Melatonin Supplementation from Early Morning Auto-Urine Drinking

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Abstract — Drinking one's morning urine ('amaroli') is a traditional practice of the yogic religion still widely performed. The pineal hormone melatonin and its conjugated esters are present in morning urine in significant quantities. Drinking the first morning urine restores plasma night-time melatonin levels due to deconjugation of its esters to melatonin. Exogenous melatonin, by either regulation of the sleep-wake cycle or enhancement of the physiological prerequisites for meditation (decreased body awareness (i.e. analgesia) and claimed slowed brain wave activity and heightened visualization ability) may be the mechanism behind the alleged benefits of 'amaroli'.

Introduction

'Behind the unintelligent practice, which doubtless to some extent exists amongst the multitude of every faith, I felt sure there must be a rational principle, since men on the whole do not continue throughout the ages to do that which is in itself meaningless and is therefore without result'(1).

Drinking one's own morning urine ('amaroli') is a traditional practice of the yogic religion still widely performed, particularly in the Indian Subcontinent. The ancient yogic text 'Damara Tantra' calls auto-urine 'shivambu kalpa vidhi' ('urine, revitalise technique'). In the popular literature it is widely extolled as a prophylactic, an adjunct to traditional therapies and a cure for ailments ranging from skin disorders to AIDS and cancer (2-6). 'Amaroli' seems originally, however, to have been incorporated into yoga as an aid to meditation (7). The following description is from the 'Damara Tantra':

'A sensible man gets up early in the morning when three quarters of the night has passed (i.e. about 3 or 4 o'clock), faces east and passes urine' (verse 6). 'The initial and concluding flow of urine is to be discarded. The intermediate flow is to be consumed. This is the most suitable method' (verse 7) (8).

The 'Hatha Yoga Pradipika' explains that the first part of the flow is discarded because 'it is a mixture of too much bile' and the last part as it is 'useless' (7, 9). The practice was traditionally performed in secret, in isolation, together with prolonged fasting and meditation as well as a diet of fruit and veg-
etables (8). Whether the fasting and ‘amaroli’ were designed to encourage meditation or meditation and fasting to enhance ‘amaroli’ (i.e. by increasing the levels of hormonal ‘breakdown products’ in the urine (10)) is unclear.

Two common contraindications are:

1. a diet containing excess animal products, processed or refined foods or spices; or
2. recent use of drugs (i.e. alcohol, tobacco) or medications (7).

One current practitioner of South Indian ‘siddha’ medicine requires patients to consume, for one month, approximately 100mls (‘three handfuls’) of midstream morning urine after a prior monofruit diet, alleging that the urine of prepubescent has the greatest potency (11).

Little research has been done on whether ‘amaroli’ has any beneficial effects and on the possible mechanisms by which they might arise. Bartsch and Bartsch, however, reported that: ‘urine from healthy individuals shows an anti-tumor effect, which, we think, may be due to the presence of pineal gonad-inhibiting substances’ (12).

Discussion

Melatonin in urine

Amongst the substances normally excreted in morning human urine (13) is the pineal hormone melatonin and its liver conjugated metabolite 6-hydroxymelatonin sulfate (6HMS) (14). Maximum plasma melatonin levels are found between midnight and 06.00h with mean peak levels of 40–80pg/ml at 02.00h (15). Day-time levels are very low, often undetectable by radioimmunoassay techniques (less than 10pg/ml) (14).

The clearance of melatonin is unusual. Circulating melatonin is taken up by all tissues, including the brain. It is rapidly metabolised by hydroxylation at position 6 of its indole ring; afterwards the ester is conjugated in the liver with sulfate (70%) and glucuronic acid (6%) (16). This rapid metabolism may explain why, despite high levels of conjugation, urine concentrations of the remaining melatonin are equivalent to those of plasma (17). Lynch, for example, found a urinary melatonin maximum of 52 pg/ml (18), Akcrsteck 93 pg/ml (19) and Mills 41 pg/ml (20). Diurnal urinary variations of melatonin and 6HMS levels correlate with those of plasma (21).

Light and melatonin

Production and hence release of melatonin is inhibited for a subject with open eyes by 2500 lux light with a 509 nm wavelength (2500 lux is approximately three times the artificial indoor light intensity but only 3–5% the intensity outside on a sunny day) (22, 23). Light stimulates the retinal rod cells. The resultant signal, via the optic nerve, inhibits the suprachiasmatic nucleus (SCN) signal to the pineal.

The SCN has projections to the superior cervical ganglion which in turn projects to the pineal via the conarian nerve (24). (Kneisley et al reported abolition of the day-night urinary melatonin rhythm in 6 patients with clinical evidence of transected cervical spinal cords (25)). When so inhibited by light, this pathway results in the abolition of noradrenaline release from pinealocyte sympathetic endings. Normally noradrenaline acts on beta-adrenergic receptors, which are coupled to adenyl-cyclase. This enzyme triggers protein phosphorylation mediated induction of the pineal enzyme N-acetyl transferase (NAT), which in turn governs the conversion of serotonin (5HT) to melatonin. Deactivation of pinealocyte beta adrenergic receptors with 40 mg oral propranolol (a beta receptor blocker) at 20.00 h can also prevent nocturnal release of melatonin (26). It is by this mechanism that light entrains the pineal gland to produce melatonin (27).

In total darkness, or in blinded humans (28), melatonin is secreted with a mean periodicity of approximately 25 h (29). This means, for example, that if peak release occurs at midnight on day 1 of a measured period, it will arise at 01.00 h on day 2 and 02.00 h on day 3. For a person with open eyes this pattern is ‘chopped off’ by the sunlight of dawn each morning through the circuit previously described. The intrinsic rhythm persists for 4 days upon rapid travel through 12 time zones (i.e. abrupt change of the daily dark period from 23.00 h to 07.00 h to 11.00 to 19.00 h) indicating that the source of the signal may be a ‘clock’ such as the SCN (30).

Effects of melatonin administration

Soon after melatonin was isolated and its structure determined (31) it was reported to facilitate sleep when introduced directly into the cat hypothalamus (32). Reports show that melatonin administration altered sleep and behaviour (33, 34). In 100 humans who had taken exogenous melatonin the only consistent effects were mild sedative and analgesia (35, 36). A 1.7 mg nasal spray melatonin produced sleep (not polygraphically reported) and subsequent feelings of ‘emotional balance’ in 7 of 10 subjects as against 1 in 10 for placebo (37). Administration of three 80 mg
doses at noon, 13.00 h and 14.00 h to 14 healthy male volunteers produced similar changes, on validated questionnaires of vigor, mood and sleepiness to those produced by hypnotic drugs (38).

The time of administration appears to be crucial in exogenous melatonin's effects on circadian sleep cycles. Chronic 1 mg/kg/day melatonin given at the onset of daily activity entrains the daily activity rhythms of rats kept in constant darkness (39). Exogenous melatonin administration may have similar effects in humans (40). Birkeland reported that melatonin is released in pulses during the light phases of human sleep and proposed that this may prevent awakening and restore deep sleep (41). Claustrat, however, found no correlation between melatonin peaks and minima and sleep stages or spontaneous waking in 6 healthy humans (42). Ingestion of melatonin at the bedtime of a new destination has been suggested to minimise jetlag (43). The mechanism may involve melatonin's ability to lower body temperature (44), the onset and duration of sleep depending on the phase of the circadian core body temperature rhythm (45).

Anton-Tay administered up to 1.25 mg/kg melatonin dissolved in 10ml of 1% ethanol solution intravenously at 16.00 h to 5 males and 5 females aged 18–25 years. Compared to three prior sessions involving only 1% ethanol, subjects experienced slowing of the electroencephalographic readings, increased visual imagery, a subjective sense of elation and an alteration in time estimation (33).

**Urinary melatonin supplementation**

The doses of exogenous melatonin experimentally administered to humans to date have exceeded the maximum night-time plasma level by a factor of 10–100 (46). Both oral and endogenous melatonin are rapidly metabolised (46), and the level in urine as a maximum 100 ml bolus dose of between 52–93 pg/ml would increase the mean peak plasma values of 40–80 pg/ml only be approximately 2 pg/ml or about 2%, if 'amaroli' is performed before dawn. However, melatonin is stable in acidic environments such as that of the stomach, whereas esters (47) such as 6HMS are unstable and could readily deconjugate to produce an two-fold increase of approximately 200 pg/ml of plasma melatonin. Thus such deconjugation could easily restore plasma melatonin to night-time levels.

**Conclusion**

There are certain elements in the practice of 'amaroli' which suggest a connection with melatonin. First, the recommended time of ingestion coincides with the greatest amount of melatonin in urine. Second, the urine of prepubescents is regarded as being superior in efficacy. Waldhauser has shown that a reduction in nocturnal secretion of melatonin occurs at puberty (48) coinciding with a diminution in sleep quality (49). Third, it is traditionally recommended that auto-urine be taken daily over a month, melatonin requiring similar chronic administration to entrain sleep cycles. Exogenous melatonin at the commencement of daily activities may 'convince' the body that it has had more sleep. Fourth, the traditionally recommended period for yogic meditation is from 04.00 h to 06.00 h (8). The injunction to perform 'amaroli' prior to this may involve a recognition of its ability to promote analgesia (a constant sitting posture may need to be maintained for 2 h) along with slowing of brain-wave activity and the visualization capacity considered necessary for many meditative practices (8). In fact we suggest that such effects are mediated via exogenous melatonin. Romijn, for example, has claimed that the rise in urinary indoleamine metabolites observed during a standardised meditation technique 'ought not to be primarily attributed to the stimulated enterochromaffine cell system but rather to enhanced activity of the pineal gland' (50).

**Further research**

Before and after 'amaroli', radioimmunoassays (14, 15, 36, 41, 42, 49) or HPLC analyses (20) could be performed for melatonin on plasma and urine (in free and conjugated form) to verify our hypothesis that an increase in the levels of plasma melatonin occurs. Subjects' melatonin levels and mental states could be compared when using their night urine to when using their (stored) day urine in a 'double-blind' manner to verify our hypothesis that melatonin supplementation from early morning auto-urine drinking promotes tranquility during meditation.

**References**

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