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SHORT REPORT

The first-night effect and the consistency of short sleep in insomnia disorder

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Summary

The nature and degree of objective sleep impairments in insomnia disorder remain unclear. This issue is complicated further by potential changes in sleep architecture on the first compared with subsequent nights in the laboratory. Evidence regarding differential first-night effects in people with insomnia disorder and controls is mixed. Here, we aimed to further characterize insomnia- and night-related differences in sleep architecture. A comprehensive set of 26 sleep variables was derived from two consecutive nights of polysomnography in 61 age-matched patients with insomnia and 61 good sleeper controls. People with insomnia expressed consistently poorer sleep than controls on several variables during both nights. While poorer sleep during the first night was observed in both groups, there were qualitative differences regarding the specific sleep variables expressing a first-night effect. Short sleep (total sleep time < 6 hr) was more likely during the first night and in insomnia, although approximately 40% of patients with insomnia presenting with short sleep on night 1 no longer met this criterion on night 2, which is important given the notion of short-sleeping insomnia as a robust subtype.

KEYWORDS
electroencephalogram, first-night effect, insomnia, polysomnography, sleep architecture

Jennifer R. Ramautar and Eus J.W. Van Someren share senior authorship.

1 | INTRODUCTION

Insomnia disorder is characterized by self-reported sleep difficulties with adverse effects on daytime functioning (Riemann et al., 2022). Although complaints are often severe and the diagnosis increases the odds of developing other mental disorders (Hertenstein et al., 2019), objective sleep as measured by laboratory-based polysomnography (PSG) typically shows mild differences between patients with insomnia and good sleeper controls. Meta-analytic evidence indicates systematic but modest differences across many sleep measures, with patients exhibiting reductions in total sleep time (TST; mean group difference: −24 min), sleep efficiency (SE; −7%), N3 sleep (−20 min, −2%) and rapid-eye movement sleep (REM; −11 min, −2%), as well as increases in sleep-onset latency (SOL; +6 min), wake after sleep onset (WASO; +22 min, +6%) and number of Awakenings (+6; Baglioni et al., 2014). At the same time, individual studies vary substantially regarding the precise sleep variables affected.

Assessing PSG disturbances in insomnia is complicated further by the first-night effect (FNE), whereby individuals may sleep worse during their first night in a sleep laboratory, usually ascribed to unfamiliarity with the laboratory setting, discomfort of sleeping with electrodes and/or being under scrutiny. For controls, FNEs have been reported fairy consistently (Agnew et al., 1966; Edinger et al., 1997; Hirscher et al., 2015; Toussaint et al., 1995), with meta-analytic evidence indicating systematic first-night reductions in TST (mean night difference: −18 min), SE (−3%) and REM (−2%), and first-night increases in SOL (+3 min), WASO (+7 min), REM latency (+16 min) and N1 (+1%; Ding et al., 2022).

First-night effects have also been reported in insomnia (Edinger et al., 1997; Hirscher et al., 2015; Newell et al., 2012; Riedel et al., 2001), though some studies found no effect (Morin et al., 1993), or even suggest subgroups with a reverse FNE (Hauri & Olmstead, 1989; Riedel et al., 2001). Studies including both groups suggest comparable FNEs (Edinger et al., 1997; Hirscher et al., 2015; Newell et al., 2012) or attenuated FNEs in patients (Toussaint et al., 1995), though groups were not always compared statistically and individual studies again vary regarding the precise sleep variables expressing effects. As such, it remains unclear whether and to what degree a single PSG night captures typical group differences, or allows separation between short (TST < 6 hr) and normal sleep duration insomnia subtypes with potentially different prognoses and treatment requirements (Vgontzas & Fernandez-Mendoza, 2013).

Given the unresolved status of both insomnia- and night-related differences in sleep architecture, we set out to comprehensively characterize how PSG-derived sleep variables differ between patients with insomnia and controls, and between laboratory nights.

2 | METHODS

The current sample concerns archival PSG records stemming from four studies with comparable inclusion/exclusion criteria and overnight protocols, but varying in daytime experimental procedures. We included participants: (1) who either had a confirmed insomnia disorder diagnosis according to current DSM and ICSD criteria or qualified as good sleeper controls; (2) who spent two consecutive nights in the lab under unperturbed PSG registration; (3) whose PSG recordings had durations > 4 hr and were of sufficient quality for sleep scoring. Individual study protocols were approved by the ethics committees of either VU Medical Center or University of Amsterdam. All participants adhered to their own habitual sleep timing, underwent an assessment of the Insomnia Severity Index (Bastien et al., 2001), gave written informed consent in accordance with the Declaration of Helsinki, and were paid for participation.

The 256-channel electroencephalography (EEG; 1000 Hz sample rate, Cz reference) with additional bipolar physiological signals (Electrical Geodesic, Eugene, OR, USA) was converted to a PSG montage of standard EEG, electrooculography and electromyography for visual scoring of sleep stages and arousals (Berry et al., 2018). Sleep architecture variables were calculated using SleepTrip. Defining sleep onset/offset as the first/last epoch scored as sleep, we considered 26 sleep measures: time in bed (TIB; lights out to sleep offset); total sleep period (TSP; sleep onset to sleep offset); TST (minutes of sleep in TSP); SOL (lights out to sleep onset); sleep efficiency (SE_TIB: TST/TIB × 100%; SE_TSP: TST/TSP × 100%); time in minutes (e.g. N2_Dur) and percentages (e.g. N2%) spent in each stage during/relative to TSP; latency in minutes from sleep onset to N2, N3 and R (e.g. N2_Lat); Awakenings (total number); stage transition density (StageTrans, number per hour); global (AI_TST) and stage-wise arousal index (e.g. AI_N2, number per hour).

Statistical analyses were performed in R. Following logarithmic transformations of N2_Lat, N3_Lat and AI_N3, a mixed-effect model (hereafter: full model) implemented in *lme4* estimated overall differences between groups and nights for each dependent sleep variable. In each model, we included Group, Night and their interaction as predictors, Age and Sex as covariates, and Subject as a random intercept. Standardized coefficients were taken as effect sizes. Next, to facilitate comparisons to previous studies considering only a single night or group, we computed post hoc tests using *emmeans* for four pairwise contrasts (two group comparisons, one for each night; two night comparisons, one for each group). Cohen's *d* was taken as effect size. Given our aim to estimate sleep architecture comprehensively across and within groups and nights, we report all relevant effect sizes and use uncorrected $p < 0.05$ as a pragmatic cutoff to select effects to discuss, but the reader is free to consider what conclusions might be drawn under alternative statistical approaches.

3 | RESULTS

The present sample consists of 61 patients with diagnosed insomnia disorder (48 female, 79%; 50.3 ± 13.1 years, range: 23–69) and 61 good sleeper controls (41 female, 67%; 46.1 ± 14.2 years, range 23–70). Groups did not differ in terms of sex ($\chi^2 = 2.0$, $p = 0.15$) or age ($t_{120} = 1.7$, $p = 0.09$). The Insomnia Severity Index was higher for patients than controls (16.1 ± 4.6 versus 3.7 ± 4.0, $t_{117} = 16.8$, $p < 10^{-15}$). Table 1 shows descriptive statistics for 26 sleep

TABLE 1 Means (M) and standard deviations (SD) for 26 sleep architecture variables by Group and Night

	Control ($N = 61$)							Insomnia ($N = 61$)								
	Night 1		Night 2		Change (%)			Night 1		Night 2		Change (%)			Comparison	
PSG variable	*M*	SD	*M*	SD	↑	↓	=	*M*	SD	*M*	SD	↑	↓	=	χ^2	*p*
TIB (min)	467.7	48.2	479.5	55.0	62	38	0	461.3	50.9	477.9	50.8	69	31	0	0.6	0.446
TSP (min)	451.7	46.8	459.4	54.9	61	39	0	430.3	62.2	454.4	52.7	67	33	0	0.6	0.451
TST (min)	404.2	51.2	424.0	58.5	67	33	0	375.4	74.0	398.8	59.5	61	39	0	0.6	0.451
SOL (min)	13.8	10.1	16.5	19.3	**52**	**48**	0	21.0	23.8	13.3	14.2	**30**	**66**	5	**5.6**	**0.018**
SE_TIB (%)	86.5	7.1	88.5	7.2	67	33	0	81.4	12.6	83.8	10.7	57	43	0	1.3	0.262
SE_TSP (%)	89.5	6.8	92.2	5.5	69	31	0	87.1	10.2	87.9	8.6	54	46	0	2.8	0.094
N1 (%)	5.1	3.5	4.6	3.1	39	61	0	4.7	3.2	4.8	3.3	52	48	0	2.1	0.146
N2 (%)	49.3	12.4	50.0	10.7	54	46	0	49.4	11.0	47.8	10.0	38	62	0	3.3	0.069
N3 (%)	18.4	8.8	20.5	10.3	48	51	2	18.0	7.9	19.7	8.5	61	38	2	2.2	0.142
R (%)	16.7	6.6	17.2	7.3	51	49	0	15.0	5.8	15.5	6.5	67	33	0	3.4	0.066
WASO (%)	10.5	6.8	7.8	5.5	31	69	0	12.9	10.2	12.1	8.6	46	54	0	2.8	0.094
N1 (min)	22.9	15.4	21.5	15.4	38	62	0	19.6	11.8	21.7	14.6	49	46	5	2.4	0.124
N2 (min)	223.9	64.2	229.9	57.2	64	36	0	213.6	59.6	218.0	55.2	52	46	2	1.4	0.236
N3 (min)	81.8	37.1	93.8	50.0	54	44	2	77.0	33.7	88.9	36.2	62	33	5	1.4	0.243
R (min)	75.7	32.3	78.8	35.7	56	44	0	65.3	29.3	70.2	30.4	64	34	2	1.1	0.298
WASO (min)	47.5	31.3	35.4	25.1	**30**	**69**	2	54.9	43.8	55.6	40.8	**51**	**48**	2	**5.8**	**0.016**
N2_Lat (min)	2.6	2.5	2.6	4.3	39	46	15	3.5	6.8	2.4	3.2	30	41	30	0.2	0.675
N3_Lat (min)	24.1	23.6	23.5	19.0	53	42	5	35.5	30.4	34.0	33.5	45	52	3	1.1	0.304
R_Lat (min)	121.1	50.8	108.7	59.6	36	64	0	137.0	72.2	121.8	66.4	41	59	0	0.3	0.577
Awakenings (#)	22.5	8.0	19.4	7.2	34	64	2	22.3	8.9	24.1	11.7	49	48	3	3.1	0.081
StageTrans (#/hr)	11.0	3.2	10.2	3.1	43	57	0	11.2	3.8	11.4	4.1	56	44	0	2.1	0.147
AI_TST (#/hr)	17.8	6.6	16.6	7.7	34	66	0	19.5	9.8	18.6	7.8	44	56	0	1.2	0.266
AI_N1 (#/hr)	40.3	18.5	37.5	16.4	43	57	0	38.9	17.2	35.7	17.1	51	49	0	0.8	0.364
AI_N2 (#/hr)	16.2	8.1	15.7	9.4	39	61	0	18.9	11.6	17.9	9.0	48	52	0	0.8	0.361
AI_N3 (#/hr)	1.9	1.6	1.4	1.3	37	53	10	2.1	2.5	2.2	1.6	57	42	2	3.2	0.073
AI_R (#/hr)	24.0	13.0	21.4	13.2	**36**	**64**	0	21.5	15.7	21.9	14.7	**57**	**43**	0	**5.6**	**0.018**

Note: Also shown are percentages of participants within each group exhibiting night-to-night increases (↑), decreases (↓) or identical (=) values, and the results of chi-square tests comparing increase/decrease percentages between groups ($p < 0.05$ indicated in bold).
Abbreviation: AI, arousal index; SE, sleep efficiency; SOL, sleep-onset latency; TIB, time in bed; TSP, total sleep period; TST, total sleep time; WASO, wake after sleep onset.

architecture variables by Group (Control versus Insomnia) and Night (1 and 2). Details of the full and pairwise models are presented in Tables S1 and S2, respectively.

The full model yielded a main effect of Group on five outcome measures (Figure 1a), with patients showing reduced TSP, TST and SE_TIB, as well as increased SOL and N3_Lat relative to controls. Separate post hoc group comparisons for the 2 nights (Figure 1b) showed consistent group differences for TST, SE_TIB and N3_Lat on both nights, while effects on TSP and SOL were significant only on night 1. On night 2, patients also showed decreased SE_TSP, as well as enhanced WASO%, WASO_Dur, Awakenings and AI_N3 relative to controls, effects not significant on night 1 or in the full model.

The full model showed a main effect of Night for seven variables (Figure 1c), with reduced TST, SE_TSP and N3_Dur, as well as increased WASO%, WASO_Dur, Awakenings and AI_N3 during the first night. Separate post hoc night comparisons for the two groups (Figure 1d) indicated that only TST and N3_Dur were consistently shorter during the first night in both controls and patients. FNEs found in the full model for the five other variables remained significant in the control group only, and no other variables with FNEs specific to controls appeared. In contrast, three FNEs specific to patients appeared, being reduced TIB and TSP, as well as increased SOL.

The full model Group*Night interactions (Figure 1e) showed differential FNEs for SOL (more pronounced in patients) and Awakenings (more pronounced in controls). In a complementary approach, individuals were categorized according to whether their values on a sleep variable increased, decreased, or remained unchanged from night 1 to night 2 (Table 1). Proportions of increasers and decreasers were

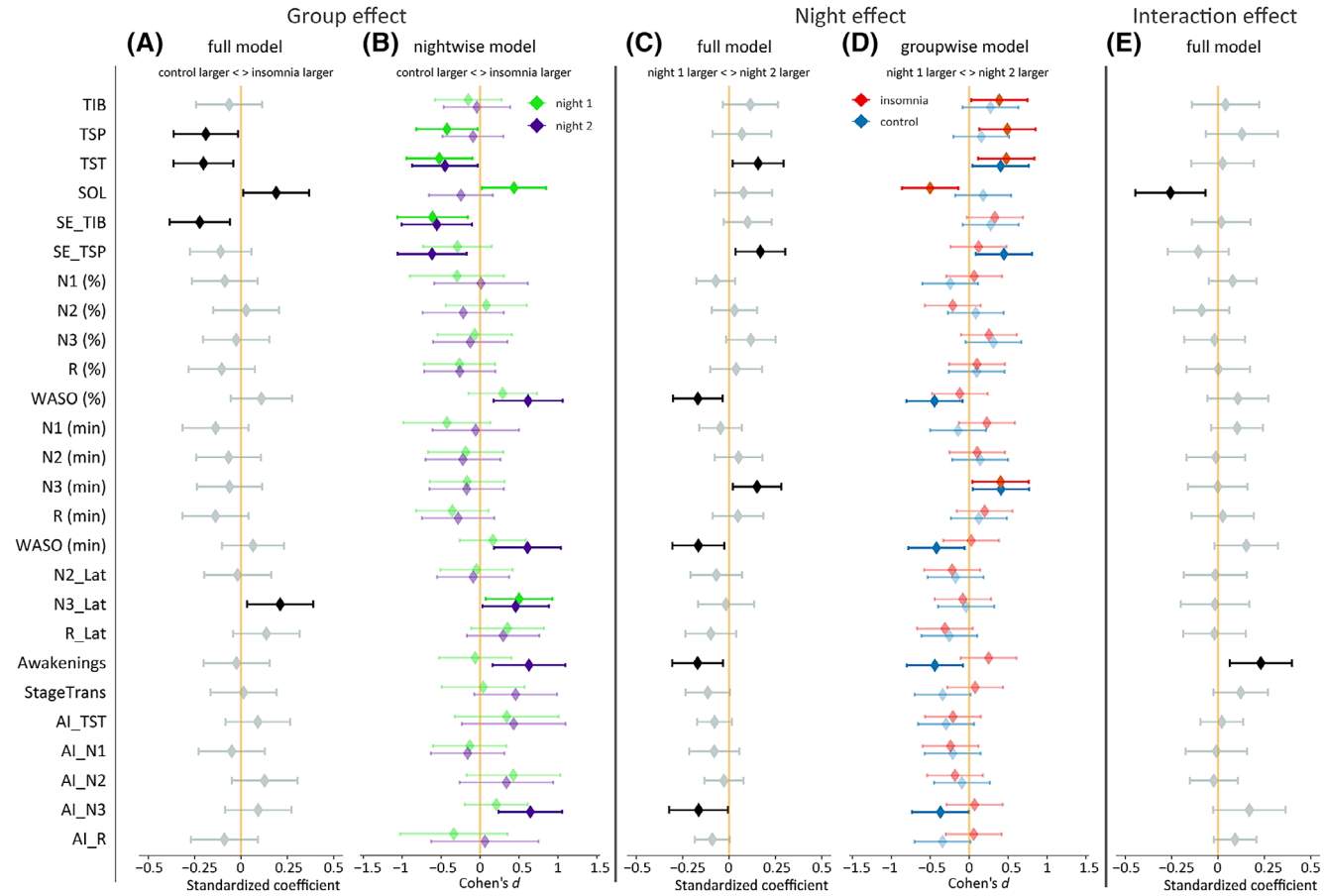

FIGURE 1 Effect size estimates with 95% confidence intervals for full and pairwise models. (a) Main effect of Group in full model. (b) Effect of Group, separated by Night. (c) Main effect of Night in full model. (d) Effect of Night, separated by Group. (e) Group*Night interaction effect in full model. Effects with uncorrected *p*-values below/above 0.05 indicated in black/grey (full model) or saturated/desaturated colours (pairwise models)

	Control ($N = 61$)		Insomnia ($N = 61$)		Comparison	
Night	**Short**	**Normal**	**Short**	**Normal**	χ^2	***p***
Night 1 (%)	12 (19.7%)	49 (80.3%)	24 (39.3%)	37 (60.7%)	5.7	**0.02**
Night 2 (%)	6 (9.8%)	55 (90.2%)	20 (32.8%)	41 (67.2%)	9.6	**0.002**
Condition (night 1–night 2)						
Short-short (%)	2 (3.3%)		14 (23.0%)		10.4	**0.001**
Short-normal (%)	10 (16.4%)		10 (16.4%)		0	1
Normal-short (%)	4 (6.6%)		6 (9.8%)		0.4	0.51
Normal-normal (%)	45 (73.8%)		31 (50.8%)		6.8	**0.009**

TABLE 2 Number and percentages of participants within each group expressing short/normal sleep duration for each of the 2 nights (top half), or each sequence across nights (bottom half), and the results of chi-square tests comparing proportions between groups

Note: Bold values indicate statistical significance.

generally similar between groups, but patients more often showed a decrease in SOL, while controls more often showed decreases in WASO_Dur and AI_R.

Covariates age and sex were associated with 13 and 6 outcome variables, respectively (Table S1), but removal of covariates from the models did not alter the pattern of results (Table S3).

Finally, we considered the occurrence of short (TST < 6 hr) and normal sleep duration subtypes across nights and groups. Whereas 39.3% (24/61) of patients with insomnia presented with short sleep during their first laboratory night (Table 2, top), almost half of them (41.7%, 10/24) no longer showed short sleep on night 2 (short-short: 23.0% [14/61]; short-normal: 16.4% [10/61]; Table 2, bottom). In contrast, of the 60.7% (37/61) of patients starting with a normal sleep duration, only 16.2% (6/37) shifted to short sleep on night 2 (normal-short: 9.8% [6/61]; normal-normal: 50.8% [31/61]).

4 | DISCUSSION

Overall, our findings confirm that patients with insomnia have poorer objective sleep relative to controls, with reduced TST and increased N3_Lat seen consistently across nights. At the same time, the outcome measures distinguishing groups vary between nights, with case–control differences being expressed across a larger set of variables on night 2.

We also confirm that the first night in the sleep laboratory negatively impacts sleep, with similar first-night reductions in TST and N3_Dur across patients with insomnia and controls. However, groups expressed FNEs in different ways. Across nights, SOL decreased for patients but not controls, whereas Awakenings decreased for controls but not patients. Similar results emerged from categorical analyses, which additionally revealed that the share of individuals showing reductions in WASO_Dur and AI_R was larger in controls than patients. More broadly, separate comparisons for the two groups indicated night-to-night changes occur across a larger set of variables for controls than patients. All told, our data do not support strong claims of attenuated FNEs in insomnia (Toussaint et al., 1995), nor of an absence of group differences (Edinger et al., 1997; Hirscher et al., 2015; Newell et al., 2012). Rather, FNEs are present in both groups but with qualitative differences regarding the precise variables expressing an effect.

Based on a single night of PSG, Vgontzas and Fernandez-Mendoza proposed an insomnia subtype with objective short sleep duration (Vgontzas & Fernandez-Mendoza, 2013). Our findings as well as a previous report (Johann et al., 2017) indicate that a large share (42% and 68%, respectively) of insomniacs showing short sleep on night 1 no longer show short sleep on night 2. These findings indicate that a considerable portion of insomniacs presumed to have a short sleep subtype do not chronically suffer from short sleep. Insomniacs' short sleep during the first PSG night could be reflective of an increased insomnia response to stress (Drake et al., 2004). Alternatively, it could merely reflect the additive effects of Group and Night on TST, such that patients are particularly likely to meet the TST < 6 hr threshold on night 1. Only SOL showed a Group*Night interaction such that the FNE was exacerbated in patients, so this feature might reflect the insomnia response to stress more sensitively.

Overall, modest group differences confirm that standard PSG metrics are of limited use for directly diagnosing insomnia disorder. Equally modest night differences in PSG metrics suggest valid insights may be obtained from a single night for many purposes, but multi-night recordings may nonetheless be valuable to refine insomnia subtyping based on sleep duration.

AUTHOR CONTRIBUTIONS

Conceptualization: Roy Cox, Lara Rösler, Frederik D. Weber, Tessa F. Blanken, Eus J.W. Van Someren. Data collection: Rick Wassing, Jennifer R. Ramautar. Analysis: Lara Rösler, Roy Cox, Frederik D. Weber. Interpretation: Roy Cox, Lara Rösler, Frederik D. Weber, Tessa F. Blanken, Rick Wassing, Jennifer R. Ramautar, Eus J.W. Van Someren. Writing: Roy Cox (with input from all authors). Funding acquisition: Eus J.W. Van Someren. All authors approved the final version of the manuscript.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data and R code for running analyses and reproducing tables and plots are available from http://doi.org/10.17605/OSF.IO/57EY6

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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