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

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 previous reports that CBH promotes neuronal energy hypometabolism leading to cognitive dysfunction and AD. If CBH is a vital element in the development of AD, then interventions that prevent or delay neuronal hypometabolism could be a therapeutic target in patients at high risk of AD. Transcranial infrared laser stimulation (TILS) offers a non-invasive approach to raise neurometabolic energy levels that can improve cerebral hemodynamics and cognitive function in humans. TILS may work by increasing brain cytochrome-c-oxidase to boost mitochondrial ATP production and neuronal energy capacity. Preliminary studies in normal adult human volunteers indicate that using TILS in the prefrontal cortex significantly improved memory tasks compared to a placebo group. Pilot randomized, placebo-controlled studies have reported that MCI patients improved memory function following 12 weeks of daily 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.

### Speaker 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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