CHAPTER V
Occupational Environment of Workers of a Tea Processing Factory with Specific Reference to Excess Risk Estimates of Hearing Impairment and Associated Biochemical Changes

Abstract

This study was undertaken on 24 healthy male workers of a tea-processing factory of Eastern India. The objective was to estimate an excess risk of hearing impairment in two groups of workers. Additionally, an attempt was made to examine whether there exist relationship between estimates of excess risk of hearing impairment and stress biochemical factors. Equal number of subjects were sampled from office workers (n=12) and factory workers (n=12). Compared to the noise exposure level [94.6-101.0 dB(A)] of subjects of factory workers, the office workers were considered as low-noise exposed [63.0 dB(A)] control group. Noise exposure of factory workers of this processing unit thus was found exceeded the acceptable limit of 90.0 dB(A) for 8 hour exposure stipulated by Occupational Safety and Health Administration (OSHA). Audiometric analyses of both groups of subjects were conducted at various frequencies (0.125-10.0 kHz). The average estimated excess risks of hearing impairment of the subjects were calculated from audiometric data using five standard model equations. The hearing threshold levels of office workers at audiometric test frequencies of 0.125-2.0 kHz did not exceed 25.0 dB (A) to cause hearing disability. On the contrary, hearing threshold levels of factory workers exceeded 25.0 dB (A) at all audiometric test frequencies (0.125 – 10.0 kHz) and showed signs of hearing disability in both right and left ears. Octave band analyses of noise of the factory environment further showed the presence of high sound pressure level [95.3 dB (A)] at 4.0 kHz frequency, which may be a major reason to cause hearing handicap in these subjects. Simple linear correlation analyses between stress hormone (cortisol), pro-oxidative (NO, TBARS) and anti-oxidative (GSH, CAT and SOD) markers and estimates of excess risk of hearing impairment revealed a significant association between these variables indicating that hearing handicap of factory workers might be an oxidative-stress mediated systemic response. This suggestion was further supported by results of two liver function enzymes, aspartate aminotransferase (AST) and alanine aminotransferase (ALT), which were also seen significantly associated with estimates of excess risk of hearing impairment.
Relevance to Industry. This study emphasizes the need for introduction of hearing conservation programme for factory workers. It further highlights the importance of periodic checking of the factory workers for determining their auditory threshold shift. Additional care also may be taken to monitor their adverse health effects regularly, and to provide noise-insulated area during work-rest cycle.
1. Introduction

Noise is one of the most menacing industrial pollutants, involving every industry and causing severe hearing loss of workers in every country in the world. Occupational hearing loss includes acoustic traumatic injury and noise-induced hearing loss (NIHL), and can be defined as a partial or complete hearing loss in one or both ears as the result of one's employment. NIHL is the second most common form of acquired hearing loss after age-related loss (presbycusis), with studies showing that people who are exposed to noise levels higher than 85.0 dB suffered from NIHL [1]. A typical NIHL is of a sensory neural type involving injury to the inner ear. It is bilateral and symmetrical, usually affecting the higher frequencies (3.0, 4.0 or 6.0 kHz) and then spreading to the lower frequencies (0.5, 1.0 or 2.0 kHz) [2]. The International Standards Organization (ISO), Eastern Economic Community (EEC) and other developed countries have recommended a maximum permissible occupational noise exposure limit in the range of 85.0 - 90.0 dB(A) Leq for 8 hours per day (40 hours per week), but in India, model rules under Indian Factories Act – 1948 stipulate a limit of 90.0 dB (A) for 8 hours exposure. But due consideration shall be given to the fact that most of the factories in India operate 6 days in a week, which means 48 hours per week. Most of the skilled and unskilled workers employed in the industries are either illiterate or semi literate, having no information about the noise regulations and adverse effects of noise on their performance and health [3]. Unfortunate though, being mostly economically underprivileged and anxious to retain their jobs, majority of the workers accept adverse working conditions and do not demand health and safety measures.

It is well established that exposure to high-intensity noise causes hearing loss that can be temporary (temporary threshold shift) or permanent (permanent threshold shift), depending on the duration and the intensity of the noise exposure [4]. According to American Speech Language Hearing Association (ASHA, 1981) [5], hearing impairment or hearing loss usually denotes a change for the worse in auditory structure or auditory function, outside the range of normal hearing. Hearing handicap is usually denoted as an average hearing threshold level (HTL) of greater than 25.0 dB(A) for both ears at selected frequencies [6]. According to an earlier report [7], continual exposure to high noise levels damage and destroy hair cells within the ear; making noise-induced hearing loss an irreversible impairment. Additionally, there is evidence that the noise damage is induced in the affected tissues at molecular level by the elevation of substances (for example, free radicals), which, together, have been called reactive oxygen species (ROS) [8-11]. Although ROS are normal byproducts of cellular aerobic metabolism, these unstable molecules can impair cellular lipids, proteins and nucleic acids in DNA if the balance of corresponding antioxidants is disrupted [11]. Reports also indicate that health effects of noise are substantial and studies clearly have established a relation between exposure to noise and stress responses [12]. Several studies have reported about
adverse effects of industrial noise on metabolic processes and psychological status [13-15]. Further, elevation of cellular levels of ROS following stress and injury has been reported not only in the cochlea but also in other tissues, including the brain and heart [4]. Literature survey also has revealed that, in addition to cochlea, noise-induced oxidative DNA and lipid damage was found in liver that has high metabolic functions [11]. Moreover, there is both direct and indirect biochemical and histological evidence of cochlear oxidative stress [11].

In this perspective, the present study was undertaken in a tea-processing factory of Dooars, North-Eastern India to examine the effects of noise exposure on hearing disability of factory workers based on their audiometric data and to estimate an excess risk by using a set of standard equations. Attempt was also made to examine whether this intense chronic noise could produce downstream noise stress responses like oxidative and antioxidative marker changes and alteration in liver function enzymes. This study was further intended to make a comparative assessment of various noise effects between the factory workers and office workers.

### 2. Materials and Methods

#### 2.1. Recruitment of Study Population

This cross sectional study was undertaken in a tea-processing unit of Dooars, West Bengal, India. Healthy male subjects of similar age, height and weight group with no previous history of systemic disease and 8-10 years of continuous professional record were randomly selected for this study from both factory and office staff. Informed consent was obtained from all subjects according to the guidelines of a study protocol approved by the Institutional Scientific Committee. They were interviewed by using a formatted questionnaire to obtain relevant information, including general health profile, current medication, use of personal protective equipment, and lifestyle characteristics such as cigarette smoking, alcohol and tea consumption. Both the groups of selected workers were used to attend 8 hours shift duty every day, 6 days per week. As far as the nature of works at factory and office are concerned, factory workers were engaged in cutting and fermentation of raw tea leaves with no significant load carrying activities, whereas, office workers were engaged in usual paper and file works. The selected factory workers (n=12) were exposed to equivalent noise levels [L_Aeq] of 94.6-101.0 dB(A) during work, while the selected office workers (n=12) who served as control were exposed to...
equivalent noise levels \([L_{eq} \text{ (m)}]\) of 63.0 dB(A) during work. The different physical and physiological variables of the selected subjects are reported (mean \(\pm\) SD) in Table 1. The body surface area (BSA) and body mass index (BMI) were calculated respectively by using the Dubois body surface nomographic chart [16] and BMI formula as given elsewhere [17]. Resting heart rate and blood pressure (mmHg) of the subjects were recorded respectively by using POLAR heart rate monitor (S610i, POLAR Electro Oy, Finland) and sphygmomanometer.

Table 1. Measurements of different physical and physiological parameters of the factory workers and office workers (n = 24).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>Factory workers (n =12)</td>
<td>32.25 (± 3.19)</td>
</tr>
<tr>
<td></td>
<td>Office workers (n =12)</td>
<td>35.58 (± 0.95)</td>
</tr>
<tr>
<td>Body height (m)</td>
<td>Factory workers (n =12)</td>
<td>1.59 (± 0.02)</td>
</tr>
<tr>
<td></td>
<td>Office workers (n =12)</td>
<td>1.62 (± 0.02)</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>Factory workers (n =12)</td>
<td>49.33 (± 1.25)</td>
</tr>
<tr>
<td></td>
<td>Office workers (n =12)</td>
<td>53.46 (± 1.25)</td>
</tr>
<tr>
<td>Body surface area (m²)</td>
<td>Factory workers (n =12)</td>
<td>1.48 (± 0.03)</td>
</tr>
<tr>
<td></td>
<td>Office workers (n =12)</td>
<td>1.55 (± 0.03)</td>
</tr>
<tr>
<td>Body mass Index (kg/m²)</td>
<td>Factory workers (n =12)</td>
<td>19.57 (± 0.40)</td>
</tr>
<tr>
<td></td>
<td>Office workers (n =12)</td>
<td>20.52 (± 0.40)</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>Factory workers (n =12)</td>
<td>121.67 (± 3.26)</td>
</tr>
<tr>
<td></td>
<td>Office workers (n =12)</td>
<td>128.08 (± 2.47)</td>
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<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>Factory workers (n =12)</td>
<td>73.75 (± 2.68)</td>
</tr>
<tr>
<td></td>
<td>Office workers (n =12)</td>
<td>77.33 (± 0.75)</td>
</tr>
<tr>
<td>Resting heart rate (beats/min)</td>
<td>Factory workers (n =12)</td>
<td>74.92 (± 3.12)</td>
</tr>
<tr>
<td></td>
<td>Office workers (n =12)</td>
<td>74.17 (± 1.19)</td>
</tr>
</tbody>
</table>

2.2. Assessment of Environmental Variables

Dry bulb, wet bulb and globe temperatures at work places of both groups of subjects (factory and office) at different hours of the day shift were recorded on several occasions. Relative humidity was determined. Wet Bulb Globe Temperature (WBGT) Index is one of the simplest and most convenient
heat stress index to ascertain the thermal stress in a warm humid environment and it was determined by following the formula of Yaglou & Minard (1957) [18].

 Formula: \( \text{WBGT (indoor)} = 0.7 (T_{nwb}) + 0.3 (T_g) \)

 where, \( T_{nwb} \) = natural wet bulb temperature  \
 \( T_g \) = Vernon globe temperature

2.3. Recording of Noise Level and Octave Band Analysis

Prior approval from authority of the factory as well as consent from the workers was obtained before recording of noise and audiometric tests. Noise level of the processing factory was measured by using a Sound Level Meter (Type 2235; Brüel and Kjaer, Naerum, Denmark). It was calibrated before each measurement with the sound level calibrator Type 4230. During measurement of sound care was taken to note that calibration period of the measuring equipment was not expired. Acoustic test of microphones were undertaken before all measurements. Two measurements were performed at day shift (between 9.30 to 10.30 A.M. and 2.30 to 3.30 P.M.). Duration of noise measurement in each series was 1 hour. Noise was measured from five different points according to the formula for determining number of measuring points (\( N_b \)). Each measurement was taken for at least 10 minutes with an interval of 2 minutes between each measurement, since the noise was persistent and unchangeable in nature. There was no recordable side-noise and care was taken that surrounding objects do not cause measuring errors. While measuring, at least 1 m distance was left from walls and other reflecting surfaces, if any; 1.2 m – 1.5 m above the floor and, if the measuring point was near the window, 1.5 m was left from such windows. Since noise was recorded within the shed of the processing factory, result of two measuring points if exceeded by 7.0 dB was discarded. Octave band analysis was also performed. The Octave Filter Set (Type 1624) of Sound Level Meter (Type 2235; Brüel and Kjaer, Naerum, Denmark) was used for the purpose. Equivalent noise levels (\( L_{eq} \)) were automatically calculated as continuous steady noise, which was measured according to the International Organization for Standardization for the measurement of community noise [19].

2.4. Audiometric Measurements

Tests were carried out in an audiometric test room that was designed to meet the background noise requirements for ANSI S3.1-1999 [20]. Background noise level in the test room was monitored intermittently throughout each testing session. On entering into the test room, each subject was asked to sit and rest for half an hour. Audiometric testing was conducted at lower frequencies (0.125, 0.25 kHz) and at higher frequencies (8.0 and 10.0 kHz) in addition to ISO 1999 (1990) [21] and National
Institute of Occupational Safety and Health (NIOSH) (1998) [22] recommended frequencies (0.5, 1.0, 1.5, 2.0, 3.0, 4.0 and 6.0 kHz) to assess the effect of prolonged (8-10 years) noise exposure on hearing disability of workers. The audiometric testing consisted of air conduction, pure tone, hearing threshold measurement of left and right ears alternatively at different frequencies. An Arphi 700MK IV (India) Audiometer that was calibrated following the recommended protocols of the manufacturer was used for the auditory sensitivity measurements. Prior to each measurement, instrument was checked. Before each specified experimental condition was tested, an audiometric baseline hearing threshold test (BHT) was routinely performed on each subject. The randomly selected pure tone of different frequencies and intensities was heard by right ear through earphone. The test was started from 0 dB(A) for all tested frequencies. As the intensity was increased or decreased, each subject was asked to indicate when he could hear the tone by pressing the indicator bulb switch, or when it ceases to be audible by releasing the indicator bulb switch. The lowest sound intensity that could barely be heard by the subject for each tested frequency was determined and recorded as hearing threshold level. The threshold levels at eleven selected frequencies (0.125 – 10.0 kHz) were plotted as an audiogram to show graphically hearing loss for the selected frequencies. The same procedure was repeated for left ear.

2.5. Calculation of Excess Risk of Hearing Impairment

The estimated excess risk of hearing impairment was calculated from the measured audiometric data of the selected subjects by using five most commonly used model equations (Table 2). These equations are taken from the American Academy of Otolaryngology (AAO, 1979) [23], American Academy of Ophthalmology and Otolaryngology (AAOO, 1959) [24], National Institute of Occupational Safety and Health (NIOSH, 1972, 1998) [25, 22] and British Society of Audiology (BSA, 2004) [26]. These equations determined average hearing loss for a range of frequencies (0.5 - 4.0 kHz). These equations used low and high fences of 25 and 92 dB (A), representing 0% and 100.0% hearing handicap limits, respectively. The low fence of 25.0 dB(A) represents normal hearing. The formula developed by Kavanagh (1992, 2001) [27, 28] and housed cm World Wide Web http://www.occupationalhearingloss.com was used to calculate estimated excess risk of hearing impairment of noise-exposed subjects of tea processing factory by using selected model equations.

2.6. Blood Sample Collection

Venous blood samples were collected. After separation of plasma, the buffy coat was removed and packed cells (RBCs) were washed thrice with cold physiological saline. RBC lysate was prepared by
lysing a known volume of RBC with cold phosphate buffer (pH 7.4). The hemolysate was separated by
centrifuging at 3000 g for 10 min at 2°C and used to measure the activities of superoxide dismutase
(SOD) and catalase (CAT) [29].

2.7. Analytical Procedure

Biochemical assays of hepatic enzymes aspartate aminotransferase (AST) and alanine
aminotransferase (ALT) were performed from serum by using kit methods marketed by E. Merck (India)
Ltd.

Plasma cortisol level was estimated by using the assay kit obtained from ElAgen cortisol kit,
Adaltis Italia S.p.A, Italy. All samples were assayed in duplicate. The intra assay variation was 6.7%. To
avoid inter assay variation all samples were run at one time.

Nitric oxide (NO) decomposes rapidly in aerated solutions to form stable nitrite/nitrate
products. In the present study, nitrite accumulation was estimated by Griess reaction [30] and was used
as an index of NO production. The amount of nitrite in the sample (μmolar/unit) was calculated from a
sodium nitrite standard curve.

Quantitative measurement of lipid peroxidation was performed by following the thiobarbituric
acid (TBA) test [31]. The amount of thiobarbituric acid reactive substances (TBARS) formed, an
indicator of lipid peroxidation, was quantitated with TBA and was used as an index of lipid peroxidation.
The results were expressed as nmol TBARS/dL plasma.

SOD was assayed according to the method of Misra and Fridovich (1972) [32]. The change in
absorbance due to the conversion of epinephrine to adrenochrome could be markedly inhibited by the
presence of SOD. The reaction was initiated by addition of epinephrine and an increase in absorbance
at 480nm was measured by a UV-Double Beam Spectrophotometer (Shimadzu 160A).

CAT was assayed by the method of Cohen et al. (1970) [33]. The enzyme-catalyzed
decomposition of H₂O₂ was measured at 480nm in a UV-Double Beam Spectrophotometer (Shimadzu
160A).

Plasma GSH level was estimated according to the method of Ellman (1959) [34].

2.8. Statistics

Student's t-test was performed to find out whether or not scores of different groups differ significantly.
We determined the association between different variables studied by simple correlation analysis.
StatsDirect software (Version 2.6.5, StatsDirect Ltd., UK) was used for statistical data analysis. Chosen
level of significance in each case was \( p = 0.05 \) or lower.
3. Results and Discussion

3.1. External Physical Factors

During the study period at the tea-processing factory, the external physical influencing factors, namely WBGT Index and relative humidity of factory and office respectively were 25.3°C, 79.0% and 23.9°C, 72.5%. The values of WBGT index show that both the groups of workers did not exceed the recommended level of American Conference of Governmental Industrial Hygienists (ACGIH) WBGT threshold limit value (TLV) for continuous moderate work [35], suggesting that both the groups of workers were not under thermal environmental stress while working during 8 hour duty.

3.2. Measurement of Noise Level

The recorded noise level at factory was 94.6-101.0 dB(A), while at office it was 63.0 dB(A). This indicated that the factory workers were chronically exposed to a noise level which exceeded the acceptable limit of 90.0 dB (A) for 8 hour exposure stipulated by Occupational Safety and Health Administration (OSHA) [3].

Since exposure to high-intensity noise causes hearing loss that can be temporary (temporary threshold shift) or permanent (permanent threshold shift), depending upon the duration and the intensity of the noise exposure [4], we examined the audiometry threshold profile of the workers of both factory and office. Figure 1 shows the variation of mean hearing threshold levels (dB) as a function of audiometric frequencies (kHz) for left and right ears of two groups of subjects.
Figure 1. Auditory threshold profile of right ears and left ears of the selected subjects (n = 24) of a tea estate. Error bars represent mean ± SD.

It was observed that mean hearing threshold levels were lower for office workers and higher for factory workers. The binaural average of hearing threshold levels of office workers at the audiometric test frequencies of 0.125, 0.25, 0.5, 1.0 and 2.0 kHz in both the ears did not exceed 25.0 dB(A) to cause hearing handicap. The reason of this lower hearing threshold level in office workers might be due to
their exposure to low equivalent noise level \( [L_{eq}(8 h)] \) of 63.0 dB(A) during 8 hours of work. However, average hearing threshold levels of both ears of factory workers exceeded 25.0 dB(A) at all the audiometric test frequencies. Results indicated that hearing threshold levels increased with exposure to high noise level, which in the present study was 94.6–101.0 dB(A). The one-tail \( t \)-test indicated that there was a highly significant \( (p<0.001) \) difference in auditory threshold levels of both ears at most of the tested frequencies including 4.0 kHz between factory and office workers. This finding is consistent with earlier Hearing Disability Assessment Report of Ireland (1998), which indicated that noise induced permanent threshold shift usually occurs first around 4.0 kHz and then progresses to involve adjacent frequencies, and such hearing loss at 4.0 kHz progresses over the first ten years of noise exposure and then tends to stabilize [36]. The result of the present study further finds its support from earlier studies [6, 37, 38] and suggest that high noise exposure of the factory workers [96.0–101.0 dB(A)] possibly was responsible for their significant alteration in hearing threshold level.

Figure 2 depicts that mean threshold level at most of the audiometric frequencies (0.125–10.0 kHz) for the right ear was higher as compared to left ear for both groups of subjects, except at some lower frequencies (0.5, 1.0 and 2.0 kHz) for office workers where changes were either indistinguishable or less.

**Figure 2.** Audiogram of right and left ears for two groups of subjects \( (n=24) \) of a tea estate.

Previous reports although indicate that noise effects in the left ear is stronger compared to the right [39, 40], but this does not agree with our results, which suggest a stronger noise effects in right ear rather than left ear in both factory and office groups of workers. The plausible reason for such right-
left variation although is not known, but a differential sensory-neural hearing loss in two ears might be an acceptable suggestion [41,42].

3.3. Excess Risk Estimates

Table 2 shows that highest values of average estimated excess risk of hearing impairment are 2.36% and 28.40% for office workers and factory workers, respectively, with different model equations. The average estimated excess risk of hearing impairment was the highest with American Academy of Otolaryngology (AAO, 1979) [23] model for office workers. This model calculates the average excess risk of hearing impairment at audiometric test frequencies of 0.5, 1.0, 2.0 and 3.0 kHz. However, the average estimated excess risk of hearing impairment was the highest with NIOSH 1998 model [22] for factory workers. This model calculates the average excess risk of hearing impairment at audiometric test frequencies of 1.0, 2.0, 3.0 and 4.0 kHz. The NIOSH 1998 [22] model is more sensitive to high frequency (3.0 and 4.0 kHz) noise. The detailed audiometric analysis of the subjects of office workers indicated that 50.0% subjects of this group had no excess risk of hearing impairment at all. However, the remaining 50.0% subjects although showed sign of hearing impairment, but that too was negligible, and maximally 2.36% could only be recorded. The average estimated excess risk of hearing impairment with different models ranged from 24.67% - 28.40% for factory workers and 0.68% - 2.36% for office workers (Table 2).

Table 2. Estimated excess risk of incurring hearing impairment by the office workers and factory workers (n=24).

<table>
<thead>
<tr>
<th>Procedural Model</th>
<th>Frequencies (kHz)</th>
<th>Average excess risk (%) (Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AAO (1979)</td>
<td>0.5,1,2,3</td>
<td>2.36 (0-16.21)</td>
</tr>
<tr>
<td>AAOO (1959)</td>
<td>0.5,1,2</td>
<td>0.95 (0-7.22)</td>
</tr>
<tr>
<td>NIOSH (1972)</td>
<td>1,2,3</td>
<td>0.68 (0-3.93)</td>
</tr>
<tr>
<td>NIOSH (1998)</td>
<td>1,2,3,4</td>
<td>1.91 (0-12.86)</td>
</tr>
<tr>
<td>BSA (2004)</td>
<td>0.5,1,2,4</td>
<td>1.89 (0-11.33)</td>
</tr>
</tbody>
</table>

The audiometric analyses further revealed that, in case of factory workers, peak hearing damage was expected to occur at 4.0 kHz for both right and left ears, while for office workers such hearing damage in both ears was progressively increased from 3.0 kHz to 10.0 kHz and no definite peak frequency could be seen. Although the small sample size limits the power of this study, but data generated strongly suggests that, compared to office workers, estimated excess risk of hearing impairment of the
factory workers is higher due to their high noise exposure level. This emphasizes the need to take certain preventive measures to reduce potential harmful effects of loud noise to factory workers.

3.4. Octave Band Analysis

Figure 3 depicts the octave band analysis of factory noise during day shift. In the present study, recording of equivalent sound pressure level ($L_{Aeq}$) indicated that noise level was ranged between 94.6 and 101.0 dB(A) at the factory, and octave band analysis further revealed that high sound pressure level is mostly predominating particularly at 4.0 kHz frequency. The high sound level present in this frequency can be a major reason for causing noise induced hearing loss, since the threshold of human ear varies with the pitch of the sound, the greatest sensitivity being in the 1.0–4.0 kHz range [43]. Similar observation was made by Bedi (2006) [3] in different sections of two textile plants in Northern India, where he found presence of high sound level in 4.0 kHz frequency [11]. Furthermore, this particular observation finds its support from the Hearing Disability Assessment Report of Ireland (1998), which had indicated that noise induced permanent threshold shift usually occurs first around 4.0 kHz and then progresses to involve adjacent frequencies, and such hearing loss at 4.0 kHz progresses over the first ten years of noise exposure and then tends to stabilize [36].

![Octave band analysis](image)

**Figure 3.** Octave band analysis of the tea processing factory at day shift (between 9.30 to 10.30 A.M and 2.30 to 3.30 P.M.). All data indicate mean of two $L_{Aeq}$ values taken at different time on 4 consecutive days
3.5. Assessment of Hepatic Function

Literature survey has revealed that, in addition to cochlea, noise-induced oxidative DNA and lipid damage was found in brain and liver (which has high metabolic functions) [11]. To elucidate whether liver function was affected by exposure to high noise level [94.6-101.0 dB(A)], we examined two marker enzymes, AST and ALT. Aminotransferases (AST and ALT) are regarded as most sensitive markers of hepatocellular injury since liver is the major site of metabolism [44, 45]. Results of measurements of hepatic function enzymes are depicted in Table 3. Results show that, compared to office workers, activities of both AST and ALT were significantly higher by 44.91% (p<0.01) and 90.00% (p<0.001) respectively in factory workers, suggesting that possibly significant liver damage and alteration in function has occurred in these subjects who were exposed to such high noise level for 8-10 years.

Table 3. Biochemical measurements of liver function enzymes and of different oxidative and antioxidative markers of factory workers and office workers of a tea estate.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Office workers (n = 12)</th>
<th>Factory workers (n = 12)</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST (U/dL)</td>
<td>23.47 ± 2.48</td>
<td>34.01 ± 2.01</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>ALT (U/dL)</td>
<td>15.42 ± 1.26</td>
<td>29.33 ± 2.55</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>NO (µmol/dL)</td>
<td>1001.62 ± 42.27</td>
<td>1639.03 ± 88.82</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>TBARS (nmol/dL)</td>
<td>28.47 ± 2.82</td>
<td>45.15 ± 3.34</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>Cortisol (ng/ml)</td>
<td>90.53 ± 4.03</td>
<td>140.31 ± 4.33</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>SOD (µg/ml RBC lysate)</td>
<td>0.112 ± 0.005</td>
<td>0.184 ± 0.006</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>CAT (First order reaction rate constant/ml RBC lysate)</td>
<td>1106.44 ± 19.63</td>
<td>727.38 ± 35.00</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>GSH (mmol/dL)</td>
<td>6.88 ± 0.24</td>
<td>4.94 ± 0.15</td>
<td>p&lt;0.001</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SE.

3.6. Assessment of Oxidative and Antioxidative Status

Earlier studies clearly have indicated a relation between exposure to noise, oxidative stress responses and hair cell damage for the development of physiopathology of noise-induced hearing loss [12, 40, 46, 47]. Although ROS are normal byproducts of cellular aerobic metabolism, these unstable molecules can impair cellular lipids, proteins and nucleic acids in DNA if the balance of corresponding antioxidants is disrupted [11]. Table 3 shows results of plasma cortisol, NO, TBARS, SOD, CAT and GSH in both
factory and office workers. Results indicate that, compared to office workers, factory workers showed 54.99%, 63.64% and 58.59% significantly higher values respectively for cortisol, NO, TBARS ($p<0.001$). However, values of two antioxidant markers, CAT (34.26%) and GSH (28.20%), were found significantly low in factory workers, compared to office workers ($p<0.001$). On the contrary, the value of SOD, although it is an antioxidant, was found significantly higher (64.73%; $p<0.001$) in factory workers. Results indicate that, in the present study, exposure to high sound level at 4.0 kHz possibly could develop significant oxidative stress in factory workers. Because, it is well known that circulating products of lipid peroxidation and DNA repair represent the oxidative status of the whole body, and they can serve as biomarkers of oxidative stress if elevated above the normal levels [11, 48]. Furthermore, it is well known that an increase in SOD activity causes an increase in the production of $\text{H}_2\text{O}_2$ whereas a decrease in catalase activity reduces the conversion of $\text{H}_2\text{O}_2$ to $\text{H}_2\text{O}$ [49]. Since $\text{H}_2\text{O}_2$ is a more potent oxidant to cause cellular damage [50], an increase in SOD activity and a decrease in CAT activity in the present study suggest that possibly there was an imbalance in between free radical formation and its removal under the stressful working environment in the tea-processing factory. Non-protein thiols like glutathione are one of the important factors of defence mechanism that counteract the oxidative stress [29]. Results of the present study showed that, compared to office workers, there was a lower plasma glutathione level in factory workers, suggesting that this reduction in glutathione level possibly was due to utilization of non-protein thiols by increased oxygen free radicals [51] in these subjects.

According to the compelling theory of noise exposure reactions by Ising and Braun [52], habitual noise produces sympathetic activation with an increase in plasma cortisol level, an adrenal stress hormone. Results of cortisol assay in the present study corroborates well with this suggestion because, compared to office workers, cortisol level was found increased by 55% ($p<0.001$) in factory workers (Table 3), suggesting a noise-induced environmental stress in these subjects.

Table 4 shows simple linear correlation studies between excess risk of hearing impairment (NIOSH, 1998) and stress hormone, prooxidative and antioxidative markers, and hepatic function enzymes. This simple correlation analysis revealed that, in factory workers, noise-induced excess risk estimates of hearing impairment have positive correlation with cortisol, NO, TBARS and SOD as well as with hepatic function enzymes, while negative correlation with CAT and GSH. As the rationale for changes in the level of cortisol, oxidative stress markers and hepatic function enzymes are related to alteration in hearing impairment, most likely these are associated.
Table 4. Simple linear correlation between excess risk of hearing impairment (NIOSH, 1998) and stress hormone, prooxidative and antioxidative markers, and hepatic function enzymes of factory workers.

<table>
<thead>
<tr>
<th>Estimated excess risk of hearing impairment</th>
<th>r value (n = 12)</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortisol</td>
<td>+ 0.65</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>NO</td>
<td>+ 0.58</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>TBARS</td>
<td>+ 0.63</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>GSH</td>
<td>- 0.75</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>CAT</td>
<td>- 0.67</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>SOD</td>
<td>+ 0.72</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>AST</td>
<td>+ 0.65</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>ALT</td>
<td>+ 0.66</td>
<td>p&lt;0.05</td>
</tr>
</tbody>
</table>

4. Conclusions

Despite limitations of this study because of sample size, in summary, it may be proposed that, compared to office workers, the audiometric threshold levels were much higher in factory workers and the average estimated excess risk of hearing impairment also was higher in this group of subjects. It is speculated that generation of increased free radicals possibly was responsible to cause cochlear damage and metabolic changes, which possibly were responsible for excess risk of hearing impairment.

5. Our Recommendations

1. Hearing conservation programme for factory workers.
2. Use of earplugs or earmuffs.
3. Noise-insulated area during work-rest cycle.
4. Regular health checks.
6. References


