

Predictability of Sleep in Patients with Insomnia

Annie Vallières, PhD; Hans Ivers, PhD; Simon Beaulieu-Bonneau, MPs; Charles M. Morin, PhD

École de psychologie, Université Laval, Centre d'étude des troubles du sommeil, Institut universitaire en santé mentale de Québec, Québec, Canada

Study Objectives: To evaluate whether the night-to-night variability in insomnia follows specific predictable patterns and to characterize sleep patterns using objective sleep and clinical variables.

Design: Prospective observational study.

Setting: University-affiliated sleep disorders center.

Participants: 146 participants suffering from chronic and primary insomnia.

Measurements and Results: Daily sleep diaries were completed for an average of 48 days and self-reported questionnaires once. Three nights were spent in the sleep laboratory for polysomnographic (PSG) assessment. Sleep efficiency, sleep onset latency, wake after sleep onset, and total sleep time were derived from sleep diaries and PSG. Time-series diary data were used to compute conditional probabilities of having an insomnia night after 1, 2, or 3 consecutive insomnia night(s). Conditional probabilities were submitted to a *k-means* cluster analysis. A 3-cluster solution was retained. One cluster included 38 participants exhibiting an unpredictable insomnia pattern. Another included 30 participants with a low and decreasing probability to have an insomnia night. The last cluster included 49 participants exhibiting a high probability to have insomnia every night. Clusters differed on age, insomnia severity, and mental fatigue, and on subjective sleep variables, but not on PSG sleep variables.

Conclusion: These findings replicate our previous study and provide additional evidence that unpredictability is a less prevalent feature of insomnia than suggested previously in the literature. The presence of the 3 clusters is discussed in term of sleep perception and sleep homeostasis dysregulation.

Keywords: Insomnia, sleep, night-to-night variability, predictability, sleep patterns, sleep diary

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INTRODUCTION

Sleep in insomnia is characterized by extensive night-to-night variability and unpredictability. Night-to-night variability is seen as the result of coping strategies used initially to compensate for the sleep loss but which subsequently become maladaptive behaviors perpetuating insomnia. For instance, an individual may go to bed earlier on the night following an insomnia night in order to recover. However, this increased time in bed may fragment sleep and create variability across nights. Several empirical studies have confirmed patients' clinical reports of extensive night-to-night variability.¹⁻⁵ Most of these studies have observed sleep variability while comparing good sleepers and individuals with insomnia sleep profiles. One recent study specifically addressed this issue in a sample of elderly insomnia sufferers.⁶ This study demonstrated greater night-to-night variability in insomnia compared to non-insomnia sample.

In our previous study,⁷ night-to-night variability in insomnia was investigated to evaluate if sleep on poor nights was predictable. If specific sleep patterns are present in insomnia, it would be expected that poor nights are occurring at regular intervals and not at random. That study was conducted with 106 participants with primary insomnia. Despite the presence

of high night-to-night variability, 3 specific sleep patterns were found. The first represented a group exhibiting a constant high probability of having a poor night's sleep (HPP; 21% of the sample). The second pattern was a group of participants presenting a low and decreasing probability of having a poor night's sleep (LPP; 42.9%). The third group exhibited a constant median probability of having a poor night's sleep (UP; 36.1%). The HPP group was characterized by more severe insomnia, with a predominance of sleep maintenance insomnia. The LPP group presented moderate insomnia severity with a predominance of sleep maintenance insomnia as well; and the UP group presented severe insomnia with the lowest proportion of sleep maintenance insomnia. These groups were similar on night-to-night variability, insomnia duration, age, and anxiety or depression symptoms.

Since these data were published, one review paper and 3 empirical studies have examined this topic. The review paper⁸ hypothesized that specific sleep patterns might be present in the night-to-night variability because insomnia is a periodic disorder. They added that the sleep homeostat in primary insomnia is affected in such a way that it takes more poor sleep nights to sufficiently increase the sleep pressure. They also underlined the possible reinforcement of taking sleeping pills when the good night expected by the sleep pattern is paired with the intake of a sleeping pill. This same group ran a pilot study with 10 participants to assess temporal patterning of poor sleep in patients with primary insomnia who completed sleep diaries for an average of 43 nights.⁹ Temporal patterning analysis suggested that a sleep pattern in which a "better than average" night's sleep most frequently occurred after 1 to 2 nights of poor sleep. Buisse et al.⁶ assessed 61 elderly participants with primary insomnia and compared them to 31 elderly participants without insomnia. Sleep diaries were completed and actigraphs were worn for 14 days before treatment. This study demonstrated

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Address correspondence to: Annie Vallières, PhD, FAS-Local 1044, 2325, rue des Bibliothèques, Université Laval, Québec (Qc), CANADA, G1V 0A6, Centre d'étude des troubles du sommeil, Centre de recherche Université Laval-Robert Giffard, Centre de recherche du Centre hospitalier universitaire de Québec; Tel : +1 418 656-2131, ext. 8258; Fax: +1 418 656-3646; E-mail: annie.vallieres@psy.ulaval.ca

that night-to-night variability in sleep was greater for people with insomnia. Their results do not support that a poor night's sleep is followed by a recovery night, which would imply the presence of a specific sleep pattern. Finally, Cervena et al.¹⁰ evaluated the presence of a sleep pattern within a sample of 36 participants with primary insomnia who underwent 3 nights of polysomnography (PSG). As there were no diary data, they conducted conditioned probability on these 3 PSG nights. No pattern was found in sleep, further suggesting that sleep patterns might be related to sleep perception but not to objective sleep.

Convergences can be drawn among the 4 studies cited. First, both our study⁷ and that of Buysse et al.⁶ confirmed extensive night-to-night variability in sleep of chronic insomnia. When these studies are considered together, they demonstrate that sleep seems to be unpredictable with no temporal structure. Secondly, 2 studies provide converging evidence that sleep in insomnia might be predictable.^{7,9} The 10 participants of Perlis et al.⁹ seem to be similar to those of our LPP group ($n = 45$).⁷ Indeed, in Perlis et al.'s study participants experienced a better than average night after an interval of 1 to 3 poor nights. Nevertheless, there are also divergences among studies, as Buysse et al.⁶ and Cervena et al.¹⁰ did not find sleep patterns in insomnia. Two reasons might explain these divergences: one is that insomnia can be seen as a periodic disorder; a second is the length of the series of data analyzed.

Sleep is regulated by mechanisms that act on circadian rhythm leading to periodicity. Clearly, in insomnia, this natural sleep periodicity is impaired, given that good night's sleep does not seem to happen regularly anymore. Sleep periodicity in insomnia could be affected in 2 ways: it could be affected at random providing an unpredictable sleep pattern, or it could be affected in such a way that good and poor nights occur alternately following a specific pattern. To examine sleep periodicity in insomnia, it is important to assess sleep over a long time period, as the likelihood of detecting stable sleep patterns increases with the number of nights assessed. This is the first problem that can explain divergence among studies, as one study¹⁰ computed conditional probabilities on 3 PSG nights. Clearly this period of time is too short to detect periodicity. Moreover, long series of data are important because the sleep pattern of each individual is not necessarily at the same point in the periodicity when the sleep evaluation begins. In other words, the first day reported in the sleep diary can potentially be any day of the sleep pattern when assuming the existence of a stable temporal pattern. The analysis of Buysse et al.⁶ implies that, for each participant, day one of the sleep diary is also day one of the sleep pattern, providing an arbitrary assignment of each night. This methodological choice may explain why these authors did not find stable temporal structure in sleep. By analogy, let us consider a researcher comparing the temporal course of cortisol levels of several participants regardless of the time the first sample was taken. When data are plotted from all participants together, results will show that, on average, cortisol does not exhibit a predictable temporal course. However, this unexpected conclusion would rather be explained by the arbitrary assignment of the first sample as being the starting point of the cortisol pattern. The difference between patterns in insomnia nights and cortisol level is

that at present, no periodicity has been identified for insomnia nights, while it is known for cortisol. Therefore, to determine if there are specific sleep patterns in insomnia, nights have to be considered periodic as is the case with diurnal cortisol level. **Sleep patterns should be studied first within each individual** to overcome an arbitrary assignment. Then, group analyses should investigate whether subgroups of participants exhibit similar sleep patterns.

Because results are equivocal concerning presence or absence of sleep pattern in primary insomnia, the objective of the present study was to replicate the study of Vallières et al.⁷ in a larger sample to examine if sleep in insomnia follows specific patterns despite the night-to-night variability observed. In addition, the present study aims at characterizing sleep patterns using objective and subjective sleep and clinical variables.

METHODS

Participants

Participants were recruited for a larger treatment study comparing combinations of cognitive-behavior therapy (CBT) and medication.¹¹ Only data derived from baseline measures were used for the current project. Participants were individuals with persistent insomnia evaluated for a treatment study. They were recruited through newspaper advertisements and referrals from outpatient clinics. Inclusion criteria were: (a) age ≥ 30 years, and meeting criteria for chronic insomnia based on a combination of diagnostic criteria from the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR)¹² and the International Classification of Diseases, 10th Edition (ICD-10)¹³: (b) difficulties initiating and/or maintaining sleep, defined as sleep onset latency and/or wake after sleep onset > 30 min, with a corresponding sleep time < 6.5 h and a sleep efficiency (SE) $< 85\%$; (c) difficulties initiating and/or maintaining sleep ≥ 3 nights per week for > 6 months; and (d) the sleep disturbances (or associated daytime fatigue) causing significant distress or impairment of social, occupational, or other areas of functioning (rating ≥ 2 on a 0-4 scale for individual items of the Insomnia Severity Index [ISI]). In addition to these general criteria from the main treatment study, the present study included only participants who had completed sleep diaries for ≥ 21 consecutive days with no more than 3 missing days.

Exclusion criteria were: (a) active and progressive physical illness or neurological degenerative disease; (b) use of medications known to alter sleep; (c) lifetime diagnosis of any psychotic or bipolar disorder; (d) current diagnosis of major depression, dysthymia, or anxiety disorders, unless currently in remission; (e) > 2 past episodes of major depression; (f) history of suicide attempt/contemplation within the past year; (g) alcohol or drug abuse within the past year; (h) evidence of sleep apnea, restless legs, or periodic limb movements during sleep; and (i) night-shift work or irregular sleep pattern (usual bedtime after 01:00). The large majority of patients enrolled in this clinical trial suffered from primary insomnia without any comorbid psychiatric disorders. However, patients with comorbid anxiety (e.g., GAD) or affective disorders (e.g., major depression, dysthymia) were included in the protocol if these coexisting conditions were not the primary cause of insomnia, and only if they were treated and in complete or partial remission.

Participants underwent a multi-step screening evaluation. Of the 486 individuals who completed an initial telephone screening, 242 were considered eligible for the study and went through a second evaluation consisting of (a) Insomnia Interview Schedule (IIS)¹⁴ and clinical sleep evaluation/history; (b) Structured Clinical Interview for DSM-IV (SCID-IV)¹⁵ and psychological screening; (c) medical history and physical examination; and (d) all-night PSG laboratory evaluations. Eighty-two persons were excluded after this screening phase because of psychological (n = 24), medical (n = 4), or other sleep disorders (n = 10); not meeting insomnia criteria (n = 9); lack of interest (n = 26); or use of hypnotic drugs (n = 5) or alcohol/substance abuse (n = 4). For the present study, 14 additional participants were excluded because they did not complete a sleep diary for ≥ 21 consecutive days.

The final sample included a total of 146 participants (60% women). As 29 of these 146 participants were part of the previous study,⁷ the replication was conducted with 117 participants (146-29). In addition to these 29 participants, the previous study included 59 participants from 2 other studies published at that time.^{16,17} Then, if results were replicated, the total sample to be studied for further analysis would be 146. Mean age was 51 years (SD = 10.3), mean education level was 14.7 years (SD = 3.58; range, 2-24), and mean insomnia duration was 16.4 years (SD = 13.7; range, 0.4-62). Participants completed daily sleep diaries for an average of 48 baseline nights (SD = 18.6, range 21-118) before receiving insomnia treatment. Results regarding treatment efficacy are reported elsewhere.¹¹ Overall, 2.7% of the total sample of participants presented initial insomnia, 23.3% sleep maintenance insomnia, 1.4% terminal insomnia, and 72.2% mixed insomnia.

Measures

Sleep diaries

Participants were instructed to complete their diaries every morning at breakfast time. Although sleep diary data do not reflect absolute values obtained from PSG, daily morning estimates of specific sleep parameters (e.g., sleep onset latency [SOL] and wake after sleep onset [WASO]) yield a reliable and valid clinical index of insomnia¹⁸ and represent standard outcome assessment in insomnia research.¹⁹ Several parameters were monitored on the diaries (bedtime, wake time, SOL, number and duration of awakenings, medication intake). Main outcome variables were SOL, WASO, early morning awakening (EMA; time awake between the last awakening and the rising time), TWT (SOL + WASO + EMA), TST, SE, total time spent in bed (TIB), and sleep quality rating (1-5 Likert scale). Despite some limitations (e.g., reactivity), which are inherent to all forms of self-monitoring, the sleep diary remains a practical, economical, and widely used assessment instrument in insomnia outcome research. Its most important feature is that it allows for prospectively tracking sleep patterns over extended periods of time in the subjects' natural environment.

Polysomnography (PSG)

Participants underwent 3 baseline nights of sleep laboratory evaluation. Bedtime and awake time in the sleep laboratory were kept within 30 min of the participants' habitual sleep schedule at home, as determined by sleep logs kept during the

2 weeks preceding recording. Standard PSG montage including electroencephalographic (EEG), electromyographic (EMG), and electroculographic (EOG) monitoring was used.²⁰ Respiration (air flow, tidal volume, and oxygen saturation) and anterior tibialis EMG were also monitored during the first night, which was used as a screening night for ruling out sleep apnea and periodic limb movements during sleep. Sleep stages were scored by experienced technicians, blind to participants' conditions, and according to standardized criteria.²⁰ Dependent variables were SOL (time from turning off lights to persistent stage 2 sleep), WASO (time awake from initial sleep onset until last awakening), number of awakenings (NWAK), total wake time (TWT; SOL + WASO and wake time before light on), total sleep time (TST), and SE (ratio of total sleep time to the actual time spent in bed and multiplied by 100). Measures of sleep architecture included percentages of time spent in NREM (stages 1, 2, and 3-4) and REM stages. Outcome measures were based on the average of pre-treatment nights 2 and 3, as the first night was used as an adaptation to the laboratory.

Insomnia Severity Index (ISI)

The ISI¹⁴ is a 7-item questionnaire assessing the severity of (a) sleep onset, (b) sleep maintenance, and (c) early morning awakening problems; (d) satisfaction regarding sleep; (e) perceived interference of sleep difficulties with daytime functioning; (f) noticeability of sleep problems by others; and (g) distress caused by the sleep difficulties. A 5-point Likert scale ("0" = not at all, "4" = extremely) is used to rate each of these items, yielding to a total score ranging from 0 to 28. A higher score indicates more severe insomnia, within 4 severity categories: absence of insomnia (0-7); sub-threshold insomnia symptoms (8-14); moderate insomnia (15-21); severe insomnia (22-28). The ISI has adequate psychometric properties.²¹

Dysfunctional Beliefs and Attitudes about Sleep Scale, 30-item version (DBAS-30)

The DBAS-30¹⁴ is a measure of sleep related cognitions. Participants rate each item on a 0-10 Likert scale (0 = strongly agree, 10 = strongly disagree). Total score is the mean of the 30 items (0 to 10), higher scores indicating higher endorsement of dysfunctional beliefs and attitudes.

Multidimensional Fatigue Inventory (MFI)

The MFI²² is composed of 20 statements for which the responder has to indicate, on a 5-point Likert scale, to what extent the particular item applies to his or her situation in recent times. The questionnaire measures 5 dimensions of fatigue: (1) general fatigue, (2) mental fatigue, (3) physical fatigue, (4) reduced activity, and (5) reduced motivation. For each scale, the score varies between 4 and 20, a higher score indicating a higher level of fatigue. The internal consistency and the construct validity of this scale are adequate.

Beck Depression Inventory (BDI) and Beck Anxiety Inventory (BAI)

The BDI²³ and BAI²⁴ are 2 of the most widely used instruments to assess psychological symptoms. Each of these questionnaires contains 21 items rating depression/anxiety symptoms experienced during the past week on a 4-point Likert scale (0-3). Total scores range from 0 to 63, with higher

scores suggesting higher depression/anxiety symptomatology. The psychometric properties (i.e., reliability, validity) of these instruments have been studied extensively.

Procedure

Participants completed self-report measures and daily sleep diaries before entering a treatment protocol. Measures were mailed to participants who were asked to complete baseline sleep diaries and to return them by fax each week after completion. Waiting time before entering treatment was different for each participant, which consequently led to different baseline lengths for sleep diary data. For more details on studies' procedure, see Morin et al.¹¹ Each participant thus presented a series of sleep diary nights in which each night was dichotomized into either "poor" or "good." Therefore, data were transformed into 146 series (one per participant) of nights labelled as either poor or good. As 29 participants were part of a previous study,⁷ the replication was conducted with 117 series. Then, if clusters of the previous study were replicated, comparison of clusters was done with the 146 participants. Because the purpose of this study is to evaluate the prediction of a poor night, nights labelled as poor should not raise doubt about the fact that they were effectively poor. Given that using the standard criteria of 30 min could increase the risk of arbitrary assignment of a poor or good night, the criteria used to dichotomize nights were thus strengthened. A poor night was then defined as SOL and/or WASO \geq 60 min associated with SE \leq 80%, as derived from daily sleep diaries. Nights that did not match both criteria were considered as good nights.

Data Analysis Plan

All data were carefully inspected to identify missing data and outliers and to assess normality.²⁵ Computations of missing data percentage yielded an average of 2.7 missing nights per participant (total of missing nights within the sample = 106 of 7896 nights). There was no relationship between number of nights and percentage of missing data, $r(105) = -0.05$, $P = 0.64$. Following statistical guidelines in these particular set of data cases,²⁶ no missing data imputation was performed and only complete sequences of 2, 3, or 4 nights were included in the computation of conditional probabilities. Descriptive and inferential statistics were completed using SAS 8.2 statistical software.²⁷ Alpha level was fixed at 5% (2-tailed) for all inferential tests.

Consecutive daily sleep data were conceptualized as time-series data. Each night was dichotomized as either a good or a poor night according to criteria described in the procedure section. Conditional probabilities to have a poor night after 1, 2, or 3 consecutive poor nights were computed for each participant. This analysis was used to predict the *probability* of an event, which in this study context was a poor night, rather than the *magnitude* of this event, which in sleep, would be the severity. However, the classic conditional probability formula was not appropriate because some time-series had non-consecutive (missing) data. Thus, the formula was slightly modified to take into account only consecutive sleep data (see Appendix). Conditional probabilities to have a poor night after 1, 2, or 3 consecutive poor nights were submitted to an exploratory *k-means* (least squares) cluster analysis in order to identify subgroups of

participants showing similar levels of conditional probabilities. Solutions ranging from 2 to 5 clusters were investigated. The final solution was selected based on 3 criteria: (a) the parsimony of the solution, (b) the sample size of each cluster, and (c) the clinical interpretability of each cluster. One-way ANOVAs, mixed model analysis, and χ^2 tests were computed to compare clusters on demographics, clinical, sleep, and psychological measures. One-way ANOVAs and χ^2 tests were computed to compare clusters on demographics, clinical, sleep, and psychological measures. Mixed model analysis was performed comparing the variability of sleep across clusters (3) and the severity of night (2) (fixed effects) while controlling for patient covariance (random effects).

RESULTS

Replication of Sleep Patterns and Level of Predictability

When computed on the totality of the replication sample ($N = 117$ participants), results of conditional probabilities suggested that sleep was unpredictable. Indeed, conditional probabilities to have a poor night after 1, 2, or 3 poor nights were respectively [$p(p|p) = 0.61$, $p(p|pp) = 0.63$, and $p(p|ppp) = 0.65$]. These results indicated that there was between 61% and 65% chance of experiencing a poor night following 1, 2, or even 3 consecutive poor nights.

Based on the 3 criteria mentioned previously, the *k-means* cluster analysis supported a 3-cluster solution ($R^2 = 75.7\%$), with each participant being part of only one cluster. Moreover, each participant within a given cluster experienced the same sleep pattern over time. The 3 clusters replicated the same clusters as in the previous study and were labelled the same. The first was called "high probability pattern" (HPP) and included 49 participants (42% of the replication sample) who displayed a predictable sleep pattern. Their mean probabilities (SD) to have a poor night after 1, 2, or 3 consecutive poor nights were high and constant [$p(p|p) = 0.82$, $p(p|pp) = 0.86$, and $p(p|ppp) = 0.87$; SD = 0.12, 0.09, and 0.09, respectively]. The second cluster, labelled "low probability pattern" (LPP), comprised 30 participants (26% of the replication sample) showing a low and decreasing probability to have a poor night following previous poor night(s) [$p(p|p) = 0.33$, $p(p|pp) = 0.25$, and $p(p|ppp) = 0.12$; SD = 0.17, 0.22, and 0.17, respectively]. For the second cluster, poor sleep appeared predictable as the probability was low and decreasing with the number of previous poor nights. The third cluster, labelled "unpredictable pattern" (UP), contained 38 participants (32% of the replication sample) showing a constant median probability to have either a poor night or a good night following poor nights [$p(p|p) = 0.55$, $p(p|pp) = 0.60$, and $p(p|ppp) = 0.61$; SD = 0.12, 0.11, and 0.15, respectively]. For the last cluster, a poor night appeared to be unpredictable and unrelated to the number of previous poor nights.

After having replicated the same 3 clusters, conditional probabilities and *k-means* cluster analyses were conducted again with the whole sample ($N = 146$). The solution still supported a 3-cluster solution ($R^2 = 76.4\%$). Means probabilities (SD) for the HPP group ($n = 54$ participants 37% of the sample) were exactly the same as within the replicated sample. For the LPP group ($n = 54$, 37%), probabilities followed the

Table 1—Means and standard errors of night-to-night variability (individual standard deviations) of sleep variables for poor nights and good nights among clusters

Sleep Variables	Clusters		
	HPP M (SE)	LPP M (SE)	UP M (SE)
SOL (minutes)			
Total variability	22.90 (1.99)	24.38 (2.32)	22.76 (1.92)
Poor nights	37.43 (2.72)	39.16 (3.13)	35.97 (2.72)
Good nights	8.37 (2.91)	9.60 (3.24)	9.55 (2.72)
WASO (minutes)			
Total variability	34.38 (1.73)	34.64 (2.02)	32.31 (1.67)
Poor night	50.91 (2.36)	51.67 (2.90)	47.88 (2.36)
Good night	17.85 (2.53)	17.60 (2.82)	16.74 (2.36)
SE (percentage)			
Total variability	11.75 ^a (0.50)	9.74 ^b (0.59)	11.82 ^a (0.49)
Poor night	13.60 (0.69)	11.20 (0.84)	13.83 (0.69)
Good night	0.91 (0.74)	8.28 (0.82)	9.80 (0.69)

Means of poor and good nights for each sleep variable differ at $P < 0.0001$, except for SE where $P < 0.01$. Means in the same row that do not share the same superscripts differ at $P_s < 0.05$ according to the REGW multiple range test. HPP, high probability pattern of insomnia; LPP, low probability pattern of insomnia; UP, unpredictable pattern of insomnia; SOL, sleep onset latency; WASO, wake after sleep onset; SE, sleep efficiency.

Table 2—Means and standard deviations for sociodemographic variables among clusters

Variables	Clusters		
	HPP M (SD)	LPP M (SD)	UP M (SD)
Age (years)	53.2 ^a (10.2)	47.8 ^b (10.5)	49.8 ^{ab} (9.7)
Education (years)	14.2 (2.7)	14.1 (4.2)	15.2 (3.4)
Insomnia duration (years)	16.8 ^{ab} (13.8)	12.2 ^b (9.5)	18.9 ^a (15.5)
	%	%	%
Gender (women)	66.7	50.0	61.1
Married status (married/ common-law)	68.5	65.8	72.2
Occupation (employed)	64.2	78.9	75.0
Insomnia subtype (mixed)	85.2	89.5	64.8

Means in the same row that do not share the same superscripts differ at $P_s < 0.05$ according to the REGW multiple range test. HPP, high probability pattern of insomnia; LPP, low probability pattern of insomnia; UP, unpredictable pattern of insomnia.

same pattern being low and decreasing nearly to 0% [$p(p|p) = 0.32$, $p(p|pp) = 0.24$, and $p(p|ppp) = 0.07$; SD = 0.17, 0.21, and 0.14, respectively]. Finally, the UP group ($n = 38$, 26%) still had roughly 1 chance out of 2 to have a poor night regardless of the number of previous poor nights [$p(p|p) = 0.55$, $p(p|pp) = 0.59$, and $p(p|ppp) = 0.59$; SD = 0.12, 0.11, and 0.15, respectively].

Threshold and Cluster Appropriateness

Two different analyses were carried out to ensure that criteria used did not create arbitrary assignment of good or poor night. Table 1 includes means and standard errors of night-to-night variability (i.e., standard deviation computed for each participant) of sleep variables for each cluster. First, linear mixed models showed that clusters did not differ significantly regarding night-to-night variability in SOL and WASO ($F_{S_{2,143}} = 0.17$ and 0.53 , n.s.), whereas clusters differed significantly on SE ($F_{2,143} = 4.49$, $P = 0.01$). Multiple comparison tests revealed that the LPP cluster presented significantly less variability than the 2 other clusters. Also, results showed that for each cluster, poor nights were significantly different for each sleep variable compared to good nights ($F_{S_{1,134}} = 138.48$, 244.44 , and 33.91 , $P_s < 0.0001$). As in the previous study, a percentage of nights on the edge of the threshold was computed for each participant following these criteria: WASO or SOL of 60 ± 15 min and SE of $80\% \pm 5\%$. Results showed that 10.3% of nights from the overall sample, which included a total of 7896 nights, met these “near the threshold” criteria. Also, 23 of 146 participants presented the highest percentage of nights near the threshold, which was from 20% to 42%. Nine of these 23 participants were in the LPP, 4 were among

the HPP cluster, and the remaining 10 were in the UP. Third, the number of missing data and length of the series were computed again per cluster. Participants in the HPP cluster presented an average of 46.3 nights with an average of 0.9 missing data per series; LPP, 45.8 nights with 0.8 missing data per series; and UP, 48 nights and 1.6 missing night. There was no significant difference between clusters on length of the series and number of missing data. Therefore, the variability among nights seemed equivalent across clusters at least for wake time variables; poor nights were significantly different from good nights. Moreover, none of the participants present a constant pattern of “near the threshold” nights that could lead to arbitrary assignment. Finally, length of the series was similar between clusters.

Differences among Clusters

Sociodemographic variables

Means and standard deviations for sociodemographic variables according to clusters are presented in Table 2. One-way ANOVAs revealed significant differences among clusters regarding age ($F_{2,143} = 3.39$, $P = 0.04$). Multiple comparisons test revealed that the LPP group was significantly younger than the UP group. Although the UP cluster seems to present insomnia for a longer time than the HPP, this difference was not significant ($F_{2,143} = 2.75$, $P = 0.07$). There were no significant difference on education level, gender, marital status, occupation, and insomnia subtype.

Subjective and objective sleep variables

Means and standard deviations for subjective and for objective sleep variables according to clusters are presented in Table 3. One way ANOVAs revealed significant differences among clusters concerning subjective sleep variables such as SOL ($F_{2,143} = 5.53$, $P = 0.005$), WASO ($F_{2,143} = 16.64$, $P < 0.0001$), TWT ($F_{2,143} = 12.82$, $P = 0.0001$), TST ($F_{2,143} = 8.69$, $P = 0.0003$), and SE ($F_{2,143} = 12.90$, $P < 0.0001$). Multiple comparisons test

Table 3—Means and standard deviations for subjective and objective sleep variables among clusters

Sleep Variables	Clusters		
	HPP M (SD)	LPP M (SD)	UP M (SD)
Subjective			
SOL (minutes)	44.1 ^a (42.1)	22.8 ^b (19.7)	30.5 ^b (24.6)
WASO (minutes)	83.0 ^a (43.7)	41.6 ^b (25.9)	61.7 ^c (27.8)
TWT (minutes)	190.9 ^a (79.2)	122.5 ^b (65.4)	146.8 ^b (51.2)
TST (minutes)	317.7 ^a (77.6)	377.0 ^b (64.3)	350.9 ^b (60.6)
SE (%)	62.0 ^a (15.4)	75.8 ^b (12.2)	70.1 ^c (10.9)
Objective			
SOL (minutes)	12.9 (7.8)	14.6 (9.2)	15.4 (10.8)
WASO (minutes)	63.2 (39.4)	52.1 (30.5)	61.7 (37.4)
TWT (minutes)	80.8 (43.4)	72.2 (38.7)	80.6 (43.9)
TST (minutes)	372.9 (45.4)	378.7 (41.5)	374.0 (52.3)
SE (%)	82.3 (8.9)	84.2 (8.3)	82.4 (8.9)
Stage 1 (%)	4.8 (2.5)	5.5 (3.8)	5.2 (3.2)
Stage 2 (%)	63.4 (5.9)	64.6 (8.2)	63.8 (6.4)
Stage 3-4 (%)	7.4 (6.5)	7.3 (6.8)	6.6 (7.6)
Stage REM (%)	24.4 (3.6)	22.8 (4.3)	24.3 (5.0)
FNA	2.7 ^a (1.7)	1.8 ^b (1.2)	2.1 ^{ab} (1.4)

Means in the same row that do not share the same superscripts differ at $P < 0.005$ according to the REGW multiple range test. HPP, high probability pattern of insomnia; LPP, low probability pattern of insomnia; UP, unpredictable pattern of insomnia; SOL, sleep onset latency; WASO, wake after sleep onset; TWT, total wake time; TST, total sleep time; SE, sleep efficiency; FNA, frequency of night awakenings.

revealed that HPP presented a longer SOL and TWT and lower TST ($P < 0.05$) than the 2 other clusters. For WASO, the 3 clusters were different, with HPP presenting the longest and LPP the shortest WASO ($P < 0.05$). The 3 clusters differed also on SE, with LPP having the highest SE and HPP the lowest ($P < 0.05$). Clusters did not differ for any of the objective sleep variables and on any of the sleep stages. However, HPP and UP presented significantly more awakenings than the LPP cluster ($P < 0.02$).

Comparisons were conducted to determine if participants within a given cluster overestimated sleep variables. Ratios of sleep diary variables on PSG measures were used. Table 4 presents means and standard deviations of the percentage of under- or overestimation of sleep variables compared to PSG for each cluster. The 3 clusters presented overestimation of SOL and TWT. However, a multiple comparison test revealed that HPP significantly overestimated SOL when compared to LPP and UP and overestimated TWT compared to LPP. The HPP cluster also underestimated TST less than to LPP and UP.

Clinical Variables

Means and standard deviations for clinical variables according to clusters are presented in Table 5. Results showed that clusters differ for insomnia severity as assessed with the ISI ($F_{2,143} = 5.58$, $P = 0.004$). Multiple comparison tests revealed that the HPP cluster presented more severe insomnia than the LPP cluster ($P < 0.05$). Clusters also significantly dif-

Table 4—Means and standard deviations of percentage of under- or overestimation of subjective sleep variables compared to objective ones among clusters

Sleep Variables	Clusters		
	HPP M (SD)	LPP M (SD)	UP M (SD)
Difference SOL (%)	333.3 ^a (448.3)	100.8 ^b (196.8)	166.3 ^b (313.8)
Difference WASO (%)	71.3 (118.9)	29.5 (159.5)	47.8 (135.6)
Difference TWT (%)	188.6 ^a (151.8)	104.0 ^b (123.5)	134.3 ^{ab} (146.7)
Difference TST (%)	-13.8 ^a (23.7)	0.8 ^b (20.8)	-5.4 ^b (15.8)

Means in the same row that do not share the same superscripts differ at $P < 0.02$ according to the REGW multiple range test. HPP, high probability pattern of insomnia; LPP, low probability pattern of insomnia; UP, unpredictable pattern of insomnia; SOL, sleep onset latency; WASO, wake after sleep onset; TWT, total wake time; TST, total sleep time; Difference SOL = (SOL(sleep diary) - SOL(PSG)) / SOL(PSG) × 100.

Table 5—Means and standard deviations for clinical variables among clusters

Clinical Variables	Clusters		
	HPP M (SD)	LPP M (SD)	UP M (SD)
Insomnia Severity Index	18.7 ^a (3.7)	16.1 ^b (4.2)	17.5 ^{ab} (3.4)
DBAS-30, q.19 (unpredictability)	7.7 (2.5)	7.1 (2.8)	7.3 (2.8)
FMI: General fatigue ¹	13.0 (3.2)	12.1 (3.3)	11.7 (3.6)
FMI: Physical fatigue	9.4 (3.6) ^{ab}	10.1 (3.2) ^a	8.5 (3.2) ^b
FMI: Mental fatigue	11.6 (3.5) ^a	10.6 (4.0) ^{ab}	9.7 (3.3) ^b
BAI: Anxiety symptoms ¹	8.5 (6.3)	8.0 (5.9)	5.9 (5.0)
BDI: Depression symptoms	8.2 (5.1)	8.8 (5.2)	7.9 (6.4)

¹ $P = 0.08$. Means in the same row that do not share the same superscripts differ at $P < 0.02$ according to the REGW multiple range test. HPP, high probability pattern of insomnia; LPP, low probability pattern of insomnia; UP, unpredictable pattern of insomnia; DBAS-16, Dysfunctional Beliefs and Attitudes about Sleep scale-16 items; FMI, Fatigue Multidimensional Inventory; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory.

ferred for physical and mental fatigue as evaluated by the MFI ($F_{2,143} = 2.87$ and 3.69 , $P = 0.05$ and 0.03 , respectively). A multiple comparison test revealed that physical fatigue was higher for the LPP cluster than the other two, while mental fatigue was higher for the HPP cluster compared to the other two clusters ($P < 0.05$). General fatigue and anxiety seemed to be lower for the UP cluster, but this was not significant. Depression symptoms did not differ among clusters ($F_{2,139} = 0.29$, $P = 0.75$). Regarding beliefs toward sleep, mixed model analysis was performed on item 19 of the DBAS-30 to evaluate if clusters differ on their expectations regarding sleep unpredictability. Results demonstrated that there were no differences between clusters in item 19 (“I can’t ever predict whether I’ll have a good or poor night’s sleep”) pertaining to sleep unpredictability, showing that participants of each cluster similarly endorsed the unpredictability feature of their sleep ($F_{2,143} = 0.75$, $P = 0.47$).

DISCUSSION

Sleep in insomnia may follow specific patterns, although there is extensive night-to-night variability among nights. These findings replicated our previous study and demonstrated that poor sleep might be predictable, following specific patterns for 68% of people with chronic insomnia. Poor sleep is predictable for two subgroups of participants: one cluster (HPP) presenting a constant high probability of having a poor night, and another (LLP) cluster for which the probability of having a poor night is low and decreasing to achieve nearly 0% after three poor nights. The third cluster is the UP, representing participants for whom poor sleep is unpredictable regardless of the previous number of poor nights' sleep. The night-to-night variability in sleep is high and similar within clusters, except for sleep efficiency for which LPP presents the lowest night-to-night variability. Furthermore, none of the participants had a constant pattern of near the threshold nights, ensuring that the label of "poor" night was adequately used.

The replication study identified the characteristics of each cluster. The HPP cluster was older and presented more severe sleep impairment (i.e., longer SOL, WASO, TWT, and shorter TST). The UP cluster presented insomnia for a longer time. No objective sleep variables distinguished the three clusters, except that the frequency of night awakenings was lowest for the LPP cluster. The three clusters presented moderate insomnia severity as measured by the ISI, with LPP having the least severe insomnia. Each cluster overestimated sleep onset latency and total wake time. However, the overestimation of these two variables was greatest for the HPP cluster. The three clusters were similar in their degree of endorsement of the belief that their sleep is unpredictable. The LPP cluster presented the highest physical fatigue level and the HPP cluster the highest mental fatigue level.

These findings regarding distinct patterns of poor sleep are unique because they are the first to demonstrate that poor sleep is predictable in insomnia despite extensive night-to-night variability. Although some patients with insomnia state that they can predict a poor night's sleep, no model has included such predictability in explaining insomnia. Moreover, if people with insomnia were able to predict their poor night's sleep, it would be expected that at least participants in the HPP cluster would be able to predict their poor sleep. However, participants strongly agreed that they could not predict whether they would have a good or poor night's sleep. It seems then that participants are unaware of their sleep pattern. Future prospective studies would benefit from asking participants to attempt predicting their nightly sleep in order to answer this question of predictability of good and poor night's sleep in insomnia.

In addition to being a replication of our previous study, part of these results are consistent with the findings of Perlis et al.⁹ The LPP group was similar to participants in the Perlis study, who had a better than average night after one to two poor nights' sleep. However, our study used a larger sample and demonstrated that this situation is not representative of all patients with insomnia. Sleep was unpredictable for about 37% of our sample. The fact that clusters were similar on objective sleep measures supported the results of Cervena et al.¹⁰ Our results also converged with those of Buysse et al.,⁶ demonstrating that high night-to-night variability is a characteristic for patients

with insomnia. However, our results diverged in that we found distinctive sleep patterns. Methodological differences among our studies might explain this difference. The first difference was the age of the sample. The HPP cluster participants were older than the others and presented a constant high probability of having a poor night's sleep. Buysse et al.⁶ conducted their study within an elderly population. As such, it is possible that their sample included participants similar to those in the HPP cluster. Two other methodological issues pertaining to the number of nights included in the analysis may also help explain the divergence in results. Having very long series of data increases the likelihood of finding a pattern in a supposed periodic behavior. In addition, the analyses used, as in our study, did not arbitrarily assume that the first night of the sleep diary was the first night of the (supposed) periodic cycle in sleep.

A few hypotheses can be drawn in trying to explain the three clusters. First, they may represent a continuum in the severity of insomnia. For example, when participants are young, insomnia is less severe and a good night is likely to occur after two or three poor nights. As participants get older, insomnia severity increases, sleep begins to be unpredictable, and then becomes constantly poor (representing HPP). Although this interpretation seems to make sense, it is not entirely supported by results. The presence of the unpredictable pattern group who are as old as the HPP cluster and present the same insomnia severity do not support the continuum in the severity explanation. In addition, younger persons with insomnia are thought to have predominantly sleep onset latency insomnia followed by mixed and maintenance insomnia as they get older. Even if the LPP cluster includes the youngest participants, it also has the shortest sleep onset latency.

Another hypothesis which attempts to understand the three clusters is related to sleep estimation. The three clusters were identified based on subjective reports of sleep. In our previous study,⁷ we hypothesized that the HPP cluster represented people who constantly misperceived their sleep, while the LPP cluster was more accurate by their estimation. Results on sleep estimation partially support this explanation showing that HPP clearly overestimated sleep onset latency and total wake time and underestimated total sleep time compared to LPP. However, this explanation still does not explain UP so readily, as they are similar to LPP in sleep onset latency estimation and similar to HPP in total wake time estimation. In addition, the hypothesis about a more positive way of thinking for LPP cannot explain the difference in misperception as the three clusters strongly endorse the belief that their sleep is unpredictable.

The third possible explanation for the three clusters is related to the dysregulation of the sleep mechanisms. As hypothesized previously,⁷⁻⁹ a greater level of sleep deprivation might be necessary to produce a sufficient pressure to sleep that will bypass all other factors contributing to poor sleep. With mild but cumulative sleep loss, the pressure to sleep reaches a threshold to produce a better night. This hypothesis is supported by one study²⁸ that demonstrated that individual with sleep maintenance insomnia have a weaker but functional sleep pressure. Following this explanation, LPP sleep homeostasis should still be working, but it would require a greater amount of sleep debt to generate a better night. For the HPP cluster, sleep homeostasis may be impaired such that even if poor sleep is occur-

ring every night the pressure to sleep is rarely high enough to bypass the threshold level. For UP, sleep regulation seems to be affected at random. The explanation about the homeostasis mechanism emphasizes the possible periodicity in sleep of people with insomnia.

A fourth explanation would be that the three clusters represent a continuum in the level of arousal. Sleep mechanisms would be working properly but concomitantly with a hyper physiological and/or cognitive arousal state. For instance, the LPP cluster would present a certain amount of arousal that could be bypassed by an increasing sleep pressure related to the number of poor nights. For the HPP cluster, level of cognitive and/or physiological arousal would be higher bypassing an efficient sleep pressure. Under such explanation, more sleepiness during the day as well as more sleep-related mental activities in the period preceding sleep would be expected for the HPP cluster. The fact that the HPP cluster presents more mental fatigue than the LPP cluster might partially support this explanation. Unfortunately, this explanation does not entirely support the UP cluster for which the arousal degree would be varying at random.

These results also supported one previous hypothesis⁹ regarding the fact that the predictability in sleep may reinforce maladaptive thoughts and behaviors. In our studies, participants strongly endorsed the belief that whether sleep would be good or not was unpredictable even if poor sleep was predictable in two-thirds of the sample. At least, the HPP cluster should have presented a lower endorsement of unpredictability knowing that their sleep would be poor. Surprisingly, it seems that the sleep pattern was not related to participants' belief of unpredictability. It seems obvious that thinking that one's own sleep is unpredictable when in fact it is may reinforce maladaptive behaviors that can contribute to perpetuate insomnia. For the LPP cluster, anything that would be done concomitantly to the "better night" occurrence would reinforce the fact that what had been done contributed to good sleep. What might have been done might be taking a sleeping pill, having a glass of milk, or staying in bed longer, which may all contribute to maintaining insomnia. For the HPP cluster, participants can try so many unsuccessful tactics that they lose the expectation to sleep well.

The current study presents some methodological limitations. First, all data were based on subjective and self-reported measures. Compliance to daily retrospective measures may be questionable, and the use of actigraphy could be a useful alternative to polysomnography. Second, reactivity to a self-report repeated measure could affect the external validity. For instance, participants may have enhanced severity in their self-reported sleep difficulties in order to be accepted in the treatment study. The replicated study used the same criteria as our previous study⁷ to define a poor night's sleep. As a consequence, there is still a limit in the interpretation toward "good" sleep. For example, in this study as in the previous one, the fourth night, in the LPP cluster, was one of recovery but not necessarily a "good" one. Moreover, because participants were not asked to predict if their night would be "good" or "poor," it was impossible to corroborate investigators' criteria of poor night. Thus, the distinction between a "poor" night sleep and a "better" night may not reflect participants' representation of "poor" and "good" sleep. Finally, the present sample represented a population with

primary insomnia who were expecting treatment for their difficulties, precluding generalization to other insomnia population.

In conclusion, sleep in insomnia might follow specific patterns despite the presence of high night-to-night variability. Poor sleep is predictable for about two-thirds of the sample. Again, these results demonstrated that unpredictability of sleep does not seem to be a general characteristic of all people with insomnia. Moreover, this study underlined that insomnia sufferers are not a homogeneous group having unpredictable poor nights. These results emphasize four possible explanations of these three clusters. They reflect the course of insomnia severity, a continuum in the sleep perception, a degree in the dysregulation of the sleep homeostat, or a continuum in the arousal state. Whatever the complete explanation is, it seems, from these results that sleep in insomnia may follow a periodicity which is affected but still present. Nevertheless, none of the four hypotheses clearly explain the "Unpredictable Pattern," which might reflect distinct functioning or randomness in the sleep regulation system. The fact that participants endorsed the belief that sleep was unpredictable, even when it was, supports the previous hypothesis⁸ that the occurrence of a better night might reinforce maladaptive beliefs or behaviors. The specific sleep patterns found did not inform on the sequence that follow "poor" and "better" nights in sleep periodicity. Therefore, further studies could focus on identifying the sequence of nights within each sleep pattern. Other studies should evaluate the hypotheses explaining clusters and investigate the course of the clusters in term of treatment response and process.

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Appendix—Conditional Probability Formula

The *classic* conditional probability formula states that the probability to observe a good night after a poor night is:

$$\begin{aligned} p(G | P) &= \frac{\text{number of one poor night followed by one good night}}{\text{number of poor nights}} \\ &= \frac{n(PG)}{n(P)} \end{aligned}$$

However, missing data *within* the series affected the number of available two-night sequences for the numerator of the formula. Thus, the solution was to correct the denominator of the formula, and replace it by the correct number of available two-night sequences for the participant. The *modified* conditional probability formula is:

$$\begin{aligned} p(G | P) &= \frac{\text{number of one poor nights followed by one good night}}{\text{number of poor nights followed by good or poor night}} \\ &= \frac{n(PG)}{n(Px)} \end{aligned}$$