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Prevention of Alzheimer's disease may be achieved with transcranial infrared laser stimulation

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Alzheimer's disease (AD) and dementia is probably the most worrying health problem facing the Western world today. A large number of clinical trials have failed to show any benefit of the tested drugs in stabilizing or reversing the steady decline in cognitive function that is suffered by dementia patients. Although the pathological features of AD consisting of beta-amyloid plaques and tau tangles are well established, considerable debate exists concerning the genetic or lifestyle factors that predispose individuals to developing dementia. Photobiomodulation (PBM) portrays the helpful utilization of red or close infrared light to invigorate mending, soothe torment and irritation, and keep tissue from passing on. As of late PBM has been applied for a various scope of cerebrum issue, every now and again applied in a non-intrusive way by sparkling light on the head (transcranial PBM). The current survey talks about the instruments of activity of tPBM in the mind, and sums up examines that have utilized tPBM to treat creature models of AD. The aftereffects of a predetermined number of clinical preliminaries that have utilized tPBM to treat patients with AD and dementia are talked about.

Photobiomodulation (PBM) portrays the restorative utilization of red or close infrared light to animate mending, soothe agony and irritation, and keep tissue from kicking the bucket. PBM used to be called "low-level laser (or light) therapy" (LLLT) but the name was changed to reflect the fact that the term "low" was undefined, lasers were not absolutely required, and inhibition of some processes was beneficial

Objective

Dementia is the clinical term used to describe a broad range of brain disorders that affect cognitive and executive functioning and memory. The diagnosis of dementia requires a change in mental function with a more pronounced decline than one would expect due to the normal aging process. In 2015, 46.8 million people throughout the world were estimated to be suffering from dementia, with 58% living in low and middle income countries and this number is expected to double every 20 years.

Alzheimer's disease (AD) is the most common type of dementia (60% to 70% of cases) followed by vascular dementia (25%), and Lewy body dementia (15%). AD was first described by Alois Alzheimer (1864–1915) who published his report in 1911. About 70% of the risk is probably genetic, with many genes proposed to be involved. Other risk factors include a history of head injury, depression, and hypertension. AD is characterized by diffuse atrophy of the entire brain (especially of the cortex), accompanied by extracellular beta-amyloid plaques and intraneuronal neurofibrillary tangles composed of hyperphosphorylated tau protein. A wide variety of other investigational drugs have been tested in clinical trials, but so far without much success.

There is now compelling evidence that chronic brain hypoperfusion (CBH) during advanced aging is not only a major contributor to cognitive impairment but may also be the underlying cause of Alzheimer's disease (AD). This conclusion forms part of the vascular hypothesis of AD which argues that AD development is dependent on the presence of vascular risk factors for AD and on the progressive age-related decline of cerebral blood flow.(1) Over time, this combination of events can lead to significant cerebrovascular insufficiency. Neuroimaging studies of aged persons with mild cognitive impairment (MCI), a presumed precursor of AD, have shown marked reduction of cerebral perfusion in brain regions later attacked by Alzheimer-related neurodegeneration. These brain regions include prefrontal, temporoparietal and posterior cingulate cortices. We have proposed in past reports that CBH advances neuronal vitality hypometabolism prompting psychological brokenness and AD. On the off chance that CBH is an indispensable component in the improvement of AD, at that point mediations that forestall or postpone neuronal hypometabolism could be a helpful objective in patients at high danger of AD. Transcranial infrared laser incitement (TILS) offers a non-intrusive way to deal with raise neurometabolic vitality levels that can improve cerebral hemodynamics and psychological capacity in people. TILS may work by expanding cerebrum cytochrome-c-oxidase to support mitochondrial ATP creation and neuronal vitality limit. Primer examinations in typical grown-up human volunteers demonstrate that utilizing TILS in the prefrontal cortex altogether improved memory assignments contrasted with a fake treatment gathering. Pilot randomized, fake treatment controlled examinations have revealed that MCI patients improved memory work following 12 weeks of day by day TILS. These and other findings using TILS to enhance mitochondrial ATP synthesis in dysfunctional brain cells require randomized clinical trials to evaluate the merit of this technique Conclusion

The fact that PBMT may produce a large range of beneficial changes in the brain, and is without any major side-effects, suggests it should be more widely tested for AD and dementia in large controlled trials. Exposing the head to light at power levels less than that received in direct sunlight (but without harmful ultraviolet wavelengths) is intrinsically safe. Any side-effects reported have been rare, mild and transient, consisting of slight headache, difficult sleeping and mild itching on the scalp. It is likely that tPBM for AD will need to be continued indefinitely, as regressions have been observed when PBM treatments have ceased. Moreover, unrelated health problems such as urinary tract infections or falls can lead to loss of the benefits achieved with tPBM. Home use tPBM devices can be applied by the caregivers, who consistently report improvements in their own quality of life.

Biography

Jack de la Torre began his research studies of Alzheimer's disease in 1990. He has written over 200 peer-reviewed articles and edited or coedited ten volumes on the vascular pathophysiology of dementia which he proposed in 1993 as the cause of Alzheimer's disease. He is the author of 4 books including the recent Alzheimer's Turning Point: A Vascular Approach to Clinical Prevention (Springer 2016). He has held

professorial appointments in neurosurgery and neuroscience departments at the University of Chicago, Northwestern University and the University of Ottawa, and is presently continuing his research as a Professor in Neuropsycholog at the University of Texas, Austin.

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