Available online at www.sciencedirect.com
ScienceDirect
Int. J. Hyg. Environ. Health 212 (2009) 37–44
International Journal of Hygiene and Environmental Health
www.elsevier.de/ijheh

Salivary cortisol levels, subjective stress, and tinnitus intensity in tinnitus sufferers during noise exposure in the laboratory

Sylvie Hébert[a,b,*], Sonia J. Lupien[c]

[a]*École d'Orthophonie et d'Audiologie, Université de Montréal, Montréal, Canada H3C 3J7*
[b]*Centre de Recherche, Institut Universitaire de Gériatrie de Montréal, Montréal, Canada H3C 3J7*
[c]*Stress Laboratory, Department of Psychiatry, Douglas Hospital Research Centre, McGill University, Montréal, Canada H3C 3J7*

Received 21 March 2007; received in revised form 22 November 2007; accepted 27 November 2007

Abstract

Tinnitus, a chronic internal noise, is thought to increase in intensity during or following external noise exposure. Yet there is no empirical evidence for this complaint in the extant literature. Recently, cortisol has been advanced as a useful tool for studying the physiological effects of noise on stress, but few, if any, studies have examined the short-term effects of noise on cortisol levels in tinnitus sufferers. This study assesses the effects of noise exposure on cortisol levels and subjective stress in tinnitus participants and controls without tinnitus. Twenty tinnitus participants and 20 controls without tinnitus were exposed to a 20-min broadband noise with amplified low frequencies. Saliva samplings for cortisol analysis and subjective stress and tinnitus intensity ratings (for tinnitus participants) were performed at regular intervals throughout testing. Results show higher cortisol levels for both groups immediately before, immediately after, and 10 min after the end of noise than at other time points. The tinnitus group had lower overall cortisol levels than controls. In contrast, subjective stress ratings were higher for the tinnitus group, and higher at midpoint and immediately after the noise ended. Tinnitus subjective intensity increased throughout testing, especially for the group with high tinnitus-related distress. Overall results show that noise exposure influences cortisol response, subjective stress, and tinnitus intensity.

Keywords: Noise; Stress; Cortisol; Tinnitus; Distress; HPA axis

Introduction

Subjective chronic tinnitus is the perception of sound in the ears or head, usually ringing or buzzing, with no external sound source. Tinnitus prevalence is conservatively estimated at about 6.75% in the over-50 population. A small proportion of the overall population is moderately or severely affected, and prevalence of the disabling type increases with age (Davis, 1995). Tinnitus prevalence is expected to increase across all age groups in the near future. Indeed, the most important predictor

Abbreviations: HPA, hypothalamic–pituitary–adrenal; CRF, corticotropin-releasing factor; ACTH, adrenocorticotropin hormone; S.D., standard deviation; S.E., standard error of the mean; PTA, standard pure tone average for 500-, 1000-, and 2000-Hz frequencies; HF PTA, pure tone average for 4000- and 8000-Hz frequencies; TRQ, Tinnitus Eeaction Questionnaire; µg/dl, micrograms per decilitre; BDI-II, Beck Depression Inventory.

*Corresponding author at: École d'Orthophonie et d'Audiologie, Faculté de Médecine, Université de Montréal, BRAMS, Pavillon 1420 Mont-Royal, C.P. 6128, succ. Centre-ville, Montréal, Canada H3C 3J7. Tel.: +1 514 343 6111x2594; fax: +1 514 343 2175.
E-mail address: sylvie.hebert@umontreal.ca (S. Hébert).

1438-4639/$ - see front matter © 2007 Elsevier GmbH. All rights reserved.
doi:10.1016/j.ijheh.2007.11.005

of tinnitus presence is hearing loss, especially sensorineural loss associated with age and noise exposure. In the elderly, sensorineural hearing loss is the third most prevalent chronic condition after arthritis and hypertension (Weinstein, 2000). Moreover, prevalence of sensorineural occupational noise hearing loss is increasingly found in young populations (Vio and Holme, 2005), and the World Health Organization is currently compiling burden of disease statistics for tinnitus caused by environmental noise.

Tinnitus primarily produces psychological distress. Earlier studies have reported mood disorders and high levels of anxiety in tinnitus patients (e.g., Budd and Pugh, 1996). Sufferers report that tinnitus severity is exacerbated by stress and fatigue. Although this causal relationship has been incorporated in the classical knowledge on tinnitus (e.g., Tyler, 2000; Henry and Wilson, 2001), it derives from anecdotal and clinical observations (e.g., Alpini and Cesarini, 2006). Unfortunately, empirical data are lacking. Nevertheless, there is indirect evidence of an association between stress or distress and the clinical course of tinnitus. One retrospective study that measured several stress-related parameters using questionnaires reported that patients with sudden hearing loss and tinnitus had a greater number of stressful events and daily hassles in their lives, along with fewer coping abilities, compared to another clinical group, suggesting that tinnitus is a consequence of stress (Schmitt et al., 2000). Another study found beneficial effects of a relaxation therapy programme on the stress-related immunological parameter tumour necrosis factor of tinnitus patients, especially those with high distress (Weber et al., 2002). Predictive factors for tinnitus *severity* (not presence) are anxiety disorders (Holgers et al., 2000, 2005) and poor well-being at onset (Holgers et al., 2005; Schmitt et al., 2000). Hearing loss plays only a moderate role. Psychosocial stress and environmental problems are also more common in patients with severe tinnitus than in patients with less severe tinnitus, where severity is defined as absence from work for more than 1 month and more than three visits to the therapist or audiologist physician (Holgers et al., 2005). In addition, cognitive behaviour therapy is deemed the most promising therapy for tinnitus individuals, possibly because it reduces stress and distress through the development of coping strategies (Andersson et al., 2002).

In a recent study (Hébert and Lupien, 2007), we found that subjective tinnitus intensity did *not* increase in participants undergoing psychosocial stress (e.g., public speaking), although subjective stress levels *did*. This suggests the absence of a time-locked relationship between stress increase and tinnitus intensity, at least in the time window we considered (i.e., about 1 h). It is also possible that the subjective measure, a Likert-type scale, was insufficiently sensitive to capture tinnitus variations. Finally, psychosocial stress may not be the right stimulus to increase tinnitus intensity. In any case, the precise relationship between tinnitus intensity and stress remains largely unclear, as well as the conditions that trigger tinnitus' increase. Note that we observed a blunted and delayed cortisol response to psychosocial stress in tinnitus participants compared to controls in that study (Hébert and Lupien, 2007).

Loud noise (i.e., unwanted sound) is reported to increase tinnitus intensity. The effects of external noise (or sound) on tinnitus fluctuations are mixed. Presented at low levels, external sound is known to mask tinnitus, especially when the sound's spectral properties closely match those of tinnitus (Vernon and Meikle, 2003). Tinnitus may then disappear for durations of several seconds to several minutes, after which it reappears. This is known as "residual inhibition." When sounds are presented at moderate and high levels, however, the effects on tinnitus intensity are unknown. Studies have shown that tinnitus patients have abnormal brain responses to moderately loud external sounds (Lockwood et al., 1998; Walpurger et al., 2003), although the impact on tinnitus intensity was not reported. In a previous study (Hébert et al., 2004), we measured sensitivity to external noise psychometrically (using a questionnaire) in tinnitus participants and controls without tinnitus. Tinnitus participants were divided into two groups according to tinnitus-related distress (low vs. high). We found high levels of self-reported noise sensitivity in both groups compared to controls, and particularly in the high-distress group, where it approached strong auditory sensitivity (two standard deviations above the mean for the general population). This group also exhibited behaviours that interfered with day-to-day functioning, for instance, avoiding social situations involving noise exposure. Here again, no direct evidence linked tinnitus intensity to loud noise exposure.

Another, independent line of research has shown deleterious effects of noise exposure on the body, particularly the endocrine stress system. One hormone that has received particular attention is salivary cortisol, considered an excellent stress marker (Kirschbaum and Hellhammer, 1994). Cortisol has recently been suggested as a useful tool to measure noise-related stress (Bigert et al., 2005). Cortisol is the principal hormonal product of the human hypothalamic–pituitary–adrenal (HPA) axis, a close-looped endocrine system. It is secreted naturally following a circadian rhythm (basal cortisol) and is also secreted periodically following perceived stress (reactive cortisol). Thus, following the rapid release of corticotropin-releasing factor (CRF) by the hypothalamus, the pituitary releases the adrenocorticotropin hormone (ACTH), which in turn triggers the adrenal gland to release glucocorticoids (human cortisol).

Several animal and human studies, in both natural and laboratory settings, have shown that noise acts as a stressor and enhances cortisol levels. For instance, the above-mentioned workers who were exposed to high ambient noise levels had lower cortisol secretion, fatigue, and irritability when wearing earmuffs to reduce ambient noise levels (Melamed and Bruhis, 1996).

Noise sensitivity may modulate cortisol release. Waye et al. (2002) recently demonstrated that a 2-h exposure to low levels of low-frequency noise (similar to a common air-conditioner) during mental work increased cortisol levels compared to a flat frequency spectrum noise. However, only subjects who were generally noise sensitive, as determined by their score on a noise-sensitivity questionnaire, were affected.

To our knowledge, no study has examined the effects of short-term noise on human cortisol and subjective stress (for a review, see Babisch, 2003) or subjective tinnitus intensity. Yet noise is an increasingly annoying presence in our surroundings, due to social/leisure (audio devices, rock concerts, fireworks, toys, etc.) and environmental (urban life, traffic, etc.) phenomena. Since noise and stress are ever more pervasive and tinnitus is ever more prevalent, the relationships between them need to be investigated.

This study aimed to investigate whether short exposure to loud noise increases cortisol levels, subjective stress, and tinnitus intensity ratings in tinnitus participants and controls without tinnitus. Carefully selected participants were exposed to an 80 dBA continuous noise lasting 20 min with a 2-min midpoint pause. Salivary cortisol sampling, subjective stress ratings, and tinnitus intensity ratings (in tinnitus participants) were performed at regular intervals throughout testing and until 60 min afterwards. In view of our previous results (Hébert and Lupien, 2007), we hypothesized that the tinnitus group would display a flat cortisol response to stress compared to controls, but would rate tinnitus intensity increasing higher throughout the noise exposure and show subjective stress similar to controls. Controls were expected to display maximum cortisol levels at about 30 min after noise initiation (at time +30), with gradual decrease in the silence that followed.

Method

Participants

Twenty adults with tinnitus and 20 adults without tinnitus, all having similar socioeconomic backgrounds, participated in the study. Most participants ($n = 35$) took part in a second protocol reported elsewhere (Hébert and Lupien, 2007). The two protocols were separated by several days and the order was counterbalanced across groups.

The tinnitus group contained 10 women and 10 men and the control group contained nine women and 11 men. All women were post-menopausal. Socio-demographic variables, hearing status, and tinnitus characteristics (tinnitus participants) are presented in Table 1. Both groups had similar education ($t(36) = -1.46$, $p = .16$) and age ($t < 1$) profiles.

Inclusion criteria for all participants were strict. All participants were in good health, and none had a medical condition or took medication that could interfere with the functioning of the HPA axis responsible for cortisol secretion. None had a recent history of psychiatric or neurological disease or dependence on alcohol or other drugs. All participants were non-smokers. Although the presence of depressive symptomatology (as assessed by the BDI-II questionnaire) was

Table 1. Socio-demographic, hearing, and tinnitus characteristics of groups

	Tinnitus ($n = 20$)	Controls ($n = 20$)	p
Age			
Mean (S.D.)	67.9 (6.1)	68.8 (5.5)	n.s.
Range	56–78	61–77	
Education			
Mean (S.D.)	12.9 (4.7)	14.9 (3.3)	n.s.
Range	7–23	10–19	
Hearing threshold			
Standard PTAs (S.D.)	34.4 (15.8)	18.5 (6.9)	$<.001$
PTAs HF (S.D.)	54.2 (16.3)	42.8 (16.1)	$<.04$
Tinnitus duration in years			
Mean (S.D.)	14.5 (9.7)	–	
Range	1.5–35		
Tinnitus-related distress (S.D.)	19.9 (10.3)	–	

not an exclusion criterion *per se*, participants who took anti-depressant drugs were excluded due to possible interference with HPA activity. None of the 19 post-menopausal women were taking hormone replacement therapy at the time of testing.

Auditory testing

Hearing loss was assessed by audiogram, using the standard Hughson–Westlake procedure in a soundproof booth with a clinical audiometer and insert phones (Interacoustics AC40, ANSI S3.6 norms, 1989). Pure tone averages (PTA) were calculated for standard frequencies (500–1000–2000 Hz, hereafter PTAs) and higher frequencies (4000 and 8000 Hz, hereafter HF PTAs), since tinnitus is usually associated with hearing loss in this frequency range. No significant differences were found between men and women on PTAs or HF PTAs (both $ts < 1$) or between left and right ear on HF PTAs ($p = .30$), although there was a difference in favour of the left ear on the PTAs, $t(39) = 2.13$, $p < .04$. PTAs and HF PTAs were averaged across ears for further analysis. As expected, the tinnitus groups had slightly higher detection thresholds than the control group for both PTAs ($t(38) = 4.12$, $p < .001$) and HF PTAs ($t(38) = 2.15$, $p < .04$).

Behavioural data were obtained on each participant using questionnaires with good psychometric properties. Tinnitus-related distress was assessed by a French version of the Tinnitus Reaction Questionnaire (TRQ) (validated French version: Meric et al., 2000; original English version: Wilson et al., 1991). Sensitivity to external sounds was assessed with the Hyperacusis questionnaire (Khalfa et al., 2002). Global scores on this questionnaire are divided into three subscales corresponding to various aspects of intolerance to external sounds in everyday life: attentional, social, and emotional. A score of 28 (mean for a general population sample + 2 S.D.) was determined as strong auditory hypersensitivity. Presence of depressive symptomatology (BDI-II, Beck, 1997) was also assessed.

Subjective rating scales

At each sampling time, participants were asked to rate their subjective stress and subjective tinnitus intensity (for tinnitus participants) on a 10-point Likert-type scale. The stress scale ranged from 0 (no stress) to 10 (very stressed), and tinnitus scale ranged from 0 (no tinnitus) to 10 (tinnitus very intense).

Saliva samples analyses

Salivary samples were taken with Salivettes (Starsteadt) at regular intervals (see procedure section) and stored at −80 °C. All 216 salivary samples were recoded for blind analysis before being sent to the Douglas Hospital Centre in Montreal. Cortisol levels were determined by specific radioimmunoassay using a commercial kit (DSL-2000, Sanofi Diagnostics, Montreal, Canada). The inter-assay coefficient of variation from previous studies at the Douglas Hospital Research Laboratories was 7.5% (on a range of 0.8–1.2 μg/dl dose).

Procedure

Participants were exposed to a low-frequency flat spectrum hissing noise (125–2000 Hz) with additional sound pressure in the 31.5–125-Hz frequencies (similar to the method used by Waye et al., 2002) resembling a fan. The noise was constructed using Cool Edit and delivered by a PC-controlled Technics SA-EX140 amplifier. It was presented to all participants through TDH-39P (Telephonics Co., Farmingdale, NY, USA) earphones at 80 dBA in each ear, as measured by a Larson Davis sound level meter, an AEC101 artificial ear and a 2559 microphone. Participants were exposed to the noise for 20 min with a 2-min midpoint pause for cortisol sampling. Noise level was chosen within safety standards and to minimize potential loudness differences between groups due to loudness recruitment (i.e., high-level loudness is perceived as similar despite hearing threshold differences), thus preventing lengthy level adjustments for each participant.

Participants were seated before a computer screen and asked to perform a standard Stroop task (read the name of a colour or identify ink colour) followed by a target detection task (press the space bar when a red square appears on the screen among foils at random time intervals). These tasks are known to have no effect on cortisol secretion (Biondi and Picardi, 1999), meaning that any observed cortisol increase would be attributable to noise exposure.

Time of testing (12:30 or 3:00 p.m.) was counterbalanced across groups. The entire procedure took about 1.5 h. The experiment was approved by the Institutional Ethics Committee at the *Institut universitaire de gériatrie de Montréal* and was conducted with the understanding and consent of each participant.

Results

Cortisol data

Mean raw cortisol for each group at each timepoint was used as the dependent variable in an ANOVA, with group (tinnitus vs. control) as the between-subjects variable and time of sampling (0, +10, +20, +30, +40, and +60 min) as the within-subjects variable. This

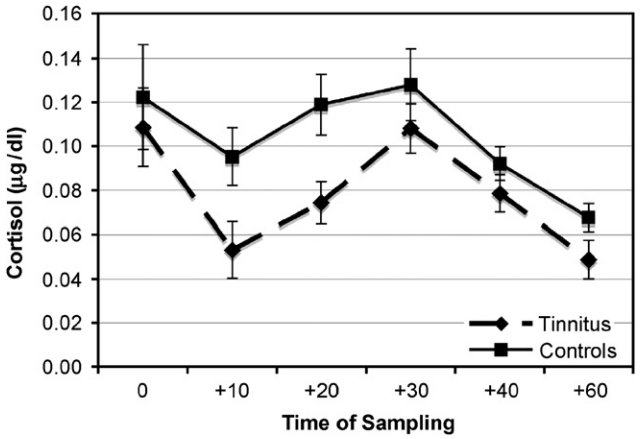

Fig. 1. Mean raw cortisol levels (with S.E.) for tinnitus and control groups.

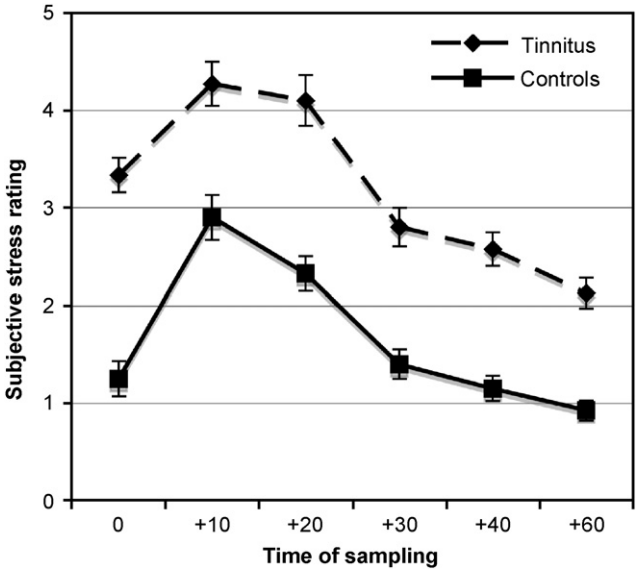

Fig. 2. Subjective stress ratings (with S.E.) for tinnitus and control groups.

analysis yielded no interaction between group and time of sampling ($F<1$). A main effect of group was found ($F(1, 38) = 4.40$, $p<.05$). The tinnitus group had overall *lower* cortisol levels than the control group (with means of 0.079 and 0.104, respectively). The main effect of time of sampling was also significant: $F(5, 190) = 8.81$, $p<.001$. Overall cortisol at +30, i.e., 10 min after the end of noise (mean = .12) was significantly higher than at +10, +40, and +60 (means of .07, .09, and .06, respectively, $p<.05$), but not higher than +20 or 0 (means = .10 and .12, respectively, $p>.05$) (Fig. 1).

Subjective stress rating scales

On subjective stress ratings (Fig. 3), the ANOVA, with group (tinnitus vs. control) as the between-subjects variable and time of sampling (0, +10, +20, +30, +40, and +60 min) as the within-subjects variable, yielded no interaction between group and time of sampling ($F(5, 190) = 1.03$, $p = .40$), but a significant main effect of group ($F(1, 38) = 12.60$, $p<.002$). The tinnitus group had overall higher ratings than the control group (means = 3.2 and 1.66, respectively).

There was also a significant main effect of time of sampling ($F(5, 190) = 25.77$, $p<.001$). Post-hoc comparisons revealed that ratings at +10 and +20, taken at midpoint and immediately at the end of noise, did not differ from one another (means = 3.59 and 3.21, $p>.05$), and were higher than those at 0, +30, +40, +60 (means = 2.29, 2.10, 1.86, and 1.53, respectively, $p<.05$). T0 and +60 also differed from one another ($p<.05$) (Fig. 2).

Tinnitus intensity rating scales

A main effect of time of sampling was found on tinnitus intensity rating ($F(5, 95) = 2.92$, $p<.02$) (Fig. 3, thick line). The only significant difference was between tinnitus intensity rating at T+60 and at T+10 ($p<.05$ for Tukey post-hoc comparisons). The remaining intensities did not differ from one another (all p's $>.05$).

Tinnitus intensity rating and tinnitus-related distress

A median split on the TRQ scores was performed to examine the contribution of tinnitus-related distress to tinnitus intensity ratings. As the median score on the TRQ was 17, 10 participants were assigned to the low distress group (<17) and 10 to the high distress group (>17). An ANOVA was performed on tinnitus ratings with group (low, high distress) as the between-subjects factor and time of sampling (0 to +30) as the within-subjects factor. A significant interaction between group and time of sampling was found ($F(5, 90) = 3.071$, $p<.02$), but not a main effect of group ($F(1, 18) = 1.425$, $p = .25$). Ratings for the high distress group were higher than for the low distress group at +10 (means = 6.28 and 4.17, $p<.04$), and marginally so at +20 (means = 6.17 and 4.17, $p<.06$) and +30 (means = 5.9 and 4.11, $p<.06$), but *not* at 0, +40, or +60 (all p's $>.05$) (see Fig. 3).

This effect was further supported by significant correlations between tinnitus intensity ratings and TRQ scores at sampling timepoints +10, +20, and +30 (with r values between .50 and .61, p values between .001 and .03).

Questionnaire data

Data are summarized in Table 2. Groups differed on total Hyperacusis scores ($F(1, 37) = 8.18$, $p<.008$), with means of 20.5 and 12.68 for tinnitus and controls,

respectively. The tinnitus group also had higher scores on the social (means = 9.10 and 4.12, $p<.001$) and emotional (means = 6.75 and 4.74, $p<.04$) subscales, but not on the attentional subscale (means = 4.65 and 3.74, $p = .34$). The two groups differed only marginally on depressive symptomatology ($t(38) = 1.9$, $p<.07$).

Correlations between measures

Correlations were run between psychometric measures (total Hyperacusis and BDI-II scores), PTAs, PTAs HF, and cortisol data (see Table 2). Significant correlations were found between BDI-II and Hyperacusis scores ($r(38) = .48$, $p<.003$), and between Hyperacusis and PTAs ($r(38) = .43$, $p<.006$). Total cortisol secretion was also negatively correlated with Hyperacusis ($r(38) = -.38$, $p<.02$), but not with PTAs or PTAs HF (both p's $>.10$).

For the tinnitus groups only, additional significant correlations were found between TRQ and BDI-II scores ($r(19) = .65$, $p<.002$) and between TRQ and Hyperacusis scores ($r(19) = .62$, $p<.004$).

Finally, to determine whether the BDI-II, hyperacusis, or PTA-high scores best explain total cortisol secretion, they were entered as predictors in a stepwise multiple regression with total cortisol secretion as the dependent variable. Hyperacusis explained 14% of the variance ($R^2 = 0.144$, $p<0.02$). PTA-high and Beck scores did not add to the explained variance and were not considered in the model.

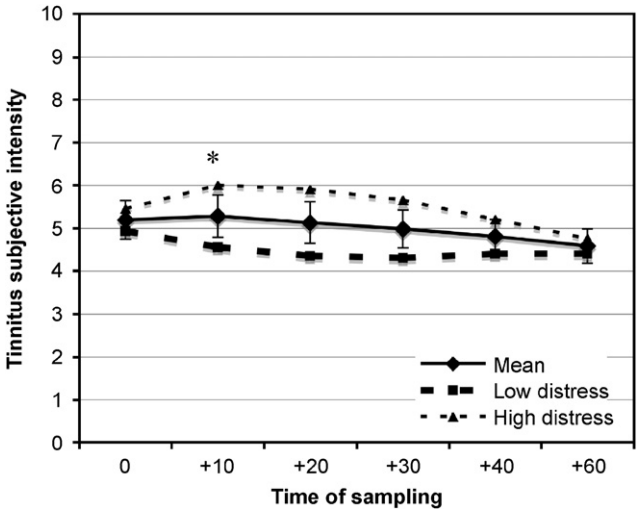

Fig. 3. Tinnitus subjective intensity ratings (with S.E.).

Discussion

The main findings of this study are that noise exposure at well below current safety standards increase (1) cortisol secretion in well-screened adults with tinnitus and healthy controls; (2) subjective stress levels; and (3) tinnitus intensity, especially in tinnitus sufferers that are already distressed by the condition.

Previous human and animals studies that have found cortisol increases with noise have used either longer exposure time, higher noise levels, or both. No data are currently available on specific dose/response in relation to cortisol in a laboratory setting. Until studies examine which specific types of noise, and at what levels, are needed to elicit a stress response from the HPA axis, it will be difficult to compare noise with, for instance, psychosocial stress tasks, which are widely reported in the stress literature. Here, after an initial drop from time 0, which could likely be attributed to stressful laboratory testing conditions, cortisol levels increased steadily during noise exposure, typically peaked after about 30 min, and then dropped when the noise was stopped. Both groups displayed this response, although lower cortisol levels were found in the tinnitus group, consistent with our previous study (Hébert and Lupien, 2007). A similar time course was used in the present study, but a stronger effect is seen than with the techno music used in our previous study (Hébert et al., 2005). It is noteworthy that current provincial standards allow workers to be exposed to continuous noise at 85 dBA up to 16 h. Given that our findings show increased cortisol, subjective stress, and tinnitus intensity with a common noise type fan at 80 dBA for only 20 min in relatively healthy people, this does not bode well for the long-term impacts of lengthy exposures on health and well-being. Exposing participants to higher noise levels in the present study would have posed serious ethical problems.

Table 2. Mean scores (S.D.) on the Hyperacusis and BDI-II questionnaires

	Tinnitus ($n = 20$)	Controls ($n = 20$)	p
Hyperacusis			
Attentional subscale	4.65 (2.94)	3.60 (2.95)	n.s.
Social subscale	9.10 (3.81)	4.21 (3.74)	<.001
Emotional subscale	6.75 (2.73)	4.74 (2.94)	<.04
Total	20.50 (8.33)	12.68 (8.73)	<.008
BDI-II	8.75 (6.37)	5.25 (5.2)	<.07

Paradoxically, the hearing loss group also showed greater sensitivity to sound, a finding that we have reported previously (Hébert et al., 2004; Hébert and Lupien, 2007; Hébert and Carrier, 2007). It may be related to mechanisms of tinnitus, such as decreased central inhibition (Eggermont and Roberts, 2004). More importantly, we show that participants with hearing problems (hearing loss, hyperacusis, and tinnitus) are more subjectively stressed under noise exposure, and that tinnitus increases, especially in those who are most distressed by it. These are also the most sensitive to noise. Indeed, in keeping with previous work, noise sensitivity was found to explain part of the variance related to total cortisol secretion (Waye et al., 2002). To our knowledge, our study is the first to demonstrate these effects. We may therefore conclude that noise exposure is more effective in increasing tinnitus intensity than psychosocial stress, even in short time frames.

Our study is limited by the fact that only cortisol levels were examined. The perception of a stressor by the brain triggers a complex cascade of events that include secretion of corticotropin-releasing hormone (CRH; a neuropeptide) by the hypothalamus, and adrenocorticotropin (ACTH) from the pituitary. The production of CRH and ACTH is triggered at a much faster rate following the perception of a stressor, when compared to the production of cortisol, which occurs after activation from ACTH on the adrenal glands. Since we did not measure CRH (in cerebrospinal fluid) or ACTH (in blood), it is thus possible that the time-course of activation of the HPA axis in tinnitus patients and controls is different than what we report for cortisol. However, it is important to note that salivary cortisol levels have been shown to be sensitive to an acute stress response, and consequently, we feel confident that the cortisol effects we see in this study are a result of noise exposure rather than of long-term exposure to stress in our groups of participants.

In conclusion, noise exposure, even within safety limits, has an effect on the HPA axis responsible for cortisol secretion, subjective stress, and tinnitus intensity. Our results point to the need for further studies to examine the effects of various noise levels and types on healthy as well as clinical populations.

Acknowledgements

We thank the Fonds de la Recherche en Santé du Québec and the Canadian Institutes for Health Research for salary supports awarded to S.H. and S.J.L., respectively. This research was made possible, thanks to research grants from The Hearing Foundation of Canada and, for S.H., from the Canadian Institutes for Health Research.

References

Alpini, D., Cesarini, A., 2006. Tinnitus as an alarm bell: stress reaction tinnitus model. ORL 68, 31–37.

Andersson, G., Stromgren, T., Strom, L., Lyttkens, L., 2002. Randomized controlled trial of internet-based cognitive behavior therapy for distress associated with tinnitus. Psychosom. Med. 64, 810–816.

Babisch, W., 2003. Stress hormones in the research on cardiovascular effects of noise. Noise and Health 5 (18), 1–11.

Beck, A.T., 1997. Beck Depression Inventory-II. The Psychological Corporation. Harcourt Canada Assessment Company, Toronto.

Bigert, C., Bluhm, G., Theorell, T., 2005. Saliva cortisol – a new approach in noise research to study stress effects. Int. J. Hyg. Environ. Health 208 (3), 227–230.

Biondi, M., Picardi, A., 1999. Psychological stress and neuroendocrine function in humans: the last two decades of research. Psychother. Psychosom. 68, 114–150.

Budd, R.J., Pugh, R., 1996. Tinnitus coping style and its relationship to tinnitus severity and emotional distress. J. Psychosom. Res. 41, 327–335.

Davis, A.C., 1995. Hearing in Adults. Whurr, London, UK.

Eggermont, J.J., Roberts, L., 2004. The neuroscience of tinnitus. Trends Neurosci. 27, 876–882.

Hébert, S., Carrier, J., 2007. Sleep complaints in elderly tinnitus patients: a controlled study. Ear Hear. 28 (5), 649–655.

Hébert, S., Lupien, S.J., 2007. The sound of stress: blunted cortisol reactivity to psychosocial stress in tinnitus sufferers. Neurosci. Lett. 2007. Epub 2006 November 2; January 10; 41(2):138–42.

Hébert, S., Paiement, P., Lupien, S.J., 2004. A physiological correlate for the intolerance to both internal and external sounds. Hear. Res. 190, 1–9.

Hébert, S., Béland, R., Dionne-Fournelle, O., Crête, M., Lupien, S.J., 2005. Physiological stress response to video-game playing: the contribution of built-in music. Life Sci. 76, 2371–2380.

Henry, J.L., Wilson, P.H., 2001. The Psychological Management of Chronic Tinnitus. Allyn Bacon, Boston.

Holgers, K.M., Erlandsson, S.I., Barrenas, M.L., 2000. Predictive factors for the severity of tinnitus. Audiol. 39, 284–291.

Holgers, K.-M., Zöger, S., Svedlund, K., 2005. Predictive factors for development of severe tinnitus suffering – further characterisation. Int. J. Audiol. 44, 584–592.

Khalfa, S., Dubal, S., Veuillet, E., Peretz-Dias, F., Jouvent, R., Collet, L., 2002. Psychometric normalization of a hyperacusis questionnaire. ORL J. Otorhinolaryngol. Relat. Spec. 64, 436–442.

Kirschbaum, C., Hellhammer, D.H., 1994. Salivary cortisol in psychoendocrine research: recent developments and applications. Psychoneuroendocrinology 19, 313–333.

Lockwood, A.H., Salvi, R.J., Coad, M.L., Towsley, M.L., Wack, D.S., Murphy, B.W., 1998. The functional neuroanatomy of tinnitus: evidence for limbic system links and neural plasticity. Neurology 50, 114–120.

Melamed, S., Bruhis, S., 1996. The effects of chronic industrial noise exposure on urinary cortisol, fatigue and irritability: a controlled field experiment. J. Occup. Environ. Med. 38, 252–256.

Meric, C., Pham, E., Chéry-croze, S., 2000. Validation of a French version of the Tinnitus reaction questionnaire : a comparison between data from English and French versions. J. Speech Lang. Hear. Res. 43, 184–190.

Schmitt, C., Patak, M., Kröner-Herwig, B., 2000. Stress and the onset of sudden hearing loss. Int. Tinn. J. 6, 41–49.

Tyler, R.S., 2000. Tinnitus Handbook. Singular, San Diego.

Vernon, J.A., Meikle, M.B., 2003. Tinnitus: clinical measurement. Otolaryngol. Clin. North Am. 36 (2), 293–305.

Vio, M.M., Holme, R.H., 2005. Hearing loss and tinnitus: 250 million people and a US $10 billion potential market. Drug Discov. Today 10, 1263–1265 (Editorial).

Walpurger, V., Hebing-Lennartz, G., Denecke, H., Pietrowsky, R., 2003. Habituation deficit in auditory event-related potentials in tinnitus complainers. Hear. Res. 181, 57–64.

Waye, K.P., Bengtsson, J., Rylander, R., Hucklebridge, F., Evans, P., Cow, A., 2002. Low frequency noise enhances cortisol among noise sensitive subjects during work performance. Life Sci. 70, 745–758.

Weber, C., Arck, P., Mazurek, B., Klapp, B.F., 2002. Impact of a relaxation training on psychometric and immunologic parameters in tinnitus sufferers. J. Psychosom. Res. 52, 29–33.

Weinstein, B.E., 2000. Geriatric Audiology. Thieme Medical Publishers Inc., New York.

Wilson, P.H., Henry, J.L., Bowen, M., Haralambous, G., 1991. Tinnitus reaction questionnaire: psychometric properties of a measure of distress associated with tinnitus. J. Speech Hear. Res. 34, 197–201.