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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-

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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, alledging that the urine of prepubescents 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 gonal-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), Akerstedt 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 adminstration

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 mediation 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

1. Woodroffe J. The garland of letters: studies in the mantra-sastra. Ganesh and Co, Bombay, 1974.
2. Patel RM. Auto-urine therapy. Bharat Sevak Samaj Publications, Ahmedabad, 1978.
3. Jagdish B. Practical guide to auto-urine therapy. Jagdish B Publications, Bombay, 1978.
4. Karlekar RV. Auto-urine cure. Shree Gajnan Book Depot, Bombay, 1972.
5. Armstrong JW. The water of life. Health Science Press, Rustington, 1971.
6. Mithal CP. Urine therapy. Pankaj Publications, New Delhi, 1978.
7. Muktibodhananda S, Satyananda S. Hatha yoga pradipika — the light on hatha yoga. Bihar School of Yoga, Munger, Bihar, India, p441, 1983.

8. Satyananda S. A systematic course in the ancient tantric techniques of yoga and kriya. Grafika, Bombay, 1981.
9. Bernard T. Hatha yoga — the report of a personal experience. Rider and Co, London p71, 1971.
10. Karmananda S. Yoga and cardiovascular management. Bihar School of Yoga, Munger, Bihar, India, p121, 1982.
11. Poomanand G. Jyothishmathi, Nagalur Road, Yercaud-2, Salem, India (Personal communication).
12. Bartsch H, Bartsch C. Effect of melatonin on experimental tumors under different photoperiods and times of administration. Journal of Neural Transmission 52: 269, 1981.
13. Harper HA. Review of physiological chemistry. Lange Medical Publications, Los Altos, p416, 1975.
14. Arendt J. Assay of melatonin and its metabolites: results in normal and unusual environments. p11 in Melatonin in Humans, Journal of Neural Transmission (Supplementum 21) (RJ Wurtman, F Waldhauser, eds) Springer-Verlag, Wien-New York, 1986.
15. Arendt J, Wetterberg L, Heyden T, Sizonenko PC, Paunier L. Radioimmunoassay of melatonin: human serum and cerebrospinal fluid. Hormone Research 8: 65, 1977.
16. p374 in ref 13.
17. Lang V, Kornemark M, Aubert ML, Paunier L, Sizonenko PC. Radioimmunological determination of urinary melatonin in humans: correlation with plasma levels and typical 24 hour rhythmicity. Journal of Clinical Endocrinology and Metabolism. 53: 645, 1981.
18. Lynch HJ, Ozaki Y, Wurtman RJ. The measurement of melatonin in mammalian tissues and body fluids. p251 in The Pineal Gland, Journal of Neural Transmission (Supplementum 13) (I Nir, RJ Reiter, RJ Wurtman, eds) Springer-Verlag, Wien-New York, 1978.
19. Akerstedt T, Forberg JE, Friberg Y, Wetterberg L. Melatonin excretion, body temperature and subjective arousal during 64 hours of sleep deprivation. Psychoneuroendocrinology 4: 219, 1979.
20. Mills MH, King MG, Keats NG, McDonald RA. Melatonin determination in human urine by high-performance liquid chromatography with fluorescence detection. Journal of Chromatography 377: 350, 1986.
21. Tetsuo M, Markey SP, Kopin IJ. Measurement of 6-hydroxymelatonin in human urine and its diurnal variations. Life Sciences 27: 105, 1980.
22. Lewy AJ, Wehr TA, Goodwin FK, Newsome DA, Markey SP. Light suppresses melatonin secretion in humans. Science 210: 1267, 1980.
23. Brainard GC, Lewy AJ, Menaker M, Fredrickson RH, Miller LS, Weleber RG, Cassone V, Hudson D. Effect of light wavelength on the suppression of nocturnal plasma melatonin in normal volunteers. Annals of the New York Academy of Science 453: 376, 1985.
24. Ebadi M, Govitrapong P. Neural Pathways and neurotransmitters affecting melatonin synthesis. p125 in Melatonin in Humans, Journal of Neural Transmission (Supplementum 21) (RJ Wurtman, F Waldhauser, eds) Springer-Verlag, Wien-New York, 1986.
25. Kneisley LW, Moskowitz MA, Lynch HJ. Cervical spinal cord lesions disrupt the rhythm in human melatonin excretion. p311 in The Pineal Gland, Journal of Neural Transmission (Supplementum 13) (I Nir, RJ Reiter, RJ Wurtman, eds) Springer-Verlag, Wien-New York, 1978.
26. Moore DC, Paunier L, Sizonenko PC. Effect of adrenergic stimulation and blockade on melatonin secretion in the human. Progress in Brain Research 52: 517, 1979.
27. Mills MH, King MG. Applications of HPLC in neurochemistry. p72 in The Clinical Biochemist: High Performance Liquid Chromatography in the Clinical Laboratory, AACB Publications, Sydney, Australia, 1986.
28. Lewy AJ, Newsome DA. Different types of melatonin secretory rhythms in some blind subjects. Journal of Clinical Endocrinology and Metabolism. 50: 204, 1983.
29. Lewy AJ, Sack RL, Miller LS, Hoban TM, Singer CM, Samples JR, Kraus JL. The use of plasma melatonin levels and light in the assessment and treatment of chronobiologica sleep and mood disorders. p311 in Melatonin in Humans, Journal of Neural Transmission (Supplementum 21) (RJ Wurtman, F Waldhauser, eds) Springer-Verlag, Wien-New York, 1986.
30. Lynch HJ, Jimmerson DC, Ozaki Y, Post RM, Bunney WE, Wurtman RJ. Entrainment of rhythmic melatonin secretion from the human pineal to a 12-hour phase shift in the light-dark cycle. Life Sciences 23: 1557, 1978.
31. Lerner AB, Case JD, Takahashi Y, Lee TH, Mori W. Isolation of melatonin, the pineal gland factor that lightens melanocytes. Journal of the American Chemical Society. 80: 2587, 1958.
32. Marczynski TJ, Yamaguchi N, Ling GM, Grodzinska L. Sleep induced by the administration of melatonin (5-methoxy-N-acetyltryptamine) to the hypothalamus in unrestrained cats. Experientia 20: 435, 1964.
33. Anton-Tay F, Diaz JL, Fernandez-Guardiola A. On the effect of melatonin upon human brains: its possible therapeuitc implications. Life Sciences 103: 841, 1971.
34. Cramer H, Rudolph J, Consbruch U, Kendel K. On the effects of melatonin on sleep and behavior in man. Advances in Biochemistry and Psychopharmacology 11: 187, 1974.
35. Lerner AB, Nordlund JJ. Melatonin: clinical pharmacology. p339 in The Pineal Gland, Journal of Neural Transmission (Supplementum 13) (I Nir, RJ Reiter, RJ Wurtman, eds) Springer-Verlag, Wien-New York, 1978.
36. Wetterberg J. Melatonin in serum. Nature 269: 696, 1977.
37. Vollrath L, Semm P, Gammel G. Sleep induction by intra-nasal application of melatonin. In: Birau N, Schloot W (eds) Melatonin Current Studies and Perspectives, Pergamon Press, London, 1975.
38. Lieberman HR, Waldhauser F, Garfield G, Lynch HJ, Wurtman RJ. Effects of melatonin on human mood and performance. Brain Research 325: 201, 1984.
39. Rerdman J, Armstrong S, Ng KT. Free-running activity rhythms in the rat: entrainment by melatonin. Science 219: 1089, 1983.
40. Wurtman RJ, Lieberman HR. Melatonin secretion as a mediator of circadian variations in sleep and sleepiness. Journal of Pineal Research 2: 301, 1985.
41. Birkeland AJ. Plasma melatonin levels and nocturnal transitions between sleep and wakefulness. Neuroendocrinology 34: 126, 1982.
42. Claustrat B, Brun J, Garry P, Roussel B, Sassolas G. Repetitive study of nocturnal plasma melatonin profile and sleep recording in 6 healthy human males. p483 in Melatonin in Humans, Journal of Neural Transmission (Supplementum 21) (RJ Wurtman, F Waldhauser, eds) Springer-Verlag, Wien-New York, 1986.
43. Armstrong SM, Cassone VM, Chesworth MJ, Redman JR, Short RV. Synchronisation of mammalian circadian rhythms by melatonin. p375 in Melatonin in Humans, Journal of Neural Transmission (Supplementum 21) (RJ Wurtman, F Waldhauser, eds) Springer-Verlag, Wien-New York, 1986.
44. Ralph CL, Firth BT, Gern WA, Owens DW. The pineal complex and thermoregulation. Biological Reviews 54: 41, 1979.
45. Zulley J, Wever R, Aschoff J. The dependence of onset and duration of sleep on the circadian rhythm of rectal tempera-

ture. Pflugers Archives. 391: 314, 1981.
46. Arendt J, Bojkowski C, Folkard S, Franey C, Minors DS, Waterhouse JM, Wever C, Wildgruber C, Wright J. Some effects of melatonin and the control of its secretion in man. p266 in Ciba Foundation Symposium: Photoperiodism, The Pineal Gland and Melatonin. Pitman, London, No 117, 1985.
47. p418 in ref 13.
48. Walderhauser F, Weisenbacher G, Frish H, Zeitlhuber U, Walderhauser M, Wurtman RJ. Fall in nocturnal serum melatonin during prepurberty and pubescence. The Lancet 1: 362, 1984.
49. Carskadon MA, Harvey K, Duke P, Anders TF, Litt IF, Dement WC. Pubertal changes in daytime sleepiness. Sleep 2(4): 453, 1980.
50. Romijn HJ. The pineal, a tranquilizing organ? Life Sciences 23: 2257, 1978.