Research Journal of Medical Sciences 3 (3): 115-122, 2009
ISSN: 1815-9346

Health Hazards of Noise: A Review Article

A.O. Olaosun, O. Ogundiran and J.E. Tobih
Department of Otorhinolaryngology,
Ladoke Akintola University of Technology, Teaching Hospital,
Osogbo, Osun State, Nigeria

Abstract: This is a review of definition, sources and health hazards of noise. Noise is defined operationally as audible acoustic energy that adversely affects, or may affect the physiological and psychological wellbeing of people. Sources of environmental noise include machinery, mechanical devices in residential areas, road traffic, rail traffic, aircraft operations, building construction and earth works, street services such as garbage disposal, military activities, building services and noise from leisure activities. Noise is more than just an annoyance. It is a significant hazard to public health. The most clearly identifiable hazard is hearing loss. Many studies have also clearly identified noise as an important cause of physical and psychological stress which, in turn, has been directly linked with many health problems. In this way, noise is associated with many diseases, including heart disease, high blood pressure, headaches, fatigue and irritability. Noise has also been shown to disturb sleep, affect optimum physical and mental performance and interfere with children's learning and with normal development of the unborn child. It is also reported to accelerate and intensify the development of latent mental disorders, predispose to disability and even loss of life. It is concluded that there is an urgent need for concerted effort to control noise.

Key words: Noise, acoustic energy, sources, health hazards, public health, noise control

INTRODUCTION

Calling noise a nuisance is like calling smog an inconvenience. Noise must be considered a hazard to the health of people everywhere. So said Dr. William H. Stewart, former U.S. Surgeon General; words not only true but which also, deserve serious consideration.

Noise is probably the most widespread nuisance. However, more than that it constitutes a real hazard to health. It can produce serious physical and psychological stress and though we seem to adjust by ignoring noise, the ear never closes and the body responds. Although, annoyance is the most common symptom of irritability and has been made the basis of many noise abatement programs, there are more subtle and more serious health hazards caused by noise that have been given much less attention. Following is a review of definition, sources and health hazards of noise.

DEFINITION OF NOISE

Noise is often arbitrarily defined as an unpleasant or undesired sound (Churchill, 1989) a subjective definition. Physically, sound is produced by mechanical disturbance propagated as a wave motion in air or other media and physical sound evokes physiological responses in the ear and auditory pathways (Berglund and Lindvall, 1995). Psychologically, sound is a sensory perception originating as a mental event evoked by physiological processes in the auditory and other parts of the brain. Thus, it is merely through the perceptual analysis of sounds that the complex pattern of sound waves may be classified and labeled noise, music, speech, etc.

From a physical point of view, therefore, there is no difference between the concepts of sound and noise, although it is an important distinction for the human listener (Berglund and Lindvall, 1995). Thus, sound can have a range of different physical characteristics, but it only becomes noise when it has an undesirable physiological or psychological effect on people.

Experts therefore, agree that it is not possible to define noise exclusively on the basis of physical parameters of sound. Rather, noise is defined operationally as audible acoustic energy that adversely affects, or may affect the physiological and psychological wellbeing of people (Berglund and Lindvall, 1995).

Corresponding Author: A.O. Olaosun, Department of Otorhinolaryngology, Ladoke Akintola University of Technology, Teaching Hospital, Osogbo, Osun State, Nigeria

MEASUREMENT OF NOISE

In measuring noise, the temporal pattern and characteristics of sound are taken into consideration. The temporal patterns of environmental noise are typically described as continuous, fluctuating, intermittent or impulsive. Continuous noise remains relatively constant, fluctuating noise rises and falls in level over time and intermittent sounds are interrupted for varying time periods. Impulsive or impact noises are caused by explosive or metal-on-metal mechanical events and have rapidly changing pressure characteristics consisting of intense, short-lasting (milliseconds) wave forms, followed by much smaller reverberations and echoes that can last many seconds (Lonsbury-Martin and Martin, 1993).

Two basic characteristics of sound, frequency and amplitude are related to how loud and annoying a sound is (Lonsbury-Martin and Martin, 1993). Frequency (or Pitch) describes the rate of vibration-how fast the object is moving back and forth. The more rapid the movement, the higher the frequency of the sound pressure waves created. The unit for measuring frequency is the Hertz (Hz), equivalent to one wave per second passing a given point.

The normal hearing range for humans is 20-20,000 Hz though there is individual variation in the ability to perceive very high or very low frequency sounds. The main source of irritation related to community noise is low frequency noise (e.g., traffic noise, bass sounds from amplified music) because they are more penetrating and more difficult to control by shielding than high frequency sounds (Nadakavukaren, 2000).

Amplitude, also called the Sound Pressure Level (SPL) is the intensity of the sound-how much energy is behind the sound wave, a measure of the 'amount' of the sound. Sound waves of the same frequency can be heard as very loud or very soft depending on the force with which, they strike the ear. Technically, amplitude is the maximum displacement of a sound wave from its resting or equilibrium position but is perceived psychologically as loudness. It is conventionally measured by a sound pressure level meter in decibels (dB) using a weighting formula called the A-scale (dBA). This A-scale measure of sound level essentially mimics the threshold-sensitivity curve for the human ear, so that low and high-frequency components are given less emphasis as auditory hazards. This is so done because a listener's perception of sound as loud does not depend entirely on its amplitude, being affected to some extent by its frequency as well.

Though, hearing ability is 20-20, 000 Hz, not all of these frequencies sound equally loud to the human ear, maximum sensitivity occurring between 1000-5000 Hz while, sounds at the very low and very high ends of the full audible range seem much fainter. Thus, for example an extremely low-pitched sound must have amplitude many times greater than a sound of medium pitch in order for both to be perceived as equally loud.

Other less used scales are the B- and C-scales which, simulate equal-loudness contours for human ears at medium and high SPLs, respectively and the linear scale, which, enables the unmodified signal to be analyzed.

The decibel scale ranges from 0, which is regarded as the threshold of hearing for normal healthy ears, to 194, regarded as the threshold for pure tones. Because any increase of 10 decibels results in a doubling of perceived loudness, even a small rise in decibel values can make a significant (Nadakavukaren, 2000) difference in noise intensity.

SOURCES OF ENVIRONMENTAL NOISE

These are numerous (Berglund and Lindvall, 1995) and include:

- Machinery, for example, in mechanized industry. It is estimated that 15-20% or more of the working population is affected by sound pressure levels of 75-85 dBA
- In residential areas, noise may stem from mechanical devices (e.g., generators), powered domestic appliances (vacuum cleaners, washing machines, lawn mowers, etc.), systems for music reproduction, TV sets, hobby activities, parties and other social activities
- Road traffic, especially in areas where the traffic movements involve a change in engine speed and power, such as at traffic lights, hills and intersecting roads
- Rail traffic, railway noise depends primarily on the speed of the train but variations are present depending upon the type of engine, wagons and rails
- Aircraft operations, more research has been devoted to aircraft noise than to any other environmental noise problem (Berglund *et al*., 1990)
- Building construction and earth works, through a variety of sounds present from cranes, cement mixers, welding, hammering, boring and other work processes
- Street services such as garbage disposal and street cleaning
- Military activities: an important noise source such as noise produced by heavy vehicles (tanks), helicopters and small and large fire arms

- Building service noise can affect people both inside and outside the building. Ventilation and air conditioning plants and ducts, heat pumps, plumbing systems and lifts, for example, can compromise the internal acoustic environment
- Noise from leisure activities: for example, shooting activities, music played back in headphones and impulse noise from toys and fireworks
- Discotheques and music concerts may exceed hearing damage risk criteria for the musicians, employees and the audience

The extent of exposure to noise is large. There are no local figures for Nigeria but in the EU countries about 40% of the population is exposed to road traffic noise exceeding 55 dB in the daytime and 20% are exposed to levels exceeding 65 dB. More than 30% are exposed at night to noise levels exceeding 55 dB, which are disturbing to sleep. Annoyance to community noise is widespread: in some EU-countries 20-25% is being annoyed by road traffic, 2-15% by aircraft and 2-4% by railway noise (Lambert and Vallet, 1994).

HEALTH HAZARDS OF NOISE

Hearing loss (noise-induced hearing loss): Of the many health hazards related to noise, hearing loss is the most clearly observable and measurable. Noise-induced hearing loss is of a sensorineural type involving injury to the inner ear. In the normal auditory process, sound vibrations in the air travel through the ear canal and cause the eardrum to vibrate. The vibrations are then transmitted by the bones of the middle ear to the sensory organ of the inner ear (cochlea). Here, they are transduced by hair cells into nerve impulses and transmitted to the brain, where they are perceived as sound (e.g., noise).

Blasts and other intense or explosive sounds can rupture the eardrum or cause immediate damage to the structures of the middle and inner ear, while, hearing loss due to prolonged noise exposure is generally associated with destruction of the hair cells of the inner ear. The severity of loss depends on both the location and the extent of damage in the organ of Corti, in the inner ear, which, in turn, depend on the intensity and frequency of the sound exposure as the number of hair cells damaged or destroyed increases with increasing intensity and duration of noise and, in general, progressive loss of hair cells is accompanied by progressive loss of hearing (Miller *et al*., 1971).

The mechanisms involved in the destruction of the Corti organ are not completely clear, although numerous experiments have been performed with animals and several explanations been proposed. For example, mechanical stresses could destroy cells (Hamernik *et al*., 1984). Noise may alter cochlear blood flow that in turn may alter the metabolic status of the cells and the local temperature leading to damaged proteins (Ward, 1991).

Occupational hearing loss and dangerous levels: Virtually every study of Occupational hearing loss has revealed that workers exposed to intense noise daily, for several years, showed noise-induced hearing loss. Considerable hearing loss was rare at lower frequencies but frequent at higher frequencies. A clear relationship was generally seen between increasing incidence of hearing loss and increasing noise level (Bauer *et al*., 1991).

Also, it has been found that the proportion of workers with noise-induced deafness (25 dB average loss at 0.5, 1 and 2 kHz) was as high as 60% in the metal industry (with sound pressure levels equal to and above 95 dBA (Rey, 1974). Mean hearing levels of exposed workers compared with those of a control group for several noise intensities and several durations of exposure showed that sound pressure levels between 85 and 88 dBA (or more) could be harmful to the ear (Cohen *et al*., 1970). A definite risk of hearing loss is found associated with prolonged exposure to sound levels between 85 and 90 dBA, or more (Martin *et al*., 1975).

Noise-induced temporary threshold shift: A person exposed to harmful levels of noise may experience a measurable loss in hearing but may recover some time after returning to a quiet environment. This can be measured as a reversible or temporary shift in audiometric thresholds and is called Noise-Induced Temporary Threshold Shift (NITTS). The extent varies considerably between individuals. Recovery can take hours, days, or weeks after exposure and depends on the severity of the hearing shift, individual susceptibility and the type of exposure. If recovery is not complete before the next noise exposure, there is a possibility that some of the loss will become permanent (Bohne, 1976).

Noise-induced permanent threshold shift: Although, noise-induced hearing loss occurs gradually, usually over a period of years, an abrupt process cannot be ruled out. The rate and extent of loss depends on the severity and duration of exposure, though individual susceptibility also seems to have a considerable effect on the rate of progression. Noise-induced losses are similar to losses due to aging (presbyacusis) and the two types of losses are difficult to distinguish (Corso, 1992).

Several studies have confirmed that loud levels of music can produce considerable temporary threshold shift and even permanent threshold shift though some suggest that hearing loss in musicians are not as large as suspected. This is attributed in part to the frequent pauses, allowing some recovery that characterize this kind of exposure (Royster *et al*., 1991).

Effects of impulsive noise: Studies of hearing loss due to impulsive noise e.g., gunfire prove that danger exists and ear protection should be worn when impulsive noises exceed a sound pressure level of 140 dB for >5 ms. Higher maximum levels may be tolerable for durations of <5 ms. Sound pressure levels in excess of 165 dB even for short durations, are likely to cause acute cochlear damage (Acton, 1967). Exposures to impulsive noise may arise even from noisy toys and in use of fireworks. To avoid hearing deficits, performers and audience should not be exposed to >140 dB peaks of impulsive sounds (Axelsson and Jerson, 1985).

Combined effects: The adverse effects of noise on hearing may be enhanced by a variety of ototoxic drugs (i.e., drugs that are toxic to the ears) and environmental chemicals. This means that noise exposures, which would otherwise not disrupt hearing may become damaging due to the presence of such a co-factor.

The aminoglycoside antibiotics, cis-platin, loop diuretics and salicylate have significant ototoxic potential. The effect ranges from permanent auditory threshold elevation after exposure to aminoglycosides and cis-platin, to temporary impairment with the loop diuretics. Chronic, high-dose aspirin therapy usually produces reversible tinnitus rather than a primary disruption in auditory thresholds. (Collins, 1988). Potentiation of dysfunction and cochlear damage has also been reported in animals co-administered the anti-tumor agent cis-platin and octave band noise at exposure levels of 85 dB continuously for periods of 5 days (Gratton *et al*., 1990).

Ototoxic chemicals include chemical asphyxiants, organic solvents and metals. All of these agents are used in occupational settings and some of the organic solvents are also used within households in glues, stain removers and paints. Some organic solvents, notably toluene, are abused because of their psychopharmacological properties. A variety of chemical asphyxiants alone can disrupt auditory function in laboratory animals including carbon monoxide, cyanide (Konishi and Kelsey, 1968) and hypoxic hypoxia (Nuttall, 1984) under severe conditions. However, evidence shows that exposure to very high carbon monoxide levels can potentiate hearing loss in subjects exposed to noise simultaneously and destroy outer hair cells in the cochlea (Young *et al*., 1987).

It has also been shown that noise-exposed smokers had an excess rate of hearing loss compared to non-smokers. Carbon monoxide is one constituent of cigarette smoke (Prince and Matanoski, 1991).

Other combined effects include the effects of combinations between noise and head injury and/or ear disease. Combined exposure to steady-state and impulse noise showed lower temporary threshold shifts than the same noises when presented alone. However, significant differences were found only at 30 min main exposure (Kundi *et al*., 1984).

The combination of high noise exposure and whole body vibration may lead to a significant aggravation of hearing losses (Manninen, 1993).

Other auditory effects: It has been shown that children in a sound-attenuated school had better auditory discrimination scores than children matched by social class who attended an unattenuated school (Moch-Sibony, 1984). Acute as well chronic tinnitus has also been frequently reported among subjects exposed high levels of aircraft noise (Tarnopolsky *et al*., 1980).

Sensory effects

Pain: The threshold of pain for sound exposures in normal hearing persons is around 110-130 dB and the threshold for physical discomfort-Loudness Discomfort Level (LDL) or Uncomfortable Loudness Level (ULL) is 80-100 dB SPL. There is however, a lowering of the threshold of aural discomfort and pain in abnormal hearing, e.g., inflammation, pain may be caused in the ear by sound pressure levels of about 80-90 dB as well as in many cases of sensorineural hearing disorders, such as Ménière's disease. In addition, discomfort associated with exposure to sudden loud noises, loud music and even raised voices is a common complaint of people who wear hearing aids (Gabrielsson *et al*., 1974).

Other sensory effects include tinnitus, which commonly accompanies hearing loss (Gabrielsson *et al*., 1974). Certain sensorineural disorders and most frequently noise-induced hearing losses, are accompanied by abnormal loudness perception, which is known as loudness recruitment (Hallpike, 1967). Some sounds may be perceived distorted. This is called paracusis. For example, a tone is heard but the pitch of the tone is inappropriate (Hallpike and Hood, 1959).

Interference with communication: It is known that noise interferes with speech discrimination and this results in a great proportion of person disabilities and handicaps such as problems with concentration, fatigue, uncertainty and lack of self-confidence, irritation, misunderstandings,

decreased working capacity, problems in human relations and a number of reactions to stress. The masking effect of noise in speech discrimination is more pronounced in the hearing impaired, the elderly and for children in the process of language acquisition, than in persons with normal hearing (Hygge *et al*., 1993).

Sleep disturbance: Many people experience sleep disturbance due to noise and sleep disturbance is considered to be a major environmental noise effect (Rovekamp, 1983).

Exposure to noise can induce disturbances of sleep in terms of difficulty to fall asleep-sleep latency, the need for using sleeping pills or ear plugs, the time to fall asleep, alterations of sleep pattern or depth and awakenings. These effects are referred to as primary sleep disturbance effects. Other primary physiological effects that can be induced by noise during sleep are vegetative reactions such as increased blood pressure, increased heart rate, increased finger pulse amplitude, vasoconstriction and change in respiration and cardiac arrhythmia, as well as body movements (Rovekamp, 1983).

Secondary effects or aftereffects (that can be measured in the morning or the day after the noise exposure) include reduced perceived sleep quality, increased fatigue, decreased mood or wellbeing and decreased performance. Long-term effects on psychosocial wellbeing have also been related to noise exposure during the night and annoyance during the night influences the total daily annoyance level. Sleep is also a necessary prerequisite for good physiological and mental health (Ohrstrom, 1993).

Psychophysiological effects: Noise affects both mental and physical wellbeing. It has been postulated that noise acts as a general stressor and as such may activate several physiological systems leading to changes such as increases in blood pressure and heart rate and vasoconstriction. The magnitude and duration of these effects are determined in part by individual susceptibility, lifestyle behaviors and environmental conditions. (Rovekamp, 1983).

Many studies have focused on the possibility that noise may be a risk factor for cardiovascular disease (Thompson, 1993). Studies in occupational settings have indicated that workers exposed to high levels of industrial noise for durations of 5-30 years have significantly increased blood pressure compared to workers in control areas (Lang *et al*., 1992).

Similarly, there is a tendency for blood pressure to be higher among persons living in proximity to airports and on streets with higher levels of traffic noise than among control subjects (Elwood *et al*., 1993).

Mental health effects: The evidence relating noise to mental illness is scanty and much of it is based only on clinical impression. However, there is some evidence that points to possible negative effects of noise on mental health, manifested in the presence of medical drug use, psychiatric symptoms and mental hospital admission rates in communities with high levels of community noise. Exposure to high levels of occupational noise has been associated with development of neurosis and irritability and exposure to high levels of environmental noise with mental health (Cohen *et al*., 1986).

Noise is not believed to be a direct cause of mental illness but might accelerate and intensify the development of latent mental disorders (Stansfeld *et al*., 1985).

Performance effects: The effects of noise on human performance are very complex. Acute noise exposure is reported to disrupt tasks that demand attention to multiple cues, tasks, in which high levels of working memory capacity are required and tasks where continuous and detailed attention to frequent signals is required (Smith, 1989).

There are well documented effects, particularly of uncontrollable noise, on human performance that demands sustained effort. Chronic noise exposure impacts reading acquisition in children. This may be related to deficits in auditory discrimination associated with chronic noise exposure in the home or at school (Hygge, 1994).

Effects on residential behavior and annoyance: The effects of noise on social and behavioral variables are often complex, subtle and indirect and though some reactions may be the result of interactions with a number of non-auditory variables, observed social and behavioral effects on residential behavior and annoyance, include overt behavior patterns such as opening windows, using balconies, writing petitions and complaining to authorities; effects on social behavior leading to aggression, unfriendliness, etc. and residential mobility (Miller, 1978).

Noise and the unborn: The fetus is responsive to sounds in the mother's environment. Particularly loud noises have been shown to stimulate the fetus directly, causing changes in heart rate and that late in pregnancy, it can respond to noise with bodily movements such as kicking. It is also not fully protected from its mother's response to stress, whether due to noise or other factors. When, her body reacts to noise, the physical changes she experiences may be transmitted to the fetus, which is capable of responding. This indirect response may

threaten fetal development if it occurs early in pregnancy. The most important period is about 14-60 days after conception.

A Japanese study of over 1,000 births produced evidence of a high proportion of low-weight babies in noisy areas. Also, the difference between the hormone levels of pregnant mothers in noisy versus quiet areas increased as birth approached.

Studies have also shown that stress causes constriction of the uterine blood vessels, which supply nutrients and oxygen to the developing baby. Links between noise and birth defects have been noted in a recent preliminary study on people living near a major airport. The abnormalities suggested included harelips, cleft palates and defects in the spine (United States Environmental Protection Agency, 1978).

Danger to Life: Noise can obscure warning signals, causing accidents to occur. It can interfere with shouts for help, preventing rescue attempts. Inability to hear auditory warning signals or shouts of caution because of noise has also been implicated in many accidents.

A study of medical and accident records of workers in several industries found that a significantly higher number of reported accidents occurred in noisier plant areas.

Thus, it is an unfortunate result of high background noise levels that people cannot respond in life and death situations when they are unable to hear approaching hazards or shouts of alarm (United States Environmental Protection Agency, 1978).

CONCLUSION

Truly, noise must be considered a hazard o the health of people everywhere. From the foregoing, it is clear that noise is more than just an annoyance. It is a significant hazard to public health. It is true that except for the serious problem of hearing loss, there is no human illness known to be directly caused by noise but very many studies have clearly identified noise as an important cause of physical and psychological stress which, in turn, has been directly linked with many health problems. In this way, noise is associated with many diseases, including heart disease, high blood pressure, headaches, fatigue and irritability. Noise has also been shown to disturb sleep, affect optimum physical and mental performance and interfere with children's learning and with normal development of the unborn child. It is also reported to accelerate and intensify the development of latent mental disorders, predispose to disability and even loss of life. There is therefore, an urgent need for concerted effort to get in control this silent but certain foe.

REFERENCES

Acton, W.I., 1967. A review of hearing damage risk criteria. Ann. Occup. Hyg., 10: 143-153.

Axelsson, A. and T. Jerson, 1985. Noisy toys: A possible source of sensorineural hearing loss. Pediatrics, 76: 574-578.

Bauer, P., K. Korpert, M. Neuberger, A. Raber and F. Schwetz, 1991. Risk factors for hearing loss at different frequencies in a population of 47 388 noise-exposed workers. J. Acoust. Soc. Am., 90: 3086-3098.

Berglund, B. and T. Lindvall, 1995. Community noise. Archives of the Center for Sensory Research, Stockholm, Sweden, 2 (1): 3, 14-19.

Berglund, B., T. Lindvall and S. Nordin 1990. Adverse effects of aircraft noise. Environ. Int., 16: 315-338.

Bohne, B., 1976. Mechanisms of Noise Damage in the Inner Ear. In: Henderson, D., R.P. Hamernik, D.S. Dosanjh and J.H. Mills (Eds.). Effect of Noise on Hearing. New York, Raven, pp: 41-67.

Churchill, 1989. Churchill's Illustrated Medical Dictionary, Churchill Livingstone Incorporated, New York, pp: 1284.

Cohen, A., J.R. Anticaglia and H.H. Jones, 1970. Noise-induced hearing loss: Exposures to steady-state noise. Arch. Environ. Health, 20: 614-623.

Cohen, S., G.W. Evans, D. Stokols and D.S. Krantz, Behavior, 1986. Health and Environmental Stress. New York, Plenum Press.

Collins, P.W.P., 1988. Synergistic interactions of gentamicin and pure tones causing cochlear hair cell lossin pigmented guineapigs. Hear. Res., 36: 249-260.

Corso, J.F., 1992. Support for Corso's hearing loss model relating aging and noise exposure. Audiology, 31: 162-167.

Elwood, P.C., H. Ising and W. Babisch, 1993. Traffic Noise and Risk Factors for Cardiovascular Disease. In: Ising, H. and B. Kruppa (Eds.). Noise and Disease. Stuttgart: Gustav Fischer, pp: 128-137.

Gabrielsson, A., B. Johansson, B. Johansson, A.C. Lindblad and L. Persson, 1974. Assessment of comfort and discomfort levels for pure tone. A methodological study. Stockholm: Karolinska Institute, Technical Audiology, Report TA No. 74.

Gratton, M.A., R.J. Salvi, B.A. Kamen and S.S. Saunders, 1990. Interaction of cisplatin and nose on the peripheral auditory system. Heart Res. Newslett., 50: 211-224.

Hallpike, C.S., 1967. The Loudness Recruitment Phenomenon: A Clinical Contribution to the Neurology of Hearing. In: Graham, A.B. (Ed.). Sensorineural Hearing Processes and Disorders, pp: 489-499.

Hallpike, C.S. and J.D. Hood, 1959. Observatrions upon the neurological mechanism of the loudness recruitment phenomenon. Acta Otolaryngol., 50: 472-486.

Hamernik, R.P., G. Turrentine, M. Roberto, R. Salvi and D. Henderson, 1984. Anatomical correlates of impulse noise-induced mechanical damage in the cochlea. Hear. Res., 13: 229-247.

Hygge, S., 1994. Classroom experiments on the effects of aircraft, road traffic, train and verbal noise presented at 66 dBA Leq and of aircraft and road traffic noise presented at 55 dBA Leq, on long-term recall and recognition in children aged 12-14 years. Gävle, Sweden: Royal Institute of Technology, Report to the Swedish Environmental Protection Agency.

Hygge, S., G.W. Evans and M. Bullinger, 1993. The Munich Airport Noise Study: Psychological, Cognitive, Motivational and Quality of Life Effects on Children. In: Vallet, M. (Ed.). Noise as a Public Health Problem. Arcueil Cedex, France: INRETS, 3: 301-308.

Konishi, T. and E. Kelsey, 1968. Effect of cyanide on cochlear potentials. Acta Oto-Laryngologica, 65: 381-399.

Kundi, M., U. Weninger, H.G. Stidl and M. Haider, 1984. Effects of Combined Exposures to Steady-state and Impulse Noise on Inner Ear Functions. In: Manninen, O. (Ed.). Combined Effects of Environmental Factors. Tampere, Finland: Keskupaino Central Printing House, pp: 169-178.

Lambert, J. and M. Vallet, 1994. Study Related to the Peparation of a Communication on a Future EC Noise Policy. Bron Cedex, France: INRETS, LEN report No. 9420.

Lang, T., C. Fouriaud and M.C. Jacquinet-Salord, 1992. Length of occupational noise exposure and blood pressure. Int. Archiv. Occup. Environ. Health, 63: 369-372.

Lonsbury-Martin, B.L. and G.K. Martin, 1993. Auditory Dysfunction from Excessive Sound Stimulation. 2nd Edn. In: Cummings, C.W., J.M. Fredrickson, L.A. Harker, C.J. Krause and D.E. Schuller (Eds.). Otolaryngology-Head and Neck Surgery: Ear and Cranial Base. St Louis, Mosby-Year Book, 4: 2885-2886.

Manninen, O., 1993. Synopsis of Studies on Combined Effects. In: Vallet, M. (Ed.). Noise as a Public Health Problem. Arcueil Cedex, France: INRETS, 3: 511-516.

Martin, R.H., E.S. Gibson and J.N. Lockington, 1975. Occupational hearing loss between 85 and 90 dBA. J. Occup. Med., 17: 13-18.

Miller, J.D., 1978. General Psychological and Sociological Effects of Noise. In: E.C. Carterette and M.P. Friedman (Eds.). Handbook of Perception. Vol. VI: Hearing. New York: Academic Press, pp: 641-676.

Miller, J.D., S.J. Rosthenberg and D.H. Eldredge, 1971. Preliminary observations on the effects of exposure to noise for 7 days on the hearing and inner ear of the chinchilla. J. Acoust. Soc. Am., 50: 1199-1203.

Moch-Sibony, A., 1984. Aspects congitifs des stress de l'environnement (Cognitive aspects of environmental stress). Le Travail Humain, 47: 155-165.

Nadakavukaren, A., 2000. Our Global Environment, A Health Perspective. 5th Edn. Illinois, Waveland Press, pp: 511-512.

Nuttall, A.L., 1984. Dynamic aspects of guinea pig inner hair cell receptor potentials with transient asphyxia. Hear. Res., 16: 1-16.

Ohrstrom, E., 1993. Research on Noise and Sleep since 1988: Present State. In: Vallet, M. (Ed.). Noise as a Public Health Problem. Arcueil Cedex, France: INRETS, 3: 331-338.

Prince, M.M. and G.M. Matanoski, 1991. Problems in Ascertaining the Combined Effects of Exposure. In: Fechter, L. (Ed.). Results of an Occupational Cohort Study of the Joint Effects of Noise and Smoking on Hearing Acuity. Proceedings of the 4th International Conference on the Combined Effects of Environmental Factors. Baltimore: Johns Hopkins University, Center for Environ. Health Sci., pp: 87-91.

Rey, P., 1974, La surdité professionnelle. Sozial Praventivmedizin, 19: 177-183.

Rovekamp, A.J.M., 1983. Physiological Effects of Environmental Noise on Normal and More Sound-sensitive Human Beings. In: Rossi, G. (Ed.). Noise as a Public Health Problem. Milano, Italy: Centro Ricerche e Studi Amplifon, 1: 605-614.

Royster, J.D., L.H. Royster and M.C. Killion, 1991. Sound exposures and hearing threshold of symphony orchestra musicians. J. Acoust. Soc. Am., 89: 2793-2803.

Smith, A.W., 1989. A review of the effects of noise on human performance. Scandinavian J. Psychol., 30: 185-206.

Stansfeld, S.A., C.R. Clark, L.M. Jenkins and A. Tarnopolsky, 1985. Sensitivity to noise in a community sample: I. Measurement of psychiatric disorder and personality. Psychol. Med., 15: 243-254.

Tarnopolsky, A., G. Watkins and D.J. Hand, 1980. Aircraft noise and mental health: I. Prevalence of individual symptoms. Psychol. Med., 10: 683-698.

Thompson, S.J., 1993. Review: Extraaural Health Effects of Chronic Noise Exposure in Human. In: Ising, H. and B. Kruppa (Eds.). Noise and Disease. Stuttgart: Gustav Fischer, pp: 106-117.

United States Environmental Protection Agency, 1978. Office of Noise Abatement and Control: Noise: A Health Problem, Washington DC, pp: 10 -11, 20-21.

Ward, W.D., 1991. Noise-Induced Hearing Damage. In: Paparella, M.M., D.A. Shumrick, J.L. Gluckman and W.L. Meyerhoff (Eds.). Otolaryngology. Vol II: Otology and Neuro-Otology. Philadelphia, PA: Saunders, Ch., 45: 1639-1652.

Young, J.S., M.B. Upchurch, M.J. Kaufman and L.D. Fechter, 1987. Carbon monoxide exposure potentiates high-frequency auditory threshold shifts induced by noise. Hear. Res., 26: 37-43.