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NON-AUDITORY HEALTH EFFECTS OF NOISE: UPDATED REVIEW

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1. CHANGES IN THE NOISE - PHYSIOLOGICAL HEALTH PARADIGM

Interest in nonauditory physiological effects of noise on man continues to be high as noise exposures in the living and work environments of most populations worsen. Research on long-term nonauditory noise effects has been motivated primarily by findings from laboratory studies showing that noise activates endocrine and sympathetic systems triggering acute physiologic changes identical to a general stress response. The major health effects studied are cardiovascular, in particular elevated blood pressure (BP) and ischemic heart disease (IHD); mental health problems; adverse reproductive outcomes; and immune responses.

In early research, investigators tended to assume that noise produced direct effects and gave little attention to the individual differences in response to noise as a stressor or to the role of other stress producing factors . Most of these studies were cross-sectional in design, lacked from sufficient control of confounding, had sample sizes too small to detect the small differences expected, and poorly differentiated the level of noise between the high and low exposed groups being compared. Although, as a whole, the research suggested an association between noise exposure and chronic health conditions, results from these methodologically weak studies in very diverse populations were far from being consistent [1].

Better designed studies in the 1980's added to the understanding of the reasons for observed disparate findings. The more methodologically rigorous studies tended to show no or weak associations between high noise and elevated blood pressure, heart disease, mental health problems, low birthweight and birth malformations when major confounding factors were controlled. Varying responses to noise due to different acoustical characteristics, socio-situational factors and contextual factors in the physical/social environment, and modifying factors were recognized but rarely considered.[2] Findings from studies of subjective reactions to noise on health raised the most questions as to how physical effects of noise might be modified. Job's [3] recent critical analysis of these findings, suggests there are many

complex and interrelating roles of psychological factors in determining any physical effect of long-term noise exposure. He also posits that negative subjective reactions to noise predict health outcomes over and above the prediction available from the direct exposure to noise itself. Some of the factors which are believed to result in large individual differences in response to noise include appraisal of the personal significance of noise and perceived possibilities for its control, general attitude toward the noise source, learned helplessness after long exposure, sensitivity to noise and general coping styles.

Inconsistent results and the possibility that direct noise effects are modified by other factors have led to recent changes in studies of non-auditory health effects of noise. Investigators seem more cautious about making strong causal inferences from less than longitudinal data. There is clear recognition of the need for more rigorously designed studies and the necessity for controlling for confounding variables. Measures of reactions to noise as a stressor and other effect modifiers are being introduced into study designs in addition to measures of sound level. This change toward studying the indirect routes whereby noise may influence health requires new conceptual and statistical approaches to the data. While some researchers continue to focus on adjusting for covariates and examining direct noise effects, a few investigators are beginning to search for indirect effects by stratifying on potential modifying factors or testing for interactions between these modifiers and noise exposure. Several of the more recent studies are reviewed below.

2. EVIDENCE OF NOISE EFFECTS

Long-term Cardiovascular Effects

Studies have consistently shown that the strongest evidence of a non-auditory health effect of noise is for an association between high noise levels and BP with prevalence ratios for hypertension ranging between 1.6 and 2.8 [2]. Several investigators [4,5,6,7] have reconstructed cross-sectional data to resemble historical prospective or case-control study designs. Lang et al. found that workers exposed for 25 or more years to 85-100 dB noise had higher BPs than workers with shorter exposures [4]. Fogari et al. matched workers on age, years employed and body mass index, and reported significantly higher systolic (SBP) and diastolic blood pressures (DBP) among workers exposed to noise >=85 dB than among those exposed to <80 dB noise. In these case-control data the hypertension odds ratio was 1.77 (p=.03), slightly higher than the overall prevalence ratio of 1.59 in the cross-sectional data.[5] The study which offers the strongest evidence of an association between occupational noise and BP covered the range of noise from 75 to 104 dBA and demonstrated a dose-response relationship between level of noise and prevalence of hypertension among women who had worked their entire lives in high noise with unprotected ears. The odds of hypertension increased by 1.2 for each 5 dB increase in noise (odds ratio=1.8 at 95 dBA), after adjusting for age, working years, salt intake and family history of hypertension.[6]

At least three studies have shown no cardiovascular effects of noise [7,8,9]. Hirar et al., controlling for temperature conditions at work, found similar increments in blood pressure and in hypertension rates for the noise exposed and

controls when comparing baseline and 10 year followup measures [7]. Hessel and Sluis-Cremer followed miners over a 27 year period by examining historic records at approximately three year intervals and estimating noise levels for individual jobs and work areas. The cross-sectional, the longitudinal analysis of change in noise and BP and the longitudinal categorical analyses all showed no association between noise exposure and BP. [8] Neither univariate nor multivariate analyses showed associations of noise exposures with blood pressure in the CORDIS study. A large number of possible modifying factors were considered including noise exposures at home and noise outside the work department as an indicator of noise over which the worker had no control. There was no evidence that noise beyond one's control produces more adverse effects than controllable noise, or that noise at home contributes to the work-noise-BP relationship. As the authors noted, the lack of an association may be related to the fact that the noise levels were relatively low and the possibility of a healthy worker effect could not be ruled out. [9] Similar to an earlier finding by Green et al. [10], and contrary to expectation, ear protector use was related positively to heart rate suggesting such usage may serve as a stressor.

Several investigators have suggested that nonauditory noise effects may be more closely correlated with suitable subjective noise parameters than with the actual sound level. In the Berlin population-based case-control traffic noise study, subjective work noise assessments were compared to the median of measured sound levels in a sample of subjects making it possible to express risk by noise levels. The risk for myocardial infarction (MI) at noise levels 79-99 dB and > 100 dB were 2.0 and 3.8 respectively, after controlling for multiple confounders.[11] In a community survey Lercher et al. found expressed annoyance with work noise had a small significant effect on DBP, but a nonsignificant effect on SBP [12]. Apparently no attempt was made to relate noise annoyance to noise level.

Recent findings from studies of physiological effects of relatively low environmental noise levels point to the importance of ambient noise as a widespread stressor. The recent Luebeck study indicated a slightly higher prevalence of hypertension for men living in high traffic areas compared to low traffic noise areas (odds ratio 1.3) [13]. While blood pressure effects were not observed in another well controlled cross-sectional study, significant associations were found between noise and heart disease risk factors including platelet count, glucose, plasma viscosity and total triglycerides [14]. One Austrian study of the effects of road traffic noise showed individuals with a situation-oriented coping style or an optimistic attitude to have lower BPs than individuals with other coping styles [15].

Two cross-sectional studies suggest that subtle changes in blood pressure from noise exposure may begin early in life. A study of 3-7 year old children exposed to traffic noise levels of <60 dBA to >70 dBA near their kindergartens and residences showed a significant increase in mean SBP and DBP and decrease in heart rate with higher traffic noise levels. Moreover, the mean BP for children from quiet homes did not rise with age, unlike in children from noisy areas. [16]. Another study of 7-12 year olds showed that both SBP and DBP were lower in children who were deaf before one year of age than in normal-hearing children [17].

The evidence for an increased risk of IHD for people living in traffic areas of

more than 65-70 dBA noise is somewhat stronger than that for blood pressure changes. After 4 years of followup of men in Caerphilly and Speedwell, results suggest a marginal increase in risk for IHD incidence (RR 1.1, based on 9 risk factors) for men exposed to traffic noise >66-70 dBA compared to men exposed to the lowest noise level, 51-55 dBA [18]. Slightly stronger risks for IHD were observed in the Berlin population based case-control study. Although not significant, there was a trend of increasing risk for MI of 1.1 and 1.5 in the higher noise categories of 71-75 dBA and 76-80 dBA compared to the lowest noise level of 51-60 dBA. Among men who had not moved in 15 years, the risks were a little larger; when the noise categories were combined, the risk was 1.3, $p < 0.10$. [19]

Experimental research indicates that noise events produce arousals and sleep stage changes and their likelihood varies with the sleep stage at noise onset. Frequent arousals due to noise could increase total autonomic nervous system activity with possible long-term effects. Slow-wave sleep can be reduced by intermittent traffic noise. If slow-wave sleep is necessary for brain restitution, chronic depletion of this stage of sleep due to noise may indirectly affect long term health in many ways. [20,21] Further research in the form of examination of sleep loss suffered by noise exposed and nonexposed individuals at baseline with long-term followup of physiological health effects has been suggested [3].

Although the effect of noise on blood pressure and other cardiovascular functions does not appear to be as strong as indicated by early studies, the evidence is strong enough and the effects are so biologically plausible that further research is clearly warranted. This research should focus on effect modifiers such as coping styles and sleep loss which may help explain the weak and inconsistent effects currently observed in population studies.

Effects of noise on pregnancy

In most countries few pregnant women are exposed to extremely high noise levels. Thus the literature on noise effects of reproductive outcomes remains rather limited. The continuing studies of Finnish researchers show no differences in fetal mortality or birthweight between exposed and nonexposed women in industries where noise levels are relatively low [22,23]. One case-control study showed that self reported exposure to occupational noise during pregnancy increased risk to antepartum fetal death, but had no effect on birthweight or birth defects [24] .

Another study showed that the perception of noise does not appear to be more important than the assessed noise intensity. Birthweights of babies born to mothers who reported exposure to noise and those who did not were similar; mothers who were assessed as noise exposed by industrial hygienists more often had infants who were small for gestational age than mothers assessed not to be exposed. Wearing of hearing protectors did not safeguard against the risk of low birthweight.[25]

No clear relationship between noise exposure and pregnancy outcomes can be established at present. With the increasing numbers of women continuing to work throughout pregnancy and the possibility that hearing protectors may not safeguard against pregnancy risks, more attention should be given to the study of effects of even moderately high noise exposure on pregnancy outcomes.

Mental Health Effects

Determining the effects of environmental noise on psychiatric disorders has been complicated by the complex interplay between noise, annoyance, noise sensitivity and mental health disorders/symptoms. In two studies designed to disentangle these relationships, Stansfeld concluded that environmental noise does not cause clinically defined psychiatric disorder, but in certain subgroups of the population the meaning of noise for an individual, typified by noise sensitivity, may be a moderator between noise and psychological morbidity [26]. This conclusion remained when cross-sectional data from the Caerphilly and Speedwell Collaborative Studies were analyzed using logistic regression to test for interactions of noise sensitivity on noise exposure level and psychiatric caseness [27].

Overall, the evidence of a relationship between psychiatric disorders and noise is weak. Nevertheless, prospective studies are warranted to clarify the role of noise sensitivity in moderating relationships between noise and psychologic morbidity.

Immune System Effects

It has recently been suggested that noise may suppress the immune system through its effects on non-specific stress responses. In 1993, Bly et al. [28] found the scant data on noise and the immune system to be inconclusive and recommended further study. About the same time, Altena and Beersman [29], concluded that nocturnal noise which disturbed the distribution of sleep stages should be considered a health risk because of direct immunosuppressive effects (specifically inhibition of eosinophils and basophils which usually proliferate during sleep). They further concluded that the threshold for immunosuppressive effects may lie at about 40 dBA continuous and intermittent noise. While Mackhe et al. [30] found evidence of a stress response with deterioration of sleep quality among subjects exposed to intermittent nocturnal aircraft noise, Carter et al. [31] did not.

Although the evidence is inclusive, the information on noise effects on sleep taken together with evidence of an association between sleep and the immune response, suggests that immune responses should be studied in people exposed to noise during sleep, especially those exposed to intermittent transportation noise.

3. CONCLUSION

There has been progress in understanding the non-auditory effects of noise; a wide array of potential modifiers of the relationship between noise and health indicators have been identified. These effect modifiers may be viewed, not as confounders, but as intervening factors in the causal chain either reinforcing or diminishing the original stress response. Complex interactive effects between environmental, social/cultural and personal attributes and the individual's direct and indirect response to noise seem to account for many of the weak associations observed in population-based studies. The manner in which these interactions are conceptualized will direct future studies. The challenge of future research is to tease out those factors which are 'necessary' in the causal chain and which offer evidence that noise exposure precedes the given physiological changes.

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