Enhancing Melatonin Secretion: The Methodical Consumption of Tryptophan from Whole Cow's Milk to Regulate Sleep Quality in Individuals Aged 18-30 with Delayed Sleep-Wake Phase Disorder

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Abstract: Young adults in the United States are increasingly affected by delayed sleep-wake phase disorder (DSPD), a prevalent circadian rhythm disorder that delays evening sleep and morning wake times relative to the solar cycle. Although medicinal supplements have shown to produce effective results on immediate sleep induction, they lack the ability to aid in regulation and maintenance of routine sleep schedules. Alternatively, a dietary method may be able to adjust the deficits of supplements. A review of the literature on clinical nutrition and endocrinology suggests that dietary alterations through the timed consumption of tryptophan-abundant whole cow's milk may be an auxiliary option of improving sleep quality and morning alertness in individuals with DSPD. Studies on chrono-nutrition indicate that dietary components absorbed by the bloodstream can alter the circadian schedule of melatonin secretion from the pineal gland, and the timed consumption of tryptophan-abundant foods, such as whole cow's milk, can consequently spike melatonin levels before a DSPD patient's desired sleep time and promote circadian rhythm advancement. Based on the stated studies, this research proposes the Melatonin Intake through Lactalbumin (a-lac) Consumption (MILC) treatment, or the consistent, timed consumption of milk. The MILC treatment may decrease a DSPD patient's morning sleepiness on the basis that disordered, high-stressed, and sleep-deprived individuals are susceptible to minimal changes in hormones because their bodies naturally attempt to attain homeostatic equilibrium. The correlation between chrono-nutrition and dietary effectiveness is a novel idea, and testing is needed to quantify the optimal timings and ranges of dietary tryptophan that can produce a significant effect on the sleep quality of a DSPD patient.

Keywords: delayed sleep-wake phase disorder, chrono-nutrition, alpha-lactalbumin

1. Introduction

Delayed sleep-wake phase disorder (DSPD) is a circadian rhythm disorder in which an individual lacks the ability to fall asleep within a socially acceptable time. The symptoms of DSPD patients are variable based on their individual routines and biological predispositions for sleep disorders. Some display signs of insomnia while others struggle with sleep latency, or the time that it takes an individual to actively initiate sleep at night. On the other hand, some patients lack total sleep efficiency timing, or the total percentage of time that individuals spend sleeping in bed. Others may have delayed sleep patterns because of unavoidable circumstances such as night shifts and demanding responsibilities. Among all categories of patients and their symptoms, researchers have found that the greatest overarching problems are morning alertness, drowsiness, inability to focus, and decreased retention (Berendsen et al., 2020).

This sleep disorder can be traced back to the sleep-inducing hormone, melatonin; its secretion from the pineal gland is a determinant of the consistency and extent of circadian offset in sleep-deprived individuals. Those with delayed circadian schedules encounter fluctuations in hormones, and, as a result, the pineal gland's secretion of melatonin is unable to adjust to a regular, timely pattern. The dim light melatonin onset (DLMO), or the timing of melatonin secretion patterns from the pineal gland, shifts in accordance with an individual's average delayed sleep schedule; at minimum, the DLMO shift is 2 hours after the socially accepted sleep onset time (Micic et al., 2007). This shift determines the extent of correlation between DLMO and sleep timing, and with the large delay for DSPD patients, adjustment is necessary through external or exogenous factors such as light exposure or overall dietary intake.

Melatonin is only released by the pineal gland at night, but it can also be synthesized from the dietary intake of the essential amino acid, tryptophan. Tryptophan conversion can increase melatonin concentrations and induce sleep through the tryptophan–serotonin–melatonin pathway (Richard et al., 2009). Consumption of naturally high-tryptophan foods or drinks may advance DLMO timings and increase melatonin secretion levels up to 6 hours after intake. If timed accurately, the high-tryptophan food can be used to enhance sleep quality in DSPD patients by accelerating melatonin secretion before the pineal gland starts its delayed release of melatonin. Through this method, the increased melatonin levels can induce deeper sleep and enhance sleep quality in terms of morning alertness.

One specific sleep inducing, high-tryptophan drink is whole cow's milk; it has the highest tryptophan content of all milk types due to its alpha-lactalbumin protein levels (Markus et al., 2000). Cow's milk also contains nutrients, such as calcium and potassium, that work through alternative pathways to aid in sleep-induction. Although plant-based and other mammalian-milk types have other defining properties and nutritional contents, cow's milk is largest in terms of tryptophan; it is also one of the highest produced milk types and is widely available to the public (Yamaguchi et al., 2014). The newly proposed Melatonin Intake through Lactalbumin (a-lac) Consumption (MILC) treatment consists of DSPD patients intaking 1 cup of whole cow's milk 1-2 hours before their desired sleep time, which may improve their overall sleep health and morning functionality, even if their circadian schedule does not shift times.

2. Delayed Sleep-Wake Phase Disorder and Phase Shifting

Because young, DSPD patients have specific difficulties in regulating their sleep and circadian rhythms to the socially acceptable times, research suggests that the best way to improve their sleep quality would be to manipulate endogenous factors by altering melatonin levels through the intake of tryptophan.

Chang et al. (2009) claimed that individuals with delayed sleep wake phase disorder have an offset sleep episode later than desired, and this can lead to difficulty in morning functionality and struggles in awakening. They added that individuals have a specialized circadian schedule based on their health, age, and lifestyle, and a delay or shift in sleep schedules based on solar cycles may lead to insufficient and non-restorative sleep when it comes to duration and quality.

Those with DSPD struggle in terms of long-term physical and mental functionality rather than overall health, but the symptoms can differ between geographical time zones, as countries have different socially acceptable sleep timings. These differences create a wide range of sleep habits across the globe as determined by Krueger & Friedman (2009), who reasoned that individuals in the United States have an average sleep initiation time of 10 PM–12 AM and awaken at 7:00 AM. On the other hand, DSPD patients are generally unable to fall asleep before 2 AM, and if their schedules permit, they don't wake up until 11:00 AM–1:00 PM. This wide distribution of sleep times can further be attributed to individual schedules and variable intensity of the disorder.

With age being crucial to the body's ability to adjust to stimuli, researchers have performed studies to determine the prevalence of DSPD in the young adult age range. Berendsen et al. (2020) studied patients with DSPD aged 13-20 due to a 7-16% prevalence of circadian disorders within that age range. Micic et al. (2007) added to the validity of that finding in their study about melatonin profiles in DSPD patients. They determined that young adults between the ages of 20-30 have the lowest melatonin secretion of all other age ranges; researchers interpreted that DSPD patients fall within that age range due to the delayed hormonal secretory rates and high stress levels. Both studies concluded that DSPD patients who need the greatest amount of aid in resetting their circadian rhythm are young adults, and the age controls can be used to quantify the "normal" amount of melatonin secretion and sleep that an average individual should maintain.

Akerstedt & Gilberg (1986) measured the subjects' daytime sleepiness through the EEG (electroencephalogram) power density analysis, a test that records brain activity and electrical signals through small sensors in the scalp. The researchers found that individuals who were restricted to fewer hours of night sleep were increasingly prone to daytime sleepiness, and they required greater

recovery time to return to a "normal" level of alertness. Although this test was conducted on healthy subjects, the results can be applied to DSPD patients who have increasingly restricted sleep schedules because of their consistent non-restorative sleep; thus, they tend to "crash" whenever they get the chance. This crash is an effort to recover sleep loss as described by Akerstedt & Gilberg (1986). Those with sleep loss need to recover minimum power density, or adequate frequencies and intensities of sleep waves, to restore sufficient energy from prior fatigue. Exhaustion from an incomplete sleep cycle can be carried over in individuals, and this can reduce attention in the morning or cause sleep crashing on off days. This extended sleep crash in healthy individuals is a homeostatic attempt to recover a percentage of the prior loss. DSPD patients generally have compounding sleep deficits and the effects from time constraints can prevent normal homeostatic sleep recovery. Whenever patients "give in" to their homeostatic sleep struggles, then prior loss can be partially recovered through an excessively long sleep, usually during the weekends.

Chang et al. (2009) found that DSPD patients were going to bed nearly 29 minutes later on weekends and waking up 50 minutes later as compared to a regular weekday ($p < 0.05$). DSPD patients and healthy individuals had similar sleep efficiency and latency when placed under unstressed conditions. This finding allowed them to shift the focus of their study to sleep quality, melatonin rhythms, and general alertness because these aspects are highly variable in patients with sleep disorders. All three factors can be shifted through exogenous changes such as melatonin supplements, solar light cues, or bright light exposure. This modification can initiate the endogenous melatonin secreted from the pineal gland to realign and form a stable temporal relationship to obtain an optimal sleep-wake rhythm.

Although an exogenous shift in circadian phase can occur through light therapy and photic stimuli, researchers are exploring dietary methods as an alternative. Richard et al. (2009) discussed that the hormone melatonin correlates with sleep based on its concentration, production, and secretion from the pineal gland during the dark phase of the solar cycle. An additional mechanism of melatonin production is through its synthetization from its essential amino acid precursor, tryptophan. Manipulating tryptophan levels could be used as a dietary alternative to supplements to increase melatonin levels and the induction of phase shifts to improve sleep quality. Micic et al. (2015) emphasized that those with sleep disorders can utilize exogenous factors such as the tryptophan content in food to instigate melatonin production.

The melatonin secretion of young adults can become offset due to social times dependent on their age range, and the continuous shifting of circadian phases can lead to the formation of delayed sleep-wake phase disorder. Research suggests that manipulation of exogenous and endogenous stimuli can shift sleep quality and establish a normal temporal rhythm, and the manipulation of melatonin through dietary means is crucial to understanding how to manage DSPD symptoms.

3. The Effects of Exogenous Factors on the Melatonin Synthesis Pathway

Because tryptophan is the sole precursor of melatonin, an increase in tryptophan intake may alter the overall melatonin profiles and concentrations to boost mood and potentially increase sleep quality through phase shifting.

As previously mentioned, Micic et al. (2015) studied the nocturnal melatonin profiles in patients with DSPD and healthy sleepers to determine how offset circadian timing can contribute to the development of delayed sleep-wake phase disorder. They determined that there was a 2–6-hour delay in the circadian rhythms of the DSPD group as compared to healthy sleepers. However, the greatest determinant in sleep health between both types of sleepers was the initial burst of melatonin in the early part of the night and the melatonin production fluctuations throughout the night.

Shibui et al. (1999) established a positive correlation between sleep phase markers and melatonin phase markers. Whenever melatonin levels increase during the night, the individual enters a deeper state of sleep, and the lower the melatonin levels, the closer an individual is to an alert state. An average individual has peaks in melatonin secretion from the pineal gland between 2 AM and 4 AM, gradually decreasing afterwards. However, a DSPD individual has melatonin peaks during morning sunrise hours, and if they have a morning time constraint to awaken for, their sleep cycle won't

be completed. Not only does this lead to excessive lethargy and decreased alertness from the high melatonin concentrations in the morning, but it can also add to the non-restorative sleep percentage. This cycle can be combated by advancing the melatonin onset through exogenous factors that can increase melatonin production at a desired time.

Melatonin can be obtained through the intake and conversion of tryptophan, an essential amino acid that can *only* be obtained from food. Richard et al. (2009) stated that the recommended daily intake of tryptophan is 250-425 milligrams per day, and foods high in tryptophan are beneficial for sleep regulation because they can adjust melatonin levels and diurnal rhythms through conversion. Once tryptophan enters the body through food intake, it can enter numerous pathways to create necessary building blocks and molecules in the body. One specific path, called the melatonin synthesis pathway, drives tryptophan to be converted into melatonin through a series of chemical reactions.

To determine the potential of diet-induced tryptophan as a treatment method for DSPD patients, researchers have attempted to use the amino acid to raise the serotonin and melatonin levels in the body, yielding positive changes in sleep quality through relaxation and sharpened morning alertness.

4. Correlation Between the Trp:LNAA Ratio and the a-lac Content of Whole Cow's Milk

Because tryptophan (Trp) is an essential amino acid, it must be derived from food and must compete with other large neutral amino acids (LNAA) to get accepted into a transporter on the blood brain barrier (BBB), a highly selective semipermeable membrane that controls what molecules enter the brain from the bloodstream. Once a molecule passes through the transporter, it will have a greater chance of undergoing catabolism and entering its various synthesis pathways. This highly competitive transportation process between tryptophan and other amino acids in the body determines what molecule will have the greatest influence in the brain. If tryptophan has a greater concentration than other amino acids inside the BBB, then it is classified as having a high Trp:LNAA ratio. This ratio raises the chances of tryptophan being synthesized into melatonin, increasing the efficacy of sleep regulation.

Regarding the synthesis of melatonin in the brain, Markus et al. (2000) established that proteins and carbohydrates are both macromolecules that can release varying quantities of tryptophan, thus altering the ratio between amino acids. The concentrations of these amino acids can determine the level of brain serotonin, stress management, depression, and mood. To understand the optimal efficiency of raising tryptophan levels through serotonin synthesis, Richard et al. (2009) studied the interaction of amino acids in the blood brain barrier. Once ingested, nearly 95% of the tryptophan that enters the circulatory system is bound to albumin while the rest remains unbound. Unbound tryptophan has a high chance of entering the BBB, but the bound tryptophan also gets pulled towards the BBB transporter; therefore, it must compete with neutral amino acids in order to be accepted by the transporter. All competing amino acids take part in competitive inhibition to be transported across the barrier and contribute to the Trp:LNAA ratio gradient; the more tryptophan available to compete, the greater the chance that it will enter the BBB and continue its path towards serotonin synthesis.

Addressing tryptophan levels, Richard et al. (2009) emphasized that one of the best ways to increase tryptophan concentration in the brain can be through the increased consumption of carbohydrates and decreased consumption of proteins. These macromolecules do not immediately change the Trp:LNAA ratio, but rather contribute to the levels of amino acids circulating in the bloodstream that are available to compete. Specifically, carbohydrates decrease the LNAA concentrations and increase the tryptophan levels, while proteins increase the LNAA concentrations and decrease the tryptophan levels. Proteins can deplete plasma tryptophan concentrations and decrease the Trp:LNAA ratio through increased peptide chain formation from protein synthesis, and this produces a net increase in the LNAA concentrations. A balance of both macromolecules needs to be achieved for obtaining tryptophan (proteins) and converting tryptophan (carbohydrates) into serotonin.

Markus et al. (2000) narrowed down one notable exception to the CR-PP rule: alpha-lactalbumin (a-lac). A-lac is a whey protein that has qualities that are conducive to serotonin synthesis, and its tryptophan content could be able to overcome

competitive transporter blocks, making it a key component to enhance sleep quality; this protein has the highest tryptophan levels of all bovine proteins at around 4.8g/100g, and the researchers tested its success at raising the Trp:LNAA ratio despite it being categorized as a protein with high quantities of competing amino acids. Yamaguchi and Takai (2014) determined that a-lac constitutes nearly 3.5% of the total proteins in bovine milk, and one cup of whole cow's milk (237 mL) contains 284 milligrams of alpha-lactalbumin and 100 milligrams of tryptophan. These quantities are relatively high, considering that one cup of milk nearly contains half of the daily recommended amount of tryptophan.

Zeng et al. (2014) found that milk intake can stimulate serotonergic activity and melatonin synthesis to induce enhanced sleep. Whole cow's milk is enriched with omega-3, polyunsaturated fats, calcium, and potassium; these molecules can affect sleep based on the pathways that they enter, and the products they form in the bloodstream. High levels of omega-3 correlate with increased conversion of serotonin into melatonin. Similarly, polyunsaturated fatty acids are positively associated with increased sleep efficiency and REM deep sleep. Calcium and potassium have the ability to promote sleep because of their role in the modulation of voltage-dependent channels that generate slow waves and sleep spindles. Slow waves occur in non-rapid eye movement (NREM), stage 3 sleep, also known as deep, delta sleep, in which individuals are difficult to awaken. The deeper the sleep, the more substantial the restorative process in DSPD patients. In context to the overarching problem, milk has the components available to work on different aspects of sleep; when consumed by a DSPD patient with sleep deprivation and excessive lethargy, the milk can enter pathways and synthesize hormones that can regulate a healthier circadian rhythm.

Markus et al. (2000) measured the extent to which mood, digestion, and stress levels were affected by a-lac intake in vulnerable individuals. When testing subjects with various intensities of stress tolerances, the low-stress control group received casein protein, a protein high in large neutral amino acids, while the high-stress experimental group received a-lac whey protein. Markus et al. (2000) gathered university students aged 17-34 as their optimal test subjects because of their high vulnerability to stress, while the control group consisted of low-stress students with a mean age of 20.9. The highest prevalence of DSPD patients also fall within this age range, and many of them are considered high stress with regards to both circumstance and sleep health. Results from the researchers' study indicated that the high-stressed, high-cortisol test subjects were exceptionally prone to the effects of alpha-lactalbumin in raising their plasma Trp:LNAA ratio; there was a statistically significant 48% increase in the ratio for those who were on the a-lac diet as compared to the casein diet. The experimental a-lac diet also boosted mood and reduced cortisol response in high stress (HS) individuals as compared to the low stress (LS) individuals when exposed to stressors. Before conducting the experiment, the HS group had greater serotonin breakdown, creating a rise in cortisol levels as a biological response to stress, and this catabolism led to depression. However, when raising tryptophan availability in the brain using a-lac, the HS group experienced a decrease in depressive moods because of greater serotonin synthesis.

The studies mentioned above include different parameters and testing conditions, yet all reached the same conclusion that alpha-lactalbumin consumption yields the highest Trp:LNAA ratio, thus increasing sleep quality through melatonin production.

5. Changes in Sleep Quality Through Chrono-nutrition

Because research suggests that the effectiveness of dietary components can be associated with intake time, then milk consumed within 2 hours of a DSPD patient's desired sleep time could yield a net improvement in sleep quality and morning functionality on the following day because of the patient's susceptibility to changes in bodily hormones. Richard et al. (2009) briefly introduced the idea that tryptophan availability and serotonin synthesis efficiency can be impacted through the timing of consumption. The macromolecules in foods can break down and enter different synthesis pathways depending on their homeostatic necessity at that specific time. Berendsen et al. (2020) researched the phenomenon of timed dietary intake, or chrono-nutrition, and diet quality in adolescents with DSPD to measure the conjoined effect of diet and timed consumption to create a circadian balance. They

experimented on relatively healthy DSPD patients aged 13 to 20 years due to a 7-16% prevalence of DSPD within the age range. Individuals who have decreased sleep duration tend to have lower leptin levels and increased ghrelin levels, meaning that there is decreased satiation and increased hunger; researchers noted that this was associated with adverse health effects and a high BMI. Zeng et al. (2014) added that sleep restrictions can overstimulate the hypothalamus, the brain region that is sensitive to food stimuli, to decrease leptin circulation and increase ghrelin concentrations. Zellner et al. (2006) asserted that increased cortisol and ghrelin levels cause individuals to stray from healthy, low-fat foods to high-fat foods; the fat content tends to make individuals feel emotionally secure. In application to DSPD patients with abnormal circadian schedules and decreased sleep durations, unhealthy foods tend to stimulate their hunger hormones, making them susceptible to having a high BMI. In DSPD patients with high cortisol levels, their hypothalamus will increase ghrelin secretion to address poor sleep health with increased food intake. Increasing cortisol levels is a sympathetic response that causes a domino effect on decreasing digestive powers and increasing ghrelin. Since the digestive juices are not functioning at potential due to high cortisol, the food intake will have a high chance of being stored directly as fat. All these researchers similarly concluded that disruptive sleep schedules can increase ghrelin levels and contribute to adverse health effects.

Regarding the previously mentioned idea of chrono-nutrition, Markus et al. (2005) found that the consumption of a-lac in the evening can be timed properly to peak at specific times and induce sleep. They determined that the Trp:LNAA levels reached an apex at 3 hours after the initial consumption of a-lac, and the effects settled down to a base rate 5 hours after intake; within that interval, the DSPD patients reported better sleep quality due to a deeper, REM sleep stage, and they had greater morning alertness on the following day. As stated before, supplements can have excess side effects if they are not taken at an exact time, but the same cannot necessarily be stated with milk. Milk has quantities of a-lac and tryptophan that are high in comparison to other foods, but not enough to create nausea or other side effects when taken at the wrong time. Yet, DSPD patients should aim to drink the milk approximately 2 hours before they plan on going to sleep because their melatonin will begin to surge during that time, allowing them to have a shorter sleep latency and maximum sleep efficiency (Markus, 2005). It is indeterminate whether milk is substantial enough to phase shift the circadian rhythm to a desired time, but it can allow the body to adjust accordingly.

Some individuals cannot shift their schedule to a desired time because of life constraints; therefore, this method can primarily be used to improve the sleep that they receive rather than advance their circadian schedules to an alternative time. Chrono-nutritional differences in young adults with DSPD can alter how the molecules from foods can be synthesized. In the evening, the nutrients and macromolecules obtained from milk can be utilized towards synthesizing melatonin as a preparation for sleep, working in conjunction with the nightly activated pineal gland.

6. Subjective Testing and Experimental Proposal for the MILC Treatment

Because there is subjectivity in testing for sleep quality, multiple quantifiable tests, individual ratings, and sleep EEGs are required to measure the sleep activity and depth of sleep to create an overall interpretation of sleep quality in DSPD patients.

Patients with DSPD have specific definitions of a healthy, restorative sleep because of biological predispositions, but most generally have normal-high BMIs because of their susceptibility to unhealthy lifestyles. To quantify a patient's health and diet quality, Berendsen et al. (2020) referred to the Dutch Healthy Diet (DHD) index that places exercise, food quality, and drinks on a scale. Information on chrono-nutrition and alpha-lactalbumin are scarce, but researchers are expanding on the idea that chrono-nutrition and intake of a-lac can conjunctively alter an individual's sleep quality. To verify this connection between tryptophan, sleep, and chrono-nutrition, Markus et al. (2005) tested 14 subjects who were considered "good sleepers" and 14 subjects who were considered "poor sleepers." The morning after providing the subjects with evening a-lac, Markus et al. (2005) measured the effectiveness of the protein on sleep quality by subjectively asking the participants to rate their alertness levels on the Stanford Sleepiness Scale. Another alertness quantification method was through physical and mental challenges in which the participants completed continuous performance tasks (CPT)

to determine their agility, accuracy, and speed. As a last form of data quantification, the researchers performed EEGs and event-related potential (ERP) tests to analyze the eye-movement signals and deep sleep stages. All these methods of measurements accurately showed net improvement in the poor sleepers who consumed a-lac, and the results from each test correlated with this result, supporting the accuracy of these alertness tests. These alertness and sleep tests were performed to acquire an average, quantifiable measurement of sleep quality, and in future follow-up studies, the same tests should be performed to conduct baseline comparisons.

A follow up study needs to be conducted to test the efficacy of the MILC treatment, consisting of DSPD patients within the ages of 18-30. Subjects should be considered high-stressed based on the Perceived Stress Scale (PSS) in which individuals' stress levels are categorized and assessed using subjective life situations. Researchers should determine the length of the study depending on parameters, but control and experimental groups should have the same diet quality based on the Dutch Healthy Diet (DHD) index. The experimental group should have a set milk intake time in the evening, preferably 2 hours before the subjects go to sleep. The amount of light exposure for the test subjects should also be kept similar because external exogenous factors can negate the effects of milk intake. To obtain results, the Trp:LNAA ratio should be measured in individuals before, during, and after the experimental manipulation of chrono-nutrition and milk consumption. As for melatonin concentrations, the DLMO profiles should be assessed through the collection of saliva to evaluate the effectiveness of the endogenous circadian rhythm and the pineal gland melatonin releases. A sleep EEG can be used to determine the sleep stages and rhythms that an individual experiences over the course of the night. To acquire a combined, quantifiable result, standardized sleep scales and deep sleep brain waves should be utilized to determine sleep quality in patients with DSPD. Alterations to the MILC treatment, such as adjusting the temperature of milk, can possibly affect its sleep inductive qualities and affinity to the BBB, but further research is needed on the subject.

The MILC treatment suggests that a potential solution for DSPD patients' sleep quality is the ingestion of one cup of whole cow's milk to raise melatonin levels at night and induce deeper sleep. Considering chrono-nutrition, high-stress individuals who consistently time their high-tryptophan diets can yield the greatest increase in their Trp:LNAA ratio, enabling melatonin synthesis to effectively enhance agility and morning alertness.

7. Discussion

Individuals who maintain a relatively healthy lifestyle and diet, even when struggling with an unbalanced sleep schedule, can counteract negative symptoms through small alterations in their lifestyles. Consider DSPD patients who are high-stressed and unhealthy in terms of both sleep and diet; these adversities inhibit internal regulation of hormonal concentrations and can pave a path for serious illnesses. At the core of treating their disorder, these individuals need to obtain a balance of the sleep hormone, melatonin, to rise and fall at set times. To achieve this goal, the MILC treatment acts upon the concept of chrono-nutrition, or timed consumption, to raise the chances of molecular components entering a specific synthesizing pathway. For this theoretical treatment, DSPD patients will consistently consume 1 cup of whole cow's milk at a predetermined time based on individual needs. Over time, the individuals should sense an improvement in morning alertness and enhanced sleep quality, averting negative health effects. Synthesizing melatonin and combating an altered circadian schedule through milk can be a non-supplemental method to naturally reestablish a healthy sleep cycle. Although consumption of milk once will not establish results immediately, a consistent sleep, diet, and exercise schedule is crucial in efficiently regulating the body. Body types can contribute to an individual's response to the MILC treatment, but further research is needed to determine and adjust specific quantities of milk and timing of consumption. This potential treatment is meant to be an alternative method to supplements, and it can only work if an individual does not hinder the process through unnecessary exposure to delaying exogenous factors. A DSPD patient needs to be receptive towards alternative treatment methods that target specific symptoms, and the only way to figure out the best needs for a specific individual is through trial and error.

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