

Photobiomodulation (PBM) for Alzheimer's Disease Can Light Boost Brain Power?

VIDEO

Can Light Boost Brain Power? Groundbreaking tPBM Study Reveals Promising Results

Transcranial Photobiomodulation

- ▶ NIR wavelengths are highly absorbable by Cytochrome-c-oxidase (CCO)
- ▶ They convert the suppressed CCO into an active (oxidized) state
- ▶ The oxidized state →
 - Increased efficiency of reactions along the electron transport chain
 - Higher O₂ consumption
 - Sharper proton gradient
 - More ATP production
- ▶ More neuronal ATP activates the firing rate and connectivity among neurons

Biochemical Process

1. CCO near-infrared photon absorption
2. CCO oxidation
3. CCO proton pumping and NO release
4. CCO oxygen reduction to water
5. ATP synthesis
6. Hb oxygen transport

Physiological process of tPBM inside of a mitochondrion [1]

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https://www.youtube.com/watch?v=f_jUpdJVjP8

Summary of Benefits of Neuronic PBM on the Brain

Reduction of amyloid-beta protein levels

PBM can reduce the levels of amyloid-beta proteins in the brain, as demonstrated in several studies published in the Journal of Alzheimer's Disease in 2020 and 2018.

Stimulation of autophagy – cell turnover & clean-up

PBM has been shown to stimulate autophagy (internal clean up) in brain cells, which can help to clear amyloid-beta proteins out of the cells and blood.

Neuroprotection

Photobiomodulation can protect neurons from damage by maintaining cell internal ops.

Anti-inflammatory effects

Photobiomodulation has anti-inflammatory effects that can reduce inflammation in the brain. It has also been shown to improve microvascular blood flow in the brain.

Increased BDNF production – Brain derived Neurotrophic Factor

Photobiomodulation can increase BDNF production, which can improve neuronal function.

PBM may help reduce β -amyloid protein levels in Alzheimer's brains

J Photochem Photobiol B. 2013 Jun 5;123:13-22.

Non-invasive infra-red therapy (1072 nm) reduces β -amyloid protein levels in the brain of an Alzheimer's disease mouse model, TASTPM

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Abstract

Background: Alzheimer's disease (AD) is the most prevalent neurodegenerative disease and common cause of dementias in the Western world. This study investigated the expression profile of heat-shock proteins (HSPs) involved in maintaining healthy neurons in the TASTPM AD mouse model, and whether chronic treatment with 1072 nm infra-red (IR1072) modified the expression profiles of HSPs and amyloidopathy in female TASTPM mice.

Methodology/principal findings: Quantitative immunoblotting and immunohistochemistry were used to examine the expression of proteins such as HSPs, phosphorylated tau (tau-P), amyloid precursor protein (APP), β -amyloid1-40 ($A\beta$), and $A\beta$ 1-42. TASTPM mice at 3, 7 and 12 months were investigated as well as female TASTPM mice which had undergone a chronic, 5 month, IR1072 treatment. During the first 12 months of age, a critical period of AD progression, reduced HSP40 and HSP105 were observed. α B-crystallin, $A\beta$ 1-42 and tau-P increased over this period, particularly between 3 and 7 months. Chronic IR1072 treatment of female TASTPM mice elicited significant increases in HSP60, 70 and 105 and phosphorylated-HSP27 (P-HSP27) (50-139%), together with a concomitant profound decrease in α B-crystallin, APP, tau-P, $A\beta$ 1-40 and $A\beta$ 1-42 (43-81%) protein levels at 7 months of age. Furthermore, IR1072 treatment elicited a modest, but significant, reduction in $A\beta$ 1-42 plaques in the cerebral cortex.

Conclusions/significant findings: IR1072 treatment provides a novel non-invasive and safe way to upregulate a panel of stress response proteins in the brain, known to both reduce protein aggregation and neuronal apoptosis. This approach recently entered clinical trials for AD in the USA, and may provide a novel disease modifying therapy for a range of neuropathologies.

PBM helps with energy production and better cognition, improves sleep in ALZ patients

Brain Photobiomodulation Therapy: a Narrative Review

Mol Neurobiol. 2018 Aug;55(8):6601-6636.

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Abstract

Brain photobiomodulation (PBM) therapy using red to near-infrared (NIR) light is an innovative treatment for a wide range of neurological and psychological conditions. Red/NIR light is able to stimulate complex IV of the mitochondrial respiratory chain (cytochrome c oxidase) and increase ATP synthesis. Moreover, light absorption by ion channels results in release of Ca^{2+} and leads to activation of transcription factors and gene expression. Brain PBM therapy enhances the metabolic capacity of neurons and stimulates anti-inflammatory, anti-apoptotic, and antioxidant responses, as well as neurogenesis and synaptogenesis. Its therapeutic role in disorders such as dementia and Parkinson's disease, as well as to treat stroke, brain trauma, and depression has gained increasing interest. In the transcranial PBM approach, delivering a sufficient dose to achieve optimal stimulation is challenging due to exponential attenuation of light penetration in tissue. Alternative approaches such as intracranial and intranasal light delivery methods have been suggested to overcome this limitation. This article reviews the state-of-the-art preclinical and clinical evidence regarding the efficacy of brain PBM therapy.

Keywords: Brain function; Cortical neurons; Dementia; Depression; Low-level laser therapy; Photobiomodulation therapy; Stroke; Traumatic brain injury.

PBM improves cognitive performance, blood flow & connectivity in ALZ when used at home

Effects of Home Photobiomodulation Treatments on Cognitive and Behavioral Function, Cerebral Perfusion, and Resting-State Functional Connectivity in Patients with Dementia: A Pilot Trial

[Linda L Chao](#)^{1,2,3}

Abstract

Objective: To examine the effects of transcranial and intranasal photobiomodulation (PBM) therapy, administered at home, in patients with dementia. **Background:** This study sought to replicate and build upon a previously published case series report describing improved cognitive function in five patients with mild-to-moderate dementia after 12 weeks of transcranial and intranasal near-infrared (NIR) PBM therapy. **Materials and methods:** Eight participants (mean age: 79.8 ± 5.8 years old) diagnosed with dementia by their physicians were randomized to 12 weeks of usual care (UC, $n = 4$) or home PBM treatments ($n = 4$). The NIR PBM treatments were administered by a study partner at home three times per week with the Vielight Neuro Gamma device. The participants were assessed with the Alzheimer's Disease Assessment Scale-cognitive (ADAS-cog) subscale and the Neuropsychiatric Inventory (NPI) at baseline and 6 and 12 weeks, and with arterial spin-labeled perfusion magnetic resonance imaging (MRI) and resting-state functional MRI at baseline and 12 weeks. **Results:** At baseline, the UC and PBM groups did not differ demographically or clinically. However, after 12 weeks, there were improvements in ADAS-cog (group \times time interaction: $F_{1,6} = 16.35$, $p = 0.007$) and NPI (group \times time interaction: $F_{1,6} = 7.52$, $p =$

0.03), increased cerebral perfusion (group \times time interaction: $F_{1,6} = 8.46$, $p < 0.03$), and increased connectivity between the posterior cingulate cortex and lateral parietal nodes within the default-mode network in the PBM group. **Conclusions:** Because PBM was well tolerated and associated with no adverse side effects, these results support the potential of PBM therapy as a viable home treatment for individuals with dementia.

Keywords: Alzheimer's disease; LED; dementia; neuroimaging; photobiomodulation.