

Pharmacological Induction of Lucid Dreams

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Abstract

This article explores the effectiveness of using supplements to induce lucid dreams. The method of combining a cholinergic stimulating substance (galantamine & choline) with a deep sleep suppressing substance (sulbutiamine, caffeine, or desmopressin) is examined. Eleven nights of EEG data were recorded from a subject skilled in lucid dreaming techniques. On each experimental night, the subject slept for 3.5-4.5 hours before consuming either a deep sleep suppressing substance (DSS), a cholinergic stimulating substance (CS) or a combination of the two. The subject experienced a lucid dream in five out of five trials that included a combination of cholinergic stimulation with deep sleep suppression. Furthermore, the five lucid dream events occurred in the span of 10 calendar days and were all characterized as Wake Initiated Lucid Dreams (WILDs) in which conscious awareness remained during the transition from the waking state to the dream state. A second observation relating the influence of dopamine uptake to the subject's control over the dream environment is also discussed.

Key Words: Lucid, dreaming, dreams, sleep, galantamine, supplements, LDS

Introduction

Lucid dreaming is a specific state of consciousness defined as consciously perceiving and recognizing that one is in a dream while one is sleeping. More simply stated: “dreaming while knowing that you are dreaming.” [LaBerge, 1993]

Over the centuries many mental exercises have been developed to help improve the capacity to dream consciously. Nonetheless, learning to reliably induce

lucid dreams is quite difficult for most people [Holzinger, LaBerge, & Levitan, 2006]. Traditionally those interested in learning how to induce lucid dreams have relied on a combination of increased dream recall, state testing (i.e. performing reality checks), and recognizing dream signs [LaBerge & Rheingold, 1990]. This approach has been successful for some, but many struggle to achieve only limited or fleeting success.

Another characteristic of traditional induction techniques is they tend to favor a specific type of lucid dream referred to as a DILD (Dream Initiated Lucid Dream) [LaBerge & Rheingold, 1990]. A DILD occurs while a person is asleep and dreaming, they notice something odd and suddenly realize they are in the midst of a dream. DILDs start as regular non-lucid dreams. There is however, a second type of lucid dream often associated with a greater sense of awareness, increased control of the dream environment and enhanced reasoning abilities [Yuschak, 2006]. This type of lucid dream is referred to as a Wake Initiated Lucid Dream (WILD) [LaBerge & Rheingold, 1990]. Unlike DILDs, a WILD is characterized by moving directly from the waking state into the dream state while maintaining an unbroken thread of self awareness of both one's identity and one's situation. One moment a person is conscious in their physical body and the next moment they are conscious in their dream body with a continuity of self that spans the transition. Although WILDs generally include increased mental clarity, the traditional Western-developed induction techniques can rarely produce them. It has been reported that less than 20% of successful lucid dream attempts can be characterized as WILDs when using the established techniques [LaBerge & DeGracia, 2000].

There is however a pharmacological alternative that acts to either substitute or compliment the conventional induction methods (e.g. Wake-Back-To-Bed). This approach is referred to as the Lucid Dream Supplement (LDS) technique [Stride, 2006]. The LDS technique is not a faddish alternative to the conventional methods; it significantly out performs them in terms of lucid dream frequency, duration, and in the number of WILDs produced [Yuschak, 2006]. With the many potential benefits of lucid dreaming, it would be a worthwhile achievement to provide a more reliable means of making lucid dreaming accessible to the general public. If this could be achieved, the ability to dream lucidly could undergo a transformation from merely finding reliable ways of inducing lucidity to better exploring what can be accomplished in the dreaming state. The LDS technique provides one such path to accomplish this goal.

Although the LDS approach is not a new idea, there is very little information available to the public and scientific community describing how to use safe and commercially available substances to enhance dreaming and promote lucid dreaming. It is generally understood, and documented, that dreams can be dramatically influenced by ingesting certain foods or substances (drugs) that affect the levels of various neuro-chemicals within the brain. For example, neurotransmitters that have demonstrated the biggest impact on nightmares are dopamine, serotonin, and norepinephrine; with possible associations existing with acetylcholine, GABA, and histamine [Pagel & Helfter, 2003].

It has been previously shown that certain substances can promote lucid dreaming [Sergio, 1988; LaBerge, 2003; Yuschak, 2006], however there remains

much more experimental work that needs to be completed in order to fully understand and optimize the approach. The emphasis thus far has been on substances that mainly stimulate the cholinergic system, either by increasing the production of acetylcholine [Sergio, 1988] or by inhibiting its break down [LaBerge, 2003]. The cholinergic system is known to play an important role in the regulation of sleep, primarily by providing the stimulus to initiate and prolong REM sleep [McCarley, 2007], the phase where most lucid dreaming is thought to occur [Holzinger, LaBerge, & Levitan, 2006]. This system also plays an important role in regulating and maintaining the memory function [Gron, Kirstein, Thielscher, Riepe, & Spitzer, 2005] which is involved not only with dream recall but also in the recall of one's personal identity and situation within a dream. Thus a theoretical framework exists which seems to partially explain the effectiveness of substances that lead to increases of acetylcholine and the promotion of lucid dreams. These supplements have opened the world of lucid dreaming to a much wider audience than was ever possible using the traditional methods. Either way there is still room for significant improvement.

General Hypothesis and Description

Increased acetylcholine levels are a necessary, although insufficient condition of REM sleep, so any LDS theory limited to cholinergic stimulation is incomplete. Just as acetylcholine is viewed to be the primary REM promoting neurotransmitter, adenosine is seen as the primary deep sleep promoting neurotransmitter [McCarley, 2007]. Since these two systems seem to be controlled by different mechanisms it seems plausible that combining substances which promote REM sleep with those that

block the deeper sleep stages may produce favorable results in terms of inducing lucid dreams. Furthermore, such a combination might significantly increase the odds of directly entering REM sleep from the waking state, which would greatly increase the odds of successfully inducing a WILD. The monoamines serotonin, dopamine, and norepinephrine have been shown to have a significant affect on dreaming [Pagel & Helfter, 2003]. Hence, their potential role in the LDS system should be evaluated. Dopamine, in particular may play a role in supporting the dreamer's control over their dreamscape, possibly through increased motivation and confidence levels [Yuschak, 2006]. This study, although limited in scope, provides a preliminary glimpse at the interplay between REM promoting/enhancing substances and deep sleep suppressing substances as they relate to lucid dream induction.

The study involved 11 nights of EEG recording on a subject who consumed either a cholinergic stimulating substance, a deep sleep suppressing substance, or a combination of the two. The aim of the study is to show the efficiency and drawbacks of the components and combinations in terms of allowing one to move directly from the waking state into a light sleep / REM state as well as giving an overall indication of the effectiveness of the LDS method on lucid dream induction.

Supplement Profiles

This study primarily focuses on the effects of four substances as possible aids to lucid dreaming: Galantamine, Sulbutiamine, Caffeine, and Desmopressin. The study also includes Choline Bitartrate and L-Theanine as secondary, supporting supplements. It is worth noting that, with the exception of Desmopressin, all of these

substances are available without a prescription within the United States and other countries.

1. Galantamine

Galantamine is a natural substance that greatly increases the likelihood of experiencing a lucid dream [LaBerge, 2003; Yuschak 2006]. Galantamine has two primary modes of action: inhibition of acetylcholinesterase (the substance that breaks down acetylcholine) and modulation of the nicotinic cholinergic receptors to increase the release of acetylcholine [Woodruff-Pak, Vogel, & Wenk, 2001]. Galantamine has been shown to reduce REM latency and increase REM density [Riemann, Gann, Dressing, Muller, & Aldenhoff, 1994] as well as having a positive affect on memory [Koontz, Baskys, 2005]. The time required for galantamine to reach peak plasma concentrations is within one hour of ingestion.

One of the primary criteria in determining whether or not galantamine is effective at inducing a lucid dream is determined by the efficiency with which the subject enters into REM sleep after taking the dose. The odds are significantly increased if the subject enters the REM sleep phase directly from the waking state. If a typical sleep cycle proceeds however, in which the subject first passes through the deeper sleep stages, the odds of lucidity are reduced. Due to this characteristic, galantamine is usually taken after first sleeping for some period of time such that the body is already tending to naturally enter longer REM periods [Yuschak, 2006]. The normal sleep cycle favors the deeper sleep stages in the first part of the night but as morning approaches the deeper sleep stages become less dominating. Although this

trend increases the odds that galantamine will cause a person to directly enter REM sleep from the waking state, provided it is ingested after 4 or 5 hours of normal sleep, there is still a reasonable chance that one will enter the deeper sleep stages for some period of time before REM is initiated and this can lead to inconsistent results for lucid dreaming.

2. Sulbutiamine

Sulbutiamine is described as a Vitamin B analog that can significantly reduce the symptoms of asthenia: feelings of weakness that often accompanies illness [Shah et al, 2003]. Sulbutiamine has also been shown to significantly decrease the percentage of the deeper sleep stages in favor of stage 1 sleep, without having a significant impact on REM [Balzomo, & Vuillon-Cacciuttolo, 1982]. There are also reports that Sulbutiamine may have a positive impact on memory [Bizot, Herpin, Pothion, Pirot, Trovero, & Ollat, 2005]. Sulbutiamine is readily absorbed and the blood concentration peaks in 1-2 hrs after oral administration.

3. Caffeine

Caffeine acts as an adenosine antagonist and has a dramatic effect on the sleep cycle. In moderate doses caffeine can increase sleep latency and cause a reduction in total sleep time. However, low doses of caffeine can be used to significantly inhibit the deeper sleep stages, in favor of stage 1 sleep without increasing wakefulness or altering REM sleep [Yanik, Glaum, & Radulovacki, 1987]. The time required to reach peak plasma concentrations is within one hour of ingestion.

4. Desmopressin (DDAVP)

Desmopressin is a synthetic analog to the hormone arginine vasopressin. Although DDAVP is primarily used as a treatment for Nocturia (frequent need to urinate during the night), it has also been shown to have positive affects on short term memory [Gaffori & De Wied, 1986] and to significantly lighten sleep [Eggert, Fritz, Stecker, & Muller, 1996]. Although the exact mechanism of DDAVP is not fully understood there is evidence that it causes an increase in central dopamine turnover [Di Michel, Sillen, Engel, Hjalms, Rubenson, & Soderpalm, 1996]. Peak plasma concentrations of desmopressin are reached within 0.9 - 1.5 hours after ingestion.

5. Choline Bitartrate and L-Theanine

These two substances are used as supporting supplements and not a primary focus of this study. Choline Bitartrate is used to support galantamine by building up acetylcholine levels more rapidly. Choline acts as a precursor to acetylcholine and is very closely related to the vitamin B family. Choline is considered an essential nutrient by the US Food and Drug Administration (FDA). L-Theanine is used to help reduce sleep latency without having a significant impact on the various sleep stages. L-Theanine is an amino acid found in green tea and is commonly used as an anti-stress agent. L-Theanine blocks the binding of glutamic acid to glutamate receptors within the brain [Kimura, Ozeki, Juneja, & Ohira, 2007] which leads to a relaxed, calm feeling that is beneficial when returning to sleep after the more stimulating LDS substances are consumed.

Experimental Methods

Test Subject

A male, 40 years old, experienced in the techniques of lucid dreaming, was used for this analysis. The subject was well accustomed to using supplements, under controlled conditions, for the purpose of lucid dream induction. The subject has no reported illnesses (mental or physical) or history of illness. The subject does not smoke and abstained from drinking alcohol for a period of at least 24 hours prior to each experiment.

Test Procedure

On each experimental night, the subject slept naturally for approximately 3.5 to 4.5 hours before the testing period. The subject was awakened and then attached to a 4 channel EEG recorder using a referential montage. The electrodes were placed using the standard international 10-20 system to the F3, P3, F4, and P4 positions. The earlobes were used as both reference and ground. A specially prepared cap was used to maintain the electrodes position during sleep. Signal quality was confirmed prior to the start of the recording. The subject has undergone fairly regular sleep EEG experiments, so no special adaptation night was required specifically for this study.

The subject underwent eleven nights of EEG recording in the span of nineteen calendar days. On each night the subject consumed a combination of supplements immediately before the start of the EEG recording. Next he immediately laid down and lights were turned off. He rested with eyes closed while lying on his back (not his

preferred position but considered necessary to maintain a quality electrical signal). For all trials the subject was to try to induce a lucid dream. If successful, he was required to consciously exit the dream before lucidity started to wane. Immediately upon awakening, the subject recounted the experience; stating whether he experienced a DILD, WILD, or no lucid dream. If lucidity did occur, he estimated the length of the lucid period and ranked parameters such as vividness, control, ability to reason, etc.

Each of the nights fell into one of four categories: Baseline (BN), cholinergic stimulation (CS), deep sleep suppression (DSS), or a combination of cholinergic stimulation plus deep sleep suppression (LDS). The actual supplements taken on each of the nights is summarized below.

1. Baseline night (BN1): 200mg L-Theanine taken ~15 minutes prior to the start of the EEG recording.
2. Caffeine night (DSS-CN1): 200mg of L-Theanine taken ~ 15minutes prior to taking 50mg of caffeine. The caffeine was taken at the start of the EEG recording
3. Caffeine night (DSS-CN2): 200mg of L-Theanine taken ~ 15minutes prior to taking 25mg of caffeine. The caffeine was taken at the start of the EEG recording.
4. Sulbutiamine night (DSS-SN1): 200mg of L-Theanine taken ~ 15minutes prior to taking 200mg of sulbutiamine. The sulbutiamine was taken at the start of the EEG recording.

5. Desmopressin night (DSS-DN1): 10mcg of DDAVP (via nasal spray) was taken ~30minutes prior to taking 200mg of L-Theanine. After another 15 minutes another 10mcg of DDAVP was taken. The second dose of DDAVP was taken at the start of the EEG recording.
6. Cholinergic only night: (CS1): 200mg of L-Theanine taken ~ 15minutes prior to taking a combination of 8mg Galantamine and 500mg Choline Bitartrate. The cholinergic combination was taken at the start of the EEG recording.
7. LDS night 1 (LDS1): 200mg of L-Theanine taken ~ 15minutes prior to taking a combination of 8mg Galantamine, 500mg Choline Bitartrate, and 200mg of sulbutiamine. The LDS combination was taken at the start of the EEG recording.
8. LDS night 2 (LDS2): 10mcg of DDAVP (via nasal spray) was taken ~30 minutes prior to taking 200mg of L-Theanine. After another 15 minutes a combination of 8mg Galantamine, 500mg Choline Bitartrate, and a second dose of 10mcg of DDAVP was taken. The LDS combination was taken at the start of the EEG recording.
9. LDS night 3 (LDS-3): 200mg of L-Theanine taken ~ 15minutes prior to taking a combination of 8mg Galantamine, 500mg Choline Bitartrate, and 20mcg of DDAVP (via nasal spray). The LDS combination was taken at the start of the EEG recording.
10. LDS night 4 (LDS4): 200mg of L-Theanine taken ~ 15minutes prior to taking a combination of 8mg Galantamine, 500mg Choline Bitartrate, and 25mg of caffeine. The LDS combination was taken at the start of the EEG recording.

11. LDS night 5 (LDS-5): 10mcg of DDAVP (via nasal spray) was taken ~30 minutes prior to taking 200mg of L-Theanine. After another 15 minutes a combination of 8mg Galantamine, 500mg Choline Bitartrate, and a second dose of 10mcg of DDAVP was taken. The LDS combination was taken at the start of the EEG recording.

Data Analysis

The subject's EEG was recorded using a rate of 256 samples per second and a range of 0 - 48 Hz. The resulting data for each of the four EEG channels was averaged together and then filtered into 1 Hz bins that covered the range from 1 to 47hz. The 1 Hz bin included all frequencies from 0.5 Hz to 1.5 Hz, the 2 Hz bin included all frequencies from 1.5 Hz to 2.5 Hz, and so on. The final binned data was then plotted using 10 second epochs. A special delta bin was also created for this paper, by averaging the 1-3 Hz bins together. The plots included in this paper include only the delta bin and the 10Hz frequency bin. The 10Hz bin adequately captures the subjects dominate alpha frequency and can be used in correlation with the delta bin to distinguish between the sleep state and the waking state. Furthermore, the relative magnitude of signal strength between the two bins can be used to distinguish the deeper sleep stages (3 and 4) from the lighter stages (1, 2, and REM). All plots show the relative normalized signal strength as function of time.

Test Results

The subject reported experiencing a lucid dream on 5 of the 11 experimental nights. Lucid dreaming was reported on all five LDS nights (cholinergic stimulation plus deep sleep suppression). Non lucid dreaming was reported each of the remaining nights. A brief description of results is included below and an overall summary of each night's results is included in table 1.

BN1 is the baseline night and includes only L-Theanine as the supplement taken. Note that the EEG shows a standard sleep cycle in which the subject transitioned from the waking state into progressively deeper sleep as indicated by the relative signal strength between the delta and dominate alpha bins. The subject reached the deepest sleep approximately 50 minutes after the start of the recording and then gradually transitioned to light sleep followed by REM sleep. After awakening, the subject reported experiencing non-lucid dreams. See figure 1 for details.

A total of one cholinergic stimulation *only* night was included in this study (CS1). A cholinergic stimulating combination of galantamine plus choline Bitartrate was used. Although the subject has reported using this combination to successfully induce lucid dreams on many occasions in the past, he failed to experience a lucid dream on this particular night. In contrast to previous successful attempts in which the subject moved directly from the waking state into the REM sleep state (not included in this paper), this time he experienced a relatively typical sleep cycle that closely resembled that of BN1. This mimics the results of a previous study by the author (unpublished) which concluded that galantamine is most effective at inducing lucid dreams if one can directly enter REM sleep from the waking state (see figure 1).

Each of the DSS nights showed that the deeper sleep stages were significantly suppressed when compared to the baseline night (BN1) (see figure 2). Sulbutiamine seemed to show the greatest efficiency at reducing deep sleep, followed by caffeine, and then DDAVP. Two caffeine nights were included. The dose on the first night (50mg), although very effective at suppressing deep sleep, led to frequent awakenings during the EEG recording. The dose was reduced to 25mg for the second night which remained effective and did not disrupt sleep. Although deep sleep was reduced on all DSS nights, the subject was not successful at inducing a lucid dream. This emphasizes the role of acetylcholine in lucid dreaming.

Each of five LDS nights resulted in a lucid dream. Furthermore, all five lucid dreams were described as WILDs and the corresponding EEGs showed that the deep sleep stages were avoided (see figure 3). The EEG recordings suggest that the transition into the dream occurred during stage1 sleep instead of during REM but then continued into REM sleep. On each of the five nights, the subject reported experiencing an intense sense of motion as he transitioned from the waking state into the lucid dream state. The motion was described as either a linear acceleration or rotational in nature. The subject reported the strongest of these feelings occurred during LDS1 (sulbutiamine). He also reported enhanced dream control on all three of the nights that included DDAVP (LDS2, LDS3, and LDS5) and these nights also tended to produce the longest lucid dreams (see table 1). The night that included caffeine (LDS4) was reported to be the most bizarre of the lucid dreams and included a high level of lucidity with a reported unstable dream environment.

Discussion

These results lend support to the hypothesis that galantamine is most effective at supporting lucid dreaming if one can avoid entering the deeper sleep stages immediately after consuming it. Furthermore, supplements that suppress the deeper sleep stages can increase the effectiveness of galantamine even if those supplements do not necessarily promote lucidity when used alone. Table 1 contains a summary of all the testing done during this study. Note that this was a fairly aggressive testing schedule and although the subject regularly sleep for 3.5-4.5 hours prior to each experiment, deep sleep was repeatedly reduced in the second half of the night. Even with this less than ideal sleep schedule the subject reported a successful WILD on each night that galantamine was combined with a deep sleep suppressing substance; this lead to 5 lucid dream nights in a span of 10 calendar days. Furthermore when DDAVP was combined with galantamine, the subject experienced long duration lucid dreams and reported enhanced control of the dreamscape in 3 out of 3 attempts. This may be due to the enhanced utilization of available dopamine that DDAVP is thought to produce.

From these results, one conclusion is that an ideal approach of using supplements to induce lucid dreaming is to combine an REM promoting substance with a deep sleep suppressing substance. The inclusion of deep sleep suppressing substances may allow other cholinergic stimulating substances like alpha-GPC, Huperzine A, DMAE, CDP-choline, and Centrophenoxine to become powerful lucid dream aids which will be a valuable outcome for those who live in countries where galantamine is not widely available. Furthermore, the role of dopamine seems to have

a profound impact one's ability to manipulate a dream, and therefore may have powerful therapeutic applications. Finally, the effectiveness of the LDS approach to lucid dreaming promises to make the ability available to a wide range of people from different backgrounds and warrants serious study from researchers.

Pharmacological Induction of Lucid Dreams

Table:1 Summary of Results

Study Summary									
Calendar Day	Title	Cholinergic Stimulating Substances	Deep Sleep Suppressing Substances	Type of lucid dream	Lucid Dream Duration	Level of Control reported	Level of Vividness reported	Level of Reasoning abilities reported	Notes:
1	CS1	500mg Choline Bitartrate 8mg Galantamine	None	None	N/A	N/A	N/A	N/A	Failure likely due to not directly entering REM from the awake state
2	Off night								
3	DSS-SN1	None	200mg Sulbutiamine	None	N/A	N/A	N/A	N/A	EEG shows significant suppression of deeper sleep stages
4	Off night								
5	Off night								
6	LDS1	500mg Choline Bitartrate 8mg Galantamine	200mg Sulbutiamine	WILD	~20 minutes	Good	Very Good	Very Good	Took a long time to fall to sleep. Should consider lowering dose of Sulbutiamine
7	Off night								
8	Off night								
9	LDS2	500mg Choline Bitartrate 8mg Galantamine	10mcg DDAVP 45minutes prior to other supplements 10mcg DDAVP with other supplements	WILD	90 minutes	Excellent	Very Good	Very Good	High level of control possibly a result of enhanced dopamine utilization
10	DSS-CN1	None	50mg Caffeine	None	N/A	N/A	N/A	N/A	EEG shows significant suppression of deeper sleep stages however subject woke up several times during the recording
11	LDS3	500mg Choline Bitartrate 8mg Galantamine	20mcg of DDAVP taken with other supplements	WILD	25 minutes	Excellent	Very Good	Good	Although the subject reported losing lucidity after about 25 minutes he continued with non-lucid dreaming for another 40 minutes before waking up
12	DSS-CN2	None	25mg caffeine	None	N/A	N/A	N/A	N/A	The smaller dose of caffeine suppressed the deeper sleep stages but did not disrupt sleep.
13	Off night								
14	LDS4	500mg Choline Bitartrate 8mg Galantamine	25mg caffeine	WILD	30 minutes	Very Good	Good	Very Good	Subject reported that this dream was the most bizarre with the dreamscape going through constant transformations
15	LDS5	500mg Choline Bitartrate 8mg Galantamine	10mcg DDAVP 45minutes prior to other supplements 10mcg DDAVP with other supplements	WILD	50 minutes	Excellent	Excellent	Very Good	Dream included a brief and bizarre false awakening at the very end.
16	Off night								
17	BN1	None	None	None	N/A	N/A	N/A	N/A	Used to for comparison purposes
18	Off night								
19	DSS-DN1	None	Two doses of 10mcg DDAVP (first dose 45 minutes prior to second)	None	N/A	N/A	N/A	N/A	Effectively suppressed deep sleep, especially in the first part of the sleep cycle

Pharmacological Induction of Lucid Dreams

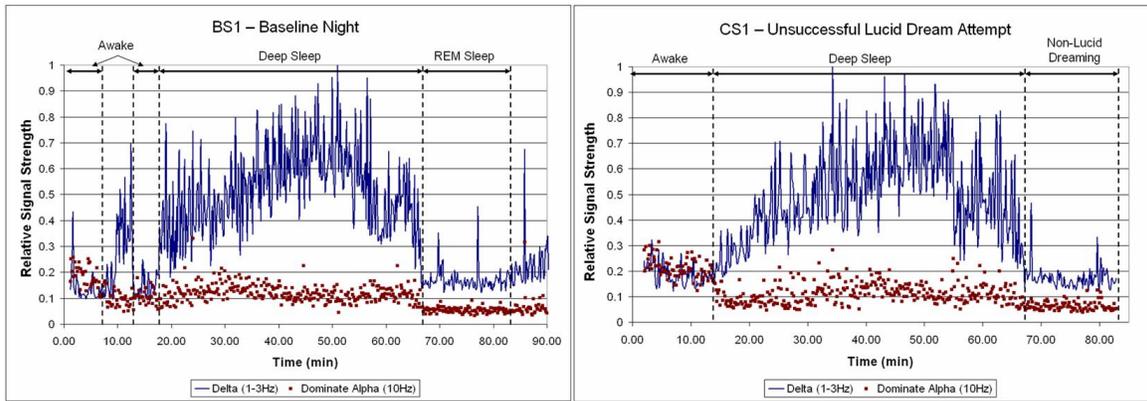


Figure 1: Baseline night (BN1) and Cholinergic only night (CS1) failed to produce lucid dreams, possibly due to the tendency to enter the deeper sleep stages directly from the waking state.

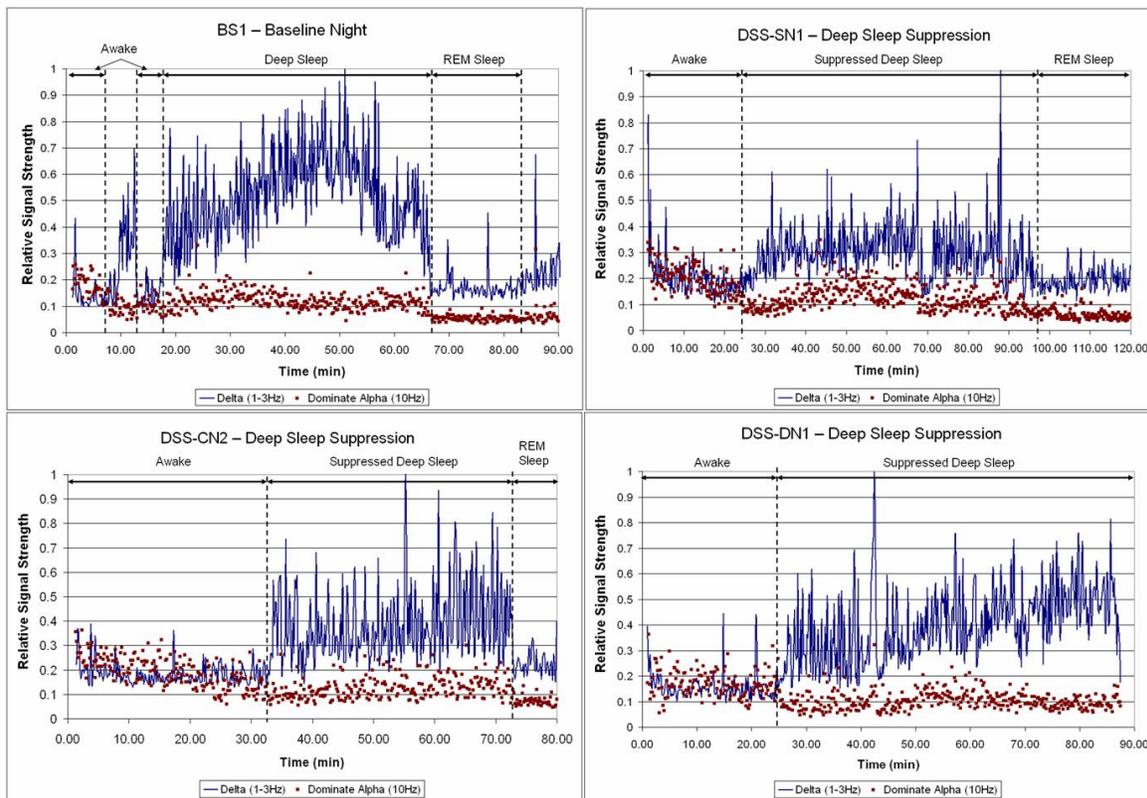


Figure 2: Each of the DSS nights showed a strong tendency to suppress the deeper sleep stages compared to the baseline night (BN1) as shown by relative power between the delta band and dominate alpha band.

Pharmacological Induction of Lucid Dreams

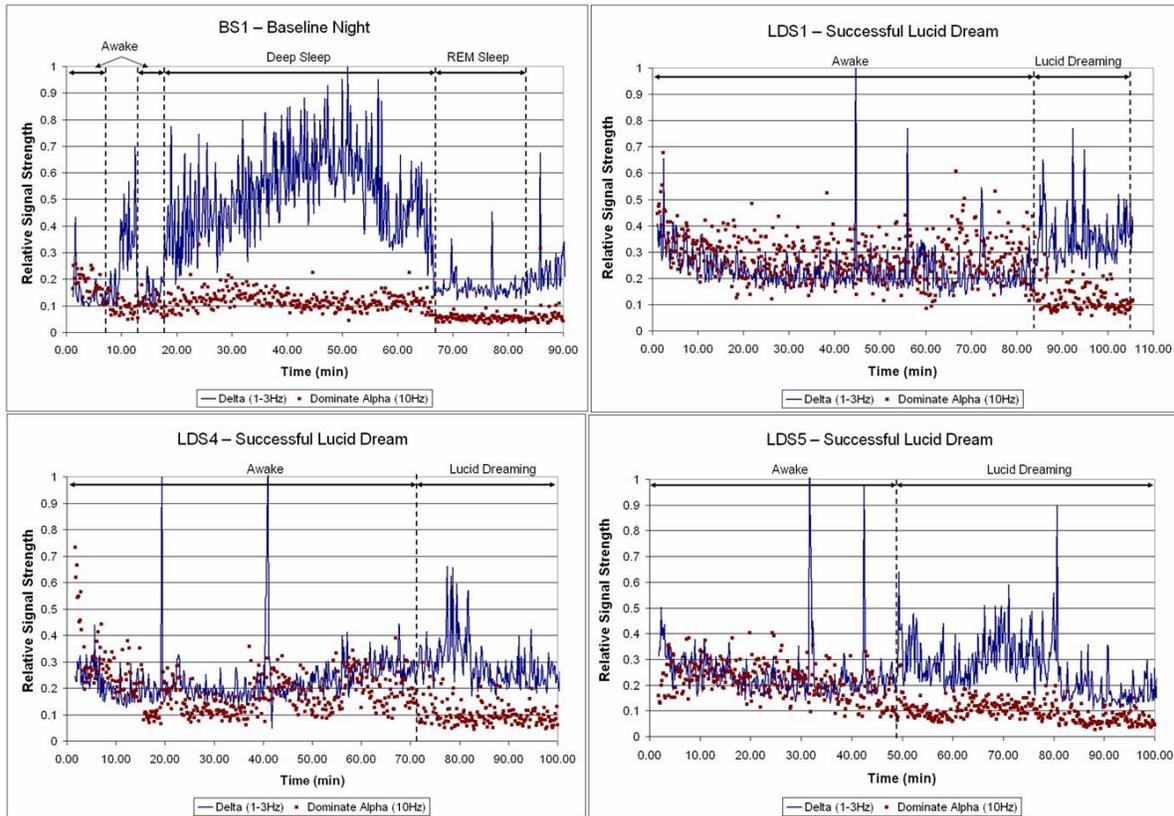


Figure 3: All five of the LDS nights (three shown above) produced Wake Initiated Lucid Dreams (WILDs). It is hypothesized that the success of this approach is due to the combination of a cholinergic stimulating substance with a deep sleep suppressing substance which allowed the subject to move from the waking state directly into a very light sleep with increased acetylcholine levels.

References:

- Balzomo, E., Vuillon-Cacciuto, G., (1982). Enhancement of the waking state by Sulbutiamine (Arcalion) during subchronic treatment in *Macaca mulatta*. *Revue d'Electroencéphalographie et de Neurophysiologie Clinique*, 12 (4), 373-378
- Bizot, J., Herpin, A., Pothion, S., Pirot, S., Trovero, F., & Ollat, H., (2005). Chronic treatment with sulbutiamine improves memory in an object recognition task and reduces some amnesic effects of dizocilpine in a spatial delayed-non-match-to-sample task. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 29 (6), 928-935

- Carrier, J., Fernandez-Bolanos, M., Robillard, R., Dumont, M., Paquet, J., Selmaoui, B., & Filipini, D. (2007). Effects of caffeine are more marked on daytime recovery sleep than on nocturnal sleep. *Neuropsychopharmacology*, 32(4), 964-972
- Di Michel, S., Sillen, U., Engel, J., Hjalmus, K., Rubenson, A., & Soderpalm, B. (1996). Desmopressin and Vasopressin Increase Locomotor Activity in the Rat Via a Central Mechanism: Implications for Nocturnal Enuresis. *The Journal of Urology*, 156 (3), 1164-1168
- Eggert, P., Fritz, A., Stecker, B., & Muller, D. (2004). Desmopressin has an influence on the arousability of children with primary nocturnal enuresis. *The Journal of Urology*, 156 (3), 1164-1168
- Gaffori, O., & De Wied, D. (1986). Time-related memory effects of vasopressin analogues in rats. *Pharmacology Biochemistry and Behavior*, 25 (6), 1125-1129
- Gron, G., Kirstein, M., Thielscher, A., Riepe, M., & Spitzer, M. (2005). Cholinergic enhancement of episodic memory in healthy young adults. *Psychopharmacology*, 182(1), 170-179
- Kimura, K., Ozeki, M., Juneja, L., & Ohira, H. (2007). L-theanine reduces psychological and physiological stress responses. *Biological Psychology*, 74 (1), 39-45
- Koontz, J., Baskys, A., (2005). Effects of galantamine on working memory and global functioning in patients with mild cognitive impairment: A double-blind placebo-controlled study. *American Journal of Alzheimer's Disease and Other Dementias*, 20 (5), 295-302

- Holzinger, B., LaBerge, S., & Levitan, L. (2006). Psychophysiological correlates of lucid dreaming, *Dreaming*, 16 (2), 88-95
- LaBerge, S., & Rheingold, H. (1990). *Exploring the world of lucid dreaming*. The Ballantine Publishing Group
- LaBerge, S., (1993). Lucidity research, past and future, *Nightlight*, 5(3)
- LaBerge, S. & DeGracia, D.J. (2000). Varieties of lucid dreaming experience. In R.G. Kunzendorf & B. Wallace (Eds.), *Individual Differences in Conscious Experience* (pp. 269-307).
- LaBerge S. (2003). Substances that enhance recall and lucidity during dreaming, *United States Patent Application 604138*
- McCareley, R. (2007). Neurobiology of REM and NREM sleep. *Sleep Medicine*, 8, 302-330
- Pagel, J. F., & Helfter, P. (2003). Drug induced nightmares – an etiology based review, *Human Psychopharmacology*, 18, 59-67
- Riemann, D., Gann, H., Dressing, H., Muller, WE., Aldenhoff, JB. (1994). Influence of the cholinesterase inhibitor galanthamine hydrobromide on normal sleep. *Psychiatry Research*, 51 (3), 253-267
- Sergio, W. (1988). Use of DMAE (2-Dimethylaminoethanol) in the Induction of Lucid Dreams. *Medical Hypotheses*, 26, 255-257
- Shah SN., Sulbutiamine Study Group, (2003). Adjuvant role of vitamin B analogue (sulbutiamine) with anti-infective treatment in infection associated asthenia. *J Assoc Physicians India* 51: 891–895

Stride, S. (2006). Foreword. *Advanced Lucid Dreaming - The Power of Supplements*, Lulu Enterprises, 1-4

Woodruff-Pak, D., Vogel, R., & Wenk, G. (2001), Galantamine: Effect on nicotinic receptor binding, acetylcholinesterase inhibition, and learning. *Proc Natl Acad Sci*, 98(4), 2089-94.

Yanik, G., Glaum, S., & Radulovacki, M. (1987). The dose-response effects of caffeine on sleep in rats, *Brain Research*, 403, 177-180

Yuschak T. (2006). *Advanced Lucid Dreaming - The Power of Supplements*. Lulu Enterprises