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Research report

Comparison of sleep/wake parameters for self-monitoring bipolar disorder

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ABSTRACT

Background: Psychosocial interventions may teach patients with bipolar disorder to successfully detect warning signs of relapse. These interventions often include ongoing self-monitoring of sleep. We previously reported that a change in sleep duration (sleep plus bedrest) of >3 h may indicate that a mood change is imminent. This analysis further investigated whether sleep duration, sleep onset or sleep offset was the most useful sleep/wake parameter to monitor for an oncoming mood change.
Methods: 101 adult outpatients receiving treatment as usual recorded mood, sleep and medications every day on a home computer for a mean of 265 ± 103 days. A daily time series of mood, sleep duration (sleep plus bedrest), sleep onset and sleep offset was constructed for each patient. After applying an ARIMA (0,1,1) filter, a cross correlation function was used to analyze the temporal relationship between the residuals for lags of ±7 days.
Results: Less frequent significant correlations were found between a change in either sleep onset or sleep offset and mood, than between sleep duration and mood. Patients with a significant correlation between sleep duration and mood included 86% of those with a significant correlation between sleep onset or sleep offset and mood. Mean sleep duration when euthymic was long (≥8 h in 89% of patients, ≥9 h in 51% of patients).
Limitations: Self-reported data, naturalistic study, and computer access required.
Conclusions: Self-monitoring of sleep duration is recommended for patients with bipolar disorder. Better understanding of the long sleep duration of euthymic patients is required.

1. Introduction

Psychosocial interventions that focus on recognition of warning symptoms, stabilization of activity patterns, and increasing medication adherence may improve the course of illness for patients with bipolar disorder (Beynon et al., 2008; Morriss et al., 2007; Rouget and Aubry, 2007). Sleep disturbances are a frequent prodromal sign of both mania and depression (Molnar et al., 1988; Jackson et al., 2003), and can

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Table 1
Demographics by patients with and without significant negative cross-correlations using sleep duration.

	All patients ($N=101$)	Cross-correlation		Test	Value	p
		Not significant ($N=59$)	Significant ($N=42$)			
Age of onset	22.1	21.7	22.63	t	−.430	.668
Hospitalizations	2.1	2.4	1.6	t	1.523	.131
Age	37.8	36.9	39	t	−.990	.325
Mean daily medications	3.2	3.0	3.4	t	.199	.199
Years of illness	15.7	15.1	16.5	t	−.621	.537
Disabled [a], n (%)	17 (16.8%)	6 (35.3%)	11(64.7%)	X^2	4.499	.034
Male:Female	34:67	18:41	16:26	X^2	.632	.426
BP I:BP II	64:37	38:21	26:16	X^2	.066	.797

[a] Received government disability payments due to bipolar disorder.

be easily identified by patients or family members (Lam et al., 2001). Since early recognition of recurrence requires persistent self-monitoring, it is important to understand which sleep/wake parameter is the most effective indicator of an upcoming mood change in bipolar disorder. We previously found that a change in sleep duration (sleep plus bedrest) of more than 3 h is a late prodromal sign, signifying that a large mood change is imminent, and that it was more useful to monitor sleep duration than either bedrest, or sleep without bedrest (Bauer et al., 2006, 2008a). In this analysis, we have further compared sleep onset and sleep offset with sleep duration to determine the most useful variable to monitor for an oncoming mood change.

2. Methods

Data were collected for an analysis of the temporal relation between sleep duration and mood in patients with a DSM-IV diagnosis of bipolar disorder, following the protocol described previously (Bauer et al., 2006, 2008a). The diagnosis of bipolar disorder was made during a clinical interview by the prescribing psychiatrist. The study included 101 outpatients aged 18 years or older who received pharmaceutical treatment as usual throughout the study. See Table 1 for the demographic characteristics, and Table 2 for medications taken throughout the study. At least 100 days of data with 2 or less sequential days of missing data was required from each patient. The 101 patients returned a mean of 265 ± 103 days of data. All patients were volunteers, and provided written informed consent.

2.1. Data collection

Every day, the patients recorded mood, sleep, and medications taken using the previously validated ChronoRecord software in their native language on a home computer (Bauer et al., 2004, 2008b). The patient recorded mood using a 100-unit visual analogue scale between the extremes of mania and depression that the patient ever experienced. A mood entry less than 40 was considered depression, a rating from 40 to 60 was considered euthymia, and greater than 60 hypomania/mania. Each daily mood rating was categorized accordingly.

The patient recorded sleep by selecting one of 3 icons, awake, asleep or bedrest, with awake as the default, for each hour in a 24-hour period. Bedrest was defined as the patient being in bed trying to sleep but awake. In relation to objective measurement by actigraphy, some patients with bipolar disorder overestimate the time it takes to fall asleep and underestimate the total time spent sleeping (Harvey et al., 2005). Therefore, to minimize the potential impact of self-reporting errors, sleep duration was defined as the time asleep plus bedrest.

2.2. Time series and cross-correlation function (CCF) analysis

The CCF methodology was described previously (Bauer et al., 2006) and is summarized here. A daily time series of self-reported mood, sleep duration, sleep onset and sleep offset were developed and analyzed for each patient. The CCF was used to evaluate the linear relationship between two time series as the paired values were separated across a range

Table 2
Medications taken [a] by patients with and without significant negative cross-correlations using sleep duration.

	All patients ($N=101$)	Cross-Correlation		Test	Value	p
		Not significant ($N=59$)	Significant ($N=42$)			
Benzodiazepines (n, % taking)	22 (21.8%)	8 (36.4%)	14 (63.6%)	X^2	5.631	.018
Antidepressants [b]	48 (47.5%)	29 (60.4%)	19 (39.6%)	X^2	.151	.698
Antipsychotics [c]	42 (41.6%)	26 (61.9%)	16 (38.1%)	X^2	.360	.548
Lamotrigine	40 (39.6%)	19 (47.5%)	21 (52.5%)	X^2	3.249	.071
Lithium	30 (29.7%)	17 (56.7%)	13 (43.3%)	X^2	.054	.817
Valproate	20 (19.8%)	15 (75.0%)	5 (25.0%)	X^2	2.824	.093
Non-benzodiazepine sedative-hypnotics [d]	19 (18.8%)	10 (52.6%)	9 (47.4%)	X^2	.322	.570
Thyroid (T4 or T3)	16 (15.8%)	7 (43.8%)	9 (56.3%)	X^2	1.683	.194
Estrogens	12 (11.9%)	8 (66.7%)	4 (33.3%)	X^2	.382	.537
Gabapentin	11 (10.9%)	6 (54.5%)	5 (45.5%)	X^2	.076	.783
Carbamazepine/oxcarbazepine	10 (9.9%)	6 (60%)	4 (40%)	X^2	.011	.915
Topiramate	7 (6.9%)	4 (57.1%)	3 (42.9%)	X^2	.005	.944

[a] Patient took medication at least 50% of days.
[b] 1 patient took a tricyclic; 2 took tranylcypromine; all others took newer antidepressants.
[c] 6 patients took typical antipsychotics; all others took atypical.
[d] Eszopiclone, ramelteon, zaleplon, zolpidem, amitriptyline, doxepin, mirtazapine, trazadone.

of time periods. Since serial trends in the data can produce false CCF results, each time series for each patient was first passed through the Auto-Regressive Integrated Moving Average (ARIMA 0,1,1) linear filter. After filtering, the CCF estimated the relationship in the residuals between sleep duration on one day and mood on another day, for time offsets of ±7 days. This analysis was repeated for sleep onset and mood, and sleep offset and mood. The results of the CCF

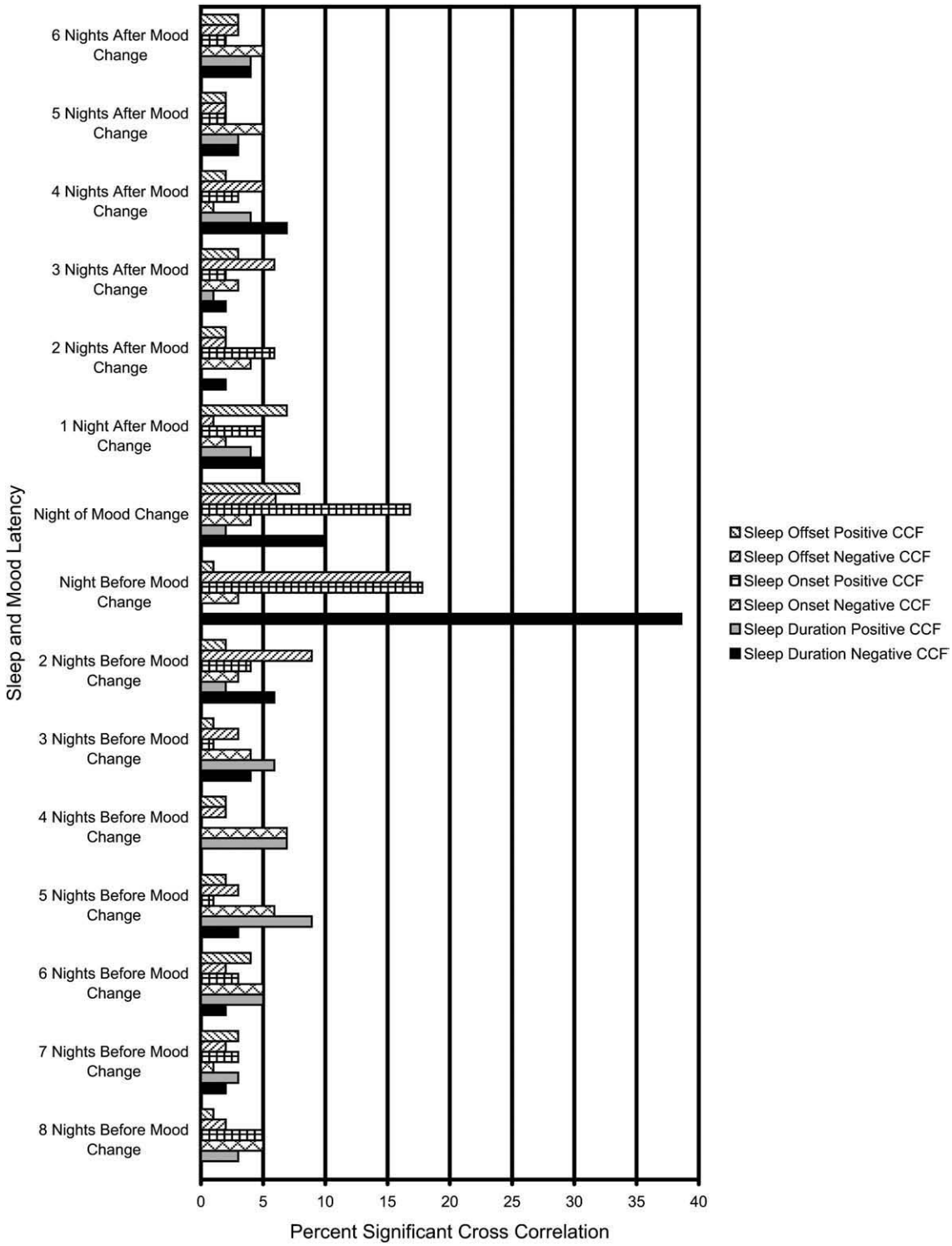

Fig. 1. Percent significant cross-correlations for all patients ($n = 101$): sleep duration, sleep onset and sleep offset with mood.

were summarized for all patients. A CCF value greater than twice the standard error indicated the correlation was statistically significant from zero.

2.3. Statistical analyses

The demographic characteristics, medications taken, and sleep patterns of the patients were compared using the Pearson 2-sided X^2 test for distributions, and the independent sample 2-sided *t*-test for mean values. To analyze for circadian sleep disturbances, double raster plots for each day of sleep for each patient were evaluated. A *p* value of less than 0.05 was considered statistically significant for all 2-sided tests. SPSS 14.0 was used for all time series and statistical calculations.

3. Results

3.1. Cross-correlation function results

The results of the CCF for all patients are shown in Fig. 1. Sleep duration and mood were significantly negatively correlated in 42 of the 101 patients (42%), on either the day before or the day of a mood change (Bauer et al., 2008a). With a negative cross-correlation, a decrease in sleep duration is accompanied by a mood change towards hypomania/mania and visa versa. With a one-day latency the change in sleep duration occurred the night before the change in mood. On the day before a mood change, sleep duration and mood were negatively correlated in 39 patients (39%), not correlated in 62 patients (61%), and there were no positive correlations. When there was no time lag between sleep duration and mood, sleep duration and mood were negatively correlated in ten patients (10%), not correlated in 89 patients (88%), and positively correlated in two patients (2%).

Sleep onset and mood were significantly positively correlated in 28 of the 101 patients (28%), on the day of or the day before the mood change. At zero offset between sleep onset and mood, 17 of these patients (17%) were positively correlated, 80 patients (79%) showed no significant cross-correlation, and 4 patients (4%) were negatively correlated. On the day before the mood change, sleep onset and mood were positively correlated in 18 patients (18%), not correlated in 80 patients (79%) and negatively correlated in 3 patients (3%). In this case the mood change preceded the sleep change by one day.

Sleep offset and mood were significantly negatively correlated in 21 of the 101 patients (21%), on the day of, day before, or 2 days before the mood change. On the day before the mood change, sleep offset and mood were negatively correlated in 17 patients (17%), not correlated in 83 patients (82%), and positively correlated in one patient (1%). When there was a 2-day time lag, sleep offset and mood was negatively correlated in 9 patients (9%), not correlated in 90 patients (89%), and positively correlated in 2 patients (2%).

Only 42 of the 101 patients showed significant cross-correlations between sleep duration and mood. However, 11 of the 101 patients reported little variation in mood, having a daily mood rating in the normal range for greater than 98% of the study days. None of these 11 patients showed any significant cross-correlations between sleep duration, sleep onset, or sleep offset and mood. The 42 patients with a significant negative cross-correlation between mood and sleep duration included 24 of the 28 (86%) patients with a significant cross-correlation between sleep onset and mood, and 18 of the 21 (86%) patients with a significant cross-correlation between sleep offset and mood.

Table 3
Distribution of mean euthymic sleep duration for patients with and without significant negative cross-correlations using sleep duration.

Mean hours of sleep [a]	Cross-correlation		Total
	Not significant	Significant	
≤7	5 (45.5%)	6 (54.5%)	11 (11.0%)
8	22 (57.9%)	16 (42.1%)	38 (38.0%)
9	23 (74.2%)	8 (25.8%)	31 (31.0%)
≥10	9 (45.0%)	11 (55.0%)	20 (20.0%)
Total [b]	59 (59.0%)	41 (41.0%)	100 (100.0%)

$X^2 = 5.432$, $df = 3$, $p = .143$.
[a] Rounded mean, e.g., 8 = 7.50 to 8.49 h.
[b] One patient had no euthymic days.

3.2. Sleep pattern results

When only considering days with euthymic mood, the mean sleep duration for all patients was 8.75 h, 8.69 h for those with a significant cross-correlation between sleep duration and mood versus 8.84 h for those without. The distribution of sleep duration when euthymic was not significantly different between those with or without a significant cross-correlation, as shown in Table 3.

None of the patients displayed a circadian sleep disturbance that lasted throughout the study period. For a length of 1–3 weeks, 10 patients displayed irregular sleep wake syndrome and 2 patients displayed a non-24 hour sleep wake cycle. Of these 12 patients with evidence of circadian rhythm disruption for short time periods, 6 patients had a significant cross-correlation between mood and sleep duration.

4. Discussion

Ongoing self-monitoring is an integral component of psychosocial interventions for coping with bipolar disorder, so it is important to optimize what patients are instructed to track. This analysis investigated sleep/wake parameters and found that sleep duration was more useful than sleep onset or sleep offset to monitor for an oncoming large mood change. Additionally, the patients with a significant cross-correlation between sleep duration and mood included 86% of those who had a significant cross-correlation with either sleep onset or sleep offset and mood. Changes to sleep onset and sleep offset of less than 1 h can also be due to weekend effects, which are observed in normal volunteers as well (Monk et al., 2000; Bauer et al., 2006), and complicate the analysis of these parameters. Moreover, in a study of 54 healthy working adults, personality characteristics were associated with both and sleep onset and sleep offset, but not with sleep duration (Soehner et al., 2007).

We have previously shown that monitoring sleep duration is clinically significant since a change in sleep duration

of greater than 3 h may signify a large mood change on the next day, and since patients with a significant cross-correlation between sleep duration and mood experienced the majority of all large changes in both mood and sleep, and larger changes from euthymic sleep duration when depressed or manic (Bauer et al., 2006, 2008a). However, patients differed in their susceptibility to the impact of sleep changes on mood, as one-third of these large changes in sleep duration occurred in those without a cross-correlation. In normal subjects, there are large differences in vulnerability to neurocognitive impairment after sleep loss and this individual variability is a stable, reproducible trait (Van Dongen et al., 2004; Leproult et al., 2003). It is not clear if the differential vulnerability to sleep changes in bipolar disorder reflects a trait (Lenox et al., 2002).

The patients in this study were long sleepers, with a mean sleep duration when euthymic of 8.75 h. However, methodological differences among the studies reporting sleep duration should be noted (Grandner and Drummond, 2007). Unlike this longitudinal study, most studies have a cross-sectional design. A variety of self-reporting instruments are used to obtain data. Sleep duration is defined inconsistently, as time asleep at night, or as time asleep plus in bed awake at night, or as total time asleep across 24 h. The cutoff to denote long sleep duration also varies, generally from >8 h (Yaggi et al., 2006; Hall et al., 2008) to ≥9 h (Patel et al., 2006; Gruber et al., 2009). In studies of bipolar disorder, some researchers only analyze data from patients with euthymic mood (Millar et al., 2004; Harvey et al., 2005), while others include data from patients in any mood state (Gruber et al., 2009). Nevertheless, the long sleep duration noted here is consistent with prior findings from patients with bipolar disorder who were euthymic (Millar et al., 2004; Salvatore et al., 2008). In contrast, the mean sleep duration for adults in the US is between 6 and 7 h (Kripke et al., 2002; NSF, 2005). In a study of over one million adults aged 30–102 years who were asked "On the average, how many hours do you sleep each night," only 7.7% slept 9 or more hours each night (Kripke et al., 2002) as contrasted with 51% of the patients in this study. Moreover, when compared to objective measurement, self-reported sleep duration was overestimated by normal, middle-aged adults (Lauderdale et al., 2006; Walsleben et al., 2004) but underestimated by patients with bipolar disorder (Harvey et al., 2005) or insomnia (Mercer et al., 2002, Harvey et al., 2005), as adjusted for in our definition of sleep duration. The long sleep duration noted in this analysis is of concern because multiple epidemiological studies have associated long sleep duration with an increased risk of morbidity and mortality although causality was not proven, and depression, low socioeconomic status, sleep disorders, and medical comorbidities may contribute to the statistical relationship (Qureshi et al., 1997; Kripke et al., 2002; Ayas et al., 2003; Patel et al., 2006; Yaggi et al., 2006; Grandner and Drummond, 2007; Hall et al., 2008).

Multiple factors may contribute to the long sleep duration in euthymic bipolar patients. Evidence from twin studies, and family-based genetic linkage studies suggests there may be significant heritability in habitual sleep duration (Heath et al., 1990; Partinen et al., 1983; Linkowski, 1999; de Castro, 2002; Gottlieb et al., 2007). Furthermore, individual variation in the circadian pacemaker may result in a longer biological night in long sleepers than in short sleepers (Aeschbach et al., 2003). In a study of 57 sighted patients with circadian sleep disorders, about half of whom had psychiatric illness, the mean sleep duration was 9.3 h (Hayakawa et al., 2005). Psychotropic medications may also contribute to the long sleep duration of the patients in this study. Mood stabilizers, antidepressants, antipsychotics, and benzodiazepines may produce strong effects on sleep, both detrimental and beneficial, as reviewed in detail elsewhere (DeMartinis and Winokur 2007; Wilson and Argyropoulos, 2005). However, studies of the impact of psychotropic drugs on the sleep architecture and the sleep/wake cycle of patients with bipolar disorder are rare. There is a need to investigate the clinical relevance of differences among these medications, as well as the potentially confounding effect of vulnerability to sleep changes.

Some patients with bipolar disorder also have circadian sleep disorders (Wehr et al., 1983, 1998), but only 12% of patients in this study displayed circadian sleep instability for a limited time period. In a short-term study of remitted outpatients with bipolar disorder using actigraphy, no objective evidence of sleep disturbances was detected (Jones et al., 2005). However, this same study and another study of bipolar patients using actigraphy observed circadian activity disruptions that persisted outside of episodes (Jones et al., 2005; Salvatore et al., 2008). Disruptions of circadian rhythms and alterations of genes involved in the molecular clock may be implicated in the pathophysiology of bipolar disorder, as recently reviewed (McClung, 2007).

There are several limitations to the analytical approach used in this study. Not all cross-correlations between a change in sleep duration and mood signify a mood switch that is clinically significant, indicate the start of an episode, or last beyond one day. However, severe symptoms may occur outside of episodes, and cumulative morbidity may have a stronger association with functional limitation in bipolar disorder than the number of episodes (Gitlin et al., 1995; Goldberg and Harrow, 2004; Bauer et al., 2008). Another drawback of this analytical approach is that cross-correlation analysis cannot be used to determine causality.

There are other limitations to this study. Total sleep time (including daytime naps) was not investigated and may be a valuable monitoring parameter. The design used a naturalistic approach, included heterogeneous patients, required use of a home computer and collected self-reported mood and sleep data. The analysis did not address prodromal symptoms unrelated to sleep including idiosyncratic signs, or comorbid psychiatric diagnoses. Although patients with a significant cross-correlation between mood and sleep duration were more likely to use benzodiazepines, we cannot determine if this was a cause or a result of the sleep and mood changes (Bauer et al., 2006).

In conclusion, we recommend that patients with bipolar disorder be taught be to monitor sleep duration for an oncoming mood change, rather than sleep onset or sleep offset. Ongoing monitoring is especially important for the patients who are vulnerable to the impact of sleep changes on mood. Additionally, there is a need to understand the impact of psychotropic medications on the sleep of patients with bipolar disorder, and to clarify the clinical significance of the long sleep duration of euthymic patients.

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This project was funded entirely by local university funds. The local funding sources had no involvement in the study design, collection, analysis and interpretation of data, in writing the report, and in the decision to submit for publication.

Conflict of interest

The ChronoRecord Association is a 501(c)(3) nonprofit organization that aims to increase understanding of mood disorders (www.chronorecord.org). None of the authors receive financial compensation from the Association. Tasha Glenn and Peter C Whybrow share a patent for ChronoRecord software. Michael Bauer, Paul Grof, Natalie Rasgon, and Peter C Whybrow are on the Medical Advisory Board. There are no other conflicts of interest.

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