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Ultraweak Photon Emission in the Brain: Mechanisms and Prospects for Non-invasive Neural Monitoring

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ABSTRACT

Ultraweak photon emission (UPE) or biophoton emission is a spontaneous emission of low intensity photons by living cells as a byproduct of metabolic and oxidative activities. According to recent research, the brain being a high metabolism and oxidative environment is a significant source of UPE. Though the amount of these emissions is nowhere near the threshold that the human eye can sense, the emissions can be sensed with a sensitive photomultiplier tube or charge-coupled device, which offers a potential non-invasive look at the functioning of the neuron and systemic oxidative stress. In this view, the mechanistic basis of the brain UPE with a particular focus on its connection with the reactive oxygen species production, mitochondrial activity, and cellular signaling pathways is explored. We will talk about the possible relationship between UPE intensity and changes in neuronal metabolic states, neuroinflammatory conditions, and oxidative burden and present a possible biomarker of neurological activity and disease. Moreover, the article addresses the theoretical consequences of UPE in brain monitoring, such as the opportunities of optical-based neuroimaging, the early diagnosis of neurodegenerative diseases, and real-time evaluation of the treatment mode. The problems of measuring such as signal-noise constraints, standardization of detection procedures and environmental interference are also identified to guide future studies. Given that such synthesis of current evidence and conceptual frameworks, this perspective will be used to offer a foundation to integrate brain UPE into experimental and clinical neuroscience. Further development of the given field could eventually allow new methods of diagnostics and monitoring, as well as allow us to learn more about the physiology and pathophysiology of the brain under the influence of non-invasive optical signals.

Keywords: Brain, Ultraweak Photon Emission, Biophotons, Neural Metabolism, Oxidative Stress, Neuroimaging

Introduction

Biophoton emission, or ultraweak photon emission (UPE), is the production of very weak-strength photons by living cells as a byproduct of metabolic and oxidative reactions [1]. These photons are many folds weaker than the visible light and they are visible to the human eye. The development of highly sensitive technologies of detecting UPE, including photomultiplier tubes and charge-coupled device (CCD) cameras has allowed investigators to measure UPE in different biological systems like plant tissues, cultured cells and mammalian organs [2]. There is also emerging evidence that the brain due to its high metabolic rate and large numbers of mitochondria is a highly active source of UPE [3].

The UPE of the brain has attracted interest since it can provide a non-invasive optical view of the neural activity and oxidative stress [4]. Reactive oxygen species (ROS) are also formed in the process of neuronal metabolism and these ROS reactions are a significant source of biophoton emission [5]. The differences in the intensity of UPE may, thus, indicate modification of the metabolic conditions, neuroinflammatory events or burden of oxidation, which might be a preliminary signal of neurological impairment [6]. Although the

interest is increasing, the area is still very conceptual with little empirical evidence on the functional role or physiological importance of brain UPE. Experimental studies are limited by several technical difficulties that require a very low level of intensity of the emissions, the interference of background noise, and the absence of a standardized protocol of measurements [7]. However, theoretical proposals allow believing that UPE can be useful as biomarker to monitor neural activity, assess neural degradation process, and measure therapeutic intervention in a non-invasive way [8].

This review will summarize the existing knowledge of brain UPE, its mechanistic basis and write about its possible applications and research directions. bringing together the new information that has been gathered thus far, the work is intended to give a conceptual framework of how ultraweak photon emission can be used to study the brain and its translationational potential as a new optical measure of brain activity.

Mechanistic Discussion

UPE in the brain is a result of interactions among the cellular metabolism and oxidative processes that are complex [9]. The major neuronal and glial pathways, reactive oxygen species dynamics, and biochemical processes involved in the photon generation are outlined in what follows.

The Mitochondrial ROS: the Original Cause of Brain UPE.

The aspect of UPE in the brain is directly connected with the oxidative metabolism of the neurons and glial cells [10]. Mitochondria, which are highly present in the neuronal tissue, produce adenosine triphosphate (ATP) under the process of oxidative phosphorylation [11]. In the process, there is sometimes a leak of electrons along the electron transport chain, leading to the formation of reactive oxygen species (ROS) including: superoxide anions, hydrogen peroxide, and singlet oxygen. These ROS react with the biomolecules such as lipids, proteins and nucleic acids to form electronically excited intermediates [12]. The release of excited molecules results in the release of photons, mostly in the ultraviolet to visible range (200-800 nm) [13]. These emissions are very weak, typically between 10 and 1,000 photons/s $^{-1}$ cm $^{-2}$, and therefore very sensitive instruments like photomultiplier tubes or cooled charge-coupled devices (CCD) are required [14].

Neuronal Response and Dynamic Modulation of UPE

Transient metabolic demands exist due to neuronal firing and synaptic activity and have the potential to regulate ROS production and, thus, affect UPE intensity [15]. Action potentials need an elevated rate of ATP consumption, and the oxidative responses resulting from this can heighten photon emission in the immediate surroundings. There is evidence to indicate that the areas of high synaptic performance might have a minor elevation of UPE, as indications of dynamic metabolic conditions [16]. Also, calcium flux and cycling of neurotransmitters are also sources of indirect ROS, which introduce time variability to the UPE patterns [17] The implication of these processes is that UPE is not a fixed signal, but a varying measure of neuronal energy expenditure and metabolic homeostasis.

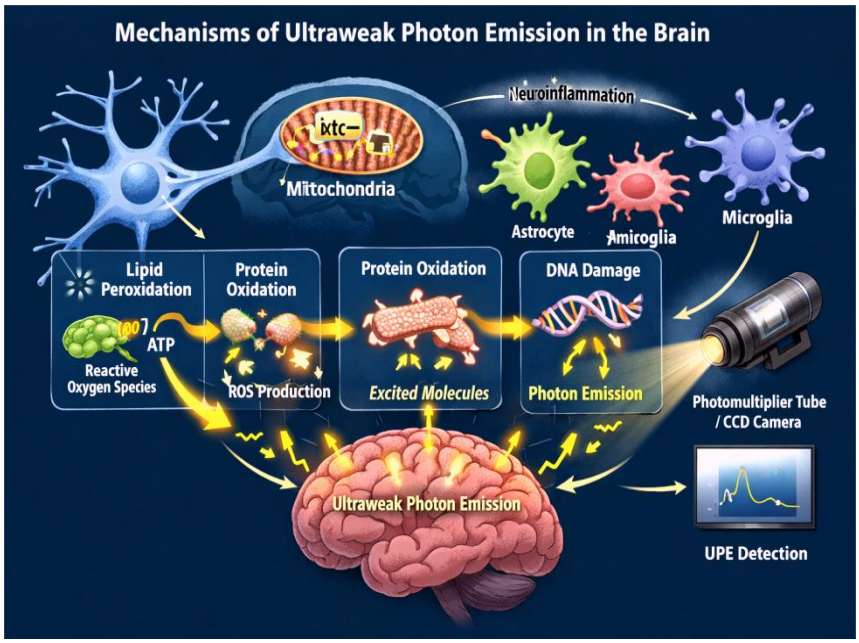

Figure 1: Basic Understanding of Mechanisms of Ultraweak Photon Emission in the Brain *(Image Source: GPT- 5 mini, OpenAI, 2025)**. This diagram shows the cellular and molecular mechanisms involved in UPE in the brain tissue. The reactive oxygen species (ROS) are produced in the neurons and glial cells (astrocytes and microglia) by the mitochondrial oxidative metabolism. ROS interacts with lipids, proteins and DNA to generate electronically excited molecules. The relaxation of these molecules produces ultraweak photons (UPE), indicated by wavy arrows of yellow color. The intensity of photon emission can indicate neurophysiological processes, the role of glia and neuroinflammation. UPE is detected with sensitive optical sensors (i.e. photomultiplier tubes or charge-coupled device (CCD) cameras) which offer a possible non-invasive biomarker of brain oxidative stress and metabolic activity. **Neurons:** The main source of metabolic ROS because it is in high demand of ATP; oxidative byproducts of mitochondrial activity are associated with photon emission. **Astrocytes and Microglia:** Part of the oxidative metabolism and regulation of local UPE through neuroinflammatory signaling. **ROS mediated processes:** These processes are lipid peroxidation, protein oxidation, and DNA damage, and produce excited molecules that can emit photons. **Photon Emission:** The ultraviolet and visible spectrum (200 800 nm) of the light produce ultraweak photons that are emitted in the region that is significantly below the capability of human eyes to detect. **Detection Systems:** Photomultiplier tubes and CCD cameras can be used to quantify and spectrum analyse UPE and, thus, may be used to monitor potential metabolic and oxidative states.*

Glial Contributions and Neuroinflammatory Modulation

The glial cells especially the astrocytes and microglia are important in the sustenance of neuronal metabolic and redox homeostasis [18]. In neuroinflammatory processes or oxidative stress, glial metabolism may increase producing other ROS and promoting photon emission. Microglial activation such as the one caused by oxidative bursts that increase due to microglial activation can raise UPE locally [19]. The ability of the astrocytes to determine neuronal oxidative stress, via glutamate uptake and metabolic maintenance, indirectly impacts the intensity of the UPE [20]. These interactions are indicative of the possibility that UPE patterns could represent a composite signal that is caused by both neuronal and glial activities, and it gives us a view into the metabolic and inflammatory milieu of the brain.

Biological Pathways between ROS and Photon Emission

The biochemical processes that support UPE are those that involve oxidative interactions and form electronically excited species that can emit photons. One of these pathways is lipid peroxidation, in which the ROS start attacking polyunsaturated fatty acids in neuronal membranes, creating excited carbonyl groups, which release photons during relaxation.[21] The oxidation of proteins and oxidation of nucleic acids also produce excited intermediates that have the ability to emit light. Singlet oxygen produced during ROS reactions may impart energy to chromophores, which create additional energy to UPE [22]. All these pathways together form a continuum of photon emission which reflects the general state of the brain tissue in terms of oxidation.

Functional Brain Monitors Implications

Despite the theoretical direct functional role of UPE, its vulnerability to changes in metabolic and oxidative states implies that it could be used as a non-invasive biomarker [23]. The local neuronal activity, local metabolic demand, or local oxidative stress levels at the level of the UPE may be correlated with spatial and temporal patterns, which provide additional insights to the conventional electrophysiological or imaging modalities [24]. It is possible that computational modeling and spectral analysis would be able to associate emission intensity with individual biochemical pathways or neuronal circuits [25]. Moreover, the UPE changes can indicate the initial neurodegenerative impairments or reactions to the treatment interventions, indicating its possible application in both research and clinical neuroscience.

Future Prospects

UPE of the brain is a new non-invasive marker of neuronal metabolic and oxidative activity. Future studies can be directed at high-resolution spatiotemporal mapping of UPE with state-of-the-art photomultiplier arrays or ultrasensitive CCD/CMOS detectors to match the photons flux with localized neural circuit activity. Combination with optogenetic or functional imaging systems could enable simultaneous measurement of metabolic, electrophysiological and oxidative responses. The UPE spectral patterns can allow the development of biomarkers to detect neurodegenerative disorders, ischemic stress, or neuroinflammatory states early with the help of computational modeling of these patterns. Moreover, investigation of the possibility to modulate UPE using pharmacological or antioxidant treatments might determine the application of the UPE in the assessment of therapeutic response. Reproducibility will be dependent on standardization of protocols to measure the spectral, spectral calibration, and noise reduction methods. On the whole, accurate quantification of UPE can revolutionize the monitoring of the brain by offering a direct optical measure of mitochondrial and oxidative physiology as an addition to traditional imaging and electrophysiology modes.

Conclusion

UPE of the brain is indicative of the metabolism, ROS processes, and oxidative relations in neurons and glial cells. Conceptual and mechanistic studies propose that UPE is an intensity which might be a sensitive measure of local metabolic activity, neuroinflammation, and oxidative stress. This recent progress in high-sensitivity optical detection and computation modeling might make UPE a non-invasive biomarker that can be used to monitor neural activity, disease progression, and treatment reactions. Standardized measurement protocol and incorporation of UPE with complementary imaging and electrophysiological measures will be essential in the application of this new concept into a viable neuroscience research and clinical practice.

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Conflict of Interest

None

Authors' Contribution

All authors contributed equally as per ICMJE

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