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A Multistudy Analysis of the Effects of Early Cocaine Abstinence on Sleep

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Abstract

Objective—To describe the sleep patterns of early cocaine abstinence in chronic users by polysomnographic and subjective measures.

Methods—28 cocaine-dependent participants (ages 24-55) underwent polysomnographic sleep (PSG) recording on the 1st, 2nd and 3rd weeks of abstinence on a research dedicated inpatient facility. Objective measures of total sleep time, total REM time, slow wave sleep, sleep efficiency and a subjective measure (sleep quality) along with demographic data were collected from three different long term research studies over a five year period. Data were reanalyzed to allow greater statistical power for comparisons.

Results—Progressive weeks of abstinence had main effects on all assessed PSG sleep measures showing decreased total sleep time, REM sleep, stage 1 and 2 sleep, and sleep efficiency; increases in sleep onset and REM latencies and a slight increase in slow-wave sleep time were also present. Total sleep time and slow wave sleep were negatively associated with years of cocaine use. Total sleep time was positively associated with the amount of current ethanol use. Sex differences were found with females having more total REM time and an increase at a near significance level in slow wave sleep. Subjective measures were reported as improving with increasing abstinence over the same time period.

Conclusions—Chronic cocaine users show a general deterioration in objective sleep measures over a three-week period despite an increase in subjective overall sleep quality providing further evidence for “occult insomnia” during early cocaine abstinence.

Keywords

Cocaine; sleep; polysomnograph; early abstinence

1.0 Introduction

Cocaine dependence remains a major medical and social problem. Compounding the difficulty for cocaine dependence is that there is no current FDA approved medicine for treatment despite numerous clinical trials of candidate medications. Thus individuals seeking help for their cocaine addiction are managed with counseling and social support when available but continue to have high relapse rates. An important strategy in finding an

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effective pharmacologic treatment for cocaine dependence is to consider the neurophysiologic adaptations associated with cocaine use and normalize them (Dackis and O'Brien, 2002). A recent and growing body of literature suggests the persistent sleep abnormalities related to chronic cocaine use may be a target for such treatment (Dackis and O'Brien, 2010; Morgan et al., 2010).

Early studies of cocaine withdrawal found that cocaine users had subjective reports of poor sleep and fatigue in the first few days of abstinence but those resolved within a week or two of abstinence (Gawin and Kleber, 1986; Weddington et al., 1990). Another large, subjective study that measured sleep in cocaine abstinence also found no persistent deficits (Coffey et al., 2000). Hence, sleep abnormalities did not appear to contribute substantially to long-term treatment outcome. However, small polysomnographic studies suggested that sleep disturbances worsened rather than improved over that same time period, with no evidence of stabilization (for review see Morgan and Malison, 2007). Consistent findings from these studies included decreased total sleep time, decreased sleep efficiency, and increased sleep onset latency. These studies were limited, however, by their small size and methodological and participant differences (Morgan and Malison, 2007; Valladares, 2007). Nevertheless, there appeared to be a substantial difference between objective measurements of sleep in cocaine dependent persons and how they experienced their sleep, with the possibility that significant abnormalities worsened as abstinence from cocaine progressed.

A more recent study addressed this apparent difference between objectively measured and subjectively experienced sleep quality in persons with cocaine dependence without co-morbid substance dependence (Morgan et al., 2006). This work suggested that subjective improvement and objective worsening of sleep were indeed concurrent, introducing the idea of an "occult insomnia", a functional sleep abnormality that is not recognized as such by the individual experiencing it.

Although this study (Morgan et al., 2006) was larger than prior studies and methodologically sound, the still relatively small sample size (N=12) was limiting, particularly as the contributions of important demographic and baseline variables like gender (Morgan et al., 2009) and quantity of cocaine and other substance use could not be considered. Such information is particularly relevant to the interpretation of a possible sleep-directed pharmacologic treatment for cocaine, as these variables may contribute substantially to variation in treatment response (e.g. co-morbid alcohol dependence; Anderson et al., 2009).

Here we present results from an analysis of objective and subjective sleep data from the largest sample of polysomnographically measured sleep in persons with cocaine dependence. To obtain this sample we combined data from three relatively homogenous polysomnographic studies of sleep during early abstinence from cocaine.

2.0 Methods

2.1 Participants

28 cocaine-dependent participants (ages 24-55) were actively recruited from the community for participation in three long-term inpatient research studies over a five-year period. Data from the individual studies has been published separately (Morgan et al., 2006, 2008a, 2010). To be included in the studies participants underwent clinical screening including unstructured clinical interviews, physical examination, and laboratory tests as part of their admission to the research unit. Participants also had positive urine tests for cocaine metabolites on pre-admission screening and the admission day (while being negative for cannabis, opiates, PCP, amphetamines, benzodiazepines, and barbiturates). Participants with verbal history of ETOH and cannabis use were permitted although no participants exhibited

alcohol withdrawal symptoms during the study as determined by the Clinical Institute Withdrawal Assessment for Alcohol (CIWA).

Participants were excluded for a history of dependence on alcohol, cannabis, or other addictive substances except cocaine and nicotine, and for any non-substance related psychiatric co-morbidity or major medical conditions (including diabetes, cardiovascular disease, liver disease, HIV-seropositivity, and neurological conditions). In addition no participants reported having been evaluated, diagnosed, or treated for any sleeping problem including sleep apnea, restless leg syndrome, periodic leg movements during sleep, or somnambulism. All participants were free from prescribed psychoactive medications in the past year. The local institutional review board approved all studies and participants gave written informed consent prior to enrollment.

2.2 Inpatient facility

Participants were admitted to a 12-bed state psychiatric research facility. The unit includes a structured daily routine and participants participated in all activities (including group and individual therapy) except for substance abuse treatment. All meals and snacks were provided caffeine-free and four times per day. Fifteen minute 'fresh-air' breaks were scheduled to allow for smoking. All participants were checked by staff at least once every 15 minutes while on the unit, and were monitored constantly if off the unit for protocol related testing or 'fresh-air' breaks. Daytime napping was not permitted and was enforced strictly by unit staff. Urine toxicology screens were administered three times per week and were negative with the exception of positive tests for cocaine consistent with laboratory cocaine administration.

2.3 Sleep Measurement

PSG measurement was performed using a TEMEC Universal8 polysomnograph or Grass Instruments Colleague system in a dedicated sleep laboratory room, and included at a minimum two EEG channels (C3/A2, C4/A1), right and left EOG, chin EMG, and ECG. PSG records were scored according to accepted criteria (Iber et al., 2007) while blinded to participant and study night. All participants had a minimum of 8 hours in bed each night. Reported sleep variables were defined using standard criteria: sleep onset latency was defined as the time from lights out until the appearance of the first epoch of sleep; REM latency was the time from sleep onset to the first epoch of REM sleep; total sleep time was defined as the time from sleep onset until final awakening minus the time awake after sleep onset. The time spent in stages N1-2 and in REM sleep was included and N3 (formerly stage 3 and 4 sleep) is described as slow wave sleep. Sleep efficiency was defined as the ratio of time spent asleep (total sleep time) to the amount of time spent in bed.

In each of the studies from which data is reported, PSG measurement was performed multiple times over the course of three weeks of abstinence. Data reported here reflects the mean values for all sleep measurements during each week of abstinence. The main between study differences were in the administration of medication (tiagabine, lorazapam or modafinil) or placebo. To help minimize these differences, only data from placebo nights (Morgan et al., 2008a) or from participants randomized to receive placebo (Morgan et al., 2010) or no medication (Morgan et al., 2006) were included in the analysis. In the case of the study in which all participants received study medication (Morgan et al., 2008a), the data from the first and second week of abstinence reflected data prior to the experimental medication being given, and the data from the third week of abstinence occurred 5 days after the most recent medication dose. All data reported here was obtained following a minimum of 3 nights of prior accommodation to the sleep laboratory and PSG measurement.

Subjective measures of sleep quality and alertness were assessed every morning and daytime alertness was assessed every evening using the Sleep Quality Questionnaire (SQQ) (Pace-Schott et al., 2005). Upon awakening, participants rated their "overall quality of sleep," "depth of sleep," "morning alertness" and "feeling well-rested" based on a visual analog scale (VAS) ranging from 1-100. In the evening just prior to going to sleep, participants retrospectively rated their "level of alertness today" using the same VAS. All objective and subjective measures obtained from days 1-7, 8-14, and 15-21 herein correspond to abstinence weeks 1, 2, and 3.

2.4 Data analysis

All outcomes were tested for normality using Kolmogorov-Smirnov test statistics and normal probability plots. Sleep latency and slow wave sleep were approximately normal after log-transformation. Linear mixed models were developed to assess each sleep outcome across abstinence week 1 through week 3 (within-subjects factor). We considered each study as a between subjects factor to test for interactions with abstinence, but none were present and thus study was dropped as a factor. Tukey's multiple comparisons procedure was used to test pair-wise differences between periods of abstinence. Covariates including gender, age, years of cocaine use, whether seeking treatment, race, amount of cocaine use per week, amount of ethanol per week, and marijuana use per week were also considered one at a time in the above models. Results were considered significant at $p<.05$. All data was analyzed using SAS, version 9.1 (Cary, NC)

3.0 Results

3.1 Demographics

Demographic data for the 28 cocaine dependent participants separated by original study are given in Table 1. Of the 28, 19 (68%) were African-American and 9 (32%) were Caucasian. None were Hispanic. The majority of subjects were males (n=23, 82%) with a mean age of 39±8 [S.D.] years. Lifetime cocaine use was chronic and severe in regards to years used (17±8) and current use (8±7 grams per week). Cannabis use was negligible in this study with a mean of 1 joint smoked in the last three months. Alcohol use was significant in a subset of the sample with six subjects drinking more then 20 drinks per week.

3.2 Polysomnographic Sleep Data

Polysomnographic sleep data are shown in Figures 1, 2 and 3. Between study differences were only significant in sleep efficiency and sleep latency measures. There were main effects of abstinence week on all assessed PSG sleep measures: total sleep time ($F[2,54]=13.4$; $p=<0.0001$), REM sleep ($F[2,54]= 13.6$; $p=<.0001$), slow wave sleep ($f[2,54]= 3.4$; $p=0.04$; stage 1 and 2 sleep ($F[2,54]=7.4$; $p=0.001$); sleep efficiency ($F[2,54]=13.7$; $p=0.001$); sleep latency ($F[2,54]=10.0$; $p=0.002$); and REM latency ($F[2,54]=11.21$; $p=0.001$).

Post-hoc tests revealed significant decreases from abstinence week 1 to week 3 in total sleep time ($t[54]=5.17$, $p<0.001$), REM sleep time ($t[54]=5.08$, $p<0.001$), stage 1 and 2 sleep ($t[54]=3.82$, $p<0.001$), and sleep efficiency ($t[54]=5.0$, $p<0.001$). Latencies in both sleep onset ($t[54]=-4.48$, $p<0.001$) and REM ($t[54]=4.66$, $p<0.001$) showed increases. There was also an increase in slow wave sleep ($t[54]=-2.37$, $p<0.05$) shown over the same period.

3.3 Subjective Sleep Quality Measurement

Figure 4 depicts results of the SQQ. All subjective measures of sleep improved from week 1 to week 3. Quality of sleep improved with abstinence ($F[2,52]=9.4$, $p=0.0003$), particularly from week 1 to week 3 ($t[52]=-4.27$, adj. $p=0.0002$).

3.4 Covariate analysis

Total sleep time was negatively associated with years of cocaine ($F[1,26]=6.74$, $p=0.015$) and, at a trend level, marijuana use per week ($F[1,26]=3.23$, $p=0.08$). A positive association was seen with the amount of ethanol use per week ($F[1,26]=4.4$, $p=0.05$). However, adjustment for these associations did not affect the observed effects of abstinence. At the near significance level females showed greater sleep time compared to males ($F[1,26]=3.92$, $p=0.06$). No significant findings were seen with treatment seeking individuals, race, amount of cocaine use per week, or age in regards to total sleep time.

REM sleep time was greater in females ($F[1,26]=6.19$, $p=0.02$). Slow wave sleep time was negatively associated with years of cocaine ($F[1,26]=7.49$, $p=0.011$). In addition, slow wave sleep also had a negative association with age ($F[1,26]=11.97$, $p=0.0019$). No other associations were seen with treatment seeking individuals, race, amount of cocaine use per week, amount of ethanol per week, or marijuana use per week in regards to total REM time or slow wave sleep.

No associations between covariates and subjective sleep quality were observed.

4.0 Discussion

Our results confirm previous findings of significant deterioration in polysomnographically measured sleep across 3 weeks of abstinence in chronic cocaine users. In addition, by combining data from small but methodologically similar studies, we were able show effects of years of cocaine use, amount of alcohol use, age, and gender on polysomnographically measured sleep variables.

Consistent with prior polysomnographic studies of sleep in cocaine users (Thompson,1995; Gillian, 1994), we found deteriorations in total sleep time, REM sleep time, stage 1 and 2 sleep time, and sleep efficiency, as well as increases in sleep onset and REM onset latencies from the first to the third week of abstinence. In contrast to polysomnographic measures of sleep, subjective sleep measures improved over the same time period. This intriguing though not unexpected (Morgan et al., 2006) finding provides further evidence supporting the notion of an “occult insomnia” in early abstinence in chronic cocaine users. It has been hypothesized that the juxtaposed worsening PSG measures and improved self reported measures could be due to a dysregulation of the homeostatic sleep drive (Morgan et al., 2006).

Although prior studies were not sufficiently powered to detect changes in slow-wave sleep time over abstinence, we found that slow-wave sleep time, like slow-wave activity (Morgan et al., 2006), increased over three weeks of abstinence. Although this increase was modest, it occurred despite the decrease in total sleep time. Slow wave sleep, typically increased following sleep deprivation and associated with “deep” sleep, has been found to correlate positively with self-reports of sleep-quality in insomniacs (Krystal et al., 2002). This finding may generalize to the chronic cocaine population and also be associated with the sensation of better quality sleep later in abstinence. However, slow-wave sleep in cocaine dependence appears to be significantly less than in healthy persons even after 2-3 weeks of abstinence (Morgan et al., 2010). Hence, the modest improvement in slow-wave sleep time over this period does not reflect a normalization of slow-wave sleep time. Although further improvements may occur, in alcohol dependence populations normalization of slow-wave sleep time may take up to 6 months or more (Ishibashi et al., 1987; Williams et al., 1981).

The initial REM latencies were shortened in early cocaine abstinence when compared to the normal 90 minutes latency seen in healthy volunteers (Pollack et al., 1999). While this result

was not found when cocaine dependent participants were compared directly to healthy controls in a previous study (Morgan et al., 2010), a likely explanation for the current result is the larger sample size of this study. Furthermore, other studies have found a similar effect of shortened REM latency in cocaine users (as well as other substance abusers) when examined in the first week of abstinence (Bolla et al., 2008; Morgan et al., 2008b). This phenomenon could stem from an initial rebound from cocaine induced REM suppression. As abstinence progresses our results showed an increasing REM latency and, while it did not move into the normal range after three weeks, we would predict that it would continue to trend in that direction.

The observed effect of years of cocaine use on sleep – decreased sleep time associated with longer use – supports the hypothesis that chronic cocaine use itself contributes to some of the observed sleep deficits. However, it is also possible that poor sleep contributes to drug use (e.g. Mednick et al., 2010; Roehrs et al., 2004); the association of longer periods of use with greater sleep deficits does not rule out this possibility, as longer periods of use may themselves reflect factors that contribute to an increased baseline risk for cocaine dependence. In addition to years of cocaine use, recent quantity of alcohol use affected sleep. In particular, greater use of alcohol was associated with higher total sleep times. In a comparison of sleep in alcohol and stimulant users during 2 weeks of abstinence (Thompson et al., 1995), total sleep time decreased in stimulant users but increased in alcohol users. Hence, the present findings may reflect an effect of abstinence from alcohol that is present despite or in addition to the effect of abstinence from cocaine.

Intriguingly, we did not find an effect of quantity of recent cocaine use on any of the tested sleep measures. Although such an effect is not ruled-out (i.e. the studies were not designed to test this rigorously), the lack of an observed effect lends credence to the notion that the sleep deficits reflect abstinence from chronic use more than withdrawal from current use. That the deficits develop over weeks of abstinence and are positively associated with number of years of use also support this idea.

As anticipated, age was a significant factor in slow-wave sleep, with increased age associated with decreased slow-wave sleep time. Age was not a contributing factor, however, in any other measured outcome despite known age related effects in sleep with healthy controls. This was most likely due to the relatively narrow age range of participants in this study that would make it unlikely to find other age-related differences (Williams et al., 1974).

Despite having only five females in this sample, gender related differences were found with females having longer total sleep time and REM sleep time compared to their male counterparts. This is the first polysomnographic evidence of a gender difference in sleep in cocaine dependence and is consistent with non-polysomnographic findings (Morgan et al., 2009). While this finding should be confirmed in a larger sample, such differences, if real, could be significant in determining future sleep-related treatment options.

This study employed a multistudy design and attempts were taken to limit the cohort effects including strict inclusion criteria, similar protocols and equipment, and the same outcome measures. That withstanding, weaknesses inherit in a multistudy design should give caution when generalizing these results because of the heterogeneity of combining studies.

In addition, it was beyond the scope of the current study to measure cognitive changes associated with sleep changes. This remains an open and important question because of the links of cognitive difficulties to worse treatment outcomes in cocaine dependent individuals (Teichner et al., 2001; Aharonovich et al., 2006). A multistudy analysis by Pace-Schott et al. in 2008 did offer insight into cognitive findings during early cocaine abstinence by finding a

decrease in attentional and learning capacity over the weeks of abstinence observed. In addition, Morgan in 2006 also found sleep-related cognitive disruptions in early abstinence. This evidence lends weight to the idea that learning deficits in chronic cocaine users are similar to the well-established differences in sleep-dependent learning in healthy subjects.

The present work has further clarified sleep disturbances in early cocaine abstinence. If and when these disturbances normalize remains an interesting query that warrants further studies and could help elucidate the importance of sleep on cocaine dependence and relapse.

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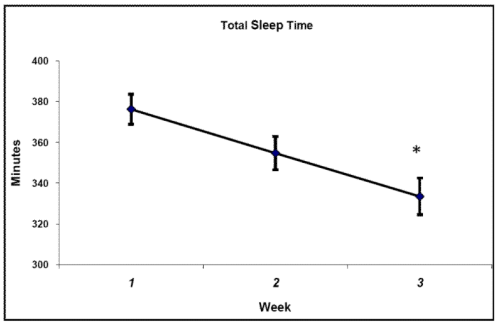

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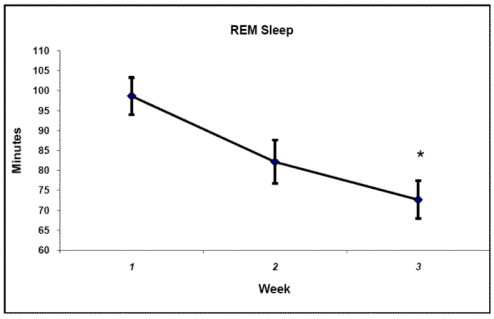

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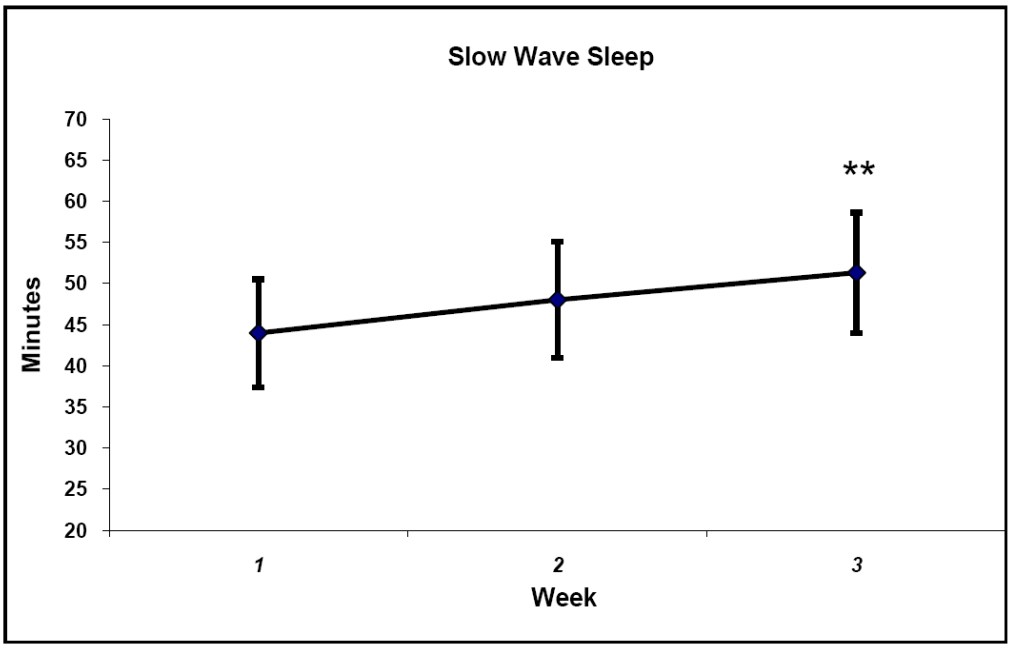

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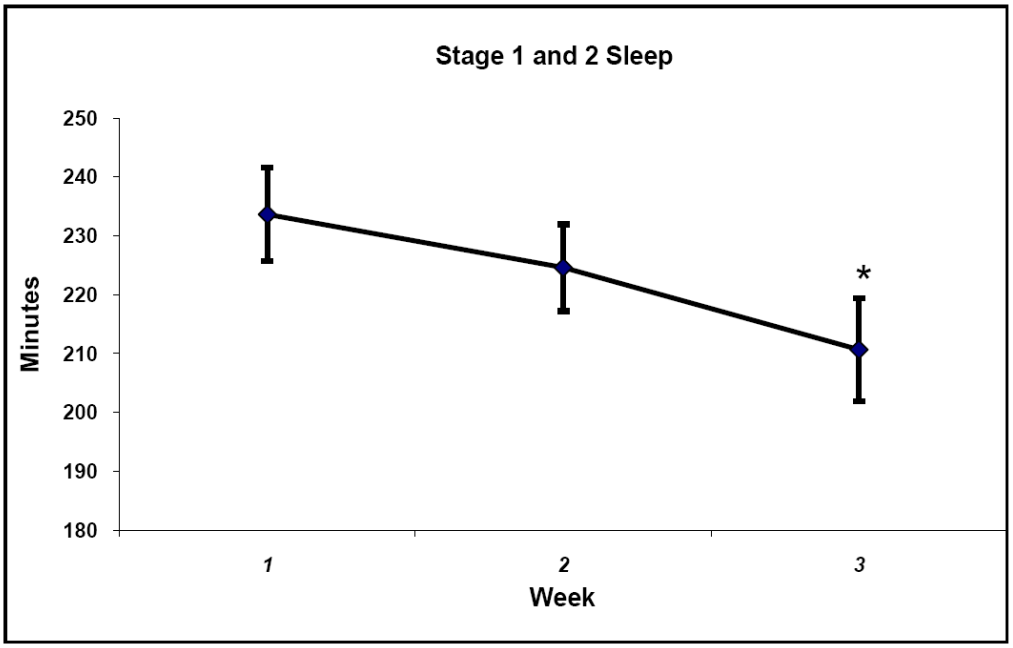

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Figure 1.

(a,b,c,d): Total Sleep Time, REM Sleep, Slow Wave Sleep, and Stage 1 and 2 Sleep (in minutes) at 1, 2, and 3 weeks of abstinence in chronic cocaine users. All error bars denote standard error. Main effects of abstinence week were present on all measures (see text). * denotes significant differences between week 1 and 3 for total sleep time, REM Sleep and sleep stages 1 and 2 ($p<0.001$) ** denotes significant differences between weeks 1 and 3 for slow wave sleep at the $p<.05$ level. Other significant differences were found between weeks 1 and 2 and weeks 2 and 3 in Total Sleep Time and REM Sleep at $p<.05$ or above.

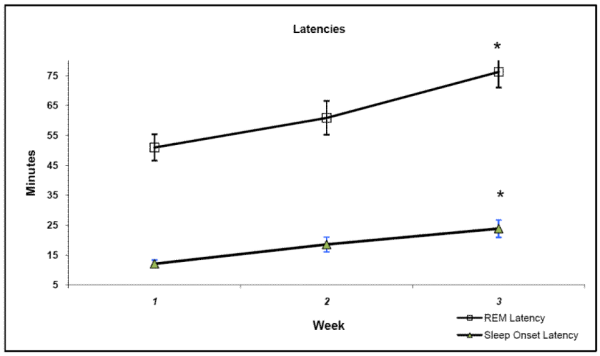

Figure 2.

REM and Sleep Onset Latency are both shown (in minutes) at 1, 2, and 3 weeks of abstinence in chronic cocaine users. All error bars denote standard error. * denotes increased latencies between weeks 1 and 3 that were significant at the $p<0.001$ level. Significant differences were also found at week 2 and 3 in REM latency at the $p<0.002$ level.

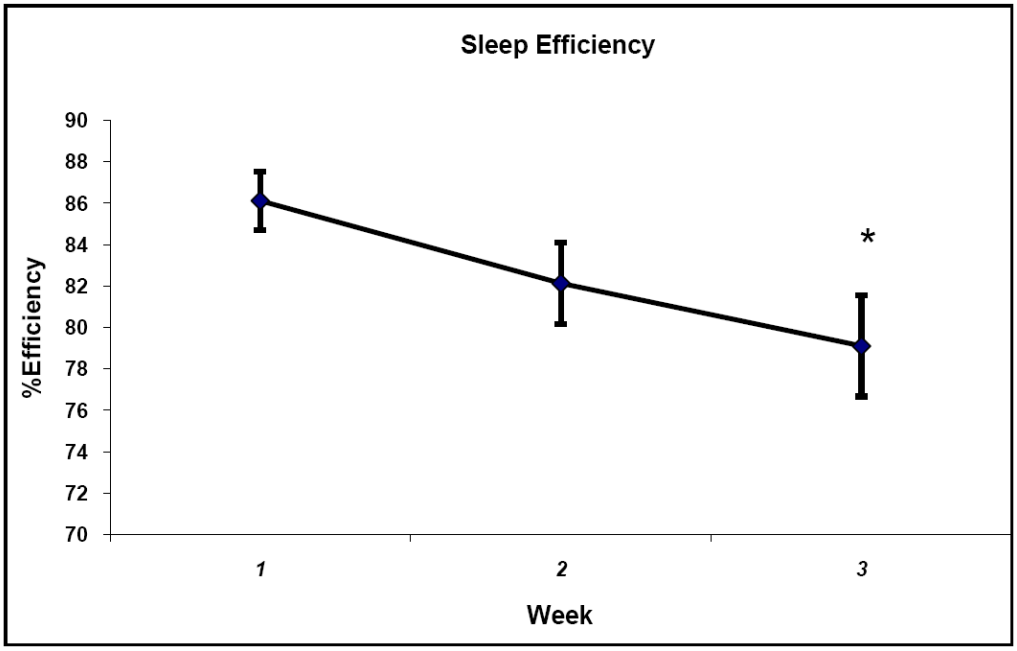

Figure 3.

Sleep efficiency (percentage of time sleeping while in bed) is shown above at 1, 2, and 3 weeks of abstinence in chronic cocaine users. All error bars denote standard error. * denotes significant differences at week 1 and 3 ($p < .001$). Significant differences were also found at weeks 1 and 2 and week 2 and 3 at $p < .05$ or above.

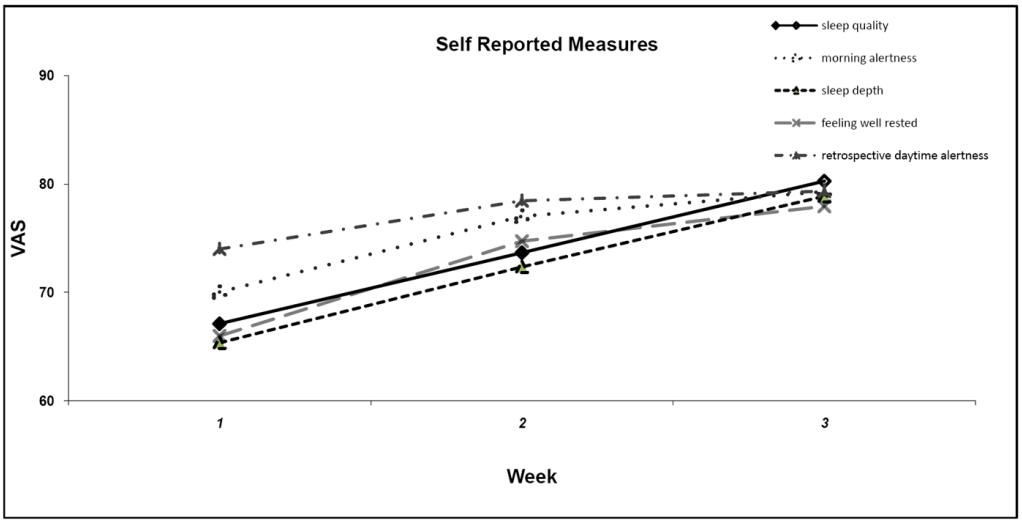

Figure 4.
Subjective measures from the visual analog scale (VAS) at 1, 2, and 3 weeks of abstinence in chronic cocaine users. Significant differences between week 1 and 3 for sleep quality, morning alertness, sleep depth, and feeling well rested were found at the P<.05 or above.

Table 1

Participant demographics with mean and standard deviation.

Study	Number of subjects	Age (years)	Race (African-American/Caucasian)	Years of cocaine use	Current weekly cocaine avg (g)	Current weekly alcohol use (drinks)	Cannabis use in last 90 days (joints)	Currently Seeking Treatment
2006	12 (10 males, 2 females)	39±7	9 AA 3 EA	17±7	7±4	5±8	1±2	0
2008	6 (4 males, 2 females)	37±9	4 AA 2 EA	14±9	15±10	18±17	2±2	0
2010	10, (9 males, 1 female)	42±9	6 AA 4 EA	19±9	4±3	10±11	0±1	8
Total	28 (23 males, 5 females)	39±8	19 AA 9 EA	17±8	8±7	9±12	1±2	8