

HOSTED BY



ELSEVIER

Available online at www.sciencedirect.com

ScienceDirect

www.elsevier.com/locate/ssci

Environmental noise and sleep disturbances: A threat to health?

Demian Halperin*

Department of Psychiatry, Barzilai Medical Center, Haistadrut Street 2, Ashkelon 78278, Israel

ARTICLE INFO

Article history:

Received 2 July 2014

Received in revised form

29 August 2014

Accepted 1 September 2014

Available online 15 November 2014

Keywords:

Environmental noise

Sleep disturbances

Health outcomes

ABSTRACT

Environmental noise, especially that caused by transportation means, is viewed as a significant cause of sleep disturbances. Poor sleep causes endocrine and metabolic measurable perturbations and is associated with a number of cardiometabolic, psychiatric and social negative outcomes both in adults and children. Nocturnal environmental noise also provokes measurable biological changes in the form of a stress response, and clearly affects sleep architecture, as well as subjective sleep quality. These sleep perturbations are similar in their nature to those observed in endogenous sleep disorders. Apart from these measurable effects and the subjective feeling of disturbed sleep, people who struggle with nocturnal environmental noise often also suffer the next day from daytime sleepiness and tiredness, annoyance, mood changes as well as decreased well-being and cognitive performance. But there is also emerging evidence that these short-term effects of environmental noise, particularly when the exposure is nocturnal, may be followed by long-term adverse cardiometabolic outcomes. Nocturnal environmental noise may be the most worrying form of noise pollution in terms of its health consequences because of its synergistic direct and indirect (through sleep disturbances acting as a mediator) influence on biological systems. Duration and quality of sleep should thus be regarded as risk factors or markers significantly influenced by the environment and possibly amenable to modification through both education and counseling as well as through measures of public health. One of the means that should be proposed is avoidance at all costs of sleep disruptions caused by environmental noise.

© 2014 Brazilian Association of Sleep. Production and Hosting by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/3.0/>).

Background

The World Health Organization (WHO) has documented seven categories of adverse health and social effects of noise pollution, whether occupational, social or environmental: hearing impairment, interference with spoken communication, cardiovascular disturbances, mental health problems, impaired cognition, negative social behaviors and sleep disturbances [1]. The latter is considered the most deleterious

non-auditory effect because of its impact on quality of life and daytime performance [2–4]. Environmental noise, especially that caused by transportation means, is a growing problem in our modern cities [5]. It is considered a major cause of exogenous sleep disturbances, after somatic problems and day tensions [6,7].

Sleep is an important modulator of hormonal release, glucose regulation and cardiovascular function. In particular slow-wave sleep, the most restorative sleep stage, is

*Tel.: +972 50 327 84 24.

E-mail address: demianhalperin@gmail.com

associated with decreased heart rate, blood pressure, sympathetic nervous activity and cerebral glucose utilization, compared with wakefulness. During this sleep stage, growth hormone is released while stress hormone cortisol is inhibited [8,9]. Healthy sleep plays also an important role in memory consolidation [10]. Poor sleep causes measurable changes on these systems. Experimental studies demonstrated that both sleep restriction and poor quality sleep affect glucose metabolism by reducing glucose tolerance and insulin sensitivity [11-13] and that sleep restriction dysregulates appetite (lower levels of leptin and higher levels of ghrelin) [14,15] as well as cortisol levels [8]. Sleep restriction has also been shown to increase blood pressure [16,17] and affect immune processes [18]. It has been hypothesized that these perturbations cause long-term consequences on health [19].

Indeed there is increasing evidence that quantitative and qualitative sleep disturbances may play a role in the development of cardiometabolic disease. A number of cardiovascular risk factors and cardiovascular outcomes have been associated with disturbed sleep: coronary artery calcifications, atherogenic lipid profiles, atherosclerosis, obesity, type 2 diabetes, hypertension, cardiovascular events [19,20]. Increased mortality from all causes has also been observed [21]. During the past years, the relationship between insomnia and psychiatric disorders has come to be considered synergistic, including bi-directional causation. It has become clear that insomnia is not merely a symptom of psychiatric disorders, but contributes also to the risk of future relapse or the development of new onset mood, anxiety, and substance use disorders, as well as to the severity of psychiatric symptoms [22]. Disturbed sleep has also been associated with increased frequency of violent acts as well as domestic violence, work and vehicle accidents, increased work absenteeism [23-26]. The observed associations between poor sleep and obesity, diabetes, depression, aggressive and delinquent behaviors concern children and adolescents, too [27-30]. As a result of sleep disturbances, children also suffer from impaired cognition and worsening of attention deficit hyperactivity disorder symptoms [31].

Nocturnal environmental noise also provokes measurable metabolic and endocrine perturbations (increased secretion of adrenaline, noradrenaline, cortisol), increased heart rate and arterial pressure, and increased motility. These biological responses to noise during sleep are most of the time unnoticed [32-34]. Noise also affects sleep architecture, as well as subjective sleep quality. Nocturnal air traffic causes nocturnal awakenings at levels as low as 48 dB, and physiological reactions in the form of increased vegetative hormonal secretions, cortical arousals and body movements at even lower levels, probably around 33 dB [33,35,36]. Nocturnal noise has been shown to fragment sleep, and as a consequence lead to a redistribution of time spent in the different sleep stages, typically increasing wake and stage 1 sleep and decreasing slow wave sleep and REM sleep, i.e. causing a shallower sleep [7,32,37]. Basner et al. showed that although these effects on sleep structure and continuity are relatively modest, they have a significant impact on subjective assessments on sleep quality and recuperation: Subjects experience their sleep as disturbed and with low recuperative value.

Also, despite being most of the time in an unconscious state, subjects are able to distinguish between nights with low and high degrees of traffic noise exposure. Their reaction time at next day performance test is also slightly but significantly increased [7]. These findings corroborate with previous observations that noise is indeed a widespread factor of self-reported sleep disturbances [38].

Apart from these measurable effects and the subjective feeling of disturbed sleep, people who struggle with nocturnal environmental noise often also suffer the next day from daytime sleepiness and tiredness, annoyance, mood changes as well as decreased well-being and cognitive performance [1,4,10,39-42]. Associations between exposure to aircraft noise and the following health complaints and health indicators have been demonstrated: headache, poor self-rated health status, use of medication for cardiovascular diseases and use of sleep medication [40,43]. Could these short-term effects be also followed by long-term adverse health outcomes? Data show that exposure to traffic noise, not specifically at night, is associated with increased incidence of diabetes [44], hypertension [45] and stroke among the elderly [46], as well as increased incidence and mortality from coronary heart disease [47-50]. But interestingly some epidemiological data support the hypothesis that exposure to noise at night time may be especially relevant in terms of negative cardiovascular outcomes, perhaps due to the fact that repeated autonomic arousals habituate to a much lesser degree to noise than cortical arousals [2,7]. Indeed data show that exposure to traffic noise especially at night increases the risk for hypertension [43], also in children [51], as well as the risk for heart disease and stroke [52]. These results confirm previous findings of studies looking at the association between subjective responses to community noise and cardiovascular outcomes that suggested that night-time noise may be more a determinant of noise-induced cardiovascular effects than daytime exposure [53-55].

As we previously described, poor sleep triggers biological mechanisms contributing to the deterioration of somatic health and is clearly associated with significant psychiatric morbidity, too. Whereas exposure to traffic noise around-the-clock seems to be clearly associated with adverse health outcomes, the question of health consequences of noise exposure specifically at night still needs to be further explored, since the majority of evidence so far comes either from observational field studies that look at the immediate consequences of nocturnal noise exposure or from epidemiological studies that do not usually separate nocturnal from diurnal exposure. However, although sleep structure perturbations in the context of nocturnal noise seem less severe than in sleep pathology such as obstructive sleep apnea, they tend to be similar in their nature [2]. It is thus reasonable to hypothesize that poor sleep may act as a mediator between nocturnal noise pollution and increased risk of cardiovascular morbidity, through impaired endocrine and metabolic functions [11]. Also, by affecting sleep architecture, environmental noise pollution causes sleep disturbances that lead to subjective distress in the form of daytime sleepiness and tiredness, decreased well-being and cognitive performance, as well as mood changes and potentially more serious psychopathology and psychiatric morbidity, although this remains

to be proven. Finally, as we have seen, by affecting biological systems in the form of a stress response causing the release of stress hormones which in turn affects factors such as blood pressure and heart rate, noise, especially at night, may also increase the risk of cardiovascular morbidity by a direct mechanism. In 2009, considering the evidence so far, a group of experts working WHO regional office for Europe officially recommended that an $L_{\text{night, outside}}$ (average level of sound pressure at night) of 40 dB (and 55 dB as an interim target) should be the target to be achieved in order to prevent nocturnal noise deleterious health consequences [55].

Conclusion

There is clear evidence that sleep disturbances are associated with health deterioration, and growing evidence that exposure to noise pollution, around-the-clock, negatively affects health, too. It has also been proven that nocturnal noise pollution significantly impairs sleep, objectively and subjectively. Whether these noise-induced sleep disturbances represent the link between environmental noise exposure and negative health outcomes still remains uncertain. However, the emerging data suggest that indeed nocturnal environmental noise may be the most worrying form of noise pollution in terms of its health consequences, possibly because of its synergistic direct and indirect (through sleep disturbances) influence on biological systems. Duration and quality of sleep should thus be regarded as risk factors or markers significantly influenced by the environment and possibly amenable to modification through both education and counseling as well as through measures of public health. One of the means that should be proposed is avoidance at all costs of sleep disruptions caused by environmental noise. Furthermore, more large scale prospective studies are needed. These studies should involve representative samples of the population including vulnerable groups like children, elderly and mentally ill subjects, have a sufficient follow-up period, assess health outcomes according to daytime versus nighttime exposure, assess hormonal and polysomnographic measures, and take into consideration potential confounders. Subgroup sleep analyses should also be performed. This would help to better understand to what extent sleep disturbances indeed mediate between exposure to environmental noise and negative health consequences.

Acknowledgment

I would like to express my gratitude to Prof. Mathias Basner for his kind advice.

REFERENCES

- [1] Goines L, Hagler L. Noise pollution: a modern plague. *South Med J* 2007;100(3):287–94.
- [2] Basner M, Babisch W, Davis A, Brink M, Clark C, Janssen S, et al. Auditory and non-auditory effects of noise on health. *Lancet* 2014;383(9925):1325–32.
- [3] Fritschi L, Brown A, Kim R, Schwela D, Kephelopoulos S. Burden of disease from environmental noise. Bonn: World Health Organization; 2011.
- [4] Muzet A. Environmental noise, sleep and health. *Sleep Med Rev* 2007;11(2):135–42.
- [5] Carlos D. A different route to health: implications of transport policies. *Br Med J* 1999;318:1686–9.
- [6] Stansfeld SA, Matheson MP. Noise pollution: non-auditory effects on health. *Br Med Bull* 2003;68:243–57.
- [7] Basner M, Muller U, Elmenhorst EM. Single and combined effects of air, road, and rail traffic noise on sleep and recuperation. *Sleep* 2011;34(1):11–23.
- [8] Copinschi G. Metabolic and endocrine effects of sleep deprivation. *Essent Psychopharmacol* 2005;6(6):341–7.
- [9] Van Cauter E, Spiegel K, Tasali E, Leproult R. Metabolic consequences of sleep and sleep loss. *Sleep Med* 2008;9 (Suppl. 1):S23–8.
- [10] Basner M. Nocturnal aircraft noise exposure increases objectively assessed daytime sleepiness. *Somnologie* 2008;12 (2):110–7.
- [11] Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet* 1999;354 (9188):1435–9.
- [12] Buxton OM, Pavlova M, Reid EW, Wang W, Simonson DC, Adler GK. Sleep restriction for 1 week reduces insulin sensitivity in healthy men. *Diabetes* 2010;59(9):2126–33.
- [13] Spiegel K, Tasali E, Leproult R, Van Cauter E. Effects of poor and short sleep on glucose metabolism and obesity risk. *Nat Rev Endocrinol* 2009;5(5):253–61.
- [14] Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Ann Intern Med* 2004;141(11):846–50.
- [15] Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Med* 2004;1(3):e62.
- [16] Lusardi P, Zoppi A, Preti P, Pesce RM, Piazza E, Fogari R. Effects of insufficient sleep on blood pressure in hypertensive patients: a 24-h study. *Am J Hypertens* 1999;12:63–8 (1 Pt 1).
- [17] Tochikubo O, Ikeda A, Miyajima E, Ishii M. Effects of insufficient sleep on blood pressure monitored by a new multibiomedical recorder. *Hypertension* 1996;27(6):1318–24.
- [18] Miller MA, Cappuccio FP. Inflammation, sleep, obesity and cardiovascular disease. *Curr Vasc Pharmacol* 2007;5 (2):93–102.
- [19] Knutson KL. Sleep duration and cardiometabolic risk: a review of the epidemiologic evidence. *Best Pract Res Clin Endocrinol Metab* 2010;24(5):731–43.
- [20] Knutson KL. Sociodemographic and cultural determinants of sleep deficiency: implications for cardiometabolic disease risk. *Soc Sci Med* 2013;79:7–15.
- [21] Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. *Sleep* 2010;33(5):585–92.
- [22] Krystal AD. Psychiatric disorders and sleep. *Neurol Clin* 2012;30(4):1389–413.
- [23] Daley M, Morin CM, LeBlanc M, Gregoire JP, Savard J, Baillargeon L. Insomnia and its relationship to health-care utilization, work absenteeism, productivity and accidents. *Sleep Med* 2009;10(4):427–38.
- [24] Grano N, Vahtera J, Virtanen M, Keltikangas-Jarvinen L, Kivimaki M. Association of hostility with sleep duration and sleep disturbances in an employee population. *Int J Behav Med* 2008;15(2):73–80.
- [25] Hoshino K, Campregheer Pasqualini J, Pessanha D'Oliveira E, Pires da Silva C, Esteves Modesto A, Silveira R. Is sleep

- deprivation involved in domestic violence? *Sleep Sci* 2009;2(1):14–20.
- [26] Leger D, Bayon V, Ohayon MM, Philip P, Ement P, Metlaine A, et al. Insomnia and accidents: cross-sectional study (EQUINOX) on sleep-related home, work and car accidents in 5293 subjects with insomnia from 10 countries. *J Sleep Res* 2014;23(2):143–52.
- [27] Chen X, Beydoun MA, Wang Y. Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. *Obesity (Silver Spring)* 2008;16(2):265–74.
- [28] Gregory AM, Eley TC, O'Connor TG, Plomin R. Etiologies of associations between childhood sleep and behavioral problems in a large twin sample. *J Am Acad Child Adolesc Psychiatry* 2004;43(6):744–51.
- [29] Johnson EO, Roth T, Breslau N. The association of insomnia with anxiety disorders and depression: exploration of the direction of risk. *J Psychiatr Res* 2006;40(8):700–8.
- [30] Matthews KA, Dahl RE, Owens JF, Lee L, Hall M. Sleep duration and insulin resistance in healthy black and white adolescents. *Sleep* 2012;35(10):1353–8.
- [31] Owens JA. A clinical overview of sleep and attention-deficit/hyperactivity disorder in children and adolescents. *J Can Acad Child Adolesc Psychiatry* 2009;18(2):92–102.
- [32] Basner M, Samel A. Effects of nocturnal aircraft noise on sleep structure. *Somnologie* 2005;9(2):84–95.
- [33] Basner M, Samel A. Nocturnal aircraft noise effects. *Noise Health* 2004;6(22):83–93.
- [34] Selander J, Bluhm G, Theorell T, Pershagen G, Babisch W, Seiffert I, et al. Saliva cortisol and exposure to aircraft noise in six European countries. *Environ Health Perspect* 2009;117(11):1713–7.
- [35] Maschke C, Hecht K, Wolf U. Nocturnal awakenings due to aircraft noise. Do wake-up reactions begin at sound level 60 dB(A)? *Noise Health* 2004;6(24):21–33.
- [36] Basner M, Samel A, Isermann U. Aircraft noise effects on sleep: application of the results of a large polysomnographic field study. *J Acoust Soc Am* 2006;119:2772–84 (5 Pt 1).
- [37] Griefahn B, Marks A, Robens S. Noise emitted from road, rail and air traffic and their effects on sleep. *Sound Vib* 2006;295:129–40.
- [38] Miedema HM, Vos H. Associations between self-reported sleep disturbance and environmental noise based on reanalyses of pooled data from 24 studies. *Behav Sleep Med* 2007;5(1):1–20.
- [39] Babisch W, Houthuijs D, Pershagen G, Cadum E, Katsouyanni K, Velonakis M, et al. Annoyance due to aircraft noise has increased over the years—results of the HYENA study. *Environ Int* 2009;35(8):1169–76.
- [40] Franssen EA, van Wiechen CM, Nagelkerke NJ, Lebet E. Aircraft noise around a large international airport and its impact on general health and medication use. *Occup Environ Med* 2004;61(5):405–13.
- [41] Basner M, Griefahn B, Berg M. Aircraft noise effects on sleep: mechanisms, mitigation and research needs. *Noise Health* 2010;12(47):95–109.
- [42] Elmenhorst EM, Elmenhorst D, Wenzel J, Quehl J, Mueller U, Maass H, et al. Effects of nocturnal aircraft noise on cognitive performance in the following morning: dose–response relationships in laboratory and field. *Int Arch Occup Environ Health* 2010;83(7):743–51.
- [43] Jarup L, Babisch W, Houthuijs D, Pershagen G, Katsouyanni K, Cadum E, et al. Hypertension and exposure to noise near airports: the HYENA study. *Environ Health Perspect* 2008;116(3):329–33.
- [44] Sorensen M, Andersen ZJ, Nordsborg RB, Becker T, Tjønneland A, Overvad K, et al. Long-term exposure to road traffic noise and incident diabetes: a cohort study. *Environ Health Perspect* 2013;121(2):217–22.
- [45] Babisch W, Kamp I. Exposure–response relationship of the association between aircraft noise and the risk of hypertension. *Noise Health* 2009;11(44):161–8.
- [46] Sorensen M, Hvidberg M, Andersen ZJ, Nordsborg RB, Lillelund KG, Jakobsen J, et al. Road traffic noise and stroke: a prospective cohort study. *Eur Heart J* 2011;32(6):737–44.
- [47] Huss A, Spoerri A, Egger M, Roosli M. Aircraft noise, air pollution, and mortality from myocardial infarction. *Epidemiology* 2010;21(6):829–36.
- [48] Selander J, Nilsson ME, Bluhm G, Rosenlund M, Lindqvist M, Nise G, et al. Long-term exposure to road traffic noise and myocardial infarction. *Epidemiology* 2009;20(2):272–9.
- [49] Sorensen M, Andersen ZJ, Nordsborg RB, Jensen SS, Lillelund KG, Beelen R, et al. Road traffic noise and incident myocardial infarction: a prospective cohort study. *PLoS One* 2012;7(6):e39283.
- [50] Babisch W. Updated exposure–response relationship between road traffic noise and coronary heart diseases: a meta-analysis. *Noise Health* 2014;16(68):1–9.
- [51] van Kempen E, van Kamp I, Fischer P, Davies H, Houthuijs D, Stellato R, et al. Noise exposure and children's blood pressure and heart rate: the RANCH project. *Occup Environ Med* 2006;63(9):632–9.
- [52] Floud S, Blangiardo M, Clark C, de Hoogh K, Babisch W, Houthuijs D, et al. Exposure to aircraft and road traffic noise and associations with heart disease and stroke in six European countries: a cross-sectional study. *Environ Health* 2013;12:89.
- [53] Babisch W, Beule B, Schust M, Kersten N, Ising H. Traffic noise and risk of myocardial infarction. *Epidemiology* 2005;16(1):33–40.
- [54] Babisch W, Ising H, Gallacher JE, Sweetnam PM, Elwood PC. Traffic noise and cardiovascular risk: the Caerphilly and Speedwell studies, third phase—10-year follow up. *Arch Environ Health* 1999;54(3):210–6.
- [55] World Health Organisation (WHO). Night noise guidelines for Europe. Copenhagen, Denmark: World Health Organisation (WHO); 2009. (http://www.euro.who.int/__data/assets/pdf_file/0017/43316/E92845.pdf) (accessed July 20, 2014).