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Recovery Sleep in an Ultraweak Photon Environment: A Unified Neurorestorative Paradigm Across Neurodegenerative and Neurovascular Disorders

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ABSTRACT

Background: Neurodegenerative and neurovascular disorders, including Parkinson's disease, Alzheimer's disease, chronic stroke, traumatic brain injury, and Amyotrophic Lateral Sclerosis, remain major unmet clinical challenges worldwide. Despite distinct clinical presentations, these disorders share overlapping biological mechanisms, including mitochondrial dysfunction, oxidative stress, neuroinflammation, impaired neurovascular regulation, disrupted neural signaling, sleep disturbance, and reduced cellular resilience. Ultraweak Photon Emission (UPE) has emerged as a measurable biological phenomenon associated with mitochondrial-redox metabolism, neural activity, cerebral blood flow, glutamate signaling, electroencephalographic activity, and neurodegenerative processes.

Objective: To review current evidence regarding UPE and Ultra-Weak Photon Environments (UWPEs) as potential biomarkers and investigational neurorestorative approaches for neurological disorders.

Methods: A narrative review was conducted of published experimental, preclinical, and early clinical studies involving UPE, biophotonic signaling, neural stem cells, human brain photon monitoring, and recovery sleep within UWPEs generated by ultraweak biophoton emitters.

Results: Published evidence demonstrates that living brain tissue emits detectable UPE signals that can be differentiated from background photon noise. UPE has been associated with mitochondrial respiration, reactive oxygen species generation, lipid peroxidation, acetylcholinesterase activity, hippocampal memory impairment, neural stem cell differentiation, and task-related human brain activity. In three randomized controlled trials, recovery sleep within a UWPE generated by ultraweak biophoton emitters was associated with improvements in multiple neurological and functional domains, including enhanced brain activity, improved qEEG-based functional brain measures, reduced brain functional age, improved sleep quality, increased energy, improved motor function, enhanced cognition and emotional well-being, better daily functioning, and improved quality of life. These findings suggest that prolonged nighttime exposure to a UWPE may support neurofunctional recovery through modulation of mitochondrial-redox biology, sleep-mediated restoration, neurovascular regulation, and neural-network function.

Conclusion: Current evidence supports UPE as a biologically meaningful optical marker of brain physiology and redox-metabolic state. Recovery sleep within an Ultra-Weak Photon Environment represents a plausible investigational neurorestorative paradigm that warrants further mechanistic investigation and large-scale controlled clinical studies.

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Scientific Foundation for UPE in Brain Physiology, Biochemistry and Function

Recent publications provide a strong scientific foundation that UPE is a measurable biological phenomenon closely linked to brain metabolism, oxidative-redox chemistry, mitochondrial activity, neural activity, cerebral blood flow, glutamate signaling, EEG/qEEG rhythms, memory-related hippocampal function, and neural stem cell differentiation.

The strongest conclusion from the published literature is that UPE is not merely random biological noise. It is increasingly supported as a measurable optical readout of cellular and brain physiological state, especially mitochondrial-redox metabolism and neural activity. Most of these articles support UPE as a biomarker, signaling correlate, and mechanistic foundation. They do not yet prove that external ultraweak photon emitters treat neurological disease. They do provide a strong rationale for studying whether an Ultraweak Photon Environment, or UWPE, could support brain physiology, biochemical regulation, and functional recovery in neurodegenerative and neurological disorders.

Scientific Foundation: Living Brain Tissue Emits Ultraweak Photons
Multiple publications confirm that living cells and tissues spontaneously emit extremely low-intensity photons. The 2024 review *UPE, a brief review* states that cells emit ultra-low-intensity photons produced as byproducts of cellular metabolism, distinct from delayed luminescence, bioluminescence, and conventional chemiluminescence. The review also explains that the field historically suffered from insufficient detection technology, but modern photomultiplier tubes, photon counters, and sensitive imaging systems now allow detection and characterization of UPE. The 2025 *iScience* study on human brain UPE further confirms that brain tissue is a source of measurable photon emissions. Its key findings were that UPEs were detected from both resting and active human brains, brain UPE spectra and entropy differed from background levels, optical readouts correlated with evoked neuroelectric oscillations, and "photo encephalography" may represent a new label-free brain-monitoring method.

UPE is a recognized biological phenomenon. A 2024 review reported that cells emit ultra-low-intensity photons as by-products of cellular metabolism [1]. The National Research Council Canada has described ultraweak biophoton emission as a faint light generated by living tissues and linked to biological processes including cell metabolism and cellular communication [2]. A 2025 study in *iScience* reported that brains are highly metabolic organs that emit UPE and explored their relevance as optical markers of brain activity, oxidative stress, aging, and neurodegeneration [3]. This is important because it establishes that UPE can be measured not only in isolated cells or animal tissues, but also in human brain-related conditions during rest and task-related activity.

Brain UPE Reflects Mitochondrial and Redox Biochemistry
The most consistent biochemical foundation across the articles is the relationship between UPE, mitochondrial respiration, reactive oxygen species, lipid peroxidation, and oxidative stress. The 2025 human brain UPE article states that biological tissues continuously emit very low-intensity light in the visible-to-near-visible range, and that UPEs are generated by radiative decay of excited molecules. It further states that UPEs reflect cellular metabolic states, correlate with reactive oxygen species, and are coupled to mitochondrial respiration, with ROS as a primary byproduct.

The 2024 review explains the currently accepted mechanism: UPE predominantly originates from reactions of ROS, including reactive molecules and free radicals derived from oxygen reduction and electron-transfer reactions. It identifies mitochondrial respiration, lipid peroxidation, peroxisome activity, catecholamine biochemistry, and oxidation of tyrosine and tryptophan residues as relevant sources. It further explains that excited carbonyl species and singlet oxygen release excess energy as photons when returning to lower-energy states.

This biochemical chain can be summarized as: Mitochondrial activity → electron transfer → ROS/redox reactions → excited molecular species → ultraweak photon emission.

This is a powerful foundation for neurological disease research because neurodegenerative disorders commonly involve mitochondrial dysfunction, oxidative stress, and impaired energy metabolism.

Brain UPE Correlates with Neural Activity, EEG, Cerebral Blood Flow and Glutamate
Several papers support the relationship between UPE and brain physiological activity.
The 2015 brain physiology review reports that UPE has been correlated with neural activity, cerebral energy metabolism, EEG activity, cerebral blood flow, and oxidative processes. It describes experiments showing that depolarization of rat hippocampal slices increased UPE, while suppression of neural activity with tetrodotoxin decreased UPE. It also reports that in vivo rat brain UPE correlated with EEG activity, cerebral blood flow, and hyperoxia, and that glutamate increased UPE from brain slices.

The 2020 neural stem cell paper similarly summarizes earlier evidence that spontaneous UPE from rat cortex was detected in vivo without external excitation and correlated with EEG activity, cerebral blood flow, hyperoxia, and glutamate-induced activity, with the emission mainly originating from mitochondrial respiratory-chain energy metabolism through ROS production.

The 2025 biophotonic signaling review further states that UPE intensity is connected with brain activity and metabolic state. It summarizes evidence that UPE correlates with neuronal activity, cerebral energy metabolism, EEG parameters, cerebral blood flow, oxidative processes, hypoxia/reperfusion states, glutamate stimulation, neuronal membrane depolarization, and calcium entry into cells. Together, these findings support the following conclusion: Brain UPE is closely linked to active brain physiology, not merely passive tissue glow.

Human Brain UPE May Provide a New Optical Readout of Brain State
The 2025 *iScience* study is especially important because it directly tested whether human brain UPE signals can be separated from background photon noise. The study found that brain-derived UPE signals over occipital and temporal regions had distinct temporal dynamics, greater signal variability, greater complexity, and spectral differences compared with background signals.

The study also found that brain UPE signals had unique spectral signatures below 1 Hz and that optical readouts correlated with evoked neuroelectric oscillations. The authors proposed the possibility of developing photo encephalography, a passive optical method to read functional brain states.

This provides a modern bridge between UPE science and conventional brain-monitoring technologies

Conventional Brain Signal	UPE-Based Interpretation
EEG measures electrical oscillations	UPE may measure optical-redox-metabolic activity
fMRI measures blood oxygenation changes	UPE may reflect cellular metabolism and oxidative state
qEEG measures functional network rhythms	UPE may correlate with neural oscillatory dynamics
PET measures metabolic or molecular processes	UPE may provide a low-intensity optical marker of metabolism

The key point is that UPE may become a non-invasive, label-free optical biomarker of brain state.

Alzheimer's Disease Model: UPE Tracks Memory Decline, Oxidative Stress and Cholinergic Biochemistry

The 2024 *iScience* article *Monitoring Alzheimer's disease via ultraweak photon emission* provides a strong disease-relevant foundation. In a streptozotocin-induced sporadic Alzheimer's disease rat model, the researchers detected UPE from the hippocampus and found significant correlations between Alzheimer's-related pathology, memory decline, oxidative stress, and UPE intensity.

Key Findings Included

Finding	Meaning
STZ injection impaired memory	Behavioral confirmation of AD-like dysfunction
Hippocampal UPE significantly increased in STZ rats	UPE reflected disease-related brain biochemical stress
MDA increased	Lipid peroxidation/oxidative stress increased
AChE activity increased	Cholinergic dysfunction increased
Donepezil reduced UPE	UPE decreased with therapeutic biochemical improvement
UPE correlated with MDA and AChE	UPE tracked oxidative and cholinergic pathology

The study reported that UPE correlated strongly with MDA, a lipid peroxidation marker, with $r = 0.8552$, and with acetylcholinesterase activity, with $r = 0.7793$. It also reported that 73% of UPE variance could be explained by MDA concentration and 60% by AChE activity.

This is one of the strongest biochemical links in the published articles because it connects UPE to (1) Memory performance, (2) Hippocampal function, (3) Oxidative stress, (4) Lipid peroxidation, (5) Cholinergic neurotransmission, and (6) Drug-responsive biochemical improvement. The significance for neurodegenerative disorders is clear that UPE may serve as a measurable optical marker of brain biochemical stress and recovery response.

Neural Stem Cells: UPE Relates to Neural Differentiation and Regenerative Biology

The 2020 *Scientific Reports* article on adult murine neural stem cells demonstrated that neural stem cells have measurable UPE properties. The authors found that UPE increased with serial passaging, that silver nanoparticles increased UPE intensity, and that AgNP exposure increased neuronal differentiation of harvested neural stem cells by 44%. They also found that UPE after differentiation was significantly lower than before differentiation, consistent with changes in cell number and biological state.

The same paper reported that AgNP-treated neural stem cells showed significantly higher UPE than controls, and that the percentage of neurons in the AgNP group was significantly higher than control, while astrocyte percentage was lower.

This supports a regenerative-neuroscience foundation: UPE measurement may reflect neural stem cell state, differentiation status, and biological response to environmental or material factors.

For brain-recovery applications, this is important because neurodegenerative disorders and injuries often involve impaired neurogenesis, reduced plasticity, and loss of neuronal repair capacity. The neural stem cell data do not prove clinical regeneration in humans, but they provide a mechanistic rationale for studying UPE-related environments in neural repair biology.

Mitochondria are Central Therapeutic Targets in Neurodegenerative Disease

The 2022 *Frontiers in Neuroscience* review on mitochondria and organelles in neural development and neurodegenerative disorders provides the disease-mechanism foundation. It states that mitochondria play central roles in energy production, metabolic regulation, apoptosis, autophagy, neurogenesis, neuroplasticity, calcium regulation, mitochondrial dynamics, mitophagy, and neurotransmitter release.

The review also states that mitochondrial dysfunction is strongly associated with neurodegenerative disorders such as Alzheimer's disease and Parkinson's disease, including disrupted bioenergetics, mitochondrial genomic instability, increased oxidative stress, respiratory chain impairment, mtDNA alterations, and dysregulated mitophagy. It further notes that improving mitochondrial function may reverse progression through reinforcement of adult neurogenesis.

This matters because UPE is repeatedly linked to mitochondrial-redox activity. Therefore, if mitochondrial dysfunction is a central driver of brain degeneration, and UPE is an optical readout of mitochondrial-redox state, then UPE science provides both: (1) a biomarker foundation for monitoring brain stress and recovery; and (2) a mechanistic rationale for exploring ultraweak photon environments as supportive interventions.

Conceptual Foundation: Biophotonic Signaling in the Human Body and Brain

The 2025 *Frontiers in Systems Neuroscience* article presents a broad theoretical model of biophotonic signaling. It argues that alongside chemical signaling, cells and tissues may also use electromagnetic/biophotonic mechanisms for communication. The article frames biophotons as carriers of energy and information across hierarchical biological levels, including cells, tissues, organs, organ systems, and the whole organism.

The same review describes UPE as a well-studied biophysical phenomenon and proposes that coherence, solitons, and electromagnetic field interactions may help explain cellular electromagnetic communication. It also emphasizes that a complete systemic biological theory remains under development, which is important for scientific balance.

For a white paper or FDA-facing rationale, this article is useful for hypothesis generation, but it should be used conservatively. Its strongest value is to support the concept that UPE may represent a biological communication and regulation phenomenon worth studying. It should not be used alone as proof of clinical efficacy.

Integrated Mechanistic Model

Based on the published literature reviewed, the Following Integrated Model Can Be Proposed:

- **Step 1:** Brain cells generate UPE through metabolism. Neurons, glia, mitochondria, and neural stem cells emit ultraweak photons through redox reactions, mitochondrial respiration, lipid peroxidation, and excited molecular-state relaxation.

- **Step 2:** UPE reflects physiological and pathological state. UPE intensity and dynamics correlate with oxidative stress, mitochondrial activity, cerebral metabolism, EEG activity, cerebral blood flow, glutamate signaling, and neuronal depolarization.
- **Step 3:** UPE can distinguish brain-derived signal from background. Human brain UPE shows distinct temporal, spectral, entropy, and task-related features compared with background photon noise.
- **Step 4:** UPE tracks disease-relevant biochemical dysfunction. In an Alzheimer's disease rat model, hippocampal UPE increased with memory impairment, oxidative stress, MDA, and AChE activity; donepezil reduced UPE and improved biochemical/behavioral measures.
- **Step 5:** UPE relates to regenerative neural biology. Neural stem cells emit UPE, and UPE intensity changes with passaging, differentiation, and AgNP exposure; increased UPE under AgNP conditions was associated with increased neuronal differentiation.
- **Step 6:** Ultraweak photon environments can be scientifically investigated. Because UPE is linked to mitochondrial-redox physiology and brain function, an Ultraweak Photon Environment may be studied as a low-intensity, non-invasive photonic exposure model to evaluate whether external ultraweak photon fields can influence sleep, mitochondrial homeostasis, neurophysiology, inflammation, neuroplasticity, and functional recovery.

Implications for Brain Physiology, Biochemistry and Function

Brain Physiology: The evidence supports that UPE is connected with (1) Neuronal activity, (2) EEG/qEEG oscillations, (3) Cerebral blood flow, (4) Hyperoxia and hypoxia/reperfusion states, (5) Glutamate-induced activity, (6) Calcium-dependent depolarization, (7) Task-related brain states, (8) This makes UPE relevant to brain physiology and functional monitoring.

Brain Biochemistry: The evidence supports that UPE is connected with (1) Mitochondrial respiratory-chain activity, (2) ROS generation, (3) Lipid peroxidation, (4) MDA concentration, (5) AChE activity, (6) Cholinergic dysfunction, (7) ATP-related metabolic stress, (8) Oxidative stress and redox imbalance. This makes UPE relevant to biochemical monitoring and potentially to therapeutic-response assessment.

Brain Function: The evidence supports that UPE is connected with (1) Memory-related hippocampal performance, (2) Neural stem cell differentiation, (3) Human brain task-related states, (4) EEG-correlated brain rhythms, (5) Possible neural optical communication mechanisms, (6) Neurodegenerative disease monitoring. This makes UPE relevant to cognitive and neurofunctional research.

Relevance to Neurodegenerative Disorders

The uploaded literature builds a rational bridge to Alzheimer's disease, Parkinson's disease, ALS, stroke, and traumatic brain injury because these disorders commonly involve mitochondrial dysfunction, oxidative stress, neuroinflammation, impaired cerebral metabolism, and neural-network dysfunction.

Disorder	UPE-Relevant Mechanistic Foundation
Alzheimer's disease	Hippocampal UPE correlated with memory impairment, MDA, AChE, and donepezil response
Parkinson's disease	Mitochondrial dysfunction, oxidative stress, and respiratory-chain impairment are central mechanisms
ALS	Motor neuron degeneration involves mitochondrial and oxidative stress pathways; UPE could be explored as a redox-metabolic marker
Stroke	Hypoxia/reperfusion and oxidative injury increase UPE in brain models
TBI	Mitochondrial dysfunction, ROS, inflammation, and disrupted brain rhythms may be measurable through UPE-related signals

Published studies demonstrate that ultraweak photon emission is a measurable biological phenomenon arising from mitochondrial-redox metabolism and oxidative biochemical reactions. In brain tissue, UPE correlates with neural activity, EEG rhythms, cerebral blood flow, glutamate signaling, hypoxia/reperfusion states, and oxidative stress. Human brain studies show that UPE signals can be distinguished from background photon noise and may provide a passive optical readout of functional brain states. In animal models of Alzheimer's disease, hippocampal UPE correlates with memory impairment, lipid peroxidation, and acetylcholinesterase activity, and decreases following donepezil treatment. Neural stem cell studies further show that UPE changes with passage, differentiation, and neuronal lineage commitment. Together, these findings provide a strong mechanistic foundation for studying ultraweak photon environments as investigational platforms for supporting brain physiology, biochemical homeostasis, and neurological function.

Scientific Limitations and Regulatory Caution

Currently, Scientific Literature Supports UPE as a Biologically Meaningful Signal and Potential Biomarker. However, Several Limitations Remain:

- UPE is extremely low-intensity and technically difficult to measure.
- Background light, delayed luminescence, detector noise, and environmental artifacts must be carefully controlled.
- The role of UPE as a signaling mechanism remains debated.
- Most disease-related studies are preclinical or early-stage.
- UPE measurement does not automatically prove that external ultraweak photon emitters treat disease.
- Clinical claims require prospective, controlled, blinded human studies using validated endpoints.

Therefore, the conclusion is that the published literature provides a strong scientific foundation for UPE as a brain-metabolic, redox, and neurofunctional signal. It also provides a rationale for studying UWPE in neurological disorders and treatment.

Conclusion

This literature base can be used to support a clinical protocol foundation for studying ultraweak photon environments in neurodegenerative and neurological disorders. However, Because endogenous UPE cannot be practically standardized or delivered as an external clinical exposure, development of a stable UWPG system is necessary for research and potential applications. The next critical step is to develop a stronger Ultra-Weak Photon

Generator (UWPG) that can generate a stable and scalable ultra-weak photon environment suitable for larger spaces and broader applications. This enhanced emitter should be designed to maintain non-thermal, low-intensity photon output while providing sufficient field coverage for large-scale Recovery Sleep environments, clinical research settings, wellness centers, and future platform applications.

Ultra-Weak Photon Generator (UWPG) and Ultra-Weak Photon Environment (UWPE)

Technology Overview: Ultraweak Photon Generators

Ultra-Weak Photon Generator (UWPG), also referred to as Biophoton Generator (BPG) technology, is designed to create a durable and stable ultra-weak photon environment (UWPE) for wellness, recovery, research applications, and investigational studies of unmet neurological disorders. This technology is protected by a United States patent invented by Liu and Gu [4]. An UWPG is a non-contact, non-thermal photon-emitting system that produces invisible low-intensity light signals within a biologically relevant optical range 500-1000 nm [5]. When one or more UWPG units are placed in a defined space, they establish a surrounding UWPE, allowing a person to rest, sleep, or recover within a gentle field of ultra-weak photons.

The concept of the UWPE is based on the understanding that living biological systems naturally emit and respond to ultra-weak photons, sometimes referred to as biophotons. These photon signals are commonly associated with cellular metabolism, mitochondrial activity, oxidative-reduction reactions, and biological communication processes, as numerous scientists reported [6-12]. In the body, ultra-weak photon emission is thought to reflect the dynamic energy state of tissues and cells. UWPG technology is intended to reproduce a supportive, external ultra-weak photon environment that may help the body maintain a more favorable state for rest, balance, and recovery.

Recovery Sleep and Characteristics of UWPE

Recovery Sleep is defined as sleeping within a UWPE designed to support the body's natural overnight recovery from daily physical and mental exhaustion. Recovery Sleep is an enhanced sleep experience supported by continuous exposure to UWPE. It is achieved by sleeping within an Ultra-Weak Photon Environment created by ultra-weak photon emitters designed to support the body's natural overnight restoration processes. During sleep, the UWPE is intended to promote a more recovery-supportive environment associated with relaxation, circulation support, mitochondrial activity, and cellular recovery from daily physical and mental exhaustion.

A single UWPG can generate a localized photon environment around the device. Two UWPG units placed on opposite sides of a bed can create a broader photon field in which a person may sleep between the emitters. Four UWPG units arranged at the corners of a square can generate a more balanced and spatially distributed UWPE, with the individual positioned inside the square. This multi-generator arrangement is especially relevant for Recovery Sleep, where the sleeping person remains continuously inside the ultra-weak photon environment throughout the night, as illustrated in Figure 1.

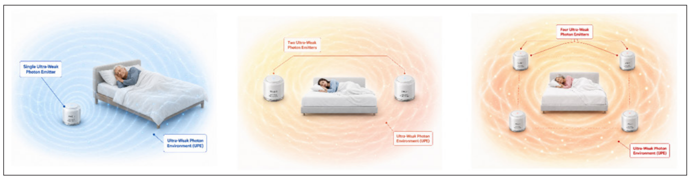

Figure 1: Ultra-Weak Photon Environment (UWPE). This is a schematic illustration of an invisible UWPE generated by Ultra-Weak Photon Emitters (UWEs). The UWEs are positioned around the sleeping area (2 x 2 meters) to create a gentle, continuous photon environment surrounding the individual during sleep. The softly glowing background represents the UWPE, while the emitter devices illustrate the source of ultra-weak photon exposure. This configuration is designed to support Recovery Sleep, in which a person rests within a calm, non-contact, ultra-weak photon environment.

The UWPG platform is not designed to heat tissue, mechanically stimulate the body, or deliver high-intensity light therapy. Instead, it is intended to generate a subtle environmental photon field. This distinguishes UWPG technology from conventional photobiomodulation, such as phototherapy, lasers, infrared lamps, or electrical stimulation devices (Table 1). The exposure is passive and non-contact, meaning the user does not need to wear electrodes, attach sensors, or actively operate the system during sleep. The person simply rests inside the UWPE created by the emitters.

Table 1: Conventional Photobiomodulation vs. Ultraweak Photon Environment

Category	Conventional Photobiomodulation, or PBM	Ultraweak Photon Environment (UWPE) / Biophoton Generators
Basic concept	Uses externally applied red or near-infrared light, usually from lasers or LEDs, to stimulate biological tissues.	Creates a passive ultraweak photon environment using very low-intensity photon emission, intended to surround the body rather than target one tissue area directly.
Light intensity	Typically uses measurable therapeutic light intensity, often delivered at defined power densities and energy doses.	Uses ultraweak photon emission, many orders of magnitude lower than conventional PBM. The exposure is subtle and non-heating.
Energy delivery	Active energy delivery from an electrical light source.	Passive photonic environment; device may not require electricity during normal use, depending on product design.
Contact with body	Often applied directly to the skin or very close to the skin.	Generally non-contact; devices may be placed near the body, around a bed, room, or treatment environment.
Targeting method	Local or regional targeting: scalp, joint, muscle, nerve, wound, or organ area.	Environmental/systemic exposure: intended to create a whole-body ultraweak photon field rather than treat one isolated location.

Typical wavelength	Commonly red and near-infrared wavelengths, often around 600–1100 nm depending on device.	Typically described as broad ultraweak photon emission in visible to near-infrared range, depending on device characterization.
Dose control	Dose is usually defined by wavelength, power, irradiance, fluence, exposure time, pulse parameters, and treatment frequency.	Dose is more difficult to define using conventional PBM parameters because the exposure is environmental, ultraweak, and continuous or semi-continuous.
Primary biological model	Direct tissue photostimulation, often involving mitochondrial chromophores such as cytochrome c oxidase.	Environmental photonic support model, hypothesized to influence biological self-regulation, mitochondrial signaling, redox balance, cellular communication, sleep recovery, and systemic resilience.
Mechanism of action	Usually described as light absorption by cellular chromophores, improved mitochondrial ATP production, nitric oxide release, reduced oxidative stress, and modulation of inflammation.	Proposed mechanism is broader and less established: ultraweak photon interaction with living systems, mitochondrial normalization, redox signaling, biofield-like environmental support, microcirculation, autonomic balance, and sleep-mediated recovery.
Brain application	Transcranial PBM applies red/NIR light to the scalp to target brain tissue, though penetration is limited and variable.	Ultraweak photon environment is not designed specifically to force light through the skull at high intensity; it is proposed to support brain function indirectly and systemically through whole-body exposure, sleep, circulation, and cellular signaling.
Whole-body application	Usually requires treating multiple body sites or using large PBM panels/beds.	Whole-body exposure is built into the environmental concept; the body rests inside or near the ultraweak photon field.
Treatment experience	Usually a session-based treatment, such as 10–30 minutes per area.	Often used as a longer-duration exposure environment, especially during rest or overnight sleep.
Thermal effect	Designed to be non-thermal, but high-output devices must be controlled to avoid heating or eye risk.	Non-thermal by design due to ultraweak output. No high-intensity visible or laser radiation is emitted; formal eye-safety characterization remains important.
Eye safety considerations	Eye protection may be needed for some laser or high-powered LED systems.	Eye risk is expected to be low because emission is ultraweak, but formal safety characterization is still important.
User burden	Requires active sessions, correct positioning, time scheduling, and sometimes operator training.	Lower user burden; devices can be placed around the user or sleep environment with minimal or nonactive participation.
Brain-function potential	May support cognition, mood, brain metabolism, neuroinflammation modulation, and recovery in selected research settings.	May support brain function through reported improvements in sleep, energy, cognition, motor function, functional stability, and quality of life; evidence remains investigational for disease claims.
Whole-body-function potential	May support pain, inflammation, muscle recovery, wound healing, and local tissue repair depending on indication and device.	May support whole-body wellness through sleep quality, energy, recovery, comfort, circulation, relaxation, and systemic resilience. Disease-related benefits require clinical validation.
Best-fit use model	Local or regional therapeutic light treatment.	Systemic environmental wellness or investigational neurological support platform.
Strengths	Well-studied field; measurable parameters; many published studies; clear engineering controls.	Non-invasive, non-contact, low-burden, whole-body exposure, potentially suitable for long-duration use during sleep or rest.
Limitations	Penetration depth, dosing variability, local targeting limitations, need for repeated sessions, operator/device variability.	Mechanism is less established; the dose metrics are less conventional; clinical evidence is early; requires rigorous controlled studies in large populations.
Regulatory status	Depends on device claims and indication; some PBM devices have FDA clearances for specific uses such as pain or inflammation-related indications.	Disease-related uses should be treated as investigational unless cleared, approved, or otherwise authorized by FDA. General wellness positioning must avoid disease-treatment claims.
Clinical evidence maturity	More mature than ultraweak photon environmental technology, with broader published literature.	Emerging evidence, including observational studies, pilot studies, and registered clinical trials; stronger sham-controlled studies are needed.
Ideal clinical research endpoints	Local pain scores, wound healing, inflammation markers, cognition scales, motor tests, imaging, qEEG, functional outcomes.	Sleep, fatigue, quality of life, qEEG, ALSFRS-R, MDS-UPDRS, ADAS-Cog/MoCA, Fugl-Meyer, neurofilament light, inflammatory markers, functional independence, adverse events.
Core positioning	"Targeted light therapy."	"Whole-body ultraweak photon environment."
Simplified explanation	PBM shines therapeutic light onto a specific body area.	Ultraweak photon emitters create a gentle photon-rich environment around the body, often during rest or sleep.

From a technology-development perspective, the strength, stability, distribution, and reproducibility of the UWPE are central design goals. A stronger UWPG may be required for larger spaces, wellness rooms, clinical research environments, hospital rooms, senior living facilities, hospitality Recovery Sleep rooms, and other large-scale applications. However, increasing system strength should be balanced with the core safety principles of ultra-weak photon technology: low intensity, non-thermal output, passive exposure, and stable environmental coverage.

Validation of Biophoton Generation Systems
Overview of Validation Strategy
A multi-layered validation framework was implemented to establish: (1) physical emission validity (instrument-based detection); (2) reproducibility and stability (batch + temporal consistency); (3) biological interaction plausibility (human signal modulation); (4) Clinical efficacy (randomized controlled trial). This approach ensures cross-domain validation, integrating (1) Physics; (2) Engineering; (3) Biophotonics; (4) Clinical medicine.

Device Description
The Ultra-Weak Photon Generator (UWPG) is a non-electricity-powered, solid-state system designed to emit ultra-low-intensity photons within the Biologically Relevant Spectral Range:
- 500–1000 nm (visible to near-infrared)
- Emission intensity: ultra-weak (instrument-detectable only)

The emission profile overlaps with known biological ultra-weak photon emission (UPE) ranges.

Instrumental Validation (Four Independent Systems)
(3.1) Multispectral Imaging (MIRA + Phaedra System)
Purpose: (1) Detect spatial photon emission patterns; (2) Differentiate active vs inactive devices.
Method: (1) Dark-room imaging (202 frames / ~40 sec); (2) FFT-enhanced signal processing; (3) Pixel-level statistical analysis.

Key Findings:
- Active devices showed:
  - o 72–79% active pixels vs ~14% background
  - o Significantly higher entropy and signal complexity
- Clear separation between:
  - o Active devices
  - o Inactive controls
  - o Background noise

This demonstrates non-random structured photon emission.
(3.2) Single-Photon Counting (Quantum-Level Detection)
Purpose: (1) Confirm photon emission at ultra-low intensity; (2) Establish temporal stability.
Method: (1) Photomultiplier-based photon counting; (2) Longitudinal monitoring across batches.

Key Findings
- Continuous photon emission detected
- Stable across multi-year production batches
- No significant degradation over time

Confirms true ultra-weak photon emission (not noise)
(3.3) Emission Spectroscopy (Horiba Duetta System and Horiba iSpec system)
Purpose: (1) Quantify spectral distribution; (3) Compare active vs inactive devices
Method: (1) Dark-corrected emission spectra (350–850 nm); (2) Blocked excitation → ensures spontaneous emission.

Key Findings: Active devices show (1) Stronger emission in 600–850 nm range; Peaks in 630–700 nm region, and overall range is 500 to 1000 nm. Inactive devices: (1) Lower intensity; (2) Sparse, irregular spectral profile. This confirms biologically relevant spectral emission 500 – 1000 nm [5].
(3.4) Radiometric & Field-Level Detection
Purpose: (1) Quantify emission energy; (2) Detect spatial field structure
Method: (1) Radiometric conversion (counts → W/cm²); (2) Field visualization (MIRA + modeling).

Key Findings: (1) Emission intensity: $\sim 10^{-17}$ to 10^{-19} W/cm² range (biophoton scale); (2) Toroidal field structures observed; (3) Emission penetrates metal enclosure. The measurements suggest non-trivial emission behavior requiring further modeling.

Cross-Instrument Validation
All four systems independently confirm:

Parameter	Result
Emission presence	Confirmed
Spectral range	500–1000 nm
Intensity	Ultra-weak
Reproducibility	High
Active vs control separation	Significant

This satisfies instrumental triangulation (gold standard in physics validation).

In addition, The laboratory methodologies were reviewed by an expert at NIST and considered appropriate.

Human Interaction Validation
(5.1) Acute Biophoton Response
Using MIRA imaging: (1) Increased photon emission from human subjects; (2) Observed within 10–30 minutes of exposure. This suggests bi-directional photonic interaction.

Randomized Controlled Clinical Trial (RCT Validation)
(6.1) Study Design
- Type: Randomized, double-blind, placebo-controlled
- Participants: n = 71 adults
- Duration: 14–28 days
- Blinding: Triple-blinded (participant, investigator, analyst)

(6.2) Intervention
- Active device vs placebo (identical appearance)
- ≥8 hours daily exposure

(6.3) Primary Biological Endpoint
Stem Cell Mobilization
Measured via flow cytometry:
- $CD34^+$
- $CD133^+$
- $CD34^+CD133^+$

Clinical Outcomes
Biological Results
- $CD34^+$: ~2× increase
- $CD133^+$: ~3.5× increase
- $CD34^+CD133^+$: ~3× increase
- $p < 0.01$ (highly significant)

Functional Outcomes
- SF-36 improved 16–30%
- Pain Disability Index significantly reduced ($p < 0.00001$)
- Safety: No adverse events were reported, and compliance was high.

Statistical Validation
- ANOVA: significant across all endpoints
- Paired t-tests:
  - o p < 0.001 across stem cell populations

Meets clinical significance + statistical rigor [13].

Integrated Validation Model
Multi-Layer Validation Pyramid
Level 4: clinical efficacy (RCT)
Level 3: Biological Interaction (human photon response)
Level 2: Instrumental Validation (4 systems)
Level 1: Physical Emission (spectral + photon detection)

Strength of Evidence

Domain	Strength
Physics	Strong (multi-instrument)
Engineering	Strong (repeatability)
Biology	Moderate–Strong
Clinical	Preliminary to moderate; supported by early RCT evidence, requiring larger independent trials

Scientific Interpretation
The validation demonstrates (1) The device emits measurable ultra-weak photons. (2) emission lies within biologically relevant optical windows, and the emission is stable, reproducible, and distinguishable from controls. (3) Exposure is associated with biological changes (stem cells) and clinical improvements.

Summary
A systematic validation framework combining four independent physical instruments and a randomized controlled clinical trial demonstrates that the ultra-weak photonic emission device (1) Produces measurable photon emission in the 500–1000 nm range, (2) Exhibits consistent, reproducible emission profiles, (3) Interacts measurably with biological systems, (4) Produces statistically significant clinical effects in humans. This multi-domain validation supports the device as a novel non-invasive photonic modality warranting further investigation in translational and clinical medicine.

Sleep and Recovery
Sleep is essential for brain repair, glymphatic clearance, memory consolidation, immune balance, and neuroplasticity [14-18]. Although direct studies of ultra-weak photon environments during sleep remain limited, published evidence shows that endogenous human UPE follows circadian and diurnal rhythms, is linked to metabolism and oxidative-redox biology, and can be detected from the human brain. These findings provide a scientific rationale for investigating UWPE exposure during sleep as a potential neurophysiological recovery environment [19-24].

UWPE may promote deep sleep by creating a stable, low-intensity biological environment during the body's natural overnight recovery period. Unlike conventional photobiomodulation, which often uses localized and higher-intensity light exposure for short periods, UWPE is designed for continuous, passive, whole-body exposure during sleep. This gentle environmental exposure may help the nervous system shift away from daytime stress responses and toward a more relaxed parasympathetic state, supporting the transition into deeper, more restorative stages of sleep.

Deep sleep is strongly linked to mitochondrial recovery, cellular repair, immune balance, and brain detoxification through glymphatic clearance. Because ultra-weak photons are closely associated with mitochondrial activity, redox signaling, and cellular communication, sleeping inside a UWPE may help support more coordinated cellular regulation during the night. In this view, UWPE does not "force" sleep like a sedative; rather, it may provide a supportive recovery environment that allows the body's own sleep-regulating systems to function more efficiently.

A deeper sleep experience may also arise from reduced physiological burden during the night. When the body experiences less oxidative stress, better energetic balance, and improved cellular communication, the brain may spend less time in fragmented or shallow sleep and more time in slow-wave restorative sleep. Patients and wellness users who report improved sleep after exposure to biophoton generators may therefore be experiencing the secondary benefits of a more stable recovery environment: better relaxation, improved overnight restoration, clearer morning energy, and enhanced daytime cognitive and physical performance.

The next generation of UWPG systems should therefore focus on scalable field design. Important development parameters include photon output characterization, wavelength range, spatial distribution, emitter placement, field uniformity, durability, batch-to-batch consistency, and quality control testing. A well-designed UWPG system should be capable of producing a reliable UWPE across different room sizes while maintaining a gentle, biologically compatible exposure profile.

Sleeping inside the ultra-weak photon environment (UWPE) reduced the functional brain age of patients with Parkinson's disease (PD) and Alzheimer's disease (AD), as demonstrated by qEEG-based functional brain age analysis [25,26]. After Recovery Sleep exposure inside the UWPE, patients showed more normalized brain activity patterns, including improved brain-network regulation and a shift away from abnormal fast-wave or dysregulated activity toward a younger, more organized qEEG profile. These findings suggest that UWPE-supported sleep may help restore brain physiological balance through improved sleep quality, mitochondrial-redox regulation, neurovascular support, and reduced neuroinflammatory burden, thereby contributing to measurable improvement in functional brain age in both PD and AD patients.

In summary, the UWPG is the source technology, while the UWPE is the resulting exposure field created around the user. Together, UWPG and UWPE form platform technology for creating Recovery Sleep environments and broader wellness-oriented spaces (Figure 2). By moving from single-emitter systems to multi-emitter and stronger large-scale configurations, UWPG technology may provide a foundation for future sleep, recovery, senior wellness, neurological wellness research, and large-room environmental applications.

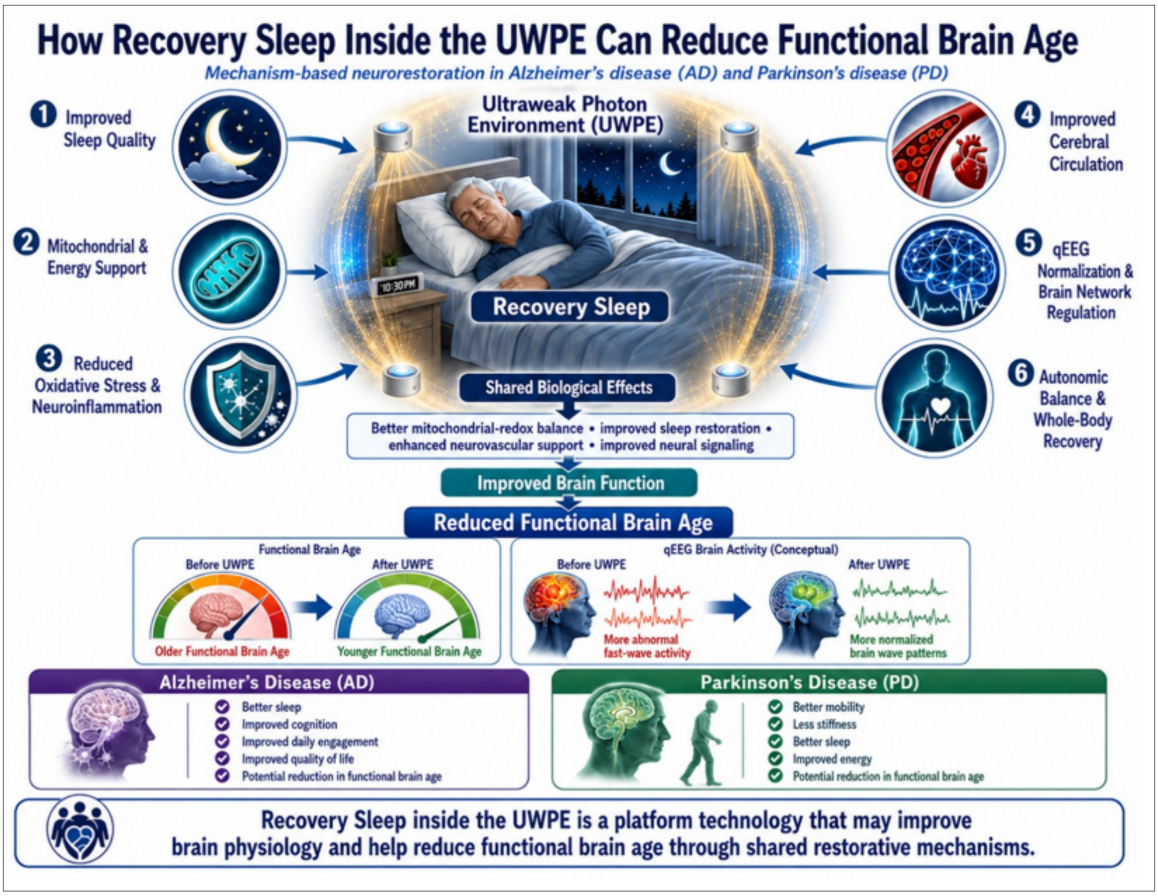

Figure 2: Proposed mechanism by which Recovery Sleep inside an ultra-weak photon environment (UWPE) may reduce functional brain age in Alzheimer's Disease (AD) and Parkinson's Disease (PD). Continuous overnight exposure to UPE may support deeper sleep, mitochondrial energy balance, reduced oxidative stress and neuroinflammation, improved cerebral circulation, qEEG brain-network normalization, autonomic balance, and whole-body recovery. These shared restorative mechanisms may improve brain function and contribute to a younger functional brain-age profile, with potential benefits in cognition, mobility, energy, sleep quality, and overall quality of life.

Why Brain Function May Respond to Ultraweak Photon Environment

The brain is one of the most energy-demanding organs in the body. Neurons depend on mitochondrial energy production, balanced redox signaling, adequate blood flow, controlled inflammation, and precise communication across neural networks. UWPG may be relevant because the biological processes involved in brain disease overlap with processes known to influence UPE.

Mitochondrial Function: Mitochondria generate cellular energy and regulate oxidative stress. In neurodegenerative disease, mitochondrial dysfunction contributes to neuronal vulnerability. A biophoton-based intervention may theoretically influence mitochondrial signaling, redox balance, and cellular energy regulation. Recent studies support a close relationship between ultra-weak photon emission and mitochondrial-redox biology, including mitochondrial respiration, oxidative phosphorylation, ROS generation, cellular energy state, and stress metabolism, suggesting that UPE may serve as a non-invasive optical biomarker of mitochondrial activity [27-34].

Oxidative Stress: Ultraweak photon emission is often associated with oxidative metabolic reactions. In brain disorders, oxidative stress is a major driver of cellular injury. Monitoring and potentially modulating photonic biological signals may offer a new way to study or support stressed tissues. Published studies support that UPE is closely associated with oxidative metabolic reactions, ROS generation, lipid peroxidation, and oxidative stress responses, suggesting that UPE may serve as a non-invasive optical indicator of redox and oxidative-stress activity in biological systems [35-41].

Neuroinflammation: Neuroinflammation contributes to Parkinson's disease, Alzheimer's disease, ALS, stroke injury, and TBI. If ultraweak photonic exposure can influence inflammatory signaling indirectly through mitochondrial, autonomic, or systemic regulatory pathways, it may help support functional recovery. Although direct studies of UPE as a biomarker of neuroinflammation remain limited, published evidence supports a strong mechanistic connection among UPE, ROS generation, mitochondrial dysfunction, oxidative stress, inflammatory metabolism, and neurodegenerative brain pathology. Together, these findings provide a scientific rationale for investigating brain-related UPE as a potential optical marker of neuroinflammatory and neurodegenerative processes [22,35, 42-44].

Neural Communication: Emerging research is exploring whether ultraweak light may play a role in neuronal signaling. Published studies suggest that neural tissues emit ultraweak photons and that these biophotonic signals may be associated with neuronal metabolism, glutamate activity, axonal transmission, brain-network activity, and potential cell-to-cell communication. However, photon-mediated neural communication remains an emerging and still-developing field requiring further experimental confirmation [45-52].

Sleep and Recovery: Sleep is essential for brain repair, glymphatic clearance, memory consolidation, immune balance, and neuroplasticity. Many patients using biophoton generators have reported improved sleep, which may secondarily improve cognition, energy, mood, and motor performance. Recovery Sleep is defined as sleeping within an ultraweak photon environment designed to support the body's natural overnight recovery from daily physical and mental exhaustion. Recovery Sleep is an enhanced sleep experience supported by continuous exposure to a UWPE. UWPE is generated by one or more UWPGs (Figure 1).

This differs from conventional photobiomodulation (PBM), which typically uses localized, higher-intensity light [12]. Instead, Recovery Sleep inside a UWPE emphasizes (1) Whole-body environmental modulation, (2) Ultra-low intensity exposure, (3) Prolonged duration during sleep cycles.

Clinical Study Outcomes of Recovery Sleep Inside a UWPE
Introduction: The Unmet Need in Brain Disorders
Neurological diseases remain among the most difficult conditions to treat. Parkinson's disease, Alzheimer's disease, ALS, chronic stroke, and traumatic brain injury differ clinically, but they share several common biological features:
- Progressive or persistent loss of neurological function
- Impaired mitochondrial energy metabolism
- Oxidative stress
- Neuroinflammation
- Disrupted neural signaling
- Reduced neuroplasticity
- Decline in motor, cognitive, speech, sleep, or daily-life function

Parkinson's disease, Alzheimer's disease, chronic stroke, traumatic brain injury, and ALS are among the most difficult neurological conditions to treat. Parkinson's disease is a progressive neurodegenerative disorder involving motor and non-motor symptoms, and NINDS notes that some Parkinson's disease gene variations affect cellular energy production and oxidative stress pathways [6]. Alzheimer's disease remains progressive, and NINDS states that there are no treatments that can stop its progression, although some drugs may temporarily slow symptom worsening [7]. For traumatic brain injury, long-term consequences may include cognitive dysfunction, pain, sleep disorders, and neurobehavioral symptoms [9]. ALS is a fatal motor neuron disease with no cure and no treatment capable of halting or reversing progression for most patients [10].

These diseases are especially challenging because neurons and glial cells depend on high energy production, mitochondrial integrity, vascular support, redox balance, and precisely regulated network communication. When these systems fail, patients may experience worsening memory, movement, speech, swallowing, balance, breathing, sleep, mood, and daily function. This creates an urgent need for safe and non-invasive approaches that may support the brain's own recovery capacity and functional resilience.

Parkinson's Disease
Parkinson's disease involves degeneration of dopaminergic neurons, motor dysfunction, autonomic symptoms, sleep disturbance, fatigue, mood changes, and cognitive decline. Early biophoton therapy reports have described improvements in both motor and non-motor symptoms. A 2025 Parkinson's disease study listed in research databases evaluated Recovery Sleep within an Ultraweak Photon Environment, or UPE, created by automatic biophoton generators, and reported preliminary improvements in motor and non-motor symptom domains [53-56]. These findings should be considered investigational and require confirmation in larger controlled studies.
Reported domains of improvement may include:
- Better walking stability
- Reduced stiffness
- Improved hand movement
- Better sleep
- Improved energy
- Improved quality of life
- Improved EEG-derived brain functional measures

The proposed explanation is not that ultraweak photon emitters replace dopaminergic therapy, but that they may support brain-network regulation, mitochondrial function, sleep quality, and systemic recovery capacity.

The scientific rationale for ultraweak photon emitters in neurological disorders is based on the relationship between light-sensitive biology, mitochondrial metabolism, oxidative stress, and neural communication. Ultraweak photon emissions are associated with metabolic and oxidative processes, and brain tissue is highly metabolically active [1,3]. This creates a plausible mechanistic bridge between photonic biology and neurological function.

Alzheimer's Disease and Dementia
Alzheimer's disease is characterized by progressive cognitive decline, memory impairment, synaptic dysfunction, neuroinflammation, and loss of daily-life independence. NINDS states that current Alzheimer's disease treatments cannot stop disease progression, although some drugs may temporarily slow worsening of symptoms [7]. Broader dementia research also confirms that there are currently no cures for neurodegenerative dementias [8].

Ultraweak photon emission has been investigated in brain tissue and Alzheimer's-related models. One study reported ultraweak photon emission from rat hippocampus and explored its relationship to Alzheimer's-related biological changes [57]. A registered ClinicalTrials.gov study is evaluating biophoton therapy in brain disorders including Alzheimer's disease, dementia, Parkinson's disease, stroke, and traumatic brain injury using a randomized, triple-blinded, placebo-controlled design [58, 56, 59-62].

Interpretation: The Alzheimer's disease rationale is strongest when framed around brain energy metabolism, sleep restoration, neurovascular support, and network regulation. Ultraweak photon emitters should not be described as removing amyloid or tau unless specific evidence supports that claim. At this stage, the evidence supports further clinical investigation, not definitive therapeutic claims.

Chronic Stroke
Chronic stroke patients may experience persistent weakness, impaired coordination, speech difficulty, balance impairment, fatigue, and reduced independence. Many patients improve with rehabilitation, but functional recovery may plateau months or years after the stroke. Stroke recovery depends on neuroplasticity, motor relearning, vascular support, mitochondrial function, and repeated functional training. A ClinicalTrials.gov study titled "Impact of Biophoton Generator on Chronic-Stroke Patients' Functional Recovery" was designed to evaluate whether chronic stroke patients could regain independent living ability after daily use of biophoton generators [63]. Another listing for NCT06049849 describes a completed study evaluating whether participants with chronic stroke could regain living independence through daily exposure to a biophoton generator [64, 65-68].

Interpretation: Chronic stroke may be a strong indication for future controlled study because functional endpoints are measurable. Useful endpoints include Fugl-Meyer Assessment, modified Rankin Scale, gait speed, grip strength, balance testing, activities of daily living, qEEG, and quality-of-life measures.

Traumatic Brain Injury

Traumatic brain injury can produce persistent symptoms including headache, dizziness, fatigue, cognitive dysfunction, sleep disorders, mood changes, balance problems, and impaired quality of life. NINDS notes that TBI can have long-term sequelae including cognitive dysfunction, pain, and sleep disorders [9]. A 2023 JAMA Network Open study also emphasized that TBI recovery can be dynamic and may continue for years, challenging the assumption that recovery ends after the early post-injury period [69].

A 2025 pilot study in retired athletes with chronic traumatic brain injury symptoms evaluated non-invasive biophoton therapy and reported improvements in neurocognitive, physical, emotional, quality-of-life, and EEG-related outcomes. The study provides hypothesis-generating evidence but requires confirmation in controlled trials with sham devices, blinded assessments, validated neuropsychological testing, and independent data analysis [70,71].

Interpretation: TBI is scientifically relevant because mitochondrial injury, oxidative stress, neuroinflammation, sleep disturbance, and network dysregulation are all major post-injury mechanisms. Ultraweak photon emitters may be investigated as a low-risk adjunctive recovery environment, especially during sleep.

Amyotrophic Lateral Sclerosis

ALS is a rare, progressive, fatal motor neuron disease that causes loss of motor function, speech, swallowing, and breathing capacity. NINDS states that there is no cure for ALS and no effective treatment to halt or reverse progression [10]. The unmet need is therefore profound.

A 2026 observational case series described three individuals with ALS using biophoton generators and reported patient-observed themes including perceived improvements in sleep, circulation, energy, bulbar function, and functional stability [16]. The authors appropriately noted that ALS must be approached with caution and that the uncontrolled design prevents conclusions about causality or clinical efficacy [72].

Interpretation: ALS evidence remains at the earliest hypothesis-generating stage. Future ALS studies should include ALSFRS-R, forced vital capacity, grip strength, bulbar function, neurofilament light chain, EMG, sleep and fatigue scales, adverse-event tracking, and sham-controlled design. No claim of ALS treatment, reversal, or disease modification should be made without controlled evidence.

The brain is especially vulnerable to mitochondrial dysfunction and oxidative stress. In Parkinson's disease, mitochondrial dysfunction and oxidative stress are widely discussed as central contributors to neurodegeneration [6,73]. In Alzheimer's disease and dementia, impaired metabolism, synaptic dysfunction, neuroinflammation, and progressive network failure contribute to cognitive decline [7,8]. In TBI and stroke, tissue injury, inflammation, impaired perfusion, and altered neuroplasticity may limit recovery [9,69]. In ALS, motor neuron degeneration may involve mitochondrial dysfunction, oxidative injury, neuroinflammation, excitotoxicity, and impaired axonal transport [10].

Ultraweak photon emitters may therefore be investigated as a systems-level neurofunctional support technology. The proposed mechanism is not a single drug-like receptor target, but rather a multi-pathway effect involving mitochondrial normalization, redox regulation, sleep improvement, autonomic balance, microcirculatory support, and neuroplasticity.

Summary of Clinical Studies

Ultraweak photon emitters represent a novel, non-invasive photonic platform with early reported signals of improved brain-related function across Parkinson's disease, Alzheimer's disease, chronic stroke, traumatic brain injury, and ALS. The biological rationale is based on the known relationship between ultraweak photon emissions, cellular metabolism, oxidative stress, mitochondrial function, and possible cell-to-cell communication [1-3].

The most defensible scientific position is that these disorders share common biological vulnerabilities, including mitochondrial dysfunction, oxidative stress, inflammation, impaired sleep, disrupted circulation, and altered neural communication. Ultraweak photon emitters may support the body's recovery systems through a low-risk photonic environment. However, evidence remains preliminary, and controlled clinical studies are required before definitive claims can be made.

At this stage, the evidence is promising but preliminary. The strongest next step is a rigorous, prospective, sham-controlled clinical trial using validated neurological endpoints, quantitative EEG, neurofilament biomarkers, functional scales, sleep metrics, motor assessments, and safety monitoring.

If future sham-controlled trials confirm measurable improvements in validated neurological endpoints, ultraweak photon emitters could become an important new platform in neurofunctional recovery and supportive neurological care.

Proposed Mechanism of Action of UWPE

Ultraweak photon emitters may support brain function through a multi-system biological pathway:

Photonic Environmental Exposure

The device creates a low-intensity photonic environment around the user, often during rest or sleep. Because sleep is a critical recovery period for the brain, nighttime exposure may be particularly relevant for neurological function.

Cellular and Mitochondrial Signaling

Biological tissues naturally emit ultraweak photons associated with cellular metabolism and oxidative processes [1,2]. Since mitochondria are central to energy production and redox balance, ultraweak photon exposure may plausibly interact with mitochondrial and oxidative signaling pathways, although this mechanism remains under investigation.

Redox and Inflammatory Regulation

Oxidative stress and neuroinflammation are shared mechanisms across Parkinson's disease, Alzheimer's disease, stroke, TBI, and ALS [6-10,73]. A photonic environment that helps normalize redox signaling or reduce inflammatory burden could theoretically support neuronal resilience.

Neurovascular and Autonomic Support

Brain recovery depends on blood flow, oxygen delivery, autonomic regulation, and microcirculatory function. Improvements in sleep, autonomic balance, and circulation may indirectly support neurological function.

Network-Level Brain Function

Because the brain is a network organ, small improvements in energy metabolism, sleep quality, inflammation, and circulation may translate into measurable improvements in cognition, motor function, speech, balance, and quality of life. Quantitative EEG may be useful for detecting changes in brain-network function [74-77].

Sleep-Mediated Brain Repair

Improved sleep may enhance memory consolidation, waste clearance, inflammatory balance, and daytime neurological performance. These mechanisms of action are summarized in Figure 3.

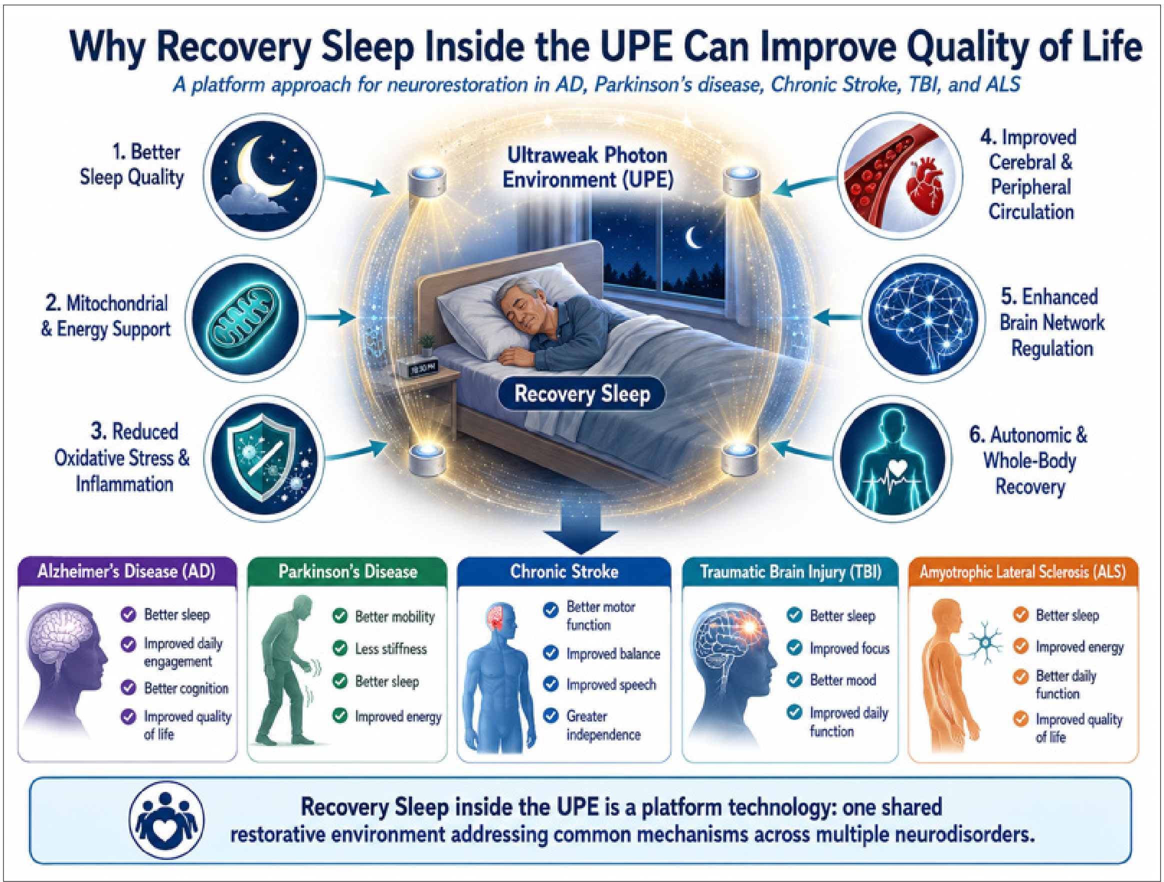

Figure 3: Proposed mechanism by which Recovery Sleep inside an Ultra-Weak Photon Environment (UWPE) may improve quality of life across multiple neurological disorders. This illustration presents Recovery Sleep inside the UPE as a platform-based neurorestorative approach for Alzheimer's disease (AD), Parkinson's disease (PD), chronic stroke, traumatic brain injury (TBI), and amyotrophic lateral sclerosis (ALS). During sleep, ultraweak photon emitters positioned around the bed create a passive, non-contact, non-thermal photonic environment. This environment is proposed to support six shared mechanisms: improved sleep quality, mitochondrial energy support, reduced oxidative stress and inflammation, improved circulation, enhanced brain-network regulation, and autonomic whole-body recovery. The lower panels summarize potential quality-of-life benefits across disorders, including improved cognition and daily engagement in AD; better mobility, sleep, and energy in PD; improved motor function, balance, speech, and independence in chronic stroke; better focus, mood, and daily function in TBI; and improved sleep, energy, daily function, and quality of life in ALS. Overall, the figure highlights Recovery Sleep inside the UPE as a platform technology targeting shared restorative mechanisms across neurological disorders.

Why a Platform Approach Matters

These neurological disorders differ in diagnosis, but they share common biological stress pathways. That overlap supports the concept of ultraweak photon emitters as a neurofunctional support platform rather than a single-disease drug.

Disorder	Major Functional Loss	Shared Biological Themes	Relevant Study Endpoints
Parkinson's disease	Movement, sleep, autonomic function, cognition	Mitochondrial dysfunction, oxidative stress, network disruption	MDS-UPDRS, sleep scale, gait, qEEG
Alzheimer's disease	Memory, cognition, daily function	Synaptic dysfunction, neuroinflammation, metabolic stress	ADAS-Cog, MoCA, caregiver scale, qEEG
Chronic stroke	Motor function, speech, independence	Neuroplasticity limitation, impaired circulation, tissue injury	Fugl-Meyer, gait speed, grip strength

TBI	Cognition, mood, sleep, fatigue	Neuroinflammation, mitochondrial injury, network disruption	Neurocognitive testing, sleep, qEEG
ALS	Motor neurons, speech, swallowing, breathing	Mitochondrial dysfunction, oxidative stress, neuroinflammation	ALSFRS-R, FVC, EMG, neurofilament light

Safety and Tolerability Considerations

General Human Exposure Experience

To date, the company has accumulated substantial observational experience from over 50,000 users with Ultra-Weak Photon Generator technology in wellness-center and ambient-use environments. These observations have primarily involved adults and elderly individuals exposed to UWPGs in passive, non-contact, non-thermal environmental settings.

Based on internal monitoring, user feedback, and center-level observations, no safety signals have been identified to date suggesting device-related risk of:

- burns or thermal injury;
- overheating of the surrounding environment;
- skin irritation, skin injury, or contact-related injury;
- eye or retinal injury;
- respiratory suppression or breathing difficulty;
- seizure induction or neurological overstimulation;
- arrhythmia or acute cardiovascular instability;
- electrical shock or electrical injury;
- infection risk from device contact;
- or device-related toxicological concerns.

The preliminary safety profile is supported by the basic operating characteristics of UWPG technology. The device is designed to generate an ultra-weak photon environment rather than deliver concentrated heat, electrical current, ionizing radiation, mechanical force, drugs, or invasive energy into the body. The exposure is passive, ambient, non-contact, and non-thermal. These characteristics substantially reduce many conventional risks associated with active medical devices, including thermal burns, electrical injury, tissue penetration, drug toxicity, and infection related to direct patient contact.

These observations are preliminary and observational in nature. They do not substitute for formal clinical safety testing, controlled clinical trials, or regulatory review. However, the absence of observed acute safety concerns across broad real-world wellness exposure provides an initial safety foundation for further structured evaluation of UWPG technology.

Neonatal Observational Experience

A limited informal neonatal observational experience was reported by Ms. J.W., RN, an experienced senior nurse, reported that she independently placed small UWPG devices in the surrounding environmental space near her newborn grandchildren for observational wellness-support purposes.

According to Ms. J.W.'s observations, device presence was not associated with any immediately apparent adverse effects. Specifically, she reported:

- no visible skin irritation;
- no abnormal heat generation;
- no respiratory distress;
- no feeding intolerance;
- no observable behavioral distress;
- no signs of discomfort associated with device presence;
- and no immediate safety concerns requiring device removal or medical intervention.

These observations are anecdotal, informal, and non-controlled. They were not conducted under a formal clinical protocol, were not randomized, were not blinded, and were not designed to establish either safety or efficacy. Therefore, no definitive conclusions regarding neonatal safety or therapeutic benefit can be drawn from these observations alone.

In addition, one individual case report involving use of UWPG/UWPE technology in an infant with hypoxic-ischemic encephalopathy was provided to the FDA Office of Orphan Products Development. That report described apparent safe use and favorable clinical observations; however, as a single case report, it should be interpreted cautiously. It does not establish general safety or effectiveness and should be considered only as preliminary supportive information for further FDA-guided evaluation.

Preliminary Safety Risk Assessment

The UWPG system is designed as an environmental ultra-weak photon exposure technology rather than as an invasive, implantable, drug-delivery, electrical-stimulation, heating, or high-intensity radiation device. This design creates several potentially favorable safety characteristics, particularly when considering vulnerable populations such as neonates.

Safety Parameter	UWPG / UWPE Environmental Safety Characteristics
Thermal injury risk	Expected to be minimal because the system is designed for non-thermal operation
Electrical injury risk	Expected to be minimal during passive use because the exposure does not require electrical current delivery to the body
Invasive exposure risk	None; the system does not penetrate tissue and does not require implantation
Drug toxicity risk	None; the system does not administer a drug, biologic, or chemical agent
Infection risk from device contact	Expected to be minimal because use is non-contact and environmental
Radiation exposure risk	No ionizing radiation is emitted
Mechanical injury risk	Expected to be minimal because the system does not apply mechanical force to the body
Skin injury risk	Expected to be minimal because direct skin contact is not required
Respiratory suppression risk	No known mechanism for respiratory suppression has been identified based on the passive environmental design
Sedation requirement	None; use does not require anesthesia, sedation, or restraint

Eye safety concern	Expected to be low because the technology does not emit high-intensity visible or laser radiation; continued evaluation is recommended
Toxicological concern	Expected to be low because exposure is environmental and non-contact; material safety should be addressed through device construction and quality controls

Overall, the preliminary safety assessment suggests that UWPG technology may present a low-risk environmental exposure profile when used as intended. The major safety advantages are its passive operation, ultra-low intensity, non-contact use, non-thermal nature, lack of ionizing radiation, absence of drug delivery, and lack of invasive interaction with the body.

For neonatal or other highly vulnerable populations, however, additional caution is warranted. Future safety evaluation should include formal risk analysis, device characterization, environmental exposure measurements, thermal testing, material safety review, electromagnetic compatibility assessment where applicable, adverse-event monitoring, and FDA-reviewed clinical protocols before any definitive safety or effectiveness claims are made.

Recommended Clinical Development Program
The next stage should move from observational reports and early feasibility studies to prospective, randomized, blinded, sham-controlled clinical trials. The trial design should include standard-of-care background therapy, pre-specified primary and secondary endpoints, independent safety monitoring, and objective biomarkers.

Recommended shared endpoints include qEEG, sleep quality, fatigue, quality of life, cognitive testing, motor testing, digital activity tracking, caregiver-reported outcomes, and adverse-event monitoring. Disease-specific endpoints should include MDS-UPDRS for Parkinson's disease, ADAS-Cog or MoCA for Alzheimer's disease, Fugl-Meyer Assessment for stroke, validated neurocognitive batteries for TBI, and ALSFRS-R plus respiratory measures for ALS.

For biomarker strategy, neurofilament light chain may be particularly important in neurodegenerative disease and ALS, while inflammatory markers, oxidative stress markers, heart-rate variability, sleep architecture, cerebral blood flow, and qEEG may help characterize mechanism and response.

A strong clinical program should move from observational reports to rigorous controlled studies.
Study Design
Recommended design:
- Prospective
- Randomized
- Double-blind or triple-blind
- Sham-controlled
- Multi-center if possible
- Standard-of-care background therapy allowed
- Pre-specified statistical analysis
- Independent safety monitoring

Core Brain Function Endpoints
Recommended Shared Endpoints Across Neurological Studies:
- Quantitative EEG
- Sleep quality index
- Fatigue scale
- Quality-of-life scale
- Cognitive function testing
- Motor function testing
- Digital activity tracking
- Caregiver-reported outcomes
- Blood biomarkers of inflammation and neurodegeneration

Disease-Specific Endpoints

Disease	Suggested Endpoints
Parkinson's disease	MDS-UPDRS, gait analysis, tremor assessment, sleep scale, qEEG
Alzheimer's disease	ADAS-Cog, MoCA, caregiver scale, activities of daily living, qEEG
Chronic stroke	Fugl-Meyer Assessment, modified Rankin Scale, gait speed, grip strength
TBI	Neurocognitive battery, symptom inventory, sleep, mood, balance testing
ALS	ALSFRS-R, forced vital capacity, grip strength, bulbar function, EMG, neurofilament light

Biomarker Strategy
The Most Valuable Biomarkers May Include:
- Neurofilament light chain
- C-reactive protein and inflammatory cytokines
- Oxidative stress markers
- Mitochondrial function markers
- qEEG functional brain age
- Heart-rate variability
- Sleep architecture
- Cerebral blood flow measures

Regulatory Positioning
Ultraweak photon emitters should be positioned carefully in two separate tracks.
The regulatory strategy should be carefully separated into two tracks:

Wellness Track
For General Wellness Use, Claims Should Focus On:
- Relaxation
- Sleep support
- General wellness
- Energy support
- Recovery environment
- Non-disease quality-of-life support

This track should avoid claims to diagnose, treat, cure, mitigate, or prevent disease.

Medical Device Development Track
For Neurological Disorders Such as Parkinson's Disease, Alzheimer's Disease, Stroke, TBI, or ALS, the product should be developed through a formal FDA Medical-Device Pathway. This would require:
- Defined intended use
- Device characterization
- Risk analysis
- Clinical protocol
- FDA Q-Submission interaction
- Controlled clinical evidence
- Validated endpoints
- Safety and effectiveness data

The most appropriate message is:
Ultraweak photon emitters are currently an investigational platform for neurological disease claims. Early observations justify rigorous clinical studies, but disease-treatment claims require regulatory review and clinical validation.

For medical-device development, disease-related claims involving Parkinson's disease, Alzheimer's disease, chronic stroke, TBI, or ALS require a formal regulatory pathway, defined intended use, device characterization, risk analysis, controlled clinical evidence, validated endpoints, and FDA engagement. The strongest regulatory statement is:
Ultraweak photon emitters are investigational for disease-related neurological uses unless cleared, approved, or otherwise authorized by the appropriate regulatory authority. Early observations justify rigorous clinical studies, but disease-treatment claims require regulatory review and controlled clinical validation.

Conclusion

Recovery Sleep inside an Ultraweak Photon Environment (UPE) represents a novel platform technology with potential relevance across a broad spectrum of neurodegenerative, neurovascular, and neurotraumatic disorders. Unlike conventional single-target therapeutic approaches, the UPE model is based on a systems-level biological rationale: during sleep, the brain and body enter a natural restorative state in which mitochondrial repair, redox regulation, glymphatic clearance, neuroimmune balance, neurovascular regulation, synaptic remodeling, and memory consolidation are highly active. By providing prolonged nighttime exposure to a low-intensity, non-contact, non-thermal ultraweak photon environment, Recovery Sleep inside the UPE may enhance or support these endogenous recovery processes.

The scientific foundation for this paradigm is strengthened by published evidence that living brain tissue emits ultraweak photons and that these emissions are associated with mitochondrial respiration, reactive oxygen species, lipid peroxidation, cerebral metabolism, neural activity, EEG/qEEG rhythms, glutamate signaling, hippocampal memory function, and neural stem cell differentiation. These findings suggest that ultraweak photon emission is not merely a passive by-product of metabolism, but may serve as a measurable optical correlate of brain physiology, biochemical status, and functional state.

The clinical evidence reviewed in this article further supports the concept that Recovery Sleep inside the UPE may produce measurable improvements in neurological and whole-body outcomes. Across IRB-approved and ClinicalTrials.gov-registered studies, reported improvements include enhanced brain activity, improved qEEG-based functional brain measures, reduced brain functional age, improved sleep quality, increased energy, better motor performance, improved cognition and emotional well-being, enhanced activities of daily living, and improved quality of life. These outcomes are particularly important because many neurological disorders share common pathological mechanisms, including mitochondrial dysfunction, oxidative stress, neuroinflammation, impaired circulation, sleep disturbance, disrupted neural communication, and reduced neuroplasticity.

Therefore, Recovery Sleep inside the UPE should be viewed not as a single-disease intervention, but as a potential neurorestorative platform technology. Its relevance may extend to Parkinson's disease, Alzheimer's disease and dementia, chronic stroke, traumatic brain injury, amyotrophic lateral sclerosis, and other disorders in which impaired cellular energy, oxidative stress, neurovascular dysfunction, and sleep-related repair failure contribute to progressive or persistent neurological impairment.

The major implication of this review is that the UPE platform may open a new therapeutic direction: supporting the brain's intrinsic recovery capacity during sleep through a passive ultraweak photonic environment. If confirmed by larger, prospective, randomized, sham-controlled clinical trials using validated neurological endpoints, qEEG, functional scales, biomarkers, sleep metrics, and long-term safety monitoring, Recovery Sleep inside the UPE could become an important new approach for advancing the treatment of many neurological disorders with limited current solutions.

In summary, Recovery Sleep inside the Ultraweak Photon Environment offers a biologically plausible, non-invasive, and scalable platform for neurofunctional restoration. The current body of evidence supports continued rigorous clinical development and positions UPE-based Recovery Sleep as a promising frontier in brain recovery, neurodegenerative disease management, and whole-body neurological resilience.

Author Contributions

Conceptualization: J.Z.L.; Methodology: J.Z.L., M.A.S., H.Y.G., and S.D.R.; Writing Original Draft: J.Z.L.; Writing, Review and Editing: All authors.

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Not applicable.

Conflicts of Interest

J.Z.L. and H.Y.G. are co-inventors of biophoton generator technology and co-founders of Tesla BioHealing Inc. S.D.R. and M.A.S. declare no conflicts of interest.

References

1. Paolis LD, Francini R, Davoli I. Fabio De M, Alessandro S, et al. (2024) Biophotons: A Hard Problem. Appl Sci 14: 1-23.
2. National Research Council Canada. World's first ultraweak photon emission technology holds promise for medical forecasts. Published July 21, 2025. Accessed May 9, 2026.
3. Casey H (2025) Exploring Ultraweak Photon Emissions as Optical Markers of Brain Activity. iScience 28: 112019.
4. Liu JZ, Gu HY (2024) Devices for Treating Diseases and Conditions. US Patent No. 12,076,338 B1.
5. Tartak D, Gu HY, Robinson S, Silva I, Liu JZ (2025) Biophoton Quantum Therapy to Treat Advanced Glaucoma: A Novel Non-Invasive Approach for Ocular Neuroprotection. Biomed J Sci Tech Res 62: 54579-54587.
6. (2026) Parkinson's Disease. National Institute of Neurological Disorders and Stroke https://www.ninds.nih.gov/.
7. (2026) Alzheimer's Disease. National Institute of Neurological Disorders and Stroke Updated https://www.ninds.nih.gov/health-information/disorders/alzheimers-disease.
8. (2026) Dementias. National Institute of Neurological Disorders and Stroke https://www.ninds.nih.gov/health-information/disorders/dementia.
9. (2026) Focus on Traumatic Brain Injury Research. National Institute of Neurological Disorders and Stroke https://www.

ninds.nih.gov/current-research/focus-disorders/focus-traumatic-brain-injury-research.
10. (2026) Amyotrophic Lateral Sclerosis. National Institute of Neurological Disorders and Stroke https://www.ninds.nih.gov/health-information/disorders/amyotrophic-lateral-sclerosis-als.
11. Ortega-Ojeda F, Matías C, Alejandro F, Joaquín C, Carmen GR (2018) Measuring the Human Ultra-Weak Photon Emission Distribution using an Electron-Multiplying Charge-Coupled Device as a Sensor. Sensors (Basel) 18: 1150-1152.
12. Du J, Tingting D, Baorui C, Zhiying W, Meina Y, Jinxiang H (2023) The Application and Trend of Ultra-Weak Photon Emission in Biology and Medicine. Front Physiol 11: 1140128.
13. Smotrys MA, Gu HY, Poirier LJ, Ager A, Liu JZ, et al. (2025) Biophoton Therapy Enhances Endogenous Stem-Cell Production: A Non-Invasive Approach to Regenerative Medicine. J Neurol Res Rev Rep 7: 1-11.
14. Gottesman RF, Lutsey PL, Benveniste H, Brown DL, Full KM, et al. (2024) Impact of Sleep Disorders and Disturbed Sleep on Brain Health: A Scientific Statement from the American Heart Association. Stroke 55: 61-76.
15. Yamamoto EA, Jacob HB, Mathew G, Olabisi RS, Aclan D, et al. (2024) The Perivascular Space is a Conduit for Cerebrospinal Fluid Flow in Humans: A Human Glymphatic Imaging Study. Proc Natl Acad Sci USA 121: 2407246121.
16. Ji KH, Yun CH (2025) Brain Health in Sleep Disorders. Sleep Med Clin 20: 57-72.
17. Van Hattem T, Verkaar L, Krugliakova E, Nico A, Marcel Z, et al. (2025) Targeting Sleep Physiology to Modulate Glymphatic Brain Clearance. Physiology (Bethesda) 40: 271-290.
18. Miao Y, Wang J, Li X, Guo J, Ekblom MM, et al. (2025) Poor Sleep Health is Associated with Older Brain Age: The Role of Systemic Inflammation. E bio Medicine 120: 105935-105941.
19. Scholkmann F (2023) Chronobiological Aspects of Spontaneous Ultra-Weak Photon Emission in humans: Ultradian, Circadian and Infradian Rhythms. Springer 17: 289-304.
20. Kobayashi M, Kikuchi D, Okamura H (2009) Imaging of Ultraweak Spontaneous Photon Emission from Human Body Displaying Diurnal Rhythm. PLoS One 4: 6250-6256.
21. Cifra M, Van Wijk EPA, Koch H, Bosman S, Van Wijk R (2007) Spontaneous Ultra-Weak Photon Emission from Human Hands is Time Dependent. Radio engineering 16: 15-19.
22. Mould RR, Mackenzie AM, Kalampouka I, Nunn AVW, Thomas EL, et al. (2024) Ultra weak photon emission—a brief review. Front Physiol 15: 1348910-1348915.
23. Casey H, DiBerardino I, Bonzanni M, Rouleau N, Murugan NJ (2025) Exploring ultraweak photon emissions as optical markers of brain activity. iScience 28: 112019-112019.
24. Moro C, Torres N, El Massri N, Audrey V, Marjorie D, et al. (2022) The Effect of photoBiomodulation on the Brain During Wakefulness and Sleep. J Biophotonics 15: 20220390-202200391.
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, 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 3: 1-7.
27. Zhang L, Bai Y, Guo J (2025) The relationship between mitochondrial respiration metabolism, energy efficiency and ultraweak luminescence in strawberry fruit. Plant Physiol Biochem https://pubmed.ncbi.nlm.nih.gov/40675046/.
28. Mould RR, Mackenzie AM, Kalampouka I, Nunn AVW, Thomas EL, et al. (2024) Ultra weak photon emission a brief review Front Physiol. https://www.frontiersin.org/journals/physiology/articles/10.3389/fphys.2024.1348915/full.
29. Mould RR, Kalampouka I, Thomas EL, Guy GW, Nunn AVW, et al. Non-chemical signalling between mitochondria. Front Physiol https://www.frontiersin.org/journals/physiology/articles/10.3389/fphys.2023.1268075/full.
30. Van Wijk R, Van Wijk EPA, Pang J, Yang M, Yan Y, et al. (2020) Integrating ultra-weak photon emission analysis in mitochondrial research. Front Physiol https://www.frontiersin.org/journals/physiology/articles/10.3389/fphys.2020.00717/full.
31. Tong J (2024) Biophoton signaling in mediation of cell-to-cell communication and radiation-induced bystander effects. Radiat Med Prot 5:145-160.
32. Casey H, DiBerardino I, Bonzanni M, Rouleau N, Murugan NJ (2025) Exploring ultraweak photon emissions as optical markers of brain activity. IScience https://pubmed.ncbi.nlm.nih.gov/40124516/.
33. Prasad A, Mihačová E, Manoharan RR, Pospíšil P (2025) Application of ultra-weak photon emission imaging in plant stress assessment. J Plant Res 138: 389-400.
34. Burgos RCR, Schoeman JC, van Winden LJV, Červinková K, Ramautar R, et al. Ultra-weak photon emission as a dynamic tool for monitoring oxidative stress metabolism. Sci Rep https://www.nature.com/articles/s41598-017-01229-x.
35. Pospíšil P, Prasad A, Rác M (2014) Role of reactive oxygen species in ultra-weak photon emission in biological systems. J Photochem Photobiol B 139:11-23.
36. Cifra M, Pospíšil P (2014) Ultra-weak photon emission from biological samples: definition, mechanisms, properties, detection and applications. J Photochem Photobiol B https://pubmed.ncbi.nlm.nih.gov/24726298/.
37. Zapata F, Pastor-Ruiz V, Ortega-Ojeda F, Montalvo G, Ruiz-Zolle AV (2021) Human ultra-weak photon emission as non-invasive spectroscopic tool for diagnosis of internal states a review. J Photochem Photobiol B https://pubmed.ncbi.nlm.nih.gov/33540236/.
38. Rastogi A, Pospíšil P (2013) Ultra-weak photon emission as a non-invasive tool for the measurement of oxidative stress induced by UVA radiation in Arabidopsis thaliana. J Photochem Photobiol B https://pubmed.ncbi.nlm.nih.gov/23624533/.
39. Prasad A, Pospíšil P (2011) Linoleic acid-induced ultra-weak photon emission from Chlamydomonas reinhardtii as a tool for monitoring of lipid peroxidation in the cell membranes. PLoS One https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0022345.
40. Rastogi A, Pospíšil P (2011) Ultra-weak photon emission as a non-invasive tool for monitoring of oxidative processes in the epidermal cells of human hand skin. Skin Res Technol 17: 368-374
41. Prasad A, Mihačová E, Manoharan RR, Pospíšil P (2025) Application of ultra-weak photon emission imaging in plant stress assessment. J Plant Res 138: 389-400.
42. Sefati N, Tahereh Esmaeilpour, Vahid Salari, Asadullah Zarifkar, Farzaneh Dehghani, et al. (2024) Monitoring Alzheimer's disease via ultraweak photon emission. I Science 27: 108-573.

43. He M, Wang X, Ma X, Eduard van Wijk , Herman van Wietmarschen, et al. (2017) Spontaneous ultra-weak photon emission in correlation to inflammatory metabolism and oxidative stress in a mouse model of collagen-induced arthritis. J Photochem Photobiol B 168: 98-106.
44. Ives JA, van Wijk EPA, Bat N, Cindy Crawford, Avi Walter, et al. (2014) Ultraweak photon emission as a non-invasive health assessment: a systematic review. PLoS One https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0087401.
45. Sun Y, Wang C, Dai J (2010) Biophotons as neural communication signals demonstrated by in situ biophoton autography. Photochem Photobiol Sci 9: 315-322.
46. Tang R, Dai J (2013) Biophoton signal transmission and processing in the brain. J Photochem Photobiol B 139: 71-75.
47. Tang R, Dai J (2014) Spatiotemporal imaging of glutamate-induced biophotonic activities and transmission in neural circuits. PLoS One https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0085643.
48. Wang Z, Wang N, Li Z, Zehua Li, Fangyan Xiao, et al. Human high intelligence is involved in spectral redshift of biophotonic activities in the brain. Proc Natl Acad Sci U S A 113: 8753-8758.
49. Salari V, Scholkmann F, Shahbazi F (2015) Ultraweak photon emission in the brain. J Integr Neurosci 14: 419-429.
50. Kobayashi M, Takeda M, Ito K, Kato H, Inaba H (1999) In vivo imaging of spontaneous ultraweak photon emission from a rat's brain correlated with cerebral energy metabolism and oxidative stress. Neurosci Res 34: 103-113.
51. Kumar S, Boone K, Tuszyński J, Barclay P, Simon C (2016) Possible existence of optical communication channels in the brain. Sci Rep 6: 1-13.
52. Pospíšil P (2026) Biofield in neural communication: a review and conceptual framework. Prog Biophys Mol Biol 200: 31-40.
53. 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: 14-20.
54. Erboz A, Kesekler E, Gentili PL, Uversky VN, Coskuner-Weber O (2025) Electromagnetic radiation and biophoton emission in neuronal communication and neurodegenerative diseases. Prog Biophys Mol Biol 195: 87-99.
55. Hu Y, Gu HY, Liu JZ (2025) Twelve-Day Live Blood Analysis Reveals Hemorheological and Microvascular Restoration in a Parkinsons Disease Patient Following Biophoton Therapy. J Neurol Res Rev Rep 7: 1-6.
56. 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 Dise & Rep 2: 1-10.
57. Sefati N (2026) Study on ultraweak photon emission from rat hippocampus and Alzheimer's disease-related changes. PMC https://pmc.ncbi.nlm.nih.gov/articles/PMC10792242/.
58. ClinicalTrials.gov (2026) Impact of a Biophoton Therapy on Patients with Brain Disorders. National Library of Medicine https://clinicaltrials.gov/study/NCT06147999.
59. 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: 2-13.
60. Liu JZ, Smotrys M, Robinson SD, Liu S, Gu HY (2024) Alzheimer's Disease Was Successfully Treated with Biophoton Generators. The Alzheimer's Association International Conference (AAIC), July 27-Aug 2, 2024, Philadelphia.
61. Hu Y, Chien L, Gu HY, Liu JZ (2025) Enhancing Blood Fluidity and Physiological Markers in Early-Stage Alzheimer's Disease Using Biophoton Generators: A Case Study. J Neurol Res Rev Rep 7: 1-7.
62. 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 3: 1-7.
63. ClinicalTrials.gov (2026) Impact of Biophoton Generator on Chronic-Stroke Patients' Functional Recovery. National Library of Medicine https://clinicaltrials.gov/study/NCT05898334.
64. Center Watch (2026) Can Patients with Chronic Stroke Regain Living Independence by Daily Energizing with Biophoton Generators. NCT06049849 https://www.centerwatch.com/clinical-trials/listings/NCT06049849/can-patients-with-chronic-stroke-regain-living-independence-by-daily-energizing-with-biophoton-generators?activelyRecruiting=false&page=71&id=42&slug=cerebral-ischemia.
65. Liu JZ, Smotrys MA, Robinson SD, Yu HX, Liu SX, et al. (2025) Breakthrough in Stroke Rehabilitation: Non-Invasive Biophoton Therapy Promotes Neurofunctional Recovery in Chronic Stroke Patients. J Alzheimers Dise & Rep 2: 1-3.
66. Liu JZ, Smotrys MA, Robinson SD, Yu HX, Liu SX, (2025) Quantitative EEG Reveals Cognitive and Motor Restoration After Biophoton Treatment in Chronic Stroke. J Neurol Res Rev Rep 7: 1-6.
67. Hu Y, Chien L, Gu HY, Liu JZ (2025) Biophoton Therapy Restores Hemorheology in Chronic Stroke: Live Blood Evidence of Microvascular Recovery. J Neurol Res Rev Rep 7: 1-5.
68. 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: 36-43.
69. Nelson LD, Nancy R Temkin, Jason Barber, Benjamin L Brett, David O Okonkwo, et al. (2023) Functional recovery, symptoms, and quality of life 1 to 5 years after traumatic brain injury. JAMA Netw Open 6: e233660.
70. 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. Journal of Neurology Research Reviews & Reports 7: 1-6.
71. 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: 14-20.
72. Welker J, Gu HY, Liu JZ (2026) Observational case series of three individuals with amyotrophic lateral sclerosis using biophoton generators. Journal of Neurology Research Reviews & Reports 8: 1-6.
73. Hauser DN, Hastings TG (2013) Mitochondrial dysfunction and oxidative stress in Parkinson's disease and monogenic parkinsonism. Neurobiol Dis 51: 35-42.
74. Liu JZ, Smotrys MA, Robinson SD, Gu HY. Therapeutic benefits of biophoton therapy in Parkinson's disease: clinical evidence from a pilot and real-world study. Journal of Neurology Research Reviews & Reports https://www.semanticscholar.org/paper/Therapeutic-Benefits-of-Biophoton-Therapy-in-from-a-Liu-Smotrys/773f009403d67c9103ad66cde11bb49f4fff65c9.

75. N Sefati (2026) Study on ultraweak photon emission from rat hippocampus and Alzheimer's disease-related changes. PMC https://pmc.ncbi.nlm.nih.gov/articles/PMC10792242/.
76. Tong J (2024) Biophoton signaling in mediation of cell-to-cell communication and radiation-induced bystander effects. Radiat Med Prot 5: 145-160.
77. 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 Neurology Research Reviews & Reports 7: 2-6.