

Optimizing Brain Biology Through Near-Infrared-Induced Mitochondrial Melatonin Synthesis

Analysis by [Dr. Joseph Mercola](#)

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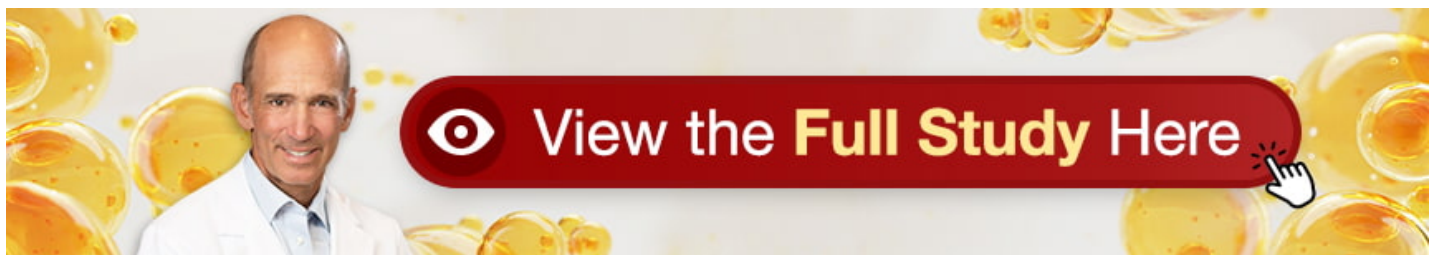
STORY AT-A-GLANCE

- › Near-infrared (NIR) light absorbed by mitochondria triggers local melatonin production, creating an antioxidant cascade that offers neuroprotective benefits independent of the sleep-related melatonin system
- › The brain is especially vulnerable to oxidative damage due to high energy demands, fragile cell membranes, limited cleanup mechanisms, and age-related decline in melatonin levels
- › Two coordinated defense systems activate in response to NIR light – one reduces damaging oxygen byproducts inside mitochondria, while the other protects cell membranes from toxic fat-breakdown products that spread cellular damage
- › Light exposure follows a "Goldilocks principle." Moderate doses produce benefits, while insufficient or excessive exposure yields no effect or may even cause harm, especially in sensitive brain tissue
- › Alzheimer's disease represents a primary application since patients show early mitochondrial dysfunction and dramatically reduced melatonin levels, though human trials remain limited and inconclusive

In the past, I've written about the benefits of [near-infrared \(NIR\) light and its role in activating photobiomodulation](#), which is a therapy wherein exposure to specific light wavelengths stimulate beneficial processes in your tissues. For example, it can promote

collagen production,¹ which can help rejuvenate skin health. Moreover, it has anti-inflammatory and wound-healing properties, making it useful for faster tissue repair and recovery from injuries.² But that's not all NIR light can offer.

Recently, I've been doing a deep dive into the published literature involving NIR light, **mitochondrial melatonin**, and its related biological processes. In my paper, "Optimizing Brain Biology Through Near-Infrared-Induced Mitochondrial Melatonin Synthesis," now published in the journal Cureus, I examine how NIR can be used to activate mitochondrial melatonin synthesis to create an antioxidant cascade that offers neuroprotective benefits. You can view the published study below.



For your convenience, I've also simplified it into a format that can be understood easily without having to learn complicated scientific terms. You can download that simplified version here. The remainder of this article also summarizes the key points of this work.



Introduction and Methodology

Your brain is a power-hungry organ. Even though it only accounts for 2% of your body by weight, it uses 20% of your body's energy. That heavy workload happens mostly inside the neurons, each containing thousands of mitochondria.

- **High energy use comes with a cost** – When your cells make energy, they also create oxidative byproducts that can damage nearby parts of the cell. While this process is normal, it becomes a bigger problem when the system is already under strain.
- **Your diet creates another layer of weakness** – Many brain cell membranes are embedded with **polyunsaturated fats (PUFs)** that make them more susceptible to lipid peroxidation.

On top of that, your brain has fewer built-in cleanup tools than other tissues. Specifically, mitochondria lack catalase and need to lean more on glutathione to handle certain kinds of damage. If that glutathione-based system is overwhelmed, trouble spreads fast.

- **Your environment also shapes brain health** – More people are now spending more time indoors than humans did for most of history, which means you get far less NIR light from the sun. This has implications that go beyond vitamin D synthesis, because NIR light is the signal mitochondrial chromophores that set the stage for endogenous neuroprotection.
- **Melatonin is another piece of the story, and it fades with age** – Melatonin from your brain declines over time, and in Alzheimer's disease, the drop is especially strong – showing up even before clear symptoms in some cases.
- **Your mitochondria are key to better brain health** – These energy factories contain the tools needed to produce melatonin right where damage starts. From my research, I discovered that melatonin made inside the cells follow a different pattern than the nighttime melatonin tied to sleep. In this model, melatonin isn't only a sleep hormone – it's a shield produced inside the parts of the cell that need it most.

The Proposed NIR-Melatonin-Glutathione Cascade

The core hypothesis of my paper can be summarized as follows: NIR light starts a protective cascade inside your cells that ends with stronger neuroprotection via mitochondrial antioxidant defense. Basically, NIR light interacts with a key energy-related part of your cells, triggering changes that support your cells' own protective systems.

- **The first step in the chain begins where your cells make energy** – NIR light is absorbed in the cytochrome c oxidase (CCO, also known as Complex IV) of your mitochondria. CCO is a core enzyme in your mitochondria's energy-producing machinery. It sits at the end of the electron transport chain (ETC) and controls the final step of cellular respiration, where electrons combine with oxygen to drive ATP production.

Because this step sets the pace for energy flow, CCO acts as a metabolic gatekeeper rather than a passive component.

CCO also happens to contain metal centers – copper and heme groups – that respond to specific wavelengths of light. NIR light interacts with these metal centers, altering how the enzyme functions. This makes CCO light-responsive. When NIR light reaches CCO, it dislodges nitric oxide (NO), a small molecule that can bind to the enzyme and slow electron flow. Once NO releases, electron transfer resumes, oxygen use improves, and mitochondrial energy production increases almost immediately.

- **How melatonin is made inside your cells** – NIR also triggers melatonin production inside your mitochondria. Your mitochondria contain the full enzyme machinery to synthesize melatonin locally, creating a rapid antioxidant supply exactly where oxidative stress occurs. This mitochondrial melatonin does not follow a daily rhythm, distinguishing it from nighttime melatonin released into the bloodstream
- **The speculative part of the paper's hypothesis** – Light-driven energy activity increases carbon dioxide inside the cell. That change sets off a chain involving bicarbonate and calcium signals, which flip on an internal messenger system. That

directly activates the enzyme that drives melatonin production inside the mitochondria.

- **What matters most is the outcome** — If the mitochondria can make their own melatonin, then each one can protect itself right where damage begins. This local defense is far more efficient than relying only on antioxidants delivered from elsewhere in the body.

Melatonin, then, strengthens another major defense system: glutathione. These two work together — melatonin helps restore glutathione after it neutralizes damage, and glutathione helps preserve melatonin when stress levels are high. This creates a stronger and longer-lasting neuroprotective shield.

- **A key limiter in this process** — Glutathione levels only rise when your body has enough basic building blocks, especially **glycine** and cysteine. As you age, these building blocks drop, which weakens this defense even if the signal is strong. Restoring these nutrients corrects age-related glutathione loss.

Coordinated Mitochondrial Defense: The SIRT3-SOD2 and Glutathione-GPX4 Axes

Your mitochondria need more than one line of defense because damage doesn't happen in just one place. Inside brain cells, two main threats dominate. First, dysfunctional energy production in the ETC leads to excess superoxide, a reactive oxygen species that damages mitochondrial proteins and DNA.

Second, fragile cell membranes rich in PUFs are vulnerable to lipid peroxidation. This process produces toxic byproducts like 4-hydroxynonenal (4-HNE), which spreads damage throughout the cell and accelerates neurodegeneration. These two threats — internal oxidative stress and membrane breakdown — require coordinated antioxidant systems to keep damage in check.

- **How the first defense line works** – Melatonin activates the SIRT3-FOXO3a pathway, a core mitochondrial defense system. FOXO3a is a transcription factor that turns on genes involved in antioxidant defense and autophagy, including the enzyme SOD2 that neutralizes damaging superoxide radicals at their source. This signaling axis enhances mitochondrial protection and improves cell survival across models of cardiac, intestinal, and lung injury.

Reduced FOXO3a activity is linked to Alzheimer's disease, where areas near amyloid plaques show weaker antioxidant signaling and greater metabolic stress.

- **The second defense line targets your cell membranes** – When fats in cell membranes oxidize, they produce toxic byproducts that can trigger a destructive process called ferroptosis. GPX4 is the key enzyme that neutralizes these fat-based toxins. Without it, membrane damage accelerates and cells fail rapidly.

Melatonin strengthens this system by boosting glutathione production, enhancing GPX4 expression, and supporting the enzymes that regenerate used glutathione. Together, these actions help keep your cell membranes stable under stress.

- **The reason coordination matters becomes clear when both systems are viewed side by side** – One pathway controls damage inside the mitochondria, while the other stops damage from spreading through the cell's outer layers. In Alzheimer's disease tissue, both systems show signs of failure at the same time.

Automitocrine Signaling: How Cells Use Their Own Melatonin to Defend Mitochondria

Your cells have the ability to produce melatonin locally, right inside the mitochondria – the power centers most vulnerable to metabolic stress. This process doesn't depend on signals from the pineal gland or bloodstream. Instead, melatonin acts at its point of origin to protect against the very threats that triggered its production. This is the basis of the automitocrine hypothesis, a self-contained cellular defense system.

- **How local melatonin signaling works** – Unlike circulating melatonin, which follows a circadian rhythm and travels system-wide, mitochondrial melatonin is made on-site and used immediately. This allows rapid, targeted protection during cellular stress, such as when oxidative byproducts spike or calcium levels surge. Think of it as an internal emergency system that flips on when the mitochondria sense danger.
- **Receptor activation at the source** – Evidence suggests that melatonin receptors called MT1 are located directly on the inner mitochondrial membrane, at least in brain cells. When mitochondrial melatonin binds to these receptors, it triggers survival pathways that stabilize energy production and improve resilience under stress. One of the most important actions is blocking cytochrome c release – a key early step in the apoptotic (cell death) cascade triggered by calcium overload.
- **Why it matters: calcium and cell death** – Mitochondria can only buffer so much calcium before they fail. Excess calcium opens pores in the mitochondrial membrane, allowing cytochrome c to leak into the cell. Once that happens, a death program is initiated. Activating MT1 receptors through automitocrine melatonin signaling prevents this leak, preserving cell integrity even under extreme stress.

This mechanism has been observed in neural models exposed to glutamate toxicity, which mimics excitotoxic brain injury.

- **Where NIR comes in** – NIR light activates this entire system. Exposure to NIR stimulates CCO, a mitochondrial enzyme that enhances oxygen consumption and ATP production. That same boost in mitochondrial function appears to promote local melatonin synthesis and amplify its protective effects. In animal studies, NIR exposure raised levels of mitochondrial melatonin, reduced markers of oxidative stress, and improved cell survival.

The Circadian Dimension: Timing and the Dual Melatonin System

When it comes to light exposure, knowing the "when" is important, not just that it happens in the first place. Your overall exposure changes throughout the day and seasons, and your body's internal timing systems interact with this process.

- **The role of sunlight as a delivery system** – Sunlight provides a natural, daily source of NIR light, but the amount your body receives depends on several factors. NIR is strongest around midday when the sun is highest in the sky. In contrast, early mornings, late afternoons, winter months, cloud cover, clothing, and limited skin exposure all reduce your NIR dose.

Your body doesn't need an extreme amount of NIR to benefit – just the right amount at regular intervals. Like many biological signals, NIR follows a dose-response curve. Too little does nothing. Too much can backfire. The goal isn't to max out exposure, but to build a rhythm of moderate, repeatable doses. This is how sunlight supports mitochondrial health most effectively – through a consistent, Goldilocks-style delivery system.

- **Time-of-day effects on cellular responses to NIR light** – Evidence from animal studies shows that the cell structures responding to NIR light are more active at certain times of day. This means your cells are not equally responsive at all hours. While this effect has been clearly observed in specific brain regions in animals, broader confirmation in the human brain is still needed.
- **The dual melatonin system hypothesis** – There are two functionally distinct melatonin systems. The first one follows a daily rhythm tied to darkness and sleep. This melatonin enters your bloodstream and brain fluid at night and helps coordinate sleep, repair, and waste removal while you rest.

The second source works differently. This melatonin is produced locally inside cells and does not follow a day-night rhythm. It is a rapid response system designed to protect cells exactly where stress occurs.

- **The two systems go hand in hand** – These two melatonin systems complement each other. Daytime light supports immediate, local protection inside cells, while nighttime darkness supports whole-brain coordination and cleanup during sleep. One system handles fast defense. The other handles long-term maintenance.

The Biphasic Dose-Response Relationship: The Goldilocks Principle

As hinted earlier, your body responds best to the right amount of stimulation, not the most. After reviewing the literature, the findings point to a clear pattern: Biological responses to light follow a bell-shaped curve. Too little light creates no change. A middle range produces benefits. Too much light shuts the system down or causes stress.

- **The pattern shows up repeatedly across different experiments and outcomes** – Whether researchers look at energy production, cell survival, or protective signaling, the same rule applies. The system responds when stimulation lands in a narrow Goldilocks zone. Outside that, the response fades or flips in the wrong direction.

This makes appropriate dosing particularly important when it comes to using NIR for brain health, as brain cells are particularly sensitive and have a narrower therapeutic window than many other tissues. This helps explain why results vary so widely between studies using different settings.

- **Why timing and spacing sessions matter** – Some effects of light exposure last for more than a day. When sessions are stacked too closely together, the system doesn't have time to reset. That overlap can weaken results or push exposure into the harmful range. Very few studies have carefully tested spacing, but existing data show that recovery time matters.

It's important to note that this is not a flaw of light-based biology. It's a safety feature. Your cells use small concentrations of ROS as signals to turn on protection. When stress stays low and controlled, those signals strengthen the system. When

stress becomes too strong, protective systems shut down to avoid damage.

- **At the center of this response is how your cells handle oxidative stress** – Small, brief increases act like alarms that trigger repair and defense programs. Large increases can overwhelm cleanup systems and damage cell parts instead. NIR light works by nudging this balance, not blasting through it.

This balance also explains why results differ so much between tissues. Muscle, skin, and brain all absorb and respond to light differently. The brain's tight safety margin makes precise dosing especially important.

Potential Risks and Adverse Effects

While the hypothesis presented in my paper shows promise in the field of neuroprotection, it's important to examine what is known – and not yet known – about safety. Case in point, I also reviewed existing human trials and theoretical concerns to determine where caution is still warranted.

- **NIR light exposure is relatively safe** – Very few reported side effects in human studies so far have been reported. However, limited long-term data leaves important questions unanswered.

Most reported side effects in human brain studies are mild and short-lived. Participants occasionally report headaches. These effects typically resolve quickly and do not require medical intervention. Across studies reviewed, serious adverse events are rare.

- **Large clinical trials also support this safety profile** – In a major stroke trial that tested NIR light shortly after brain injury, researchers found no increase in harmful outcomes compared to standard care. Even though the treatment did not improve recovery, it did not introduce new safety risks.

- **Theoretical concerns** – This pertains to tumor biology. Because NIR light boosts energy activity inside cells, it could also stimulate the growth of cancer cells. For this reason, people with known or suspected brain tumors have not been adequately studied and need to be treated with special care.

Alzheimer's Disease: A Primary Application

Alzheimer's disease stands out as the most logical place to explore the hypothesis of my research – specifically, whether the biological problems seen in this disease line up with the protective systems described earlier. Based on the literature, the same cell systems weakened in Alzheimer's are the ones the light-driven defense pathway targets.

- **Reframing how Alzheimer's is viewed** – Instead of starting with the premise of protein buildup alone, another angle of pathology is the early breakdown in the brain's mitochondria. This loss starts a downward spiral – less energy, more damage, and further decline.
- **As energy output drops, unstable ROS rise** – Those byproducts damage the mitochondria further, creating a loop that drives disease forward. Interrupting this cycle early matters far more than trying to clean up damage later.
- **Melatonin loss adds another layer** – Published literature shows that people with Alzheimer's disease have dramatically lower cerebrospinal melatonin levels – around 20% of age-matched control levels. This drop appears very early, sometimes before symptoms are fully apparent. In addition, structural changes in the pineal gland are more common in Alzheimer's.

While I do not claim melatonin loss causes Alzheimer's outright, its early vanishment removes a key line of defense. Because melatonin protects brain cells from both protein stress and energy-related damage, restoring its influence becomes a logical target.

- **A review of current evidence** – Small trials and case reports show improvements in thinking ability, daily function, and brain blood flow. More recent controlled studies involving older adults report gains in memory along with increases in brain-supporting growth factors. These findings are encouraging but, again, are not definitive.

In another example, which was a large trial, using NIR light after acute stroke did not improve outcomes, even though it remained safe. Several factors can explain why no benefit occurred, such as a severely compromised mitochondrial function, which limited CCO-mediated responses.

Frequently Asked Questions (FAQs) About Near-Infrared Light and Neuroprotection

Q: How does near-infrared (NIR) light protect the brain?

A: NIR light is absorbed by cytochrome C oxidase (CCO) in your mitochondria, which improves cellular energy production and triggers local melatonin synthesis. This pathway works with glutathione to create an antioxidant cascade that defends brain cells against oxidative damage right where it occurs.

Q: What makes the brain particularly vulnerable to oxidative damage?

A: The brain uses 20% of your body's oxygen and energy despite accounting for only 2% of your total body weight. This high metabolic activity generates harmful byproducts, while brain cell membranes contain fragile fatty acids prone to damage due to a poor diet. Additionally, mitochondria lack certain cleanup enzymes and need to rely heavily on glutathione, which declines with age.

Q: What is the dual melatonin system hypothesis?

A: There are two proposed melatonin systems – one follows a day-night rhythm tied to sleep and enters the bloodstream at night for whole-brain repair, while the other is produced locally inside mitochondria in response to stress, providing rapid, targeted protection independent of circadian timing.

Q: Why does dosing matter so much with NIR light therapy?

A: Biological responses follow a Goldilocks principle – too little light produces no effect, while too much can shut down protective systems or cause harm. Brain cells have an especially narrow therapeutic window, which helps explain inconsistent results across studies using different settings.

Q: Is NIR light therapy proven to treat Alzheimer's disease?

A: Not yet. While Alzheimer's patients show dramatically reduced melatonin levels and the same defense systems NIR targets are impaired in the disease, the hypothesis hasn't been confirmed in humans. Small trials show promise, but large, long-term clinical studies are still needed.

Sources and References

- [1 Platinum Therapy Lights, February 24, 2023](#)
- [2 Platinum Therapy Lights, November 1, 2024](#)