mackenzie AM, Kalampouka I, Nunn AVW, Thomas EL, et al. (2024) Ultra weak photon emission - a brief review. *Front Physiol* 15: 1348915.
- Bodis J, Berke J, Nagy B, Gulyas I, Hersics P, et al. (2026) Ultra-weak photon emission: from oxidative metabolism to DNA-based communication - a review of biochemical, biophysical and quantum biological perspectives. *Front Endocrinol (Lausanne)* 17: 1861061.
- de Freitas LF, Hamblin MR (2016) Proposed mechanisms of photobiomodulation or low-level light therapy. *IEEE J Sel Top Quantum Electron* 22: 7000417.
- Wong Riley MTT, Liang HL, Eells JT, Chance B, Henry MM, et al. (2005) Photobiomodulation directly benefits primary neurons functionally inactivated by toxins: role of cytochrome c oxidase. *J Biol Chem* 280: 4761-4771.
- Chen ACH, Arany PR, Huang YY, Tomkinson EM, Sharma SK, et al. (2011) Low-level laser therapy activates NF- κ B via generation of reactive oxygen species in mouse embryonic fibroblasts. *PLoS One* 6: e22453.
- Hamblin MR (2018) Mechanisms and mitochondrial redox signaling in photobiomodulation. *Photochem Photobiol* 94: 199-212.
- Huang YY, Sharma SK, Carroll J, Hamblin MR (2011) Biphasic dose response in low level light therapy - an update. *Dose Response* 9: 602-618.
- Lin MT, Beal MF (2006) Mitochondrial dysfunction and oxidative stress in neurodegenerative diseases. *Nature* 443: 787-795.
- Liu JZ, Gu HY, Smotrys MA, Robinson SD, Yu HX, et al. (2025) A Breakthrough in Stroke Rehabilitation: Non-Invasive Biophoton Therapy Promotes Neurofunctional Recovery in Chronic Stroke Patients. *J Alzheimers Dis Rep* 2: 1-13.
- Liu JZ, Smotrys MA, Robinson SD, Yu HX, Liu SX, et al. (2025) Quantitative EEG Reveals Cognitive and Motor Restoration After Biophoton Treatment in Chronic Stroke. *J Neurol Res Rev Rep* 7: 1-10.
- Liu JZ, Smotrys M, Robinson SD, Liu S, Gu HY (2025) Therapeutic Benefits of Biophoton Therapy in Parkinson's Disease: Clinical Evidence from a Pilot and Real-World Study. *J Neurol Res Rev Rep* 7: 1-6.
- Liu JZ, Smotrys MA, Robinson SD, Yu HX, Liu SX, et al. (2025) Quantitative EEG Evidence of Functional Brain

- Recovery in Parkinson's Disease Following Biophoton Therapy. J Neurol Res Rev Rep 7: 1-9.
25. Liu JZ, Smotrys MA, Ager A, Liu DR, Liu SX, et al. (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 8: 1-7.
26. Liu JZ, Smotrys MA, Robinson SD, Yu HX, Liu SX, et al. (2025) Multimodal Evidence of Cognitive Restoration in Alzheimer's Disease Following Biophoton Therapy: A Neurophysiological and Energetic Assessment. J Alzheimers Dis Rep 2: 1-10.
27. Liu JZ, Smotrys MA, Robinson SD, Yu HX, Liu SX, et al. (2025) Quantitative EEG Evidence of Cognitive Restoration in Alzheimer's Disease Following Biophoton Generator Therapy. J Neurol Res Rev Rep 7: 1-11.
28. Thomas T, Gu HY, Smotrys MA, Robinson SD, Liu JZ (2025) Non-Invasive Biophoton Therapy for Neurocognitive and Physical Recovery in Retired Athletes with Traumatic Brain Injury: A Pilot Study in Former NFL Players. J Neurol Res Rev Rep 7: 1-6.
29. Liu JZ, Smotrys MA, Robinson SD, Yu HX, Liu SX, et al. (2025) Biophoton Therapy Reverses Electrophysiological Deficits in Chronic Traumatic Brain Injury: Quantitative EEG Evidence of Cognitive and Network Recovery. J Neurol Res Rev Rep 7: 1-11.