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Acute increases in night-time plasma melatonin levels following a period of meditation☆

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Abstract

To determine whether a period of meditation could influence melatonin levels, two groups of meditators were tested in a repeated measures design for changes in plasma melatonin levels at midnight. Experienced meditators practising either TM-Sidhi or another internationally well known form of yoga showed significantly higher plasma melatonin levels in the period immediately following meditation compared with the same period at the same time on a control night. It is concluded that meditation, at least in the two forms studied here, can affect plasma melatonin levels. It remains to be determined whether this is achieved through decreased hepatic metabolism of the hormone or via a direct effect on pineal physiology. Either way, facilitation of higher physiological melatonin levels at appropriate times of day might be one avenue through which the claimed health promoting effects of meditation occur.

Keywords: Circadian rhythms; Meditation; Melatonin; Pineal; Relaxation

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1. Introduction

Meditation has been reported to have numerous acute effects on a diverse range of endocrine, metabolic and autonomic parameters (Jevning et al., 1992). Early scientific investigations of the transcendental meditation (TM) technique concluded that it produced a unique hypometabolic state (Wallace, 1970), but this claim was disputed in later studies which described TM merely as a biochemical resting state, perhaps no different to relaxation (Michaels et al., 1976). Since that time however, a number of studies have demonstrated clear differences between the physiological effects of TM and those of eyes-closed rest (Jevning et al., 1992), and there can now be little doubt that the technique can produce a physiological state which is qualitatively different to that resulting from simple rest.

Although the uniqueness of the physiological effects of meditation has not been clearly demonstrated against other more active relaxation strategies, such as biofeedback or progressive muscle relaxation (Morse et al., 1977; Shapiro, 1982), meditators consistently report greater subjective gains than have been reported for other relaxation methods (Shapiro, 1982). One may conclude from this that either there is a specific placebo effect for meditation, or that the physiological variables responsible for the meditative state have not been identified. Until recently (Massion et al., 1995), melatonin, the major hormone produced and secreted by the pineal gland had not been investigated as a variable in meditation research. However, a number of parallels between the reported effects of meditation and those of melatonin suggest a possible connection may exist between the two.

The pineal is situated in the epithalamus at the center of the brain and receives sympathetic enervation from the superior cervical ganglia via the conarian nerves (Moore, 1978). In mammals, the pineal produces and releases melatonin (Lerner et al., 1958) only during darkness and it is not detectable during the day, irrespective of whether the species is nocturnal or diurnal in its behavioural activity (Klein, 1985). The pineal rhythm of nocturnal melatonin release is generated by the biological clock located in the suprachiasmatic nuclei of the anterior hypothalamus (Moore and Eichler, 1972; Stephan and Zucker, 1972). In humans, melatonin release is robust and appears resistant to major change by sleep, general activity, a variety of stressors (Vaughan, 1986) except when severe (Monteleone et al., 1992), diet, alcohol or beverage ingestion (Norman et al.). While bright light can inhibit melatonin release (Lewy et al., 1980), and studies indicate that extended periods of rigorous night-time physical exercise can decrease or phase delay the onset of the melatonin rhythm (Van Reeth et al., 1994), no known natural substances or phenomena have been reported to reliably increase plasma melatonin levels, although some pharmacological compounds, e.g. monoamine-oxidase A inhibitors, are able to do so (Murphy et al., 1986). The stability of the melatonin rhythm makes it an ideal candidate for a biological timing hormone, a role which is indisputable for rhythms in seasonal breeding of photosensitive mammals (Arendt, 1986) and has been postulated also for daily rhythms (Armstrong, 1989).

In addition to the biological rhythm functions of melatonin, other seemingly non-rhythmic roles have been advanced, including; anti-cancer (Gupta et al., 1988;

Panzer and Viljoen, 1997), immunoaugmentation (Maestroni et al., 1986), anti-aging (Maestroni et al., 1988; Pierpaoli, 1998) and anti-stress (Pierpaoli and Maestroni, 1987). Although these properties have not yet been clearly established in humans, the fact that melatonin has been found to be an extremely potent antioxidant and free-radical scavenger (Reiter et al., 1997) suggests that it may have an important role in reducing the cellular damage associated with the wear and tear of normal day to day life.

In this context, it is interesting to note that anti-cancer (Solberg et al., 1995; Meares, 1979), immunoaugmenting (Wallace, 1989), anti-aging (Wallace et al., 1982) and anti-stress (Jevning et al., 1978a,b; MacLean et al., 1997) properties have also been claimed in relation to meditation. While the validity of the anti-cancer and anti aging claims in particular is debatable, the parallels with those made for melatonin are intriguing, and invite speculation that one of the mechanisms by which meditation might achieve some of its health benefits may be through an effect on circulating melatonin levels. With the above in mind, the following investigations were undertaken in order to test whether a period of meditation could acutely affect plasma melatonin levels.

2. Methods

Two groups of meditators were studied in two separate experiments. The protocol used entailed a repeated measures design consisting of control and intervention conditions conducted on separate nights, 2 weeks apart. A crossover design was used, with random allocation to either the control or intervention condition on the first night, followed by the alternative condition on the second night. The studies were carried out during winter months at the Austin-Repatriation Medical Centre, Heidelberg, Melbourne, and the experimental protocol was approved independently by the human ethics committees of both the medical centre and La Trobe University. Prior to participation, potential subjects underwent a screening interview to ensure that they were suffering from no medical or psychiatric illness, nor taking any medication, that might affect experimental outcome.

In the first experiment, 11 experienced meditators (six male and five female) who practised the advanced transcendental meditation Sidhi (TM-Sidhi) program were recruited. The mean age of this group was 38.82 years (S.D., 6.2) while mean experience in the TM technique was 10.8 years (S.D., 6.19). Mean usual bed-time was 22:44 (S.D., 46.69 min).

The intervention condition entailed a meditation session between 00:00 and 01:00 on one of the two nights. Although midnight is an unusual time to meditate, it was chosen because melatonin levels normally begin to peak and plateau around this time (between midnight and 04:00) and it is, therefore, a period of relative stability of the melatonin curve (Arendt, 1995). Subjects were instructed not to meditate on the afternoon prior to experimentation, to eat before 19:00, to avoid bright light, but otherwise keep to their normal daily activities. One participant was subsequently excluded for failing to keep to these guidelines, leaving ten subjects.

Between 21:30 and 22:00 a butterfly needle was inserted into a suitable vein in the cubital fossa region of the forearm. Baseline samples were collected at 22:00, 23:00. 23:30 and 00:00. Post-treatment samples were taken at 20 min intervals between 01:00 and 02:00. Apart from during the treatment period itself, subjects were free to talk, watch videos and imbibe light beverages, and the procedure was exactly the same on both nights. When undergoing the intervention condition, participants carried out their usual meditation practice, albeit at the unusual time. On the control night, participants spent the h between midnight and 01:00 sitting quietly. Lighting levels were monitored throughout the experiment and did not exceed 15 lux, a level well below that known to influence melatonin secretion (McIntyre et al., 1988; Trinder et al., 1996).

Blood was centrifuged immediately and separated plasma was stored at -20°C until assayed. Plasma melatonin levels were assayed using a radioimmunoassay (Fraser et al., 1983) with the intra-assay precision for $n = 10$ samples being 4.9% (cv%) for a melatonin pool of 82.4 ± 4.1 pg/ml, and an inter-assay precision of 9.9%. Formal post-experimental questioning of our subjects indicated no sleep during meditation, however, no objective validation of these subjective reports are available since subjects were not monitored via EEG. Nevertheless, previous polysomnographic evaluation of TM indicates that it is not the homogenous, unique state of consciousness as was originally thought, for in addition to the occurrence of the well publicised alpha levels, EEG patterns normally classified as stages of sleep can occur (Pagano et al., 1976). Nevertheless, there is no evidence that either the total amount of sleep (Jimmerson et al., 1977; Vaughan et al., 1976), or any sleep stage (Vaughan et al., 1979) is associated with an increase in melatonin release.

In the second experiment, seven practitioners (two males and five females) of another internationally well known form of yoga[1] were recruited. It was normal practice for these subjects to meditate for around 30 min, which is half that of the usual TM-Sidhi routine. As a result, we collected two extra post-treatment blood samples at 00:30 and 00:45 in this group. Apart from this, the experimental protocol was exactly the same as for the TM-Sidhi group. The mean age of subjects was 32.1 years (S.D., 7.01) while the mean number of years meditation experience was 5.77 (S.D., 4.45). Mean usual bed-time was 23:27 h (S.D., 52.2 min).

3. Results

Mean control and treatment night plasma melatonin concentrations are shown for each group in Fig. 1. It can be seen that the melatonin levels of the TM–Sidhi group rose steadily during the pre-treatment period, from similar points and at similar rates on both experimental nights. Post-treatment levels then diverged, with control night values plateauing while meditation night values contin-

[1] Practitioners agreed to participate in the research on the condition that their organization was not identified.

ued their rise for some time before they peaked and fell back to the final control night value. The Yoga group, represented in Fig. 1(B), followed a similar trend, even though they meditated for only half as long as the TM–Sidhi group. S.D.s are indicative of the normal inter-subject variation in normal melatonin secretion patterns, rather than within subject variance, which was the focus of the analysis.

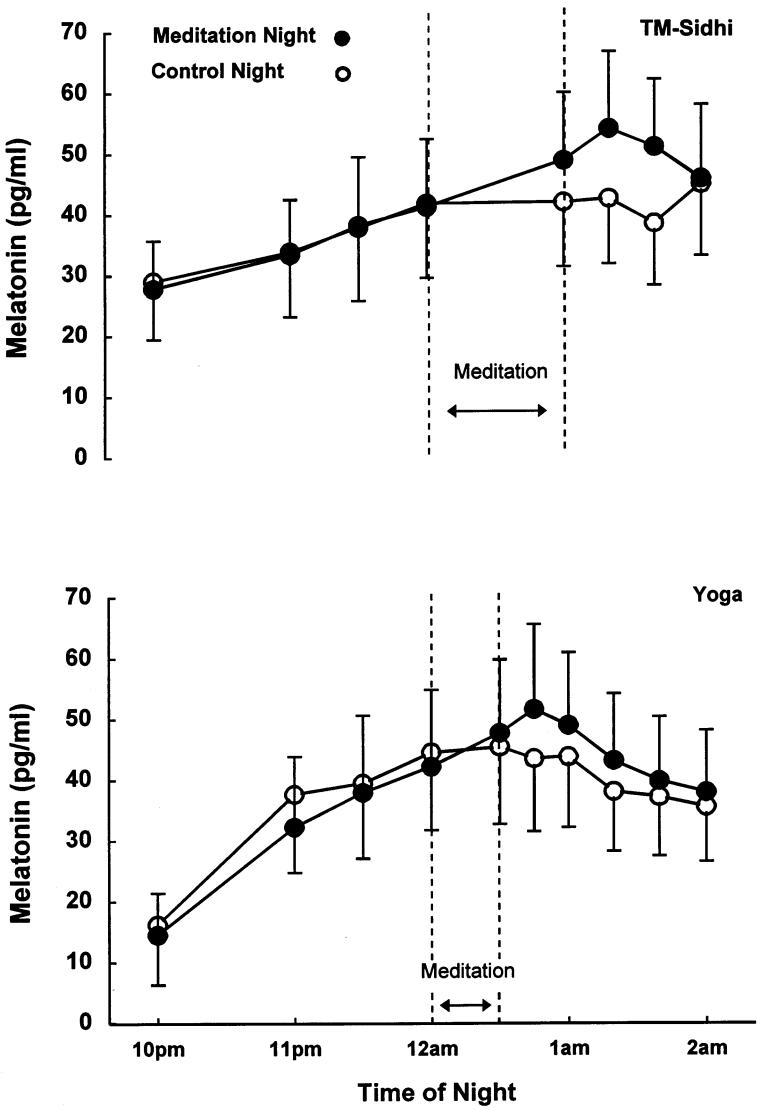

Fig. 1. Means and standard errors for plasma melatonin levels at each sampling point for the ten TM-Sidhi subjects and seven Yoga subjects on control and meditation nights.

Table 1
Individual values and group means for post-treatment area under the curve (melatonin pg/ml) on control and meditation nights

	TM-Sidhi 01:00–02:00h		Yoga 00:30–02:00h	
Participant number	Control	Meditation	Control	Meditation
1	54.00	90.00	173.85	195.40
2	66.50	141.00	46.90	68.35
3	45.00	58.50	25.00	26.45
4	209.00	278.00	167.55	198.50
5	154.00	160.00	254.30	261.20
6	122.00	129.00	384.40	423.55
7	141.00	158.00	369.90	412.00
8	28.00	42.50		
9	66.00	66.00		
10	363.00	405.00		
Group means	124.85	152.80	203.13	226.49
S.D.	101.41	111.85	142.33	153.42
F-test	$F(1,9) = 11.12,\ P < 0.01$		$F(1,6) = 16.19,\ P < 0.01$	

In order to statistically examine the hypothesis that meditation would cause an acute increase in melatonin levels, the summary measure of area under the curve (AUC) was calculated for each participant pre and post-treatment on each night using the trapezoidal rule. Pre-treatment AUC was calculated using the four samples collected between 22:00 and 0:00 h. For the TM-Sidhi group, pre-treatment melatonin AUC means were 87.77 pg/ml (S.D., 81.32) on the control night and 82.54 pg/ml (S.D., 77.14) on the meditation night. Yoga group control night pre-treatment levels were 135.29 pg/ml (S.D., 117.42) and 132.43 pg/ml (S.D., 103.27) on the meditation night. Post-treatment AUC encompassed samples collected immediately post-treatment (01:00 for TM-Sidhi and 00:30 for the Yoga group) until 02:00. Mean post-treatment melatonin AUC for the TM-Sidhi group was 124.85 pg/ml (S.D., 101.47) on the control night and 152.8 pg/ml (S.D., 111.83) post-meditation. Corresponding figures for the yoga group were 203.13 pg/ml (S.D., 142.33) on the control night and 226.49 pg/ml (S.D., 153.42) post-meditaton.

A two-way repeated measures ANOVA was conducted with treatment (meditation/control) and time (pre-/post-treatment) as independent variables. Due to the fact that the Yoga group meditated for only half an h, the post-treatment period over which AUC was calculated was a half h longer than for the TM-Sidhi group. Individual data for the post-treatment periods only and relevant statistics are presented in Table 1. Analysis of melatonin AUC TM-Sidhi group found a significant treatment by time interaction ($F(1,9) = 12.32,\ P < 0.01$). While there was no difference between control and meditation night AUC during pre-treatment ($F(1,9) = 0.88,\ P > 0.05$), post-treatment melatonin levels were significantly higher on the meditation night($F(1,9) = 11.12,\ P < 0.01$). Analysis of the yoga group data

resulted in similar findings, with a significant treatment by time interaction ($F(1,6) = 6.89$, $P < 0.05$), no difference in pre-treatment levels ($F(1,6) = 0.25$, $P > 0.05$), and significantly higher post-treatment melatonin AUC on the meditation night ($F(1,6) = 16.19$, $P < 0.01$).

Results indicate that, for both groups of meditators, while pre-treatment plasma melatonin levels did not differ between control and meditation nights, post-treatment levels were significantly higher following meditation than they were during the same period on the control night.

4. Discussion

Because the control period entailed sitting quietly with eyes open while meditation was mostly practiced with eyes-closed, it is reasonable to assume that participants were exposed to more light on the control night. Clearly, this could have implications with respect to the findings of this study, given that light of sufficient intensity and duration can have a suppressing effect on melatonin secretion (Lewy et al., 1980). However, the levels of light exposure during this study (below 15 lux) were well below the minimum intensity that has been documented to have a significant effect on melatonin levels (Aoki et al., 1998; McIntyre et al., 1989).

The results of this study support the hypothesis that a period of meditation can acutely increase night-time plasma melatonin levels. Although a recent pilot study (Massion et al., 1995) has offered preliminary evidence that meditators may consistently produce higher levels of melatonin, as far as we can determine, this is the first demonstration of acute increases in plasma melatonin levels as a result of a behavioural intervention. These increases are impressive given that there are no known substances which will reliably increase melatonin other than some classes of psychotropic drugs such as monoamine-oxidase A inhibitors (Murphy et al., 1986).

The mechanism by which meditation might induce an increase in plasma melatonin levels is uncertain. However, reduced hepatic blood flow has been reported to occur during meditation (Jevning et al., 1978a,b) and this may well slow the rate of melatonin metabolism, leading to higher plasma levels of the hormone. A mechanism by which meditation might lead to increased production and secretion of melatonin by the pineal is difficult to hypothesise at this stage, however other investigators have demonstrated that meditation can increase blood levels of noradrenaline (Lang et al., 1979) and urine levels of the serotonin metabolite 5HIAA (Bujatti and Riederer, 1976). Melatonin is synthesised in the pineal from serotonin, and the process is stimulated by noradrenaline acting on beta-adrenergic receptors on the pinealocytes (Moore, 1978).

Regardless of the mechanism, higher plasma levels of melatonin at the appropriate time of day could, theoretically, have a health promoting effect, for example, by prolonging its antioxidant activity. But the finding that melatonin is increased after meditation at midnight should not be interpreted as meaning that this occurs whenever a person meditates. Indeed, in the yoga literature it is usually suggested that meditation should be carried out in the early morning or evening. These are

times when melatonin levels are low, being likely to either have just fallen or about to rise. It may be that melatonin levels can be increased at these times, however it is both unlikely and undesirable, from a physiologic standpoint, that the hormone would be released during daytime as a result of meditation, since activity of the rate limiting enzyme, *N*-acetyl transferase, is low (Klein, 1985) and light would inhibit the release of the hormone even if its synthesis did occur (Klein, 1985). Furthermore, the health benefits of meditation are expected to accrue over years of practice, rather than from single episodes, and an effect on plasma melatonin levels is likely to be just one of the mechanisms by which meditation can have a positive effect on the well-being of practitioners.

The results of the present study raise the possibility that it is via the pineal and its principal hormone, melatonin, that at least some of the health benefits claimed for meditation could occur. It has been suggested previously that the TM Sidhi program may act as a zeitgeber for synchronised hormone release (Werner et al., 1986), and it is perhaps through an effect on melatonin, which is postulated as a mammalian internal zeitgeber (Armstrong, 1989), that it might achieve this.

Clearly, further research is required to establish the acute effects of meditation on melatonin levels at different times of the day, particularly at the time it is normally practiced. Research should also focus on comparing the effects of meditation with those of other relaxation strategies that are more active than simple eyes-closed rest, such as biofeedback and progressive muscle relaxation. Researchers might also look at the different yogic techniques within meditation to assess whether any of these is particularly capable of affecting melatonin levels.

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