

It has taken far too long for cognitive neuroresearchers to discover the fact that blue light waves can impact nearly all that is known about behavioral health. That being said, the following Chargaff maxim still applies.

“Science is wonderfully equipped to answer the question 'How?' but it gets terribly confused when you ask the question 'Why?'”

Erwin Chargaff - Member of Watson and Crick Team (1895-2002)

The issue of HOW blue light can impact behavioral health is explained in the following article. However, the facts for WHY the outcomes occur have never been elucidated until MCFIP applied quantum computation biology to the process.

Our findings are outlined in the document affixed to this article.

https://academic.oup.com/sleep/article-abstract/42/Supplement_1/A355/5451237

0884 Morning Blue Light Exposure Improves Sleep and Fear Extinction Recall in PTSD

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Abstract

Introduction

Sleep disruption is considered to be the “hallmark symptom” of post-traumatic stress disorder (PTSD). In addition to sleep deficits, patients with PTSD who undergo experimental fear

conditioning also typically show a deficit in the ability to recall extinction memories relative to those without the disorder. As memory consolidation is strongly influenced by sleep, we hypothesized that an intervention that regulates sleep and circadian rhythms (i.e., morning exposure to blue-wavelength light) might enhance consolidation and retention of learned extinction memory during a fear conditioning/extinction protocol among patients with PTSD.

Methods

Thirty-eight individuals with PTSD (18 male; Age=30.8, SD=9.0) underwent a well-validated fear conditioning and extinction protocol and were then randomly assigned to receive either BLUE (469 nm; n=20) or placebo AMBER (578 nm; n=18) morning light therapy for 30-minutes daily for 6-weeks. Participants returned after 6 weeks to undergo post-treatment extinction recall when exposed to the same previously conditioned stimuli. Extinction recall magnitude (ERM) at follow-up was calculated as the difference in skin conductance response (SCR) between the “extinguished” and the “never-extinguished” stimuli.

Results

BLUE light was associated with an increase in sleep duration relative to AMBER ($p=.016$). Based on the ERM, participants in the BLUE group showed sustained retention of extinction memory, while those in the placebo AMBER group showed a resurgence of the fear response after 6-weeks ($p=.016$). Moreover, retention of ERM was correlated with improvement in sleep on the Insomnia Severity Index for the BLUE ($r=.44, p<.05$) but not the AMBER group ($r=-.09, ns$).

Conclusion

Compared to placebo, 6-weeks of daily morning BLUE-wavelength light exposure was associated with increased sleep duration and greater retention of extinction learning in patients with PTSD. We speculate that increased sleep quantity or quality during the intervening weeks after learning led to greater consolidation of the fear extinction memory. Prominent exposure treatments for PTSD are based on principles of fear extinction, and our findings suggest that blue light treatment may facilitate treatment gains by stabilizing sleep in a manner that promotes consolidation of extinction memory.



The following explanation uses quantum computational modeling and it can be confusing without a step-by-step explicit demonstration of the

roles of the neuropeptides and consequences of imbalances as part of their interactions.

We used neuropeptide modeling and the interactions between the catecholamines in NPY that encompass the norepinephrine for focus and memory as the foundation from which long term memory can be stored and retain in the hippocampal cells. This process was used to correlated the energy (vibrations aka wave ripples) to activate the dopamine modulation of the trigeminal nerve.

Our theory has been supported by the recent FDA approval of trigeminal nerve stimulation of the trigeminal nerve to treated ADHD that can be verified as having excessive dopamine (impulsivity) as a primary factor.

Using quantum mechanics and the principles of $E=MC^2$, Einstein's Photoelectric Effect and optogenetics, we have connected the dots to develop an actionable theory for the use of vibration within the range of 256 nm - 280 nm (blue light waves) to stimulate the aromatic amino acids as the means of activating autophagy to prevent mutation of the catecholamines in short term memory for their ability to interface with BDNF in pancreatic polypeptide (PPY) that drives long term memory.

Bioinformatic search can verify the links between the catecholamines and short term memory with the correlation to long term memory.

https://neurosciencenews.com/eeg-ripples-working-memory-14234/?utm_source=feedburner&utm_medium=feed&utm_campaign=Feed%3A+neuroscience-rss-feeds-neuroscience-news+%28Neuroscience+News+Updates%29

Researchers identify hidden brain signals behind working memory

Summary: Artificially prolonging hippocampal sharp wave ripples improves working memory.

Source: NYU Langone

Making a specific type of brain pattern last longer improves short-term memory in rats, a new study finds.

Published online by the journal *Science* on June 14, the study addressed “working memory,” the temporary activation of brain cells that happens as we tour a new neighborhood, for instance, and remember our way around later that day.

Led by researchers at NYU School of Medicine, the new study finds that signals created by brain cells (neurons) – called sharp wave ripples – are longer by tens of milliseconds and capture more information when an animal is learning about a new place than when in a familiar setting.

When the research team artificially doubled the length of the signals involved in memory recall of the best route through a maze, rats with extended ripples were found to be 10-15 percent better at finding a sugary reward than rats without the manipulation.

“Our study is the first in our field that made artificial changes to intrinsic neuronal firing patterns in the brain region called the hippocampus that increased the ability to learn, instead of interfering with it like previous attempts,” says György Buzsáki, MD, PhD, the Biggs Professor in the Department of Neuroscience and Physiology at NYU School of Medicine. “After decades of study, we finally understand the mammalian brain well enough to alter some of its mechanisms in ways that may guide the design of future treatments for diseases that affect memory.”

The study results revolve around nerve cells, which “fire” – or bring about quick swings in the balance of their positive and negative charges – to transmit electrical signals that coordinate memories. Buzsáki’s team in recent years discovered that sets of neurons fire within milliseconds of each other in rhythmic cycles – creating closely connected sequences of signals that can encode complex information.

This observed pattern — where hippocampal cells in different parts of the circuit fire together briefly – creates “sharp wave ripples.” The patterns are named for their shape when captured graphically by electro-encephalography or EEG, a technology that records brain activity with electrodes.

Buzsáki says the ripples represent the ‘replaying’ and combining of fragments of learned information, part of the process that weaves them into an animal’s memory.

Within the Ripple

In the current study, the team designed experiments such that the correct route to get sugary water alternated between the left and right arms of a maze each time a rat was placed in it. To get their reward, the rats had to use working memory, recalling which way they had gone on the previous trial, and choosing the opposite way the next time.

Studies in recent years in many labs have established that hippocampal “place cells” encode each room, or each arm of a maze, when entered, and then fire again as rats or humans remember going there, or plan to go there again. The study authors recorded the firing of place cells as a rat performed the memory task in the maze, and predicted the route taken as reflected in the cell firing sequence captured in each sharp wave ripple.

To artificially double the duration of just the ripples made by rat’s brain cells during task-driven navigation, researchers engineered hippocampal cells to include light-sensitive channels. Shining light through tiny glass fibers activated neurons, adding more neurons to the naturally occurring sequence, thereby encoding more detail of the maze representation.

Importantly, the study also found that the extended ripples enabled slower-firing neurons to be recruited into their sequences. The authors’ past studies had shown these sluggish neurons to be better at changing their properties (more plastic) as something new is learned.

In contrast, faster firing partners in a ripple tended to start the sequence regardless of which route the rat took. Buzsáki’s team has been building the case that such ‘rigid’ neurons generalize across experiences, encoding the familiar (instead of the newfound) aspects of each newly encountered location.

“Our next step will be to seek to understand how sharp wave ripples can be prolonged by non-invasive means, which if we succeed would have implications for treating memory disorders,” says first author Antonio Fernandez-Ruiz, PhD, a postdoctoral fellow in Buzsáki’s lab.

Along with Buzsáki and Fernandez-Ruiz, authors from the New York University Neuroscience Institute were first author Azahara Oliva, Eliezyer Fermino de Oliveira, Florbela Rocha-Almeida, and David Tingley.

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ABOUT THIS NEUROSCIENCE RESEARCH ARTICLE

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[“Long-duration hippocampal sharp wave ripples improve memory”](#). Antonio Fernández-Ruiz, Azahara Oliva, Eliezyer Fermino de Oliveira, Florbela Rocha-Almeida, David Tingley, György Buzsáki.

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Abstract

Long-duration hippocampal sharp wave ripples improve memory

Hippocampal sharp wave ripples (SPW-Rs) have been hypothesized as a mechanism for memory consolidation and action planning. The duration of ripples shows a skewed distribution with a minority of long-duration events. We discovered that long-duration ripples are increased in situations demanding memory in rats. Prolongation of spontaneously occurring ripples by optogenetic stimulation, but not randomly induced ripples, increased memory during maze learning. The neuronal content of randomly induced ripples was similar to short-duration spontaneous ripples and contained little spatial information. The spike content of the optogenetically prolonged ripples was biased by the ongoing, naturally initiated neuronal sequences. Prolonged ripples recruited new neurons that represented either arm of the maze. Long-duration hippocampal SPW-Rs replaying large parts of planned routes are critical for memory.