EFFECT OF SHORT-TERM EXPOSURE TO BELOW DAMAGE RISK CRITERIA NOISE ON TEMPORAL PROCESSING AND SPEECH PERCEPTION

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This dissertation is submitted as a part fulfilment for the

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(Audiology)

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May-2017

CERTIFICATE

This is to certify that this dissertation entitled "Effect of Short-term Exposure to Below

Damage Risk Criteria Noise on Temporal Processing and Speech Perception" is a

bonafide work submitted as a part for the fulfilment for the degree of Master of Science

(Audiology) of the student Registration Number: 15AUD013. This has been carried out

under the guidance of the faculty of this institute and has not been submitted earlier to

any other University for the award of any other Diploma or Degree.

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This is to certify that this dissertation entitled "Effect of Short-term Exposure to Below

Damage Risk Criteria Noise on Temporal Processing and Speech Perception" is the

result of my own study under the guidance of Dr. Sandeep M., Reader in Audiology,

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been submitted earlier to any other University for the award of any other Diploma or

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Chapter 1

INTRODUCTION

Human beings are exposed to various sounds of different levels in their daily life. Most of these sounds, in spite of being within the audible frequency band are irrelevant to the individual and therefore are categorized as Noise. The effects of noise on the human auditory system has been extensively studied in past, and based on its effects on hearing sensitivity, the permissible noise exposure levels have been clearly delineated. According to Occupational Safety and Health Administration (OSHA,1983), damage risk criterion is 90 dBA for 8 hours with a time-intensity tradeoff of 5dB. Any noise exposure that extends beyond this criterion is likely to cause noise induced hearing loss (NIHL). Similarly, NIOSH (1998) recommends an exposure limit of 85 dBA for 8 hours per day, and uses a 3 dB time-intensity tradeoff. Exposure to prolonged durations of noise is one of the major causes of cochlear hearing loss, especially in adults. NIHL can either be an immediate effect of noise exposure or it can manifest itself after years of exposure (Miller, Watson & Covell, 1963). The prevalence of NIHL has been reported to be 16% worldwide (Nelson, Nelson, Barrientos & Fingerhut, 2005).

The extent of the damage can range from loss of individual sensory cells to disintegration of an entire portion of the organ of Corti and its corresponding afferent nerve fibers depending on the intensity and duration of noise exposure, (Lurie, 1942; Hawkins, Lurie & Davis, 1943; Habermann, 1906). With intense exposures of greater than 140 dB SPL, traumatic changes have been reported by the end of the exposure itself (Hawkins, Lurie & Davis, 1943; Lurie, 1942; Covell, Smith & Eldredge, 1954). On the other hand, in ears exposed continuously to relatively lower levels of noise (e.g. 95-108 dB SPL), damage to the sensory cells may lead that in the supporting

cells and nerve fibres by several hours to several days (Bohne, 1971). To account for damage to the sensory cells following moderate noise exposures, several theories have been proposed. Two of the most accepted theories include metabolic exhaustion of the stimulated cells (Spoendlin, 1962, Lim & Melnick, 1971; Lim, 1976) and changes in blood supply to the cochlea during prolonged stimulation (Spoendlin, 1962; Lawrence, Gonzalez & Hawkins, 1967; Kellerhals, 1972; Lipscomb & Thomas, 1972).

Noise exposure may also interfere with our other day-to-day activities and cause anxiety, restlessness, stress and sleep disturbances (Cohen, Evans, Krantz & Stokols., 1980; Cohen, Krantz, Evans, Stokols & Kelly,1981). During the recent times, with the growing population and the urban development, noise pollution is increasing at an alarming rate, the major noise sources of concern being traffic noise (rail, road and air), construction activities, industrial noises and social gatherings.

Recent studies have also found compromised central auditory processing in individuals exposed to noise, in spite of hearing sensitivity being unaffected. Kumar, Ameenudin and Sangamanatha (2012) recorded temporal processing skills and speech perception in normal hearing train drivers, exposed to occupational engine noise of approximately 86dBA. Their results showed that speech recognition scores in the presence of noise were significantly poorer compared to control individuals and it had an association with their poorer temporal processing skills. Similarly, Feng, Yin, Kiefte and Wang (2010) found deteriorated temporal resolution in the low frequency region in individuals with only the high frequency sensorineural hearing loss. They hypothesized that this deterioration in the normal hearing region is caused by the central deficits secondary to cochlear damage or subclinical cochlear damage in the lower frequencies.

The negative effects on central auditory processing has also been found for exposure levels below damage risk criteria. Norena, Gourevitch, Aizawa and Eggermont (2006) studied the effects of noise on cortical physiology in cats. In their study, cats were exposed to tone pips of 32 different frequencies (5–20 kHz) at 80dBSPL (which is below DRC) for 5 months. Post-exposure, it was observed that cortical representation of the exposure frequencies was poorer and there was a secondary cortical reorganization. Similar effects were found in adult cats by Pienkowski, Munguia and Eggermont (2011) for noise levels of 68dBSPL.

Similarly Zhou and Merzenich (2012) did a study on adult rats which were subjected to structured noise at a sound pressure level of 65dB. Results indicated that even with exposure to such noise levels which was markedly below the safety level standards, there was a significant effect on cortical functions such as frequency response selectivity and rate following ability even with normal peripheral hearing sensitivity. The above studies suggest that exposure to noise level below DRC can have effects on central auditory processing.

Contradicting the above findings, several studies have also found that exposure to moderate levels have a positive/ no effect on hearing. Hamernik, Qiu and Davis (2003) exposed chinchillas to gussian/non-gussian noise (interrupted and non-interrupted conditions) at 100dBSPL. Results suggested that following the exposure to interrupted noise, effects of trauma was lowered, attributable to toughening effect. Suo-qiang, Wei-wei and Ning (2009) did a study on Wistar rats. They took two groups in which one group was exposed to noise at lower levels for 10 hours and then to a traumatic noise. Whereas, the other group of rats were directly exposed to the traumatic noise. Pre and post exposure auditory brainstem thresholds were obtained and the results showed that Wistar rats which received sound conditioning had lesser

threshold shift than the other group. This was again attributed to toughening effect. Alvarado, Santamaría, Gabaldón-Ull, Janero-Flores, Miller and Juiz (2016) in their study in Wistar rats found that the rats that were exposed to noise had a faster recovery in the wave amplitude and latencies of auditory brainstem responses. They were associated with protective mechanisms related to toughening effect in the auditory system.

1.1 Justification for the Study

The environmental noise in India has increased over the years and human beings are exposed to these noises in their daily life. Although most of these noise are below prescribed damage risk criteria, based on the findings in animals one can speculate that even these low levels of noise could have deleterious effects on central auditory processing. Probing into such influences secondary to short-term noise exposure in humans is the interest of the present study. Specifically, it is of interest to study the effect of short-term noise exposure on temporal processing and speech in noise perception, considering their importance in verbal communication. The earlier studies have reported deleterious effects on central auditory processing for exposure duration of several days. However, the effect of short-term exposure of few hours is not known. In most natural instances, noise exposure is only for few hours. Therefore it is important to study the effects of noise exposure of few hours on temporal processing abilities and speech perception.

The effects of short-term noise exposure on cochlea is well known (Furst, Reshef, & Attias, 1992; Kværner, Engdahl, Arnesen & Mair, 1995; Pawlaczyk-Łuszczyńska, Dudarewicz, Bąk, Fiszer, Kotyło & Śliwińska-Kowalska, 2004; Keppler et al, 2010). Moore (2007) has reported that subclinical cochlear damage can

lead to temporal processing abilities. Therefore it is important to understand the role of cochlear functioning in the possible deviations in central auditory processing, secondary to short-term noise exposure. Hence recording of otoacoustic emissions in the pre and post exposure conditions and relating it to the temporal processing and speech perception abilities is likely to help delineate the role of cochlea.

Noise is also known to influence the cognitive abilities (Stansfeld et al., 2005; Gomes, Pimenta & Branco, 1999). Therefore, any observed difference in the temporal processing and speech perception abilities between pre and post exposure conditions could be because of influence of noise exposure on cognitive abilities. In such a case, it is important to control the role of working memory while understanding the effects of short-term noise exposure on temporal processing and speech perception abilities. Hence assessing working memory in the pre and post exposure conditions and relating it to the temporal processing and speech perception abilities is likely to help delineate the role of cognition.

1.2 Aim of the Study

The aim of the study is to investigate effect of short-term, continuous, below DRC levels of noise exposure on outer hair cell functioning, temporal processing, speech perception and cognitive abilities.

1.3 Objectives of the Study

- i.To compare transient evoked otoacoustic emissions (TEOAEs), before and after exposure to 2 hours of 65dBSPL broad band noise
- ii.To compare gap detection thresholds(GDT) and temporal modulation transfer function (TMTF), before and after exposure to 2 hours of 65dBSPL broad band noise

- iii.To compare signal to noise ratio (SNR-50) before and after exposure to 2 hours of 65dBSPL broad band noise
- iv. To compare working memory as assessed on operation span task, before and after exposure to 2 hours of 65dBSPL broad band noise
- v.To study influence of pre-post difference in TEOAEs on pre-post difference in GDT, TMTF and SNR-50

1.4 Hypotheses

- i. There is no significant difference in gap detection thresholds and TMTF, before and after exposure to 2 hours of 65dBSPL broad band noise
- ii.There is no significant difference inSNR-50 before and after exposure to 2 hours of 65dBSPL broad band noise
- iii. There is no significant difference in working memory as assessed on operation span task, before and after exposure to 2 hours of 65dBSPL broad band noise
- iv. There is no significant difference in TEOAEs, before and after exposure to 2 hours of 65dBSPL broad band noise
- v.There is no significant pre-post difference in TEOAEs on pre-post difference in GDT, TMTF and SNR-50

Chapter 2

REVIEW OF LITERATURE

The effects of noise exposure on audition are well known. The different criteria that exist to prevent noise induced hearing loss consider hearing thresholds assessed using audiometry. But the effects of noise exposure can be wide spread, including auditory and non-auditory effects. Nonauditory effects of noise include sleep disturbance, mental health, physiological function, and annoyance, as well as effects on cognitive outcomes such as speech communication, and cognitive performance (WHO, 2000).

2.1 Pathophysiology of Noise Induced Hearing Loss

Permanent Noise Induced Hearing Loss is due to obliteration of cochlear hair cells or damage to their mechano-sensory hair bundles (Liberman & Dodds, 1984). Hair cell damage can be visible within minutes after exposure to noise, and hair cell death can continue for days (Wang, Hirose & Liberman, 2002). Noise-induced loss of spiral ganglion cells (SGCs) and the cell bodies of the cochlear afferent neurons contacting these hair cells are delayed by months and can progress for years (Kujawa & Liberman, 2006). Exposure to noise can result in detrimental effects varying from one to several focal losses of OHCs to total loss of OHCs and IHCs. Several studies have indicated the pathophysiological changes associated with noise induced hearing loss which include mechanical damage to the structures in the organ of corti and degeneration of sensory cells, (Habermann, 1906; Lurie, 1942; Hawkins, Lurie & Davis, 1943; Bohne, 1976; Bohne & Rabbitt, 1983; Bohne & Harding, 2000; Ou, Bohne & Harding, 2000). Some studies suggest the reduced blood flow to the inner ear following intense noise exposure. (Perlman & Kimura, 1962; Hawkins,

1971; Hawkins, Johnson & Preston, 1972; Lipscomb & Roettger, 1973; Santi & Duvall, 1978; Axelsson, Vertes & Miller, 1981; Axelsson & Dengerink, 1987; Duvall & Robinson, 1987; Scheibe, Haupt & Ludwig, 1993). Henderson, Bielefeld, Harris & Hu (2006) found that noise exposure drives mitochondrial activity and free radical production, results in reduction of cochlear blood flow, leads to excitotoxic neural swelling, and induces both necrotic and apoptotic cell death in the organ of corti.

Kujawa and Liberman (2009) found that noise-induced damage to the ear has progressive consequences that are significantly more widespread than are revealed by conventional threshold testing. Reversibility of noise-induced threshold shifts is reported to mask the progressive underlying neuropathology that is likely to have profound long-term consequences on auditory processing. This primary neurodegeneration has been found to result in difficulties including hearing in noisy environments.

2.2 Effect of Noise on Cochlea and Brainstem

Covell (1963) conducted a study in which thirty three adult cats were exposed to different amount of sound exposure. Microscopic examination was conducted for confirmation of tissue injury. He found that wide band noise at 115 dBSPL for one and a half hours resulted in mild injuries; for 2-hour exposures the injuries were moderate to severe while for 8-hour exposures there were severe injuries. A total exposure of two hours at 115 dBSPL was interrupted with then divided into 16 doses of 7.5 minutes each, with one hour inter-exposure intervals. This resulted in minor to moderate changes. The same total energy in the same number of doses for 7.5 minutes, with an inter-exposure interval of 6 hours, was found to produce comparatively lesser injuries.

Mannstrom, Kirkegaard and Ulfendhl (2015) studied the effects of varying levels of repeated moderate noise exposures on hearing. Female rats were subjected to broadband noise exposure for 90 minutes at different levels of intensity. Every 6 week exposure was repeated for a maximum of six repetitions or until a permanent hearing loss was observed. Auditory brainstem responses were used to assess hearing. Rats exposed to the higher intensities of 107 and 110 dB SPL showed permanent threshold shifts subsequent to the first exposure, whereas rats exposed to 101 and 104 dB SPL could be exposed at least six times without inducing a continued change in hearing thresholds. The animals were subjected to high-intensity noise exposure of 110 dB for 4 hours to test for possible change in noise susceptibility following the repeated moderate noise exposures. Rats previously exposed repeatedly to 104 dB SPL were slightly more resistant to high-intensity noise exposure than non-exposed rats or rats exposed to 101 dB SPL indicating the phenomena of toughening.

Keppler, et al. (2010) conducted a study to determine the output levels of a commercially available MP3 player and evaluated changes in hearing after 1 hour of listening to the MP3 player. Twenty-one participants were included in the study. They were exposed to pop-rock music in 6 different sessions using 2 types of headphones at multiple preset gain settings of the MP3 player. 28 participants were included in the control group. The output levels at the full gain setting were found to be 97.36 dBA and 102.56 dBA for the supra-aural headphones and stock earbuds, respectively. In the participants who were exposed to music, significant changes in hearing thresholds and transient-evoked otoacoustic emission amplitudes were found between pre exposure and post exposure measurements. However, such a pattern was not seen in distortion product otoacoustic emissions. The findings indicated temporary changes in hearing sensitivity and the potential harmful effects of listening to an MP3 player.

Kværner, Engdahl, Arnesen and Mair (2009) studied temporary threshold shift (TTS) and otoacoustic emissions after industrial noise exposure. Pure-tone thresholds, otoacoustic emissions (OAEs) and tympanograms were recorded in 13 healthy employees on three successive days pre and post noise exposure for a duration of 7 hours. They found significant pure-tone air-conduction threshold elevation in the region of 4 and 6 kHz in the employees exposed to an industrial noise level of 85–90 dBA. Results also indicated a significant reduction of the amplitude of TEOAEs. There was no correlation found between temporary threshold shift (TTS) and reduction in TEOAEs.

Prendergast, Guest, Léger, Munro, Kluk, and Plack, (2016) investigated the effects of noise exposure on young adults with hearing sensitivity within normal limits at octave frequencies from 500 to 8 kHz. One hundred and twenty six adult participants (75 females) with a wide range of noise exposures were included. Participants had a wide range of lifetime noise exposures. Audiometric thresholds did not differ across noise exposures up to 8 kHz. Auditory brainstem responses and Frequency-following responses were measured. The bandwidth of the ABR stimuli and the carrier frequency of the transposed tones were selected to target the 3-6 kHz characteristic frequency region which is usually linked with noise induced damage in humans. The results indicated no relation between noise exposure and the amplitude of the ABR. The results suggested either that noise-induced cochlear synaptopathy is not a significant problem in young, audiometrically normal adults, or that the ABR and FFR are relatively not sensitive to this disorder in young humans, although it is possible that the effects become manifested with age.

2.3 Effect of Noise on Temporal Processing

Kumar, Ameenudin and Sangamanatha (2012) recorded temporal processing skills and speech perception in normal hearing train drivers, exposed to occupational engine noise more than 80dBA. A total number of 118 participants comprising of three groups of train drivers in the age range of 30–40 (n = 13), 41–50 (n = 9), and 51–60 (n = 6) years and their non-noise-exposed counterparts (n = 30 in each age group) participated in the study. Participants of all the groups had hearing sensitivity within 25 dB HL in the octave frequencies between 250 and 8 kHz. The tests used to assess temporal processing were gap detection, modulation detection and duration pattern. Speech recognition was evaluated in the presence of multi-talker babble presented at -5dB SNR. Their results showed that speech recognition scores in the presence of noise were significantly poorer compared to control individuals and it had an association with their poorer temporal processing skills suggesting that processing of suprathreshold temporal cues can be significantly distorted due to noise exposure which may contribute to the difficulties in hearing in adverse listening conditions.

Kujala and Brattico (2009) assessed the performance in visuo-motor target tracking task and simultaneously recorded the mismatch negativity for /pa/ and /ka/ contrasts on healthy individuals who were exposed to high levels of occupational noise. All their subjects had hearing thresholds that were comparable to the control group. Results showed poorer syllable-discrimination in the left hemisphere of noise exposed individuals in silence and increased N2b complex for the novel sounds. in addition, attention control and ability to focus on visuo-motor tasks were abnormal in noise-exposed group. These results suggested that long- term exposure to occupational noise effects both sound discrimination mechanism and attention control mechanism.

Similarly, Feng, Yin, Kiefte and Wang (2010) determined whether highfrequency sensorineural hearing loss is accompanied by deterioration in temporal resolution in the low-frequency region where hearing sensitivity is within normal range and evaluated whether such temporal processing deficits contribute to difficulty in speech perception in noise. Subjects either with or without high-frequency hearing loss matched by age were taken for the study. Temporal resolution was evaluated using amplitude modulation (AM) detection and gap detection tasks. Since the auditory sensitivity was virtually normal in the low frequency regions, low-pass noise carriers (for AM detection) and gap markers (for gap detection) were used to limit evaluation to these regions. Hearing in noise tests (HINT) was used with varied time compression rates of the speech materials to evaluate the impact of temporal processing deficits on speech perception. They found deteriorated temporal resolution in the low frequency region in individuals with only the high frequency sensorineural hearing loss. They hypothesized that this deterioration in the normal hearing region was caused by the central deficits secondary to cochlear damage or subclinical cochlear damage in the lower frequencies. Similar findings were also reported earlier by Fitzgibbons and Gordon-Salant (1987).

2.4 Effect of Noise on Cognition

Cohen, Evans, Krantz and Stokol (1980) studied the physiological, motivational and cognitive effects of aircraft noise on children. The subjects of experimental group were children attending four noisy elementary schools located around an airport. Children from three quiet schools were taken as control. Peak sound levels in these schools were as high as 95dBA. They found the children in the noisy schools had higher blood pressure and were more likely to give up on a cognitive task than those from matched control (quiet) schools.

Cohen, Krantz, Evans, Stokol and Kelly (1981) conducted a similar study in which they used longitudinal data to determine whether children adapt to the air craft noise over a period of one year. The results indicated that there was little evidence for adaptation to noise over a period of one year. Noise abatement had small unsatisfactory effect on cognitive performance, children's ability to listen to their teachers and their social achievement.

Gomes, Pimenta and Branco (1999) studied the effects of occupational noise exposure to low frequency noise on cognition. Subjects were forty male workers employed as aircraft technicians (aged 35–56 yrs), exposed to occupational noise of large pressure amplitude (> or =90 dB SPL) and low frequency (≤5500 Hz) LPALF noise for a long period of time (range 13-30 yrs). Thirty adult males who were education and age-matched served as controls. P300 event-related brain potential elicited with an auditory discrimination task, and the psychological tests were performed to record any change in cognition. Results indicated that there was deterioration in memory but not in attention as a result of long-term exposure to LPALF.

Elmenhorst, et al. (2010) investigated whether noise-induced sleep has an effect on cognitive performance in laboratory and field. 112 participants were subjected to aircraft noise for 9 successive nights in a laboratory set up. Similarly, 64 participants were examined during 9 successive nights in the neighbourhood of an airport in the field condition. Reaction time, signal detection performance and subjective task load were recorded using psychomotor vigilance task and a memory search task. Results indicated that reaction time significantly reduced in the following morning.

2.5 Effects of Noise Below DRC Levels

In all the aforementioned studies, the exposure levels were high. However a literature review also reveals that he negative effects on central auditory processing are present even for exposure levels below damage risk criteria. Norena, Gourevitch, Aizawa and Eggermont (2006) studied the effects of noise on cortical physiology in juvenile cats. In their study, cats were exposed to tone pips of 32 different frequencies (5–20 kHz) at 80dBSPL (which is below DRC) for 5 months. Post-exposure, it was observed that cortical representation of the exposure frequencies was poorer, cortical representation in the neighbouring frequency regions was increased and there was a secondary cortical reorganization. Similar effects were found in adult cats by Pienkowski, Munguia and Eggermont (2011) for noise levels of 68dBSPL.

Zhou and Merzenich (2012) in their study on 4 adult rats, subjected them to structured noise (4-20 kHz) continuously at 65dBSPL. Neural recordings and analysis procedure indicated that even with exposure to such noise levels which was markedly below the safety level standards, there was a significant effect on cortical functions such as frequency response selectivity and rate following ability despite normal peripheral hearing sensitivity being normal. The above studies suggest that exposure to noise level below DRC can also have effects on central auditory processing.

2.6 Moderate Noise Exposure having a Positive/no effect on Hearing

Contradicting the above findings, several studies have also found that exposure to moderate levels have a positive/ no effect on hearing. Miller, Watson and Covell (1963) were the first to propose the existence of resistance to noise trauma.

Sound conditioning is an active process induced by low-level, nondamaging noise exposure that creates long-term protective effects to succeeding detrimental forms of noise trauma (Niu & Canlon, 2002). Studies have employed two different paradigms to reduce the susceptibility of the inner ear to noise trauma. The first paradigm uses a low-level, non damaging continuous acoustic stimulus prior to the exposure to a traumatic noise. This phenomenon termed as 'sound conditioning' has been demonstrated on a number of species including guinea pigs, gerbils, rabbits and rats (Rajan & Johnstone, 1983; Canlon, Borg & Flock, 1988; Canlon, Borg & Löfstrand, 1980; Ryan, Bennett, Woolf & Axelsson, 1994; Boettcher & Schmiedt, 1995; Dagli & Canlon,1997; Kujawa & Liberman, 1997; Pukkila, Zhai, Virkkala, Pirovola & Ylikoski, 1997; White, Boettcher, Miles & Gratton, 1998).

The second paradigm uses an interrupted schedule of noise at sound levels that induce a temporary threshold shift during the first few days of noise exposure. However, as the daily exposure continues, the degree of threshold shift is reduced, in some cases to no threshold shift regardless of an ongoing exposure. This reduction has been termed 'toughening' or resistance to noise-induced hearing loss (NIHL). Toughening has been demonstrated in different animals including chinchillas, guinea pigs, gerbils and humans (Clark, Boettcher & Bohne, 1987; Sinex, Clark & Bohne, 1987; Campo, Subramaniam & Henderson,1991; Franklin, Lonsbury-Martin, Stagner & Martin, 1991; Subramaniam, Campo & Henderson, 1991; Boettcher, Spongr & Salvi, 1992; Miyakita, Hellström, Frimanson & Axelsson, 1992; Subramaniam, Henderson, Campo & Spongr, 1992; Boettcher, 1993; Henselman, Henderson, Subramaniam & Sallustio, 1994; Henderson, Subramaniam, Papazian & Spongr, 1994; McFadden, Henderson & Shen, 1997; White, Boettcher, Miles & Gratton, 1998).

Campo, Subramaniam and Henderson (1991) explored the effect of different levels of noise exposure on the development of progressive resistance to temporary threshold shift in chinchillas caused by an octave band of noise centered at 0.5 kHz. For six hours a day for ten days, the animals were exposed to either 85, 95 or 100 dB SPL. Hearing thresholds of the animals were recorded electrophysiologically, prior to and after each exposure on a daily basis. At all the levels of exposure, tendency toward decreasing threshold shift with increase in the number of exposures was observed. The amount of threshold shift depended upon the level as well as the test frequency.

Canlon, Borg and Flock (1988) studied whether pre-exposing guinea pigs to a low level acoustic stimulus can reduce the permanently damaging effects of noise. Total of thirty-two guinea pigs were considered for the study. Seven guinea pigs were pre-exposed to a low level acoustic stimulus of 1 kHz tone at 81 dB SPL presented continuously for 24 days prior to exposure to a traumatising noise of 1 kHz tone at 105 dB SPL for 72 hours. Twenty five guinea pigs which were taken as controls were exposed directly to the traumatising noise. Threshold shift was reduced by approximately 20 dB for the experimental than the control group.

Bohne, Yohman and Gruner (1987) exposed four groups of chinchillas to an octave band of noise with a center frequency of 4 kHz at an intensity of 80 or 86 dB SPL on interrupted schedules with 18,42 or 162 hours of rest between consecutive 6-hours exposures. Damage in these ears was compared to that in ears which were exposed to continuous noise equal in total energy. In ears damaged by continuous and interrupted exposures, there was no change in pattern of cell loss observed. In all the ears exposed to interrupted noise, the incidence and average size of the lesion in the basal turn were reduced. When eighteen hours of rest was provided between

consecutive 6 hours, there was a significant protection from damage for the basal turn of the cochlea in chinchilla.

Hamernik, Qiu and Davis (2003) exposed 36 chinchillas (1year to 2 years old) to gussian/non-gussian noise (interrupted; approximately 6 hours/day for 20 days and non-interrupted conditions; approximately 24 hours/day for 5 days) at 100dBA. Results suggested that following the exposure to interrupted noise (gaussian/non-gaussian), effects of trauma was lowered, attributable to toughening effect.

Suo-qiang, Wei-wei and Ning (2009) did a study on Wistar rats. They took two groups in which one group was exposed to 4 kHz octave band noise exposure at lower levels (95 dB SPL) for 10 hours and then to a traumatic noise dose (105 dB SPL for 13 hours) delivered 12hours later. The other group of rats were directly exposed to the traumatic noise. Pre and post exposure auditory brainstem thresholds were obtained and the results showed that Wistar rats which received sound conditioning had lesser threshold shift than the other group. This was again attributed to auditory toughening effect.

Alvarado et al., 2016 carried out a study in Wistar rats. ABRs were evaluated before and after exposures to a sound conditioning protocol consisting of a broadband white noise of 118 dBSPL for 1hour every 72 hours, four times. They found that the rats exposed to noise had a faster recovery in the wave amplitude and latencies of ABRs. They were associated with protective mechanisms related to toughening effect in the auditory system.

2.7 Effect of Temporal Processing on Speech Perception

Studies indicate that there is a direct correlation between temporal processing and speech perception. Tyler, Summerfield, Wood and Fernandes (1982) obtained different measures of temporal processing from 32 participants (16 normal hearing and 16 individuals with a hearing loss of heterogeneous origin). These measures were temporal integration, gap detection, temporal difference limen and gap difference limen. Speech identification in noise was measured with the Four Alternative Auditory Feature (FAAF) test. Most of the hearing-impaired listeners displayed poorer temporal analysis than the normals on all of the psychoacoustical tasks. The hearing-impaired listeners displayed a decreased ability to discriminate subphonemic cues for the voiced-voiceless distinction but their identification of that distinction in stop consonants remained unaffected. The hearing-impaired group made about twice as many errors as did the normals on each of the consonant features of place, manner and voicing when identifying speech in noise. Increased temporal difference limen and gap-detection thresholds were found to correlate significantly with the reduction in speech intelligibility in noise, even though the effects of the pure-tone threshold loss were partialed out.

Feng, Yin, Kiefte and Wang (2010) in their study also suggested that the reduction of temporal resolution may be related to the poorer performance in speech perception in the high frequency SNHL subjects.

The literature review suggests that noise exposure even if may not result in a shift in audiometric thresholds, it may have an effect on cognition or higher auditory functions such as temporal processing and speech perception in difficult listening conditions. The effects of noise exposure below damage risk criteria for a shorter

duration on temporal processing, speech perception and cognition have not been probed into. Hence the present study was taken.

Chapter 3

METHOD

A quasi-experimental (Non equivalent control group) design was used for the study. The following method was used to test the proposed hypothesis of the study.

3.1 Participants

Forty five normal hearing adults in the age range of 18 to 35 years participated in the study. They were ensured for normal hearing sensitivity and normal middle ear functioning using puretone audiometry and immittance tests respectively. Individuals with a present/past history of middle ear pathologies and/or neurological disorders were excluded from the study. None of the participants had a complaint of difficulty in understanding speech in noisy situations. They were also screened out for auditory processing disorders using SCAP-A (Vaidyanath & Yathiraj, 2014).

An informed consent was obtained from all the participants prior to their inclusion in the study. The selected participants were randomly divided into control and experimental groups. The participants in the experimental group were subjected to noise exposure whereas participants in the control group were not exposed to noise. The exposure level was 65dBSPL for duration of 2 hours. There were a total of 25 participants in the control group and 20 in the experimental group.

3.2 Test Environment

All the tests were carried out in an acoustically shield room with the ambient noise levels well within the permissible limits (ANSI S.3, 1991).

3.3 Instrumentation

Several technical equipments were used for the preliminary and experimental audiological evaluations. They included;

- a) A calibrated GSI-61 audiometer for pure tone audiometry. The same was used to present broad band noise to the participants in the experimental group to induce noise exposure.
- b) A calibrated immittance meter to assess middle ear functioning.
- c) SCAP-A to screen for auditory processing disorders.
- d) A laptop computer with MATLAB 2010 to measure gap detection threshold (GDT), temporal modulation transfer function (TMTF), signal to noise ratio 50 (SNR 50) and working memory. A HDA200 headset was used to deliver the test stimuli in these tests.
- e) An ILO-V6 to record and analyse transient evoked otoaccoustic emissions (TEOAE).

3.4 Test Procedure

Those individuals who satisfied all the selection criteria served as participants of the study. The participants involved in the study were blindfolded to the objectives of the study to avoid subject bias in their test performance. The participants were randomly assigned into two groups; experimental and control. The participants in the experimental group were exposed binaurally to broadband noise for 2 hours at 65dBSPL.

Temporal processing as measured on GDT and TMTF, speech perception as measured on SNR-50, cochlear functioning as measured on otoaccoustic emissions

and working memory as on operation span test were recorded individually from each participant of the two groups. The control group was taken to ensure that the difference found between the pre and post noise exposure measurements, if any, was not due to any other variable but for the noise exposure. The procedure used in each of the above mentioned tests is given in the following sections.

3.4.1 *Gap Detection Test:* In this test, noise bursts of 750ms duration with onset and offset linearly ramped for 20ms were used as stimuli. The gaps/silences were introduced at the temporal center of the noise bursts. The duration of such gaps were varied from 1ms to 20ms. The noise bursts without gap served as a reference while the noise bursts with gap served as the target stimuli. A three interval three alternate forced choice procedure was used to estimate the gap detection threshold (minimum gap in noise that the participant can detect).

The stimuli were presented using PC/Laptop using Sennheisser HDA 200 headphones. The tests were performed through the psychoacoustics tool box implemented in MATLAB by Grassi and Soranzo (2009). Every trial involved the presentation of three noise bursts in which two were the reference stimuli and one was a target stimulus. The task of the participants was to identify the noise bursts in which a gap was present by pressing the appropriate response key which was labelled as 1, 2 and 3. The order of presentation of the reference and target stimuli was randomized. The duration of the gap was varied in a two-down one-up adaptive procedure to estimate the 70.7% point on the psychometric function. A total of 12 reversals were obtained. Initial gap size was 20ms which was then altered in 5ms step sizes for the first two reversals. The subsequent reversals would then be altered in steps of 1ms gap size. The average of the last 8 reversals was considered for calculating the gap detection threshold.

3.4.2 *Temporal Modulation Transfer Functions:* This test used Gaussian noises of 1s duration was used as stimuli. The noise was sinusoidally amplitude modulated using equation 1. The noise tokens without modulation served as a reference while the noise tokens with modulation served as the target stimuli. A three interval three alternate forced choice procedure was used to estimate the minimum modulation depth the participant can detect.

Modulated Noise = $(1 + m \times \sin(2 \times pi \times mf \times t)) \times n(t)$Equation 1

Where, $m = modulation \ depth$, $mf = modulation \ frequency \ (either 32 \ or 128Hz)$, t is time, n(t) is Gaussian noise of time 't'.

Similar to GDT, the stimulus were presented using PC/Laptop using Sennheisser HDA 200 headphones and psychoacoustics tool box implemented in MATLAB by Grassi and Soranzo (2009) was used to run the test. Every trial involved the presentation of three noise tokens in which two were the reference stimuli and one was a variable or target stimulus. The task of the participants was to identify the modulated noise token. The order of presentation of reference and target stimuli was randomized. The modulation depth was varied in a two-down one-up procedure to estimate the 70.7% point on the psychometric function. A total of 12 reversals were obtained. Initial modulation depth was 0dB and was then altered in 5dB steps for the first two reversals. The subsequent reversals were then altered in steps of 1dB depth. The modulation detection threshold was assessed for two modulation frequencies, i.e., 32Hz and 128Hz. The average of the last 8 reversals was considered for calculating the modulation detection threshold.

3.4.3 *Speech Perception in Noise-SNR-50:* This test estimated minimum ratio required for 50% identification of monosyllables (SNR-50). SNR paradigm from

Smriti-Shravan developed by Kumar and Maruthy (2016) was used for this purpose. The module had 19 bisyllables mixed with broadband noise at varying SNRs. A one down one up procedure was used for tracking SNR-50. One bisyllable was presented in each trial. The test started with SNR of 2 dB. The SNR was subsequently decreased by 2 dB for every correct response and increased by 2 dB for every incorrect response. The bisyllables were displayed in the order similar to the Dravidian language script (Figure 3.1). Participants were instructed to listen carefully, recognise the bisyllable heard and indicate the response by clicking on the respective bisyllable among the 19 bisyllables displayed on a computer screen. A total of 10 reversals were used and the average of last six reversals were taken as the SNR-50.

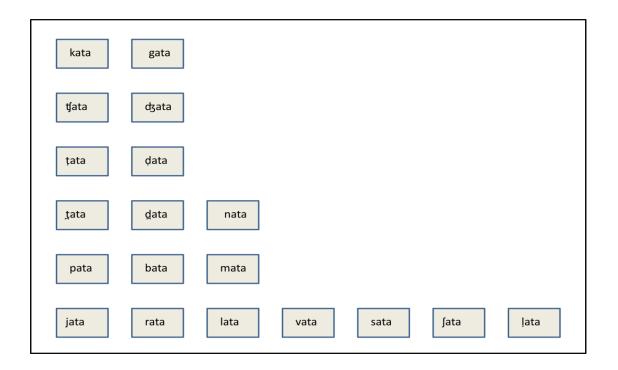
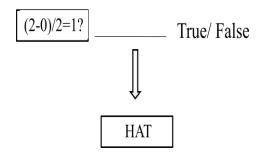


Figure 3.1: Nineteen bisyllables displayed on the computer screen.

3.4.4 Transient Evoked Otoacoustic Emissions (TEOAEs): The participants were made to sit comfortably on a chair inside a sound treated room. The probe with an appropriate ear tip was positioned in the external ear canal and was adjusted to

give flat frequency spectrum across frequency range. ILO 292 USB-II (Otodynamics, UK) equipment with V6 software was used to acquire the otoacoustic emissions. A good probe fit was ensured by adjusting the probe such that the spectrum of the click in the ear canal is relatively flat and stimulus was not ringing. TEOAEs responses for 260 sweeps of clicks (80µs) were averaged at intensity of 80 dB peakSPL. The amplitude of OAE and noise at the octave and mid-octave frequencies from 1000 Hz to 6000 Hz were noted down from the averaged response.

3.4.5 Operation Span Test: A module developed within Smriti-Sravan program developed by Kumar and Maruthy (2016) was used for this purpose. The procedure to measure working memory capacity was adapted from versions of the operation span task used by Kane, Hambrick, Tuholski, Wilhelm, Payne and Engle (2004). Guidelines recommended by Conway, Kane, Bunting, Hambrick, Wilhelm and Engle (2005) were followed during administration and scoring. To be consistent with the Conway et al.'s recommendations, the task consisted of 'items' that vary in difficulty, which were manipulated by varying the number of 'elements' per item. The operation span task consisted of 5 items and 20 elements. The number of elements per item was varied from 3 to 7. Each element included a mathematical operation which included addition/substraction and division/multiplication, and an English word at the end, as shown below



The participant's task was to read the mathematical problem aloud, then answer 'true' or 'false' to indicate whether the given answer was correct or wrong, and then say the word. After all the elements in an item are presented, the participants were shown a set of ten English words. The participants had to click on the words which came during the presentation of that particular item in the sequence of presentation. The difficulties of the items were randomized such that the numbers of elements were unpredictable at the outset of an item. The mathematical problem was taken as the distracter stimulus.

For each correct item, one mark was given, only if all the elements were repeated correctly. If some of the elements were incorrect, the number of correct elements was divided by total number of elements. For example, if three elements out of five elements of an item were correct, then a score of 0.6 was given. The scores of all five items were added and divided by five to obtain the final score for working memory. That is, if the scores obtained in five items were 1, 0.8, 0.5, 0.16 and 1, the total obtained was 3.46 and the final score for working memory was 0.693 (3.46 divided by 5). The scores for working memory ranged between 0 and 1. Participants were given a practice trial before the actual test.

The above mentioned tests (transient evoked otoacoustic emissions (TEOAEs), gap detection test (GDT), temporal modulation transfer function (TMTF), signal to noise ratio 50 (SNR 50) and operation span were administered four times on each participant of the two groups. The tests were administered only in the right ear in all the participants. The stimuli for the tests were presented at the maximum comfortable level of the participant. In experimental group, the procedure involved five steps.

Step 1: Baseline measurement 1, one hour before beginning of noise exposure

Step 2: Baseline measurement 2, immediately before the beginning of noise exposure

Step 3: Noise Exposure for 2 hours

Step 4: Post exposure 1, immediately after the cessation of noise

Step 5: Post exposure 2, one hour after the cessation of noise

In the participants of control group, the same measurements (operationally termed as M1, M2, M3 and M4) were done with the time gaps same as that in the experimental group. However, these participants were not exposed to noise. Participants in both the groups were seated in a quiet room and were watching a silent movie between baseline 2 and the next measurement. Figure 3.2 is a schematic representation of the experimental procedure.

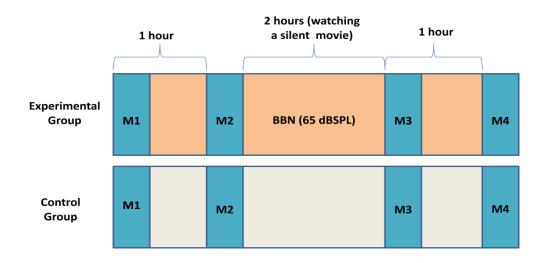


Figure 3.2: Schematic representation of the experimental procedure.

3.5 Data Analysis

Individual amplitudes (dB) of TEOAEs, gap detection thresholds (ms), modulation detection thresholds (dB), SNR-50 (dB) and working memory span were tabulated and the group data was analysed to compare the performance across the four measurements and between the two groups, to derive the effect of short term noise exposure on the aforementioned parameters.

Chapter 4

RESULTS

The present study was aimed to test whether short-term exposure to broadband noise of levels below damage risk criterion has an effect on otoacoustic emissions, temporal processing, speech perception and cognition.

In the present study, measurement condition (the four measurements: M1, M2, M3 & M4) served as the independent variable, whereas the different measures such as TEOAEs, SNR-50, GDT and TMTF served as dependent variables. The comparison across the four conditions was separately done in the two groups using repeated measures ANOVA. ANOVA was chosen based on the results of Shapiro Wilk's normality test which showed normal distribution of the data in all the variables in both the groups. The results obtained in the study are reported under the following headings:

- 1. Results of Otoacoustic emissions
- 2. Results of temporal modulation transfer functions (TMTF)
- 3. Results of gap detection test (GDT)
- 4. Results of speech perception in noise
- 5. Results of working memory

4.1 Results of Otoacoustic Emissions

Table 4.1 gives the mean and standard deviation (SD) of amplitude of transient evoked otoacoustic emissions in the four measurement conditions (M1-Measurement 1, M2-Measurement 2, M3-Measurement 3, M4-Measurement 4) in the control and experimental groups. The data showed mean differences across the

four measurements in both the groups but there was no common pattern in the way the mean amplitudes varied across the four conditions in the 5 measurement frequencies or in the overall amplitude.

Table 4.1: Mean and standard deviation (SD) of amplitude (in dBSPL) of otoacoustic emissions in the four measurements in the control and the experimental group (M1-Measurement, M2-Measurement 2, M3-Measurement 3 & M4-Measurement 4)

Frequency	Measure	Control group			Experimental group				
	Measure	M1	M2	M3	M4	M1	M2	M3	M4
1 kHz	Mean	4.46	3.85	4.49	3.26	5.91	4.85	6.20	4.78
	SD	4.75	4.88	4.69	5.09	6.38	8.03	6.10	7.62
1.5 kHz	Mean	8.46	8.07	7.67	8.49	9.85	9.30	9.51	9.15
	SD	5.01	5.20	4.78	4.54	5.22	5.19	5.19	5.07
2 kHz	Mean	6.43	5.88	6.07	6.55	9.11	9.18	8.95	8.89
	SD	5.14	5.08	5.03	4.99	4.73	4.68	4.77	8.35
3 kHz	Mean	5.75	5.55	5.58	5.84	7.86	8.3	7.93	8.25
	SD	5.19	5.17	5.24	5.10	6.28	6.26	7.05	7.20
4 kHz	Mean	8.16	8.18	8.20	8.02	7.95	8.08	8.15	8.08
	SD	7.89	7.61	7.50	7.55	7.81	7.52	7.70	8.09
Overall	Mean	15.09	14.99	15.04	15.03	16.82	16.68	16.81	16.61
	SD	5.16	5.10	5.07	4.81	4.58	4.51	4.58	5.0

The significance of observed differences in the mean amplitudes was tested using repeated measures ANOVA. This was done separately for the two groups. Results (Table 4.2) showed that there was no significant main effect (p>0.05) of

condition on the amplitude of otoacoustic emissions at any of the frequencies and also on the overall amplitude. This was true in control as well as experimental groups.

Table 4.2: Results of repeated measures ANOVA showing the effect of measurement condition on the amplitude of TEOAEs in the control and experimental group

	Contro	ol group		Experimental group			
Frequency	F	df(error)	P	F	df(error)	P	
1 kHz	2.117	3(72)	0.106	2.437	3(57)	0.074	
1.5 kHz	0.868	3(72)	0.462	0.298	3(57)	0.826	
2 kHz	1.057	3(72)	0.373	0.229	3(57)	0.876	
3 kHz	0.933	3(72)	0.429	0.798	3(57)	0.500	
4 kHz	0.207	3(72)	0.891	0.183	3(57)	0.908	
Overall	0.069	3(72)	0.976	0.175	3(57)	0.913	

The results of signal to noise ratio (SNR) of TEOAEs also was analysed in the present study. This was of particular interest as SNR is the measure of interest while using TEOAEs for clinical purposes. The results of SNR also did not show any significant main effect (p>0.05) of condition, and is given in Annexure I. It was not presented in this section as it did not provide any additional information.

4.2 Results of TMTF

4.2.1 Results of modulation detection threshold at 32Hz

Figure 4.1 gives the mean and standard deviation of the modulation detection thresholds at 32 Hz modulation frequency in the four measurement conditions in the two groups. Comparison of the mean thresholds across the four conditions showed

that there was no specific pattern in the way thresholds varied in the control group. Whereas in the experimental group, mean thresholds were elevated in the post noise exposure conditions (M3 & M4) as compared to the pre exposure measurements (M1 & M2).

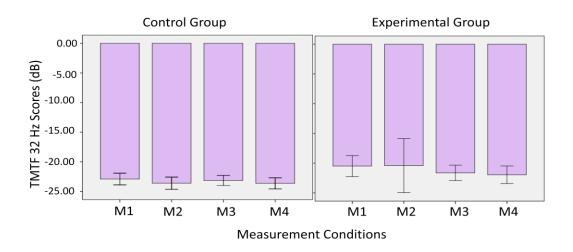


Figure 4.1: Mean and standard deviation (SD) of modulation detection thresholds at 32 Hz in the four measurement conditions, in control and experimental group.

Repeated measures ANOVA was used to test the significance of difference in mean thresholds across the four conditions. Two separate ANOVAs were done for the two groups. Results showed that there was no significant main effect of condition on temporal modulation transfer function- 32 Hz in control [F (3, 72) = 0.842, p=0.475] as well as experimental groups [F (3, 57) = 0.482, p=0.696].

4.2.2 Results of TMTF at 128Hz

Figure 4.2 gives the mean and standard deviation of the temporal modulation transfer function at 128 Hz modulations frequency in the four measurement conditions in the two groups. The data showed mean differences across the four measurements. However, there was no specific pattern in the way the thresholds varied across the four conditions.

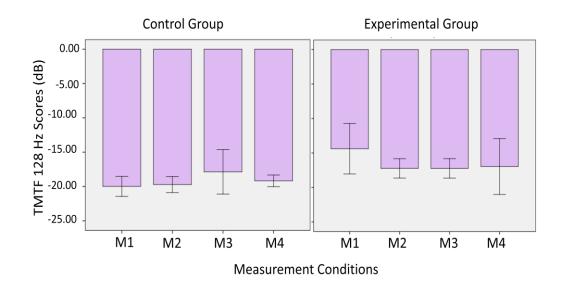


Figure 4.2: Mean and standard deviation (SD) of modulation detection thresholds at 128 Hz in the four measurement conditions, in control and experimental group.

The significance of mean differences was tested using repeated measures ANOVA, separately in the two groups. Results showed that there was no significant main effect of condition on modulations detection thresholds at 128 Hz in the control [F(3, 72) = 1.155, p=0.333] as well as experimental groups [F(3, 57) = 1.110, p=0.352].

4.3 Results of GDT

Figure 4.3 gives the mean and standard deviation of the gap detection thresholds in the four measurement conditions, in the two groups. The data showed mean differences across the four measurements. However, there was no specific pattern in the way the thresholds varied.

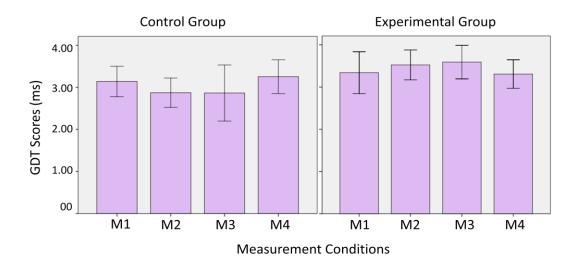


Figure 4.3: Mean and standard deviation (SD) of gap detection thresholds in the four measurement conditions, in control and experimental group.

The significance of mean differences was tested using repeated measures ANOVA, separately in the two groups. Results showed that there was no significant main effect of condition on GDT in control [F(3, 72) = 0.985, p=0.405] as well as experimental groups [F(3, 57) = 1.204, p=0.316].

4.4 Results of SNR-50

Figure 4.4 gives the mean and standard deviation of the SNR 50 in the four measurements in the two groups. The data showed mean differences across the four measurements. In the control group, mean SNR-50 increased progressively from M1 to M4. However, in the experimental group SNR-50 decreased immediately after noise exposure as compared to pre-exposure measurements.

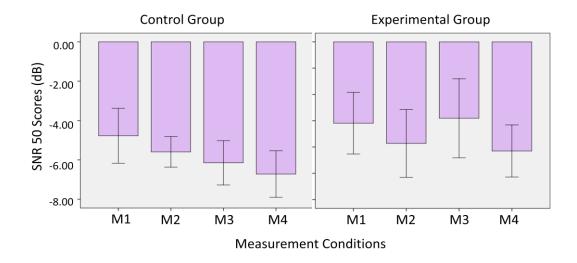


Figure 4.4: Mean and standard deviation (SD) of SNR 50 in the four measurements, in control and experimental group.

Results of repeated measures ANOVA showed that there was a significant main effect of condition on SNR-50 in control group [F (3, 72) = 3.050, p=0.034] while there was no main effect of measurement condition on SNR-50 in experimental group [F (3, 57) = 1.532, p=0.216]. A subsequent Bonferroni pair-wise comparison across the four measurement conditions in control group did not show any significant difference between any of the pairs of comparison.

4.5 Results of Operation Span Test

Figure 4.5 gives the mean and standard deviation of the operation span in the four measurements in the two groups. The data showed mean differences across the four measurements. In the control group, mean operation span scores were higher in M3 and M4 compared to M1 and M2. On the other hand in the experimental group, mean scores were decreased in M3 compared to M1 and M2. Mean scores in M4 was again higher than mean scores in M3.

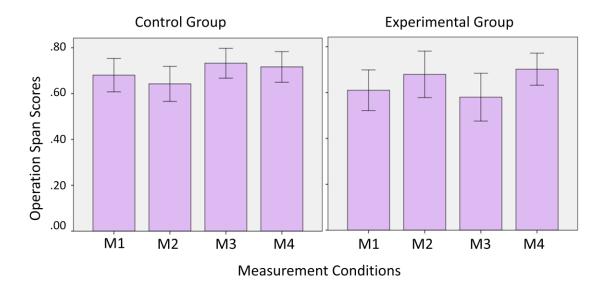


Figure 4.5: Mean and standard deviation (SD) of operation span in the four measurements, in control and experimental group.

The significance of mean differences was tested using repeated measures ANOVA, separately in the two groups. Results showed that there was a significant main effect of measurement condition on operation span in experimental group [F (3, 57) =4.134, p=0.010] whereas, no such effect was seen on control group [F (3, 72) = 2.346, p=0.080]. A subsequent Bonferroni pair-wise comparison showed that there was a significant difference between measurement conditions M3 and M4 (p=0.019) while the difference across the other pairs were not significantly different.

Chapter 5

DISCUSSION

In this study, the aim was to investigate the effect of short-term exposure to broadband noise levels below damage risk criterion on outer hair cell functioning, temporal processing, speech perception and working memory. A thorough review of literature shows equivocal results on the effect of moderate levels of noise. That is, while certain studies indicate negative influence on central auditory processing, others have shown positive effect on the auditory functioning in the form of toughening phenomena. In the present study we specifically probed into the effects of 65dBSPL noise exposed for 2 hours.

To ensure that the difference in any of the variables studied, if any, is resultant of noise exposure, two baselines were obtained. Comparison of the two baseline measurements showed that there is no significant difference in any of the measures (otoacoustic emissions, temporal processing, SNR-50 & working memory). This was true both in control and experimental groups.

Further in the control group, the participants were not exposed to noise and therefore comparison across the four measurements in this group was to give an evidence of the normal variation in the target measures. Results showed no differences across the 4 measurement conditions in the control group. This suggested that, any difference observed between pre and post noise exposure measurements in the experimental group was attributable to noise exposure and not to trial-to-trial variation in the measure.

In the results we found some interesting findings which are explained under the following headings.

- 1. Effect on cochlear functioning
- 2. Effect on temporal processing
- 3. Effect on speech perception in noise
- 4. Effect on working memory

5.1 Effect on Cochlear Functioning

In the present study cochlear functioning was assessed using otoacoustic emissions. Otoacoustic emissions specifically the transient evoked type is known to be sensitive to subtle changes in the outer hair cell functioning (Kemp, 2002). Earlier studies (Feng, et al. 2010) have attributed deficits in auditory processing abilities to subclinical damage in outer hair cell functioning. Therefore, it was of interest to study whether exposure to 2 hours of moderate level noise leads to outer hair cell dysfunction which may or may not relate to deficits in auditory processing.

Results showed that the otoacoustic emissions were comparable across the four measurements. The comparable emissions across the four measurements in the control group indicates stability in the measures across trials and absence of difference in the experimental group indicates that the noise exposure did not influence outer hair cell functioning. This was true with all the frequencies and the overall amplitude. Keppler, et al. (2010) found that there was a significant change in hearing thresholds and amplitudes of transient-evoked otoacoustic emission between preexposure and postexposure measurements for participants who listened to 1 hour of pop-rock music using the MP3 player. But in this study, the output levels at the full gain setting were higher than 90 dBA for both supra-aural headphones and stock

earbuds, respectively. Similarly evidence for cochlear damage after noise exposures was found by Kværner, et al. (2009) (115 dBSPL intensity noise for different duration of exposures from 2 hours to 8 hours) and Covell (1963) (7 hours industrial noise exposures at an intensity of 85–90 dBA). The absence of difference found in the present study can be attributed to the relatively lower levels of noise exposure. Therefore it can be inferred that the exposure broadband signals of levels up to 65dBSPL, for up to 2 hours is safe for the outer hair cells.

5.2 Effect on Temporal Processing

In the present study, temporal processing was assessed using temporal modulation transfer function (TMTF) and gap detection test. Both of these assess temporal resolution in particular. Temporal resolution in known to influence speech perception in challenging listening conditions (Tyler et al., 1982) and is a susceptible auditory process. Therefore, it was of interest to learn whether the short-term exposure to moderate levels of noise has influence on temporal processing.

Results of the present study showed that there was no significant difference in temporal processing abilities (gap detection tests, modulation detection thresholds at 32 Hz and 128 Hz) across the four measurements in control as well as experimental groups. The findings indicate that 2 hours of exposure to moderate levels of noise is safe for temporal processing abilities.

Earlier study by Kumar, et al. (2012) found reduced temporal processing abilities in subjects exposed to occupational noise. Their subjects were train drivers and the temporal processing abilities were traced using gap detection, modulation detection, and duration pattern tests. Such deficits were present even in the absence of temporary hearing threshold shift. However, the train drivers in their study were

exposed to occupational noise (8-10 hours per day) of higher than 80dBA and were exposed for several years. Thus comparing the findings of the two studies, it can be inferred that temporal processing is a susceptible measure and gets affected earlier to hearing sensitivity but not with short-term exposure to 65dBSPL noise.

Higher susceptibility of temporal processing abilities is also supported by studies in individuals with partial hearing loss. Feng, et al. (2010) found deteriorated temporal resolution in the low frequency region in individuals with only the high frequency sensorineural hearing loss. Similar findings were also reported by Fitzgibbons and Gordon-Salant (1987) who found subjects with hearing impairment restricted to frequencies above 1 kHz had processing deficits within their frequency regions of normal hearing sensitivity.

5.3 Effect on Speech Perception in Noise

Speech perception in noise is one of the important auditory ability necessary for daily life. As evident in the literature review, short-term noise exposure results in toughening phenomena leading to better auditory abilities (Campo, et al., 1991; Bohne, et al., 1987; Hamernik, et al., 2003; Suo-qiang, et al., 2009 & Alvarado, et al., 2016). On the contrary, certain studies showed poorer processing abilities secondary to short-term noise exposure. In view of this equivocal literature, it was of interest in the present study to investigate the effect of short of exposure to noise on speech perception in noise.

Results of SNR-50 showed a significant main effect of condition on SNR-50 in control group while there was no significant difference between any of the pairs of comparison in the control group. There was no such effect seen on experimental group. This indicates that the 2 hours of noise exposure to 65dBSPL noise did not

influence speech in noise perception. The present findings suggests that there is no toughening phenomena for speech in noise perception and also noise exposure of moderate levels does not negatively influence speech in noise perception. From this finding, one can conclude that a moderate levels of noise exposure for 2 hours is safe for speech in noise perception.

Speech perception in noise in individuals exposed to high levels of occupational noise was investigated by Kumar, et al. (2012). They had found poor speech in noise perception in their subject group. Similarly, Feng, et al. (2010) found reduced HINT scores for individuals with high frequency sloping hearing loss and pure tone thresholds within 25 dB HL at octave frequencies between 125 and 8000Hz. Their findings indicated that speech perception deficits accompanied by HF SNHL may not be limited to the loss of audibility to high-frequency speech information (Feng, et al., 2010). Therefore it can be inferred that speech perception in noise is affected secondary to short-term noise exposure, but not for 2 hours of exposure to 65dBSPL noise.

5.4Effect of Noise on Working Memory

Elmenhorst, et al. (2010) found a significant reduction in the reaction time in individuals who were exposed to aircraft noise for nine consecutive nights. Similar reduction in cognition due to prolonged exposure to noise is also reported in other studies (Gomes, et al., 1999; Cohen, et al., 1980; Cohen, et al., 1981). Results of the present study showed that there is a significant main effect of condition on operation span in the experimental group whereas, no such effect is seen on control group. Pairwise comparison showed that there was no significant difference between pre and post

exposure conditions (M2 and M3). This indicates that the noise exposure did not influence working memory.

On comparing the two post exposure conditions (M3 and M4), it was found that M4 had better working memory than that in M3. This shows that, post noise exposure, after a rest period, working memory is getting posted. The exact reason for the change is not clear and needs future research for conclusive derivations.

Overall, the findings of the present study indicate that there is no effect of 2 hours of noise exposure to 65dBSPL noise on outer hair cell functioning, temporal processing, speech perception noise and working memory. In our daily life, there are many instances where we are exposed to moderate levels of noise for few hours, including, watching a movie in a theatre, or attending a public function, or travelling in a train, exercising with the music on. It was speculated that such noise may have temporary effects on temporal processing and speech perception. Findings support that such exposures are safe and even temporary effects on temporal processing and speech perception are absent. In the present study the attributes were measured within 15 minutes of cessation of the 2 hours noise exposure. Yet there was no difference in measures.

The findings of the present study however do not rule out the effects if the exposure duration or the noise levels are increased. Future studies can either increase the noise duration or the levels (below DRC), and document the effects in the manner similar to that in the present study.

Chapter 6

SUMMARY AND CONCLUSIONS

With the growing population and industrialisation, environmental noise is increasing at an alarming rate. Awareness about the hazardous effects of noise exposure has to be done to the public especially in a developing nation like India. In the past, several studies were carried out to see the effects of noise exposure on auditory functioning. The findings indicate the possibility of both temporary and permanent dysfunction at different levels of the auditory system, depending on the stimulus intensity and duration. Considering that human beings are exposed to moderate levels of noise for short duration in their daily life, it was of interest in the present study to document the temporary effects of such noise, if any, on temporal processing and speech perception.

A total number of forty five participants were included, of whom, twenty five served as the control group while twenty served as participants of the experimental group. Participants in the experimental group were exposed to two hours of broad band noise at 65 dBSPL whereas, participants in the control group were not exposed to any noise. Outer hair cell functioning (as on transient evoked otoacoustic emissions), temporal processing (as on gap detection test and temporal modulation transfer function), speech perception in noise (as on SNR-50) and working memory (as on operation span) were assessed in both the groups. The tests were carried out four times in each group; two baseline conditions (one hour and fifteen minutes before the noise exposure) and two post exposure conditions (immediately and one hour after the cessation of noise). In the control group, wherein there was no noise exposure, the measurements were done with similar time intervals. The four

measurements were separately compared in the two groups using repeated measures ANOVA to derive the temporary effects of noise on the aforementioned parameters.

Results of the present study showed that there was no significant difference between the pre and post noise exposure measurements. Furthermore, there was no difference between the two baseline conditions and also between the two post exposure conditions. The absence of difference across the four measurements was seen both in control and experimental groups.

The findings in the present study indicate that there are no temporary effects of two hours of 65 dBSPL noise exposure on outer hair cell functioning, temporal processing, speech perception in noise and working memory. Therefore, moderate levels of noise exposure for up to two hours can be as safe for the auditory attributes tested in the present study.

In the present study, we used a strong research design and controlled the possible extraneous variables by adopting multiple measurements and a control group. Future studies can use the same research design to probe into the temporary effects of noise levels below damage risk criteria, by increasing the intensity and duration of the noise.

REFERENCES

- Alvarado, J. C., Santamaría, V. F., Gabaldón-Ull, M. C., Jareno-Flores, T., Miller, J. F & Juiz, J. M. (2016). Noise induced "Toughening" effecting Wistar Rats: Enhanced auditory brainstem responses are related to calretinin and nitric oxide synthase upregulation. *Frontiers in Neuroanatomy*, 10, 19.
- American National Standards Institute (1991). *American National Standards for Maximum Permissible Ambient Noise Levels for Audiometric Test Rooms*.

 ANSI S3.1 (1991). New York: American National Standards Institute.
- Axelsson, A., Vertes, D., & Miller, J. (1981). Immediate noise effects on cochlear vasculature in the guinea pig. *Acta Oto-Laryngologica*, *91*(3–4), 237–46.
- Axelsson, A., & Dengerink, H. (1987). The effects of noise on histological measures of the cochlear vasculature and red blood cells: A review. *Hearing Research*, 31(2), 183–191.
- Boettcher, F. A. (1993). Auditory brain-stem response correlates of resistance to noise-induced hearing loss in Mongolian gerbils. *The Journal of the Acoustical Society of America*, *94*(6), 3207–14. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8300955
- Boettcher, F. A., & Schmiedt, R. A. (1995). Distortion-product otoacoustic emissions in Mongolian gerbils with resistance to noise-induced hearing loss. *The Journal of the Acoustical Society of America*, *98*(6), 3215–3222. http://doi.org/10.1121/1.413811
- Boettcher, F. A., Spongr, V. P., & Salvi, R. J. (1992). Physiological and histological changes associated with the reduction in threshold shift during interrupted noise exposure. *Hearing Research*, 62(2), 217–36. http://doi.org/10.1016/0378-5955(92)90189-T
- Bohne, B. A. (1971). Scar formation in the inner ear following acoustical injury:

 Sequence of changes from early signs of damage to healed lesion. (Doctoral dissertation). Washington University, St. Louis, Missouri.

- Bohne, B.A. (1976). Mechanisms of noise damage in the inner ear. In: Henderson D., Hamernik, R.P., Dosanjh, D.S., Mills, J.H. (Eds.), *The Effects of Noise on Hearing* (pp. 41-68). New York, Raven Press.
- Bohne, B. A, & Harding, G. W. (2000). Degeneration in the cochlea after noise damage: primary versus secondary events. *The American Journal of Otology*, 21(4), 505–509.
- Bohne, B. A., & Rabbitt, K. D. (1983). Holes in the reticular lamina after noise exposure: Implication for continuing damage in the organ of Corti. *Hearing Research*, 11(1), 41–53.
- Bohne, B. A., Yohman, L., & Gruner, M. M. (1987). Cochlear damage following interrupted exposure to high-frequency noise. *Hearing Research*, 29(2), 251–264.
- Campo, P., Subramaniam, M., & Henderson, D. (1991). The effect of "conditioning" exposures on hearing loss from traumatic exposure. *Hearing Research*, 55(2), 195–200.
- Canlon, B., Borg, E., & Flock, Å. (1988). Protection against noise trauma by preexposure to a low level acoustic stimulus. Hearing Research (Vol. 34).
- Canlon, B., Borg, E., & Lofstrand, P. (1980). Physiologic and morphologic aspects to low-level acoustic stimulation. In A. L. Dancer, D. Henderson, R. J. Salvi, and R. P. Hamernik (Ed.), Noise-Induced Hearing Loss (pp. 489–499). St. Louis, MO: Mosby Yearbook, 1992.
- Clark, W. W., Bohne, B. A., & Boettcher, F. A. (1987). Effect of periodic rest on hearing loss and cochlear damage following exposure to noise. *The Journal of the Acoustical Society of America*, 82(4), 1253–1264.
- Cohen, S., Evans, G. W., Krantz, D. S., & Stokols, D. (1980). Physiological, motivational, and cognitive effects of aircraft noise on children: moving from the laboratory to the field. *The American Psychologist*, *35*(3), 231–243.
- Cohen, S., Krantz, D. S., Evans, G. W., Stokols, D., & Kelly, S. (1981). Aircraft noise and children: Longitudinal and cross-sectional evidence on adaptation to

- noise and the effectiveness of noise abatement. *Journal of Personality and Social Psychology*, 40(2), 331–345.
- Conway, A. R., Kane, M. J., Bunting, M. F., Hambrick, D. Z., Wilhelm, O., & Engle, R. W. (2005). Working memory span tasks: A methodological review and user's guide. *Psychonomic bulletin & review*, *12*(5), 769-786.
- Covell, W. P. (1963). Cumulative auditory effects resulting from multiple exposure to intense acoustic stimulation. Part ii. Histological effects of intense sound on the inner ear. Retrieved from http://oai.dtic.mil/oai/oai?verb=getRecord&metadataPrefix=html&identifier=AD0 404297
- Dagli, S., & Canlon, B. (1997). The effect of repeated daily noise exposure on sound-conditioned and unconditioned guinea pigs. *Hearing Research*, 104(1), 39–46. http://doi.org/10.1016/S0378-5955(96)00179-7
- Duvall, A. J., & Robinson, K. S. (1987). Local vs Systemic Effects of Acoustic Trauma on Cochlear Structure and Transport. Archives of Otolaryngology -Head and Neck Surgery, 113(10), 1066–1071.
- Elmenhorst, E. M., Elmenhorst, D., Wenzel, J., Quehl, J., Mueller, U., Maass, H., Vejvoda, M., & Basner, M. (2010) Effects of nocturnal air craft noise on cohnitive performance in the following morning: dose-response relationships in laboratory and field. *International Archives of Occupational and Environmental Health*, 83(7), 743-751.
- Feng, Y., Yin, S., Kiefte, M., & Wang, J. (2010). Temporal resolution in regions of normal hearing and speech perception in noise for adults with sloping high-frequency hearing loss. *Ear and Hearing*, *31*, 115-25.
- Fitzgibbons, P. J., & Gordon-Salant, S. (1987). Temporal gap resolution in listeners with high-frequency sensorineural hearing loss. *The Journal of the Acoustical Society of America*, 81(1), 133–7.
- Franklin, D. J., Lonsbury-Martin, B. L., Stagner, B. B., & Martin, G. K. (1991).

 Altered susceptibility of 2f1-f2 acoustic-distortion products to the effects of

- repeated noise exposure in rabbits. *Hearing Research*, *53*(2), 185–208. http://doi.org/10.1016/0378-5955(91)90053-C
- Furst, M., Reshef, I., & Attias, J. (1992). Manifestations of intense noise stimulation on spontaneous otoacoustic emission and threshold microstructure: experiment and model. *The Journal of the Acoustical Society of America*, 91(2), 1003-1014.
- Gomes, L. M. P., MartinhoPimenta, A. J. F., & CasteloBranco, N. A. A. (1999). Effects of occupational exposure to low frequency noise on cognition. *Aviation, space, and environmental medicine*, 70, A115-A118
- Grassi, M., & Soranzo, A. (2009). MLP: A MATLAB toolbox for rapid and reliable auditory threshold estimation. *Behavior research methods*, *41*(1), 20-28.
- Habermann, J. (1906). Contribution to teaching of the professional deafness. *Archives* of Otology, 69, 106–130
- Hamernik, R. P., Qiu. W., & Davis, B. (2003). Cochlear toughening, protection, and potentiation of noise-induced trauma by non-Gaussian noise. *The Journal of the Acoustical Society of America*, 113 (2), 969-975.
- Hawkins, J. E., Lurie, M. H., & Davis, H. (1943). *Injury of the inner ear produced by exposure to loud tones*. Committee on Medical Research, Office of Scientific Research and Development Contract OEMcmr-194.
- Hawkins, J. E. (1971). The Role of Vasoconstriction in Noise-Induced Hearing Loss.
 Annals of Otology, Rhinology & Laryngology, 80(6), 903–913. Hawkins, J.
 E., Johnson, L.-G., & Preston, R. E. (1972). Cochlear microvasculature in normal and damaged ears. The Laryngoscope, 82(7), 1091–1104.
- Henderson, D., Bielefeld, E. C., Harris, K. C., & Hu, B. H. (2006). The Role of Oxidative Stress in Noise-Induced Hearing Loss. *Ear and Hearing*, 27(1), 1–19.
- Henderson, D., Subramaniam, M., Papazian, M., & Spongr, V. P. (1994). The role of middle ear muscles in the development of resistance to noise induced hearing loss. *Hearing Research*, 74(1), 22–28.

- Henselman, L. W., Henderson, D., Subramaniam, M., & Sallustio, V. (1994). The effect of "conditioning" exposures on hearing loss from impulse noise.

 Hearing Research, 78(1), 1–10. http://doi.org/10.1016/0378-5955(94)90038-8
- Kane, M. J., Hambrick, D. Z., Tuholski, S. W., Wilhelm, O., Payne, T. W., & Engle,
 R. W. (2004). The generality of working memory capacity: a latent-variable approach to verbal and visuospatial memory span and reasoning. *Journal of Experimental Psychology: General*, 133(2), 189.
- Kellerhals, B. (1972). Acoustic trauma and cochlear microcirculation. *Advances* in *Oto-Rhino-Laryngology*, *18*, 91–168.
- Keppler, H., Dhooge, I., Maes, L., D'haenens, W., Bockstael, A., Philips, B., Swinnen, F., & Vinck, B. (2010). Short-term auditory effects of listening to an MP3 player. *Archives of Otolaryngology–Head & Neck Surgery*, *136*(6), 538-548.
- Kværner, K. J., Engdahl, B., Arnesen, A. R., & Mair, I. W. (1995). Temporary threshold shift and otoacoustic emissions after industrial noise exposure. *Scandinavian audiology*, 24(2), 137-141.
- Kujala, T., & Brattico, E. (2009). Detrimental noise effects on brain's speech functions. *Biological Psychology*, *81*(3), 135–143. http://doi.org/10.1016/j.biopsycho.2009.03.010
- Kujawa, S. G., & Liberman, M. C. (1997). Conditioning-related protection from acoustic injury: effects of chronic deefferentation and sham surgery. *Journal* of Neurophysiology, 78(6), 3095–106. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9405529
- Kujawa, S. G., & Liberman, M. C. (2006). Acceleration of Age-Related Hearing Loss by Early Noise Exposure: Evidence of a Misspent Youth. *Journal of Neuroscience*, 26(7), 2115-2123.
- Kujawa, S. G., & Liberman, M. C. (2009). Adding Insult to Injury: Cochlear Nerve Degeneration after "Temporary" Noise-Induced Hearing Loss. *Journal of Neuroscience*, 29(45), 14077-14085.

- Kumar, U. A., Ameenudin, S., & Sangamanatha, A. (2012). Temporal and speech processing skills in normal hearing individuals exposed to occupational noise. *Noise and Health, 14*, 100–105.
- Kumar, U. A., & Maruthy (2016). Effects of auditory cognitive training on some auditory and speech perception skills in individuals with sensorineural hearing loss. Unpublished research project at AIISH, funded by DSD
- Kværner, K. J., Engdahl, B., Arnesen, A. R., & Mair, I. W. (1995). Temporary threshold shift and otoacoustic emissions after industrial noise exposure. *Scandinavian audiology*, 24(2), 137-141.
- Lawrence, M., Gonzalez, G., & Hawkins, J. E. (1967). Some physiological factors in noise-induced hearing loss. *Journal of American Industrial Hygiene Association*, 28, 425–430.
- Liberman, M. C., & Dodds, L. W. (1984). Single-neuron labeling and chronic cochlear pathology. III. Stereocilia damage and alterations of threshold tuning curves. *Hearing Research*, *16*(1), 55–74.
- Lim, D. J. (1976). Ultrastructural cochlear changes following acoustic hyperstimulation and ototoxicity. *Annals of Otology, Rhinology & Laryngology*, 85(6), 740-751.
- Lim, D. J., & Melnick, W. (1971). Acoustic damage of the cochlea: A scanning and transmission electron microscopic observation. *Archives of otolaryngology*, *94*(4), 294-305.
- Lipscomb, D. M., & Roettger, R. L. (1973). Capillary constriction in cochlear and vestibular tissues during intense noise stimulation. *The Laryngoscope*, 83(2), 259–263.
- Lipscomb, D. M., & Thomas, W. A. (1972). *Indicators of environmental noise* (pp. 211–241). New York: Plenum Press.
- Lurie, M. H. (1942). The Degeneration and Absorption of the Organ of Corti in Animals. *Annals of Otology, Rhinology & Laryngology*, 51, 712-717.

- Mannström, P., Kirkegaard, M., & Ulfendahl, M. (2015). Repeated Moderate Noise Exposure in the Rat--an Early Adulthood Noise Exposure Model. *Journal of the Association for Research in Otolaryngology : JARO*, 16(6), 763–72.
- McFadden, S. L., Henderson, D., & Shen, Y.-H. (1997). Low-frequency "conditioning" provides long-term protection from noise-induced threshold shifts in chinchillas. *Hearing Research*, 103(1), 142–150. http://doi.org/10.1016/S0378-5955(96)00170-0
- Miyakita, T., Hellström, P.-A., Frimanson, E., & Axelsson, A. (1992). Effect of low level acoustic stimulation on temporary threshold shift in young humans. *Hearing Research*, 60(2), 149–155. http://doi.org/10.1016/0378-5955(92)90017-H
- Miller, J. M., Watson, C. S., & Covell, W. P. (1963). Deafening effects of noise on the cat. *Acta Oto-laryngol*. Suppl. 176,1-91
- Moore, B.C. (2007). *Perceptual consequence of cochlear hearing loss*. Cambridge: Oxford University Press.
- Nelson, D. I., Nelson, R. Y., Concha-Barrientos, M., & Fingerhut, M. (2005). The global burden of occupational noise-induced hearing loss. *American journal of industrial medicine*, 48(6), 446-458.
- Niu, X., & Canlon, B. (2002). Protective mechanisms of sound conditioning. *Advances in Oto-Rhino-Laryngology*, *59*, 96–105.
- NIOSH (1998). Criteria for a recommended standard: occupational noise exposure.

 National Institute for Occupational Safety and Health, Publication No. 98126.
- Noreña, A. J., Gourevich, B., Aizawa, N., Eggermont, J. J., & Eggermont, J. J. (2006). Spectrally enhanced acoustic environment disrupts frequency representation in cat auditory cortex. *Nature Neuroscience*, *9*(7), 932–939.
- OSHA (2012). Appendices, taken from https://www.osha.gov/dts/osta/otm/new noise/appendices_all.pdf .

- Ou, H. C., Bohne, B. A., & Harding, G. W. (2000). Noise damage in the C57BL/CBA mouse cochlea. *Hearing Research*, 145(1), 111–122.
- Pawlaczyk-Łuszczyńska, M. A. Ł. G. O. R. Z. A. T. A., Dudarewicz, A., Bąk, M., Fiszer, M., Kotyło, P., & Śliwińska-Kowalska, M. A. R. I. O. L. A. (2004). Temporary changes in hearing after exposure to shooting noise. *International journal of occupational medicine and environmental health*, 17(2), 285-294.
- Perlman, H. B., & Kimura, R. (1962). Cochlear Blood Flow in Acoustic Trauma. *Acta Oto-Laryngologica*, *54*(1–6), 99–110.
- Pienkowski, M., Munguia, R., & Eggermont, J. J. (2011). Passive exposure of adult cats to bandlimited tone pip ensembles or noise leads to long-term response suppression in auditory cortex. *Hearing Research*, 277, 117–126.
- Prendergast, G., Guest, H., Léger, A., Munro, K., Kluk, K., & Plack, C. (2016). Evidence that hidden hearing loss does not vary systematically as a function of noise exposure in young adults with normal audiometric hearing. *The Journal of the Acoustical Society of America*, *139*(4), 2122–2122.
- Pukkila, M., Zhai, S., Virkkala, J., Pirvola, U., & Ylikoski, J. (1997). The "Toughening" Phenomenon in Rat's Auditory Organ. *Acta Oto-Laryngologica*, *117*(sup529), 59–62. http://doi.org/10.3109/00016489709124081
- Rajan, R., & Johnstone, B. M. (1983). Crossed cochlear influences on monaural temporary threshold shifts. *Hearing Research*, *9*(3), 279–294.
- Ryan, A. F., Bennett, T. M., Woolf, N. K., & Axelsson, A. (1994). Protection from noise-induced hearing loss by prior exposure to a nontraumatic stimulus:

 Role of the middle ear muscles. *Hearing Research*, 72(1–2), 23–28.

 http://doi.org/10.1016/0378-5955(94)90201-1
- Santi, P. A., & Duvall, A. J. (1978). Stria vascularis pathology and recovery following noise exposure. *Otolaryngology*, 86(2), ORL 354-361.

- Scheibe, F., Haupt, H., & Ludwig, C. (1993). Intensity-related changes in cochlear blood flow in the guinea pig during and following acoustic exposure. *European Archives of Oto-Rhino-Laryngology*, 250(5), 281–285.
- Sinex, D. G., Clark, W. W., & Bohne, B. A. (1987). Effects of periodic rest on physiological measures of auditory sensitivity following exposure to noise. *The Journal of the Acoustical Society of America*, 82(4), 1265–1273. http://doi.org/10.1121/1.395829
- Smith, C. A., Covell, W. P., & Eldredge, D. H. (1954). *The effects of intense sound on the cochlea*. WADC Technical Report 54-21 Wright-Patterson AFB, Ohio.
- Spoendlin, H. (1962). Ultrastructural features of the organ of Corti in normal and acoustically stimulated animals. *Annals of Otology, Rhinology & Laryngology, 71*, 657–677.
- Stansfeld, S. A., Berglund, B., Clark, C., Lopez-Barrio, I., Fischer, P., Öhrström, E., Haines, M. M., Head, J., Hygge, S., Kamp, I & Berry, B. F. (2005). Aircraft and road traffic noise and children's cognition and health: a cross-national study. *The Lancet*, *365*(9475), 1942-1949.
- Subramaniam, M., Campo, P., & Henderson, D. (1991). The effect of exposure level on the development of progressive resistance to noise. *Hearing Research*, 52(1), 181–187. http://doi.org/10.1016/0378-5955(91)90197-H
- Subramaniam, M., Henderson, D., Campo, P., & Spongr, V. (1992). The effect of "conditioning" on hearing loss from a high frequency traumatic exposure. *Hearing Research*, 58(1), 57–62. http://doi.org/10.1016/0378-5955(92)90008-B
- Suo-qiang.Z., Wei-wei. G., & Ning. U. (2009). The protective effects against noise trauma by sound conditioning in rats. *Journal of Otology*, 4, 59-60.
- Tyler, R. S., Summerfield, Q., Wood, E. J., & Fernandes, M. A. (1982).
 Psychoacoustic and phonetic temporal processing in normal and hearing-impaired listeners. *The Journal of the Acoustical Society of America*, 72(3), 740–752.

- Vaidyanath, R., & Yathiraj, A. (2014). Screening checklist for auditory processing in adults (SCAP-A): Development and preliminary findings. *Journal of Hearing Science*, 4(1), 33–43.
- Wang, Y., Hirose, K., & Liberman, M. C. (2002). Dynamics of Noise-Induced Cellular Injury and Repair in the Mouse Cochlea. *Journal of the Association for Research in Otolaryngology*, *3*(3), 248–268.
- Ward, W. D. & Nelson, D. A.: "On the equal-energy hypothesis relative to damagerisk criteria in the chinchilla". In: Occupational Hearing Loss. D. B.Robinson (Ed), Academic Press, London and New York, 1971, p. 225.
- White, D. R., Boettcher, F. A., Miles, L. R., & Gratton, M. A. (1998). Effectiveness of intermittent and continuous acoustic stimulation in preventing noise-induced hearing and hair cell loss. http://dx.doi.org/10.1121/1.421303. http://doi.org/10.1121/1.421303
- WHO. (2000). Guidelines for Community Noise. Geneva: World Health Organisation.
- Zhou, X., & Merzenich, M. M. (2012). Environmental noise exposure degrades normal listening processes. *Nature Communications*, *3*, 843.

Annexure 1

Table 1: Mean and standard deviation (SD) of SNR (in dBSPL) of otoacoustic emissions in the four measurements in the control group and the experimental group (M1- Measurement, M2- Measurement 2, M3- Measurement 3 & M4- Measurement 4)

Frequency	Measure	Control group				Experimental group			
	Wiedsure	M1	M2	M3	M4	M1	M2	M3	M4
1 kHz	Mean	10.47	10.05	9.89	7.73	10.99	10.23	9.41	10.88
	SD	6.86	6.90	6.43	6.21	7.41	6.14	8.28	8.08
1.5 kHz	Mean	14.86	13.99	13.9	12.94	15.34	14.93	15.79	15.39
	SD	5.77	6.12	6.73	4.76	5.81	5.96	7.09	7.09
2 kHz	Mean	13.56	12.47	13.56	13.20	15.58	15.57	15.60	15.75
	SD	5.02	5.68	5.28	4.58	5.33	4.56	4.62	5.00
3 kHz	Mean	12.03	11.93	12.36	12.05	14.48	14.9	13.94	14.58
	SD	5.72	5.15	5.28	4.90	6.13	6.19	6.64	6.38
4 kHz	Mean	12.69	12.86	12.59	12.43	13.0	13.28	12.40	13.14
	SD	7.45	6.89	7.17	7.45	7.67	7.06	6.99	7.35
Overall	Mean	10.94	10.6	10.40	10.13	12.82	12.21	11.37	12.39
	SD	5.91	5.37	5.15	4.98	4.86	5.06	5.90	5.79

Table 2: Results of repeated measures ANOVA showing the effect of measurement condition on the SNR of TEOAEs in the control and experimental groups

	Contro	ol group		Experimental group			
Frequency	F	df(error)	P	F	df(error)	P	
1 kHz	2.892	3(72)	0.041	0.731	3(57)	0.538	
1.5 kHz	1.190	3(72)	0.320	0.249	3(57)	0.862	
2 kHz	0.965	3(72)	0.414	0.024	3(57)	0.995	
3 kHz	0.495	3(72)	0.687	1.627	3(57)	0.193	
4 kHz	0.298	3(72)	0.827	0.877	3(57)	0.458	
Overall	0.739	3(72)	0.532	1.813	3(57)	0.155	