NATURAL LIGHT

Natural (full spectrum) light received by the eyes is a powerful stimulus to the hypothalamus. The hypothalamus, in turn, regulates hormone production by every gland in the body, and regulates the tie-in between the hormonal system, the immune system, and the autonomic nervous system (=== maintains Sympathetic/ Parasympathetic Balance and reduces ImmunoNeuroEndocrine Stress).

Natural light is the primary control of our circadian physiological cycles. Humans are diurnal animals and our biological clock synchronizes all physiological functions. Light is the main environmental cue used by our biological clock. [NUTRI-SPEC's Diphasic Nutrition Plan is designed to maximize the amplitude of our diurnal cycle.]

Natural Law has given humans the capacity to adapt to the insufficient sunlight at higher latitudes. That is why people in Northern climates have fair skin, while those living closer to the equator have pigmented skin. But that adaptation applies only to sunlight as a vitamin D source. There are no adaptations for a deficiency of full spectrum light entering the eyes.

What Natural Law could not account for is the number of hours human beings spend indoors, with exposure to no natural light during those indoor hours. Nearly all women in Scandinavian countries suffer from osteoporosis, and many men have significant bone density loss as well. It is advisable that if adults and children spend many hours outdoors, they take 1,000 extra vitamin D during December, January, February, and March. If they do not spend many hours outdoors, then they should take 1,000 units of vitamin D eight months of the year, and 2,000 during December through March.

Even those of us who do not wear eye glasses or contact lenses are at risk for a deficiency of full spectrum light received by the retina simply because we spend entirely too much time indoors where we do not get adequate intensity or quantity of natural light --- and --- when we are indoors, much of that time we are exposed to artificial light of unnatural light frequencies.

Wearing contacts obviously compounds that tendency to deficiency since even in the limited amount of time those wearing contacts are exposed to natural light, they are not receiving it through the eyes without some wavelengths being filtered out. A far greater crippler of physical and mental health than contact lenses is tinted contact lenses. A high ratio of blue and/or green frequencies relative to red and yellow frequencies is the most devastating to physical and mental well-being.

A study sponsored by the Environmental Protection Agency finds that Americans spend about 90% of their time either indoors or in vehicles. Far too much --- far too unnatural. The stress involved is evident by looking at a study

from the University of Exeter Medical School in England. That study finds that spending a minimum of 2 hours a week in natural full spectrum light is associated with improved health. The study shows that spending time outdoors improves both our physical and mental health by several objective measures. Part of the benefits are probably from Vitamin D increase, but much of it has to do with escaping the unnatural light of TV and video screens with their high percentage of blue wavelengths.

An article published in Current Biology in 2017 shows that going camping for a week without electronics resets the body's internal biological clock and improves sleep. In that study, volunteers spent a week outdoors without any electronic devices or artificial lights. After several days, the participants found themselves going to bed earlier and getting up earlier. Not only had they effectively reset their sleep/wake cycle, but they were more alert and better rested as a result. --- Even a weekend of camping without electronics --- provides similar benefits. A breakdown of the data shows that light exposure was the biggest factor (more than fresh air, increased activity level and change in diet).

MSH (Melanocyte Stimulating Hormone); Immune System Benefits

I have seen studies suggesting that melanocyte activity is induced by exposure to light, not on the skin but received through the eyes. Whether the hormonal response to light received by the eyes involves direct stimulation of MSH, or simply a downstream activation of melanocytes, I have not seen anyone speculate. --- Even without having that mechanism specifically defined, we must maximize exposure to natural light, and to particularly allow light to enter the eyes unfiltered by contact lenses or eyeglasses.

Originally it was thought that αMSH is produced in the hypothalamus (or in the intermediate lobe of the pituitary in some animals), and that production is stimulated by exposure of keratinocytes (skin) to sunlight. However, other research shows that αMSH production is stimulated by exposure of not only the skin but also the eye to UVB. One study on mice shows that MSH in the blood increases in normal mice after UVB irradiation to either the eye or the ear. ----- (Hiramoto, et al. Ultraviolet UVB radiation to the eye induces pigmentation in the epidermis ….. Clin Exp Dermatol, 2011 Aug.)

The earliest reference I have found regarding MSH stimulation via the eyes is: Quevedo, et al. Role of light in human skin color variation. Am J Phys Anthropol, 1975 Nov. ----- This study indicates that skin pigment is regulated by radiation received through the eyes, as opposed to the skin.

There are many pro-inflammatory cytokines (of both the T-helper 1 family and the T-helper 2 family) produced by lymphocytes, macrophages, eosinophils, and mast cells that contribute to either systemic or localized inflammation, and, contribute to autoimmunity, and, are causative factors in virtually all

chronic diseases of aging. Many, if not most, of these pro-inflammatory cytokines are inhibited by alpha-MSH, and are thus kept within physiological limits by retinal exposure to full spectrum light. ----- Physiological production of alpha-MSH is clearly shown in the literature to be dependent upon full spectrum light in general, and ultraviolet light in particular, and is activated via retinal reception as opposed to, or in addition to, skin exposure.

Another interesting aspect of MSH activity is that it is modified in some respects by the Th1 inflammatory cytokine, Interleukin-1. I cannot help but wonder if some of the autoimmune diseases associated with mold exposure, (since mold exposure affects MSH), are related to this connection between Interleukin-1 and MSH, and may contribute to rheumatoid arthritis, insulin-dependent diabetes, alopecia, Hashimoto's thyroiditis, and other Interleukin-1-mediated autoimmune diseases. [Note: Interleukin-1-mediated inflammation is also induced by excess estrogen.]

A study by Hiramoto (Hiramoto et al. UVB irradiation of the eye activates a nitric oxide-dependent hypothalamo-pituitary pro-opiomelanocortin pathway and modulates functions of alpha-MSH-responsive cells. J Invest Dermatol, 2003 Jan.) may have been the first to specifically describe the stimulation of MSH via eye exposure to UVB. Hiramoto showed "that a signal evoked by ultraviolet B irradiation of the eye is transmitted in a nitric oxide-dependent manner through the ciliary ganglia involving the first branch of the trigeminal nerve to the hypothalamo-pituitary pro-opiomelanocortin system, resulting in upregulation of MSH hormone secretion and consequent stimulation of melanocytes in the skin."

----- What I have not seen is a quantification of what percentage of MSH stimulation in a critter (whether man or mouse) exposed to UVB is effected via the eye and how much through the skin. --- In other words, if you send your patients to a tanning bed (with eyes covered), will they get only a small amount of MSH stimulation relative to what they would get if their eyes were exposed to light, or, is the MSH production associated with exposure to the eyes small relative to exposure to the skin?

Lucas, et al. Sun exposure and vitamin D are independent risk factors for CNS demyelination. Neurology. 2011. ----- Shows that sunlight benefits autoimmune disease such as MS by some mechanism in addition to the production of vitamin D. So….

A connection between vitamin D and MSH? --- If the connection between Th1 (Interleukin-1) over-activation and MSH is as it seems it might be, and, since vitamin D is therapeutically beneficial for all the Th1-medicated autoimmune diseases, it might be reasonable to suspect that there is a connection between vitamin D and MSH, and that the common ground has to do with ultraviolet light exposure. I have not seen any studies making that direct link, but is suggested in this study:

Ponsonby, et al. UVR, vitamin D and 3 autoimmune diseases – Multiple sclerosis, Type I diabetes, Rheumatoid arthritis. Photochem Photobiol, 2005 Nov-Dec.

Another connection between Vitamin D, inflammatory cytokines and the TH1-medicated autoimmune diseases are as seen in MS ….

Herpes virus infection correlates with MS risk since some herpes viruses such as EBV produce an IL-10-like cytokine that may induce a dysfunction of IL-10-producing regulatory lymphocytes, thereby undermining the protective functions of sunlight and vitamin D. That IL-10-like cytokine could elicit a host immune response capable of neutralizing or depleting IL-10, or, it could compete with IL-10 but fail to perform an essential IL-10 function. In any case, a lack of sunlight exposure accompanying a herpes virus infection might synergize to induce a defect in IL-10-producing regulatory lymphocyte function.

Natural light (partially from Vitamin D, and partially from activation of the retino-hypothalamic system) prevents many other diseases in addition to MS. Depression is the condition with the most studies showing response to natural light. But natural light exposure is also associated with lower rates of pancreatic cancer. It also reduces blood pressure and reduces the risk of Type II Diabetes and all other aspects of Metabolic Syndrome. A study in Sweden even finds that people who spend more time outdoors live longer. There is also a huge boost to endorphins from spending time in the sun.

Mood; Seasonal Affective Disorder (SAD); Sleep Disorders; Pineal Gland

Deficient full spectrum light is a major cause of mood disorders --- including both depression and anxiety. It is also the cause of Seasonal Affective Disorder (SAD). Natural light is also essential to maximize mental alertness and cognitive performance. Sleep disorders are also associated with inappropriate timing of natural light, and also with a prevalence of unnatural (blue spectrum) light --- and both depression and cognitive dysfunction can also occur secondary to that lack of sleep.

One informative study is ----- Golden, et al. The Efficacy of Light Therapy in the Treatment of Mood Disorders. Am J Psychiatry, 2005 Apr. ----- Meta-analyses reveal a significant reduction in depression severity associated with bright light treatment --- both depression associated with Seasonal Affective Disorder, and non-seasonal depression. Bright light therapy administered as dawn simulation (early morning) is particularly effective.

Another study shows that not only is bright light exposure essential to maintain healthy physical and mental functions, but the entire profile of light-dark exposure influences the circadian clock, as well as mood, sleep, and vigilance quality. It is not just the brightness of the light, but the natural

spectrum of light that is clinically and physiologically beneficial. And, just as important as bright light during daylight hours is darkness at night. ----- Dumont, et al. Light Exposure in the Natural Environment: Relevance to Mood and Sleep Disorders. Sleep Med, 2007 Sep.

A study that takes a closer look at SAD shows that nearly all mood disorders (not just SAD) are significantly affected by disturbances of sleep --- and --- sleep disturbances are a primary symptom of poor quantity/quality of light exposure. Studying SAD patients, it is found that in Winter they show decreased sleep efficiency, decreased delta sleep percentage, and increased REM density --- both in comparison with themselves in summertime, and with themselves after 9 days of light therapy, and with healthy controls. ----- Anderson, et al. Sleep in Fall/Winter Seasonal Affective Disorder: Effects of Light and Changing Seasons. J Psychosom Res, 1994 May.

Bright light therapy, and particularly full spectrum light exposure, is beneficial for adults with attention-deficit hyperactivity disorder. In adults with ADHD, there is a delayed sleep/activity rhythm and/or seasonal mood symptoms that contribute significantly to the fundamental pathology and the degree of disability. Light therapy for ADHD adults showed a reduction in the Brown Adult ADD Scale and the Conners' Adult ADHD Scale, as well as improvement in the Hamilton Depression Rating Scale, as well as improvements in various neuropsychological tests. There was also a shift toward earlier circadian preference. Morning light therapy was associated with a significant decrease in both subjective and objective measures of core ADHD, improved mood, and a normalization of circadian cycle. ----- Rybak, et al. An Open Trial of Light Therapy in Adult Attention-deficit/Hyperactivity Disorder. J Clin Psychiatry, 2006 Oct.

Appropriately timed exposure to bright light can reset the timing of the sleep/wake cycle and improve both sleep quality and daytime alertness. Bright light therapy restores the circadian system so that it is appropriately synchronized with the solar day --- thus ensuring alertness and performance peak during daytime hours, and consolidated sleep at night. ----- Gooley JJ, et al. Treatment of Circadian Rhythm Sleep Disorders with Light. Ann Acad Med Singapore, 2008 August.

As part of the natural diurnal rhythm, sunlight suppresses melatonin. But the strongest response occurs in the blue wavelength of the spectrum. Exposure to light high in the blue wavelengths from fluorescent lights, from "cool" indoor lighting, and from computer and TV screens during the evening hours, unnaturally suppresses melatonin at the time of day when the natural diurnal cycle calls for it to be increasing. The result is sleep disorders, and inappropriately enhanced alertness. ----- West, et al. Blue Light from Light-emitting Diodes Elicits a Dose-dependent Suppression of Melatonin in Humans. J Appl Physiol, 2011 Mar.

Consider a study by Revell, et al. Predicting Human Nocturnal Non-visual Responses to Monochromatic and Polychromatic Light with a Melanopsin Photosensitivity Function. Chronobiol Int, 2010 Oct. ----- This study confirms that blue light sensitivity of circadian, neurobehavioral, neuroendocrine, and neurophysiological responses is attributed to melanopsin. Disturbances in melanopsin from blue light affects nocturnal melatonin levels, auditory reaction time, and subjective alertness and mood.

Since melatonin is such a popular supplement peddled by the ignorant and unscrupulous health food industry, it is important that both its benefits and its harmful effects are understood. Melatonin (like prolactin and estrogen) is a catabolic stress hormone. It is extremely damaging --- increasing catabolic oxidative damage and accelerating the aging process. As you read above in the study by West, one of the most critical benefits of natural sunlight is that it suppresses melatonin. However, during the late evening hours, it is essential that a very small physiological quantity of melatonin be produced by the pineal gland. That small surge of melatonin associated with the absence of light is a critical part of maintaining a healthy diurnal cycle, supporting restorative sleep, and preparing for arising the next morning with alertness and an elevated mood.

The problem, as West points out, is that blue wavelengths of light in the evening suppress melatonin production --- leading to enhanced alertness at an inappropriate time --- and a feeling of "stress", while it inhibits the onset of the nocturnal portion of the diurnal cycle.

So, if our natural production of melatonin is being inhibited by our computer and TV screens and our fluorescent and "cool" (blue wavelength) indoor lights, does it make sense to follow the health food industry recommendation to take supplemental melatonin to facilitate sleep? Occasionally yes --- but only in very, very small physiological quantities, and only if insomnia or failure of restorative sleep is a major concern. The problem with melatonin supplementation is that it is always recommended in harmful megadoses --- and --- many of us, despite having our melatonin production delayed by late evening exposure to blue wavelength light, end up producing too much melatonin over the course of the night. The catabolic pro-aging effects of this excess melatonin are a major concern ….

Many studies show a mood depressing effect of melatonin ----- Karman JS, et al. Negative effects of melatonin on depression. Am J Psychiatry, 1976. Melatonin exacerbated symptoms of depression in all patients, as well as causing a loss of sleep and weight and a drop in oral temperature. ----- And consider this more recent study on mice showing that inhibiting melatonin cured depression ….

----- Dubocovich ML, et al. Eur J Pharmacol, 1990. Antidepressant-like activity of the melatonin receptor antagonist in the mouse behavioral despair test.

----- Another study shows that melatonin inhibits dopamine, one of the brain neurotransmitters associated with elevated mood. ----- Dubocovich, ML. Melatonin is a potent modulator of dopamine release in the retina. Nature, 1983.

That there is an increased melatonin production response to stress, and that stress response is even more powerful than the activation of melatonin by exposure to darkness, is shown in a study by Lynch HJ, et al. The adrenal medulla may mediate the increase in pineal melatonin synthesis induced by stress, but not that caused by exposure to darkness. J Neural Transm, 1977.

The stress response to melatonin in susceptible individuals will cause an increase in blood pressure. ----- Weinstock M, et al. Seasonal variation in the development of stress-induced systolic hypertension in the rat. J Hypertens Suppl, 1985. ----- This study concluded that systolic blood pressure increased significantly after exposure to darkness and was reversed by exposure to light. The conclusion was that this phenomenon could be explained by the influence of melatonin on amplifying the sympathetic pressor activity by stress hormones adrenaline, corticosterone, and prolactin.

----- Melatonin also increases the stress hormone estrogen, while inhibiting the protective and youth-enhancing hormone progesterone. ----- Sirotkin AV. Direct influence of melatonin on steroid secretion by granulosa cells isolated from porcine ovaries. J Pineal Res, 1994. ----- It was found that melatonin inhibited progesterone and stimulated estradiol secretion.

Excess melatonin is not only a systemic catabolic stress hormone, but also can actually cause degenerative damage to the eye. ----- Arushanian EB, et al. Melatonin lowers the threshold of light sensitivity of the human retina. Eksp Klin Farmakol, 1999. ----- After chronic use of melatonin (3 mg before bedtime for 14 days), campimetry showed a significant decrease of the threshold of brilliance sensitiveness of the retina in the absence of authentic changes of the sensory motor response latency in test subjects. ----- That is to say that those who suffer from extreme light sensitivity are likely to be manifesting the effects of excess melatonin. ----- But it is not just light sensitivity that is caused by melatonin stress, but actual damage to the eye ….

Wiechmann AF, et al. Melatonin increases photoreceptor susceptibility to light-induced damage. Invest Ophthalmol Vis Sci, 1992. ----- Melatonin administration increased light-induced photoreceptor damage, while pinealectomy in experimental animals protected photoreceptors from light-induced damage.

[See the NUTRI-SPEC article, *You Need the Truth About Melatonin Supplements*.]

Blue light excess is the particularly damaging aspect of unnatural light exposure. And like melatonin, not only is blue light systemically damaging, but also damages the eye in particular ….

Chen E. Inhibition of cytochrome oxidase and blue-light damage in rat retina. Graefes Arch Clin Exp Ophthalmol, 1993. ----- The activity of cytochrome oxidase (the enzyme that drives cellular respiration), as well as outer nuclear layer thickness and edema were quantitatively evaluated in blue-light exposed rat retina. Immediately after light exposure, cytochrome oxidase activity decreased. At Day 1, the activity in the inner segments remained low, while severe edema was observed in the inner and outer segments. The outer nuclear layer thickness decreased 1-3 days after exposure. The blue-light exposure inhibited cytochrome oxidase activity and caused retinal injury. ----- And in another study by the same author it was concluded that blue light exposure is a potential hazard for vision. The blue light inhibition of cytochrome oxidase is followed by a redistribution of chloride and potassium in the inner and outer segments, damage to the mitochondria in the inner segments, edema in the inner and outer segments, and progressive degeneration of photoreceptor cells. These findings support the hypothesis that inhibition of cytochrome oxidase is one of the causes of blue-light retinal damage.

Sunlight is absolutely critical during pregnancy. Exposure to natural light in the eyes particularly, not filtered through contact lenses, is one more piece of an anti-miscarriage strategy.

- Minnerman, Kenneth, P., Wurtman, Richard, JK., "The Pharmacology of the Pineal Gland." Laboratory of Neuroendocrine Regulation, Department of Nutrition and Food Science, Massachusetts Institute of Technology, Cambridge, Massachusetts 02139.

- Nair NPV, Hariharasubramaniam N, Pilapil C, et al. "Plasma melatonin." Bio Psychiatry 1986; 21:141-50.

- Vacas, M., Sarmiento, M., Cardinale, D. Brain Research, 225, 1981.

- Pierpaoli, W., Maestroni, G. Immunology Letters 16:35-362, 1987.

Natural light is as important as any nutrient to maintain bone mass ….

Osteoporosis is probably 10% about calcium, 10% about magnesium and other minerals, 30% about sunlight and vitamin D, and 50% about exercise.

The production of vitamin D in response to sunlight begins virtually immediately. Skin has a limited capacity to utilize UV light for vitamin D production. After an interval of time that varies with the skin pigment of the individual, the vitamin D-producing mechanism shuts down. In a fair-skinned redhead, this shutdown occurs within 20 minutes. In darker-skinned people, sun exposure will continue the vitamin D production for an hour or even 2 hours.

----- So --- for example, if a patient has a vitamin D producing time threshold of 45 minutes, and exposes his face and hands to the sun for 45 minutes, he is done making vitamin D for the day unless he exposes different areas of the skin. (Quantitative Note: It is typical for a person to produce more than 10,000 – 25,000 Units of vitamin D in a day at the beach.)

Side Note: Essential to rebuilding teeth is exposure to natural sunlight. This is not solely for vitamin D, but for stimulation of the retino-hypothalamic reflex system, which in turn sets off a chain reaction in the hormonal system that maintains bone and tooth density.

There are 5 causes of osteoporosis:

1. inadequate sunlight
2. insufficient exercise
3. mineral depletion caused by drugs [Drugs taken for GERD (Proton Pump Inhibitors, Histamine 2 Blockers)], caffeine, diuretics
4. high cortisol stress hormone; low testosterone; low progesterone; either high or low thyroid
5. Metabolic Imbalances (per NUTRI-SPEC Testing)

Keep in mind that osteoporosis begins at age 23, and that a woman has already lost 2/3 of the bone density she will lose in her lifetime before she reaches menopause. So, for example, if a woman who has had a lifetime of inadequate sun and insufficient exercise reaches menopause at age 53, in the 30 years since she was 23 she has already lost 2/3 of the bone mass she will lose before death at age 86. --- Point of Emphasis = Osteoporosis is not a disease of menopause.

Nutri-Spec supplements will not reverse that pathology of 30 years duration. Such a person will need (as an adjunct to Nutri-Spec) therapeutic doses of vitamin D. The dose required can only be determined by a blood test for both 25-OH-D and 1,25-2OH-D, but may be as high as 5,000 IU daily for awhile.

These women must become physically active, and must get daily exposure to natural sunlight. The natural sunlight isn't just for the vitamin D, but for full natural spectrum light absorbed through the eyes to activate the retino-hypothalamic reflex. The retino-hypothalamic system has a major influence on hormone balance.

PubMed Results

Item 1 of 1 (Display the citation in PubMed)

1. Sleep Med. 2007 Sep;8(6):557-65. Epub 2007 Mar 23.

Light exposure in the natural environment: relevance to mood and sleep disorders.

Dumont M, Beaulieu C.

Source

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Abstract

In addition to being necessary for vision, light also plays a primary role in circadian physiology. Humans are diurnal animals and their biological clock synchronizes their physiological functions in such a way that functions associated with activity happen in the daytime while functions associated with rest occur at night. A misalignment between the endogenous circadian clock and the desired sleep schedule is the main cause of circadian sleep disorders; it may be involved in certain mood disorders as well. Since light is the main environmental cue used by the biological clock to set its own timing in relation to the day-night cycle, inappropriate light exposure can be involved in the physiopathology of circadian disorders. Conversely, when handled properly, controlled light exposure can be used to treat some mood and sleep disorders. While the earliest studies in the field focused solely on exposure to bright light, contemporary studies aim at understanding how the entire profile of light-dark exposure can influence the circadian clock and, consequently, mood, sleep, and vigilance quality. Following a brief summary of the main concepts underlying the non-visual effects of light, this paper presents some studies using ambulatory measurements of light exposure to illustrate how these concepts apply in real-life situations and discusses the clinical relevance of light exposure in the natural environment for mood, sleep, and circadian disorders.

PubMed Results

Item 1 of 1 (Display the citation in PubMed)

1. J Psychosom Res. 1994 May;38(4):323-37.

Sleep in fall/winter seasonal affective disorder: effects of light and changing seasons.

Anderson JL, Rosen LN, Mendelson WB, Jacobsen FM, Skwerer RG, Joseph-Vanderpool JR, Duncan CC, Wehr TA, Rosenthal NE.

Source

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Abstract

Disturbances of sleep are a hallmark of seasonal affective disorders (SAD), as they are of other mood disorders. Fall/winter SAD patients most often report hypersomnia. Among responses of 293 SAD patients on a symptom questionnaire, complaints of winter hypersomnia (80%) greatly exceeded insomnia (10%), hypersomnia plus insomnia (5%), or no sleep difficulty (5%). Increased sleep length in fall/winter is not unique to SAD. Among 1571 individuals across four latitudes surveyed at random from the general population, winter sleep increases of < or = 2 hr/day relative to summer were reported by nearly half. However, hypersomnia had a low correlation (r = 0.29) with the total number of other SAD symptoms that were reported in this sample. Ten SAD patients kept daily sleep logs across 1 yr that showed increases in fall and winter (sleeping most in October; least in May) whose maximum averaged 2.7 hr per day more weekend sleep than in spring and summer. These winter increases might have been somewhat attenuated since most received light therapy during part of the winter. Nocturnal EEG recordings of depressed SAD patients in winter showed decreased sleep efficiency, decreased delta sleep percentage, and increased REM density (but normal REM latency) in comparison with recordings: (1) from themselves in summer; (2) from themselves after > or = 9 days of light therapy; or (3) from age- and gender-matched healthy controls. Thus, the extent of fall/winter oversleeping recorded by our SAD patients did not differ dramatically from that reported by the general population, but sleep complaints of our SAD patients have been accompanied by features of sleep architecture that are different from healthy controls and are reversed by summer or by bright-light therapy.

PubMed Results

Item 1 of 1 (Display the citation in PubMed)

1. J Clin Psychiatry. 2006 Oct;67(10):1527-35.

An open trial of light therapy in adult attention-deficit/hyperactivity disorder.

Rybak YE, McNeely HE, Mackenzie BE, Jain UR, Levitan RD.

Source

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Abstract

OBJECTIVE:

In adults with attention-deficit/ hyperactivity disorder (ADHD), a delayed sleep/ activity rhythm and/or seasonal mood symptoms may contribute significantly to core pathology and disability. This study examined whether a chronobiologically based treatment, i.e., morning bright light therapy (LT), might have utility as an adjunctive treatment for adult ADHD in the fall/ winter period.

METHOD:

Twenty-nine adults with DSM-IV ADHD were administered a standard 3-week open trial of LT during the fall or winter months. Primary outcome measures included percentage reduction on the Brown Adult ADD Scale and the Conners' Adult ADHD Scale. Secondary measures were decrease in depression scores according to the Structured Interview Guide for the Hamilton Depression Rating Scale, Seasonal Affective Disorder version; improvements on various neuropsychological tests; and shift toward an earlier circadian preference as measured by the Horne-Ostberg Morningness-Eveningness questionnaire. Regression analyses determined which variables at baseline best predicted improvement on a given outcome measure and which variables changed in parallel with one another. The study was conducted from November 2003 through February 2004.

RESULTS:

Morning bright light therapy was associated with a significant decrease in both subjective and objective measures of core ADHD pathology, improved mood symptoms, and a significant phase advance in circadian preference. Multiple regression showed that the shift toward an earlier circadian preference with LT was the strongest predictor of improvement on both subjective and objective ADHD measures. Neither baseline global seasonality scores nor

baseline depression scores strongly predicted LT effects on most measures of ADHD.

CONCLUSION:

These findings suggest that during the fall/winter period, LT may be a useful adjunct in many adults with ADHD. Strikingly, the strongest correlate of improvement in core ADHD pathology was a phase advance in circadian preference rather than alleviation of comorbid seasonal affective disorder, suggesting important clinical benefits of LT beyond the treatment of seasonal affective disorder.

PubMed Results

Item 1 of 1 (Display the citation in PubMed)

1. Ann Acad Med Singapore. 2008 Aug;37(8):669-76.

Treatment of circadian rhythm sleep disorders with light.

Gooley JJ.

Source

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Abstract

The human circadian system is normally synchronised with the solar day, insuring that alertness and performance peak during daytime hours and consolidated sleep occurs during the night. In circadian rhythm sleep disorders, the pattern of sleep-wake is misaligned with the patient's circadian system or the external environment, resulting in insomnia, fatigue, and deterioration in performance. Appropriately-timed exposure to bright light can reset the timing of sleep and wake to the desired times, and improve sleep quality and daytime alertness. The efficacy of bright light therapy, however, is dependent on the time-of-day of the circadian cycle that the light is administered. In this article, we examine the physiological basis for bright light therapy, and we discuss the application of light in the treatment of circadian rhythm sleep disorders including advanced and delayed sleep-phase disorder, free-running disorder (nonentrained type), shiftwork disorder and jet lag disorder. We review the laboratory and field studies which have established bright light therapy as an effective treatment for sleep-wake and circadian misalignment, and we also provide guidelines for the appropriate timing and safe use of bright light therapy.

PubMed Results

Item 1 of 1 (Display the citation in PubMed)

1. J Appl Physiol. 2011 Mar;110(3):619-26. Epub 2010 Dec 16.

Blue light from light-emitting diodes elicits a dose-dependent suppression of melatonin in humans.

West KE, Jablonski MR, Warfield B, Cecil KS, James M, Ayers MA, Maida J, Bowen C, Sliney DH, Rollag MD, Hanifin JP, Brainard GC.

Source

Dept. of Neurology, Thomas Jefferson Univ., Philadelphia, Pennsylvania 19107, USA.

Abstract

Light suppresses melatonin in humans, with the strongest response occurring in the short-wavelength portion of the spectrum between 446 and 477 nm that appears blue. Blue monochromatic light has also been shown to be more effective than longer-wavelength light for enhancing alertness. Disturbed circadian rhythms and sleep loss have been described as risk factors for astronauts and NASA ground control workers, as well as civilians. Such disturbances can result in impaired alertness and diminished performance. Prior to exposing subjects to short-wavelength light from light-emitting diodes (LEDs) (peak λ = 469 nm; 1/2 peak bandwidth = 26 nm), the ocular safety exposure to the blue LED light was confirmed by an independent hazard analysis using the American Conference of Governmental Industrial Hygienists exposure limits. Subsequently, a fluence-response curve was developed for plasma melatonin suppression in healthy subjects (n = 8; mean age of 23.9 ± 0.5 years) exposed to a range of irradiances of blue LED light. Subjects with freely reactive pupils were exposed to light between 2:00 and 3:30 AM. Blood samples were collected before and after light exposures and quantified for melatonin. The results demonstrate that increasing irradiances of narrowband blue-appearing light can elicit increasing plasma melatonin suppression in healthy subjects ($P < 0.0001$). The data were fit to a sigmoidal fluence-response curve ($R(2) = 0.99$; $ED(50) = 14.19$ μW/cm(2)). A comparison of mean melatonin suppression with 40 μW/cm(2) from 4,000 K broadband white fluorescent light, currently used in most general lighting fixtures, suggests that narrow bandwidth blue LED light may be stronger than 4,000 K white fluorescent light for suppressing melatonin.

PubMed Results

Item 1 of 1 (Display the citation in PubMed)

1. Chronobiol Int. 2010 Oct;27(9-10):1762-77.

Predicting human nocturnal nonvisual responses to monochromatic and polychromatic light with a melanopsin photosensitivity function.

Revell VL, Barrett DC, Schlangen LJ, Skene DJ.

Source

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Abstract

The short-wavelength (blue) light sensitivity of human circadian, neurobehavioral, neuroendocrine, and neurophysiological responses is attributed to melanopsin. Whether melanopsin is the sole factor in determining the efficacy of a polychromatic light source in driving nonvisual responses, however, remains to be established. Monochromatic (λ(max) 437, 479, and 532 nm administered singly and in combination with 479 nm light) and polychromatic (color temperature: 4000 K and 17000 K) light stimuli were photon matched for their predicted ability to stimulate melanopsin, and their capacity to affect nocturnal melatonin levels, auditory reaction time, and subjective alertness and mood was assessed. Young, healthy male participants aged 18-35 yrs (23.6 ± 3.6 yrs [mean ± SD]; n=12) participated in 12 overnight sessions that included an individually timed 30-min nocturnal light stimulus on the rising limb of the melatonin profile. At regular intervals before, during, and after the light stimulus, subjective mood and alertness were verbally assessed, blood samples were taken for analysis of plasma melatonin levels, and an auditory reaction time task (psychomotor vigilance task; PVT) was performed. Proc GLM (general linear model) repeated-measures ANOVA (analysis of variance) revealed significantly lower melatonin suppression with the polychromatic light conditions (4000 and 17000 K) compared to the "melanopsin photon-matched" monochromatic light conditions ($p < .05$). In contrast, subjective alertness was significantly lower under the 479 nm monochromatic light condition compared to the 437 and 532 nm monochromatic and both polychromatic light conditions. The alerting responses more reflected the total photon content of the light stimulus. The demonstration that the melatonin suppression response to polychromatic light was significantly lower than predicted by the melanopsin photosensitivity function suggests this function is not the sole consideration when trying to predict the efficacy of broadband lighting. The different spectral sensitivity of subjective alertness and melatonin suppression responses may imply a differential involvement of the cone photopigments. An analysis of the photon densities in specific wavelength bands for the polychromatic lights used in this and

the authors' previous study suggests the spectral composition of a polychromatic light source, and particularly the very short-wavelength content, may be critical in determining response magnitude for the neuroendocrine and neurobehavioral effects of nocturnal light.

PubMed Results

Item 1 of 1 (Display the citation in PubMed)

1. J Biol Rhythms. 2012 Feb;27(1):70-8.

Human nonvisual responses to simultaneous presentation of blue and red monochromatic light.

Papamichael C, Skene DJ, Revell VL.

Source

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Abstract

Blue light sensitivity of melatonin suppression and subjective mood and alertness responses in humans is recognized as being melanopsin based. Observations that long-wavelength (red) light can potentiate responses to subsequent short-wavelength (blue) light have been attributed to the bistable nature of melanopsin whereby it forms stable associations with both 11-cis and all-trans isoforms of retinaldehyde and uses light to transition between these states. The current study examined the effect of concurrent administration of blue and red monochromatic light, as would occur in real-world white light, on acute melatonin suppression and subjective mood and alertness responses in humans. Young healthy men (18-35 years; n = 21) were studied in highly controlled laboratory sessions that included an individually timed 30-min light stimulus of blue (λ(max) 479 nm) or red (λ(max) 627 nm) monochromatic light at varying intensities (10(13)-10(14) photons/cm(2)/sec) presented, either alone or in combination, in a within-subject randomized design. Plasma melatonin levels and subjective mood and alertness were assessed at regular intervals relative to the light stimulus. Subjective alertness levels were elevated after light onset irrespective of light wavelength or irradiance. For melatonin suppression, a significant irradiance response was observed with blue light. Co-administration of red light, at any of the irradiances tested, did not significantly alter the response to blue light alone. Under the current experimental conditions, the primary determinant of the melatonin suppression response was the irradiance of blue 479 nm light, and this was unaffected by simultaneous red light administration.