EFFECTS OF LIGHT ON BRAIN AND BEHAVIOR*

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INTRODUCTION

It is obvious that light entering the eye permits the sensory capacity of vision. The human species is highly dependent on visual perception of the environment and consequently, the scientific study of vision and visual mechanisms is a centuries old endeavor. Relatively new discoveries are now leading to an expanded understanding of the role of light entering the eye - in addition to supporting vision, light has various nonvisual biological effects. Over the past thirty years, animal studies have shown that environmental light is the primary stimulus for regulating circadian rhythms, seasonal cycles, and neuroendocrine responses (Aschoff, 1981a; Binkley, 1990; Reiter, 1991). As with all photobiological phenomena, the wavelength, intensity, timing and duration of a light stimulus is important in determining its regulatory influence on the circadian and neuroendocrine systems (Aschoff, 1981b; Cardinali et al., 1972; Takahashi et al., 1984; Brainard et al., 1983; Brainard et al., 1986). Initially, the effects of light on rhythms and hormones were observed only in sub-human species. Research over the past decade, however, has confirmed that light entering the eyes of humans is a potent stimulus for controlling physiological rhythms (Lewy et al., 1980; Moore-Ede et al., 1982; Wurtman et al., 1985; Czeisler et al., 1986). The aim of this paper is to examine three specific nonvisual responses in humans which are mediated by light entering the eye: light-induced melatonin suppression, light therapy for winter depression, and enhancement of nighttime performance. This will serve as a brief introduction to the growing database which demonstrates how light stimuli can influence physiology, mood and behavior in humans. Such information greatly expands our understanding of the human eye and will ultimately change our use of light in the human environment.

STIMULATION OF THE CIRCADIAN AND NEUROENDOCRINE SYSTEMS BY LIGHT

In most vertebrate species, it is known that light enters the eyes and stimulates the retina. Nerve signals are sent from the retina to the visual centers of the brain and permit the sensory capacity of vision. In addition, neural signals are sent from the retina into the hypothalamus, a non-visual part of the brain. The hypothalamus is a complex neural region that influences or controls many basic functions of the body including hormonal secretion, core temperature, metabolism and reproduction as well as higher cognitive functions such as memory and emotions (Morgane and Panskep, 1979). Information about environmental light is sent from the retina to a specific part of the hypothalamus, the suprachiasmatic nucleus (SCN) (Pickard and Silverman, 1981; Moore,

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1983). This part of the brain is considered to be a fundamental part of the "biological clock", or circadian system, which regulates the body's physiological rhythms. The circadian system is thought to be responsible for controlling daily rhythms such as sleep and wakefulness, body temperature, hormonal secretion and other physiological parameters including cognitive function. It is now clear that light is the primary stimulus for regulating the circadian system, although other external stimuli such as sound, temperature and social cues may also influence the body's timing functions (Aschoff, 1981a; Binkley, 1990).

The SCN relays retinal information to many of the major control centers in the nervous system (Moore, 1983). One nerve pathway that carries non-visual information about light extends from the SCN to the pineal gland via a multisynaptic pathway with connections being made sequentially in the paraventricular hypothalamus, the upper thoracic intermediolateral cell column, and the superior cervical ganglion (Moore, 1983; Klein et al., 1983). Cycles of light and darkness relayed by the retina entrain SCN neural activity which, in turn, entrains the rhythmic production and secretion of melatonin from the pineal. In humans and all other vertebrate species studied to date, high levels of melatonin are secreted during the night and low levels are released during the day (Binkley, 1990; Reiter, 1991; Lewy et al., 1980; Vaughan et al., 1976).

THE EFFECTS OF LIGHT INTENSITY AND WAVELENGTH ON MELATONIN SUPPRESSION

In addition to entraining melatonin secretion from the pineal gland, light can have an acute suppressive effect on melatonin . Specifically, exposure of the eyes to light during the night can cause a rapid decrease in the high nocturnal synthesis and secretion of melatonin (Brainard et al., 1983; Klein and Weller, 1972; Rollag and Niswender, 1976). Early studies on humans did not demonstrate the acute suppressive influence of light on plasma melatonin (Vaughan et al., 1976; Jimerson et al., 1977; Wetterberg, 1978; Vaughan et al., 1979). However, Lewy and colleagues (1980) demonstrated that exposing the eyes of normal volunteers to 2500 lux of white light during the night induced an 80% decrease in circulating melatonin within one hour. In contrast, volunteers exposed to 500 lux of white light exhibited no significant melatonin suppression (Lewy et al., 1980). Earlier attempts at suppressing melatonin in humans with light failed when investigators used typical indoor light levels of 100 to 800 lux (Vaughan et al., 1976; Jimerson et al., 1977; Wetterberg, 1978; Vaughan et al., 1979). Whereas such typical room light would be sufficient for suppressing melatonin in many animal species (Binkley, 1990; Reiter, 1991; Brainard et al., 1983; Klein and Weller, 1972; Rollag and Niswender, 1976), and would be adequate for human vision, it was not enough to suppress melatonin in those experiments. Simply put, it takes much more light to suppress melatonin than is required for vision. The discovery that much brighter light is needed to suppress melatonin in humans provided the groundwork for numerous studies on the internal responses of humans to bright artificial light. However, the notion that only "bright" light can drive neuroendocrine and circadian responses is not entirely accurate.

To begin with, the term "bright" refers to a subjective visual sensation and is thus a relative descriptor (Kaufman, 1984). A 2500 lux light indoors indeed appears "bright" relative to typical indoor levels ranging from 100 to 800 lux. In contrast, 2500 lux of light outdoors is relatively dim compared to daylight at high noon which reaches 100,000 lux (Thorington, 1985). Several years after it was discovered that light at 2500 lux can suppress melatonin in humans, a study

was done to more precisely determine the dosages of light needed to suppress melatonin in normal volunteers (Brainard et al., 1988). In that study, six normal males were exposed to carefully controlled intensities of monochromatic green light at 509 nm for one hour during the night. Specifically, the volunteers were continuously exposed to the experimental light between 02:00 and 03:00 hours with their pupils fully dilated by a mydriatic agent, their heads held steady relative to the light source by an ophthalmologic head holder, and with translucent white integrating spheres covering both eyes. This procedure produced a constant and uniform illumination of the whole retina during the entire light exposure. The data from this experiment (Figure 1) demonstrated that light affects a human hormone in a dose-response fashion: i.e., the brighter the photic stimulus the greater the suppression of melatonin (Brainard et al., 1988).

It is interesting that all of the stimuli used in this study activated the visual system: both the volunteers and the experimenters saw all the different light intensities and accurately reported them to be green. The lower light intensities, however, did not change hormone levels whereas the higher intensities induced a 60-80% decrease in this hormone. Thus, light that activates vision does not necessarily cause neuroendocrine change. It appears to be generally true in both animals and humans that much more light is needed for biological effects than for vision. The data shown in Table 1 provide the photometric and radiometric values for the stimuli used in constructing this dose-response function.

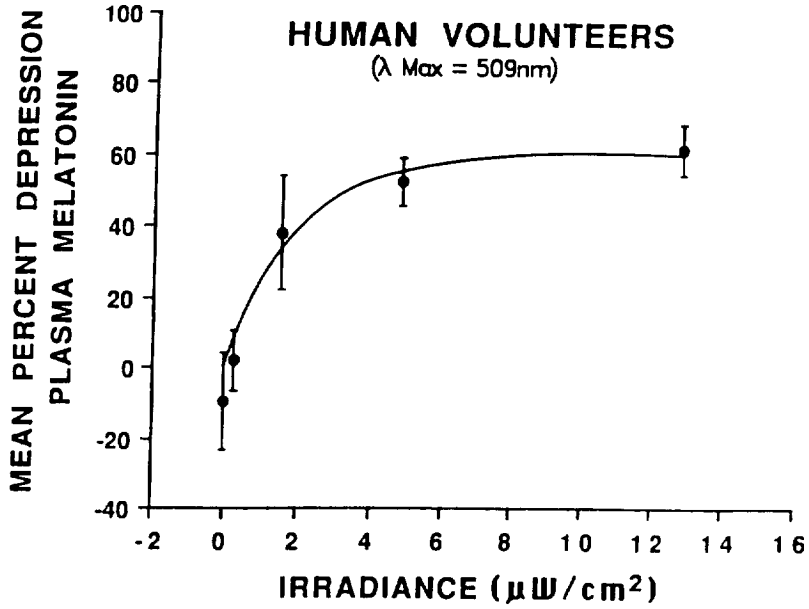

Fig. 1. The dose-response relationship between green monochromatic light (509 nm, 10 nm half-peak bandwidth) exposure of normal volunteers eyes and suppression of the hormone melatonin (Brainard et al., 1988). Data points indicate mean ± SEM.

TABLE 1 Radiometric and Photometric Stimuli Used in the Melatonin Dose-Response Curve (Brainard et al., 1988)

$\mu W/cm^2$	photons/cm^2	photopic lux	scotopic lux	% melatonin suppression
0.01	9.19×10^{13}	0.03	0.17	-9.67
0.3	2.76×10^{15}	1.03	5.25	1.83
1.6	1.47×10^{16}	5.50	27.98	37.33
5.0	4.59×10^{16}	17.18	85.90	51.67
13.0	1.19×10^{17}	44.66	227.37	60.67

The demonstration of the dose-response function for light suppression of melatonin in humans produced an unexpected result: very bright light is not necessarily needed for melatonin suppression. As demonstrated by Table 1, the mean threshold illuminance for suppressing melatonin was between 5 and 17 lux in normal volunteers - a level of illumination equal to civil twilight and well below typical indoor light . This means that under the proper conditions, 25 to 100 times less light can suppress melatonin than originally thought (Lewy et al., 1980). Why did ambient room light at levels much higher than 17 lux not suppress melatonin in earlier experiments? In those early studies (Lewy et al., 1980; Vaughan et al., 1976; Jimerson et al., 1977; Wetterberg, 1978; Vaughan et al., 1979), neither the exposure conditions nor the light stimuli were optimized. Often the experimental light stimulus consisted of turning on the overhead light provided with the experimental room. In almost any given room, it is possible to vary the light illuminance entering the eyes by a factor of 10 simply by changing the direction of gaze. Thus, in a room characterized as having "typical" illumination levels of 500 lux, the occupants may be able to see up to 500 lux if they look directly towards the light fixtures, but if they look at the floor or walls, this light reaching their eyes may be as low as 50 lux. Furthermore, the pupil of the eye adjusts dynamically to further restrict the amount of light which reaches the retina. A maximally restricted pupil can reduce the light reaching the retina to as little as one sixteenth of the light falling on the cornea (Sliney and Wolbarsht, 1980). In addition, the amount of the retina exposed to the light stimulus varies greatly with the geometry of the light source and the relative direction of gaze. A recent study by Gaddy (1992) and colleagues has shown partial retinal exposure is less effective compared to the whole retinal exposure for suppressing melatonin (Gaddy et al., 1992). Finally, the amount of light entering the eye can be further reduced by shadowing of the cornea by the bony orbit, squinting and eye blink. Thus, both behavioral and ocular factors can functionally reduce the amount of light reaching the retina to a level where it is not effective in suppressing melatonin levels. In the early studies, we presume that no efforts were made to control pupil size, direction of gaze, and retinal field exposure since none of these experimental details were reported. Hence, in those experiments "ordinary room levels of illumination" did not suppress melatonin (Lewy et al., 1980; Vaughan et al., 1976; Jimerson et al., 1977; Wetterberg, 1978; Vaughan et al., 1979) and only when much brighter light was used (Lewy et al., 1980) could hormone production be altered. However, it is clear that very low levels of light can indeed suppress melatonin when the exposure factors are optimized (Brainard et al., 1988).

In addition to exposure factors and light intensity being critical in determining if a light stimulus will suppress melatonin, the spectral quality of light is important in determining its relative biological impact. Studies done on the effects of different wavelengths on hamsters, rats and

mice suggest that wavelengths in the blue and green portion of the spectrum have the strongest impact on circadian and neuroendocrine regulation (Cardinali et al., 1972; Takahashi et al. 1984; Brainard et al., 1984; Brainard et al., 1985; Vaughan et al., 1985; Brainard et al., 1986; Bronstein et al., 1987; Podolin et al., 1987; Thiele and Meissl, 1987; Millette et al., 1987; Benshoff et al., 1987; Brainard et al., 1987; Brainard et al., 1991a). Some data have supported the hypothesis that the rod photopigment rhodopsin is the primary receptor for circadian and neuroendocrine regulation (Cardinali et al., 1972; Takahashi et al. 1984; Brainard et al., 1984; Podolin et al., 1987; Thiele and Meissl, 1987; Benshoff et al., 1987; Brainard et al., 1987). In contrast, other data have suggested that one or more cone photopigments may be involved in these regulatory effects (Brainard et al., 1984; Podolin et al., 1987; Thiele and Meissl, 1987; Millette et al., 1987; Benshoff et al., 1987; Brainard et al., 1987). It is important to note that while the highest sensitivity is in the blue-green range, this does not preclude other wavelengths from participating in circadian and neuroendocrine regulation. For example, in terms of melatonin suppression, short wavelengths in the ultraviolet region of the spectrum (Podolin et al., 1987; Benshoff et al., 1987; Brainard et al., 1987; Brainard et al., 1991a) and longer wavelengths in the red portion of the spectrum are quite capable of suppressing melatonin in rodents if the intensity is sufficiently high (Vanecek and Illnerova, 1982; Nguyen et al., 1990; Broker et al., 1990). Further studies are required to conclusively identify what specific photoreceptors and photopigments are involved in regulating the circadian and neuroendocrine systems in animals.

Only one study has specifically examined wavelength regulation of melatonin in humans (Brainard et al., 1988). That study suggested that the peak sensitivity for melatonin suppression is in the blue-green range as seems to be the case in some lower mammals. It is premature, however, to draw any conclusions as to what photoreceptors are involved in any nonvisual physiological regulation in humans.

USE OF LIGHT TO TREAT WINTER DEPRESSION

While research over the past decade has proceeded on the biological effects of light in humans, concurrent studies have tested the use of light as a therapeutic tool for improving mood and psychological status of patients diagnosed with winter depression. It has been noted since antiquity that some individuals are adversely affected by the changing seasons. More recently, the specific condition of fall and winter depression or Seasonal Affective Disorder (SAD), has been formally described in the scientific literature (Lewy et al., 1982; Rosenthal et al., 1984; Rosenthal et al., 1988; Terman et al., 1989a; Terman and Terman, 1992) and been included in the latest edition of the American Psychiatric Association's diagnostic manual (DSM-III-R, American Psychiatric Association, 1987). People affected with this malady often experience a dramatic decrease in their physical energy and stamina during the fall and winter months. As daylengths become shorter and temperatures become cooler, individuals with SAD often find it increasingly difficult to meet the demands of life - they can not function well in their jobs or can not cope with everyday family life. In addition to a general decrease in energy, they experience emotional depression and feelings of hopelessness and despair. Other symptoms of winter depression or SAD may include increased sleepiness and need for sleep, increased appetite (particularly for sweets and other carbohydrates), and a general desire to withdraw from society. People afflicted with this malady often feel compromised in meeting the ordinary demands and responsibilities of everyday life. Fortunately, among those who are accurately diagnosed with

SAD, daily light therapy has been found to effectively reduce symptoms in many patients (Lewy et al., 1982; Rosenthal et al., 1984; Rosenthal et al., 1988; Terman et al., 1989a; Terman and Terman, 1992).

There are now numerous clinics across the United States that offer light therapy for people who are afflicted with winter depression (Rosenthal, 1990; Society for Light Treatment and Biological Rhythms, 1991a). Specific treatment protocols vary somewhat between different clinics. One frequently used procedure involves a patient sitting at a specific distance from a fluorescent light panel which provides a 2500 lux exposure when looking directly at the lamp. The patient is told not to gaze steadily at the bright light, but rather to glance directly at the unit for a few seconds each minute over a two hour period. During the therapy period, a patient may read, watch television, work at a computer or do other hand work. Patients often respond to this therapy after two to seven days of light treatment and continue to benefit as long as the treatment is repeated daily throughout the months that the individual experiences winter depression (Rosenthal et al., 1984; Rosenthal et al., 1988; Terman et al., 1989a; Terman and Terman, 1992).

The white light used for treating SAD can be effectively provided by a range of lamp types including incandescent, cool-white fluorescent, and "sunlight simulating" fluorescent, (Lewy et al., 1982; Rosenthal et al., 1984; Rosenthal et al., 1988; Terman et al., 1989a; Terman and Terman, 1992; Yerevanian et al., 1986; Lewy et al., 1987; Terman et al., 1990; Stewart et al., 1990; Moul et al., 1993; Joffe et al., 1993; Terman et al, 1989b; Avery et al., 1993). Furthermore, there is an assortment of light devices available for treating SAD. Light therapy instruments come in a variety of shapes and configurations including workstations (Terman et al., 1990), head-mounted light visors (Stewart et al., 1990; Moul et al., 1993; Joffe et al., 1993) and automatic dawn simulators (Terman et al, 1989b; Avery et al., 1993). These devices are configured to shorten therapeutic time, increase patient mobility or to permit therapy during the sleep period. Doubtless there will be continued development, diversification and improvement of light therapy devices and strategies.

THE EFFECTS OF DIFFERENT WAVELENGTHS IN SAD PHOTOTHERAPY

Current evidence supports the hypothesis that light therapy for SAD works by way of light shining into the eyes as opposed to light on the skin (Wehr et al., 1987). It is not known, however, what ocular photoreceptors or photopigments mediate the therapeutic benefits of light in winter depression. To date, three consecutive studies have specifically compared different portions of the spectrum for clinical efficacy in treating SAD (Brainard et al., 1990; Oren et al., 1991; Stewart et al., 1991). In the first study, 18 patients were treated with an equal photon dose of white, blue or red light for a period of one week. The photon dose of 2.3×10^{15} photons/cm^2/sec was selected because this particular photon density of broad spectrum white light (400-760 nm half-peak bandwidth, Vitalite® lamps, Durotest Corp.) had been shown in many previous studies to be clinically effective in one week of therapy (Rosenthal et al., 1988; Terman et al., 1989a). The red and blue light sources used in this study (F40R and F40BB lamps, Westinghouse Div., Philips Inc.) had half-peak bandwidths of approximately 615-685 nm and 430-465 nm, respectively. Patients' clinical status before and after light therapy was followed by means of the 21-item Hamilton Depression Rating Scale (HDRS), a standard scale for measuring symptoms associated with depression (Hamilton, 1967). The results of this study are illustrated in Figure 2.

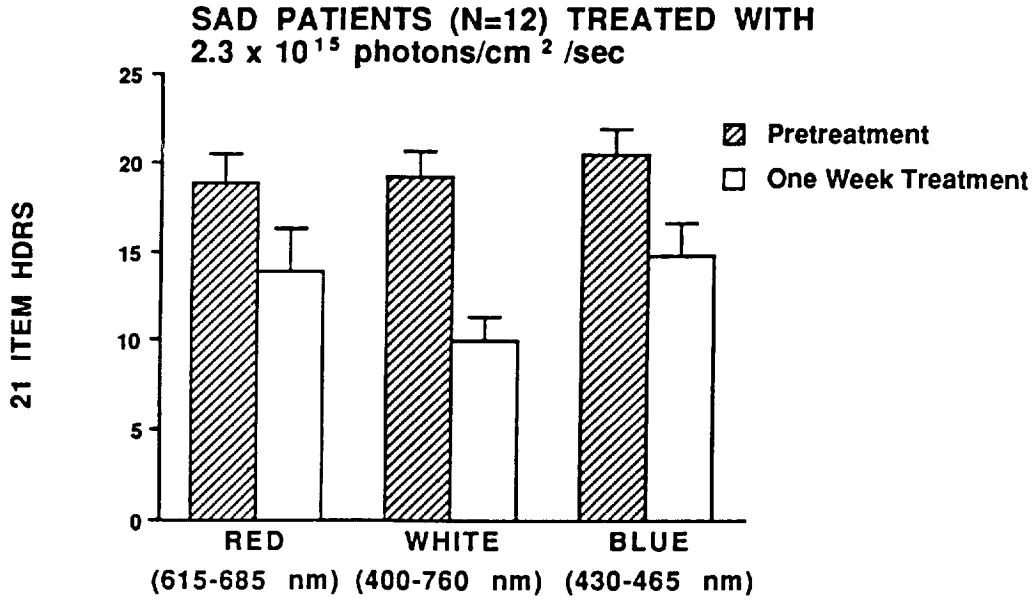

Fig. 2. The bars in this graph indicate mean + SEM Hamilton Depression Rating Scale values for patients before treatment (hatched bars) and after one week of treatment with equal photon densities of different light spectra (open bars). Numbers in parentheses indicate the half-peak bandwidth of the light source (Brainard et al., 1990).

This study was the first step towards defining an action spectrum of light therapy for winter depression. As shown in Figure 2, one week of light therapy with each of the three light sources produced an improvement in depression symptoms among the groups of patients tested. Specifically, the percent drop in mean HDRS scores were 26%, 47% and 27% for the red, white and blue light sources, respectively. Thus, the photon density emitted from the white light source elicited a significantly stronger clinical response compared to the results obtained from an equal photon density from the blue and red light sources (Brainard et al., 1990). This suggests that broad spectrum white light at this particular photon density is superior to restricted bandwidths of light in the red and blue portions of the visible spectrum. That result implies that light sources for SAD light therapy could not be improved by narrowing the wavelengths provided and shifting them towards either end of the visible spectrum. It is logical, however, to question the relative efficacy of a green bandwidth of light for treating winter depression.

To resolve that question, a second study was done comparing green light to red light at 2.3×10^{15} photons/cm^2/sec for treating SAD (Oren et al., 1991). The green and red light (F40G and F40R lamps, Westinghouse Div., Philips Inc.) had half-peak bandwidths of approximately 505-555 nm and 615-685 nm, respectively. Patients' clinical status before and after one week of light therapy was followed by means of the 21-item HDRS. The results of this study are illustrated in Figure 3.

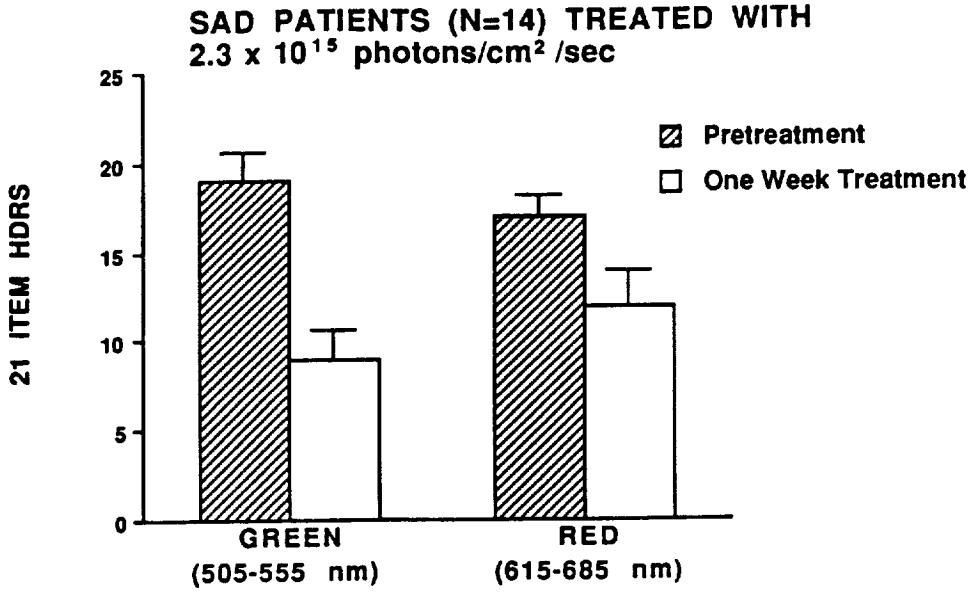

Fig. 3. The bars in this graph indicate mean + SEM HDRS values for patients before treatment (hatched bars) and after one week of treatment with equal photon densities of green or red light. Numbers in parentheses indicate the half-peak bandwidth of the light source (Oren et al., 1991).

As illustrated in Figure 3, one week of light therapy with both green and red light sources produced an improvement in depression symptoms in the groups of patients tested. The percent reduction in mean HDRS scores was 51% and 30% for the green and red light sources, respectively. Hence, at this photon density, green light was significantly stronger than the red light for treating winter depression (Oren et al., 1991). The results of this study (Figure 3) considered alongside the results from the study comparing red, white and blue light therapy at the same photon density (Figure 2) suggest that broad spectrum white light and narrower band green light are equivalent in their capacity to reduce symptoms of SAD. Between the two studies, white and green light treatments were associated with a 48% and 53% reduction in HDRS scores, respectively. Comparisons of group responses between different studies, however, are not conclusive. Are white and green light really equivalent in their phototherapeutic strength?

To answer that question, 12 patients were given one week of light therapy for SAD with either green or white light at an equal photon density (Stewart et al., 1991). Since therapy with white and green light appeared to cause roughly equivalent HDRS reductions across the first two studies, the experimental photon density was lowered to 1.23×10^{15} photons/cm^2/sec in the third study. As in the first two studies, patients' clinical status before and after one week of light therapy was followed by means of the 21-item HDRS. The results of this study are illustrated in Figure 4.

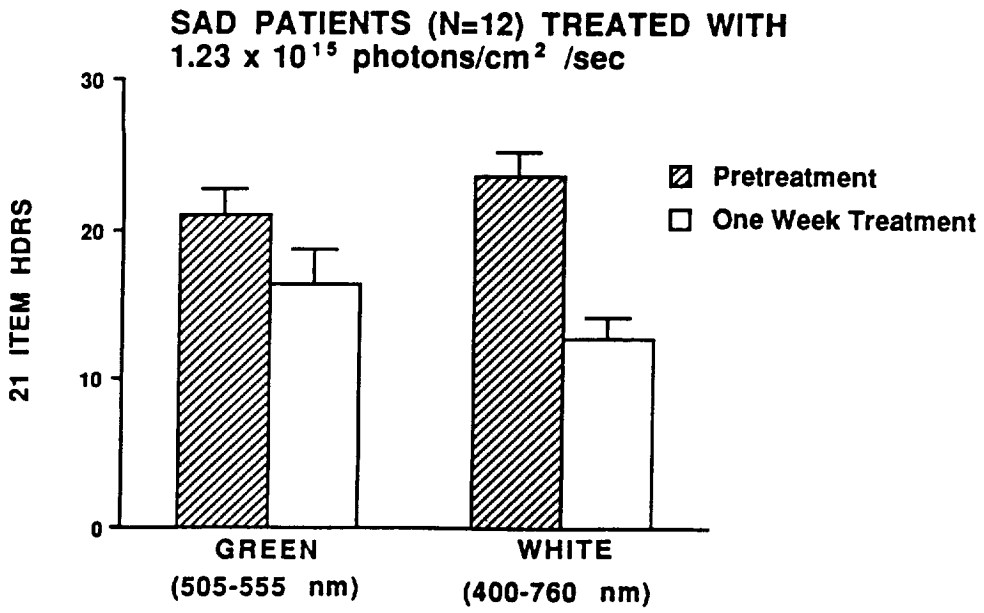

Fig. 4. The bars in this graph indicate HDRS values (mean + SEM) for patients before treatment (hatched bars) and after one week of treatment with equal photon densities of white or green light. Numbers in parentheses indicate the half-peak bandwidth of the light source (Stewart et al., 1991).

As shown in Figure 4, one week of therapy with each of the light sources produced an improvement in depression symptoms. Specifically, the percent drop in mean HDRS scores was 22% for the green light and 46% for the white light sources. At this lower photon density, white light was superior to the green light in treating SAD (Stewart et al., 1991). Hence, in this study, white and green light were not equivalent in their therapeutic efficacy as the preliminary comparison of the data from the first two wavelength studies suggested.

Together, these three studies form the ground work for determining the action spectrum for SAD light therapy (Brainard et al., 1990; Oren et al., 1991; Stewart et al., 1991). The traditional approach to defining a complete action spectrum, however, requires substantially more testing (Coohill, 1991). A thoroughly defined action spectrum can guide the development of light treatment devices that emit the optimum balance of wavelengths for treating SAD. Furthermore, an action spectrum will yield important information about the photosensory mechanism(s) responsible for the beneficial effects of light therapy. Currently, it is premature to predict what photopigment(s) or photoreceptor(s) mediate the antidepressant effects of light.

A practical issue debated among SAD researchers concerns the role of ultraviolet radiation (UV) in light therapy. Most of the early studies on SAD therapy successfully utilized fluorescent lamps that emitted white light containing a portion of UV wavelengths (Rosenthal et al., 1988; Terman et al., 1989a). Those early results erroneously led to the suggestion that UV wavelengths are necessary for successful therapy. The literature, however, shows clearly that SAD symptoms can be reduced by lamps which emit little or no UV (Yerevanian et al., 1986; Lewy et al., 1987; Stewart et al., 1990; Moul et al., 1993; Joffe et al., 1993; Brainard et al., 1990;

Oren et al., 1991; Stewart et al., 1991; Lam, 1991). Hence, UV wavelengths do not appear to be necessary for eliciting positive therapeutic results. Does this rule out UV having any role in relieving winter depression? Studies demonstrate that UV wavelengths can regulate seasonal reproduction, melatonin production, and circadian rhythms in some animal species (Brainard et al., 1985; Vaughan et al., 1985; Podolin et al., 1987; Benshoff et al., 1987; Brainard et al., 1987; Brainard et al., 1991a). Furthermore, in normal, healthy humans up to the age of at least 25 years, UV-A can be detected by the visual system (Tan, 1971; Brainard et al., 1992; Sanford et al., 1992). Although the latest studies show no decrement in therapeutic response when UV is specifically excluded in SAD treatment, they do not demonstrate that UV is totally noncontributory. Whether or not UV wavelengths can contribute to the optimum balance of wavelengths for SAD therapy remains an open question.

The data presented here make it clear that several methodological problems will have to be overcome before further progress can be made in defining an action spectrum for SAD light therapy. One complication for the wavelength studies and nearly all studies on SAD involves the fact that they are done on an outpatient basis. Hence, patient compliance on treatment timing, frequency and duration cannot be closely controlled even with the most cooperative subjects. Furthermore, very small changes in gaze direction and patient position relative to the light source can cause great variability in the amount of light transmitted to the patients' eyes (Gaddy, 1990; Dawson and Campbell, 1990). Did patients have different gaze behaviors or different patterns of light usage with the different wavelength light sources? The optimum method of comparing different wavelengths - or any other photic parameter - for SAD therapy is to work with more carefully controlled exposures. As demonstrated in the melatonin suppression studies, tight control of ocular light exposure permits substantially lower light levels to regulating hormone secretion. Could the general requirement of 2500 lux or more for SAD therapy be a compensation for differences in patient compliance and exposure variables?

Across the three wavelength studies outlined above, each light treatment produced some therapeutic improvements. Does this indicate that each light was at least partially effective in treating SAD symptoms, or are some of the therapeutic benefits of light therapy due to a non-specific or placebo response? Since patient expectations of treatment outcome are thought to contribute significantly to the placebo effect, evaluation of expectations before treatment is one strategy for approaching this question. Prior to any light treatment, subject expectations were systematically probed in each of the three wavelength studies. In general, all subjects had positive expectations about the success of light therapy but there were no differences between the expectations for the different light spectra in these studies (Brainard et al., 1990; Oren et al., 1991; Stewart et al., 1991). This evidence supports the idea that some of the therapeutic benefit of the different light spectra may have been due to a placebo response but that the differential therapeutic responses to the different light spectra were not merely an extension of the patients' preconceived beliefs.

In the medical literature it has been well documented that patients with a wide range of disorders - depression, schizophrenia and anxiety as well as cancer, diabetes and ulcers - can successfully respond to inactive or placebo treatments (Ross and Olson, 1981; Eastman, 1990a; Eastman et al., 1993). Hence it would be remarkable if SAD patients did not show some level of placebo response to light therapy. In fact, therapeutic improvements are almost always observed with

light treatments regardless of light intensity, wavelength and duration (Rosenthal et al., 1988; Terman et al., 1989a; Terman and Terman, 1992). Although it is obvious that light therapy indeed will reduce patients' depression symptoms, the critical question is how much of the patients' response to light therapy is due to a non-specific placebo response versus a genuine clinical response? This remains an open question in the SAD field and has been discussed most insightfully by Eastman (1990a). Unfortunately, until this question is resolved, a more conclusive action spectrum for SAD phototherapy may not be possible. The inability to accurately separate placebo responses from genuine clinical antidepressant responses causes an element of "noise" in phototherapy data which seriously hinders the accurate discrimination of differential wavelength effects in light therapy.

USE OF LIGHT FOR ENHANCING PERFORMANCE AND TREATING PROBLEMS OF NIGHT WORKERS

Over the past decade, most of the studies on light therapy have been concerned with winter depression. Other research, however, has begun to extend the applications of light therapy. Investigators have had some success in treating certain sleep disorders with phototherapy (Rosenthal et al., 1990; Dawson et al., 1989). In addition, studies have indicated that individuals with either non-seasonal depression (Yerevanian et al., 1986; Kripke et al., 1989) or pre-menstrual syndrome (PMS) may benefit from light therapy (Parry et al., 1987; Parry et al., 1989). Much more work needs to be done in determining the utility of light for treating these disorders. It appears that we are entering a frontier of medicine in which man's biological response to light is being harnessed to alleviate specific illnesses. Such medical developments have encouraged investigators to explore the possibilities of using light for various domestic or non-medical applications.

One area of study involves the function and dysfunction of the human circadian physiology under more challenging situations. Some preliminary studies have tested the use of strategic light exposure to prevent or ameliorate jet lag (Daan and Lewy, 1984; Wever, 1985; Cole and Kripke, 1989). The preliminary findings are generally positive and some investigators are optimistic that light will be a useful tool for quickly resetting the traveler's internal biological clock and overcoming some of the problems associated with jet travel over multiple time zones. There is a consensus among scientists however, that the data in this field - as of August, 1991 - are preliminary and insufficient for a specific prescription on how to best use light for this modern malady (Society for Light Treatment and Biological Rhythms, 1991b).

Shift work may pose problems associated with circadian desynchronization analogous to that found in jet lag (Moore-Ede et al., 1982; U. S. Congress, 1991). Instead of rapidly flying to distant countries, the shift worker stays in one place but may just as suddenly change the time period that he is awake or asleep. By the broadest definition, shift workers are individuals who do not work a standard daytime schedule. Instead, they work nights, evenings, rotating shifts, split shifts or extended shifts. It is estimated that one out of five full time workers in the United States (20 million people) is a shift worker (U. S. Congress, 1991).

As Campbell and Dawson (1992) have reported, the two most common and destructive problems associated with shiftwork are reduced quality of sleep following night work and reduced

capacity to maintain alertness while at work. Thus, shift work has drawbacks in increased accidents, decreased production and performance deficits among those who are working at night when the body has a natural tendency to be asleep. Furthermore, evidence indicates that shift workers have increased health problems including higher risk to cardiovascular disease, gastrointestinal distress, as well as cognitive and emotional problems (Moore-Ede et al., 1982; U. S. Congress, 1991; Campbell and Dawson, 1992; Folkard and Monk, 1985; Eastman, 1990b; Moore-Ede et al., 1983; Akerstedt et al., 1984). Despite these deleterious effects on worker health and efficiency, the number of people involved in shift work is likely to increase. Researchers believe that poor chronobiological adjustment to a permanent or rotating schedule causes some of these ailments (U. S. Congress, 1991). Not all of these problems, however, are solely due to a maladapted biological clock. In addition to a desynchronized circadian system, shift workers generally tend to be chronically sleep deprived and experience domestic stresses that are more or less independent of circadian adaptation (Moore-Ede et al., 1982; U. S. Congress, 1991; Folkard and Monk, 1985) Hence, there is no single solution to all of the problems associated with shift work.

On the frontiers of shift work research, some investigators are attempting to develop strategies of light stimulation to improve circadian entrainment and to enhance performance and alertness in night workers. In one study, Czeisler and colleagues simulated a night shift routine in the laboratory and tested both biological adaptation and behavioral performance under different lighting stimuli (Czeisler et al., 1990). They found that workers given 7,000 to 12,000 lux of white fluorescent light during their work hours and complete darkness to sleep in during the daylight hours, adapted better biologically and had improved alertness and cognitive performance compared to subjects who worked under 150 lux of light and had no complete darkout for sleeping during the day (Czeisler et al., 1990). Other studies on simulated shift work have shown that exposure to bright white fluorescent light at specific times can improve sleep quality, enhance performance and speed the adjustment of the circadian system (Society for Light Treatment and Biological Rhythms, 1991b; Campbell and Dawson, 1992; Eastman, 1990b). All of these studies were aimed primarily at finding a means of improving adjustment of the circadian system, sleep quality and performance of the shift worker. This experimental approach requires a minimum of 3 to 5 testing days and, under optimum conditions, even longer test periods to adequately discern circadian and sleep changes.

A different experimental approach has been to examine the immediate effects of light stimuli in a single night of work or during prolonged periods of work. The principal focus of this research has been to determine if bright light stimuli can help sustain alertness without degrading performance. In a study by French and colleagues (1990), healthy young volunteers stayed awake and worked continuously at a computer for 30 hours, taking only short breaks to eat or go to the bathroom. While working under 3,000 lux of white fluorescent light during 18:00 to 06:00 hours, the volunteers exhibited significantly improved behavioral and cognitive performance on selected tasks compared to their own performance on a separate occasion under 100 lux. In addition to these behavioral effects, there were significant differences in the body temperatures, plasma cortisol levels and plasma melatonin levels in these volunteers under the bright versus dim light condition (French et al., 1990; Brainard et al., 1991b). A similar study done in a separate laboratory has also shown that young men doing night work from 21:00 to 08:00 hours under 5000 lux of white light performed better on selected behavioral tasks versus when they

worked under light at 50 lux (Hannon et al., 1991). Again, in this study body temperatures and melatonin levels were significantly influenced by light levels. In these acute studies, it is not clear how light is influencing performance. Could the correlated biological changes in body temperature and hormone levels be directly related to improvement in behavioral tests? Is the circadian system involved in these acute effects of light? Are the acute effects of light due to a "masking" of circadian rhythms? Clearly, further studies are needed to clarify the mechanism(s) by which light enhances performance.

There are many occasions when individuals work through the night on an irregular basis, either by free choice, or by unexpected needs emerging in the home or at work. What are the longer term consequences of a single night of bright light exposure for improving alertness and performance? Will the short term gains of enhanced performance or alertness be offset by a longer term disruption of circadian physiology when the individual returns to a regular schedule? This new research raises many unanswered questions. As with jet lag applications, there is a consensus among scientists - as of September, 1991 - that it is still premature to formulate a set prescription on how to best use light for both short term and long term work applications (Society for Light Treatment and Biological Rhythms, 1991b; U. S. Congress, 1991). Much additional work is needed in both laboratory simulations and field tests before the overall consequences of using bright light stimuli can be determined and the optimum lighting strategy can be recommended for the varieties of shift work.

As with research on phototherapy for SAD and other disorders, it should be noted that the studies on using light stimuli to improve problems associated with night work may have complications of placebo responses. Simply put, most volunteers can readily see that a manipulation of light is part of the experiment. In such a circumstance, the investigator runs the distinct risk of finding a placebo reaction to the specific light treatments. There are good experimental strategies which can help address the potential problem of a placebo response and some of them are discussed above. One of the best means to avoid placebo problems in lighting studies is to collect both behavioral and biological data. Whereas behavioral variables and subjective mood states may be quite susceptible to the volunteers' mental preconceptions, objective biological variables such as circadian rhythms, hormone levels, electrophysiological responses, body temperature, urine volume and the like are much less likely to be directly influenced by a placebo response. Collecting physiological and behavioral measures together can greatly improve the reliability of data on nonvisual biological effects of light of light.

CONCLUSION

Experimental research on animals during the past thirty years and on humans in the past decade confirm that light can strongly influence the physiology and behavior of many species. With humans, light is a primary stimulus to the circadian system and can regulate many biochemical and physiological processes in the body. The critical parameters of light intensity and wavelength needed to provide this nonvisual biological stimulation are still under study. In addition to these biological effects of light, a high percentage of patients who suffer from winter depression are responsive to bright light therapy. Other clinical disorders also may be treatable with light stimuli. Further pioneering studies are now examining the use of light to improve performance and ameliorate problems associated with shift work. Taken together, these studies

provide the initial database for a frontier in medicine and biology. Beyond therapeutic applications, however, what are the potential consequences of this research?

Modern man has become very sophisticated in the specific use of light in his living and working environment. Currently, building interiors are illuminated for three main purposes: 1) providing light for visual performance; 2) providing light for visual comfort; and 3) providing light for aesthetic appreciation of the environment and its contents (Kaufman, 1984; Kaufman, 1987). The studies discussed here demonstrate that light can also influence human physiology, mood and behavior. These data may be the seeds for a revolution in architectural lighting. It is appropriate to begin exploring ways to incorporate these laboratory results into practical architectural lighting designs. Such designs will need to optimize architectural light for nonvisual biological stimulation as well as follow the traditional guidelines for providing correct visual stimulation and comfort. In the long range, this new design consideration is likely to dramatically alter illumination strategies for homes, factories, offices, schools, hospitals and most interior living spaces.

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