ABSTRACT

THE EFFECTS OF CARDIOVASCULAR HEALTH ON HEARING LEVELS AMONG MUSICIANS

By Maribeth DiSalvo

This study was designed to determine if there was a significant relationship among audiometric data, otoacoustic emissions, and cardiovascular fitness levels within a group of noise exposed individuals such as musicians versus a control group of non-musicians. Sixty-six volunteer participants were recruited from Miami University. Data was analyzed using Multivariate analyses to contrast and compare research variables as well as Pearson’s correlation coefficient and linear regression analyses to assess significance between and within groups. No difference in otoacoustic emissions, pure-tones or fitness level was found within and between the subject groups. One significant association was found within the group of musicians concerning the signal to noise ratios of the otoacoustic emissions. Significance was attributed to the absolute signal to noise ratio amplitudes between frequencies at 2000 and 4000 Hz within the musician subject group and the better results of the horn players in comparison to percussionists.
Effects of Cardiovascular Health on Hearing Levels Among Musicians

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By

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CHAPTER I

Introduction

One in ten Americans, twenty-eight million in all, possess some degree of hearing loss (Grey, 2000). The occurrence of a hearing loss is rising due to the fact people are living longer. Genetics determine how one ages, as well as how predisposed one is to a hearing loss. However, environmental injuries to the auditory system can not be ruled out as an independent contributor to a hearing loss. Everyday individuals are exposed to excessive amounts of noise such as loud music, sirens, and machinery noise. Human ears are designed to hear at low sound pressure levels (Grey, 2000). When loud sounds are introduced into the auditory system, the sensory cells or outer hair cells (OHC), which are responsible for transmitting sound energy through the auditory system, become damaged. Once the OHC are damaged, they can never be replaced. As a result, the listeners’ hearing acuity is less precise. The result of this damage is classified as a noise-induced hearing loss (NIHL). NIHL can be a significant contributor to a decline in a person’s quality of life, occupation, and hearing cardiovascular health.

A NIHL may affect speech understanding in the presence of background noise. The listener’s social activities may become frustrating and cause isolation or auditory fatigue for the listener. In an occupational setting, a NIHL may become an occupational hazard to many professions such as telephone operators, dentists, and musicians. An industrial setting with high level of machine noise is typically targeted as an environment that could cause a hearing loss. The gradual hearing loss which occurs due to over-exposure to high
intensity sounds from playing music is termed a music induced hearing loss (MIHL) (Einhorn, 1999). Recent studies have focused on cardiovascular health concerns of the entertainment industry; however, most failed to show MIHL to be of any large population problem (Kibble, 1995).

Kibble (1995) also raised the issue of the validity and risk factors associated with a MIHL. The author proposed evidence stating ‘sound which is pleasing, and therefore less stressful, may also be less damaging medically.’ Similarly, longitudinal studies have not shown a synergistic relationship between hearing loss and music (Rintelmann, Borus, Johnson, & Smitely, 1975). However, Rintelmann et al (1975) was not able to document the use of otoacoustic emission (OAE) testing as an early detector of outer hair cell dysfunction. Otoacoustic emissions were discovered in 1968 by David Kemp. It was not until 1988 that otoacoustic emissions were clinically available. However, testing of inter-octave frequencies for conventional audiometric data was not obtained in Rintellmann et al (1975). If a MIHL does appear in the audiogram, it most often occurs between 3000 and 6000 Hertz (Hall & Bulla, 1999). It should also be noted that variability among subjects has been documented in research concerning MIHL (Kolkhorst, Smaldino, Wolf, Battini, Plakke, Huddleston, & Hensley, 1997). Hall and Bulla (1999) documented a previous history of noise exposure as a protection to an individual’s predisposition to noise damaging affects (Hall & Bulla). Sound conditioning of the auditory system to impulse noise has longitudinal protective factors in preventing a permanent threshold shift (Hall & Bulla).
A MIHL can potentially hinder or end a musicians’ career. A musician requires their hearing to be acute and precise. Einhorn (1999) demonstrated that orchestral musicians are routinely exposed to sound levels ranging between 83-112 dB SPL. After exposure to high levels of noise, there is a decrease in hearing sensitivity. Susceptibility to a MIHL among musicians was found not to be affected by factors such as age, gender, eye color, and smoking (Hall & Bulla); however, potential factors exist which may influence an individual’s susceptibility to a temporary threshold shift (TTS) or MIHL such as exercise and cardiovascular health. A MIHL was assessed in this study by comparing audiometric data, cardiovascular health variables/considerations, and outer hair cell integrity in musicians and non-musicians.
Purpose of the Study

Otoacoustic emissions testing and conventional audiometric data were investigated in this study to demonstrate a systematic pattern among musicians versus non-musicians. It is hypothesized that musicians will demonstrate borderline normal conventional audiometric data, and abnormal outer hair cell functioning, while non-musicians will demonstrate normal results or data consistent with exposure to only leisure noise. The variables within the study consisted of degree of cardiovascular fitness and type of instrument played.

Hypotheses

Null Hypothesis (Ho): There is no relationship between cardiovascular fitness and the amount of MIHL among musicians versus non-musicians, specifically the protective effects on the details of otoacoustic emissions.

Alternative Hypothesis (H₁): There is a significant relationship between cardiovascular fitness and the amount of MIHL among musicians versus non-musicians, specifically the protective effects on the details of otoacoustic emissions.
CHAPTER II

Literature Review

Audiometric Data

Due to prolonged exposure to loud sounds, outer hair cells (OHC) are damaged in a tonotopic arrangement along the basilar membrane. OHC are arranged from low to high frequencies, similar to the strings on a piano. The high frequency OHC are the first to be damaged. A decline in the high frequencies is most identifiable in a MIHL or NIHL. Sounds such as high musical notes, women’s voices, and consonant sounds like /s, t, or f/ become distorted (Grey, 2000). A musician needs to understand the speech spectrum beyond conversational understanding (1000 to 4000 Hertz). Higher frequency sensitivity is needed for a musician to match frequencies over the broad spectrum of tones (Einhorn, 1999). A high frequency hearing loss may lead to a decline in performance due to an inability to monitor playing of the higher pitches. Conventional audiometric data (250 Hertz to 8000 Hertz) is most often used to monitor NIHL; however, in addition to high frequency audiometry, a MIHL may be distinguished by a loss in the upper-high frequency region (8000 Hertz to 20000 Hertz), which are important for musical clarity. It can also be used to demonstrate the innate musical talent of a musician versus a non-musician.

Measurement of hearing sensitivity above 8000 Hertz has shown to provide early detection of a MIHL. Studies performed using high-frequency thresholds from 250 to 18000 Hertz with subjects who were rifle team members and rock band musicians.
reported susceptibility to the frequency ranges above 12000 Hertz. Upper-frequency hearing sensitivity changes were not shown to be highly correlated with conventional audiometric data (Fausti, Erickson, Frey, Rappaport & Schechter, 1981). That is, audiometric data between the frequency regions of 250-8000 Hertz generally did not show early signs of a MIHL. For a musician, a high frequency hearing loss is detrimental to their career; however, exposure to music at high levels is unavoidable due to additional factors such as orchestral position, instrument, and time exposed to high impulse noise (Kahari, Axellson, Hellstrom, & Zachau, 2001).

Kahari, Axellson, Hellstrom, and Zachau (2001) demonstrated the effects of orchestral instruments on hearing sensitivity of classical musicians. The results indicated a greater hearing loss for those percussionist, woodwind, and brasswind players compared to those who played large string instruments. A contributing factor was noted in the study outlining the modernization of orchestral instruments. Current instruments of today are designed to perform at higher sound levels and frequencies. Higher sound pressure levels are present in the orchestral pit. Also, unilateral hearing losses were noted among violinist, flutist, and viola players. The potential hazardous sound pressure levels which the orchestral musicians are exposed to for long periods of time and imbalance of auditory information may attribute to associated MIHL symptoms such as tinnitus, hyperacusis, or distortion (Einhorn, 1999). Pure-tone audiometry may not be a diagnostically sensitive method in earlier identification of MIHL and possible associated symptoms. Otoacoustic emissions (OAE) testing has been shown to reflect alterations in the cochlea and its
transducers (outer hair cells) before the audiogram (Hall, 2002). High-frequency audiometry in combination with OAE testing may prove to be most sensitive to identification of a MIHL in the earliest stages.

**Otoacoustic Emissions**

Many musicians practice or perform four to eight hours a day (Sataloff, 1991). Due to the significant length of time exposed to loud sounds, protective measures have been developed such as plexiglas barriers in front of louder emitting instruments and musician ear plug attenuators. Without the use of attenuators, the amount of exposure for musicians cause the ear to anesthetize itself to the loud music (Schmich, 1994). That is, an individual acclimates to the noise without realizing the damage being done. As a precaution, other physiological measures should be used to monitor the longitudinal damaging effects of music on hearing.

Research has shown that OAE testing can detect outer hair cell damage before a significant hearing loss is present (Arnold, Lonsbury-Martin, & Martin, 1997). A person who demonstrates to have normal hearing with pure-tone testing may show a loss of sensitivity through OAE testing. In noise, some individuals with normal hearing have difficulty understanding speech or the clarity of tones; OAEs give supporting evidence to a non-organic cause of a problem.

Otoacoustic emissions testing is specifically used to evaluate outer hair cell integrity. When neural stimulation is transduced through the auditory system, the outer hair cells of
the cochlea shorten and lengthen in response to the stimulation. The amount of motility the outer hair cells possess are important for normal functioning of the cochlea. The stereocilia (sensory cells) activate the outer hair cells to act in two ways, an excitatory and inhibitative state. The otoacoustic emissions are the results of the sheering action of the outer hair cells to sounds (Hall, 2001).

Evoked OAEs measure how well the cochlea is responding and producing the sound back through the auditory system to the measuring probe microphone may be assessed through a type of otocoustic emissions referred to as evoked otoacoustic emissions. Evoked otoacoustic emissions are low level, very soft/faint sounds reproduced by the cochlea in response to sound. Stimuli (two different tones) are presented into the ear canal, conducted to the middle ear and presented to the outer hair cells of the cochlea. When the outer hair cells are stimulated, the two original stimuli plus a third product/ tone is sent back through the auditory system to the speaker of the probe microphone (Hall). The third resulting distortion product of the two stimuli is referred to as the response of the cochlea (Hall). This type of evoked otoacoustic emissions investigated in this study is called distortion product otoacoustic emissions (DPOAE).

DPOAEs have been shown to detect abnormalities within an isolated area of outer hair cells, as a result, producing greater frequency selectivity (Arnold, Lonsbury-Martin,& Martin,1997) . A study done by Arnold, Lonsbury-Martin and Martin (1997) compared high frequency thresholds with DPOAEs to investigate whether DPOAE levels at low frequencies (1000 to 5000 Hertz) were influenced by ultra-high frequency hearing. Ultra-
high frequency hearing loss due to noise or ototoxic drugs progresses in a tonotopic order along the basilar membrane. The researchers proposed that poor ultra-high frequency thresholds should be accompanied by a reduced number of outer hair cells. Also, increasingly poorer ultra-high frequency thresholds and DPOAEs should eventually affect conventional audiometric thresholds (i.e., 4000 to 8000 hertz) (Arnold et al.).

Changes in metabolism of the stria vascularis can affect DPOAEs (Rebillard, Klis, Lavigne-Rebillard, & Devaux, 1993). The stria vascularis contains endocochlear potential which is a major source of energy for the outer hair cells (Rebillard et al). Research by Rebillard et al (1993) demonstrated the effects of altering cochlear metabolism had on the presence of DPOAE levels and amplitude. Within the study, animals were anaesthetized and artificially ventilated. The animals’ vascular system was induced into a hypoxic state by decreasing the amount of oxygen within the blood. The study found that DPOAE amplitude did not recover when oxygen levels were reestablished within the vascular system. When the blood supply in the stria vascularis was reestablished, overproduction of potassium was found to pump into the scala media and produce pressure on the endolymphatic sac. As a result, the basilar membrane was then displaced on the scala tympani causing the outer hair cell processes to be immobile. The study also demonstrated that an increase in endocochlear potential did not yield an expected DPOAE’s increase in amplitude. Nuhall, Guo, Ren, & Dolan (1997) suggested that the vascular system of the cochlea is separate from the Organ of Corti; therefore, implicating
that the active processes of the outer hair cells are mediated by the auditory sensory pathways.

Professional musicians are exposed to noise daily and for sustained periods of time. Many studies have been conducted that investigate musicians and noise exposure; however, high-frequency audiometry, DPOAEs, and cardiovascular health have not been investigated solely within a population of noise-exposed subjects. In this population, the two variables, noise exposure and activity level, should demonstrate both contributing endogenous and exogenous factors to an increased or decreased hearing sensitivity. The current study is being conducted to exhibit that increased activity level will protect the musicians’ hearing acuity, as well as reduce the occurrence of a permanent TTS.

Cardiovascular Health Variables

The chronic damaging effects of a MIHL require a need for preventative measures. Cardiovascular Health concerns regarding hearing have been examined to assess a positive causal relationship between noise and exercise. Exercise and NIHL have been studied to assess whether physical fitness helps protect the cochlea from a NIHL, there is no significant difference, or hearing decreases because the cochlea is sensitive to reduced blood flow.

A study was conducted to define whether hearing sensitivity was influenced by cardiovascular fitness (CV) and muscle strength (Manson, Alessio, Cristell, & Hutchinson, 1993). Researchers found that an increase in CV increased hearing acuity.
Above average VO\textsubscript{2} maximum capability, which is defined as the point of maximum capacity for oxygen production before fatigue occurs, was theorized to be a contributor to a more enhanced circulatory system and better hearing. Muscle mass and strength was not a strong indicator of increased hearing sensitivity but was determined to be an added contribution to increased blood flow within the circulatory system.

Horton, Geer & Stuart (2001) investigated noise, exercise, and the combined effects of noise and exercise on measurements of DPOAEs and audiometric data. The premise underlying the study was to provide evidence that the cochlea is protected during physical fitness. Exogenous factors such as activity level and endogenous factors such as age and gender were compared to see if a relationship existed between subject DPOAE amplitudes. In this study, a noise induced TTS was not significantly increased when subjects engaged in physical activity; however, the study failed to find a synergistic relationship between exogenous and endogenous factors between subjects.

A TTS occurs when the action potential of the nerve fibers are reduced due to less oxygen uptake. The inhibitory effect of the efferent system is then suppressed, which indirectly affects the afferent system. A similar metabolic cause and effect, which occurs for the absence or reduced amplitude of DPOAEs. Recovery from a TTS was found to occur when metabolic capabilities were reestablished (Hawkins, 1971).

A TTS after continual exposure to noise can result in a permanent loss in hearing. It has been documented that seven hours of exposure to sound levels between 85 and 90 dBA SPL causes a TTS in the frequency regions of 3000 to 6000 Hertz (Hall & Bulla,
1999). Current research has investigated prevention of a permanent TTS by holistic measures (Henderson, 2001).

Henderson (2001) explored the role of antioxidants in preserving hearing from noise. Experiments were done on guinea pigs examined the effects of high level impulse noise on strial blood flow. When the guinea pigs were exposed to high levels of noise, there was a sharp reduction in cochlear blood flow and an increased level of free radical generation. Two hours after exposure, cochlear blood flow returned to normal, as well as, free radical activity. High levels of noise were shown to create damaging free radicals, which are potentially toxic to an individual’s hearing level. Within this study, another experiment investigated the protective effects of antioxidants on a chinchilla exposed to profound levels of noise and then treated with an ototoxic drug. The results indicated less of a TTS and hair cell loss in comparison to the control groups.

The relationship between metabolic activity and/or biophysiological capabilities within the body have been researched in combination with increased hearing acuity (Ismail, Corrigan, MacLeod, Anderson, Kasten, & Elliot, 1973). Ismail, Corrigan, MacLeod, Anderson, Kasten, & Elliot (1973) study involved an eight month exercise program done to investigate biophysiological variables such as, heart rate, blood pressure, submaximal oxygen uptake, serum levels and their effects on present audiological levels. The research attempted to prove a causal relationship between improved cardiovascular health variables and increased audiological results. The results of the experiment indicated significant changes biophysically; however, no significant pure-tone threshold
improvements occurred for the frequency ranges of 2000-14000 Hertz (Ismail et al.). However, TTS measurement taken after the eight month program demonstrated significant improvements among the subjects. On the basis of similar studies, the improvements may primarily be due to increased threshold for stress, increased metabolism and blood flow. The study proved that increased activity level demonstrated improvement in both the metabolic and circulatory systems, which are both essential to support cochlear functioning.

Previous research has confirmed metabolic changes within the Organ of Corti during noise exposure (Hamberger & Hayden, 1971). Other research supporting Hamberger and Hayden’s study demonstrated that reduced oxygen delivery to the cochlea changed endolymph and perilymph composition and caused constriction of the stria vascularis (Hawkins, 1971). With prolonged exposure to noise, strial atrophy was observed.

Noise is known as a psychological/physiological stressor (Lindgren & Axelsson, 1988). Studies have documented that noise induces an increased heart rate and blood pressure, as well as, reduced hearing sensitivity (Lindgren & Axelsson). Physiologically, vasoconstriction of the stria vascularis may prove to be most responsible for the decreased hearing sensitivity (Kolkhorst, Smaldino, Wolf, Battani, Plakke, Huddleston & Hensley, 1997). Vasoconstriction of the stria vascularis has shown to cause damage to the cochlea due to inhibited oxygen production and blood flow (Hawkins, 1971). During the onset of exercise, vasoconstriction of the stria vascularis has shown to occur, as well as, the stapedial reflex response (Colletti, Fiorino, Verlato & Montresor, 1991).
The stapedius muscle is responsible for damping loud sounds which enter into the auditory system. Colletti et al (1991) proved that physical exercise depresses the stapedial reflex. The theory is similar to the physiological response to stress and noise. The stapedial reflex protects the OHC and the cochlea from noise damaging effects. The study by Colletti et al. (1991) also demonstrated that physical exercise decreased the effects of a noise induced temporary threshold shift (TTS). It was hypothesized that increased blood flow/ temperature and metabolic changes such as, increased potassium production, lactate in the muscles, and decreased free radicals were contributing factors to the decreased TTS. The results of the study indicated the greatest stapedial reflex attenuation to be at 8000 Hertz. Other studies outlining the importance of the stapedial reflex in combination with reducing the effects of a TTS have only shown significant results in the lower frequencies (Zakrisson, 1974), therefore, the metabolic considerations in reducing a TTS can not be entirely disregarded.
CHAPTER III

Method

Subjects

There were sixty-six volunteer participants (35 males and 31 females) ranging in age from 19 to 25 participated in this study. All subjects were recruited from the Miami University campus. The subjects were placed into two groups, non-musicians and musicians. One group consisted of 33 musicians, and the other group consisted of 33 non-musicians. Age and gender data were obtained for each subject and are depicted in Figures 1 and 2.

Using the University of Houston Non-Exercise Assessment Test (Ross & Jackson, 1990), musicians and non-musicians were asked to describe their general physical activity level on a rating scale of 0 to 7. 0 rating ranging from those subjects who did not participate in recreation and/or physical activities, and 7 describing individuals with regular participation in heavy physical exercise and/or vigorous aerobic activity.

Inclusion & Exclusion Criteria

All subjects used within this study were 19 to 25 year old college males and females. Following an otoscopic examination and tympanometry, subjects within the study were found to have normal middle ear functioning.

Musicians used within this study were vocalist and instrumental players and practice 5 or more hours per week. Their mean activity level was rated on a scale of 0-7 as a 3, which is described by one hour of exercise per week. Consistent with this criteria,
musicians who played or practiced less than 5 hours per week and engaged in no physical exercise per week were excluded from this study.

The non-musicians used for the study reported that they were exposed to noise less than 2 hours per week and rated their mean activity level on a scale of 0-7 as a 5, which is characterized by running 1-5 miles per week or spending 30 to 60 minutes per week in comparable physical exercise. In addition, those subjects with more than 2 hours of noise exposure per week and an activity rating of 1, were excluded from this study.

Volunteers with middle ear pathology or abnormal functioning and those younger or older than 18 to 25 years of age were not permitted to participate in this study. Normal middle ear functioning is defined as type A tympanograms with tympanometric width <110 daPa, equivalent volume <1.5 cm³ and static compliance of >0.30 mmho (Hunter and Margolis, 1992).

Subject Consent
Mandated by the Committee on the Use of Human Subjects in Research at Miami University, subjects were given a consent form prior to testing which outlined the purpose and procedures of the study. Participants were informed of their right to withdraw from the study at any time.

Procedure
This research study consisted of five evaluation components: 1.) questionnaire; 2.) tympanometry; 3.) pure-tone hearing screening; 4.) submaximal VO₂ cardiovascular function testing; and 5.) evoked and spontaneous otoacoustic emissions testing.
A questionnaire was completed in order to obtain a short case history of each subject's hearing, health, and noise exposure (Appendix A). Tympanometry was assessed using a Madsen GSI tympanometer to measure middle ear function for each ear. Conventional audiometry data were assessed across the frequencies of 250 to 8000 Hertz in octave intervals using insert phones on a Grason Stadler model audiometer. Each subject in both groups had hearing thresholds at 25 dB HL or better. Cardiovascular fitness levels were determined by submaximal VO\textsubscript{2} testing. Subjects’ weight, height, body fat percentage, and blood pressure levels were established. Subjects exercised on a Monark Bicycle ergometer. Heart rate was monitored continuously with a Polar monitor as the subjects exercised to approximately 70% of the maximum effort. A predicted measurement of submaximal VO\textsubscript{2} levels were obtained based on the University of Houston Non-Exercise Test when actual submaximal VO\textsubscript{2} levels were not available.

Otoacoustic emissions are echoes or vibrations of the cochlea in response to non-linear movements of the outer hair cells due to sound stimulation. The intensity of otoacoustic emissions generally indicates the health or status of the cochlea (Kemp, 2003). Therefore, otoacoustic emissions do not quantify hearing loss but only detect the presence of hearing (Kemp, Bray, Alexander, & Brown, 1986). Two types of OAEs were examined: 1.) spontaneous otoacoustic emissions and 2.) evoked otoacoustic emissions. Spontaneous otoacoustic emissions (SOAE) are signals that occur in the ear without stimulation. SOAEs were assessed in this study to supplement DPOAE measurements. In certain cases, SOAE measurements may need to be subtracted from DPOAEs amplitude;
however, the presence or absence of SOAE can not be said to have any clinical significance (Kemp et al). Evoked otoacoustic emissions (EOAE) reflect the active processes associated with the outer hair cells (Attias & Bresloff, 1996). There are two types of EOAE: 1.) distortion-product otoacoustic emissions (DPOAE) and 2.) transient evoked otoacoustic emissions (Hall, 2001).

Distortion-product otoacoustic emissions and the environmental noise floor sound pressure levels were measured using a Madsen Celesta 503 Cochlear Emission Analyzer (Taastrup, Denmark). The analyzer was coupled to the IBM 486 (model number TS30MS) PC (Armond, NY) with RS232 cables to produce 2 simultaneously presented tones at the test frequencies of 1000 Hz, 1500 Hz, 2000 Hz, 3000 Hz, and 4000 Hz. Measurements were obtained at sound pressure levels of f1 (55 dB SPL) and f2 (65 dB SPL) and a f2/f1 ratio of 1.22. Research has indicated the separation in the range of the primary tone levels (f1 and f2) will be more sensitive in identify damage as a result to a noise-induced hearing loss (Lonsbury-Martin & Martin, 2003).

The DP-gram was also used to assess the signal to noise ratio in DPOAE measurements. The DP-gram pictorially represents the signal to noise ratios (SNR) at F1, F2, and 2F2-F1 at all of the test frequencies. The SNR is a result of acoustical sampling of environmental low frequency energy in the test surroundings minus the response of the test frequency within the cochlea. The noise floor is the amount of background noise that occurs in proximity to the test frequency at the time of recording (Robinette & Glattke, 1997). An emission is stated as being present if the amplitude of the emission is greater
than that of the noise (Robinette & Glattke). An increase in the SNR produces clearer and reliable DPOAE responses. To attain a more enhanced SNR, the higher primary level intensity should be established (Zhao et al.). The SNR was assessed within this study to evaluate the effects of noise exposure on the finer details of DPOAE results. Testing was performed within a sound-treated room. Before testing started, subjects were asked not to vocalize or move during testing.
Figure Captions

Figure 1: Demographic data for age in years

Figure 2: Demographic data based on gender distribution
Figure 1
Figure 2

Gender Distribution

Male 55%
Female 45%

Age Distribution

Age 18 19 20 21 22 23 24 25 26 27
Frequency 2 4 6 12 18 6 2 2 2 2

Male
Female
CHAPTER IV

Results

There were sixty-six volunteer participants within this study. Descriptive statistics of the musicians indicate an average of 11.19 years (a range of 6-17 years) of noise exposure. Conventional audiometric testing (250 Hertz to 8000 Hertz) demonstrated pure-tone average hearing levels to be 7dB HL for the musicians and 8dB HL for the non-musicians. Pure tone averages fell within the normal range based on the normal bell-shaped curve for distribution. Mean pure-tone averages for both the non-musicians and musicians are represented in Figures 3 and 4.

The activity level of the musicians spent a mean of one hour of exercise per week. Conversely, non-musician subjects mean number of hours engaged in physical activity was characterized by running 1-5 miles per week or spending 30 to 60 minutes per week in comparable physical exercise. Musicians and non-musicians rated their activity level on a scale of 0-7 to be within a range of 3 and 5. Out of the 66 participants, 11 subjects within the study reported smoking on a regular basis, while 5 out of 33 the non-musicians reportedly smoke on a regular basis. Of both the non-musicians and musicians groups, 52% of the musicians and 70% of the non-musicians reported having experienced tinnitus, monaurally or bilaterally. Descriptive statistics of the covariants used within this study are provided in Table 1. There were 6 infrequent smokers and one heavy smoker (≥ 1 pack per day) in the musician group; five nonmusicians indicated heavy smoking habits and one nonmusician reported occasional use. Given the small number and varying
degrees of smoking within each group, no further data analysis was conducted to compare results between nonsmokers and smokers within groups.

A Multivariate analysis of distortion product otoacoustic emissions, distortion product signal-to-noise ratios, and mean pure tones from the data are shown in Figure 5. Mean pure tone thresholds (500 to 4000 Hertz) for each ear were used in the analysis. The MANOVA test demonstrated no significant differences in the means between musicians and non-musicians with regard to distortion product otoacoustic emissions and mean pure-tone levels. As a result, the descriptive statistics involving distortion product otoacoustic emissions, pure-tone levels, and/or in comparing significance among and between the non-musicians and musicians groups are evaluated simultaneously for the left and right ears. A multivariate analysis was repeated within the musician group to see if any difference co-existed dependent on the type of instrument played (Table 2.). A significance at the alpha level of .05 was evident within the musicians group concerning the signal to noise ratios among DPOAEs. Bonferroni follow-up comparisons indicated a better SNRs among the horns compared to the percussionists (p=.03). Also, SNRs were better for all musicians at 4000 Hz in comparison to 2000 Hz (p<.05).

Fitness activity levels of the subjects were analyzed through either a predicted or actual analysis of submaximal VO\textsubscript{2} levels. Each subjects’ predicted maximal VO\textsubscript{2} was calculated to serve as a measure of their level of cardiovascular fitness, however, only ten of the sixty-six subjects participated in an actual VO\textsubscript{2} max measurement test. A scatterplot of the actual VO\textsubscript{2} max measurements versus the predicted VO\textsubscript{2} maximal
measurements are shown in figure 5. As evidenced by the results, there does not appear to be any significant correlation. The Pearson correlation coefficient for these measurements is -0.450 with a p-value of 0.175, indicating no significant linear correlation exists between the actual versus the predicted analysis.
Figure Captions

Table 1: Descriptive variables of two subject groups

Table 2: P-Values for MANOVA comparing musicians versus non-musicians distortion product otoacoustic emissions and pure-tone levels

Table 3: P-Values for MANOVA comparing instruments and the effects on distortion product otoacoustic emissions and pure-tone levels with-in the musician group

Figure 3: Mean pure-tone averages of non-musicians

Figure 4: Mean pure-tone averages of musicians

Figure 5: Actual VO2 levels versus predicted VO2 levels between musicians and non-musicians
Table 1

*Descriptive variables of two subject groups*

<table>
<thead>
<tr>
<th></th>
<th>Musicians</th>
<th>Non-Musicians</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (Mean)</strong></td>
<td>21.2</td>
<td>22.2</td>
</tr>
<tr>
<td><strong>Smoking</strong></td>
<td>18%</td>
<td>15%</td>
</tr>
<tr>
<td><strong>Years of Exposure (Mean)</strong></td>
<td>11</td>
<td>0%</td>
</tr>
<tr>
<td><strong>Tinnitus (% present)</strong></td>
<td>70%</td>
<td>52%</td>
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</tbody>
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Table 2

*P-Values for MANOVA Comparing Musicians and Non-Musicians*

<table>
<thead>
<tr>
<th>Variable of Interest</th>
<th>P-Value of MANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distortion Product Otoacoustic Emission</td>
<td>0.84</td>
</tr>
<tr>
<td>Pure Tone Thresholds</td>
<td>0.12</td>
</tr>
<tr>
<td>Signal to Noise Ratio</td>
<td>0.44</td>
</tr>
</tbody>
</table>
Table 3

*P-Values for MANOVA Comparing Variables within Musicians*

<table>
<thead>
<tr>
<th>Variable of Interest</th>
<th>P-Value of MANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distortion Product Otoacoustic Emission</td>
<td>0.19</td>
</tr>
<tr>
<td>Pure Tone Thresholds</td>
<td>0.58</td>
</tr>
<tr>
<td>Signal to Noise Ratio</td>
<td>0.05*</td>
</tr>
</tbody>
</table>

*significant at the .05 alpha level

Figure 3

*Non-Musicians Pure-Tone Average*
Figure 4

Musicians Pure-Tone Average

![Musicians Pure-Tone Average](image)

Figure 5

Scatterplot of Actual versus Predicted VO2 Max

![Scatterplot of Actual versus Predicted VO2 Max](image)
CHAPTER V

Discussion

The main goal of a hearing conservation program is to detect the primary stage of initial noise induced damage to the auditory system. Research has demonstrated the OHCs are the first to be affected by noise exposure (Vinck, Van Cauwenberge, Levoy, & Corthals, 1999). Therefore, an alternative approach to behavioral detection of an MIHL is an objective means of detection using otoacoustic emissions testing. This study was designed to assess the relationship between cardiovascular fitness, hearing acuity, and DPOAE amplitudes in musicians and non-musicians with no significant history of noise exposure.

The study hypothesized that musicians would demonstrate abnormal DPOAE amplitudes and non-musicians would demonstrate normal DPOAE amplitudes. In this study, there was no difference in DPOAE amplitudes between the nonmusicians and the musician group. The pure tone levels were similar between the two subject groups. Within the group of musicians, the analysis revealed a significant difference in the signal to noise ratios between the two frequencies of 2000 and 4000 Hertz. No differences were detected between pure tones and DPOAEs between instruments in the musician group. The variability for mean pure-tone levels between the different types of instruments in the musician group and within the non-musician group precluded any type of statistical difference (Figures 3 and 4).
A NIHL or MIHL on average is bilateral and affects the higher frequencies of hearing (Attias, Horovitz, El-Hatib, & Nageris, 2001). When DPOAEs are presented to the auditory system, a notch is found around 3000 Hertz, which closely resembles a hearing configuration of a noise exposed individual (Attias et al, 2001). Results of the DPOAE measurements from the current study reveal no significant findings between 1000 to 4000 Hertz between the two groups. However, DPOAE measurements at 3000 Hertz did indicate abnormally low emission amplitudes among three of the twelve individuals in the musician group who only experienced a slight loss in pure-tone hearing levels. For any test frequency, there is overlap in DPOAE data among those with normal hearing and hearing loss subjects (Hall, 2000). Figure 6 demonstrates the results of both the musicians and non-musicians absolute amplitudes DPOAEs in comparison to the normal adult range (Gorga, Neely & Ohlrich, 1997).

The signal to noise ratio amplitudes within the musician group were found to be significant (p<.05). For estimation of normal hearing, the average signal to noise ratio necessary is 6 dB greater than the noise (Robinette & Glattke, 2002). Mean SNRs vary between 19 and 30 dB at frequencies above 1000 Hz in clinically normal ears (Robinette & Glattke, 1997). The signal to noise ratios within the group of musicians were similar compared to the signal to noise ratio amplitudes of non-musicians in both ears (Figures 7 and 8). However, the standard deviations were larger at each frequency for the nonmusician group (Table 5); thus detection of statistical difference becomes more difficult. Within the musician group, the only statistical difference by instrument was the
found between the horn players in comparison to the percussionists (Figures 9 and 10). The horn players had better average SNRs compared to the percussion musicians. Use of SNRs in evaluating DPOAE test performance (sensitivity and specificity) is stronger incorporating SNRs in test measurements (Hall, 2000). The correlation of signal to noise ratios and otoacoustic emission efferent suppression are researched in the literature (Perrot, Micheyl, Khalfa, & Collet, 1999). Increased efferent activity was found to enhance signal to noise perceptions within musicians versus non-musicians.

Kahari, Axellson, Hellstrom, & Zachau (2001) found their results to indicate a greater hearing loss for those percussionists, woodwind, and brasswind players. Also, the co-occurrence of tinnitus among these musicians and others were found within this population (Einhorn, 1999). Table 6 depicts the frequency of these instruments within the present study. Our SNR results were consistent with Kahari et al (2001) within the musician group and similar to Einhorn (1999); subjects within both the experimental and control groups reported suffering from tinnitus (70% musicians and 52% non-musicians). The high occurrence of tinnitus within the non-musicians group speculates that leisure noise has had damaging effects on their auditory systems.

The efferent system’s role within the auditory system was proposed as a protector for the ear in noisy environments (Michie, LePage, Solowij, Haller, & Terry, 1996). In the human cochlea, there are two distinct parallel pathways, the afferent and efferent sensory pathways. Evidence confirms that the efferent system plays a significant role in outer hair cell physiology. However, it is difficult to discuss the efferent systems role solely
without mentioning the corresponding afferent systems added contribution. The afferent fibers are responsible for innervating 90 to 95% of the inner hair cells and only 5% of the outer hair cells. However, physiological evidence suggests that movement or innervation of the outer hair cells does influence inner hair cell motility. The active processes of the outer hair cells are directly related to movement of the tectorial membrane. OHC are embedded within the tectorial membrane along the Organ of Corti. In contrast, the inner hair cells are in the proximity, but not in direct contact of the tectorial membrane. When sounds enter into the auditory system and cause excitation or inhibition of the outer hair cells, the movement of the outer hair cells is likely to influence the position of the tectorial membrane and enhance the inner hair cells sensitivity (Stach, 1998).

The final pathway for outer hair cell innervation occurs within structures which are medial to the superior olivary complex (Hall, 2000). The superior olivary complex is the site for binaural interaction of information received from both ears. Two responses may occur within the superior olivary complex, contralateral and ipsilateral stimulation. Excitation or inhibition occurs within structures of the superior olivary complex referred to as the lateral and medial superior olive. Collectively, these structures are called the olivocochlear bundle. With a contralateral input, excitation occurs within the lateral superior complex, and ipsilateral stimulation causes inhibition of neurons within the medial superior complex. In comparing the responses to an intensity of a signal of one ear versus that of the other, it allows the suppressive affects of the bilateral auditory system to be analyzed.
Perrot, Micheyl, Khalfa, & Collet (1999) discovered that musicians possessed stronger bilateral efferent suppression capabilities versus non-musicians. This finding was in contrast to Engdahl’s study (1996) stating that the sensitivity of the outer hair cells to noise depends more on the amount of OAE saturation than to the amount of efferent activity. However, the study by Perrot et al. (1999) highlighted musicians and proposed that they require more central auditory stimulation than do non-musicians. Also, non-musicians possess a more general asymmetry or lateralization in sensory strength than musicians. The bilaterally enhanced efferent system were found to be greater in each ear for musicians versus non-musicians (Perrot et al.). Increased efferent activity was also found to enhance signal to noise perceptions and auditory fatigue in musicians. The study hypothesized that suppression was related to protection of outer hair cell damage. As a result, efferent suppression may be an earlier indicator of a NIHL before any structural damage is found in the audiogram (Desai, Reed, Cheyne, Richards, and Prasher, 1999).

It was speculated that cardiovascular fitness might protect hearing and distinguish musicians, in particularly, those who may be at higher risk than average for a MIHL. The predicted VO₂ maximal values were not significantly different between the musician and nonmusician groups in the current study. Hutchinson, Alessio, Hoppes, Gruner, Sanker, Ambrose, & Rudge (2000) found fitness and hearing to be specifically related to cardiovascular fitness; the premise being that high cardiovascular fitness levels are associated with increased circulatory capabilities within the vascular system. This study investigated whether increased cardiovascular fitness protected sensory function within
the cochlea of noise-exposed subjects such as musicians. The musicians in this study demonstrated mean VO$_2$max levels ranging between 20 to 49 ml/kg/min. According to the American College of Sports Medicine guidelines for exercise testing and prescription (1986), the musicians in the study fell within the fair to average fit levels for individuals under 29 years of age. The use of the means and standard deviations of VO$_2$ submaximal values are also depicted in Table 6.

The validity of the University of Houston Non-Exercise Test, which was used in predicting VO$_2$Max levels, is documented in the literature(Jackson & Ross, 1990). Estimation of VO$_2$ Max from submaximal methods is a feasible alternative to direct measurement of VO$_2$ Max. Subjects in each group were asked to estimate their weight, height and amount of regular exercise. As young adults, these individuals may not have been straightforward in their responses. For optimum accuracy, the test requires trained technicians and appropriate equipment, which can correctly assess cardiovascular function (Jackson & Ross, 1990). The present study involved senior level undergraduates and first year graduate students. Additionally, the equipment used in the current study to obtain submaximum fitness levels may result in higher standard error values when estimating VO$_2$max levels (Jackson & Ross). Figure 5 demonstrates the correlation between the University of Houston Non-Exercise model and the actual cardiovascular fitness test. There is some clustering of values, but no significant correlation between the two measures was found (p>.05).
Vasoconstriction of the stria vascularis was speculated as having a direct relationship in noise-induced hearing loss (Hawkins, 1971). Changes in the Organ of Corti due to high impulse noise have been suggested as contributing factors. Metabolic stress and the presbycusic effects on the auditory system have been documented in the research (Johnssan & Hawkins, 1972). In the aging ear, as well as when impulse noise occurs, the stria vascularis has shown to cause damaging effects on the cochlear mechanisms (Johnssan & Hawkins). Sanden and Axelsson (1981) reported a temporary hearing loss to occur when either blood cholesterol and/or heart rate were elevated. Thus, suggesting decrease circulation within the inner ear is indirectly caused by circulatory shifts caused by exercise. Musicians within this study did not exhibit a MIHL through abnormal DPOAEs or pure-tone averages. Also, age effects were not an issue due to the age within subject groups. As expected, we did not find abnormal pure-tone means in musicians, however, higher, less variable SNRs were found within the musician group.

A study by Oeken, Lenk, & Bootz (2000) researched the controversy between age effects and pure-tone averages as being contributing factors in increased amplitudes of DPOAEs. In the present study, pure-tone averages did not reveal a synergetic relationship between or within the musicians. The DPOAE amplitude means across the frequencies and the pure tone averages were not statistically different between groups. The type of instrument played was also not a factor in amplitude values of DPOAEs (Table 3).
Summary

The implications of a MIHL are very important to performing artists, especially musicians and vocalists. The literature stresses the importance of ear protection and hearing conservation programs to help protect or stop further damage to musicians hearing. DPOAE amplitudes were poor among several musicians in comparison to normal pure tone thresholds. The SNRs further defined differences by frequency and instrument. The results of this study indicated that cardiovascular fitness was not associated with protection against MIHL in musicians; however, this study attempted to demonstrate further protection from a MIHL through general increased cardiovascular health. The study hypothesized that increased cardiovascular health, characterized by a high VO$_2$max level, and would yield increased circulation and metabolic capabilities both of which are important in supporting cochlear function. Although our results did not demonstrate significant findings by cardiovascular fitness variables, further research is needed to facilitate an early method of identification and susceptibility to a MIHL. A musicians hearing requirements are not only detrimental to everyday conversation and listening environments but to their livelihood.
Limitations of the Study

A limitation of this research involves the amount of experimental subjects obtained within each subgroup of musicians. It was difficult to demonstrate significance with only one or two subjects’ representing a whole subgroup of instruments. There were many extraneous factors to guard against such as activity level of that individual, height, weight, and/or a significant history of noise exposure in comparison to the normative group. The data was not able to yield significant effects because of the low statistical power.

Some of the subjects used in this investigation participated in the exercise portion of the research protocol. In doing this, the researchers were able to assess the subjects’ actual $\text{VO}_2$ maximal level. Without an exercise test, a predicted $\text{VO}_2$ maximal level was calculated from the subjects estimated height, weight, and age, which may be a less precise indication of the cardiovascular health/activity level of the subjects.

A major limitation within the research was the limited amount of control the experimenter possessed over the amount of noise exposure the control group has experienced. As stated previously, the world of today is a noisy environment. It would be impossible if not unrealistic to attempt to find individuals without any exposure to hazardous amounts of noise. A major task for future research would be to have better protocol requirements estimating the experimenters’ allowable amount of previous history of noise exposure within the control group.
Future Research

Research concerning efferent suppression and submaximal levels within the older population may be of interest in future research concerning the details of otoacoustic emissions and noise-exposure. The presbycusics effect on the auditory system has been well documented yet additional information regarding the sensory pathways among musicians is of further interest.
Figure Captions

Table 4: Means and Standard Deviations For Non-Musicians Signal to Noise Ratios

Table 5: Frequency of Instruments for Horns, Woodwinds, and Percussionists

Table 6: Means and Standard Deviations of Predicted VO₂MAX Levels

Figure 6: Musicians and Non-Musicians DPOAEs in Comparison to the Normal Range

Figure 7: Mean Signal to Noise Ratio Amplitudes for the Left Ear

Figure 8: Mean Signal to Noise Ratio Amplitudes for the Right Ear

Figure 9: Distribution of Instruments

Figure 10: Non-Musicians Signal to Noise Ratios at 2000 and 4000 Hertz
Table 4

*Means and Standard Deviations For Non-Musicians Signal to Noise Ratios*

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Signal to Noise Ratio at 2,000 Hz.</td>
<td>8.95</td>
<td>9.32</td>
</tr>
<tr>
<td>Signal to Noise Ratio at 4,000 Hz.</td>
<td>16.83</td>
<td>12.40</td>
</tr>
</tbody>
</table>

Table 5

*Frequency of Instruments for Horns, Percussionists, and Woodwinds*

<table>
<thead>
<tr>
<th></th>
<th>Horns Frequency</th>
<th>Percussionists Frequency</th>
<th>Woodwinds Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tuba</td>
<td>4</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Trombone</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Euphonium</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Trumpet</td>
<td>7</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>French Horn</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>16</td>
<td>*8</td>
<td>6</td>
</tr>
</tbody>
</table>

*Vocalists (n=3) were excluded from this chart*
Table 6

*Means and Standard Deviations of Predicted VO$_2$MAX Levels*

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Musicians</td>
<td>42.01</td>
<td>9.25</td>
</tr>
<tr>
<td>Non-Musicians</td>
<td>41.61</td>
<td>6.75</td>
</tr>
</tbody>
</table>

Figure 6
Figure 7

Signal-to-Noise Ratio Left Ear

Mean Signal-to-Noise Ratio Across Amplitude

Figure 8

Signal-to-Noise Ratios Right Ear

Mean Signal-to-Noise Ratio
Figure 9

![Instruments Played](image_url)

The bar chart shows the number of subjects who played various instruments. The instruments listed include Trombone, Vocalist, Cymbals, Euphonium, Flute, Piano, Clarinet, Trumpet, Viola, Percussion, Violin, French Horn, Saxophone, and No Instrument. The chart indicates that the majority of subjects played No Instrument, with no significant number playing the other instruments.
Figure 10

[Bar chart showing mean signal-to-noise ratio (SNR) for different instrument types with three frequency bands: SNF 2kHz, SNF 3kHz, SNF 4kHz, for the right ear.]
References


Appendix 1

Research Questionnaire

Name _____________________________
Date ______________________________
Birthdate __________________________ Age __________________
Address ______________________________________________________________
Home Phone _______________________ Work Phone _____________________
Occupation ____________________________________________________________

General Information

Section 1:
Do you smoke? Yes ____________ No ____________
If so, how often? ________________________________________________________
Have you ever had major surgery? Yes ____________ No __________________
If so, please list date and type ____________________________________________
Have you ever had a head injury? Yes ____________ No __________________
Do you have a family history of hearing loss? Yes ____________ No ____________
If so, please explain: ____________________________________________________
How long has it been since you were last exposed to loud noise, music, etc.? ____________

Section 2:
Are you a musician? Yes ____________ No ____________
(If no, skip the rest of the questions in this section)
What instrument do you play? (drums, guitar, sing, etc.) _________________________
How long have you been playing? ____________________________________________
Approximately how many hours are you exposed to loud music per week? ________

Do you wear ear protection while exposure to loud music? (rate yourself on how often
you wear hearing protectors on a scale from 1 to 4) 1= most of the time, 2= less than
half of the time, 3= seldom, 4= never ________________________________________

Section 3:
(When filling out this section keep in mind the following: loud noise is characterized by the
fact that you would have to raise your voice to be heard by another person when talking to
them from arm’s length away)
Have you had a history of noise exposure? (other than music if you are a musician)
Yes ______________ No ______________
If so, please explain: ______________________________________________________

Are you exposed to noise on the job? Yes ______________ No ______________
If yes, please explain: ______________________________________________________

Are you exposed to loud noise recreationally? (hunting, woodworking, socially, etc.)
Yes ______________ No ______________ If so, please explain: ____________________
________________________________________________________________________
If so, approximately how many hours per week? __________________________________

Do you wear ear protection when exposed to this noise? (rate yourself on how often you
tear hearing protectors on a scale from 1 to 4) 1= most of the time, 2= less than half of
the time, 3= seldom, 4= never ___________________________________________

Hearing
Have you ever had a change in your hearing? Yes ______________ No ______________
(If no, skip the rest of the questions in this section)

Has the change in your hearing been Temporary? ______________ Permanent? ______________

Has the change in your hearing been gradual? ______________ Sudden? ______________
Which ear is your better ear? Right ______________ Left ______________ Both the same ______________
What is the cause of your hearing loss (if known)? _______________________________

Do you hear better in: Quiet? ______________ Noise? _______________________________

Do you hear on the telephone? Yes ______________ No _______________________________

Tinnitus
Do you have a ringing, popping, crackling, or cricket-like noise in your ears? Yes ______________ No ______________
If so, in which ear? Right ______________ Left ______________ Both ______________
(If no, skip the rest of the questions in this section)

Is the ringing continuous? Yes ______________ No _______________________________
Is the pitch of the ringing high? Yes ______________ No _______________________________
Is the pitch of the ringing low? Yes ______________ No _______________________________

Please provide any other information, which might be helpful.