



## Waking EEG signs of non-restoring sleep in primary insomnia patients



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### HIGHLIGHTS

- Insomnia patients showed no beneficial effects of sleep on EEG parameters.
- Insomniacs are hyper-aroused during morning wakefulness.
- Non-restorative sleep is correlated with post-sleep Gamma.

### ABSTRACT

**Objective:** Subjective feelings of insufficient and non-restorative sleep are core symptoms of primary insomnia. Sleep has a restorative effect on next-day waking EEG activity, whereas sleep loss has non-restorative effects in good sleepers. We proposed to explore waking EEG activity in primary insomniacs the evening before, and the morning after, a night of sleep, in order to detect signs of morning hyper-arousal and non-restoring sleep that might explain the subjective feelings despite the absence of objective signs in polysomnography.

**Method:** Pre-sleep (10 pm) and post-sleep (10 am) waking EEG activity was analyzed in 10 non-medicated primary insomniacs and matched control subjects. Beta and Gamma absolute power and EEG temporal coupling were obtained. Participants also evaluated subjective sleep quantity and quality.

**Results:** Insomnia patients evaluated their sleep as non-restorative and insufficient. Compared to pre-sleep, during post-sleep control subjects exhibited significantly decreased Beta and Gamma power and reduced synchronization among anterior and posterior regions, consistent with restoring effects of sleep. Insomnia patients showed no beneficial effects of sleep on these EEG parameters.

**Conclusion:** Insomniacs are hyper-aroused during morning wakefulness and they do not benefit from preceding sleep.

**Significance:** Our study adds new knowledge to our understanding of the physiopathology of insomnia.

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## 1. Introduction

Primary insomnia (PI) is characterized by subjective feelings of insufficient and non-restorative sleep, complaints of daytime tiredness, unwellness, and impairments in cognitive functioning in emotional, social or professional fields (Riemann et al., 2010; Shekleton et al., 2010). According to the “hyper-arousal” model of PI, insomniac patients suffer higher 24-h physiological and cognitive arousal during both sleep and wakefulness (Bonnet and Arand, 2010).

With few exceptions (see, for example, Wu et al., 2013) the results of quantitative EEG studies generally support the idea that PI patients are hyper-aroused during pre-sleep waking at bedtime. These studies have shown that PI patients are more cortically-activated than good-sleepers or normal subjects during both sleep and the wake–sleep transition period. Awake, state-related, Gamma, Beta and Alpha EEG frequencies are higher in PI patients than good-sleepers or normal subjects during the wake–sleep transition period when they are trying to fall asleep (Cervena et al., 2014; Figueredo-Rodríguez et al., 2009; Maes et al., 2014; Merica and Gaillard, 1992; Lamarche and Ogilvie, 1997; Perlis et al., 1997, 2001a, 2001b; Staner et al., 2003).

The prefrontal cortex and posterior association areas are particularly aroused in PI patients when they are trying to sleep. In a previous study, we found higher Beta absolute power and current

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density in the frontal cortex and left posterior association areas in PI patients than normal controls. Also, functional relationships or the temporal coupling of fast frequencies – which are involved in binding functions and information processing (Linás et al., 1998; Tononi, 2010; Uhlhaas et al., 2009) – that link frontal, parietal and posterior midline regions of the left hemisphere are enhanced during the wake–sleep transition in these patients, suggesting that frontal deactivation and disengagement of the brain regions involved in executive control (Fuster, 2003), inner attention (Corbetta, 1998), and self-awareness (Kjaer et al., 2002; Mazoyer et al., 2001) are impaired in PI patients during the wake–sleep transition (Corsi-Cabrera et al., 2012).

According to the 24-h hyper-arousal model, such cortical activation should also be expected during the day. One study that analyzed waking EEG activity during the day in consecutive sleep sessions of a routine diagnostic multiple sleep latency test (MSLT) it was found that insomnia patients show higher relative power of fast Beta activity (18–30 Hz) at central regions than controls (Wolynczyk-Gmaj and Szelenberger, 2011) however, temporal coupling of waking EEG activity in PI patients during the day has not yet been investigated.

On the other hand, while some studies have found that PI patients show some polysomnographic (PSG) signs of disturbed sleep, such as longer sleep latency, more stage 1 sleep and less stage 3 and 4 sleep than good sleepers (Reite et al., 1995), these differences do not fully explain the subjective feelings of tiredness and unwellness reported by PI patients that are often worse than the objective PSG signs (Orff et al., 2007; Rosa and Bonnet, 2000).

A large body of evidence from quantitative EEG analyses has demonstrated that sleep loss has profound effects on brain functioning during subsequent wakefulness, suggesting that poor sleep has a deteriorating effect on next-day brain activity; whereas sleep is involved in the restoration of brain function (Huber et al., 2013). Sleep deprivation has been associated with a compensatory increase in Beta frequencies of the EEG spectrum in next-day waking (Cajochen et al., 1995; Corsi-Cabrera et al., 1992; Dumont et al., 1999; Forest and Godbout, 2000; Koenis et al., 2013; Lorenzo et al., 1995). Functional relationships, or temporal coupling among brain regions of fast frequencies, are also disorganized after sleep deprivation: EEG temporal coupling of Beta and Gamma activity within the same hemisphere is enhanced after sleep deprivation in humans compared to after a regular diurnal or nocturnal period of sleep (Corsi-Cabrera et al., 1992). In addition, brain network topology is disrupted (Koenis et al., 2013). After recovery sleep, waking spectral power and temporal coupling return to pre-deprivation values, suggesting a reorganizing effect of sleep on cortical activation and functional relationships among cortical networks (Cajochen et al., 1995; Corsi-Cabrera et al., 1992; Dumont et al., 1999; Ferri et al., 2008; Forest and Godbout, 2000; Lorenzo et al., 1995). Thus, as PI patients often complain of insufficient and poor sleep even in the absence of objective signs on the PSG, waking activity in the morning after a night of subjectively-rated unsatisfactory sleep should show EEG signs of non-restoring sleep.

Given (1) that Beta and Gamma activity during waking is associated with brain activation, attention and information processing; (2) that the temporal coupling of Gamma electrical activity between brain regions is involved in the processing and integration of disperse information in the brain; (3) that PI patients show enhanced Beta and Gamma activity and temporal coupling; and, (4) that sleep deprivation is followed by a rebound in Beta activity and enhanced Beta and Gamma temporal coupling in the same hemisphere in normal subjects; it might be expected that PI patients would show higher Beta and Gamma activity and temporal coupling than control participants after a night of poor sleep. Thus, the present study examined Beta and Gamma absolute

power and temporal coupling during resting waking at bedtime before going to sleep, and in the morning after a night of sleep, in order to elucidate whether PI patients show signs of morning hyper-arousal and non-restorative sleep associated with subjective ratings of sleep quality compared to normal subjects. According to the 24-h hyper-arousal model of primary insomnia (Bonnet and Arand, 2010), we hypothesized that PI patients would show signs of hyper-arousal; *i.e.*, higher Beta and Gamma power and temporal coupling between frontal and posterior regions in the evening as well as in the morning, in comparison to healthy controls, and that PI patients would present EEG signs of non-restoring sleep during next-day EEG waking activity, in the form of smaller changes in power and temporal coupling after a night of subjectively-poor sleep quality compared to healthy controls.

## 2. Method

Participants were the same subjects as in our own previous study (Corsi-Cabrera et al., 2012) except for two additional control subjects; that is, 10 young (19–32-year-old) patients (4 women) who met the International Classification of Sleep Disorders (American Academy of Sleep Medicine, 2005) criteria for primary insomnia and 10 controls (5 women), matched by age and education, who reported their sleep as restorative and satisfactory, and had regular sleep habits. All participants were right-handed and none had any medical, psychiatric or neurological conditions, nor had they been medicated for insomnia or suffered other sleep disorders. All participants were free of medication, including over-the-counter drugs. The drug-free condition was corroborated before performing polysomnography using the Multi-Drug 6 Panel Urine Test (MEDIMPEX United Inc, Bensalem, PA). Women were recorded between days 3 and 5 of their menstrual cycle (Solís-Ortiz et al., 1994). All participants underwent a general medical and psychiatric structured interview and completed a 15-day log to assess subjective sleep quality and sleep habits. Insomnia symptoms, or the absence of sleep complaints, were verified using the Pittsburgh Sleep Quality Index (Buysse et al., 1989), the Athens Insomnia Scale (Soldatos et al., 2000), and the Insomnia Severity Index (Bastien et al., 2001). The absence of clinical depression was ascertained with the Beck Depression Inventory (Beck et al., 1961) and Hamilton Depression Scale (Hamilton, 1967).

There were no significant differences between the two groups in terms of age, sex distribution or education (Table 1), or with respect to any PSG variable for the entire night, as reported elsewhere (Corsi-Cabrera et al., 2012).

All participants gave their informed, written consent and were offered treatment if they so desired. The protocol was approved by the Ethics Committee of the Faculty of Medicine at the Universidad Nacional Autónoma de México.

**Table 1**  
Demographic and subject characteristics by group.

	IN	CL	<i>t</i>	<i>p</i>
Age	25.8 (1.7)	26.6 (1.6)	0.3	0.75
Education (yr)	14.6 (0.3)	16.8 (0.3)	2.1	0.08
Illness duration (mo)	64 (1.4)	–	–	–
PSQ	13.4 (0.7)	2.5(0.7)	7.5	<b>0.0001</b>
AIS	13.3(1.19)	2.3(0.7)	7.9	<b>0.00001</b>
Sleep log				
Sleep: quality	5.4 (0.6)	7.8 (0.5)	2.9	<b>0.02</b>
Sleep: bad-good	5.5 (0.6)	7.8 (0.5)	3.2	<b>0.007</b>
Sleep: refreshing	5.2 (0.8)	7.6 (0.7)	2.4	<b>0.03</b>
Sleep: depth	5.0 (0.6)	7.4 (0.5)	2.9	<b>0.008</b>
Mood	6.9 (0.4)	8.1 (0.1)	0.98	0.35
Additional sleep desired (h)	3.32 (0.6)	0.94 (0.2)	3.6	<b>0.009</b>

IN, insomnia group; CL, control group; PSQ, Pittsburgh Sleep Quality Index; AIS, Athens Insomnia Scale. Numbers in bold indicate significant differences,  $p < 0.05$ .

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