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## First-night effect in the chronic fatigue syndrome

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### Abstract

Since the magnitude of the first-night effect has been shown to be a function of medical conditions and of settings in which polysomnographies are performed, it is essential to evaluate the habituation phenomenon in each case in order to determine the optimal recording methodology. A first-night effect was evidenced in certain cases of chronic fatigue syndrome, but not in others. To clarify this issue, a large group of patients with chronic fatigue syndrome who had no primary sleep disorders were selected and recorded for two consecutive nights in a hospital sleep unit. Several parameters, frequently associated with the first-night effect, were found to be influenced by the recording methodology: Total Sleep Time, Sleep Efficiency, Sleep Efficiency minus Sleep Onset, Sleep Onset Latency, Wake Time, Slow Wave Sleep, Rapid Eye Movement Sleep, Rapid Eye Movement Sleep Latency and Number of Sleep Cycles. Bland and Altman plots determined that the difference scores between the nights included a systematic bias linked to the order of recordings (first-night effect). Factorial analysis grouped the difference scores into three factors. No significant difference was observed between patients with generalized anxiety comorbidity and those with no psychiatric comorbidity, or between those with and without psychiatric comorbidity. Chronic fatigue syndrome must thus be added on the list of conditions where a clinically significant habituation effect takes place.

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### 1. Introduction

Polysomnography presently remains the tool of choice for the study of sleep and sleep disorders. One of its drawbacks is the variability of parameters across consecutive nights, which makes generalization difficult. What is known as the

‘first-night effect’ is the set of differences observed on the first recording in comparison with consecutive ones, a phenomenon recognized since 1964 (Rechtschaffen and Verdone, 1964) and later described in more detail (Agnew et al., 1966). Although most differences are observed in the comparison of Night 1 with Night 2, it has been shown that residual effects are present beyond Night 2 (Le Bon et al., 2001a) and, for a few variables, take up to 3 weeks before reaching full

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steady-state (Wohlgemuth et al., 1999)—so that the phenomenon should in fact more appropriately be called a ‘polysomnography habituation effect.’

The main characteristics of the first night effect include the following: less Total Sleep Time (TST) and Rapid Eye Movement (REM) sleep, Lower Sleep Efficiency index (SEI), more intermittent Wake Time (Wake Time), and longer REM Latency (RL), whereas no clear patterns have been described for non-REM (NREM) sleep.

The origins of the first-night effect are probably multifactorial and may include the following: (1) discomfort caused by electrodes; (2) limitation of movements by gauges and cables; and (3) potential psychological consequences of being under scrutiny. Factors that can influence the magnitude of the habituation process are: (1) *Setting in which recordings are performed*. In most cases, studies have been performed in specialized sleep units, adding a change in environment to the list of potential causes for first night effect. It has been proposed that home recordings, or a higher level of comfort in sleep units, could reduce or suppress the first night effect, to the point that first nights could be used without habituation (Coble et al., 1974; Browman and Cartwright, 1980). However, a home study in healthy controls has shown habituation effects of a magnitude comparable to what is usually observed in hospital sleep units (Le Bon et al., 2001a). (2) *Medical condition*. The first night effect was shown to be an important issue in sleep apneic disorders (Le Bon et al., 2000a) but was found less important in inpatients with depression (Toussaint et al., 1995), insomnia (Edinger et al., 1997, 2001) and posttraumatic stress disorder (Saletu et al., 1996). It is even considered of little clinical significance in the use of plethysmography for the detection of male impotence (Kader and Griffin, 1983). (3) *Patient age*. Older age has been linked to a more severe first night effect (Webb and Campbell, 1979).

Because medical conditions can affect the magnitude of the first night effect, the presence and clinical importance of habituation effects have to be examined in each case, in order to determine the optimal recording strategy. Relevant to chronic fatigue syndrome, one study on fibrositis, a related syndrome, reported a first night effect limited to

an increased RL and an increased number of awakenings per hour (Gupta and Moldofski, 1986). Another study on fibrositis reported only an increased RL (Whelton et al., 1992). The polysomnographic studies in chronic fatigue syndrome either used a habituation night as a precaution (Greenberg et al., 1995; Fischler et al., 1997; Morehouse et al., 1998) or considered that, on theoretical grounds, no first night effect would be present and recorded only one night (Stores et al., 1997; Sharpley et al., 1997).

The present study analyzes two consecutive recordings in a large group of patients with chronic fatigue syndrome in a sleep unit setting, the null hypothesis being that no habituation effect would be present.

## 2. Methods

### 2.1. Subjects

Patients with a primary complaint of chronic fatigue ( $n=126$ ) were referred to the sleep unit by the internal medicine polyclinic of a university hospital that specializes in fatigue disorders (tertiary care setting) between 2000 and 2002. The diagnosis of chronic fatigue syndrome was performed by one of us (L.L.) using CDC criteria (Fukuda et al., 1994) after physical examination and laboratory tests for exclusion of clinically significant disorders. DSM-IV criteria were assessed by experienced clinicians (CVM and SG). Patients with sleep disorders and alcohol/drug consumption were excluded for the potential influence of these comorbidities in a sleep study, notably to avoid as much as possible an overlap between fatigue and sleepiness. Accordingly, the following types of patients were excluded: (1) Patients with acute or chronic consumption of more than 10 drinks (approx. 10 g alcohol) per week or use of illicit drugs; (2) patients who had shown apneic–hypopneic indexes or periodic limb movements above 5/h on either of the 2 nights of recording; and (3) patients with irregular sleep schedules or night phase shifts. Twenty-five of the patients were also part of a sleep study comparing patients with chronic fatigue syndrome, apneic

patients and controls on respective proportions of deep and light sleep (submitted manuscript).

A washout of all psychotropic drugs was achieved and patients were required to avoid napping at least 2 weeks before admission to the sleep unit. Patients were required to modify their lifestyle as little as possible for the last fortnight. The study was performed according to the Helsinki guidelines.

## 2.2. Polysomnography

The two sequential recordings were made between Mondays and Fridays, in order to avoid more irregular weekend schedules. The following set-up was used: three pairs of EEG electrodes (Fp2-A1; C4-A1; O2-A1), one pair of EOG electrodes, a chin and two inferior limb EMG electrodes, thoracic and abdominal gauges for respiratory movements, thermistors around the mouth and the nose, a finger oxymeter and a microphone for the detection of snoring. Patients went to bed at their usual sleep time (approx. 23:00 h) but were awakened if they had not done so spontaneously, at approximately 07:00 h, because of hospital routine.

Recordings were randomly analyzed by one of two well-trained technicians, on a 21-inch screen displaying 30-s polysomnographic epochs. Standard scoring criteria were used (Rechtschaffen and Kales, 1968). Visual scoring was in two steps: (a) determination of sleep stages and (b) detection and quantification of respiratory sleep events and periodic limb movements. The inter-rater reliability was measured recently, and kappa values exceeded 0.88 for all variables. Sleep Onset Latency was defined as the time between Lights Out and the first epoch of Stage 2. Sleep Efficiency was TST divided by time in bed (TIB); Sleep Efficiency minus Sleep Onset Latency was TST/Sleep Period Time (SPT). Intermittent Wake Time represents the time spent awake after sleep onset (WASO). RL was defined as the time between the first epoch of Stage 2 and the first epoch of Stage REM, without excluding intermittent Wake Time. An apneic episode was defined as more than an 80% reduction in airflow for at least 10 s during sleep. A hypopneic episode was defined as a 50–80%

reduction of airflow amplitude accompanied by either a 3% or greater reduction in oxygen saturation or an arousal. The number of ultradian NREM-REM cycles was defined as each REM episode and the NREM period immediately preceding it, going back to sleep onset (1st NREM-REM cycle) or to the limit of another REM episode (from the second NREM-REM cycle to the end of the night). The first NREM episode began with the first epoch of Stage 2. Each REM episode began with the first epoch of REM and ended when the last epoch of REM was followed by at least 15 min of NREM sleep or the end of the night (Feinberg and Floyd, 1979; Merica and Gaillard, 1991).

## 2.3. Statistics

Three variables (Wake Time, SOL and RL) required log-transformation to achieve a normal distribution. Student's *t*-tests for paired measures were applied to compare outcomes collected over the 2 nights for the main hypothesis. Given their relatively large number, the remaining analyses were performed in a descriptive way. Patients were grouped by direction of the first night effect ('classical' vs. 'inverted' first night effect, where the subjects slept better on Night 1 vs. Night 2) and these groups were compared using chi-square tests with diagnostic categories. *T*-tests for unpaired samples were used to compare clinical groups on difference scores between the recordings. A principal components factorial analysis on the difference between night scores was carried out with Varimax rotation when Eigenvalues were superior to 1. Pearson's product-moment correlation coefficient was used for correlations. Hypotheses tests were two-sided and carried out at the 5% significance level (trends were considered between 5 and 10%). Statistics were computed with Statview 5.0 (SAS Institute).

## 3. Results

### 3.1. Data description

The primary reasons for exclusion of patients from the original group ( $n=126$ ) were excessive

Table 1  
Comparison of major sleep variables between two consecutive nights ( $n=83$ )

Variables	Night 1		Night 2		Mean* difference	Increment/ Night 1	CI of the difference	Paired <i>t</i> - tests ( <i>P</i> )	Bland– Altman <sup>s</sup> ( <i>P</i> )
	Mean	S.D.	Mean	S.D.					
TIB (min)	433.8	56.7	432.1	54.4	−1.7	0%	−6.9; 21.5	NS	NS
SPT (min)	371.5	80.0	389.3	57.7	17.8	+5%	1.7; 39.5	(0.058)	0.001
TST (min)	304.0	86.9	340.7	69.6	36.7	+12%	19.9; 59.0	0.001	0.019
Sleep efficiency (%)	68.2	18.5	76.4	13.3	8.2	+12%	4.2; 12.1	0.001	0.001
Sleep efficiency Minus sleep onset Latency (%)	80.8	14.9	87.1	10.7	6.3	+8%	2.8; 9.8	0.001	0.002
Wake time <sup>£</sup> (min)	67.4	48.1	48.6	40.1	−18.8	−27%	−31.4; −6.2	0.002	0.048
Sleep onset latency <sup>£</sup> (min)	62.3	54.5	42.7	30.6	−19.6	−31%	−31.8; −7.3	0.041	0.001
Slow wave sleep (min)	86.2	34.4	101.9	41.7	15.7	+18%	7.1; 25.5	0.001	NS
NREM sleep (min)	267.4	74.4	292.9	62.9	25.5	+9%	9.2; 41.8	0.003	NS
REM sleep (min)	36.5	24.1	47.8	22.9	11.3	+30%	5.7; 16.6	0.001	NS
REM latency <sup>£</sup> (min)	120.9	72.8	104.0	59.8	−16.9	−14%	−35.7; 1.8	0.042	(0.072)
Number of sleep cycles (number)	2.7	1.3	3.2	1.0	0.5	+18%	0.3; 0.9	0.001	0.020

£: log-transformed in the analyses; \*: Night 2 minus Night 1; \$: Correlations between the mean of the two nights and the difference between them; TIB: Time in Bed; SPT: Sleep Period Time; TST: Total Sleep Time.

consumption of alcohol ( $n=8$ ), use of illicit drugs ( $n=6$ ), apneic-hypopneic index  $\geq 5/h$  ( $n=17$ ), periodic limb movement ( $n=4$ ), irregular sleep schedules ( $n=6$ ) and night phase shift ( $n=2$ ). Eighty-three patients thus fulfilled the selection criteria and entered the study (mean age: 39.1; S.D.: 8.9; 73 women). Thirty-eight patients (46%) suffered from generalized anxiety disorder, 14 (17%) from major depressive disorder, 30 (36%) had a history of major depressive disorder, 8 (9%)

suffered from panic disorder and 4 (5%) from post-traumatic stress disorder. A total of 46 (55%) had at least one psychiatric diagnosis.

### 3.2. Repeatability and first-night effect

Table 1 describes the main sleep variables for the two nights. As an example, Fig. 1 shows the distribution of Wake Time as observed on Nights 1 and 2. We note that the distribution on Night 2

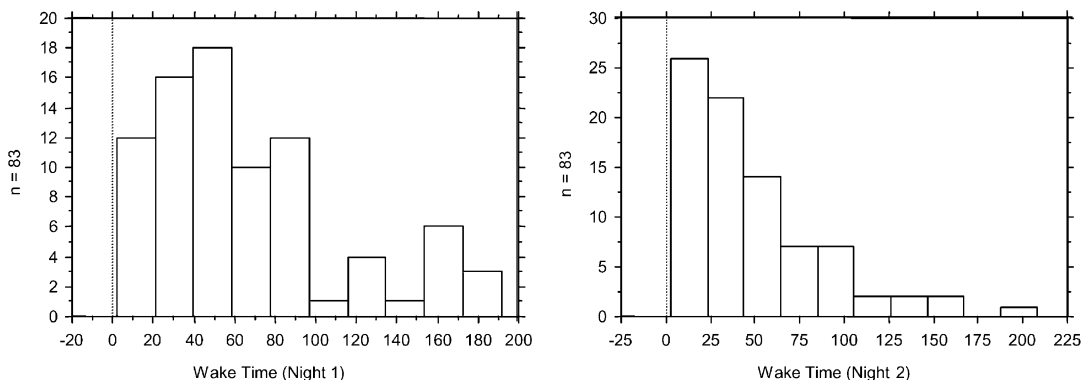


Fig. 1. Distribution histograms for Night 1 and Night 2.

is more homogeneous and largely more skewed to the left, which is classical with this variable. The other sleep variables showed similar differences in distribution (data not shown). In comparison with Night 2, the recording from Night 1 showed significantly less TST, Sleep Efficiency, Sleep Efficiency minus Sleep Onset Latency, Slow Wave Sleep (SWS), REM Sleep and Number of Sleep Cycles. Conversely, Wake Time, Sleep Onset Latency and RL showed significantly higher values. The relatively large increments observed between the two nights (percentage Night 2/Night 1) and the confidence intervals of the difference scores support clinical significance in most cases.

However, given the well-known variability of sleep parameters, a rather large degree of random variation is expected from night to night, even if, as is the case here, the same measurements were performed on the same patients on two consecutive nights. Approximately one quarter of the patients (28% for TST, 27% for Wake Time, 26% for Sleep Efficiency) even showed better sleep indices on Night 1 than on Night 2 (inverted first night effect). The extent to which the difference between the two results pertains to this general random variation or, more specifically, to the order of recordings (systematic bias) is not easy to disentangle.

Probably the best way to differentiate between the two explanations is by measuring the relationship between the mean of the two measurements and the difference between them (Bland and Altman, 1986). These correlations were significant for SPT, TST, Sleep Efficiency, Sleep Efficiency minus Sleep Onset, Sleep Onset Latency and Number of Sleep Cycles (Table 1). For higher Wake Time values, for instance (Fig. 2), the differences between the two recordings are more important, which means that a systematic bias is present, linked to the recording sequence.

### 3.3. Links with clinical subgroups

The comparisons were also performed on two subsamples (comorbidity with generalized anxiety,  $n=38$ , and patients with no psychiatric comorbidity,  $n=37$ ; Table 2). In both cases, the general pattern did not differ significantly from data on

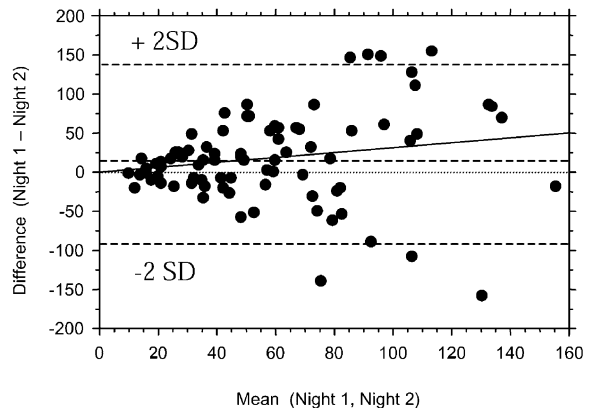


Fig. 2. As the real value for the population is unknown, the mean represents our best estimate from the sample. The difference shows by how much the two recordings differ from each other (bias). The data dispersion can be judged graphically to be more or less clinically significant. For a good agreement between two measurements, the mean difference between results should be close to zero, no correlations should be evidenced between mean values and differences, and the dispersion of the difference scores should be limited. In the present case, the mean difference is not equal to zero and the differences increase significantly with the magnitude, which means that a systematic error is linked to the order of recordings. It is also informative to note that the dispersion is more important with higher degrees of magnitude, so that prediction from one night to another will be less reliable at higher values. Note that the slope is only presented here for easier reading, as the figure does not represent a regression.

the entire sample, but the tests had less statistical power because they included fewer patients. Comparisons were also performed between the two above-mentioned subsamples, and between patients with or without psychiatric comorbidity, and were not significant for any variable.

The data were then split into two according to the direction of the first night effect, using the difference between the nights on TST. The 24 patients with inverted first night effect were thus compared with the 59 who showed classical first night effect. Chi-square tests did not demonstrate associations with the clinical subgroups mentioned above.

### 3.4. Association between variables in the first night effect

To measure whether the selected variables varied in parallel during the habituation process, a facto-

Table 2  
Comparison of major sleep variables between two consecutive nights (subgroups generalized anxiety disorder and no psychiatric diagnosis)

Variables	Generalized anxiety disorder ( <i>n</i> =38)						No psychiatric diagnosis ( <i>n</i> =37)							
	Night 1		Night 2		Mean* difference	CI of the difference	Significance ( <i>P</i> )	Night 1		Night 2		Mean* difference	CI of the difference	Significance ( <i>P</i> )
	Mean	S.D.	Mean	S.D.				Mean	S.D.	Mean	S.D.			
TIB (min)	420.7	69.4	448.4	43.7	27.7	−1.8; 57.2	(0.065)	447.8	40.4	447.2	40.1	−0.528	−15.1 ; 14.1	NS
SPT (min)	351.5	94.2	388.3	53.0	36.8	−0.978; 74.6	(0.056)	383.4	66.6	396.0	55.8	12.6	−9.4; 34.7	NS
TST (min)	292.8	94.7	338.9	67.3	46.0	6.6; 85.5	0.024	313.2	75.4	345.5	67.9	32.3	11.5; 52.9	0.003
Sleep Efficiency (%)	68.4	18.3	75.8	14.1	7.4	−0.443; 15.3	(0.063)	70.2	16.7	77.2	13.4	6.9	2.5; 11.4	0.003
Sleep Efficiency minus Sleep Onset Latency (%)	82.1	11.9	86.9	10.5	4.8	−0.537; 10.2	(0.076)	81.5	13.3	87.2	11.7	5.6	0.081; 11.2	0.047
Wake Time <sup>£</sup> (min)	58.6	37.0	49.4	39.8	−9.2	−29.0; 10.5	NS	70.2	49.4	50.5	44.0	−19.6	−41.4; 2.1	0.076
Sleep Onset Latency <sup>£</sup> (min)	64.5	56.3	52.3	38.8	−12.2	−36.6; 12.2	NS	56.4	49.2	36.0	20.4	−20.4	−5.4; −35.4	0.009
Slow Wave Sleep (min)	74.8	44.6	86.3	31.6	11.5	−3.8; 26.8	NS	89.5	36.6	107.4	48.4	17.8	3.6; 32.0	0.015
NREM Sleep (min)	264.5	78.6	294.4	60.2	29.9	−3.7; 63.5	(0.079)	278.1	63.6	299.8	62.1	21.7	3.0; 40.3	0.023
REM Sleep (min)	34.1	23.9	48.1	24.0	13.9	4.5; 23.5	0.005	40.1	24.6	48.5	24.5	8.4	0.7; 16.0	0.033
REM Latency <sup>£</sup> (min)	114.2	70.4	96.0	48.0	−18.3	−43.2; 23.5	NS	123.0	64.3	111.9	66.6	−11.0	−41.5; 19.4	NS
Number of Sleep Cycles (number)	2.5	1.4	3.3	0.8	0.7	0.1; 1.3	0.016	2.9	1.1	3.3	0.9	0.4	0.0; 8	0.041

£: log transformed in the analyses; \*: Night 2 minus Night 1; TIB: Time in Bed; SPT: Sleep Period Time; TST: Total Sleep Time.

Table 3  
Principal component factorial analysis on the differences between variable scores (Night 2 minus Night 1)

Differences (2-1)	Factor 1	Factor 2	Factor 3
TST (min)	0.548	0.028	0.048
Sleep Efficiency (%)	0.487	0.087	0.009
Sleep Efficiency Minus Sleep Onset Latency (%)	-0.168	0.654	0.066
Wake Time (min)	0.455	-0.799	-0.014
Sleep Onset Latency (min)	-0.741	0.380	-0.033
Slow Wave Sleep (min)	0.200	0.309	0.142
Non-REM Sleep (min)	0.566	0.001	0.143
REM Sleep (min)	0.183	0.118	-0.290
REM Latency	0.222	0.045	0.851
Number of sleep cycles	0.531	-0.282	-0.512

rial analysis was performed on differences between night scores (Table 3). The first factor (53% of the variance) heavily loaded with TST, Sleep Efficiency, NREM Sleep, Sleep Onset Latency, and Number of Sleep Cycles. It seems to represent night duration and variables that increase with night duration, such as the Number of Sleep Cycles. The second factor (16% of the variance) loaded with Wake Time, Slow Wave Sleep, and Sleep Efficiency minus Sleep Onset Latency. It seems to represent more intrinsic sleep quality than the first factor and is inversely linked to intermittent Wake Time. The third factor (10% of the variance) loaded with REM Sleep, RL and Number of Sleep Cycles. It probably represents variables related to REM Sleep.

#### 4. Discussion

The main result of this comparison between two consecutive nights in patients with chronic fatigue syndrome is the significant differences observed in SPT, TST, Sleep Efficiency, Sleep Efficiency minus Sleep Onset Latency, REM Sleep, Sleep Onset Latency, and RL. This is typically the kind of outcome that can be attributed to first night effect. Examination of the difference scores, incrementing percentages and confidence intervals reveals differences between the nights that should be considered as clinically significant in most cases.

As can be judged from Fig. 2, a large dispersion is apparent when the differences are compared to

the mean, which underlines a poor repeatability of the outcomes. Approximately a quarter of the patients, for instance, slept better on Night 1 than on Night 2, showing an inverted first night effect. No links were found, however, between the direction of the first night effect and clinical subgroups, so this probably merely reflects the random variation between nights. This dispersion was also clearly more elevated at higher mean values, so that the repeatability of the measurements diminished with the magnitude of the studied variable. Not all the differences between the nights were random, however, since correlations between difference scores and means were positive in most cases, which indicates a significant bias linked to the order of recordings (first night effect). The difference in distribution for Wake Time was quite visible in Fig. 1 and was similar to other sleep variables.

No difference was observed between patients with generalized anxiety disorder and those who showed no psychiatric comorbidity, nor between those who did or did not present such a comorbidity. This is in accordance with a study showing that sleep parameters in chronic fatigue syndrome were not attributable to such associations (Le Bon et al., 2000b).

No single sleep variable can summarize the first night effect, as factorial analyses isolated three factors. It is particularly interesting, from a physiological point of view, to note the grouping of difference scores of REM Sleep, RL, and Number of Cycles, since the Number of Cycles has been

shown to be associated with both RL (Merica and Gaillard, 1985; Le Bon et al., 2001b) and REM duration (Le Bon et al., 2002) in studies of healthy controls.

The large group of patients in the present study and the differences in the selection criteria are likely to explain the discrepancies with the two previous reports showing only a limited first night effect in fibromyositis (Gupta and Moldofski, 1986; Whelton et al., 1992).

The main limitation of this retrospective study was probably the use of spontaneous sleep-wake schedules and the forced awakenings of some patients at 07:00 h because of hospital routine. It is also possible that weekends immediately preceding the recordings may have influenced the recordings on Mondays and Tuesdays more than the weekdays immediately preceding the recordings on Thursdays and Fridays. The naturalistic approach used here is probably less reliable than the use of a constant routine and nap control organized beforehand with the use of sleep logs to adjust sleep periods to future sleep unit setting. It is, for instance, possible that a higher sleep pressure on Night 2 might partially explain the present outcome. However, patients with irregular schedules and night shifts were excluded. We also observed that approximately a quarter of the group actually slept better on Night 1, which is hard to explain by this rationale. Also, for clinical reasons, patients with chronic fatigue syndrome, as a rule, try to conserve their energy and do not go out late at night, a characteristic that reduces the potential weekend influence. The present results follow the usual pattern and magnitude that can be observed in most studies on the first night effect. Another potential limitation is the lack of actual controls on patient reports concerning the use of alcohol, tobacco, caffeine and street drugs.

In conclusion, a clinically significant first night effect was observed in a large group of patients with chronic fatigue syndrome. The minimal recommendation should be to analyze only the second night of polysomnographic recording. Considering the inverse evolution of a significant subsample, two consecutive habituation nights should probably be advocated for optimal comparisons with healthy controls. Studies that have not used habituation

nights, even those performed at home, should thus probably be interpreted with some caution.

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### References

- Agnew, H.W., Webb, W.B., Williams, R.L., 1966. The first-night effect: an EEG study of sleep. *Psychophysiology* 2, 263–266.
- Bland, J.M., Altman, D.G., 1986. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 8, 307–310.
- Browman, C.P., Cartwright, R.D., 1980. The first-night effect on sleep and dreams. *Biological Psychiatry* 15, 809–812.
- Coble, P., McPartland, R.J., Silva, W.J., Kupfer, D.J., 1974. Is there a first-night effect? (a revisit). *Biological Psychiatry* 9, 215–219.
- Edinger, J.D., Fins, A.I., Sullivan, R.J., Marsh, G.R., Dailey, D.S., Hope, T.V., Young, M., Shaw, E., Carlson, D., Vasilas, D., 1997. Sleep in the laboratory and sleep at home: comparisons of older insomniacs and normal sleepers. *Sleep* 20, 1119–1126.
- Edinger, J.D., Glenn, D.M., Bastian, L.A., Marsch, G.R., Daile, D., Hope, T.V., Young, M., Shaw, E., Meeks, G., 2001. Sleep in the laboratory and sleep at home. II: Comparisons of middle-aged insomnia sufferers and normal sleepers. *Sleep* 24, 761–770.
- Feinberg, I., Floyd, T.C., 1979. Systematic trends across the night in human sleep cycles. *Psychophysiology* 16, 283–291.
- Fischler, B., Le Bon, O., Hoffmann, G., Cluydts, R., Kaufman, L., De Meirleir, K., 1997. Sleep anomalies in the chronic fatigue syndrome. A comorbidity study. *Neuropsychobiology* 35, 115–122.
- Fukuda, K., Straus, S.E., Hickie, I., Sharpe, M.C., Dobbings, J.G., Komaroff, A. and the International Chronic Fatigue Syndrome Study Group, 1994. The chronic fatigue syndrome: a comprehensive approach to its definition and study. *Annals of Internal Medicine* 121, 953–959.
- Greenberg, H.E., Ney, G., Scharf, S.M., Ravdin, L., Hilton, E., 1995. Sleep quality in Lyme disease. *Sleep* 18, 912–916.
- Gupta, M.A., Moldofski, H., 1986. Dysthymic disorder and rheumatic pain modulation disorder (fibrositis syndrome): a comparison of symptoms and sleep physiology. *Canadian Journal of Psychiatry* 31, 608–616.
- Kader, G.A., Griffin, P.T., 1983. Reevaluation of the phenomena of the first-night effect. *Sleep* 6, 67–71.
- Le Bon, O., Hoffmann, G., Tecco, J., Staner, L., Nosedà, A., Pelc, I., Linkowski, P., 2000a. Mild to moderate sleep respiratory events: one negative night may not be enough. *Chest* 118, 353–359.

- Le Bon, O., Hoffman, G., Murphy, G., De Meirleir, K., Cluydts, R., Pelc, I., 2000b. How significant are primary sleep disorders and sleepiness in the chronic fatigue syndrome? *Sleep Res Online* 3 (2), 43–48.
- Le Bon, O., Staner, L., Hoffmann, G., Dramaix, M., San Sebastian, I., Murphy, J.R., Kentos, M., Pelc, I., Linkowski, P., 2001a. The first-night effect may last more than one night. *Journal of Psychiatric Research* 35, 165–172.
- Le Bon, O., Staner, L., Hoffmann, G., Pelc, I., Linkowski, P., 2001b. Shorter REM latency associated with more sleep cycles of a shorter duration in healthy humans. *Psychiatry Research* 104 (75–83), 2001.
- Le Bon, O., Staner, L., Rivelli, S.K., Hoffmann, G., Pelc, I., Linkowski, P., 2002. Correlations using the NREM–REM sleep cycle frequency support distinct regulation mechanisms for REM and NREM sleep. *Journal of Applied Physiology* 93, 141–146.
- Merica, H., Gaillard, J.M., 1985. Statistical description and evaluation of the interrelationships of standard sleep variables in normal subjects. *Sleep* 8, 261–273.
- Merica, H., Gaillard, J.M., 1991. A study of the interrupted REM episode. *Physiology and Behavior* 50, 1153–1159.
- Morehouse, R.L., Flanigan, M., MacDonald, D., Braha, D., Shapiro, C., 1998. Depression and short REM latency in subjects with chronic fatigue syndrome. *Psychosomatic Medicine* 60, 347–351.
- Rechtschaffen, A., Verdone, P., 1964. Amount of dreaming: effect of incentive, adaptation to laboratory, and individual differences. *Perceptual Motor Skills* 19, 947–958.
- Rechtschaffen, A., Kales, A. (Eds.), 1968. *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep States of Human Subjects*. Superintendent of Documents, U.S. Government Printing Office, Washington, DC.
- Saletu, B., Klösch, G., Gruber, G., Anderer, P., Udomratn, P., Frey, R., 1996. First-night effects on generalized anxiety disorder (GAD)-based insomnia: laboratory vs. home sleep recordings. *Sleep* 19, 691–697.
- Sharpley, A., Clements, A., Hawton, K., Sharpe, M., 1997. Do patients with ‘pure’ chronic fatigue syndrome (neurasthenia) have abnormal sleep? *Psychosomatic Medicine* 59, 592–596.
- Stores, G., Fry, A., Crawford, C., 1997. Sleep abnormalities demonstrated by home polysomnography in teenagers with chronic fatigue syndrome. *Journal of Psychosomatic Research* 45, 85–91.
- Toussaint, M., Luthringer, R., Schaltenbrand, N., Carelli, G., Lainey, E., Jacqmin, A., Muzet, A., Macher, J.P., 1995. First-night effect in normal subjects and psychiatric inpatients. *Sleep* 18, 463–469.
- Webb, W.B., Campbell, S.S., 1979. The first night effect revisited with age as a variable. *Waking Sleeping* 3, 319–324.
- Whelton, C.H., Salit, I., Moldofski, H., 1992. Sleep, Epstein-Barr virus infection, musculoskeletal pain and depressive symptoms in chronic fatigue syndrome. *Journal of Rheumatology* 19, 939–943.
- Wohlgemuth, W.K., Edinger, J.D., Fins, A.I., Sullivan Jr, R.J., 1999. How many nights are enough? The short-term stability of sleep parameters in elderly insomniacs and normal sleepers. *Psychophysiology* 36, 233–244.