

Oscillations and Brain Waves

by: Paul Schwen

Note: this article is incomplete and still requires data on the following:

Theta Wave Disruptions and Disorders
Alpha Wave Disruptions and Disorders
Beta Wave Disruptions and Disorders
Gamma Wave Disruptions and Disorders

The purpose of this article is to persuade practitioners to utilize laser therapy as a holistic, ancillary approach to treating neurological disorders.

Demyelinating diseases of the CNS can be classified according to their pathogenesis into several categories: demyelination due to inflammatory processes, viral demyelination, demyelination caused by acquired metabolic derangements, hypoxic-ischemic forms of demyelination, and demyelination caused by focal compression. These diseases can specifically target upper motor neurons, lower motor neurons, or both. Some of these distinctions are rather simplistic in that there is overlap in pathogenesis between the entities in the different categories, but classification provides a conceptual framework that may be useful in more accurate diagnoses.

Simply put, demyelinating diseases signify free-radicals that enter the nervous system and cause immune system and enzymatic abnormalities, thereby damaging the fragile tissues of the nervous system. Radiant Beam Energy can penetrate deeply (up to 7cm) into the brain and other tissues, and can be effective in reversing the effects of disease-causing inflammation, ischemic effects, degenerative effects of axon and myelin atrophy, and more.

Neuronal Oscillations and Brain Waves

When brain cells fire rhythmically and in sync, they produce waves, which are categorized by their firing frequencies. Delta waves (1.5 Hz to 4 Hz), for example, are produced during deep sleep, theta waves (4 Hz to 12 Hz) occur during running and deep meditation, and gamma waves (25 Hz to 100 Hz) are associated with excitement and concentration. Disruption of gamma waves could be a key contributor to Alzheimer's disease pathology, according to recent MIT studies. The restoration of these waves, researchers propose, may one day be an option for Alzheimer's disease treatment.

Delta wave activity between 0.5 Hz to 2 Hz – deep stage 3 of NREM sleep; thalamus or cortex

Theta activity between 4–8 Hz and 150–350 ms – distance running, deep meditation

Alpha activity between 8–16 Hz and 500–700 ms -

Beta activity between 16–28 Hz and 600–1000 ms.

Gamma activity between 25–76 Hz and 150–350 ms; excitement, concentration 44Hz primary; frontal lobes, prefrontal cortex

Gamma activity between 76–86 Hz and 150–350 ms

DELTA WAVES

A DELTA wave is a high amplitude brain wave with a frequency of oscillation between 0.5–4 hertz. DELTA waves are usually associated with the deep stage 3 of NREM sleep, also known as slow-wave sleep (SWS), and aid in characterizing the depth of sleep.

Delta activity stimulates the release of several hormones, including growth hormone releasing hormone GHRH and prolactin (PRL). GHRH is released from the hypothalamus, which in turn stimulates release of growth hormone (GH) from the pituitary. The secretion of (PRL), which is closely related to (GH), is also regulated by the pituitary. The release of thyroid stimulating hormone (TSH), is decreased in response to delta-wave signaling.

Delta Wave Disruptions and Disorders

Regional delta wave activity not associated with NREM sleep was first described by W. Grey Walter, who studied cerebral hemisphere tumors. Disruptions in delta wave activity and slow wave sleep are seen in a wide array of disorders. In some cases, there may be increases or decreases in delta wave activity, while others may manifest as disruptions in delta wave activity, such as alpha waves presenting in the EEG spectrum. Delta wave disruptions may present as a result of physiological damage, changes in nutrient metabolism, chemical alteration, or may also be idiopathic. Disruptions in delta activity is seen in adults during states of intoxication or delirium and in those diagnosed with various neurological disorders such as dementia or schizophrenia.

Patients suffering from fibromyalgia often report unrefreshing sleep. A study conducted in 1975 by Moldovsky et al. showed that the delta wave activity of these patients in stages 3 and 4 sleep were often interrupted by alpha waves. They later showed that depriving the body of delta wave sleep activity also induced musculoskeletal pain and fatigue.

Delta Wave Disorders

Schizophrenia

Dementia

Fibromyalgia

Narcolepsy

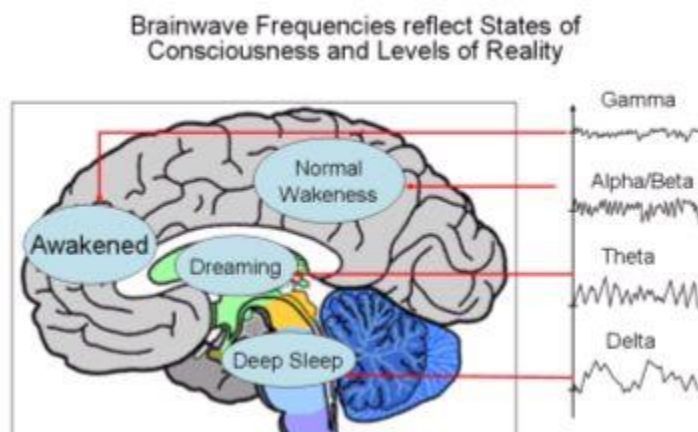
Depression

Anxiety

Obsessive-compulsive disorder

Attention deficit hyperactivity disorder (ADHD) and its three subtypes.

Juvenile chronic arthritis



Gamma Wave Disorders

Disruption of gamma waves could be a key contributor to Alzheimer's disease pathology, ; the restoration of these waves, may one day be an option for treatment of dementia, Parkinson's disease, and Alzheimer's disease.

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Stimulation of gamma waves reduced levels of amyloid- β , decreased phosphorylation of tau, and led the brain's immune cells—microglia—to perform their usual housekeeping role, clearing away cellular debris, including amyloid- β (as opposed mounting an inflammatory response as microglia do in Alzheimer's disease).

MIT's Li-Huei Tsai, Ed Boyden, and their colleagues have shown that stimulating neurons to produce gamma waves at a frequency of 40 Hz reduces the occurrence and severity of several Alzheimer's-associated symptoms in a mouse model of the disease. The researchers induced slow gamma waves using optogenetics, and by exposing lab mice to light energy at 40Hz, an approach they suggest could translate to human therapies.

"It's a pretty striking result that at one particular frequency with which they entrained the brain . . . they were able to reduce, in the mouse at least, all three hallmarks of Alzheimer's pathology," said Rudolph Tanzi, who leads genetics and aging research at Massachusetts General Hospital and was not involved in the work.

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The results are "important both for mechanistic study and also for potentially therapeutic developments," said Yadong Huang of the University of California, San Francisco, and the Gladstone Institute of Neurological Disease, who also was not involved in the work. The study suggests that "a slow-gamma deficit might be part of this [Alzheimer's disease] pathogenesis [and that] manipulating slow-gamma activity . . . could be a new way to suppress amyloid- β production and increase amyloid- β clearance," Huang added. Scientists have long hypothesized that decreasing amyloid- β accumulation could help reverse—or even prevent—symptoms of Alzheimer's disease.

Huang and colleagues previously reported that, during sharp-wave ripples in the hippocampus, patterns of brain activity thought to occur during memory replay and consolidation, gamma waves were disrupted in a mouse model of Alzheimer's disease. Gamma waves are also disrupted in the brains of people with Alzheimer's disease. But exactly how gamma waves contribute to this neurodegenerative pathology remains unclear.

To learn more, Tsai and Boyden first examined gamma waves in the hippocampi of Alzheimer's disease–model mice. Compared with those of control animals, the hippocampi of the model mice had fewer gamma waves during sharp-wave ripples, but gamma waves during theta waves were unaffected.

Next, the researchers optogenetically stimulated hippocampal neurons to produce gamma waves in Alzheimer's disease–model mice that had transgenically received both a light-responsive ion channel and a fluorescent label in their hippocampal neurons. Compared with control animals (model mice that were stimulated at stochastic frequencies or mice stimulated at 40 Hz that received the fluorescent label but not the ion channel), the gamma-stimulated mice had lower hippocampal levels of amyloid- β . Further experiments revealed that the mice that underwent gamma stimulation had reduced amyloid- β production. Additionally, gamma stimulation led microglia to shift toward their housekeeping function and engulf amyloid- β . The resulting amyloid- β reductions in gamma-stimulated animals were likely due both to lower production of the protein and to microglia clearing more of it away, the authors wrote.

"Optogenetics is very precise and therefore a good way to study how cell types and oscillations can be used in potential therapeutic prototyping," Boyden said during a press briefing this week (December 6). However the procedure, as performed on mice, drilling a hole in the skull and injecting a transgene-delivering virus into the brain. "When it came time to think about how we could translate this to humans, we started thinking about non-invasive strategies to achieve this result," said Boyden.

Their solution? Flickers of visible light—"like a strobe light, but faster," coauthor [Annabelle Singer](#) of Georgia Tech and Emory University said during the press conference—to stimulate not the hippocampus but the visual cortex.

After an hour of stimulation by an LED light flickering at 40 Hz to induce gamma waves, Alzheimer's disease–model mice had lower levels of amyloid- β than control model mice that were kept in the dark. The researchers repeated the experimental treatment, in older mice that had developed amyloid plaques, finding that the treatment—this time for an hour each day and for seven days—also led to plaque reduction. Finally, in a mouse model of tauopathy, mice subjected to the flickering-light treatment had lower levels of tau phosphorylation associated with formation of neurotoxic tangles.

It remains to be seen, however, whether gamma stimulation can prevent memory loss or rescue learning and memory deficits, Huang noted. Going forward, Tsai and colleagues hope to develop a technology based on this flickering-light treatment to treat Alzheimer's disease patients.

Tsai and some of her coauthors have started a company, Cognito Therapeutics (<https://www.cognitotx.com/>), to develop treatments for Alzheimer's disease, including technologies based on gamma stimulation via flickering light.

"While this is promising, we have many steps to go to translate these discoveries from mice into a therapy for humans using this noninvasive technique," Singer said during the press conference. "We need to do clinical studies in humans, and we're currently working hard to do that."

individual neurons can precisely time their spikes when driven by temporally fluctuating synaptic inputs (1). Narrowband oscillations mediated by inhibitory neurons are thought to be a key source of coordinated fluctuating discharges from input neurons, and they vary in power and frequency during wakeful behavior and sleep. Oscillations in the gamma range (30–80 Hz), thought to be mediated by fast-spiking inhibitory neurons expressing the calcium-binding

protein parvalbumin (2, 3), are modulated by the sensory environment (4–6), attention (7), and volition (8), as well as by specific memory tasks, causing changes in sensory responses (2) and information transfer (3) in the cortex.

The modulation is observed both in the oscillation power, which we define as the peak of a distinct “bump” in the power spectrum of the local field potential (LFP), as well as the oscillation frequency, which is the frequency at this peak in the power spectrum (5, 6). In current models of oscillations in neuronal networks, oscillations are regulated by stimulation of inhibitory neurons such that increasing stimulation mainly increases their frequency (9–11) or power (12). In the visual cortex, both the contrast and size of visual stimuli increase the stimulation to local inhibitory neurons (13, 14), but the former increases the frequency of gamma-range oscillations (6), and the latter decreases it (5). The power of gamma oscillations increases in the somatosensory, medial temporal (15), motor (8), olfactory (16), and primary visual cortex (5) with increased stimulation to local inhibitory neurons. However, the peak power of oscillations decreases with increased stimulation of inhibitory neurons with attention (17) in some cortical areas (7). In a third scenario, whereas the broadband power in the LFP signal increases with increasing visual contrast (6, 18), peak narrowband power shows no significant trend in response to increasing contrast (8), which is thought to increase the stimulation to the local inhibitory neurons (13). We show that these diverse experimental observations can be explained by the following hypothesis: The balance of two distinct pathways that activate local inhibitory neurons mediates bidirectional regulation of oscillations (Fig. 1A). We classify these pathways as monosynaptic (MS), those that make direct excitatory synaptic connections to the inhibitory neurons, and disynaptic (DS), those that act through the local excitatory neurons.

Relative strength of MS and DS stimulation to inhibitory neurons determines the power and frequency of oscillations in spiking activity. (A) Schematic of local network and the monosynaptic (solid black) and disynaptic (dotted black) pathways for stimulating the local inhibitory neurons. The model network architecture featured both excitatory (E) and inhibitory (I) neurons, with recurrent connections between and within E and I populations. (B) Example evolution of population oscillatory activity (thick traces in green palette) from baseline (1) by increasing stimulation to MS (2) and DS (3) pathways in a model network oscillating in the gamma range (30–80Hz). The data shown are for first 200 ms of an example trial. The relative strengths of individual pathways are indicated at the top of each panel. Raster plots show the spike times of all inhibitory (inside rectangle) and excitatory neurons. (C) Power spectrum of average population activity for the three cases shown in B (mean across 10 trials of 2-s duration). Dotted lines indicate the peak power and corresponding frequency of narrowband oscillations. (D) Variation in peak power and frequency of narrowband oscillations with the strength of MS and DS stimulation of inhibitory neurons. The stimulation strengths are normalized to the range of interest. (E) Modulation of peak power and frequency of oscillations with the relative strength of MS-to-DS stimulation. The power and frequency data were normalized to the range of values shown in D. The MS-to-DS stimulation ratios were calculated from the absolute values of the two inputs used in the simulation experiments.