Summary

This review of a wide-ranging and multidisciplinary topic is oriented towards those who have interests in researching into, designing or specifying lighting for working and living environments. The contents have been assembled around six sections to give coherence to such a diverse literature. After the introductory section 1, section 2 outlines the biological pathways mediating melatonin responses to light and section 3 examines the links between light, body rhythms and melatonin secretion. Section 4 reviews the relationship between melatonin response and spectral irradiance and section 5 provides an overview of the association between mood states, pineal activity and light therapy. In section 6 the interrelationship between the pineal gland with other glandular activities is briefly explored and section 7 offers a summary and conclusions. The whole paper suggests that there are many issues to be resolved before convincing generalisations can be drawn about the relationship between lighting, melatonin and mood states.

The effects of environmental illumination on melatonin, bodily rhythms and mood states: a review

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1 Introduction

The subjects reviewed in this paper have attracted much attention in the popular media, but the finer distinctions, uncertainties and debatable aspects are often ignored. The purpose here is to attempt an impartial evaluation of the effects of lighting on melatonin, bodily rhythms and mood states and to formulate something of a coherent structure from a wide diversity of material. This text has been derived from papers published mostly in biological, psychological, psychiatric and illumination engineering journals, their selection being aimed at providing a context for professional people who are concerned to provide suitable lighting conditions for environments in which people work and live. The reader who requires more information about the fundamental biology and biochemistry underlying these topics is recommended to consult further texts such as those by Arendt(1), Shafii and Shafii(2), O’Brien and Klein(3), Wurtman et al.(4) and Ariëns Kappers and Pévet(5).

This paper has its origin in a report the author(6) prepared for the Building Research Establishment. An abridged version was included in a previous BRE publication by Raw and Hamilton, the present paper offering a more comprehensive and detailed account.

2 Biological pathways mediating the melatonin response to light

The optic nerve is well known as the transmission pathway from eye to brain subserving the process of seeing, but it fulfils a second role by providing information from the eye to the pineal gland about the dark–light state of the external environment.

The anatomical pathway from the retina to the pineal gland in humans is complex and, after leaving the optic nerve, it passes to the suprachiasmatic nucleus in the hypothalamus and thence to the paraventricular nucleus. The route continues down the spinal cord along the intermedio-lateral cell columns, linking with the cervical ganglion and thence via autonomic nervous system fibres into the pineal body. The relative location of the pineal within the brain is shown diagrammatically in Figure 1 and a schematic summary of the pathway from the eye to the pineal gland is presented in Figure 2.

Sympathetic nervous system terminals release noradrenaline within the pineal gland, which initiates the biosynthesis of melatonin during darkness. Serotonin is involved in this process and many other products having peptide and protein type structures have been identified(4,7). Figure 3 provides a general summary of the biosynthetic process.

![Diagram](https://example.com/diagram.png)

**Figure 1** A diagrammatic representation of a longitudinal section through the human brain to show the location of the pineal gland in relation to other structures.
Light, body rhythms and melatonin secretion

All mammals show a rise in melatonin production during the night and much less production during daylight; three basic night patterns have been described by Reiter(12). Lewy and Markey(13) demonstrated a clear 24-hour cycle of melatonin in man, with a very large rise during the night and a decline with the onset of daylight. This rhythm is disturbed if it is interrupted by strong light during the dark phase, as was shown by

Bhatnagar(10) points out that all mammals possess a pineal gland and, in vertebrates, it is first seen evolutionarily in the lamprey. In the human it is situated deep in the brain and is about 8.8 mm in length by 5.2 mm in width. Certain lower vertebrates, fish and lizards possess a homologous structure, sometimes referred to as a third eye, but, on the evolutionary pathway from lower vertebrates to man, a change from a rudimentary photoreceptor type process to a neuroendocrine organ has evolved.

In amphibians, melatonin regulates skin colour; in birds it regulates circadian rhythms; and in mammals it probably regulates the cycle of sexual receptivity and fertility. Its role in man is not entirely clear, but it is thought to influence bodily rhythms, especially circadian rhythms such as sleeping and waking activity, although these have a more complex regulation than is ascribable to one single factor. Arendt et al.(9) suggested that ‘melatonin may not be necessary in normal physiology; however it may be sufficient to manipulate neuroendocrine function’. Oksche(16) proposes that the role for the pineal gland is to serve as a photoreceptor, biological clock and endocrine gland. This would seem to be a perfectly reasonable concept since in many animals, excluding man, there are selective times of the year when ovulation, sexual receptivity, hormonal changes and reproductive activity take place, related to lighting conditions. This implies that melatonin interacts with other neuroendocrine products to stimulate behaviour appropriate to a particular time and season. Mess et al.(14) suggest that the role of the pineal is to serve as a break or attenuator in the regulation of pituitary, trophic hormone secretion. Although the role of melatonin in man has not been defined exactly, it is clear that a simple stimulus and response theory for its actions is totally inadequate since it is mediated by and mediates a complex of hormonal activities that result in certain types of behaviour.

3 Light, body rhythms and melatonin secretion

Lewy et al.(14), who awakened healthy volunteer subjects from sleep during the night and exposed them to 2500 lx of incandescent light. Diminished concentrations of melatonin occurred some minutes after the exposure. There was less effect following exposure to 1500 lx, and 500 lx at the eye from a fluorescent lamp had no influence on melatonin concentration. Restoration of melatonin was fairly swift after the high light levels had been extinguished and soon reached the normal night-time value.

If people are exposed to a long continuous period of light, their melatonin rhythm may be markedly altered. According to Wurtman(15), restoration of the normal pineal rhythm may take several days in rats and in humans following exposure to a 24 hour period of light. Wever(16), experimenting with both incandescent and fluorescent lamps at illuminances up to 1500 lx found only very small effects on human circadian rhythms when subjects were exposed to constant illumination for periods between one and two weeks. But, in later experiments using illuminances above 3000 lx, there were marked alterations in the normal circadian rhythms of body temperature and the sleep–wake cycle, resulting in entrainments of greater than ± 5 h.

In most domestic and working interiors, illuminance levels are modest, commonly in a range of about 50 lx to 1500 lx. Simpson and Tarrant(17) found generally low levels of illumination in a survey of 50 owner-occupied houses and 51 rented houses, with a median illuminance for prolonged reading of 70 lx, hall illuminance of 30 lx and kitchens with a mean of 120 lx. Okudaira et al.(18) showed that actual illuminances received by individuals who work indoors but make some short excursions into the external environment experienced light intensities exceeding 1000 lx for less than one hour per day. Savides et al.(19) logged the continuous light exposure for 10 subjects during their working hours over the period August to November, in San Diego, California. The received illuminances were at low levels and subjects experienced more than 2000 lx of external illumination for only short periods. Cawthorne(20) conducted a survey in Cambridge, UK, monitoring the light received by subjects during the course of a day in locations with a high average, medium and a low average daylight factor (DF). The degree of glazing affected the light exposure, which in the high-daylight factor condition rarely exceeded 2500 lx, probably when the occupant was near a window or went outdoors. In the middle of the day (between 13.00 and 14.00) an average of 1551 lx was found for the high-DF interior and 155 lx for the low-DF interior.

It would seem from the above surveys that the light exposures that most people experience under normal living and working conditions are of insufficient intensity and duration to influence circadian cycles of melatonin activity to any significant degree. On the other hand, evidence shows that prolonged
exposure to abnormal lighting conditions, such as constant high illuminance or constant darkness, affects some bodily rhythms such that those rhythms having a normal 24-hour periodicity tend to run freely and the full circadian period is usually extended.

There are many rhythms in the bodily physiological activities of man and animals that exhibit different cycle times; common among them is the circadian rhythm with its 24-hour diurnal cycle, but there are also seasonal rhythms that have importance for animal behaviour. External influences (zeitgebers) such as light intensity and daylight duration (photoperiod) have a significant influence on these rhythmic patterns generated by the endogenous or internal clock. The suprachiasmatic nucleus in the hypothalamus is now considered to be the source of the body clock which generates the 24-hour rhythms. Environmental influences determine the timing and phase (entrainment) characteristics of a particular bodily rhythm.

In man the role of light as a zeitgeber was once thought to be relatively weak, with social and other stimuli playing a more dominant role. However, more recent research shows that unusual photoperiods of light exposure may influence bodily rhythms and melatonin secretion. Lewy et al. examined the time of onset of melatonin production in a group of healthy volunteer subjects when the day length was artificially changed and the onset of dusk was advanced so that their sleep period fell between 23.00 and 06.00. After one week, the plasma melatonin showed a phase advance in production of about 2.5 h, indicating that the melatonin responded to the ‘false’ evening. In a further experiment, in which dawn normally occurred between 06.00 and 07.30 but was artificially delayed to 09.00, a phase delay of 1 h in the onset and offset of melatonin was found.

Wehr showed significant lengthening of the night secretion of melatonin in healthy volunteers, during an artificially induced 4-week ‘winter’ dark–light cycles of 14 h of darkness compared with an artificial ‘summer’ period of 8 h of darkness. Sleep duration was increased in the ‘winter’ period.

Changes in bodily rhythms are known to occur following long-term exposure to environments in extreme latitudes. In the Arctic and Antarctic regions, cycles of light differ from those in the more temperate zones, with a long winter with little or no daylight and shorter summer periods when the light is almost continuous. The effect of such zeitgebers on people has long been of interest to polar and environmental scientists who have observed changes in bodily rhythms in different groups of people. There have been few studies on direct melatonin changes. One of them was conducted in the north of Finland by Martikainen et al. In this region in mid-June the day length is 22 h, and in mid-December it is 3.5 h. Over a period of 13 months, six plasma hormones including melatonin were analysed in 24 males aged 21–41 years. The results revealed two peaks in serum melatonin, in December and in May, and definite troughs in the periods March and August, a statistically significant variation in the melatonin annual rhythm. However, there was no conclusive evidence that changes in daylight were related to changes in the hormone levels—these may have been induced by some other factor. The authors suggested that any natural light effect may have been masked by living under artificial lighting conditions and by influences such as diet, mood, stress and extended physical exercise. Arendt and Broadway overcame one of these difficulties in their study in Antarctica since the artificial lighting remained constant at 250 lx. They showed a significant phase advance of the melatonin rhythm in summer by about 2 h compared with the winter rhythm. Midwinter and Arendt conducted observations on melatonin levels following night-shift duties in both winter and summer periods in Antarctica. The melatonin took a significantly longer time to return to base levels in winter, and differences were found in melatonin production during night shifts between winter and summer periods.

In summary, certain bodily functions showing a circadian periodicity in man may be disturbed in extreme latitudes, especially by seasonal light changes; but there is evidence that, even for nonindigenous settlers, normal rhythms of performance are not affected by the absence of solar time cues. However, the general evidence shows that melatonin exhibits different seasonal peaks of output in such environments and that these peaks are related to some quality of the environmental light, which is likely to be, at the least, illuminance. These effects are no doubt the basis for the sleep difficulties reported by personnel staying in polar regions. Insomnia or ‘the big-eye’, as the Americans call it, is an occupational disease in the Antarctic winter. The indications are that changes in various bodily rhythms induced by changes in photoperiods due to seasonal light changes are significant for behaviour in terms of mood, sleep states and fatigue.

4 Melatonin response to spectral irradiance

The influence of light of different wavelengths on melatonin responses is not a well-explored field, especially in man. Cardinali et al. compared the effects of coloured lights on the suppression of the enzyme hydroxyindole O-methyltransferase (a catalyst critical for the biosynthesis of melatonin; see Figure 3) in the pineal gland in rats. The light sources used were bulbs of narrow spectral band each delivering the same level of radiant energy, with peaks at 360 nm (near UV), 435 nm (blue), 530 nm (green), 590 nm (yellow) and 660 nm (red). It was shown that green light exerted the most inhibition on this pineal enzyme. In another study, by Vanacce and Illnerova, on the effects of red light on rat pineal activity, a strong red light was found to inhibit the enzyme N-acetyltransferase (see Fig 3), but the irradiance was 10 times greater than the minimally effective white light intensity required to induce the same effect. Brainard and co-workers’ study on Syrian hamsters showed that blue light (435–500 nm) had the most inhibitory effect on pineal melatonin when the animals were exposed to light at night. They showed that green wavelengths in the band 515–550 nm also had some effect, but there were no effects for red, yellow or near UV. Later they compared more specifically the effects of blue and green wavelengths at three irradiances (0.019, 0.074 and 0.186 μW cm⁻²) and they found that the blue wavelength was always more inhibiting than the green by about 25%. A study of particular interest is one by Brainard et al. on the suppression of pineal melatonin in three human subjects exposed at night to different monochromatic wavelengths. The peaks of the wavelengths to which three subjects were exposed were 448, 476, 509, 574 and 604 nm, with 10 nm half-peak bandwidths. Exposure was for an hour, following which plasma samples were analysed for melatonin content. Wavelengths and irradiances were carefully controlled and each subject received equal quanta of each wavelength on separate occasions that were at least 5 days apart. The percentage of the melatonin inhibition was greatest under the 500 nm band width (i.e. blue-green region). This result is particularly interesting and more critical since equal-energy sources were used. Typical
coloured lights in common use do not provide equal energies and are therefore not a true control condition.

In a simulated office experiment, Küller and Wetterberg(36) found no significant differences in urinary melatonin output from human subjects exposed to illuminances of approximately 450 lx and 1700 lx and no differences between 'Warm White' (3000 K) lamps and 'Daylight' (5500 K) lamps.

An early study by Wurtman and Weisal(27) examined the effects on various organs of two fluorescent lamps, one of which was described as 'Cool White' and the other as 'Vita-Lite', the latter being intended to approximate daylight. Male and female rats were exposed to the two forms of lighting at the same level of radiant energy of 630 μW cm⁻². This resulted in average illuminances under the 'Vita-Lite' of approximately 1540 lx and under the 'Cool White' source of 2120 lx. The animals were mated and the progeny were studied for up to 50 days under the experimental lighting conditions. These progeny were then killed and it was found that the testes and ovaries of rats born and reared under 'Vita-Lite' were significantly larger than those of animals exposed to the 'Cool White' source. Females under the 'Vita-Lite' had larger hearts and pineal glands than the females exposed to the 'Cool White' light. Male rats under 'Vita-Lite' had larger adrenals than those reared under the 'Cool White' source. All of these differences were statistically significant. Although the authors do not refer specifically to melatonin, there is presumably a hormonal effect induced in these rats that could be mediated by melatonin.

The significance of these findings in a biological sense is open to interpretation, but of immediate interest is the fact that different spectral irradiances have a differential influence on activity, the blue-green region appearing to have the maximum inhibitory effect in humans. In view of the long evolutionary history of the pineal body, it might be supposed that coloured light was of significance in the habitats of man's forebears and that the quality of forest light, for example, might have an important bearing on melatonin inhibition during daylight hours to maintain alertness.

5 Mood states, pineal activity and light therapy

Comments about a possible relationship between mood states, weather conditions, light and the seasons have been made since the time of the Greek philosophers, and later by clinicians, sociologists, biologists and others. But the association was largely anecdotal until work by researchers at The National Institute of Mental Health, Maryland, and at Oregon Health Services Laboratory, Portland, Oregon, USA provided a firm foundation for knowledge of the role that light can play in altering mood states. Lewy et al.(38) first showed in a manic depressive patient with a seasonal mood cycle that bright light could modify this rhythm and relieve the depression phase. Many follow-up studies have been conducted, confirming the original work. It is important to emphasise that patients have been selected from a subgroup of a more general condition known as bipolar affective disorder. The name seasonal affective disorder (SAD) has been given to the condition that typifies these subgroups. During the winter, affected individuals feel depressed, slow down, and generally oversleep, overeat and crave carbohydrates. In spring and summer they are elated, active and energetic and generally function well(39). The condition also affects children but generally to a lesser degree, although sufficiently to affect school life(40).

The light sources generally used in therapeutic procedures for SAD are so-called full-spectrum fluorescent lamps arranged in a container fitted with a plastic diffusing screen. In the phototherapy experiments with patients suffering from SAD, high-intensity stimulation is usually at 2500 lx or greater and low intensity at 300 lx or lower. Most authors state the distance from the light box at which the illuminance measurement is taken (usually ~1 m) and it is to be assumed that this illumination is on the face, but that point is not always made explicit. It is probably appropriate to treat such illuminances as notional quantities as they may vary slightly with patient orientation to the light source. Other researchers have attempted to give more precise dosages with special equipment (e.g. reference 45). In a number of early studies, melatonin assays were conducted and measures of changes in mood state were recorded, typically using the Hamilton Rating Scale(46) and the Beck Depression Inventory(22).

Further evidence for the effect of bright light therapy was obtained by Rosenthal et al.(47). A group of patients was treated by bright white light (~2500 lx) and by dim yellow light (~100 lx). Scores on the Hamilton Rating Scale and on the Beck Depression Inventory fell significantly (indicating reduced depression) following bright light exposure, whereas no significant changes in scores occurred following the yellow light treatment.

Another study by Rosenthal et al.(48) showed that changes occurred in a self-rating mood measure between the 'before' and 'during' treatment phases, and again after treatment had been withdrawn. Significant differences were found (p<0.05) between pre- and post-treatment ratings in the mood measures under the bright light treatment and again between treatment and withdrawal. No significant differences were found between the same conditions for exposure to the dim light.

Joffe et al.(45) controlled the light exposure to 105 SAD subjects more carefully than had been done previously. Use of a light visor ensured that the subjects received accurate light dosages compared with open-field viewing using a light therapy box. Three illuminances of 60 lx, 600 lx and 3500 lx were used daily for two weeks, on three groups of patients who were scored for mood change on the modified Hamilton Rating Scale for Depression, SAD Version (HRSD-SAD)(46). No significant differences were found between the three light levels, although all the light exposures created a significant reduction in scores for depression. The authors suggest two possible reasons for this result. One is lack of a dose-response relationship, and the second is that light intensities over a wide range are biologically active.

A critical factor in phototherapy has been to extend the patient's daily light exposure (photoperiod) by prolonging the evening with artificial light and starting the day early by exposing the subjects to light first thing in the morning. Both James et al.(49) and Rosenthal et al.(50) concluded that presenting high illuminances to subjects in the evenings is sufficient and that the early morning exposure did not appear to be critical for therapy. However, Dunham(46) comments that some patients profit from therapy in the morning and others from therapy during the evening. Lewy et al.(51) observed that some depressive patients have phase-advanced circadian rhythms and wake early in the morning and that these are better treated by light in the evening, but that patients with phase-delayed circadian rhythms are more successfully treated by light in the morning. Lewy therefore recommends that preliminary phase-typing is necessary to decide whether morning or evening light therapy is appropriate.

A fundamental question arising from these studies is the precise role of melatonin. Although melatonin is thought to
be an important factor in the mediation of SAD symptoms, the
details of the differences between melatonin secretion in
summer and winter in patients with SAD is not yet known[44].
A number of experimental studies have concluded that there
are lower concentrations of melatonin at night in depressed
patients. Lewy et al.[59] showed that depressed patients were
more sensitive to the suppression of melatonin by light.
Subsequent work by Lewy et al.[59] demonstrated very consid-
erable differences in sensitivity to melatonin suppression
between manic depressive patients and normal controls. It has
been suggested that supersensitivity to melatonin suppression
by light might be an individual trait marker, and that normal
subjects who show this may be predisposed to affective illness.
This could be an interesting concept for lighting research,
since such individual variations in melatonin response might
relate to differences in sensitivity to glare.

A question of particular importance is whether melatonin has
an effect on people who are not suffering from SAD. This has
been examined by administering melatonin as a drug to
normal subjects. One study[53] showed that sleep was induced
within one or two hours of melatonin administration.
Another study[52] showed an increase in fatigue in the evening
following melatonin treatment, but not all of the three
subjects reported an increase in sleepiness.

A more extensive study by Lieberman et al.[53] used perfor-
mance tests and standardised mood scales. The latter
included the Profile of Mood States (POMS) devised by
MacNair et al.[54] and the Stanford Sleepiness Scale (SSS)
devised by Hoddes et al.[55] and these were administered every
hour. The performance tests used were a simple auditory reac-
tion time, a long-duration auditory reaction time, a four-
choice visual reaction time, a grooved peg-board test, a digit
symbol substitution test, a critical flicker fusion test, and a
recall and recognition memory test. High doses of oral mel-
tatonin were administered to 14 healthy male volunteers,
subjects receiving three capsules of 80 mg of melatonin at
12.00, 13.00 and 14.00, respectively. The total dose was 240 mg
of melatonin, which ensured that melatonin would be high in
the plasma throughout the afternoon test session. The results
showed that melatonin significantly altered the scores on
several subscales of the mood tests. The SSS showed that
subjects reported increased sleepiness at 15.00 and 16.00, but
by 17.00 no significant effects were present. Compared with
baseline measures, the ‘vigour’ subscale on the POMS test was
significantly reduced by melatonin, and the ‘fatigue’ and
‘confusion’ subscales scores were significantly increased.
In the performance tests, melatonin significantly increased
response latency on the Four-Choice Visual Reaction Time
task, but the number of commissive errors was significantly
decreased. The number of errors on the Simple Reaction
Time task was also significantly reduced. None of the other
tests showed any significant effects of melatonin. Although
the plasma melatonin levels were still elevated at 17.00, the
mood state had returned to near normal baseline by that time.
According to Lieberman et al.[53], the melatonin dosage
produced changes in mood that were equivalent in magnitude
to those produced by a clinical dose of benzodiazepines.
However, there was no effect on memory impairment as there
would be with benzodiazepine ingestion. Sherer et al.[56]
administered oral melatonin to six SAD patients, and
conducted tests on psychomotor performance and memory
after one week of melatonin administration. They found that
reaction time on a simple visual reaction task was significantly
reduced, but there was no effect on memory or vigilance. This
is partly consistent with the study of Lieberman et al.[53] but
contradicts it on the reaction-time finding, which may be due
to a different dosage of melatonin or to its effects on circadian
rhythms influencing performance.

Lieberman et al.[53] conclude that the effect of bright light in
suppressing melatonin release could increase alertness or act
as a zeitgeber for circadian cycles. They also suggest that there
could be subtle and as yet unknown neurochemical and
behavioural consequences of living in the artificial lighting
environment typical of our society today. This would seem to
be unlikely for ordinary conditions in view of the work
referred to in section 3.

In many experiments and phototherapeutic trials reported
here, reference has frequently been made to the use of the
‘Daylight Simulating’ lamp or ‘Vita-Lite’ lamp, often referred
to as a ‘full spectrum’ lamp. This use has been interpreted in
more popular articles in newspapers and magazines as indic-
ating that the ‘Vita-Lite’ lamp is an ideal light source for inte-
rior lighting, and organisations have installed these lamps
when refurbishing their lighting systems. There has been a
presumption that such sources will help to dispel the depre-
sions of everyday life and provide positive stimulation for the
working day. But it must be remembered that the experiment-
tation on this subject relates only to alleviating serious clinical
depression arising from the condition known as seasonal
affective disorder. The popularity of the full-spectrum lamp
has possibly arisen from the fact that it has frequently been
used by researchers, probably because they wished to preserve
comparability between experimental trials. It was noted by
Wirz-Justice et al.[57] that when Vita-Lite lamps and other light
sources were compared, no differences were found for therape-
autic effects and Kuller and Wetterberg[58] failed to find a
differential effect on melatonin for two types of fluorescent
lamp. This suggests that spectral quality is not as significant a
parameter as illuminance level. Furthermore, Boray et al.[58], in
a laboratory study, found no significant differences in cogni-
tive task performance, in subjective ratings of room and occu-
parent attractiveness and in mood self-rating after subjects had
been exposed to ‘Cool White’ lamps (~ 4150 K) or ‘full spec-
trum’ lamps (~ 5000 K) or ‘Warm White’ lamps (~ 3000 K).
Veitch et al.[59] were similarly unable to find differences in
performance and mood among subjects working in a labora-
tory situation under either ‘full spectrum’ lighting (5000 K) or
‘Cool White’ lamps (4150 K).

From the results of these varied experiments it is clear that
unequivocal support for any superior benefit for the effects of
full-spectrum lamps on mood and performance cannot be
maintained. Furthermore, any extra value of these lamps
specifically for use in SAD phototherapy, compared with other
spectral power distributions, remains unestablished and it is
possible that high illuminance from any traditional lamp will
suffice.

6 The relationship of the pineal gland with other
glandular activities

The fact that in many mammalian and avian species light plays
an important role in seasonal rhythms of reproduction, migra-
tion, metabolic adjustments and circadian rhythms suggests
that there is a complex system response involving not only the
pineal gland or its homologue but also other glands such as
the gonads and the pituitary gland. Hormones such as oestro-
gens, androgens, progesterone and prolactin may all be
involved at appropriate times. It has been shown from human
and animal experiments that gonadotrophin and melatonin
production is decreased at the time of the preovulatory peak\(^{(60)}\). Melatonin can inhibit or stimulate activity in the pituitary, thyroid and adrenal glands as well as in the gonads. Such relationships enable the pineal to form a bridge between the photic environment and the synchronisation of different behavioural activities, although the exact way in which the pineal interacts with other endocrine mechanisms is not fully understood. Cardinali\(^{(60)}\) proposes that the pineal gland participates in the control of gonadal, adrenal and thyroid functions, and Wurtman\(^{(55)}\) suggests that the influence of the pineal is more probably exerted via the hypothalamic system rather than directly on other glands themselves.

A number of experiments have shown that lighting induces changes in adrenal gland products as well as in the gonadal system, and a strong assumption would be that the link is via the pineal gland and possibly the hypothalamus. An early experiment by Ishiu\(^{(62)}\) examined the effects of illumination at 3200 lx (the light condition) on endocrine changes in six healthy males and compared it with a dark condition (illuminance unspecified), subjects being exposed to each condition for 3 h. The light source was a 'Daylight' fluorescent lamp. Urine from the subjects was collected during the periods of each experimental condition and was assayed for a variety of steroids and urea. Blood samples were taken and serum potassium and sodium, cholesterol, blood sugar, lactic acid and pyruvic acid concentrations were measured. The results showed increases under the light condition of urinary androstosterone, androgen and 11-oxosteroid, adrenal androgen and glucocorticoid and gonadal androgen. An increase in serum potassium occurred under the light condition and decreased in the dark condition. Unfortunately, no statistical treatment was applied to these data so it is uncertain whether the results are really meaningful. They are, however, suggestive of steroid release.

Hollwich and Dieckhues\(^{(63)}\) compared four groups of people for cortisol levels over the period from 08.00 in the morning to 23.00 at night. The groups varied with respect to visual status: group 1 were people with normal sight, group 2 were blind people, group 3 were blind people with some ability to discern hand motion, and group 4 were blind people with some degree of light perception, i.e. partially sighted people. The results showed a significantly different mean level of cortisol between the blind and the fully sighted group, the cortisol level being much lower in the blind group; it was also significantly lower when compared with the group that could perceive only hand motion.

In earlier experiments, these authors\(^{(63)}\) had shown differences in cortisol and ACTH (adrenocorticotrophic hormone) levels in patients who were temporarily blind with cataracts and who later had the cataracts removed. Following cataract removal, ACTH and cortisol levels returned to normal. In a subsequent set of experiments on 18 young volunteer subjects, the effects of 'Cool White Light' fluorescent lamps were compared with natural daylight to determine whether there would be any effect on ACTH and cortisol following a 14-day exposure under each condition. The fluorescent lamps provided an illuminance of 3500 lx, but illuminance values for the daylight conditions were not stated. The results showed significant increases of ACTH and cortisol under the 'Cool White Light' fluorescent lamp condition. A final study by Hollwich and Dieckhues\(^{(63)}\) on 10 subjects compared natural daylight with 3200 lx for their effects on cortisol and ACTH excretion, but no significant differences were obtained. The conclusion was that the stress-like effects resulting from the 'Cool White' tube (previous experiment) were not present under the 'sunlight simulating' tube. These latter experiments again serve only as pointers, since they require better experimental control to allow clear conclusions to be drawn.

Erikson and Kuller\(^{(64)}\) studied 55 office workers in Sweden over 7 months from December to June, i.e. from the darkest to the lightest part of the Swedish year. About half of the subjects were exposed in their normal setting to 'Standard' fluorescent lamps and the other half to 'Daylight' fluorescent lamps. No illuminance values were stated. The two groups were working in the same building, one storey above the other, and all offices contained windows. On the first and the last days of the experiment, i.e. in December and June, urine from all subjects was collected and assayed for melatonin and cortisol. In addition, subjects were asked to complete an extensive questionnaire comprising self-rating scales. No significant differences were found for the two groups between December and June on any of the biochemical measures, but the relationships between the biochemical data and the five factors extracted from the self-rating scales are of interest. The four factors Hedonic Value (placid-RESTLESS), Activation (awake-drowsy), Social Mood (social-quiet), and In Control (dominating-submissive) all correlated with the cortisol index (a combination of melatonin and cortisol). A high value in the cortisol index meant a high rating on Activation. Furthermore, a high cortisol index differentiated the two lighting conditions on the self-rating factor of 'In Control': the higher the cortisol index, the greater the difference in rating of In Control between the Standard source (high positive ratings) and the Daylight source (high negative ratings). This interaction was significant (\(p=0.05\)). The authors state that definite conclusions about the desirability of different light sources cannot be drawn on the basis of the evidence. There are, however, indications that spectral quality is an important parameter in melatonin suppression and there is evidence for a relationship between mood state and the endocrine products cortisol and melatonin.

Further evidence for a melatonin–ACTH–cortisol link is provided by Brismar et al.\(^{(65)}\) who administered the drug metyrapone to 10 patients in order to induce cortisol reduction and release of ACTH and to observe the effect on melatonin secretion (by urinary assay). They found an increase in melatonin output with a parallel increase in ACTH and an inhibition of the cortisol response.

Küller and Wetterberg\(^{(66)}\) found no differences in either urinary cortisol or melatonin in human subjects exposed to a 'Warm White' lamp and a 'Daylight lamp' (Duro Test True Lite). It thus seems that these hormones are not necessarily sensitive to illumination by fluorescent lamps and the illuminances that are within the range of daily experience. On the other hand, if subjects are confined to windowless environments, or environments lacking adequate illumination, cortisol levels may be influenced by being delayed in attaining their seasonal level\(^{(66)}\). Table 1 summarises the results from experiments reviewed in the text of the effects of different lamp spectra and illuminances on various physiological responses in human subjects. It should be noted that some of these experiments included more variables than those reported here; the information in Table 1 has been selected in relation to the main themes of this paper.

It would appear from the evidence of the experiments reviewed here that the role of melatonin in both physiological and behavioural reactions has to be considered in terms of its reciprocal relationships with the adrenal and other glands.
This conclusion leads to a possible reconsideration of the role of melatonin in phototherapy and whether it is the main causal agent in the relief of depression. Jacobson and Rosenthal (67) pursue the following line of argument: 'Although the mechanism of phototherapy—antidepressant action in SAD is unclear, one clue might lie in the findings of several studies that light is capable of increasing arousal [Whiting, di~., Kallman and Isaac (66)], Chavez and Delay (70)].

It is tempting to develop this idea further in the light of work by Anderson et al. (80), who observed changes in urinary noradrenaline and its metabolites, other catecholamines and urinary free cortisol, before and after light therapy on a group of SAD patients. They found statistically significant decreases in noradrenaline, normetadrenaline and 3-methoxy-4-hydroxy-mandelic acid. These results suggest a possible addition or alternative to melatonin as a cause of the change in mood state following light therapy. Noradrenaline is found in the adrenal medulla, and in brain areas, especially in the hypothalamus.

Schildkraut and Kety (72) long ago drew attention to the concept of the catecholamine hypothesis of affective disorder', which contends that depression may be associated with a deficiency of noradrenaline and elation with an excess. In the experiment of Anderson et al. (80), the decrease in the urinary noradrenaline suggests an internal uptake following light therapy, resulting in a reduced depressive state, as was measured by the Hamilton Depression Scale. It is unknown whether such action could be mediated by light at the hypothalamic level via the suprachiasmatic nucleus or the paraventricular nucleus, or at the pineal gland level in the presynthesis of melatonin, or be induced by a melatonin feedback process. However, it does raise the question whether the action of light therapy is better explained by a catecholamine and arousal hypothesis rather than by an explanation in which melatonin is considered to be the central agent. An arousal hypothesis would seem to fit more readily with the idea of light as a stimulating agent.

7 Summary and conclusions

Melatonin production by the pineal gland is directly influenced by lighting conditions, its synthesis being inhibited by light whereas it is freely produced during darkness. An important function of melatonin is to synchronise many internal bodily rhythms with the external environmental circadian cycles of daylight and darkness and the changing photo-periods over the seasons of the year.

Disturbances in circadian rhythms can arise out of a mismatch between external time cues and internal timing of some aspects of bodily physiology. Such disturbances may arise, for example, from shift work, jet lag and blindness causing periods of fatigue, difficulties in sleeping, and changes in alertness and reaction time.

Experience of everyday interior lighting conditions including the occasional excursion into daylight is thought not to disturb circadian rhythms, but exposure to days of a continuous high light level is likely to cause some desynchronisation of certain bodily rhythms.

Abnormal circadian rhythms are considered to be causal factors in seasonal affective disorder. Light therapy has been shown to alleviate the depressive phase of this disorder for some patients, but the role of melatonin in such therapy is not fully understood; other physiological and biochemical factors may also be involved.

There is suggestive evidence for seasonal changes in melatonin output in northern latitudes of the world and it might be thought that SAD would feature prominently among people living in the extreme northern zones of Europe, such as in Finland. According to Wolpert (79), 'only about 5% of people in very northern latitudes experience SAD, but a much larger proportion experience milder changes in mood'. Boyce and Parker (76) raise the question whether the SAD syndrome is found in the southern hemisphere such as in Australia. They propose that the evidence supports a winter/autumn photo-period factor in SAD in the southern hemisphere.
It is important to distinguish between clinical depression and the everyday experiences of depression with which nearly everyone is acquainted. There is no doubt that prolonged darkness, grey skies and low levels of daylight will induce depressive feelings, but these are usually transitory and may be rapidly dispelled by a more cheering environment. The appearance of the sun or good interior lighting design can make an important contribution, and in this respect the factors of lightness and visual interest are significant. The effects of reduced visual variety under conditions of uniform dull grey skies or in winter darkness, and the consequent lack of perceptual stimulation, could also serve as factors that explain a depressed mood.

Lighting designers are sometimes requested to install ‘full-spectrum’ lamps in offices in the anticipation that they will add a special quality to working life. The implication is that a type of phototherapy is introduced into the office, but it can be seen from this review that these lamps are unlikely to serve that role. Practitioners need to be aware of such assumptions.

Clearly, more research is required for a deeper understanding of the role of light in mood states. It is essential to measure lighting conditions adequately in photometric or radiometric terms and to define accurately the spectral power distributions of light sources; descriptive labels such as ‘Daylight lamp’ are inadequate. It is also important to use standardised scales for subjective responses or for assessing behaviour and to employ some commonly agreed biochemical assays. For thorough-going investigations in this area, a multidisciplinary team is required who employ various standardised technical tools so that systematic relationships between the stimulus and biochemical and behavioural responses can be identified through the use of adequately measured variables.

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