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Melatonin and Sleep Qualities in Healthy Adults: Pharmacological and Expectancy Effects

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ABSTRACT. The impact of expectancy on melatonin's effects on sleep qualities was investigated. Both the pharmacological dose of 6 mg of melatonin and the expectation of receiving melatonin were predicted to improve subjective ratings of sleep qualities. The balanced placebo design varied 2 factors within-subjects: actual treatment and expected treatment. Adults (N = 53; 21 men and 32 women) between the ages of 26 and 71 years were administered either 6 mg of melatonin or a placebo for 8 nights. An instructional manipulation directed participants' expectations. Participants rated their nightly sleep experiences. Results revealed that feelings upon awakening differed between genders and that expecting melatonin increased ratings of sleep continuity. Most important, high ratings of "grogginess/tiredness" were associated with receiving melatonin, regardless of expectancy, as well as with receiving placebo when melatonin was expected. Overall, the findings underscore the need to consider expectancy and gender differences in research on melatonin and sleep experiences.

Key words: expectancy effects, melatonin, sex differences, subjective sleep experiences

NEARLY 40 years ago, Aaron Lerner discovered a hormone that produced a "mild sedation" in humans when administered intravenously (Zhdanova & Wurtman, 1997). That hormone came to be known as melatonin. Since that discovery, a great deal has been learned about this so-called sleep hormone. Melatonin is produced and secreted by the pineal gland, whose activity is regulated by the suprachiasmatic nuclei (SCN). The SCN are functional groupings of about 10,000 cells located in the hypothalamus (Dement, 1992; Haimov & Lavie, 1996; Klein, Reppert, & Moore, 1991; Sack, Lewy, Erb, Vollmer, & Singer, 1986). Changes in illumination influence the SCN and, thus, circulating levels of melatonin are at their peak during darkness (Cagnacci, 1996; Shanahan & Czeisler, 1991; Tarquini, Cornelissen, Perfetto, Tarquini, & Halberg, 1997). These findings have

inspired numerous studies examining melatonin's role in the regulation of the human sleep/wake cycle. Many studies have affirmed melatonin's involvement in the synchronization of human circadian rhythms (e.g., Armstrong, Cassone, Chesworth, Redman, & Short, 1986; Cassone, 1990; Lewy, Ahmed, Latham-Jackson, & Sack, 1992). Research has shown that melatonin possesses sleep-inducing properties when an exogenous dose is administered (Anton-Tay, 1974; Anton-Tay, Diaz, & Fernandez-Guardiola, 1971; Cramer, Rudolph, Consbruch, & Kendel, 1974; Vollrath, Semm, & Gammel, 1981). Both healthy individuals and those suffering from insomnia have experienced accelerated sleep onset after melatonin treatment (Dahlitz et al., 1991; Dijk & Cajochen, 1997; Hughes & Badia, 1997; Reid, van den Heuvel, & Dawson, 1996; Waldhauser, Saletu, & Trinchard-Lugan, 1990). This reduction in sleep onset latency appears to be the most commonly observed effect of exogenous melatonin. Other reported effects include an increase in total sleep time (Attenburrow, Cowen, & Sharpley, 1996; Hughes & Badia, 1997; MacFarlane, Cleghorn, Brown, & Streiner, 1991), a decreased number of nighttime awakenings (Waldhauser et al., 1990; Wurtman & Zhdanova, 1995), and greater sleep efficiency (Attenburrow et al., 1996; Garfinkel, Laudon, Nof, & Zisapel, 1995; Waldhauser et al., 1990; Zhdanova, Wurtman, & Lynch, 1995).

Researchers have explored melatonin's impact on diverse populations, with a wide range of doses, using various durations of treatment. In general, these researchers have examined the pharmacological properties of melatonin to account for the relationship between melatonin and sleep. There has yet to be an investigation of the cognitive effects of melatonin treatment. In light of the exuberance with which the populace has greeted the widely publicized claims regarding melatonin's positive effects, it is possible that these claims themselves have had an impact on the results of many studies. Perhaps some of the detected effects are the result of participant expectations about melatonin's impact on sleep. Expectations arise when individuals acquire reliable information regarding the association between events (Fillmore & Vogel-Sprott, 1994). For example, if a participant is presented with information stating that melatonin improves sleep, then that participant is likely to expect that taking melatonin will result in improved sleep. This tendency raises the following question: Does the simple act of taking a pill that may improve sleep lead individuals to judge that their sleep

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has improved, or is the melatonin actually having an effect on sleep quality? Previous research has not directly addressed the role of participant expectations in relation to melatonin.

Studies examining the impact of response expectancies have demonstrated that participant expectations contribute to self-reported differences. Early investigations indicated that participants' responses to a placebo were similar to their responses to an "active drug" (Lasagna, Laties, & Dohan, 1958). More recent examinations indicate that the effectiveness of a placebo ultimately depends on the participant's beliefs or expectations regarding the outcome of the treatment (Kirsch & Baker, 1993). Several studies investigating the impact of expectancy and the effects of alcohol consumption have supported this notion. Lang, Goeckner, Adesso, and Marlatt (1975) found that participants who had been told they received alcohol behaved more aggressively than those who had not been told they received alcohol. These results were independent of whether the participants had actually consumed the drug. Similarly, Rohsenow and Bachorowski (1984) found that participants' expectancies prior to consuming alcohol predicted their post-drinking verbal aggression. Another experiment (Fillmore & Vogel-Sprott, 1994) demonstrated a positive relationship between participants' expectations and their performance on a psychomotor task. Those who expected alcohol to negatively affect their performance performed more poorly on the task regardless of whether they received the drug or placebo. Interestingly, the same relationship held true for participants given caffeine, a substance thought to improve psychomotor performance. Those participants who expected the least improvement due to caffeine consumption subsequently performed the most poorly on the task under both drug and placebo conditions. In these cases, it is evident that participants' beliefs affected the outcomes of the treatments. The possibility of an expectancy effect with respect to melatonin treatment also warrants scientific investigation.

Although many studies have addressed melatonin's effects relative to a placebo, those studies have sought to control only for the expectations that participants bring into the experiment. Such a design is based on the assumption that participants carry with them certain generic expectations throughout each condition. The present study was conducted to go beyond that assumption and measure not only the effects of a treatment but also the contribution of participants' specific expectations regarding that treatment. This is an important link missing in the current literature on melatonin. Consumers of exogenous melatonin have often responded to widespread anecdotal claims. Those claims have the potential to affect the expectations of those taking melatonin and also to affect their patterns of usage. For that reason, it is necessary to examine which effects of melatonin are actual treatment effects and which can be produced by the expectation of treatment.

In the present study, we used a balanced placebo design to assess both the pharmacological and the cognitive effects of melatonin on the subjective sleep

experiences of healthy adults. This design included four conditions as the result of crossing treatment (melatonin or placebo) and expectancy (melatonin or placebo). A sample of 53 healthy adults ingested either 6 mg of melatonin or a placebo prior to their bedtime for eight nights (two trials for each condition). Their expectancies as to which substance they received on each night were manipulated through instructional methods. Each morning, participants completed a Daily Subjective Sleep Experience Questionnaire (DSSEQ), which measured specific aspects of their sleep experiences, such as feelings prior to sleep, depth and continuity of sleep, and feelings and mood upon awakening. On the basis of previous research, we expected the ease of sleep onset as well as depth and continuity of sleep to be improved when participants received melatonin (Dahlitz et al., 1991; Dijk & Cajochen, 1997; Hughes & Badia, 1997; Reid et al., 1996; Rose, Chase, Blazej, & Kahan, 1999; Waldhauser et al., 1990). In addition, through the use of the instructional manipulation, we anticipated that participants would report decreased sleep onset latency and increased depth and continuity of sleep throughout the night when melatonin was the perceived condition.

Method

Participants

Participants were recruited from the faculty and staff at Santa Clara University and the surrounding community. Potential participants received recruitment letters, which included a brief overview of the purpose and procedures and an initial prescreening questionnaire. In all, 1,046 recruitment letters were sent. A total of 108 questionnaires indicating the person's interest in participating in the study were returned (10.3% return rate). Forty of the 108 potential participants were excluded because of unstable sleep patterns, excessive intake of alcohol or caffeine, use of nicotine, or pre-existing medical conditions that presented added health risks (e.g., diabetes, hypoglycemia, clinical depression, leukemia, epilepsy, autoimmune diseases, severe allergies to food), or because they were women who were nursing or pregnant. An additional 15 individuals were excluded after beginning the experimental protocol due to noncompliance with the instructions.

In all, 53 participants (21 men, 32 women) between the ages of 26 and 71 years (M = 42.5) completed the study. These individuals displayed healthy and stable sleeping patterns as shown by the results of the prescreening process (see Table 1 for complete demographic data). Among the prescreening variables, only age differed significantly ($p < .05$) between men and women. Ninety-four percent of the sample reported going to bed within 1 hr of the same time each weeknight and waking up within 1 hr of the same time each morning. Participants reported an adequate amount of nightly sleep (between 5 and 9 hr per night) and consistency maintaining regular sleep and wake times. There was no evidence of excessive napping; participants reported taking less than one nap per week (M = 0.8). In addition, individuals were selected because of their minimal intake of sleep-altering substances. Those who participated consumed an average of 1.5 cups of caffeinated coffee or tea and less than one caffeinated soda per day. Participants also reported consuming alcoholic beverages an average of less than one weeknight per week (M = 0.8). None of those selected to participate were taking melatonin at the time of the study, nor did they regularly smoke.

TABLE 1
Demographic Characteristics of the Study Sample

Question	Men (n = 21)			Women (n = 32)		
	M	*SE*	Range	*M*	*SE*	Range
Age	47.25	3.12	27–71	40.07	1.80	26–60*
Sleep (hr/day)	6.90	0.24	5–9	6.94	0.17	5–9
Number of naps (per week)	1.00	0.30	0–4	0.58	0.17	0–4
Length of naps (min)	18.00	5.00	10–60	13.55	3.49	10–70
Sleep difficulties (nights/week)	2.00	0.36	0–5	2.26	0.29	0–5
Coffee/tea (cups/day)	1.65	0.31	0–5	1.42	0.20	0–3
Soft drinks (drinks/day)	0.95	0.28	0–4	0.74	0.15	0–3
Alcohol consumed (nights/week)	1.10	0.31	0–5	0.71	0.21	0–5

*Comparison between men and women was significant at $p < .05$.

Participation was voluntary, and all individuals gave informed consent before beginning the study. Those who completed the study received $25 as compensation for their participation. Participants were treated in accordance with APA ethical guidelines, and all materials and procedures were approved by the Santa Clara University Human Subjects Committee.

Materials

Members of the faculty and staff at Santa Clara University were mailed a recruitment letter via campus mailing services. This letter briefly described the study and provided general background information about melatonin. A prescreening questionnaire accompanied the letter, which individuals were asked to complete and return if they were interested in being in the study. Individuals from the surrounding community were recruited by word of mouth and flyers advertising the study posted on the Santa Clara University campus. Potential participants responded to questions regarding average nightly hours of sleep; consistency of bedtime and time of awakening; napping tendencies; and consumption of alcohol, caffeine, and nicotine. Persons with erratic sleeping patterns or excessive intake of alcohol, caffeine, and nicotine were not invited to participate. The informed consent document served as an additional screening device. This doc-

ument, which all participants read and signed before beginning the study, contained general information regarding the uses of melatonin, its safety, and a brief summary of the experimental procedures. The informed consent also instructed those with pre-existing medical conditions (diabetes, hypoglycemia, clinical depression, leukemia, epilepsy, autoimmune diseases, severe allergies to food) and women who were nursing or pregnant not to participate in the study. Five individuals declined to participate or were excluded for these precautionary measures after reading and discussing the informed consent.

Participants received individually coded packets that contained the experimental materials and instructions for completing the study. The code indicated the order of pill ingestion throughout the study as well as the experimental condition for any given night. Each packet contained nine envelopes, labeled Day 1 through Day 9. The Day 1 envelope contained a DSSEQ only. The remaining envelopes contained a DSSEQ and a pill envelope, which held either a 6-mg dosage of melatonin (two 3-mg capsules) or a physically identical placebo (two gelatin capsules filled with organic rice flour). Studies have shown that 6 mg of melatonin is within the range of what is considered a safe and potentially effective dosage (Hughes & Badia, 1997; Jan & O'Donnell, 1996; MacFarlane et al., 1991; Waldhauser et al., 1990). A single, 3-mg dosage may not be large enough to produce a significant effect (Mishima, Satoh, Shimizu, & Hishikawa, 1997). The melatonin pills were obtained from TwinLab, and the placebos were assembled under a laminar flow hood (to ensure sterility), using gelatin capsules and heat-sterilized organic rice flour. The melatonin and placebo capsules were physically identical with respect to size, color, and consistency of contents.

The DSSEQ took into account principles found in previously used, subjective sleep-quality scales. Many questions, such as the ease of falling asleep, the continuity of sleep, and the feelings and mood upon awakening, can be found throughout the literature on melatonin and sleep (Frankel, Buchbinder, & Snyder, 1973; James, Sack, Rosenthal, & Mendelson, 1990; Parrott & Hindmarch, 1978; Webb, Bonnet, & Blume, 1976). Research has shown that subjective assessments of sleep quality and objective measures of sleep microstructure are well correlated (Ferini-Strambi et al., 1993). The DSSEQ focused on measuring a range of subjective sleep experiences, such as the ease of falling asleep, the depth of sleep, and the ease of awakening. Each of the 12 questions was scored on an 11-point scale, with 1 being the lowest response (i.e., not continuous sleep, not easy to fall asleep, or not rested upon awakening) and 11 being the highest response (i.e., very deep sleep, very rested upon awakening, or very groggy/tired prior to falling asleep).

Included in the DSSEQ was a sleep diary, which participants used to record information about their nightly sleep. They responded to questions such as the estimated time of sleep onset, the time of pill ingestion, the number of awakenings throughout the night, the time spent awake after the initial sleep onset, and the time of awakening. We needed to ensure that individuals' use of sleep-altering substances did not vary from the patterns found in the prescreening process; therefore, the DSSEQ also asked participants to report the amount of alcohol and caffeine consumed the previous day. We informally reviewed responses to these questions to ensure that participants did not deviate from their initially reported sleep/wake patterns.

Design

A 2 (actual treatment) × 2 (expected treatment) factorial within-subjects design with repeated measures was used. This resulted in the following four conditions: (a) expected melatonin/received melatonin, (b) expected melatonin/received placebo, (c) expected placebo/received placebo, and (d) expected placebo/received melatonin. Participants experienced each condition twice over the course of the study. Although they were correctly instructed regarding their actual condition on half of the trials, participants remained blind to the experimental procedure—they were unaware of which trials were labeled correctly and which were not. This deceptive method of placebo administration has been found to more effectively produce a placebo effect than double-blind administration (Lotshaw, Bradley, & Brooks, 1996). Treatment orders were counterbalanced via block randomization, and each participant was assigned to one of eight orders. Melatonin's relatively short biological half-life (Hughes, Sack, & Lewy, 1998; Waldhauser et al., 1990) allows for the use of a within-subjects design. A nighttime dosage as high as 50 mg has been shown to pass through the body by the subsequent morning (Lamberg, 1996). Thus, it would be highly unlikely that melatonin treatment on one night would continue to influence sleep the following night.

Procedure

Individuals invited to participate attended an orientation session, at which instructions as to the purpose and the protocol of the study were given. In an attempt to control for the many preconceived ideas that individuals may have held regarding melatonin and its effects, participants were told that previous research had indicated that melatonin decreased sleep onset latency and improved depth and continuity of sleep. The experimenters also explained that this research did not show any positive or negative effects the following morning. Participants were instructed that these results were found among a sample of young adults (ages 18–22 years; Rose et al., 1999) and that they could expect to experience a greater influence because endogenous levels of melatonin decrease with age (Hughes et al., 1998; Sack et al., 1986). These instructions were given to set up the specific aforementioned expectancies. Because individuals would most likely begin with diverse beliefs concerning melatonin, it was necessary to manipulate their actual expectancies prior to the onset of the experimental protocol. Research has shown

that verbal/instructional manipulations have been successful at influencing participants' experiences (Montgomery & Kirsch, 1997). During this session, participants were also informed that they would be made aware of the experimental condition on each night by way of the labeled pill envelopes. Each individual pill envelope was clearly labeled melatonin or placebo on both the front and the back sides. These labels were intended to set up participants' expectations as to which substance they would be receiving on each experimental trial. To answer the questions that would inevitably arise regarding this procedural method, we gave a detailed explanation to each participant. We explained that this method was being used to eliminate the role of participant conjecture on the data collection. We further explained that, in the past, participants in blind studies were continually speculating on which condition occurred on each night. This speculation introduced an unwanted variable to the analysis. For this reason, those in the present study were being informed of the contents of the pills prior to ingestion. Anyone who expressed dissatisfaction with this explanation was excused from participation and immediately given complete disclosure regarding the procedural manipulation. One individual was excluded after declaring skepticism regarding the experimental instructions.

Beginning on a Sunday night, data were collected for nine nights over two weeks. Friday and Saturday evenings were excluded so that the variability of weekend sleep habits would not interfere with data collection. The first night served as a practice trial, which enabled participants to become familiar with the DSSEQ and the experimental protocol. No pills were ingested on this night, and data from this trial were not included in the analysis. On each of the following eight nights, participants were instructed to ingest the contents of the labeled pill envelopes with a full glass of water approximately 30 min prior to bedtime. The timing of pill ingestion was vital because research has shown that melatonin may produce different sleep onset effects depending on the time the pill is administered (Tzischinsky & Lavie, 1994). Within 30 min of awakening the subsequent morning, participants completed the DSSEQ. Upon completion, the DSSEQ was to be placed back into the daily envelope and sealed. Individuals then dated and signed a checklist on the outside of their packet. This procedure was repeated on each of the experimental nights.

After completing the study, participants attended a debriefing session. The materials were collected, and the true purpose of the study was revealed. Participants were informed that the investigation was intended to examine the impact of expectancy on melatonin's effects on subjective sleep quality. We explained that to achieve this goal, we had to use deception with regard to the condition on half of the trials. We explained that this deception was necessary in order to investigate the possible effects of expectations on the reported qualities of sleep. Any remaining questions or concerns were also addressed. Participants received $25 in appreciation for their taking part in the investigation and were told that the results of the study would be made available to them.

Results

Psychometric Properties of the DSSEQ

It is important to first consider the psychometric properties of the DSSEQ, because the questions in this measure were adapted from other sleep quality scales. For each of the 12 questions, a mean rating was calculated by adding the participants' ratings for a given question across the eight experimental trials and dividing by 8. We then conducted a correlational analysis (Pearson *r*) to determine the strength of the linear relationship between participants' average ratings. We expected the ratings to be highly correlated, although we also anticipated that the questions would measure distinguishable qualities of sleep.

Table 2 presents the item-item correlations for the 12 questions. A Bonferroni approach was used to adjust for the increased likelihood of Type I errors resulting from multiple correlations involving the same information; the resulting experimentwise alpha level was .004 (Cliff, 1987; Keppel & Zedeck, 1989). Individual questions were reliably correlated with between 3 other questions (Question 6: groggy/tired before sleep) and 8 other questions (Question 12: overall sleep quality; see Table 2). Of particular interest was the pattern of item-item correlations for Question 6 (groggy/tired). Participants' ratings for this question were significantly and

TABLE 2
Intercorrelations Between Questions (N = 53)

Question	1	2	3	4	5	6	7	8	9	10	11	12
1. Rested	—	.80*	.84*	.91*	.32	.06	.54*	.47*	.25	.68*	.38	.61*
2. Alert		—	.94*	.91*	.24	.04	.67*	.64*	.25	.72*	.29	.51*
3. Energetic			—	.94*	.27	.05	.67*	.64*	.27	.76*	.35	.56*
4. Refreshed				—	.32	.04	.61*	.56*	.28	.75*	.39	.61*
5. Ease of falling asleep					—	.59*	.26	.24	.50*	.32	.56*	.53*
6. Groggy/tired prior to sleep						—	.09	.04	.40*	−.02	.40*	.33
7. Ease of awakening							—	.94*	.16	.62*	.12	.28
8. Ease of getting out of bed								—	.17	.62*	.13	.27
9. Continuity of sleep									—	.34	.81*	.74*
10. Mood upon awakening										—	.36	.58*
11. Depth of sleep											—	.87*
12. Overall quality of sleep												—

Note. Questions 1–4 refer to participants' feelings upon awakening.
*$p < .004$.

positively correlated with only 3 other questions: ease of falling asleep (Question 5), continuity of sleep (Question 9), and depth of sleep (Question 11). These intercorrelations were consistent with the results of previous research showing that melatonin often affects these sleep-related experiences (Haimov et al., 1995; Nave, Peled, & Lavie, 1995; Tzischinsky & Lavie, 1994; Waldhauser et al., 1990; Zhdanova et al., 1995). Also of note is the absence of a reliable relationship between overall quality of sleep (Question 12) and groggy/tired before sleep (Question 6), ease of awakening (Question 7), and ease of getting out of bed (Question 8). All other items were highly correlated with overall quality of sleep (Question 12; see Table 2).

Table 3 presents the corrected item-total correlations. With the exception of Question 6 (groggy/tired), all correlations were greater than .3. This shows that the DSSEQ was internally consistent and that 11 of the 12 items reliably measured an aspect of the larger construct (subjective sleep experience; Anastasi & Urbina, 1997). Further evidence of the reliability of the DSSEQ could be seen in the computed Cronbach coefficient alpha of .91.

Given the importance of knowing whether the individual items were measuring different aspects of subjective sleep experience, we also computed Hotelling's T^2. This statistic tests the hypothesis that the mean ratings are not different, while controlling for the possibility that the intercorrelations among ratings vary considerably, thus controlling the Type I error rate (Hair, Anderson, Tatham, & Black, 1995). In the present case, $T^2(11, 42) = 15.26$, $p < .00001$, indicating that the questions differed from each other.

Thus, for the psychometric analyses, we found that the DSSEQ, as administered in the present study, was internally consistent and contained reliable items, that also were not redundant.

TABLE 3
Corrected Item–Total Correlations Between Questions

Question	Corrected item–total correlation coefficient
1. Rested upon awakening	.75
2. Alert upon awakening	.77
3. Energetic upon awakening	.81
4. Refreshed upon awakening	.81
5. Ease of falling asleep	.53
6. Groggy/tired prior to sleep	.25
7. Ease of awakening	.63
8. Ease of getting out of bed	.60
9. Continuity of sleep	.52
10. Mood upon awakening	.74
11. Depth of sleep	.59
12. Overall quality of sleep	.76

Analysis of the Experimental Design

Analytic strategy. Our primary interest was in the effects of the actual treatment (received melatonin vs. received placebo) and the expected treatment (expected melatonin vs. expected placebo) on participants' ratings of their sleep-related experiences. The basic design was a 2 (actual treatment) × 2 (expected treatment) factorial within-subjects design with repeated measures. Participants experienced each of the four experimental conditions twice and completed the DSSEQ each time. Hence, for each of the 12 questions, a mean rating was computed by adding the participant's ratings on that question for the two nights the given condition was experienced and dividing by 2. Table 4 presents the mean ratings for each question, as a function of actual condition and expected condition.

Participants' gender was added to the design as a blocking variable. We felt this would increase the sensitivity of the analysis to differences across conditions in light of individual differences in response to melatonin (e.g., Arendt et al., 1987; Zhdanova & Wurtman, 1997; Zhdanova, Wurtman, Morabito, Piotrovska, & Lynch, 1996).

For each of the 12 questions on the DSSEQ, we conducted an analysis of variance (ANOVA) using SPSS (1988) to determine whether participants' mean

TABLE 4
Mean Ratings and Standard Errors of Sleep Qualities as a Function of Actual (Melatonin/Placebo) and Expected (Melatonin/Placebo) Conditions

	Actual condition							
	Mel				Plc			
	Exp Mel		Exp Plc		Exp Mel		Exp Plc	
Question	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*
1. Rested upon awakening	6.57	.39	6.38	.37	6.63	.31	6.81	.37
2. Alert upon awakening	6.63	.36	6.55	.33	6.72	.35	6.79	.38
3. Energetic upon awakening	6.31	.39	6.29	.34	6.25	.32	6.35	.35
4. Refreshed upon awakening	6.23	.40	6.13	.36	6.14	.32	6.42	.37
5. Ease of falling asleep	8.19	.40	8.13	.44	8.32	.37	7.71	.40
6. Groggy/tired before sleep	7.31	.41	7.03	.37	7.01	.47	6.17	.34*
7. Ease of awakening	6.34	.46	6.53	.42	6.23	.42	6.34	.43
8. Ease of getting out of bed	6.13	.48	6.31	.41	6.15	.37	6.08	.43
9. Continuity of sleep	7.41	.44	6.50	.43	6.87	.46	7.15	.45**
10. Mood upon awakening	6.97	.36	6.63	.36	6.63	.32	6.89	.34
11. Depth of sleep	7.63	.38	7.33	.37	7.69	.39	7.49	.39
12. Overall quality of sleep	7.20	.40	6.74	.37	7.13	.36	7.26	.35

Note. Mel = melatonin. Plc = placebo. Exp = expected condition.
*Main effect of actual condition was significant at $p < .05$. **Main effect of expected condition was significant at $p < .05$.

ratings varied as a function of the between-subjects factor (gender) and/or the two within-subjects factors (treatment received: melatonin or placebo, and treatment expected: melatonin or placebo). Effect size (η^2) and power were also computed (see Appendix).

Because there were unequal numbers of men and women, we also tested for violations of the assumption of homogeneity of variance using Box's M test, which is recommended for repeated measures designs (SPSS, 1988). This test is highly sensitive to departures from normality (Bray & Maxwell, 1985; Hair et al., 1995); therefore, the recommended alpha level of .01 (Hair et al., 1995) was adopted for the present analysis. None of the ANOVAs violated the assumption of homogeneity of variance (all p values > .01).

Significant ANOVA results. The ANOVA for Question 6 (feeling groggy/tired before sleep) produced our most complex set of findings. In general, participants' mean ratings were higher when they received melatonin (M = 7.17) than when they received placebo (M = 6.59), $F(1, 50) = 7.08$, $MSE = 2.52$, $p = .01$, $\eta^2 = .12$, power = .74. This finding is consistent with our original predictions, our cover task, and past research (Anton-Tay et al., 1971; Arendt, Borbely, Franey, & Wright, 1984; Rose et al., 1999; Vollrath et al., 1981; Zhdanova et al., 1995). However, this main effect is qualified by two significant two-way interaction effects. First, a significant interaction between actual condition and expected condition revealed that the effect of treatment depended on whether participants expected melatonin or placebo, $F(1, 50) = 6.59$, $MSE = 2.87$, $p = .013$, $\eta^2 = .12$, power = .71. Figure 1 presents the pattern of means for this interaction.

When participants received melatonin, their mean ratings of grogginess/tiredness did not differ when they expected melatonin (M = 7.31) and when they expected placebo (M = 7.03), $F(1, 52) = 1.00$, $MSE = 1.72$, $p = .32$. This pattern shows a treatment effect in that melatonin had an impact on participants' ratings even when they expected placebo.

When participants received placebo, their mean ratings of grogginess/tiredness were higher when they expected melatonin (M = 7.01) than when they expected placebo (M = 6.17), $F(1, 52) = 5.35$, $MSE = 3.41$, $p = .02$. This pattern shows an expectancy effect, in that expecting melatonin (but getting placebo) either elevated ratings (in light of expectancies regarding melatonin's effects), or expecting placebo (and getting placebo) suppressed ratings (in light of expectancies regarding receiving no treatment). In short, expectancy had a greater effect on participants' ratings when they received placebo than when they received melatonin.

The second significant interaction discovered for Question 6 (feeling groggy/tired before sleep) was between gender of participant and expectancy, $F(1, 50) = 4.79$, $MSE = 2.18$, $p = .033$, $\eta^2 = .09$, power = .57. Figure 2 shows the pattern of means. The general trend was similar for men and women, in that both rated their grogginess/tiredness before sleep higher when melatonin was expected than when placebo was expected (see Figure 2). However, the difference in the mean ratings given by men and women was greater when the placebo was the expected condition (difference between means = .34).

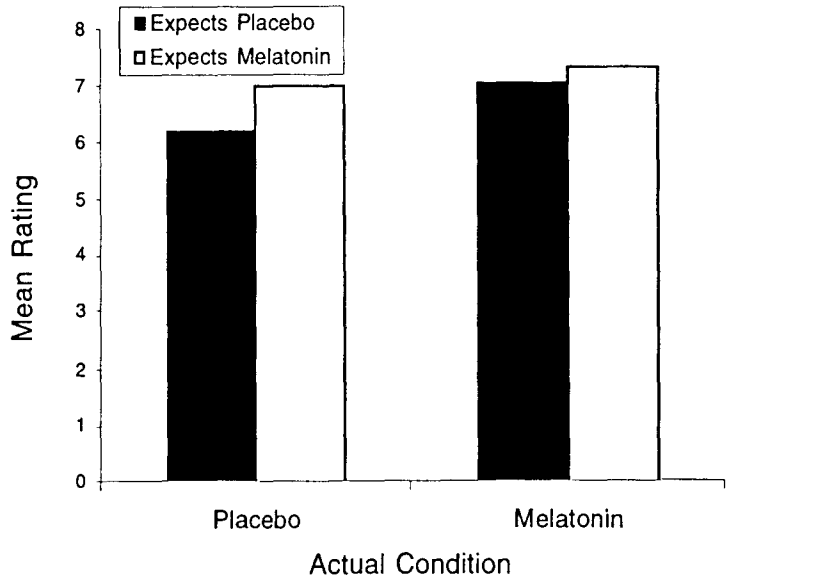

FIGURE 1. Mean ratings of grogginess/tiredness prior to sleep as a function of actual condition and expected condition.

Pairwise comparisons showed that, when placebo was the expected condition, women's mean ratings of their grogginess/tiredness before sleep were higher (M = 6.94) than men's (M = 6.07), $F(1, 51) = 3.85$, $MSE = 2.45$, $p = .05$. When melatonin was the expected condition, mean ratings did not differ for women (M = 7.29) and men (M = 6.95), $p > .10$.

In summary, both actual and expected treatment influenced participants' ratings of their grogginess/tiredness prior to sleep; however, the pattern varied for men and women.

The ANOVA for Question 9 (continuity of sleep) showed a main effect of expectancy. Participants rated their sleep as more continuous when they expected melatonin (M = 7.14) than when they expected placebo (M = 6.83), $F(1, 50) = 5.75$, $MSE = 2.21$, $p = .02$, $\eta^2 = .10$, power = .65. This result is again consistent with the information provided in our cover task, with our initial predictions, and with past research (Anton-Tay et al., 1971; Arendt et al., 1984; Rose et al., 1999; Vollrath et al., 1981; Zhdanova et al., 1995).

ANOVAs of Questions 7 (ease of awakening) and 8 (ease of getting out of

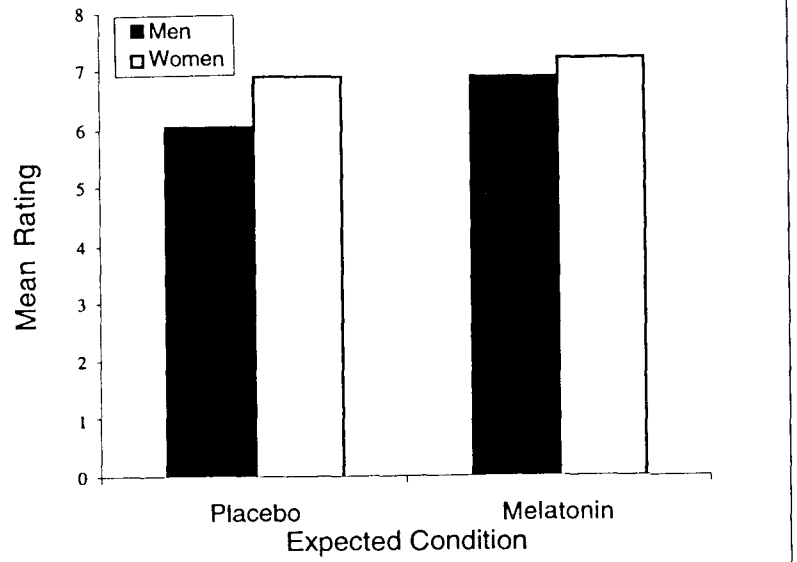

FIGURE 2. Mean ratings of grogginess/tiredness prior to sleep as a function of participant gender and expected condition.

bed) produced reliable gender differences. Men reported greater ease of awakening (M = 7.18) than did women (M = 5.84), $F(1, 50) = 7.17$, MSE = 12.29, p = .01, η^2 = .13, power = .75. Similarly, men reported greater ease of getting out of bed (M = 7.05) than did women (M = 5.62), $F(1, 50) = 8.41$, MSE = 12.02, p = .006, η^2 = .41, power = .81. No other reliable main effects or interactions ($p < .05$) emerged for the 12 questions of the DSSEQ.

Discussion

Our findings suggest that both pharmacological and expectancy factors influence how melatonin affects subjective ratings of sleep qualities by healthy adults. The significant results indicate that melatonin facilitates an increased grogginess or tiredness prior to sleep onset. However, this sleep-promoting effect is qualified by both expectancy and the gender of the participant. Additionally, the expectation of receiving melatonin resulted in ratings of greater sleep continuity. These results are consistent with our initial predictions. However, the predictions regarding subjective depth of sleep were not confirmed. Our findings also indicate that significant gender differences may exist with respect to post-sleep experiences. These particular findings do not support our original hypotheses, but they indicate a need to further explore the impact of gender in this area of research.

Pharmacological factors affected subjective reports of participants' experiences prior to sleep onset. A significant increase in grogginess/tiredness (Question 6) prior to sleep was associated with melatonin treatment. This hypnotic property is highly consistent with reports from previous research showing that melatonin both induces subjective sleepiness (Anton-Tay et al., 1971; Arendt et al., 1984; Vollrath et al., 1981; Zhdanova et al., 1995) and decreases sleep onset latency among various populations (Cramer et al., 1974; Dahlitz et al., 1991; Dijk & Cajochen, 1997; Hughes & Badia, 1997; Lushington, Pollard, Lack, Kennaway, & Dawson, 1997; Reid et al., 1996; Waldhauser et al., 1990; Zhdanova et al., 1996). It stands to reason that increased feelings of grogginess or tiredness produced by melatonin are correlated with a shorter sleep onset latency. The present results support what is the most common finding throughout the literature on melatonin: that its administration effectively promotes sleep onset in humans (Anton-Tay, 1974; Haimov et al., 1995; Nave et al., 1995; Tzischinsky & Lavie, 1994). The correspondence between our findings and those of past research also speaks to the validity of the present study. The replication of melatonin's sleep-promoting effects increases the confidence with which we view these results. When placed into the context of past research, the present findings suggest that melatonin could indeed offer relief both to those who suffer from temporary difficulties initiating sleep and to healthy sleepers alike.

Although the results of this investigation lend further support to the increasing literature on melatonin's sleep-promoting properties, the discussion about Question 6 (groggy/tired prior to sleep) does not end here. In light of the instructional manipulation (or cover task), it is not surprising that further analysis of this question revealed a two-way interaction between actual condition and expected condition. Participants' ratings of their grogginess/tiredness prior to sleep hinged on what they expected to receive. This interaction reveals the impact of both treatment and expectancy. When participants received melatonin, expectancy did not affect their ratings. Participants gave comparable ratings of grogginess/tiredness when they expected melatonin and when they expected placebo. Thus, the pharmacological effects of melatonin transcended any effects of expectancy. However, when participants received placebo, expectancy did affect their ratings. Participants' ratings of their grogginess/tiredness were lower when they expected placebo than when they expected melatonin. Thus, the combination of expecting and receiving placebo seemed to result in suppressed ratings of grogginess/tiredness. This finding was possibly attributable to participants' expectations of receiving no treatment. The corollary here is that participants' ratings of their grogginess/tiredness were elevated when they expected melatonin, even when no melatonin was administered.

The second interaction observed with Question 6 (groggy/tired prior to sleep) was between gender of the participant and expectancy. The fact that both men and women reported increased grogginess/tiredness prior to sleep when melatonin was the expected condition, compared with when placebo was expected, again supports the notion that the cover task was successful in manipulating partici-

pants' expectancies. However, it is also interesting that the shift from expecting melatonin to expecting placebo had a more substantial impact on the men than on the women. This finding implies that the cover task influenced the ratings of men more than the ratings of women. It could be conjectured that women are simply more inclined to provide higher ratings in general. This could have led to more consistent ratings across the conditions. It could also be that the men and women had different expectations about how a placebo may affect sleep. After all, the cover task was not designed to set up any specific expectations about the placebo. Fillmore and Vogel-Sprott (1994) reported that expectancies can be acquired and/or influenced in any number of ways. What is clear is that under certain circumstances, expectancy can vary with the gender of the participant.

As evidenced by the main effect of expectancy for Question 9 (continuity of sleep), participant expectations about melatonin can influence subjective ratings of sleep qualities. As was initially predicted, participants reported more continuous sleep throughout the night when melatonin was the expected condition (regardless of the actual condition). Because there were no treatment effects found for this question (possibly due to the low power associated with this study; see Appendix for a discussion of power), the difference can be attributed to the instructional manipulation, which set up this specific expectation. The participants were told that melatonin has been found to produce a deeper and more continuous sleep in past studies (Attenburrow et al., 1996; Hughes & Badia, 1997; Rose et al., 1999; Waldhauser et al., 1990). In this regard, the design was successful in manipulating participants' ratings of the continuity of their sleep. This outcome corresponds well with results of studies on alcohol and caffeine, which have indicated a relationship between participant expectations and performance (Fillmore & Vogel-Sprott, 1994; Finnigan, Hammersley, & Millar, 1995). The finding also strengthens the argument that expectations may influence the perceived effects of melatonin. Although in this case an instructional manipulation was introduced into the design, other studies are not immune to the effects of expectancy. If the present study is any indication, those effects may be powerful enough to suggest an effect when none was observed.

Men reported more favorable postsleep experiences, greater ease of awakening (Question 7), and greater ease of getting out of bed (Question 8) than did women. The fact that scores on these questions were highly correlated (.94) signifies that they tap a similar construct. The gender differences cannot be attributed to any experimental manipulation and are not consistent with our initial predictions. Rather, these results provide evidence that men and women may have dissimilar postsleep experiences, even when their basic sleep patterns and sleep hygiene do not differ dramatically (see Table 1).[1] Investigators of sleep behavior may benefit by explicitly considering gender. However, as it was not the primary intent of this study to measure differences in sleep between men and women, further research is warranted.

[1]Analyses of actual sleep time (as determined by participants' sleep diaries) and number of awakenings did not reveal statistically significant differences between men and women.

The present study has several potential limitations. First, although the results are generally consistent with our predictions regarding how expectancies about melatonin would affect participants' subjective ratings of sleep qualities, the effectiveness of the expectancy manipulation could have been formally checked in the postexperimental interview. That is, we overlooked the opportunity to ask participants, prior to debriefing, exactly how they thought melatonin would affect their sleep. This study is also limited by the lack of physiological data. Polysomnographic measures could provide more information regarding how melatonin affects sleep as well as provide objective indices of sleep variables such as onset, efficiency, and duration. However, physiological data alone would also be limited in that physiological measures do not reveal how individuals feel following their sleep experience. Moreover, one of the strengths of the present study is that participants were sleeping in familiar surroundings and were not removed from their home environment. Studies requiring participants to relocate to a sleep laboratory run the risk of measuring sleeping patterns that are altered due to the change of environment. It should also be noted that those who do use melatonin in an attempt to alleviate sleep difficulties are presumably doing so in their own home. Thus, it is clear that future studies should incorporate both subjective and physiological measures into their designs. However, they must also consider very carefully the tradeoff between an experiment set in a sleep laboratory and a field experiment.

The present study could also have benefited from an increased sample size. For most of the questions, the power was relatively low. A larger sample could increase the power and improve on the ability of the study to detect differences when they exist. It is likely that our results underestimate the impact of both melatonin and expectancy, and perhaps of gender differences as well. Furthermore, to better determine the role of gender in participants' sleep experiences, researchers would have to recruit a sample with equal numbers of men and women. The present study did not have such a sample, because it was not initially predicted that gender differences would be important. Future researchers may wish to do so in light of the present findings.

The present study reaffirms the most commonly found effect of melatonin (its sleep-inducing properties), and it adds to the discussion of melatonin's influence on sleep qualities. At this time, individuals who experience difficulties initiating sleep onset would be among those most likely to benefit from the subjective feelings of grogginess/tiredness following melatonin treatment. Findings regarding both participant expectations and gender underline the importance of considering these variables when investigating melatonin and sleep in general. As people continue to consume melatonin in search of better sleep, it is clear that their own expectations may affect their ensuing experiences. Furthermore, the present study offers evidence that gender differences also affect participants' rat-

ings of their sleep. Thus, there is a need for further examination of both expectancy and gender to determine more clearly their relationship to the effects yielded by exogenous melatonin on individuals' sleep qualities.

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APPENDIX

There were several reasons for the decision to conduct univariate ANOVAs rather than a multivariate ANOVA (MANOVA) in light of the correlations among the dependent variables. First, a repeated measures design is already a special case of MANOVA because correlations among the measures are considered in the analysis (Bray & Maxwell, 1985; Hair et al., 1995). Second, when there are many dependent variables, as in the present study, the univariate tests may be more sensitive than a MANOVA (Hair et al., 1995). Third, the significant value of Hotelling's T^2 indicated that the questions on the DSSEQ measured different aspects of subjective sleep experience; thus, information (and power) would be lost if we either combined questions or took a multivariate approach. Fourth, the results of MANOVA are often difficult to interpret. Inevitably, a significant MANOVA requires post hoc, univariate F tests to clarify which variables are contributing to differences across conditions. Finally, MANOVA requires greater sample sizes than univariate ANOVAs to maintain the same power (Hair et al., 1995); power in the present study was already low, and further decreasing power offered no advantages.

Having resolved the advantages of a univariate over a multivariate strategy for the present analyses, we do need to address the issue of inflated Type I errors as a result of conducting 12 ANOVAs. Interestingly, to the extent that the dependent variables *were* correlated, there would be less inflation in the alpha than if the dependent variables were all uncorrelated (Hair et al., 1995). Nevertheless, we were unable to directly control the experimentwide Type I error rate in the present analyses, and we acknowledge the potential for increased Type I errors. However, in any study, there is a trade-off between Type I and Type II errors (see Keppel & Zedeck, 1989, for a discussion of this issue). As control over Type I errors is increased, typically by adopting a more conservative alpha level, power is decreased. In the present study, the power associated with our analyses was generally considerably lower than the recommended .80 (Hair et al., 1995). In light of how low power was, it did not make sense to further increase the likelihood of Type II errors by decreasing the alpha level to "correct" for the multiple univariate tests. As noted by Hair et al. (1995, p. 278), "The analyst must always be aware of the implications of adjusting the alpha level, because the overriding objective of the analysis is not only avoiding Type I errors but also identifying the treatment effects if they do exist." And though it was our intention to conform to the conventional alpha level of .05, we also felt it was worth noting that this alpha level is, indeed, a conventional and arbitrary cut-off for saying something "did" or "did not" occur (also see Kirk, 1982). Some statisticians, including Scheffé (1953), have suggested using .10 as the experimentwide alpha level (cited in Klockars & Sax, 1986). The present strategy regarding Type I and II errors was also adopted in view of the exploratory nature of the present study and the potential importance of discovering how treatment and expectancy effects interact with respect to melatonin and sleep experiences.

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