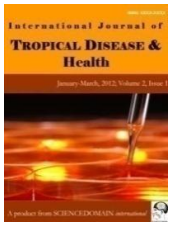

International Journal of TROPICAL DISEASE & Health
6(2): 35-43, 2015, Article no.IJTDH.2015.036
ISSN: 2278–1005

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Impact of Noise Pollution on Human Cardiovascular System

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Authors' contributions

This work was carried out in collaboration between both authors. Author EOA designed and planned the review. Author VUN carried out literature searches. Both authors prepared and approved the final manuscript.

Article Information

DOI: 10.9734/IJTDH/2015/13791
Editor(s):
(1) Thomas Britt, Department of Health Studies, College of Health Sciences, Chicago State University, USA.
(2) Giuseppe Murdaca, Clinical Immunology Unit, Department of Internal Medicine, University of Genoa, Italy.
(3) Shankar Srinivasan, Department of Health Informatics, University of Medicine & Dentistry of New Jersey, USA.
Reviewers:
(1) Anonymous, Italy.
(2) Anonymous, Serbia.
(3) Anonymous, India.
(4) Anonymous, Malaysia.
Complete Peer review History: http://www.sciencedomain.org/review-history.php?iid=852&id=19&aid=7557

Review Article

Received 3rd September 2014
Accepted 9th December 2014
Published 31st December 2014

ABSTRACT

Noise pollution is one of the man-made environmental hazards that is given the least attention. By World Health Organization's (WHO) definition, noise pollution is unwanted or excessive sound that can have deleterious effects on human health and environmental quality. Industrial facilities, entertainment joints, highway, railway, airplane traffic, construction activities and some indoor activities are major sources of noise. Prolong or frequent exposure to excessively loud noise can cause degeneration of the spiral organ resulting in high frequency deafness. Despite the awareness of noise impact on auditory function, people are still engaged in activities that generate loud noise. In some developing countries where electrical power supply is not reliable, the use of generators has contributed immensely to environmental noise. Studies have reported that noise increases the prevalence of hypertension which is one of the risk factors for cardiovascular disorders. The

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increasing number of hypertensive individuals in developing countries might be owed to incessant noise. If the governments of these countries do not take necessary measures to combat noise pollution, their countries might be populated with cardiovascular disease individuals.

Keywords: Blood pressure; cardiovascular system; hypertension; noise; pollution.

1. INTRODUCTION

Noise pollution is one of the environmental nuisances created by urbanization. Noise is gradually becoming ubiquitous, yet an overlooked form of pollution. It may not appear to be as harmful as other forms of pollution, but it is a problem that affects human health and well-being as well as environmental quality [1]. By definition, noise pollution is loud unwanted, undesired or excessive sound that can have deleterious effects on human health and environmental quality [2]. The effects of noise pollution are slow and subtle, it might be immediate in terms of annoyance, but its effect becomes deleterious after continuous exposure to harmful sounds. Unlike air and water pollution, it's contaminants are not physical particles, but waves that interfere with naturally occurring waves of a similar type in the same environment [3].

Sound waves are vibrations of air molecules carried from a noise source to the ear. Sound is characteristically described in terms of the loudness (amplitude) and the pitch (frequency) of the wave. Loudness, which is also called sound pressure level (SPL) is measured in decibels (dB) and decreases with distance from the source. The normal human ear can detect sounds that ranges between 0 dB (hearing threshold) and about 140 dB (pain threshold) [4]. The hazardous effects of noise depend on its intensity, duration, and frequency (high or low). High and low pitch is more damaging than middle frequencies, and white noise covering the entire frequency spectrum is less harmful than noise of a specific pitch [5]. Sound intensity is the amount of energy flowing per unit time through a unit area that is perpendicular to the direction in which the sound waves are travelling and it is proportional to the square of the sound pressure level (SPL). Unlike loudness, sound intensity is objective and can be measured by auditory equipment independent of observer's hearing. Frequency of a sound wave is expressed in cycles per second (cps), but hertz (Hz) is frequently used. The human eardrum is a very sensitive organ with a large dynamic range, being able to detect sounds at frequencies as low as 20 Hz (a very low pitch) up to about 20,000 Hz (a very high pitch). The intensity of one sound can be compared to that of another of the same frequency by taking the ratio of their powers. Precise measurement and scientific description of sound levels differ from most subjective human perceptions and opinions about sound, subjective human responses to noise depend on both pitch and loudness. People with normal hearing generally perceive high-frequency sounds to be louder than low-frequency sounds of the same amplitude. For this reason electronic sound-level meters used to measure noise levels take into account the variations of perceived loudness with pitch [6].

The sources of artificial noise pollution are from industrial machinery and processes, heavy automobile traffic, train, airplane, electrical appliances, public address systems, and generators used for both commercial and residential purposes. Construction activities like mining, construction of bridges, dams, buildings, stations, roads, flyovers and noise from social events make the environment unfriendly and perhaps uninhabitable [2]. Noise generated by household equipment like television, music system, blender, grinder, pressure cooker, vacuum cleaners, washing machine, dryer, cooler, air conditioners contribute minimal amount of noise in the neighborhood. The use of generators in every household in a country like Nigeria because of erratic power supply creates seemingly unbearable noise.

Noise pollution adversely affects the lives of people. Noise Induced Hearing Loss (NIHL) is the most common and often discussed health effect of noise, but studies have revealed that exposure to continuous or high levels of noise can cause several adverse health effects. It can damage psychological health; excessive noise level has been linked to the occurrence of aggressive behavior, constant stress, and fatigue. Loud noise hampers sleeping pattern resulting in fatigue, reduction in performance and may lead to sleep disorders. These in turn can cause more severe and chronic health issues later in life. High level of noise has been reported to affect the cardiovascular system. It has been

linked to high blood pressure, constriction of blood vessels and coronary artery disease [7-11]. This review therefore discusses the impact of noise on cardiovascular system.

2. NOISE POLLUTION AND CARDIOVASCULAR SYSTEM

The ability of the body to perform its normal activities is sustained by a mechanism that supplies the necessary metabolic substrates and removes by-products of metabolism. This mechanism is driven by the cardiovascular system. The contraction of the heart pumps blood and generates pressure that drives the blood through the network of blood vessels transporting substances (respiratory gases, nutrients, etc) throughout the body tissues [12]. The flow of blood through these vessels is dependent upon pressure difference which is the driving force that permits the movement of blood and resistance of the vessels which is an impediment to blood flow [13,14].

The cardiovascular system is control by several mechanisms; the autonomic nervous system, reflex mechanisms, renal-body fluid system, renin- angiotensin system and hormones. The workload on the heart and its output (cardiac output) depends on the preload which is determined by the amount of blood returned to heart through the venous system (venous return) and afterload (peripheral resistance), which is basically influenced by vascular resistance is the pressure in aorta in which the left ventricle must pump against [15]. The autonomic nervous system and reflex mechanisms which act through autonomic nervous system act directly on heart to alter the heart rate and myocardial contractility as well as on vessel to alter their resistance. Other control mechanisms act either by altering venous return and/or vascular resistance. An increase in venous return increases the cardiac output whereas high peripheral resistance raises the pressure in the aorta, causing the pressure gradient between the aorta and left ventricle to fall and consequently cardiac output decreases [15]. The effectiveness of the heart pumping action is affected by the arterial pressure; the heart can be hypo effective, if arterial blood pressure is elevated, high arterial pressure causes excessive workload on the heart and this may lead to heart failure, coronary heart disease or even death as a result of heart attack. Furthermore, high arterial pressure causes multiple hemorrhage in the kidney, consequently destroying the kidney tissues. The kidney plays an important role in normal functioning of the cardiovascular system [16].

Studies have reported that exposure to excessively loud noise increases blood pressure. The effects of industrial noise on the cardiovascular system were assessed in workers of lock factories, which were exposed to industrial noise levels exceeding 80 dB. There was significant increase in systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure, pulse pressure and heart rate in the workers compared to people who never lived or worked in a noisy environment [17]. A meta-analysis study evaluated the association between the modifications of the cardiovascular system and chronic exposure to noise in occupationally exposed subjects in articles published from 1950 to May 2008. A total of 18,658 workers were divided into three groups according to the level of noise exposure; High Exposure (HE), Intermediate Exposure (IE), Low Exposure (LE). The results showed a statistically significant increase in SBP and DBP in HE workers compared to LE and IE workers, while the heart rate was significantly higher in HE participants compared to LE participants. The prevalence of both hypertension and ECG abnormalities was significantly higher in HE workers compared to LE and IE workers [18].

Road traffic is the dominant source of community noise exposure and the assessment of the association between exposure to residential road traffic noise and hypertension in urban metropolis found out that the odds ratio (OR) for hypertension adjusted for age, smoking, occupational status and house type was 1.38 per 5 dB(A) increase in noise exposure. The association was stronger among those who had lived at that particular area for more than 10 years. Analyses of categorical exposure variables revealed an exposure–response relationship and the strongest association between exposure to traffic noise and hypertension was found among those who did not have triple-glazed windows, living in an old house and having the bedroom window facing the street [19]. A survey evaluated the prevalence of stress-related diseases like hypertension in relation to road traffic noise exposure in 1718 participants. The period of prevalence of hypertension increased steadily with the road traffic noise in the range of 55 to 70 dB during the daytime and 50 to 65 dB during the nighttime. The relative risks were high in those exposed to noise level exceeding 65 dB in

daytime and exceeding 55 dB in night time with regards to staying in the living room during the day and the bedroom during the night. When subjects were analyzed separately, for those who slept nearly every night with an open bedroom window, the relative risk was considerably higher [20]. However, a population-based cohort study enrolled 57,053 participants aged 50 – 64 years between 1993 and 1997 and conducted a follow-up survey of all eligible cohort participants between 2000 and 2002. The systolic and diastolic blood pressure was measured at enrolment and in the follow-up survey, while information on hypertension was assessed by questionnaire. It was observed that long-term exposure to road traffic noise was weakly associated with a higher systolic blood pressure and was not linked with diastolic blood pressure or hypertension [21]. Van Kempen and Babisch [22], carried out a meta-analysis in order to derive a quantitative exposure-response relationship between the exposure to road traffic noise and the prevalence of hypertension, road traffic noise was positively and significantly associated with hypertension, the data aggregation revealed an odds ratio (OR) of 1.034 per 5 dB(A) increase of the 16 h average road traffic noise level. The differences observed in studies considered were due to the age and sex of the population under study, the way exposure was ascertained, and the noise reference level used. Also, the way noise was treated in the statistical model and the minimum years of residence of the population under study gave an explanation regarding the observed differences. Therefore, no definite conclusions can be drawn about the threshold value for the relationship between road traffic noise and the prevalence of hypertension.

Aircraft noise has a significant association with the incidence of hypertension. The study of the effect of short-term changes of transportation or indoor noise levels on blood pressure (BP) and heart rate (HR) during night-time sleep in 140 subjects living near four major airports reported that the systolic blood pressure increased by 6.2 mmHg and diastolic blood pressure by 7.4 mmHg over 15 min intervals in which an aircraft event occurred [23]. A cohort study of 2754 men in 4 metropolises around the airport was followed between 1992 – 1994 and 2002 – 2004. Residential aircraft noise exposure was assessed by geographical information systems technique among those living near the airport. Incident cases of hypertension were identified by physical examinations, including blood pressure measurements, and questionnaires in which subjects reported treatment or diagnosis of hypertension and information on cardiovascular risk factors. The analyses were restricted to 2027 subjects who completed the follow-up examination, were not treated for hypertension, and had a blood pressure below 140/90 mm Hg at enrollment. For subjects exposed to noise levels above 50 dB(A) the relative risk for hypertension was 1.19. Maximum aircraft noise levels presented similar results, with a relative risk of 1.20 for those exposed to > 70 dB(A). Stronger associations were observed among older subjects, those with a normal glucose tolerance, nonsmokers, and subjects not annoyed by noise from other sources [24]. A total of 4721 subjects aged 35 – 56 years at baseline, were followed for 8 – 10 years. Their Blood pressure was measured at baseline and at the end of follow-up. Additional information regarding diagnosis and treatment of hypertension as well as various lifestyle factors was provided by questionnaires. In the overall population, no increased risk for hypertension was found among subjects exposed to aircraft noise ≥ 50 dB(A). When restricting the cohort to those not using tobacco at the blood pressure measurements, a significant risk increase per 5 dB(A) of aircraft noise exposure was found in men, but not in women. In both sexes combined, an increased risk of hypertension related to aircraft noise exposure was indicated primarily among those reporting annoyance to aircraft noise. The results suggested an increased risk of hypertension following long-term aircraft noise exposure in men, and that subjects annoyed by aircraft noise may be particularly sensitive to noise related hypertension [25]. Jarup and co-worker [26], assessed the relationship between noise from aircraft or road traffic near airports and the risk of hypertension in 4,861 persons who had lived at least 5 years near any of six major airports. They measured blood pressure and collected data on health, socioeconomic, and lifestyle factors, including diet and physical activity by questionnaire at home visits and assessed noise exposure using detailed models with a resolution of 1 and a spatial resolution of 250 × 250 m for aircraft and 10 × 10 m for road traffic noise. They found significant exposure–response relationships between night-time aircraft as well as average daily road traffic noise exposure and risk of hypertension after adjustment for major confounders. For night-time aircraft noise, a 10-dB increase in exposure was associated with an odds ratio of 1.14. The exposure–response relationships were similar for road traffic noise

and stronger for men with an odd ratio of 1.54 in the highest exposure category. The results indicated excess risks of hypertension related to long-term noise exposure, primarily for night-time aircraft noise and daily average road traffic noise. In consistence, a study of population comprising two random samples of subjects aged 19 – 80 years, one including 266 residents in airport vicinity and another comprising 2693 inhabitants in other parts of the County. The subjects were classified according to the time weighted equal energy and maximum aircraft noise levels at their residence. A questionnaire provided information on individual characteristics including history of hypertension. The prevalence of hypertension was found to be higher among subjects exposed to time weighted energy averaged aircraft noise levels of at least 55 dBA, or maximum levels above 72 dBA occurring at least three times during the average 24 hour period in 1 year. The risk of hypertension from exposure to aircraft noise seemed greater among those not reporting hearing disabilities as a result of the aircraft noise, while the other subjects who reported hearing disability prior to blood pressure measurement showed no signs of hypertension [27].

The effect of noise pollution is not restricted to increased blood pressure. Studies have linked noise to other cardiovascular disorders. The association of aircraft noise with risk of stroke, coronary heart disease, and cardiovascular disease was investigated in about 3.6 million residents living near Heathrow airport. Hospital admissions showed statistically significant linear trends of increased risk with higher levels of both daytime (average A weighted equivalent noise 7 am to 11 pm, $L_{Aeq,16h}$) and night time (11 pm to 7 am, L_{night}) aircraft noise. When areas experiencing the highest levels of daytime aircraft noise were compared with those experiencing the lowest levels (>63 dB *v* ≤51 dB), the relative risk of hospital admissions for stroke was 1.24 (95% confidence interval 1.08 to 1.43), for coronary heart disease was 1.21 (1.12 to 1.31), and for cardiovascular disease was 1.14 (1.08 to 1.20) adjusted for age, sex, ethnicity, deprivation, and a smoking proxy using a Poisson regression model including a random effect term to account for residual heterogeneity. The study concluded that high levels of aircraft noise were associated with increased risks of stroke, coronary heart disease, and cardiovascular disease for both hospital admissions and mortality in areas near Heathrow airport in London [28]. A hospital-based case-control study associated noise to risk of myocardial Infarction. The study was done in 4114 patients 20 to 69 years of age, a clear dose-response relationship of an increasing risk of myocardial infarction with increasing traffic noise levels was found. The increase in risk started at an average sound level of 60 dB(A) and male subjects that lived on streets with average sound levels of more than 70 dB(A) during the day showed a relative risk of myocardial infarction of odd ratio of 1.27 compared to those who lived on streets with less equal sound levels of 60 dB(A). In the sub-sample subjects who had been living for at least 10 years at the present address, a significant odds ratio of 1.81 was found for the same comparison [29]. In consistence, analysis of Swiss National Cohort of 4.6 million persons older than 30 years who were followed from near the end of 2000 through December 2005, revealed that people exposed to high levels of noise from aircraft were at increased risk of dying from myocardial infarction. The association was strongest in those who had lived at the same highly exposed location for at least 15 years. The report concluded that aircraft noise was associated with mortality from myocardial infarction, with a dose-response relationship for level and duration of exposure [30]. A population-based case-control study on myocardial infarction conducted by Selander and colleague [31], between 1992 and 1994 revealed that long-term exposure to road traffic noise increased the risk of developing myocardial infarction with a positive exposure-response trend.

Noise has been linked to high blood cholesterol. The study in 180 workers employed in a metal industry exposed to industrial noise levels exceeding 80dB, and to communal noise levels exceeding 70dB, and a control group consisted 90 workers that had never worked or lived in a noisy environment showed a significant increase in plasma low density lipoprotein cholesterol and triglycerides in the noise exposed group than in the control group. High blood cholesterol is one of the major factors responsible for atherosclerosis which can lead to coronary heart disease, degenerative changes in the myocardium and atherosclerotic changes on arterial blood vessels of low extremities [32].

3. POSSIBLE MECHANISM THROUGH WHICH NOISE AFFECTS CARDIOVASCULAR SYSTEM

3.1 Stress Response

Noise may not be connected to danger, but the body responds to it as a threatening signal, thus, triggering the body's stress response. This generates a cascade of actions that stimulate the release of stress hormones (adrenaline, noradrenaline and cortisol) which consequently initiate several reactions in the body. Sustained effect of chronic noise on stress hormones may be the potential mechanism by which environmental noise is connected to the occurrence of cardiovascular disorders.

The auditory system is always active, even during sleep. The excitations caused by excessively loud noise signals are acknowledged by the brain as stress signals and are sent to the amygdala. The amygdala interprets the sounds and perceives it as danger and immediately sends a distress signal to the hypothalamus [33]. The hypothalamus communicates with the autonomic nervous system, which controls the cardiovascular system; blood pressure, heart rate, and the dilation or constriction of blood vessels. The sympathetic component of the autonomic nervous system triggers the fight-or-flight responses and release epinephrine and norepinephrine from its nerve ending. The released epinephrine and norepinephrine cause acceleration of the heart rate increase in the force of myocardial contraction and constriction of arteries and in addition epinephrine triggers the release of glucose and fatty acid from their storage sites which are used by the body as fuel. Aside from the hypothalamic activation of the sympathetic nervous system, the posterior hypothalamus also acts through the sympathetic preganglionic neuron linked to the adrenal medulla to stimulate the adrenal medulla to secrete adrenaline (epinephrine) and noradrenaline (norepinephrine). These reinforce the efforts of the sympathetic drive [34].

As the first surge of epinephrine diminishes, the hypothalamus activates the second component of the stress response system; the hypothalamic-pituitary-adrenal-axis (HPA-axis). The HPA-axis releases corticotropin-releasing hormone (CRH) through the hypothalamus, which travels to the pituitary gland and triggers the release of adrenocorticotropic hormone (ACTH). This stimulates the zona fasciculata of the adrenal cortex to release cortisol. Cortisol helps to generate glucose through the degradation of protein in a process called gluconeogenesis in the liver and mobilization of fat from adipose tissue. This helps to replenish the body's energy stores that are depleted during the stress response [35].

The persistent activation of the stress response systems has adverse effect on the body. Recurrent epinephrine surges can damage blood vessels and arteries, increase blood pressure and raising risk of heart attacks or stroke [36]. The high level of cortisol in the blood increases the amount of cholesterol in the blood, thereby adding to associated artery plague buildup which results in hypertension and coronary heart disease [37]. In line with this speculation, the study by Cavatorta et al. [38], reported that exposure to high intensity industry noise raised levels of noradrenaline and adrenaline and another study reported that catecholamine secretion decreased when workers wore hearing protection against noise [39]. The nocturnal excretion of catecholamines in urine was studied in 30 – 45 years old women whose bedrooms and/or living rooms were facing streets of varying traffic volume, showed significant associations between traffic volume and noradrenaline concentrations in urine with regards to the exposure of the bedroom (not the living room), indicating a higher chronic physiological arousal in noise-exposed subjects as compared to the less exposed. Subjective measures of disturbance due to traffic noise were positively correlated with the noradrenaline level in subjects where closing the window could not reduce the perceived disturbance [40]. The measurement of cortisol concentrations in the saliva and urine before, during and after an arithmetic calculation task under 90 dB(A) white noise and quiet conditions showed that salivary cortisol level was significantly higher than the pre-task level during the task with noise, but not under quiet conditions [41]. Tafalla and Evans [42], also reported same in male college students exposed to noise while calculating the Norinder arithmetic. Twelve male test subjects were equipped with probes for multiple sampling of venous blood and exposed to the noise of a military low altitude flight for about 20 seconds and this was reproduced via loudspeakers inside an audiometric cabin. On the first day of the experiment, the maximum level was 105 dB(A) and on the second day 125 dB(A). Blood samples were taken before the noise exposure and 1, 8, 16 and 32 minutes thereafter. Before

the experiment, the bladder was emptied and urine collected after one hour. The cortisol concentration was significantly increased 8 minutes after the 125 dB(A) noise exposure when compared with the 105 dB exposure and reached a maximum at 16 minutes after exposure. The cortisol difference was found to be significant 32 minutes after exposure. Also, the excretion of free cortisol was significantly increased after 125 dB(A) as compared with 105 dB(A) [43].

4. CONCLUSION

The studies reviewed showed a strong link between noise pollution and high cardiovascular risk. Aside from the strong association of noise pollution with high blood pressure which itself is one of the risk factors for cardiovascular disorders, studies have linked noise pollution to the incidence of myocardial infarction and stroke. Despite the evidence about the adverse health effects of noise, most countries are indifferent about noise pollution. Precautions should be taken against the ill effects of noise; the government should enforce federal standards for highway and aircraft noise, proper planning for buildings, urban planning, and transportation management. Furthermore, residential houses should not be built in areas where there are 24 hour average noise levels exceeding 60 dB. The design as well as technology of machines/equipment should be improved to emit low noise. The residents of noisy cities can reduce noise considerably by insulating the buildings against noise and installing sound proof windows (dual-paned windows) and workers in noisy industries can wear hearing protection against noise. Public enlightenment is also necessary so that individuals become aware of the ill effect of noise on cardiovascular system and thus protect themselves against noise pollution by reducing noise from home appliances and generators as well as outdoor activities that engender loud noise.

Exposure to noise disturbs sleep proportional to the amount of noise experienced, this effect of noise on sleep can in turn cause more severe and chronic health issues and in addition, noise activates the body's stress response which consequently initiates several reactions in the body. Prolonged effect of noise on stress hormones might be the possible mechanism by which environmental noise adversely affects the cardiovascular system. The ill effects of noise pollution may be alleviated by undertaking measures to minimize unnecessary noise. Where that is inevitable, the use of suitable protective measures should be employed to reverse the physiological changes associated with chronic stress.

CONSENT

Not applicable.

ETHICAL APPROVAL

Not applicable.

ACKNOWLEDGEMENTS

Authors hereby salute the efforts of the secretarial staff of the Department of Physiology, University of Uyo, Akwa Ibom State, Nigeria.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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Peer-review history:
The peer review history for this paper can be accessed here:
http://www.sciencedomain.org/review-history.php?iid=852&id=19&aid=7557