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Brief report

Subjective sleep quality in relation to inhibition and heart rate variability in patients with panic disorder

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ABSTRACT

Background: Patients with panic disorder (PD) are known to report impaired sleep quality and symptoms of insomnia. PD is an anxiety disorder characterised by deficient physiological regulation as measured by heart rate variability (HRV), and reduced HRV, PD and insomnia have all been related to impaired inhibitory ability. The present study aimed to investigate the interrelationships between subjectively reported sleep impairment, cognitive inhibition and vagally mediated HRV in a sample characterised by variability on measures of all these constructs.

Methods: Thirty-six patients with PD with or without agoraphobia were included. Cognitive inhibition was assessed with the Color–Word Interference Test from the Delis–Kaplan Executive Function System (D-KEFS), HRV was measured using high frequency (HF) power (ms^2), and subjectively reported sleep quality was measured with the Pittsburgh Sleep Quality Index (PSQI).

Results: Cognitive inhibition was related to both Sleep latency and Sleep disturbances, whereas HRV was only related to Sleep disturbances. These relationships were significant also after controlling for depression.

Limitations: Correlational design.

Conclusion: Cognitive inhibition is related to key insomnia symptoms: sleep initiation and sleep maintenance. The data supports the psychobiological inhibition model of insomnia, and extends previous findings. Possible clinical implications of these findings are discussed.

1. Introduction

Insomnia has long been regarded as a disorder of hyperarousal (Perlis et al., 2011). According to the psychobiological model of insomnia (Borbély, 1982; Espie, 2002), insomnia is conceived as failure to inhibit wakefulness (e.g. arousal), rather than a conditioned state of hyperarousal. This model can accommodate findings on difficulties initiating and maintaining sleep (Bastien, 2011), and studies using Event Related Potentials suggest that decreased cognitive inhibition is related to increased sleep onset latency (Bastien et al., 2008). Cognitive inhibition, together with attentional shifting, have been found to be basic, underlying executive functions (Miyake et al., 2000), and attentional shifting is related to subjective sleep quality (Benitez and Gunstad, 2012). Still, studies on cognitive impairments in insomnia have yielded inconsistent findings. Shekleton et al. (2010) suggest that this is partly due to the heterogeneity of samples and possible clinical overlap between insomniacs and controls.

Impaired regulation of arousal has been considered a pathological mechanism in panic disorder (PD). Accordingly, PD-patients report reduced sleep quality as measured by the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) compared to healthy controls (Stein et al., 1993). These patients also show impaired inhibitory capacity (Dupont et al., 2000), and are characterised by decreased heart rate variability (HRV;

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Cohen et al., 2000; Middleton et al., 1994). Vagally mediated HRV is suggested to be an index of pre-frontal cortical (PFC) neuronal inhibitory activity (Thayer and Lane, 2000; Friedman, 2007), and this area, the PFC, is strongly implicated in executive functioning (Ridderinkhof et al., 2004). In line with this, vagally mediated HRV is positively related to executive functioning (Hansen et al., 2004; Mathewson et al., 2010), including cognitive inhibition (Hovland et al., 2012a). Furthermore, HRV is reduced compared to controls in both subjectively and objectively (polysomnography) defined insomniacs (Yang et al., 2011; Bonnet and Arand, 1998), whereas Spiegelhalder et al. (2011) only found insomniacs with reduced sleep duration to have decreased HRV.

In order to circumvent the methodological challenges associated with sample characteristics as highlighted by Shekleton et al. (2010), we investigated the relationships between subjective sleep quality and measures of cognitive and neuronal inhibition (i.e. HRV) in a sample of PD-patients, while controlling for level of depression. PD-patients frequently report insomnia and have altered physiological and cognitive inhibitory functioning. They thus represent an ideal sample for investigating the aforementioned relationships.

2. Methods

2.1. Participants

Thirty-six subjects provided written informed consent for participation in a randomised controlled treatment trial for PD. The study was approved by the Regional Committee for Medical and Health Research Ethics in Western Norway. Inclusion criterion was PD with or without agoraphobia, and exclusion criteria were psychotic disorders, substance-abuse, including the habitual use of benzodiazepines, severe major depressive episode, medical conditions that preclude participation in physical exercise, or organic brain disorder. Inclusions and exclusions based on the first three criteria were established using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First et al., 1995), whereas exclusions based on the last two criteria were determined by interviews or by consulting the participant's GP. Psychotropic medications were stabilized prior to assessments, and confirmed by self-report. Following inclusion, participants waited a mean of 68 days before further assessments were conducted. This wait was related to recruitment and randomisation not relevant to the present study. Participants were assessed on the clinical measures described below both before and after this wait, and did not change significantly on any of these during this period (please refer to Hovland et al., 2012b for details).

Table 1 describes the sample on demographic and clinical measures.

Table 1
Characteristics of participants ($N=36$).

	N	%
Female	29	80.6
Living alone	10	27.8
Medication		
SSRIs	13	36.1
Benzodiazepines[a]	5	13.9
Other psychotropic medication[b]	4	11.1
Agoraphobia	29	80.6
	Mean	*SD*
Age (years)	37.9	8.6
Years of education	13.6	2.5
Duration of PD (years)	10.1	9.5
Number of co-morbid axis 1 disorders	2.1	1.2
Clinical measures		
Panic frequency	1.8	1.4
Panic-related distress	6.2	1.7
BSQ	2.2	.5
ACQ	2.4	.7
BAI	22.2	10.5
STAI-T	50.8	10.8
PSQI	8.0	3.9
BDI-II	16.9	9.3

Note: BSQ=Body Sensations Questionnaire; ACQ=Agoraphobic Cognitions Questionnaire; STAI-T=State-Trait Anxiety Inventory-Trait version, BDI-II=Beck Depression Inventory II, PSQI=Pittsburgh Sleep Quality Index.

[a] Refers to intermittent use (e.g. in particularly stressful situations, such as long-distance travelling) of benzodiazepines (Sobril, Valium, Stesolid, Imovane; dosage 10–15 mg) within the last month. Participants were instructed not to use benzodiazepines on days of testing.

[b] Refers specifically to either of the following psychotropic medications: Tolvon, Efexor, Sarotex and Lamotrigin. Only registered if this medication was not used in addition to any SSRI.

2.2. Measures

HRV was obtained with a three-lead electrocardiogram (ECG; VU-AMS, Vrije Universiteit van Amsterdam; de Geus et al., 1995), using Ag/AgCl electrodes (1700 Cleartrace™, Conmed, Utica, NY) and a sampling rate of 1000 Hz. Recordings were conducted individually in a quiet location while participants were standing, yielding stable 4-min recordings. QRS-detection and the need for artefact correction were investigated while consulting with the corresponding ECG signal. Processing and analyses were conducted using the Kubios HRV (version 2.0; University of Eastern Finland). Trend components were removed using the smoothness priors method (Tarvainen et al., 2002). Vagally mediated HRV was based on absolute High frequency (HF)-power (ms^2) derived with an autoregressive model of order 16 and a frequency-band of .15–.40 Hz.

Cognitive inhibition was assessed with the Inhibition condition in the Color–Word Interference Test (henceforth referred to as the Stroop; Stroop, 1935) from the Delis–Kaplan Executive Function System (Delis et al., 2001) which yields age-adjusted scaled scores for completion time and performed errors. Higher scores indicate better performance.

The following inventories of PD, anxiety and depression were administered: The Body Sensations Questionnaire (Chambless et al., 1984), the Agoraphobia Cognitions Questionnaire (Chambless et al., 1984), the Beck Anxiety Inventory (Beck and Steer, 1993), the State-Trait Anxiety Inventory-Trait version (Spielberger et al., 1983), and the Beck Depression Inventory II (Beck et al., 1996). Chronbach's alpha's for these measures were .85, .72, .91, .93, .87, respectively.

Panic frequency, ranging from no panic attacks (0) to one or more panic attacks per day (4) and *panic-related distress and disability*, ranging from *not at all disturbing* (0) *to very disturbing* (8) were also assed (Clark et al., 1994).

Sleep quality was assessed with the PSQI; a 19-item self-report scale yielding a global score and seven components (listed in Fig. 1). Higher scores indicate poorer sleep. Cronbach's alpha was .77.

2.3. Statistical analysis

All analyses were conducted using SPSS version 17.0. All correlations were investigated using Pearson correlation coefficient (r). Depression was controlled for using partial correlations (r), before a modified Bonferroni procedure (Simes, 1986) was applied to control for the multiple comparisons of the relationships between HRV and cognitive inhibition and sleep components.