

# Polysomnographic and quantitative electroencephalographic correlates of subjective sleep complaints in chronic tinnitus

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**SUMMARY** Chronic tinnitus, or the perception of hearing sounds without the presence of external stimulation, is estimated at about 10–15% of the population, with highest prevalence after 50 years of age. Sleep complaints are among the most prominent complaints accompanying tinnitus, but objective data are rare. In this study, we examined prospectively the subjective and objective sleep parameters of this patient population in order to determine differences in sleep disturbances associated with chronic tinnitus compared to matched controls. Forty-four subjects (22 with tinnitus and 22 controls without tinnitus), unselected with respect to sleep complaints, participated in this study. The analysis involved 1-week sleep diaries, subjective sleep questionnaires and 1 night of polysomnographic (PSG) assessment. Compared to matched controls, the tinnitus group showed lower subjective sleep quality as measured with the Pittsburgh Sleep Quality Index (PSQI) and sleep diaries, but no significant difference in objective polysomnograph sleep parameters (i.e. sleep latency, efficiency). However, quantitative non-rapid eye movement sleep analysis revealed lower spectral power in the delta frequency band in the tinnitus group compared to controls, and this decrease was correlated with subjective sleep complaints (the lower the delta spectral power, the greater the complaints). This is the first report of an electrophysiological correlate of sleep difficulties supportive of subjective sleep complaints in the tinnitus population.

**KEYWORDS** delta spectral power, eeg sleep, polysomnographic assessment, slow wave sleep, tinnitus

## INTRODUCTION

Subjective chronic tinnitus is an auditory phantom sensation of ringing, buzzing or hissing sounds. It affects approximately 10–15% of the general population (Davis and Rafaie, 2000), with approximately 1–3% experiencing significant psychological distress that interferes with their lives (Erlandsson and Holgers, 2001). The underlying pathophysiology is as yet unknown, and diagnosis relies on subjective reports.

Sleep complaints are among the most prevalent comorbidities of tinnitus (Axelsson and Ringdahl, 1989; Folmer

and Griest, 2000; Hallam, 1996; Tyler and Baker, 1983). Previous studies have used questionnaires or general health surveys to assess retrospectively subjective and self-reported sleep difficulties, and have generally found a higher prevalence of sleep complaints in tinnitus sufferers than in the general population (Asplund, 2003; Hallam, 1996). Using a validated sleep questionnaire, a study conducted at our laboratory (Hebert and Carrier, 2007) found higher scores (more complaints) in subjects with tinnitus compared to age-matched controls.

These subjective complaints have been supported only partly by objective measures, and objective assessment of sleep characteristics was reported in only two studies. Crönlein and colleagues compared retrospectively the polysomnographic (PSG) sleep of patients referred to their sleep clinic for

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complaints of insomnia and suffering or not from tinnitus (Cronlein *et al.*, 2007). They found no significant differences in objective or subjective sleep variables between the two patient groups with insomnia complaints, with the exception of longer sleep latency in the group with tinnitus. Similarly, Burgos *et al.* (2005) found that objective and subjective sleep characteristics in 10 tinnitus patients with sleep complaints did not differ from those in insomnia patients. However, when the comparison group was healthy controls rather than insomnia patients, the tinnitus group displayed worse sleep on both subjective (Pittsburgh Sleep Quality Inventory) and objective (sleep efficiency, total sleep time and number of awake periods) measures.

While these two studies used PSG to assess the sleep characteristics of tinnitus sufferers, both assessed clinic-based tinnitus patients with concomitant sleep complaints. The interpretation of the results could therefore be confounded with the presence of sleep disorders unrelated to tinnitus, and may not reflect a specific characteristic of the presence of tinnitus.

The aim of the present study was to assess the sleep quality of tinnitus subjects by examining both subjective and objective sleep characteristics within the same group of participants and by avoiding the confound of selecting only subjects with sleep disorders. We therefore recruited prospectively tinnitus participants suffering from chronic tinnitus without referring specifically to sleep difficulties. We hypothesized that subjective sleep parameters, assessed by a validated sleep questionnaire (Pittsburgh Sleep Quality Index) and sleep diaries, as well as objective sleep parameters, measured by PSG assessment of sleep architecture (sleep latency, sleep duration, number of awakenings and increased duration of Stages 1 and 2) and quantitative sleep electroencephalogram (EEG) would be more impaired in the tinnitus group than in age-matched controls without tinnitus. Questionnaires assessing mood, depression, anxiety and tinnitus distress (tinnitus group) as well as hearing status were administered to establish the contribution to group differences.

## METHODS

### Participants

Twenty-two subjects (eight men and 14 women) with chronic tinnitus for at least 6 months [mean duration of tinnitus was 10.8 years, standard deviation (SD) 10.1] and 22 controls without tinnitus (eight men and 14 women) were recruited through newspaper advertisements, word of mouth and the Tinnitus Association of Quebec. The advertisement invited subjects with or without tinnitus to participate in a sleep study. The groups did not differ in age or educational level [mean age (SD) 62.5 (6.7) and 60.4 (6.9); mean education 14.4 (3.1) and 14.8 (3.5) for tinnitus and control groups, respectively, all  $P$ s > 0.05 by independent  $t$ -tests]. All participants were in good physical and mental health, as assessed by an in-house general

questionnaire covering concomitant medication, medical history and self-reported neurological and psychiatric conditions. Exclusion criteria were any uncontrolled medical condition that interfered with metabolic, sleep or mood function (e.g. diabetes, uncontrolled hyper- or hypotension, renal insufficiency, hyper- or hypothyroidism), a BMI index greater than 35 (severely obese) or transmeridian travel or night work in the 3 months prior to the study. Participants with known diagnosed insomnia or who were taking medication to initiate or maintain sleep were also excluded.

The two groups of 22 participants did not include 13 additional participants in whom significant sleep abnormalities were discovered after PSG assessment, i.e. periodic limb movement or apnea-hypoapnea index greater than 10 episodes per hour. The number of participants excluded on the basis of sleep apnea or periodic limb movements was higher in the tinnitus ( $n = 9$ ) than control group ( $n = 4$ ), but this difference was not significant ( $P = 0.34$  using a two-tailed Fisher's exact test).

## MATERIALS AND APPARATUS

### Hearing loss

Hearing loss was assessed by an audiogram following the standard Hughson–Westlake procedure in a soundproof booth using a clinical audiometer and insert phones (Interacoustic AC40, ANSI S3.6 norms, 1989). Pure-tone averages (PTA) were calculated for standard frequencies for each ear: PTA low (250, 500, 1 kHz, 2 kHz), PTA high (4 kHz and 8 kHz) and PTA total (250–8 kHz).

### Questionnaires

Sleep complaints were assessed with the French version of the Pittsburgh Sleep Quality Index (Buysse *et al.*, 1989). The PSQI assesses subjective sleep quality retrospectively over the past month. A single global index can be obtained from its 19 items, and three domains can be clustered to describe subjective sleep reports more accurately (Cole *et al.*, 2006): (1) sleep efficiency, (2) perceived sleep quality and (3) daily disturbances. Higher scores on the three-factor analysis and global index represent higher reported sleep disturbances.

Depressive symptomatology and anxiety were assessed respectively by validated questionnaires: the Beck Depression Inventory (BDI)-II (Beck *et al.*, 1996) and the State Trait Anxiety Questionnaire (ASTA) (Spielberger *et al.*, 1970).

Auditory sensitivity to external sounds, called hyperacusis (Khalifa *et al.*, 2002), and tinnitus-related distress (Meric *et al.*, 2000) were also assessed to compare group characteristics with those of previous studies.

### Sleep diaries

Prospective self-evaluation of subjective sleep was assessed based on a 7-day sleep diary. Thus, a French version of the Pittsburgh sleep diaries (Monk *et al.*, 1994) was used to assess

habitual sleep latency (how many minutes it took to fall asleep) and sleep duration (number of minutes spent asleep, excluding sleep latency and wake time during the night). The diary also included visual analog scales (VAS) ranging from 0 to 100 mm to assess sleep quality, mood at wake time, vigilance at wake time and perception of having had a good night's sleep (restorative sleep). Higher scores mean better quality sleep. Data were averaged across the 7 days.

### PSG sleep recording and quantitative non-rapid eye movement (NREM) sleep EEG

For PSG, EEG electrodes were placed according to the international 10–20 system using a referential montage with linked ears (10 k $\Omega$ ). Chin EMG and left and right EOG were also recorded. A nasal thermistor and EMG leg electrodes were used to screen for breathing and sleep movement disorders, respectively. A Grass Model 15 Neurodata system with 15A54 amplifiers (10 000 gain, 0.3–100 Hz bandpass, –6 dB) was used, with signals digitalized at a 256 Hz sampling rate using a commercial software (HARMONIE 5.1; Stellate Systems, Montreal, Canada). Sleep stages were scored visually on C3 (linked ears) by an experienced technician blinded for group (tinnitus versus control) on a computer screen (LUNA; Stellate Systems) according to standard criteria adapted to scoring 20-s epochs. Bedtime (lights off) and wake-up time (lights on) in the laboratory for each participant were scheduled according to their mean habitual bedtime and wake-up time derived from the 7-day sleep diary. For all PSG sleep recordings, subjects were required to stay in bed for their entire habitual sleep duration (from scheduled lights off to scheduled lights on). Sleep stage variables were computed from sleep onset to lights on. Sleep efficiency was defined as (number of minutes spent asleep  $\div$  total number of minutes from sleep onset to lights on)  $\times$  100. Sleep onset was defined as three consecutive Stage 1 epochs or one epoch of another sleep stage. The number of awakenings was defined as the number of times participants woke up and remained awake for longer than 20 s over the entire night.

For EEG analysis, spectral analyses of NREM sleep (Stages 2, 3 and 4) were computed using a commercial software package (Stellate Systems) on C3 and C4 derivations using fast-Fourier Transformation (FFT) (cosine tapering) on 4-s artifact-free sections yielding a spectral resolution of 0.25 Hz. EMG artifacts were detected automatically and rejected for spectral analysis (Brunner *et al.*, 1996). Further artifacts were eliminated by visual detection. Epochs containing artifacts were considered as missing data to preserve sleep continuity. The detection algorithm compares high-frequency spectral EEG power (26.25–32.0 Hz) in each 4-s spectral epoch with the spectral power of a moving 3-min window. A 4-s value is considered artifactual when it exceeds the background spectral power by a factor of 4. Five 4-s spectral epochs were averaged to maintain correspondence with the 20-s sleep scoring windows. The number of minutes of rejected EEG contaminated by artifacts did not differ between the two groups (means

28.4 and 34.2 for tinnitus and controls, respectively,  $P = 0.29$  by an independent  $t$ -test). Spectral power was averaged for all-night NREM sleep. Analyses were performed per 1-Hz frequency bins ranging from 1.0 to 32.0 Hz. Analyses were also performed on 0.5–1.0 Hz bins. Spectral power in delta (0.5–4 Hz), theta (4–7 Hz), alpha (8–12 Hz), sigma (12–16 Hz) and beta (16–32 Hz) bands were then calculated by summing the absolute power values of respective frequency bins. Preliminary analysis yielded no interhemispheric differences (all  $P$ s  $> 0.05$  by independent  $t$ -tests), and spectral power in each frequency band was averaged for C3 and C4.

### Procedure

#### *Visit 1 (Institut universitaire de gériatrie de Montréal)*

During the first 2-h visit, participants gave their informed consent, completed the study questionnaires (general health questionnaire, BECK-II and hyperacusis), provided blood and urine samples and underwent a hearing assessment. They were given a 7-day sleep diary to complete at home prior to the sleep laboratory assessment, and they were asked to follow their regular bedtime and wake time ( $\pm$  30 min) for the 7 days according to their habitual sleep–wake cycle.

#### *Visit 2 (Hôpital du Sacré-Coeur de Montréal)*

During the second visit to the sleep laboratory clinic, participants completed additional questionnaires (PSQI, ASTA-Trait, VAS scales). PSG was performed in private bedrooms at each subject's habitual sleep and wake time, ascertained by averaging sleep and wake times from their sleep diaries.

This study was approved by the ethics committees of the Institut universitaire de gériatrie de Montréal and Hôpital du Sacré-Coeur de Montréal.

### Statistical analysis

Missing data were not replaced. Analyses of variance (ANOVAS) with one independent factor (group: tinnitus and control) and one within-subject factor (ear: left and right) were performed to compare pure tone averages (PTA: high, low, total). Independent  $t$ -tests were performed to assess group differences (tinnitus and control) on questionnaires (ASTA, Beck-II, hyperacusis), prospective subjective sleep measure (sleep diaries), retrospective subjective sleep measure (PSQI) and objective sleep assessment [PSG and quantitative EEG (qEEG)]; qEEG data were unavailable for four subjects (two tinnitus, two controls) because EEG quality was too low to be included in quantitative sleep analyses (e.g. EKG contamination, sweat artifacts). Analyses were performed on log-transformed qEEG data, but analyses performed on the raw data yielded an identical pattern of results. A Pearson product–moment correlation analysis was performed between delta frequency band and variables on which the two groups differed. All statistical tests were two-tailed.

**Table 1.** Hearing status (a) and psychological (b) variables for the tinnitus and control groups

	Tinnitus (n = 22) Mean (SD)	Controls (n = 22) Mean (SD)	P-value
(a) Mean (SD) and P-values for PTA (high, low, total) averaged across ears			
PTA high	40.5 (19.4)	28.3 (14.3)	0.024
PTA low	21.1 (10.3)	13.7 (8.1)	0.013
PTA total	27.4 (11.9)	18.5 (8.8)	0.008
(b) Mean (SD) and P-values for ASTA-Trait, BDI-II and hyperacusis			
Asta-Trait	37.1 (10.2)	30.1 (9.6)	0.027
BDI-II	6.1 (6.6)	3.0 (3.8)	n.s.
Hyperacusis	17.2 (7.4)	12.6 (8.1)	0.055

ASTA: State Trait Anxiety Questionnaire; BDI: Beck Depression Inventory; PTA: Pure-tone averages; SD: standard deviation.

## RESULTS

### Hearing loss, anxiety, depression, hyperacusis and tinnitus-related distress

There was no main effect of ear side or any interaction with this factor on thresholds (all  $P_s > 0.05$ ). The tinnitus group showed higher hearing thresholds than the control group: PTA high ( $F_{(1, 41)} 5.5$ ,  $P = 0.02$ ), PTA low ( $F_{(1,41)} 6.8$ ,  $P = 0.01$ ) and PTA total ( $F_{(1, 41)} 7.7$ ,  $P = 0.008$ ) (Table 1a).

Mean and standard deviations for anxiety and depressive symptoms and hyperacusis measures for both groups are shown in Table 1B. Compared to the control group, tinnitus subjects were more anxious (anxiety trait: ( $t_{(40)} -2.29$ ,  $P < 0.05$ ). The two groups did not differ significantly on depressive or hyperacusis symptoms, although the tinnitus group scored higher on both questionnaires ( $t_{(41)} -1.84$ ,  $P = 0.07$  and  $t_{(41)} 1.98$ ,  $P = 0.06$ , respectively). Tinnitus subjects scored 15.6 on average (range=0–52) on the tinnitus-related distress questionnaire and had had their tinnitus for 10.8 years on average (range=1.5–42 years).

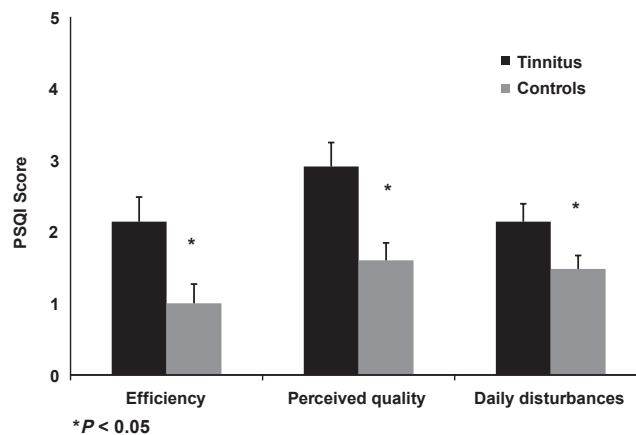
### Subjective and objective sleep parameters

#### PSQI

The tinnitus group showed a higher PSQI index than the control group, ( $t_{(39)} -3.35$ ,  $P < 0.01$ ), with means of 7.2 (SD=3.5) and 4.0 (SD=2.5), respectively. Analyses of PSQI clusters revealed that they also showed poorer self-assessment on sleep efficiency ( $t_{(41)} -2.58$ ,  $P < 0.01$ ), perceived sleep quality ( $t_{(40)} -3.06$ ,  $P < 0.01$ ) and daily disturbances ( $t_{(41)} -2.11$ ,  $P < 0.05$ ) (Fig. 1).

#### Sleep diaries

The tinnitus group reported shorter sleep duration ( $t_{(41)} 2.683$ ,  $P = 0.010$ ), poorer sleep quality ( $t_{(41)} 2.490$ ,  $P = 0.017$ ) and



**Fig. 1.** Mean scores (SE) for the tinnitus and control groups on the three factors of the Pittsburgh Sleep Quality Index (PSQI) questionnaire.

**Table 2.** Subjective prospective sleep assessment (diaries)

Item	Tinnitus (n = 22) Mean (SD)	Controls (n = 22) Mean (SD)	P-value
Sleep latency (min)	31.9 (31.5)	19.5 (15.4)	NS
Sleep duration (min)	382.3 (55.7)	431.7 (64.8)	0.010
Sleep quality	63.6 (13.8)	73.9 (13.1)	0.017
Mood at wake time	70.0 (15.5)	76.5 (15.5)	NS
Vigilance at wake time	63.0 (16.3)	73.8 (13.6)	0.024
Perception of a good night's sleep (restorative sleep)	60.1 (16.2)	70.3 (16.6)	0.050

poorer vigilance at wake time ( $t_{(41)} 2.346$ ,  $P = 0.024$ ) than the control group (Table 2). Of marginal significance is that they also reported having less restorative sleep (perception of a good night's sleep) than their control counterparts ( $t_{(41)} 2.021$ ,  $P = 0.050$ ).

#### Objective sleep (PSG)

The tinnitus group did not differ significantly from the control group on PSG sleep variables (Table 3).

#### Quantitative EEG and correlations

Spectral power in each of the five frequency bands (delta, theta, alpha, sigma and beta) was compared between groups. Spectral power in the delta frequency band was significantly lower in the tinnitus [ $t_{(38)} 2.20$ ,  $P = 0.034$ ] than the control group. The mean delta power value of 5.12 for the tinnitus group was outside the standard 95% confidence interval of the controls' mean (5.20–5.49), as well as outside the very conservative 99% confidence interval of the controls' mean (5.15–5.54). None of the other frequency band differences was significant (Table 4).

**Table 3.** Objective polysomnographic assessment [standard deviation (SD)] for the two groups

Item	Tinnitus (n = 22)	Controls (n = 22)	P-value
	Mean (SD)	Mean (SD)	
Sleep latency (min)	23.3 (35.5)	13.4 (9.8)	0.21
Sleep duration (min)	365.8 (89.3)	376.6 (59.6)	0.64
Wake time (min)	80.1 (41.6)	65.6 (41.5)	0.25
Sleep efficiency (%)	80.9 (12.2)	85.2 (8.9)	0.19
Stage 1 (% of TST)	9.5 (5.9)	8.6 (4.5)	0.57
Stage 2 (% of TST)	68.0 (6.6)	66.4 (6.6)	0.41
Stages 3 and 4 (% of TST)	3.5 (3.5)	4.8 (3.7)	0.21
REM (% of TST)	19.1 (5.5)	20.2 (5.8)	0.51
Number of awakenings	44.4 (18.1)	40.1 (18.2)	0.47

REM: rapid eye movement; TST: total sleep time.

**Table 4.** Mean spectral power [standard deviation (SD)] of the different frequency bands for the tinnitus and control groups

Frequency band	Tinnitus (n = 20)	Controls (n = 20)	P-value*
	Mean (SD)	Mean (SD)	
Delta (0.5–4 Hz)	176.9 (56.4)	219.2 (60.0)	0.034
Theta (4–7 Hz)	47.5 (20.0)	53.1 (15.5)	0.204
Alpha (8–12 Hz)	23.7 (13.1)	25.0 (14.7)	0.923
Sigma (12–16 Hz)	12.1 (5.6)	10.3 (3.7)	0.411
Beta (16–32 Hz)	4.6 (2.1)	3.9 (1.2)	0.583

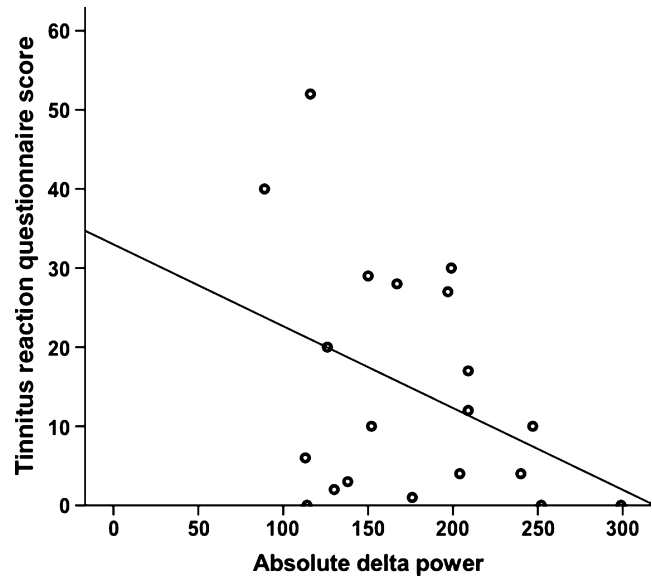
\*Although raw data are presented, the P-values correspond to analyses on log-transformed data.

Correlations were run between delta spectral power and the variables that differed between the groups, namely PSQI scores, anxiety (trait), hearing loss and the diary variables sleep duration and quality, vigilance at wake time and perception of a good night's sleep. The only significant correlation was a negative correlation between spectral power and PSQI scores:  $r_{(38)} -0.371$ ,  $P = 0.022$ . None of the other correlations was significant, with all  $P$ s between 0.12 (vigilance at wake time) and 0.67 (hearing loss). In the tinnitus group only, the correlation between tinnitus-related distress and delta power was  $r_{(19)} -0.39$ ,  $P = 0.086$  (see Fig. 2).

## DISCUSSION

The main finding of this study is that, compared to an age-matched control group, subjects with tinnitus have more sleep complaints and lower spectral power in the delta band during NREM sleep. The delta band is a frequency band associated with brain synchronization, restorative sleep and sleep depth.

A key aspect of our study is that although our tinnitus sample was similar to those described in other studies in terms of hearing status, tinnitus-related distress and psychological variables (e.g. Hebert and Carrier, 2007), subjects taking sleep medication or with diagnosed sleep disorders or other diseases that could interfere with sleep assessments were not included.

**Fig. 2.** Correlation between delta power and scores on the tinnitus reaction questionnaire in the tinnitus group.

Therefore, our findings on subjective sleep and quantitative sleep EEG can be generalized to a population of typical tinnitus subjects in this age group, and are probably a core characteristic of experiencing a phantom sound *per se*.

The patterns of subjective and objective sleep components in our tinnitus sample resembles those described in populations diagnosed with insomnia, in the sense that polysomnographic assessments only rarely corroborate subjective sleep complaints in this population (Carskadon *et al.*, 1976; Edinger and Fins, 1995; Means *et al.*, 2003). However, several studies that used the more sensitive measure of spectral power have reported decreased delta power in insomniacs compared to good sleepers (Besset *et al.*, 1998; Krystal *et al.*, 2002; Marzano *et al.*, 2008; Merica *et al.*, 1998), although some studies did not find such differences (Bastien *et al.*, 2003; Perlis *et al.*, 2001). Differences between studies have been proposed to reflect subtypes of insomnia (Marzano *et al.*, 2008). For example, the term paradoxical insomnia has been coined to designate normal PSG measures and subjective sleep complaints. In the only study where qEEG were measured in these patients (Krystal *et al.*, 2002), a negative correlation between delta power and underestimation of total sleep time was found. Classical polysomnographic (PSG) assessment might therefore not be sensitive enough to capture sleep abnormalities in some clinical populations, hence the lack of group differences on these measures. Importantly, our study is the first to evaluate quantitative sleep EEG in tinnitus patients. Future studies should determine whether tinnitus participants complaining about insomnia would show even more prominent modifications of quantitative sleep EEG.

One limitation of our study is that there was only one night of recording in the sleep laboratory. This is unlikely to have a

major impact on our results, as both groups were exposed to the same conditions, and our control sample shows similar delta values to those reported in the literature for this age group (e.g. Bastien *et al.*, 2003; Cajochen *et al.*, 2006). The negative correlations between sleep complaints and delta power and between tinnitus-related distress and delta power further support genuine sleep abnormalities rather than artifactual effects.

The physiological mechanisms and functional significance of lower delta power during NREM sleep in the tinnitus group remain unclear. The intensity and dynamics of delta power are thought to reflect the time-course of the homeostatic process (i.e. more time awake produces higher delta, whereas more time asleep is associated with lower delta) (Achermann *et al.*, 1993). Lower delta activity may therefore reflect impaired sleep homeostasis, as has been hypothesized in insomnia (Besset *et al.*, 1998). This implies that tinnitus subjects would be less able to increase sleep intensity with the accumulation of wakefulness. Future studies should address this issue by measuring the effects of multiple waking intervals on delta sleep to quantify the ability of the homeostatic process of tinnitus subjects to adapt to different wake durations.

Experimental reduction of delta activity has been linked to generalized discomfort, fatigue, attention and memory disruptions and daytime pain (e.g. Lentz *et al.*, 1999). Conversely, experimental increase of slow wave sleep has been shown to increase memory consolidation (Marshall *et al.*, 2006). Impaired attention and memory have been reported in tinnitus (e.g. Delb *et al.*, 2008; Rossiter *et al.*, 2006; Stevens *et al.*, 2007) and may be consequent to non-restorative sleep in combination with tinnitus, rather than a primary effect of tinnitus.

Finally, given our findings, future studies should examine whether and how activities and therapies that enhance NREM sleep synchronization (delta) can impact sleep quality, sleep complaints and the overall wellbeing of tinnitus patients. For example, meditation and light exercise have been shown to enhance delta sleep (Gambelunghé *et al.*, 2001; Patra and Telles, 2009). Interestingly, a 3-month intervention involving combined meditation/cognitive behavioral therapy improved wellbeing in tinnitus patients to a degree very similar to that in patients with chronic fatigue syndrome (Sadlier *et al.*, 2008; Thomas *et al.*, 2006). The effects of these therapies on delta power could reveal the mechanisms by which these therapies improve the well-being of this population, and could inform us about how NREM sleep synchronization contributes to tinnitus comorbidities.

In conclusion, our study shows that tinnitus patients without diagnosed sleep disorders have self-reported poor sleep quality, and that these complaints are not correlated with objective polysomnographic assessments of their sleep, but rather with a significant decrease in delta band spectral power.

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