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Electroencephalography during on-the-road driving in older untreated insomnia patients and normal sleepers

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ABSTRACT

Insomniacs report decreased performance in daily routines, which may have detrimental consequences for car driving. We compared changes over time in driving performance (measured as Standard Deviation of Lateral Position – SDLP) and background EEG between 20 untreated insomnia patients (52–70 years old) and 21 normal sleepers (54–73 years old) during a 1 h on-the-road driving test after a normal night of sleep, in the morning. SDLP did not differ between groups and increased slightly over time to similar degrees in both groups. EEG alpha and beta power were lower in insomniacs as compared to normal sleepers. Alpha and beta power slightly reduced during driving in normal sleepers but remained at a constant low level in insomniacs. Changes in EEG power and SDLP were not related. It is concluded that on-the-road driving performance does not differ between older insomniacs and older normal sleepers and that changes in spectral EEG measures of cortical arousal and in driving performance are not related.

1. Introduction

Insomnia is one of the most common complaints seen in medical practice. Approximately one-third of the general population experiences some insomnia symptoms occasionally, and it is estimated that approximately 10% suffers from chronic insomnia (Morin, LeBlanc, Daley, Gregoire, & Mérette, 2006; Ohayon, 2002). Insomniacs report reduced performance in daily routines, which may have detrimental consequences in particular for potentially hazardous activities such as car driving. A number of studies have shown that the consequences of sleep problems include decreased work productivity, increased risk for accidents, increased health care costs (Daley et al., 2009; Hamblin, 2007; Smolensky, Di Milia, Ohayon, & Philip, 2011; Uehli et al., 2014). Specific impairment of driving performance and increased risks for traffic accidents has been shown for sleep disturbances, such as obstructive sleep apnea, periodic limb movement disorder and sleep deprivation (e.g. Bosker et al., 2012; Gieteling et al., 2012; Philip et al., 2010; Smolensky et al., 2011). The link between insomnia and driving impairment is less clear, however (Daley et al., 2009; Komada, Asaoka, Abe, & Inoue, 2013; Léger, Massuel, & Metlaine, 2006; Philip et al., 2010; Sagaspe et al., 2010). Few epidemiological studies have found increased traffic accident risks in insomnia patients (Laugsand, Strand, Vatten, Janszky, & Bjørngaard, 2014; Léger et al., 2013), while several others do not (Lucidi, Mallia, Violani, Giustiniani, & Persia, 2013; Philip et al., 2010; Sagaspe et al., 2010; Fragoso, Araujo, Van Ness, & Marottoli, 2010). The inconsistent findings in epidemiological research are in line with those of experimental research. Experimental studies have generally failed to establish objective evidence of cognitive dysfunction, despite insomnia patients' subjective feelings of fatigue and cognitive impairments (Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2011; Shekleton, Rogers, & Rajaratnam, 2010).

Recently, two studies comparing driving performance of insomnia patients to that of normal sleepers, found significant impairment in a driving simulator task, but no differences in an

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on-the-road driving test (Leufkens, Ramaekers, de Weerd, Riedel, & Vermeeren, 2014; Perrier et al., 2014). Leufkens and colleagues (Leufkens et al., 2014) compared sleep and performance between a group of 20 older untreated insomnia patients, a group of 22 chronic users of hypnotics and a group of 21 age-matched normal sleepers, using a one-hour on-the-road highway driving tests and a battery of tests measuring driving related cognitive and psychomotor performance. Results showed no significant differences between groups in performance, despite significant sleep complaints in the insomnia patients. In contrast, Perrier and colleagues (Perrier et al., 2014), using a driving simulator, found that insomnia patients performed worse than normal sleepers. The impairment found in insomnia patients seemed to be due to a performance decrement in the second half of the test, i.e. after 20 min of monotonous driving on a highway. This suggests that insomnia may show more pronounced vigilance decrements than normal sleepers during long and monotonous tasks, such as a simulated highway driving task.

The lack of impairment during real driving in the study by Leufkens and colleagues (Leufkens et al., 2014) raises the question whether this may have been due to hyperarousal in patients. Current models of primary insomnia assume that patients are hyperaroused or unable to de-arouse, which would account for their difficulties in initiating and maintaining sleep but also for the absence of daytime sedation and associated performance impairment (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997; Riemann et al., 2010). Several studies have looked at EEG markers of arousal during sleep in insomnia patients and have found increased power in the beta and sigma frequency band around sleep onset and during NREM sleep, reflecting increased cortical activation in patients as compared to normal sleepers (see Feige et al., 2013, for review). Only two studies have been published evaluating daytime waking EEG in insomnia patients. In line with findings during sleep, Wolynczyk and Szelenberger (Wolynczyk-Gmaj & Szelenberger, 2011) report increased beta power during daytime Multiple Sleep Latency Tests, whereas Wu and colleagues (Wu et al., 2013) report no significant differences between patients and normal sleepers in 5-min resting EEG recordings with eyes open and eyes closed. It is therefore not clear whether EEG in insomnia patients during task performance such as driving will show changes as indication of increased arousal.

A few studies evaluated background EEG during on-the-road driving in healthy volunteers and found that brain activation decreases (i.e. increases in theta and alpha power spectra) with time-on-task (Brookhuis & de Waard, 1993; Schmidt, Schrauf, Simon, Buchner, & Kincses, 2011), during night-time driving (Akerstedt et al., 2013; Kecklund & Akerstedt, 1993; Sandberg et al., 2011), and after sleep deprivation (Papadelis et al., 2007). Similar changes in EEG have been reported by investigators using driving simulators (e.g. Filtness, Reyner, & Horne, 2012; Horne & Baulk, 2004; Jagannath & Balasubramanian, 2014; Lal & Craig, 2002; Phipps-Nelson, Redman, & Rajaratnam, 2011). EEG changes such as increases in alpha and theta activity, and decreases in beta activity, are often associated with decreased alertness, increased drowsiness and worse performance (Oken, Salinsky, & Elsas, 2006). Until now, no study compared EEG during driving between insomnia patients and normal sleepers.

The purpose of the present study was to assess whether changes in spectral EEG measures are related to changes in driving performance in older insomnia patients. It was expected that insomnia patients on average show increased cortical arousal during driving as compared to normal sleepers, in particular more beta power, and less alpha and theta power. It was also expected that insomniacs do not display increased indicators of fatigue and sleepiness (i.e. theta and alpha); contrary to normal sleepers. In addition it is expected that impaired driving in patients is associated with reduced beta power, rather than increased alpha or theta power compared to normal sleepers.

2. Methods

2.1. Design

Data for this paper were collected as part of study that compared sleep, on-the-road driving performance and driving related skills between older untreated insomnia patients, chronic users of hypnotics, and age-matched normal sleepers (Leufkens et al., 2014). For the present study EEG recordings during driving were analyzed and compared between two groups, i.e., untreated insomnia patients and normal sleepers.

2.2. Participants

The insomnia group comprised 20 healthy older adults (10 men, 10 women; 52–70 years old; mean ± SD age 60.8 ± 5.8 years) with insomnia, and not using sleep promoting agents for more than 3 nights per week. The control group comprised 21 normal sleepers (13 men, 8 women; 54–73 years old; mean ± SD age 61.7 ± 5.0 years). Participants were recruited via advertisement in local newspapers and through a network of local general practitioners in the region of Maastricht.

All participants had to meet the following inclusion criteria: aged between 50 and 75 years; possession of a valid driving license; average driving experience of at least 3000 km per year over the last 3 years; good health based on a pre-study physical examination, medical history, vital signs, electrocardiogram, blood biochemistry, hematology, serology and urinalysis; body mass index (BMI) between 19 and 30 kg/m^2. Exclusion criteria were history of drug or alcohol abuse; presence of a significant medical, neurological, psychiatric disorder, or sleep disorder other than insomnia; chronic use of medication that affects driving performance; for patients, use of hypnotics for more than 3 nights per week; drinking more than 6 cups of coffee per day; drinking more than 21 alcohol containing beverages per week; smoking more than 10 cigarettes per day.

Insomnia patients had to meet the criteria for primary insomnia according to DSM IV (Association & DSM-IV, 2000): (a) subjective complaints of insomnia, defined as difficulties initiating sleep (sleep latency > 30 min) and/or maintaining sleep (awakenings > 30 min); (b) duration of more than 1 month; (c) the sleep disturbance causes clinically significant distress or impairment; (d) insomnia does not occur exclusively during the course of a mental disorder and (e) insomnia is not due to another medical or sleep disorder or effects of medication or drug abuse. Participants in the control group were self-defined normal sleepers. Sleep complaints and psychopathology were evaluated by a trained psychologist using Dutch versions of validated questionnaires. In addition, sleep was evaluated during two nights of polysomnography; first during a habituation night and next during the night before the driving test. Self-report data on sleep showed that insomniacs rated their sleep significantly worse than normal sleepers (Leufkens et al., 2014). Polysomnography did also not reveal significant differences between groups in sleep duration or architecture (Leufkens et al., 2014).

During participation, use of caffeine was prohibited from 8 h prior to arrival on test days, until discharge the next morning. Alcohol intake was not allowed from 24 h prior to bedtime until discharge. Smoking was prohibited from 1 h prior to bedtime until discharge. Insomnia patients were not allowed to take hypnotic medication the night before driving.

The study was conducted in accordance with the code of ethics on human experimentation established by the World Medical Association's Declaration of Helsinki (1964) and amended in Edinburgh (2000). The protocol was approved by the medical ethics committee of Maastricht University and University Hospital of Maastricht. Participants were explained the aims, methods, and potential hazards of the study and they signed a written informed consent prior to any study-related assessments.

2.3. Procedure

All participants completed two nights of sleep evaluation and testing. The first night was a habituation and practice condition to familiarize participants with the sleeping facilities, polysomnography and test procedures, including a complete dress-rehearsal of the driving test. The second night was for actual sleep and performance assessments. Test conditions started in the evening when participants arrived at the site at 7.00 pm. Electrodes for polysomnographic recording were attached at 9.00 pm and participants retired to bed at 11.30 pm. The following morning participants were awakened at 7.30 am by an experimenter and served a standardized light breakfast. At 9.00 am they were transported to the highway to perform the on-the-road driving test between 9.30 and 10.30 am. Upon completion participants returned to the testing facilities for removal of the electrodes and were discharged. All participants completed the study between December 2007 and February 2009. Normal sleepers were enrolled largely in parallel with insomnia patients, with only a few weeks delay to allow matching for age and gender of the control group.

2.4. Assessments

2.4.1. On-the-road highway driving test

Driving performance was assessed using a standardized on-the-road highway driving test (O'Hanlon, 1984) which measures road tracking performance. In this test, participants operate a specially instrumented vehicle for approximately 1-h over a 100-km (61 miles) primary highway circuit in normal traffic, accompanied by a licensed driving instructor having access to dual controls (brakes and accelerator). The participants' task is to drive with a steady lateral position between the delineated boundaries of the slower (right) traffic lane, while maintaining a constant speed of 95 km/h (58 mph). Participants may deviate from those instructions only to pass a slower vehicle, and to leave and re-enter the highway at the turnaround points. During the drive, the vehicle's speed and lateral distance to the left lane line are continuously recorded. These signals are digitized at a rate of 4 Hz and stored on an onboard computer for later preprocessing and analysis. The signals are edited off line to remove data recorded during overtaking maneuvers or disturbances caused by roadway or traffic situations. The remaining data are then used to calculate means and standard deviations of lateral position and speed. For the present study, the dataset was split in 6 successive periods of 10 min in order to analyze performance changes with Time-on-Task (ToT). The primary outcome variable is SDLP in cm, which is an index of weaving.

2.4.2. Electroencephalography

EEG was recorded with a Vitaport portable EEG recorder at four electrodes (F4, C4, O2 and C3) with a common average (A1–A2). EEG was continuously recorded during the driving test. Data were sampled at 256 Hz and was low pass filtered at 70 Hz. In addition, a 50 Hz notch filter was applied to remove artifacts linked to electrical activity in the car. After segmentation of data into 2-s epochs data were visually inspected for artifacts that were subsequently rejected. Each epoch was then subjected to a fast Fourier transformation using a Hanning window of 10% in order to obtain the power spectrum. The following band frequencies were analyzed and quantified in absolute power for 6 consecutive 10-min periods of the driving test: theta (4–8 Hz), alpha (8–13 Hz) and beta (13–30 Hz).

EEG recordings of 9 participants were not included in the analysis: 3 participants had no valid EEG data due to technical problems during recording, data from 3 participants could not be used because of too many movement artifacts, and data from 3 participants were excluded because of outliers (mean ± 2SD). In total EEG data sets were analyzed for 32 participants: 17 insomnia patients (8 men, 9 women; 55–70 years old mean ± SD age 61.6 ± 5.8 years) and 15 normal sleepers (7 men, 8 women; 54–68 years old; mean ± SD age 60.7 ± 4.8 years). Driving data were analyzed for 34 participants: 17 insomnia patients (9 men, 8 women; 54–70 years old; mean ± SD age 61.41 ± 5.98 years) and 17 normal sleepers (9 men, 8 women; 55–68 years old; mean ± SD age 61.60 ± 4.30 years). For 5 participants driving data were omitted from analysis, because there were no corresponding EEG data. For 2 participants the datafiles could not be retrieved for analysis. Analysis of a potential linear relationship between driving performance and EEG was conducted on participants for which both driving and EEG data were available i.e. 30 participants including 16 insomnia patients (7 men, 9 women; 55–70 years old; mean ± SD age 61.88 ± 5.85 years) and 14 normal sleepers (6 men, 8 women; 55–68 years old; mean ± SD age 61.14 ± 4.57 years).

2.5. Statistical analysis

Statistical analyses were carried out using the software R 2.15.0 (www.r-project.org). The driving and EEG data that were analyzed are of longitudinal structure with both random (participant) and fixed (Group, ToT) effects. To capture these effects, we selected linear mixed effects models as methodological approach. More precisely, the models analyzed are of ANCOVA type. That is, in case of full interaction the basic model form is:

$$Pow_{EEG} = \beta o(\mathrm{Gr}) + b + \beta_1(\mathrm{Gr}) * TOT + \varepsilon,$$

where Pow_{EEG} denotes the response variable, β_o the group-varying intercept, β_1 the group-varying slope coefficient, *ToT*, the time-on-task covariate, and ε the error term, following a Gaussian distribution with expected value zero. In addition to a classical ANCOVA, the term b represents a Gaussian random effect and captures between-subject variability in the intercept. The model would also allow for the inclusion of random effects in the slope coefficient, which proved not to be necessary for obtaining a better model fit.

Mixed effects models are versatile, and able to take different data characteristics into account. In order to build models suited to the different data sets analyzed, we carried out the following steps:

1. As a preliminary step, we investigated the residuals of various models. It turned out that serial within-subject correlation was present for most series. Therefore, we investigated different correlation structures of the error term ε. Taking the Bayesian information criterion (BIC) as selection criterion, the for time series typical AR(1) error structure was selected if indicated by the criterion. Moreover, the correlation structure was verified on the finally selected model for each data set.
2. Following a similar model selection approach as described in the first point, we investigated different variance models accounting for potential heteroscedasticity of the error term ε. The variance functions investigated were: fixed variance, different variances for normal sleepers and insomniacs, ToT-varying variance, as well as group- and ToT-varying variance. The most appropriate variance structure was selected by means of the BIC.
3. When building a model, one has to evaluate if a variable entering a model has a significant effect. We followed the approach of Pinheiro and Bates (2000), and compared models with and without the respective variable by means of a likelihood ratio test (LRT). To do this, the starting point for each analysis always was the simplest model, including only a constant intercept β_0 and random effect b. Then, we added the strongest fixed effect (e.g., time-on-task, resulting in a slope potentially unequal to zero) to the model and checked if the LRT justifies the inclusion of this additional variable. If so, we continued by adding the next variable (e.g., the group effect), either additively (i.e., affecting the intercept) or in interaction with the previously included variable (i.e., affecting the slope), and compared the resulting models by LRT and BIC, until the best model was selected.
4. After carrying out the previous steps for all data sets, and determining the best model for each, we accounted for the effect of multiple comparisons: the p-values of the LRT of the final models were adjusted by the Holm–Bonferroni method. These results are represented by the associated p^{LRT} value.
5. Only when the LRT a model indicated a significant effect of one or several variables (ToT, group, or ToT and group interaction), the p-values of the coefficients (β_0, β_1) of this model were further examined.

Potential dependencies between EEG and SDLP data were investigated by treating both variables in a linear mixed effects model similar to the one previously described. In this case, the model equation was$SDLP_= \beta_o(\mathrm{Gr}) + b + \beta_1(\mathrm{Gr})^* \mathrm{ToT} + \varepsilon$,

$$\mathrm{SDLP} = \beta_o(\mathrm{Gr}) + b + \beta_1(\mathrm{Gr}) * \mathrm{TOT} + \varepsilon,$$

that is, only the response variable changed.

For all figures representing longitudinal data, we applied the approach described by Cousineau (2005). Consequently, error bars in these figures do not take variability associated with between-subject differences into account.

3. Results

3.1. Driving performance

Mean SDLP scores in both groups for 6 consecutive 10-min intervals of the driving test are presented in Fig. 1.

Results showed only a significant effect of ToT ($p^{LRT} = 0.001$). There was no significant difference between groups, and no significant interaction between Groups and ToT. The model coefficients indicate that SDLP increased with ToT ($p = 0.001$).

3.2. Electroencephalography

Table 1 shows details for the best models selected if these contained any significant fixed effects in alpha, theta and beta frequency bands.

In the theta band, no model fitted the data, indicating that there were no significant effects of group and ToT, and no interaction (see Fig. 2).

Analysis of the alpha power showed significant effects of group, ToT, and interactions of both factors ($p^{LRT} < 0.001$, Fig. 3). The model coefficients revealed that alpha power at O2 was lower in insomnia patients than in normal sleepers (2.70 vs 4.67 μV^2, $p = 0.011$). In addition, alpha power decreased with ToT in normal sleepers ($-0.22\ \mu V^2$/interval, $p < 0.001$), but not in insomnia patients ($-0.03\ \mu V^2$/interval, $p < 0.001$). There was also a significant effect of the ToT on alpha power at C3 ($p^{LRT} = 0.005$). Model coefficients indicate that alpha power increased by $0.085\ \mu V^2$ per interval at C3 for both groups ($p < 0.001$).

Analysis of power in beta frequency band showed significant effects of group, ToT, and interactions between both factors (at O2, $p^{LRT} < 0.001$; at C4, $p^{LRT} = 0.005$; at F4, $p^{LRT} = 0.026$) (See Figure 4).

Model coefficients indicated that beta power was lower in insomnia patients than in normal sleepers (at O2: 2.15 vs 4.02 μV^2, $p = 0.003$; at C4: 5.85 vs 9.06 μV^2, $p = 0.019$; at F4 5.93 vs 9.66 μV^2,

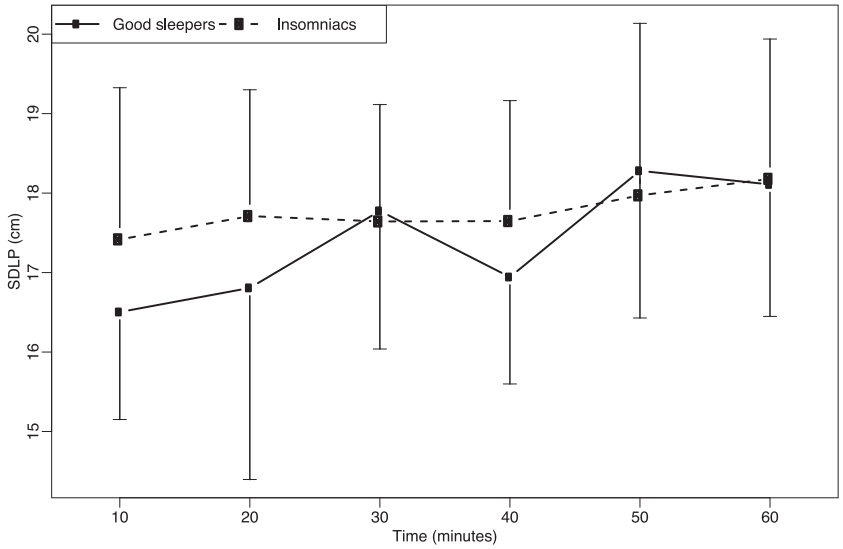

Fig. 1. Driving performance of insomnia patients and normal sleepers as measured by Standard Deviation of Lateral Position (SDLP, in cm) for each consecutive 10-min interval of the on-the-road highway driving test (means ± SD).

$p = 0.092$). The effects of ToT differed significantly between insomnia patients and normal sleepers (at O2, C4 and F4, all p's ≤ 0.001). Beta power decreased significantly with ToT in normal sleepers (at O2, $-0.24\,\mu V^2$ per 10 min interval, $p < 0.001$; at C4, $-0.41\,\mu V^2$/interval, $p = 0.001$; at F4, $-0.41\,\mu V^2$/interval, $p = 0.001$) but not in insomnia patients (at O2, $-0.02\,\mu V^2$/interval, $p = 0.001$; at C4, $-0.01\,\mu V^2$/interval, $p = 0.001$; at F4, $+0.16\,\mu V^2$/interval, $p < 0.003$). Model coefficients are presented in Table 1.

3.3. Linear relation between SDLP and EEG measures

Investigation of a relationship between EEG and SDLP data with a mixed model revealed that none of the EEG measures shows any predictive power for SDLP except for the beta band in the electrode C4. However, this result does not seem relevant as no significant relationship was found for C3 as well as no group difference (see Table 2).

Table 1
Significant results in detail for the model coefficients (group effect, time on task, and interaction).[a] The estimated values, standard errors and statistical results are presented only for significant LRT results (column p^{LRT} after Holm correction). LRT, likehood ratio test; SDLP, Standard Deviation of Lateral Position; ToT, time-of-task; df, degree of freedom.

		p^{LRT} after Holm correction	Model coefficients (β_o, β_1)	Estimated values	Standard error	df	t =	P =
SDLP		0.0012	(Intercept)	16.77	0.54	169	30.90	<0.001
			ToT	0.23	0.09	169	3.33	0.001
	Electrodes							
Alpha band	O2	<0.001	(Intercept)	4.67	0.54	157	8.70	<0.001
			Group (insomniacs)	−1.97	0.73	30	−2.71	0.011
			ToT normal sleepers	−0.22	0.05	157	−4.71	<0.001
			ToT insomniacs	0.19	0.06	157	3.26	0.001
	C3	0.0052	(Intercept)	3.47	0.42	159	8.23	<0.001
			ToT	0.09	0.02	159	3.78	<0.001
Beta band	O2	<0.001	(Intercept)	4.02	0.42	158	9.55	<0.001
			Group (insomniacs)	−1.87	0.58	31	−3.24	0.003
			ToT normal sleepers	−0.24	0.04	158	−5.49	<0.001
			ToT insomniacs	0.23	0.56	158	4.06	<0.001
	C4	0.0047	(Intercept)	9.06	0.95	158	9.55	<0.001
			Group (insomniacs)	−3.21	1.30	31	−4.30	0.020
			ToT normal sleepers	−0.41	0.10	158	−4.30	<0.001
			ToT insomniacs	0.41	0.13	158	3.25	0.001
	F4	0.0258	(Intercept)	9.66	1.55	158	6.23	<0.001
			Group (insomniacs)	−3.73	0.12	31	−3.35	0.092
			ToT normal sleepers	−0.41	0.12	158	−3.35	0.001
			ToT insomniacs	0.57	0.15	158	3.73	<0.001

[a] In the following, we illustrate the interpretation of the model coefficients by means of two examples, the C3 and O2 electrodes from the alpha band. For C3, the best model supported by LRT ($p = 0.0052$) is a rather simple one without group effect, neither as simple additive effect nor in interaction with the ToT. Therefore, the resulting model corresponds to a linear regression with common intercept ($\beta_o = 3.47$) and slope ($\beta_1 = 0.09$) for insomniacs and normal sleepers, both being highly significant ($p < 0.001$). Thus, e.g. the estimated power for both groups for the second interval of length 10 min equals 3.47 + 2·0.09. Group effects are, however, present for O2: here, the intercept for the normal sleepers equals $\beta_o(gs) = 4.67$ ($p < 0.001$), and the intercept of the insomniacs is significantly lower by 1.97 ($p = 0.011$), resulting in an intercept of $\beta_o(ins) = 2.7$ for the insomniacs. As to the slope, the results are similar. Normal sleepers have a significant slope of $\beta_1(gs) = -0.22$ ($p < 0.001$), while the slope of insomniacs is significantly higher by 0.19 ($p = 0.001$), resulting in a slope coefficient of $\beta_1(ins)$ $\beta_0(HLT) = -0.03$.

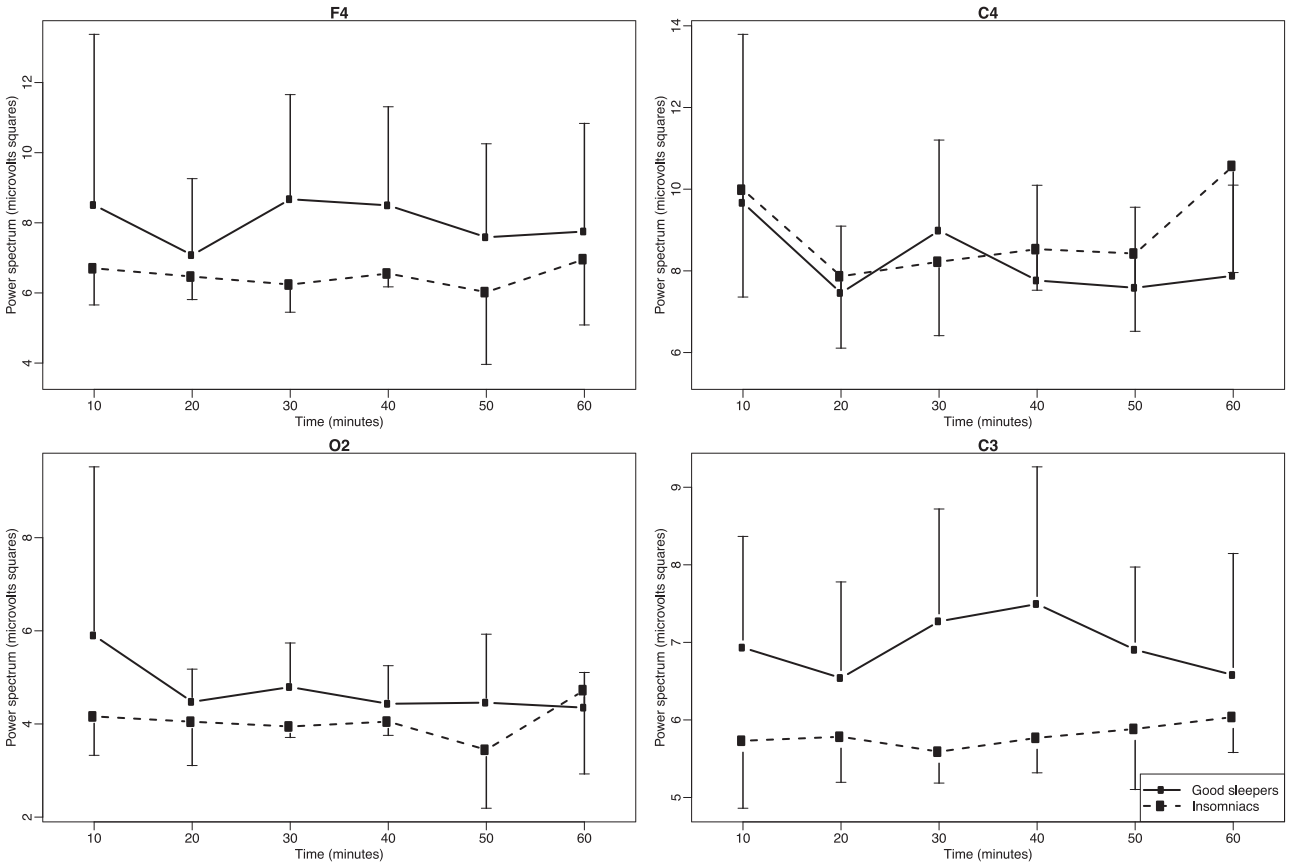

Fig. 2. EEG theta power (in μV^2) at F4, C4, O2, C3 averaged every 10 min of the driving task for both groups (means ± SD).

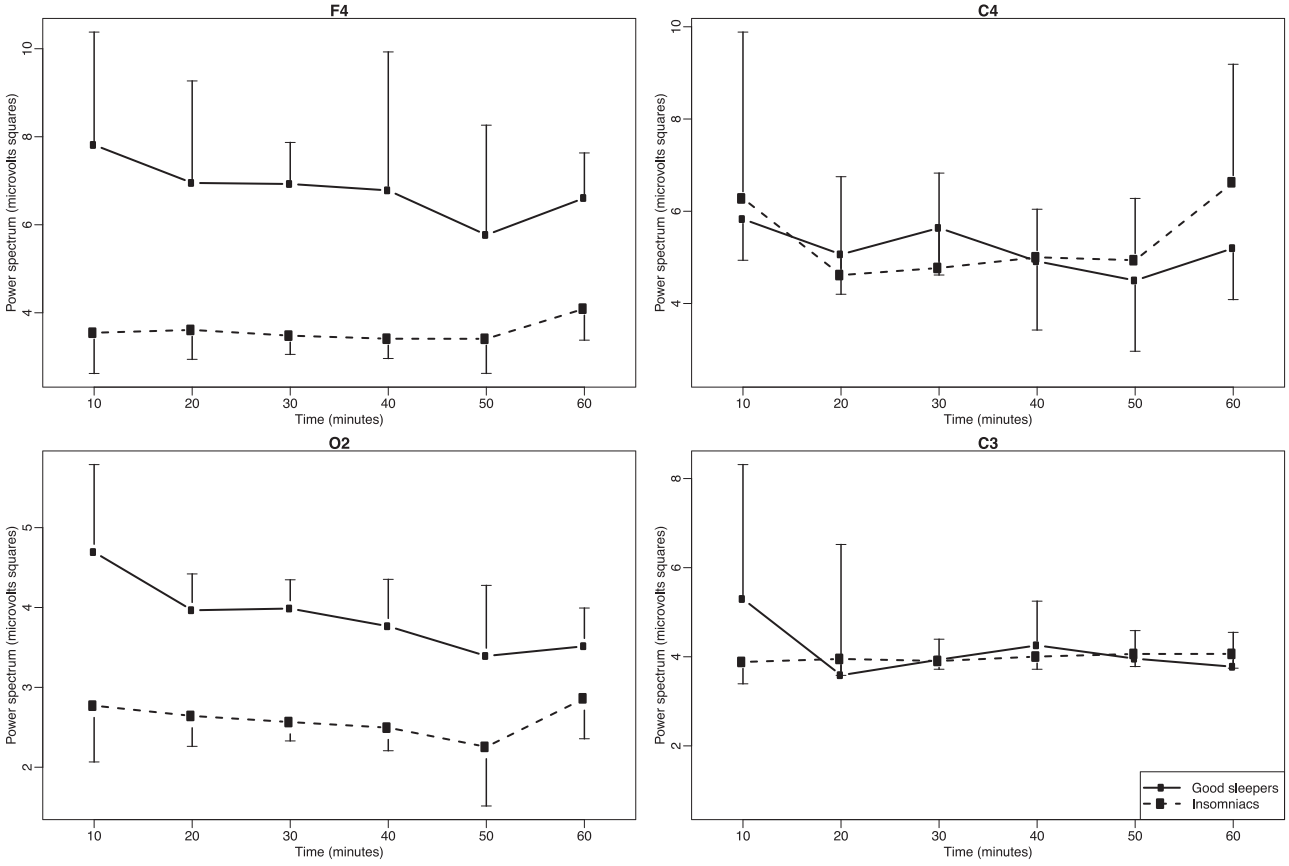

Fig. 3. EEG alpha power spectrum (in μV^2) at F4, C4, O2, C3, averaged every 10 min of the driving task for both group (means ± SD).

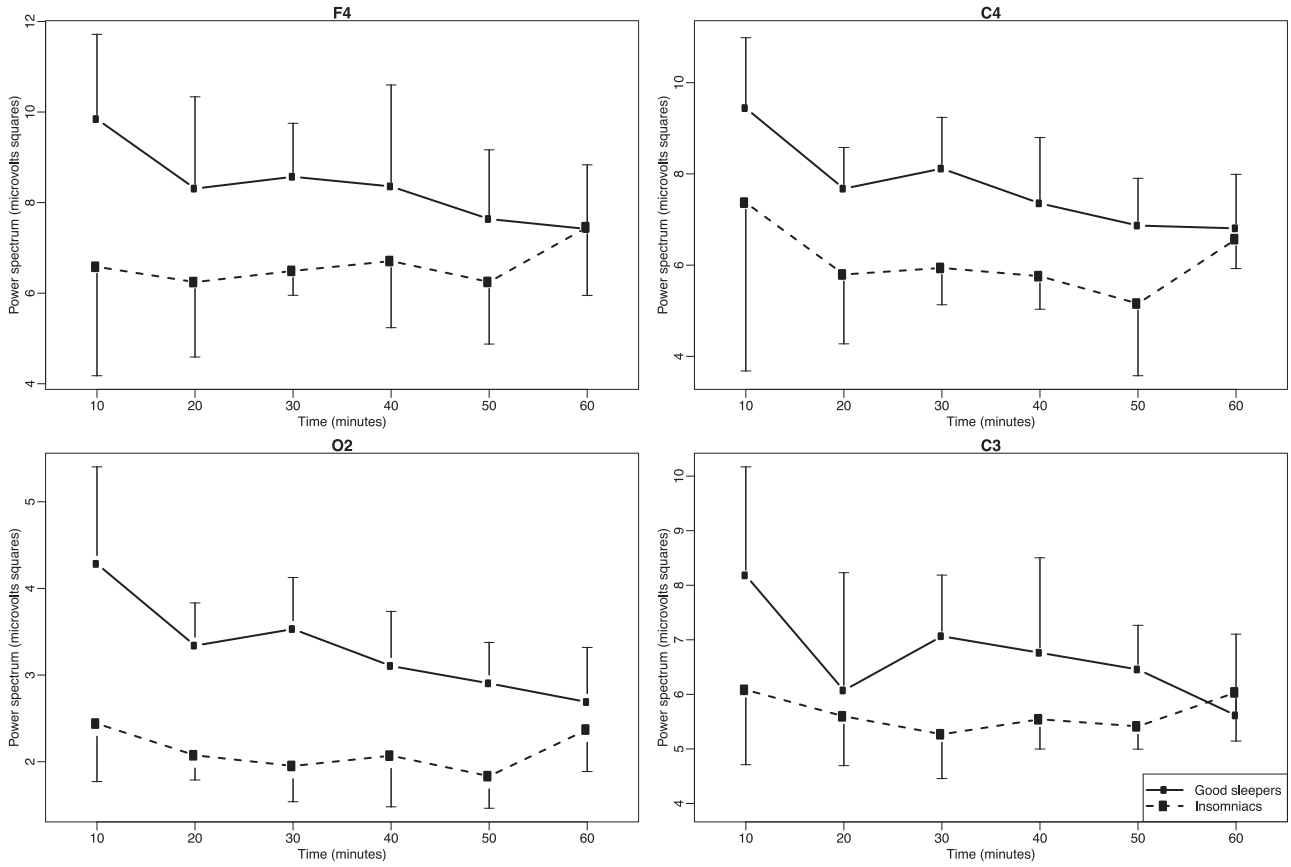

Fig. 4. EEG beta power spectrum (in μV^2) at F4, C4, O2, C3, averaged every 10 min of the driving task for both group (means ± SD).

4. Discussion

The purpose of the present study was to determine whether changes in driving performance are related to changes in cortical activity during driving in insomnia patients and normal sleepers.

Results show that overall SDLP did not differ between groups, indicating that older untreated insomnia patients were able to drive as good as normal sleepers during an on-the-road highway driving task. There was a significant effect of time on task on SDLP, without a significant interaction, indicating similar performance

Table 2
Significant results in detail for the model coefficients (power spectrum effect, time on task and interaction). The estimated values, standard errors and statistical results are presented only for significant LRT results. LRT, Likehood Ratio Test;ToT, Time-of-Task; df, degree of freedom.

		p^{LRT} after Holm correction	Model coefficient (β_o, β_1)	Estimated values	Standard error	df	*t*=	*p*=
	Electrodes							
Theta band	O2	0.012	(Intercept)	16.74	0.62	148	27.18	<0.001
			ToT	0.27	0.083	148	3.24	0.002
	C3	0.012	(Intercept)	16.72	0.60	153	27.86	<0.001
			ToT	0.26	0.081	153	3.20	0.002
	C4	0.012	(Intercept)	16.74	0.62	148	27.18	<0.001
			ToT	0.27	0.083	148	3.24	0.002
	F4	0.012	(Intercept)	16.83	0.67	128	25.23	<0.001
			ToT	0.26	0.090	128	2.90	0.004
Alpha band	O2	0.012	(Intercept)	16.74	0.62	148	27.18	<0.001
			ToT	0.27	0.083	148	3.24	0.002
	C3, C4, F4	0.012	(Intercept)	16.69	0.60	149	27.77	<0.001
			ToT	0.27	0.082	149	3.30	0.001
Beta band	O2, C3,	0.012	(Intercept)	16.69	0.60	149	27.77	<0.001
			ToT	0.27	0.082	149	3.30	0.001
	C3	0.012	(Intercept)	16.69	0.60	149	27.77	<0.001
			ToT	0.27	0.082	149	3.30	0.001
	C4	0.0014	(Intercept)	19.32	0.67	149	28.86	<0.001
			Power spectrum	−0.25	0.069	149	−3.63	<0.001
	F4	0.0085	(Intercept)	16.69	0.60	149	27.77	<0.001
			ToT	0.27	0.082	149	3.30	0.001

decrements during driving in both groups. In contrast to SDLP results, analysis of EEG power spectra revealed significant differences between patients and normal sleepers. Overall, insomnia patients displayed less alpha power and less beta power during driving compared to normal sleepers. Furthermore, patients' alpha and beta power remained relatively constant with ToT, whereas the power in both bands decreased with ToT in normal sleepers. Finally, changes in EEG power spectra were not related to changes in SDLP.

The lack of a relationship between SDLP and fluctuations in background EEG is not in line with our expectation. In the past several studies reported a link between EEG power spectra and SDLP in healthy volunteers (Brookhuis & de Waard, 1993; Lin et al., 2005), but several other studies did not (Akerstedt et al., 2010; Johnson, Dawson, & Rizzo, 2011; Lowden, Anund, Kecklund, Peters, & Akerstedt, 2009; Simon et al., 2011). The failure to find a relationship in the present study could be related to the relatively well-rested state of the participants. In their review of the literature on physiological measures of alertness and sustained attention, Oken et al. (2006) already concluded that the correlation between EEG measures and performance changes on sustained attention tasks, including simulated driving, are "not consistent or large, especially in well-rested participants." Participants in our study were tested in the morning after a normal night of sleep. Most studies trying to identify indices of drowsy driving using EEG, tested healthy young participants after inducing clinically significant levels of drowsiness through sleep restriction, and night-time driving (Akerstedt et al., 2010, 2013; Brown, Johnson, & Milavetz, 2013; Filtness et al., 2012; Sandberg et al., 2011; Schmidt et al., 2011). For example, Johnson et al. (2011) conducted a study to determine if an individualized EEG-based algorithm could be defined to track performance decrements associated with sleep loss. They concluded that, although the system could effectively track performance decrements associated with sleep deprivation, it did not work for well-rested drivers. In addition, participants in our study were older drivers, whereas most studies have used younger volunteers. In a study by Lowden and colleagues (Lowden et al., 2009) comparing simulated night-time driving performance and EEG in young and older drivers, a significant relation between driving performance and brain activity was found in young drivers, but not in older drivers. It is known that young drivers are generally more sensitive to the effects of sleep deprivation, than older drivers (Filtness et al., 2012). The individual variation in physiological parameters may be too large to detect subtle changes during daytime driving in older insomnia patients.

To our knowledge, this is the first study comparing background EEG during driving performance (i.e. eyes open and participants mentally active) between insomnia patients and normal sleepers. Contrary to studies showing elevated beta power in insomniacs during sleep (e.g. Spiegelhalder et al., 2012), we found that beta power during driving was overall lower in patients than in normal sleepers. However, alpha power was also lower in patients than in normal sleepers. It is not clear, therefore whether patients were more aroused during driving than normal sleepers. Although the lower levels of alpha support the idea that patients were less drowsy during driving, the lower levels of beta power do not support the theory of hyperarousal. It may be possible that daytime waking hyperarousal in insomnia is not reflected in increased beta power, but rather as reduced alpha levels or increases in higher frequencies, such as gamma. However, further studies are needed to confirm this assumption.

It is noteworthy that low levels of alpha and beta power in insomnia patients were very stable during the driving test and did not change with ToT. This seems to indicate that EEG power spectra in these patients reflected the underlying state of insomnia rather than any state related changes in arousal or attention that usually arise during a prolonged driving task. The majority of previous studies which investigated EEG measures during driving tasks in non-clinical populations have found slowing of cortical activity with ToT, reflected by relative decreases in beta (Jagannath & Balasubramanian, 2014; Jap, Lal, Fischer, & Bekiaris, 2009; Papadelis et al., 2007; Zhao, Zhao, Liu, & Zheng, 2012), or increases in alpha and theta power (e.g. Akerstedt et al., 2010; Brookhuis, Louwerens, & O'Hanlon, 1986; Campagne, Pebayle, & Muzet, 2004; Craig, Tran, Wijesuriya, & Nguyen, 2012; Filtness, Reyner, & Horne, 2011; Horne & Baulk, 2004; Jagannath & Balasubramanian, 2014; Lal & Craig, 2002; Lowden et al., 2009; Otmani, Pebayle, Roge, & Muzet, 2005; Simon et al., 2011; Zhao et al., 2012). In line with this we found a decrease in beta power with ToT in the normal sleepers group. SDLP in this group slightly increased as well with ToT, suggesting a vigilance decrement. Contrary to previous findings, however, we also found a decrease in alpha power in normal sleepers. Although concurrent reductions in alpha and beta power during monotonous driving have been reported before (Jap et al., 2009), this pattern of changes does not fit the hypothesis of general slowing of cortical activity with ToT. Therefore it does not support the idea that the overall increase in SDLP over time was due to a decrease in cortical arousal. However, as mentioned above previous driving studies evaluating physiological measures of driver drowsiness differ from the present study by a number of methodological issues, such as driving task (simulators vs real driving), task duration (2–4 h of driving vs 1 h of driving), time of driving (evening and night vs morning), the sleepiness of the participants (sleep deprived vs well rested), the age of the participants (young vs older drivers). These factors all affect the likelihood of measuring significant changes with ToT in physiological markers of alertness and driving performance. As shown in an on-the road study by Verster, Taillard, Sagaspe, Olivier, and Philip (2011) driving at night and for more than 2 h had significant impairing effects on performance in a group of healthy young men. Our results showed, however, that on-the-road driving in normal traffic, for only 1 h in the morning, after a normal night of sleep resulted only in very small SDLP increase in our sample of older drivers (less than 1 cm), despite their complaints of insomnia. The test used in the current study was previously calibrated for the effects of alcohol. The increase in SDLP caused by 0.5 g/L alcohol (i.e. 2.4 cm) is considered clinically meaningful since accident risk has been demonstrated to increase significantly above this BAC in large epidemiological studies (Krüger, Kazenwadel, & Vollrath, 1995; Krüger & Vollrath, 2004). Although having insomnia is not strictly comparable to given alcohol doses, this calibration gives an indication about the clinical meaning of our findings. Then, the current investigation did not reveal a clinically meaningful increase of SDLP with TOT in older drivers. We may suppose that a longer driving task could have result in larger SDLP increase in both groups. Previous on-the-road driving investigations have revealed that SDLP is an extremely reliable index of individual driving performance with high test-retest reliability (O'Hanlon, 1995; O'Hanlon & Ramaekers, 1995; Owens & Ramaekers, 2009; Verster & Roth, 2011, 2012). Concerning the relationship between SDLP and crash risk per se, comparative analyses of data obtained from on-the-road driving tests and epidemiological data on crash risks was conducted by Owens and Ramaekers (2009). It revealed robust relationships between SDLP increment and indirect risk of having a traffic accident in various conditions. These data support the value of SDLP in its relationship to traffic accident risk.

As we did not find any differences between groups performance, older insomnia participants were able to drive as good as older normal sleepers, as measured by SDLP, in a real driving monotonous condition. The present finding differs from those of Perrier et al. (2014), in which driving performance of primary insomnia patients

and normal sleepers was measured by means of a driving simulator. Results of that study revealed that insomnia patients had a larger SDLP than the normal sleepers group and the decrease in driving performance occurred especially after 20 min of driving. The discrepancies may be due to the differences in task related factors and participants sample. Patients in the study by Perrier et al. (2014) were slightly younger (mean age approximately 49 vs 61 years old) and showed significantly worse sleep than normal sleepers the night before testing as measured by polysomnography (sleep efficiency 74% vs 81%) than in the study by Leufkens et al. (2014). A recent study showed that complaints of daytime impairment were more pronounced in young than in older insomnia patients (Kierlin, Olmstead, Yokomizo, Nicassio, & Irwin, 2012). Moreover impairment is more likely to be associated with more severe insomnia. In addition, Perrier and colleagues used a driving simulator, which has been found to induce more drowsiness and larger ToT effects in monotonous driving tasks than real driving (Akerstedt, Anund, Axelsson, & Kecklund, 2014; Hallvig et al., 2012; Philip et al., 2005). The increased sensitivity and sleep problems of the younger patients, and the more monotonous nature of the simulated driving task might explain the more pronounced vigilance decrement found in patients as compared to normal sleepers in the study by Perrier et al. (2014).

It is concluded that daytime on-the-road driving performance of older insomnia patients after a normal night of sleep, as measured by SDLP, does not differ from driving performance of older normal sleepers and that changes in spectral EEG measures of cortical arousal are not related to changes in daytime monotonous highway driving performance in this group of patients.

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