Psychoneuroendocrinology (2014) 39, 21—32

Available online at www.sciencedirect.com

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journal homepage: www.elsevier.com/locate/psyneuen

Recent and long-term occupational noise exposure and salivary cortisol level

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Received 23 August 2013; received in revised form 26 September 2013; accepted 30 September 2013

KEYWORDS
Occupational noise;
Cortisol;
Hypothalamic pituitary adrenal axis;
Stress

Summary Environmental and occupational noise exposure have been related to increased risk of cardiovascular disease, hypothetically mediated by stress-activation of the hypothalamic—pituitary—adrenal (HPA) axis. The objective of this study was to investigate the relation between recent and long-term occupational noise exposure and cortisol level measured off work to assess a possible sustained HPA-axis effect. We included 501 industrial, finance, and service workers who were followed for 24 h during work, leisure, and sleep. Ambient occupational noise exposure levels were recorded every 5 s by personal dosimeters and we calculated the full-shift L_{AEq} value and estimated duration and cumulative exposure based on their work histories since 1980. For 332 workers who kept a log-book on the use of hearing protection devices (HPD), we subtracted 10 dB from every noise recording obtained during HPD use and estimated the noise level at the ear. Salivary cortisol concentration was measured at 20.00 h, the following day at awakening, and 30 min after awakening on average 5, 14 and 14.5 h after finishing work. The mean ambient noise exposure level was 79.9 dB(A) [range: 55.0—98.9] and the mean estimated level at the ear 77.7 dB(A) [range: 55.0—94.2]. In linear and mixed regression models that adjusted for age, sex, current smoking, heavy alcohol consumption, personal income, BMI, leisure-time noise exposure

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http://dx.doi.org/10.1016/j.psyneuen.2013.09.028

level, time since occupational noise exposure ceased, awakening time, and time of saliva sampling, we observed no statistically significant exposure response relation between recent, or long-term ambient occupational noise exposure level and any cortisol parameter off work. This was neither the case for recent noise level at the ear. To conclude, neither recent nor long-term occupational noise exposure levels were associated with increased cortisol level off work. Thus, our results do not indicate that a sustained activation of the HPA axis, as measured by cortisol, is involved in the causal pathway between occupational noise exposure and cardiovascular disease.

1. Introduction

Environmental and occupational noise exposure have been related to an increased risk of cardiovascular disease, hypothetically by stress-activation of the hypothalamic–pituitary–adrenal (HPA) axis (Babisch, 2003; Beelen et al., 2009; Davies et al., 2005). If so, noise can be viewed as a general and non-specific stressor, that exerts an acute as well as a sustained effect on millions worldwide every day (Bonde and Kolstad, 2012). Thirty percent of the European work force is exposed to noise so loud, that they have to raise their voice in order to talk (Eurofound, 2010), and 43% of the general population in the largest European cities is exposed to road traffic noise levels exceeding the EU-threshold of 50 dB(A) (TERM, 2012).

Activation of the HPA axis and release of cortisol are considered major components of physiological stress in humans (Kirschbaum and Hellhammer, 1999). Saliva cortisol has proven to be a valid measure of the plasma-free cortisol concentration and proficiently reflects the diurnal variation of cortisol with repeated measurements (Bigert et al., 2005).

The acute influence of industrial noise exposure on cortisol level and thus hyperactivity of the HPA axis has been shown in two intervention studies of noise exposed industrial workers. They showed a decrease in cortisol level after participants put hearing protection on (Melamed and Bruhis, 1996; Sudo et al., 1996). Lower saliva cortisol levels have also been observed in industrial workers on leisure days compared with work days (Fouladi et al., 2012). Diverging associations for environmental and occupational noise exposure and cortisol activity have also been reported (Babisch et al., 1988; Belojevic et al., 1990; Cavatorta et al., 1987; Rai et al., 1981; Selander et al., 2009). However, none of these studies addressed whether the effect of noise was only transient or persisted, but still at an increased level, after exposure had ceased and very little is known about the long-term implications of noise exposure. Demonstrating an exposure–response relation between recent and long-term noise exposure and cortisol level measured during non-exposure would provide strong evidence of a sustained stress effect. This would add further to stress-activation of the HPA axis as a pivotal link of the causal chain between noise exposure and cardiovascular disease and other extra-auditive health effects because transient increase in cortisol levels are less likely to have long-term health effects.

The objective of this study was to investigate if recent and long-term occupational noise exposure at work is associated with a subsequently dose-dependent increase in salivary cortisol level off work as an indication of sustained stress activation of the HPA axis.

2. Methods

In 2001–2002, we conducted a study of companies recruited at random from the 10 Danish industrial trades (manufactures of food, wood products, non-metallic mineral products, basic metals, fabricated metal, machinery, motor vehicles, furniture, publishing and printing, and construction) with high reporting of noise induced hearing loss according to the Danish Working Environment Authority (Danish Working Environment Authority, 2013), children day care units, and as a reference finance and service, a total of 86 companies. In 2009–10, 42 of the companies agreed to participate in a second study, as well as 34 new companies recruited according to a similar procedure as in 2001–2. From these 76 companies, 516 workers agreed to participate in the present study. Furthermore, all workers from the 44 companies of the 2001–2 study, not participating in the second round, were invited to the local hospital to participate again in 2009–10 and 149 workers agreed. In total, 665 participants participated for 2 consecutive days, and were personally instructed and handed the study material by biomedical laboratory technologists during the first study day and the material was collected approximately 24 h later. This study focused on 523 workers from the 10 industrial trades and 81 workers from the financial and other services. We excluded 43 participants invited to the local hospital that were not exposed to work-related noise on the measurement day (33 unemployed or retired, 10 on sick leave, maternity leave, or off duty).

All participants filled in the questionnaire, 526 participants provided a noise level measurement during work on the first day, and of these, 504 collected saliva samples. We excluded night-workers ($N = 3$). The study population then comprised of 501 workers, of these 415 were blue-collar workers and 86 were white-collar workers, based on their ISCO-88 codes (ILO, 1990). 132 participants filled a log-book with detailed information on usage of HPD and 200 did not use HPD according to questionnaire information.

The study protocol was approved by the local ethics committee (M-20080239) and the Danish Data Protection Agency (2009-41-3072).

2.1. Noise exposure assessment

We measured noise level as A-weighted equivalent sound level means (L_{AEq}) by personal dosimeters (Brüel & Kjær, model 4443 and 4445) recording every 5 s for 24 h with a range set to 70–120 dB(A). All measurements were synchronized with the journal information, so the timing of each 5 s measurement relative to work or leisure could be identified. Based on this we calculated the recent ambient noise exposure level as the mean L_{AEq}-value for noise exposure