TOWSON UNIVERSITY
COLLEGE OF GRADUATE STUDIES AND RESEARCH

TRANSIENT TINNITUS IN PEOPLE WITH NORMAL HEARING INDUCED BY SOUND DEPRIVATION

by

Jamie Walck

An Audiology Doctoral Thesis

Presented to the faculty of

Towson University

In partial fulfillment

of the requirements for the degree

Doctor of Audiology

May 2007

Department of Audiology, Speech-Language Pathology and Deaf Studies
Towson University
Towson, Maryland 21252

TOWSON UNIVERSITY

COLLEGE OF GRADUATE STUDIES AND RESEARCH

AUDIOLOGY DOCTORAL THESIS APPROVAL PAGE

This is to certify that the doctoral thesis prepared by Jamie Walck, entitled Transient Tinnitus in People with Normal Hearing Induced by Sound Deprivation, has been approved by this committee as satisfactory completion of the requirement for the degree of Doctorate of Audiology (Au.D.) in the department of Audiology, Speech-Language Pathology and Deaf Studies.

_____ _____

Margaret M. Jastreboff, Ph.D. Date
Chair, Audiology Doctoral Thesis Committee

_____ _____

Pawel J. Jastreboff, Ph.D. Date
Committee Member

_____ _____

Diana C. Emanuel, Ph.D. Date
Committee Member

_____ _____

Jin K. Gong, Ph.D. Date
College of Graduate Studies and Research

ABSTRACT

TRANSIENT TINNITUS IN PEOPLE WITH NORMAL HEARING INDUCED BY SOUND DEPRIVATION

JAMIE WALCK

The emergence of silence induced tinnitus perception was studied in 20 normal hearing Caucasian adults. Psychoacoustical characterization of any participants' silence-induced tinnitus was performed after sitting in a sound proof booth for 20 minutes. Participants were then moved to an anechoic chamber in order to compare their experiences to the sound booth. Tinnitus was perceived in 40% of the listeners in the sound booth and in 60% of the listeners in the anechoic chamber. Tinnitus was perceived sooner in the anechoic chamber than in the sound booth. No significant differences in tinnitus perception or emergence were seen between the sound booth and anechoic chamber or between males and females. Of the participants who perceived tinnitus, the largest percent, 33%, matched the pitch to be around 4000 Hz and 44% matched the loudness to be 0-2 dB SL. These pyschoacoustical characterizations are similar to those reported by tinnitus patients.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

Tinnitus is one of the most frequently reported patient complaints heard by audiologists and otologists. It is associated with almost every form of auditory pathology, including acoustic trauma, otosclerosis, vestibular schwanoma, and Meniere's disease (Jastreboff, 1990; Nelson & Chen, 2004). Nearly 15 million American adults are bothered by this phenomenon (Hoffman & Reed, 2004). Roughly 10-15% of the individuals who experience tinnitus require professional help (Heller, 2003). There is some research examining the exact mechanisms surrounding tinnitus, but questions still remain. Without understanding these mechanisms, treatment is difficult.

A common approach by audiologists, in treatment, is telling the patient that "nothing can be done and that they will have to learn to live with it" (Jastreboff & Hazell, 1993). For those who practice tinnitus management, which includes medications, surgery, avoidance of sounds, and masking techniques, the long-term prognosis is poor. The introduction of Tinnitus Retraining Therapy (TRT) in 1990 (Jastreboff, 1990; Jastreboff, 1996) offered a new option to tinnitus patients (Jastreboff & Jastreboff, 2003b). In order for audiologists to successfully help their clients and practice tinnitus management effectively, it would be helpful to better understand the mechanisms of tinnitus. Research is ongoing in this area and many theories surrounding the mechanisms of tinnitus generation and

the mechanisms of tinnitus-induced distress have been proposed. Several theories will be described in this paper.

Most of the research on tinnitus has been conducted on individuals who already suffer from tinnitus and, during the last 15 years, on animal models (Jastreboff, Brennan, & Sasaki, 1988). More information is needed on the emergence of tinnitus especially in normally hearing individuals, as about 20-30% of tinnitus sufferers have normal hearing (Tyler, 2000). It is well recognized that low levels of sound enhance pre-existing tinnitus or can induce tinnitus (Heller & Bergman, 1953). Mechanisms of this phenomenon are speculative and only two known studies, using normal hearing individuals without tinnitus, under controlled sound environments, were published (Heller & Bergman, 1953; Tucker, Phillips, Ruth, Clayton, Royster, & Todd, 2005). While these studies showed that tinnitus was induced by very quiet environments, the psychoacoustic parameters of silence-induced tinnitus (ex: pitch, loudness matching, maskability) were not determined. It is likely that the psychoacoustic characterization of this type of tinnitus will be helpful in delineating potential mechanisms involved in this phenomenon. Therefore, the purpose of this study was to investigate the emergence of tinnitus in normal hearing individuals under strictly controlled sound conditions, and to evaluate the psychoacoustic characteristics of silence-induced tinnitus in these individuals.

CHAPTER 2

LITERATURE REVIEW

Tinnitus

Tinnitus has been referred to as the perception of sound in the absence of external stimuli or a “phantom auditory perception” (Jastreboff, 1990). It is a symptom associated with almost every form of auditory pathology including acoustic trauma, otosclerosis, and Meniere’s disease (Jastreboff, 1990; Jastreboff, 2003b; Nelson & Chen, 2004). According to the most recent American epidemiological study (with data analyzed from the United States National Health Interview Survey Disability Supplement 1994-1995), approximately 4.4% of adults experience chronic tinnitus (lasting for 3 months or more) (Hoffman & Reed, 2004). Other studies have shown the prevalence of tinnitus in up to 20% of the general population (Hoffman & Reed, 2004). Tinnitus is neither age nor gender specific (Davis & Rafaie, 2000) and can be chronic or temporary (Hoffman & Reed, 2004). People afflicted with tinnitus often describe the sounds that they perceive as ringing, buzzing, or hissing, although this list is not exclusive.

Research has been and continues to be conducted in the hopes of finding a cure; however, results have not been promising (Jastreboff & Hazell, 1993). Until the exact mechanism of tinnitus is known, the development of an effective treatment remains difficult. Some relief has been observed with the use of

auditory masking procedures (Vernon & Meikle, 2000), electrical stimulation (Rubinstein, Tyler, Johnson, & Brown, 2003; Ruckenstein, Hedgepeth, Rafter, Montes, & Bigelow, 2001), and pharmacological methods (Johnson, Brummett, & Schleuning, 1993), but long- term results have not been successful (Jastreboff, 1990; Jastreboff & Hazell, 2004; Dobie, 1999, 2004). Therefore, a better understanding of how neuronal information related to tinnitus is processed within the nervous system and the mechanisms surrounding the different types of tinnitus generators is needed.

Classifications of Tinnitus. The terms objective and subjective are sometimes used to classify tinnitus into different groups. Tinnitus that can be detected and measured by an observer, such as sounds that may occur as a result of abnormal blood flow or swallowing, may be referred to as objective tinnitus. This form of tinnitus is rare. Subjective tinnitus, on the other hand, the most prevalent type, occurs without the physical presence of sound and can only be heard by the affected individual (Jastreboff, 1990; Nelson & Chen, 2004). Subjective tinnitus has many forms and can be anything from a minute sound occurring occasionally to a roaring sound lasting all day and interfering with everyday activities (Moller, 2003). Subjective tinnitus will be the focus throughout this literature review. Currently the terms objective and subjective tinnitus are disappearing and are being replaced by somatosound and tinnitus (Jastreboff, 2003b). Before discussing theories of tinnitus, two theories of hearing will be reviewed.

Auditory Pathways Related to Tinnitus. The most common and accepted

auditory pathway of hearing is referred to as the classic (lemniscal) pathway. In this system, sound is received by the ear and transmitted along a network of auditory nuclei leading to the cerebral cortex. Here it is then processed and interpreted as recognizable sound (Nelson & Chen, 2004).

Another pathway of hearing, supported by Jastreboff (1990) and referred to by Moller (2003) as the *nonclassic* (extralemniscal) auditory system, proposes that hearing involves more than just the passive transmission of sound along a direct line to the cerebral cortex. Projections to non-auditory sensory structures are characteristic of the nonclassic auditory system, whereas nuclei from the classic pathways project directly to the auditory cortex (Moller, 2003). These non-auditory sensory systems are also involved and contribute to the overall perception, localization, and loudness of sound in the nonclassic pathway. If the hearing pathway does include other sensory systems, then certain portions of this nonclassic pathway may be able to compensate for damage in auditory regions (Moller, 2003). This theory can be compared to the function that the somatosensory system plays in vision. When both the somatosensory and visual systems are integrated they work together to provide a sense of where one's body is in space (Moller, 2003). However, if one of these systems is not functioning properly the other system can help compensate, somewhat.

Mechanisms of Tinnitus

To date, there is no established neuronal mechanism behind subjective tinnitus (Jastreboff & Jastreboff, 2003b; Nelson & Chen, 2004). There are, however, numerous theories that have been proposed. There is a consensus

that tinnitus is caused by abnormal neural activity within the auditory pathways, and that this activity is incorrectly interpreted as sound by the auditory centers (Tondorf, 1981; Jastreboff, 1990; Moller, 2003; Bauer, 2003). This is where the similarities between theories end (Jastreboff, 1990).

Until Jastreboff's animal model of tinnitus (1988), which used both behavioral and electrophysiological approaches to look at the perceptions and possible mechanisms of tinnitus generation in pigmented rats, essentially all research was conducted on humans. Human research based on anatomic and pathophysiologic studies presents many ethical restrictions and consequences, not allowing for rigorous analysis. However, with the creation of an animal model, it has become possible to control for sources of variance, such as age and exposure to acoustic trauma, and to evaluate different possible mechanisms of tinnitus and ways to alleviate it (Jastreboff, Brennan, & Sasaki, 1988; Bauer, 2003).

Theories on where tinnitus is generated can be divided into two categories, peripheral and central, based on the possible pathology of tinnitus. Peripheral refers to the ear itself, including the cochlea, while central relates to the pathway from the VIIIth nerve to and including the brain (Nelson & Chen, 2004, Simpson & Davies, 1999). Jastreboff (1990) implies that these classifications are too simplistic and that all levels, not just peripheral or central, are involved in each tinnitus case. Although these terms are general, the hypotheses have been grouped together according to their suspected place of pathology.

Peripheral. In reference to cochlear dysfunction, there are proposed mechanisms of tinnitus generation. Morphological and/or structural changes to the cochlea alone are not believed to be directly responsible for the generation of tinnitus. This is not to say that tinnitus is not affected by pathologies in the ear. For example, hearing loss and injury to the cochlear hair cells caused by loud noise are often accompanied by tinnitus. A more acceptable theory is that abnormal input, created by pathologies in the ear, may cause changes in the central nervous system, which in turn, generate tinnitus (Moller, 2003).

Tondorf (1981) suggested that the origin of tinnitus might be based on a change in the way that hair cells are coupled to the tectorial membrane, which may result in a loss of spontaneous activity and an imbalance in nerve fiber activity. Jastreboff (1990) expanded on his theory, by proposing that mechanical decoupling may also occur at the attachment of the cilia to the outer hair cells resulting in tinnitus.

Another theory suggested by Tondorf (1987), also of cochlear origin, is based on the analogy between chronic tinnitus in the auditory system and chronic pain in the somato-sensory system. He concluded that there are many similarities between the two. Both are subjective sensations and continuous events that may change in quality. They can both be masked or alleviated, in most instances, by electrical stimulation. Neither have specific systems for transmitting or processing, and both systems consist of afferent and efferent fibers that make connections with higher order processing centers.

Jastreboff (1990) proposed that tinnitus occurs when there is a dysfunction between the outer and inner hair cell systems which is known as the discordant damage/dysfunction theory. There are a number of observations supporting this hypothesis. The damage to the basilar membrane usually begins in the basal (high frequency) end and leads to the apical (low frequency) end. It is also known that inner hair cells (innervated by afferent type I fibers) are more resilient to injury than to outer hair cells (innervated by afferent type II fibers); therefore outer hair cells are usually impacted first. Damage to the outer hair cells with intact inner hair cells may cause abnormal afferent fiber activity (Jastreboff, 1995). Outer hair cell damage changes the mechanical properties of the organ of Corti due to the disintegration of the mechanical coupling that is normally carried out by the outer hair cells and their cilia. This may lead to the regional collapse of the tectorial membrane, which will decrease the distance between cilia of the functional inner hair cells, with the possibility of physical contact of the cilia and the tectorial membrane. This may result in tonic depolarization of the inner hair cells, which gives way to abnormal activity in afferent fibers (Jastreboff, 1990; Jastreboff, 1995). Abnormal afferent fiber activity may in turn cause decreased efferent fiber activity, which impacts the length of the outer hair cells along the basilar membrane. Dysfunction of the outer hair cells along a particular section on the basilar membrane will then lead to decreased Type II fiber activity from that section.

When decreased activity of the Type II fibers occurs, a decrease in inhibition of the afferent fibers from the inner hair cells also occurs (Jastreboff,

1995). It is believed that any dysfunction of the Type II fibers will provide enhanced activity of the inner hair cells not only from the affected region of the basilar membrane, but also from the non-affected neighboring areas (Jastreboff, 1995). This increase in activity from the afferent fibers is then thought to be perceived as tinnitus (Jastreboff, 1995; Jastreboff, 2004).

These theories, although logical in nature, take into account only part of the auditory system when hypothesizing the anatomic location of the structures that generate tinnitus. It has been demonstrated that tinnitus does not occur in all individuals with hair cell injuries, thus suggesting that something other than damage to the hair cells alone is involved in the generation of tinnitus (Jastreboff, 1990; Moller, 2003).

Central. Although some forms of tinnitus may be generated in the ear, the central nervous system is believed to be the location of the physiologic abnormality that causes most forms of tinnitus. This hypothesis is supported by the fact that deaf people and individuals with severed auditory nerves can have tinnitus. It seems that most forms of tinnitus related to pathologies in the ear are caused by abnormal inputs that these pathologies are directing to the central nervous system (Jastreboff, 1990; Moller, 2003).

Neurophysiological Model. The neurophysiological model of tinnitus, proposed in the 1980s (Jastreboff, 1990) involves both parts of the auditory system (central and peripheral) and the limbic and autonomic systems. The theory behind this model provides the backbone for studying and treating tinnitus and decreased sound tolerance. This approach contains four main parts. First,

the auditory system, and other systems of the brain (limbic and autonomic), are involved in processing sound. However, when tinnitus is not perceived as bothersome, the auditory pathway is the only pathway involved, and tinnitus-related neuronal activity is restricted within the auditory system. Second, improper activation of the limbic and autonomic systems results in the behavioral manifestation of tinnitus. Third, conditioned reflexes are responsible for the interconnections between the different systems of the brain involved in tinnitus. And fourth, with habituation of the reflexes, it may be possible to eliminate the negative reactions evoked by tinnitus (Jastreboff & Jastreboff, 2003c).

Theories of Neuroplasticity

It is hypothesized that neural plasticity also shares an important role in the generation of tinnitus (Jastreboff, 1990; Moller, Moller, & Yokota, 1992; Moller, 2000). The ability of the brain to adapt or change is referred to as neuroplasticity. The central nervous system, oriented towards homeostasis, is thought to compensate for decreased information input for any modality by increasing sensitivity in the centers involved in perception of the modality. The assumption is that when structures are injured, their functions are moved to other parts of the central nervous system. This is thought to occur in all parts of the central nervous system and is mainly triggered by deprivation of input, abnormal input, or injury (Moller, 2003). A total absence of input may disrupt the balance provided by the central nervous system, resulting in the abnormal functioning of the processing centers (Jastreboff, 1990). Examples of this can be demonstrated in many systems of the body. In the case of amputees, PET scans

have shown how the brain adapts to the new input it receives and expands its sensation in the remaining areas of the damaged extremity (Moller, 2003). This can also be seen in the visual system. People who are blind often acquire greater sensation acuity in other sensory areas (Goldreich & Kanics, 2003).

Plasticity in the auditory system has also been demonstrated. Deprivation of auditory input or the generation of new input to the central nervous system may cause changes in the function of central structures (Moller, 2003). A decrease in auditory input results in an increase in the sensitivity of neurons within the inferior colliculus (Salvi, Saunders, Gratton, Arehole, & Powers, 1990). This phenomenon was observed and documented by Heller and Bergman (1953) in their landmark study. They found that when normal hearing individuals are deprived of auditory input, they will experience increased hearing sensitivity and frequently experience tinnitus. The mechanism of this phenomenon is unknown; however, it implies that decreased input may cause a redirection of information, resulting in contraction of the neuronal systems involved in automatic gain control within the auditory nervous system. This contraction, then, leads to an increase in the gain of the system, as well as a heightening of the sensitivity of those nuclei to any input including spontaneous activity. These may be perceived as tinnitus (Jastreboff, 1990).

Hyperacusis

Another possible result stemming from this neuroplasticity theory is that hyperacusis, often found to accompany tinnitus, could be a pretinnitus state (Jastreboff & Hazell, 1993). Individuals with hyperacusis may have the tendency

to develop tinnitus. Increased gain within the auditory system will enhance any pre-existing tinnitus-related neuronal activity and possibly cause it to cross the detection threshold. If this gain is decreased, however, it may reduce the tinnitus. Hyperacusis and tinnitus are sometimes thought to be manisfestations of the same internal phenomenon (i.e. increase of the gain within the auditory pathways). Because hyperacusis often occurs before the onset of tinnitus, it may offer a possibility for tinnitus prevention (Jastreboff & Jastreboff, 2003a).

Hyperacusis is a component of a decreased sound tolerance. It is characterized by a shift toward lower values of a person's Loudness Discomfort Levels (LDLs), making all sounds uncomfortable, even though the physical intensity of the sound is below what is normally judged to be of discomfort (Formby & Gold, 2002). Hyperacusis occurs when people experience negative reactions, such as pain, to sounds that would not disturb a normal listener. The neural activity occurring in response to an uncomfortably loud sound in people experiencing hyperacusis is similar to the neural activity caused by a much higher sound level in normal listeners. The reactions and discomfort, that people affected by hyperacusis experience, are mainly dependent upon the physical characteristics of the sound. They are also reliant on the limbic and autonomic nervous systems, which are activated by the connections between these systems and the auditory pathways (Jastreboff & Jastreboff, 2003c).

Misophonia (negative attitude toward sound) is another component of decreased sound tolerance. Hyperacusis and misophonia frequently coexist and they often occur in conjunction with tinnitus. According to Jastreboff and

Jastreboff (2003a), 66.4% of tinnitus patients experienced decreased sound tolerance. Misophonia occurs when the auditory system is functioning normally but there are enhanced functional connections between the auditory and limbic systems. Furthermore, phonophobia can be considered as a specific type of misophonia. It is not only determined by the physical characteristics of sound, but mainly by the patient's past experiences, psychological profile, and the context in which the sound occurs (Jastreboff & Jastreboff, 2003c). The treatments prescribed for decreased sound tolerance and tinnitus were, and often still are, the use of ear protection and avoidance of sounds. Unfortunately, these approaches are found only to worsen rather than relieve the problem (Jastreboff & Jastreboff, 2003a).

It is believed that hyperacusis is caused by a change in the central processing of sound (Nelson & Chen, 2004). More specifically, it is manifested by an increase in the central gain of the auditory system (Jastreboff, 1990). In fact, sound therapy, one of the most common strategies employed for managing hyperacusis, involves desensitization protocols that stem from the idea that a centrally mediated, compensatory gain control process is involved. Sound therapy suggests that there is a compensatory gain process that regulates a person's supra-threshold levels in the auditory system. If these levels are altered they can be set back to normal by exposure to a continual background sound. However, sound intolerance could be induced in normal listeners when sound input is attenuated for prolonged periods of time (Jastreboff & Hazell, 1993).

There is also evidence that loudness judgements can be manipulated by altering background noise levels in normal listeners. For example, Formby, Sherlock, and Gold (2003) conducted a study involving ten normal hearing participants in order to study the effects of two different treatments (earplug and noise instrument) and their elicited compensatory responses on the gain process. They found that after 2 weeks of constant treatment, the participants wearing the earplugs needed less intense tones in comparison to those individuals wearing the noise instrument to achieve the same loudness judgements when compared to their baseline judgements. These findings support the theory behind adaptive plasticity and the gain control process and can be implemented into the management of hyperacusis (Jastreboff, 1990). Because the altered perception of sound occurring in hyperacusis is similar to the distortion that occurs in tinnitus (Nelson & Chen, 2004), these findings may be further implemented into tinnitus management and research. Therefore, by acquiring a better understanding of the mechanisms surrounding hyperacusis, it is likely that a better comprehension of tinnitus and its generators will follow.

Clinical Implications and Current treatments

Many methods including medications, surgery, avoidance of sounds, and masking techniques have been tried to relieve and/or eliminate tinnitus, however outcomes have been variable. One method for providing relief that has proven effective in about 80% of patients is Tinnitus Retraining Therapy (TRT). TRT is based on the neurophysiological model of tinnitus and decreased sound tolerance (Jastreboff, 1990). Its purpose is to evoke and sustain habituation of

reactions and perception to tinnitus and other sounds by modifying the neural connections between the auditory and the limbic and autonomic nervous systems. TRT uses counseling techniques whose objectives are to remove the negative associations with tinnitus by the reclassification of tinnitus into a neutral stimuli category. In addition to counseling, the use of sound therapy is also an integral component of TRT. By increasing the background neuronal activity through the presentation of background noise, the strength of the tinnitus signal is decreased. In TRT, counseling and sound therapy are used in combination to assist in the elimination of the conditioned response (Jastreboff & Jastreboff, 2003c).

The neurophysiological model is used as the basis for TRT and has been reported to produce satisfactory responses in many patients (Jastreboff & Jastreboff, 2003b). In fact, an early outcome of TRT was the finding that, on average, approximately 14 dB LDL shifts were noted in most tinnitus patients. This reflects the adjustments to gain within the auditory system in response to the low-level background noise (Jastreboff, Gray, & Gold, 1996). More research, however, is needed in order to evaluate the efficacy of the treatment components of TRT, and to validate the concept of an adaptive auditory gain process and the mechanisms behind this neuroplasticity (Formby & Gold, 2002).

Tinnitus in individuals with normal hearing

Tinnitus is mainly associated with abnormal hearing and most theories on tinnitus generation are based on this population, although this phenomenon does occur in individuals displaying normal hearing. The discordant

damage/dysfunction theory (Jastreboff, 1990), along with automatic gain control within the auditory system, provide a possible explanation for the occurrence of tinnitus in normal hearing individuals. The discordant damage/dysfunction theory is based on the idea that when a dysfunction between the outer and inner hair cells exists, tinnitus is experienced. Hair cells may not be damaged enough to cause a hearing loss, but may be sending abnormal signals to the brain, interpreted as tinnitus. When either an abnormal signal or lack of signal is sent to the central nervous system, a redirection of the signal may result in an increase in the gain within the auditory system. This may also lead to a heightening of sensitivity to any input within the auditory nervous system, including spontaneous activity, perceived as tinnitus. The findings from Heller and Bergman (1953) suggest that auditory deprivation can cause an increase in central gain, which could lead to tinnitus in normal hearing people. Until the phenomenon of tinnitus occurring in “normal hearing” individuals is better understood, the mechanism of tinnitus generation in individuals who actually suffer from tinnitus cannot be understood.

Most of the clinical research on the mechanism of tinnitus generation has been conducted on individuals already suffering from tinnitus. Only two known studies to date, using normal hearing subjects without tinnitus, were conducted in order to determine the cause of tinnitus.

First, a study by Heller and Bergman in 1953 looked at the effects of low ambient noise levels on normal hearing individuals. The intent of this study was to determine the incidence and character of tinnitus in normal hearing people

when ambient noise levels were much lower than in ordinary living conditions. The study consisted of 80 normal hearing adults between 18 and 60 years of age without any complaints of tinnitus. During the study, participants were instructed to make notes of any sounds that they detected while in the sound-proof room. After approximately 5 minutes, the participants were removed from the sound-proof room and written details of their observations were collected. The sounds described by the participants were compared to a control group consisting of 100 hard-of-hearing patients, 73 of which had tinnitus. Eleven of the different sounds recorded were identified in both groups. Results indicated that tinnitus was experienced by 94% of the normal hearing participants when auditory input was decreased. These results suggest that tinnitus cannot be of a pathologic nature because essentially everyone experiences it.

Recently, a second study similar in design (Tucker et al, 2005) that also looked at the emergence of tinnitus in the normal hearing population was conducted. This study expanded upon Heller and Bergman's design by adding several methodology changes, including the potential impact of race and gender.

This study consisted of 120 normal hearing adults (60 male/60 female) between the ages of 18 and 30 years. Both the male and female groups contained 40 Caucasian and 20 African American participants. The Heller and Bergman study did not report race and gender differences, nor did it define normal hearing. This study determined participants to have normal hearing if they passed a 20 dB HL screening for frequencies 250 Hz through 8000 Hz and a tympanogram screening for both ears. Another modification to the earlier study

by Heller and Bergman was the amount of time spent in the sound booth and the method of reporting perceived sounds. This study placed each participant in the sound booth for a 20 minute period. Following the silent period, participants were presented with a form to record any perceived sounds. In Heller and Bergman's study participants only spent approximately 5 minutes in the booth and it was not stated exactly when the participants were instructed to write down any sounds that they detected (i.e. during the 5 minutes in the chamber or after exiting). Finally, this study clearly defined the ambient noise levels measured in SPL (within ANSI S3.1 1977 Sound Room Ambient Noise Standards) in the sound booth, which were 24 dB at 125 Hz, 15 dB at 250 Hz, 6 dB at 500 Hz, 12 dB at 1000 Hz, 7 dB at 2000 Hz, 7 dB at 4000 Hz, and 6 dB at 8000 Hz. The Heller and Bergman study only predicted the levels.

Results from this study indicated that, overall, tinnitus was perceived in 64% of the normal hearing subjects when auditory input was decreased. This was a much lower incidence when compared to the results from Heller and Bergman's earlier study (94%). This study also reported that no significant differences in perception were observed between genders, however differences were found between Caucasian and African American subjects. Tinnitus was perceived more commonly among Caucasian subjects (78%) when compared to African American subjects (38%).

These studies open the door for more research looking at the effects of decreased auditory input on the normal population. While these studies showed that that presence of tinnitus was induced by very low sound levels, they did not

determine the psychoacaoustic parameters of silence-induced tinnitus. Therefore, it would be beneficial to conduct another study, similar in design to these studies looking at the psychoacoustic parameters (ex: pitch, loudness matching, maskability). It is likely that the psychoacoustic characterization of this type of tinnitus would be helpful in delineating potential mechanisms involved in this phenomenon. Also, from an ethical standpoint, participants should be informed of the possible consequence of developing tinnitus as a result of their participation.

The observation from the previous studies (Heller & Bergman, 1953; Tucker et al, 2005) supports the idea that tinnitus might be caused by an increased gain within the subcortical centers of the auditory system. If proven, this subcortical theory would allow for the development of a mechanism-specific treatment to help relieve the effects of tinnitus and the associated phenomena. It should also help to eliminate the use of the non-effective treatments that are still being practiced today.

Statement of Purpose

To date there have been only two studies (Heller & Bergman, 1953; Tucker et al, 2005) which evaluated the emergence of tinnitus in listeners with normal hearing. The studies' results disagree in some aspects and neither assessed the psychoacoustic parameters of silence-induced tinnitus. Therefore, the current study will investigate the emergence of tinnitus in people with normal hearing under strictly controlled experimental conditions, and evaluate the psychoacoustic characteristics of silence-induced tinnitus in these individuals.

CHAPTER 3

METHODS AND MATERIALS

Participants

The study included 20 Caucasian adults (10 male/10 female) ranging in age from 18-32 years, with no reports of prior tinnitus. The participants were first screened to determine their hearing sensitivity in both ears, and only participants with normal hearing, defined as thresholds better than 20 dB HL in the frequency range of 250 Hz to 8000 Hz, participated in the study. This study was approved by the Towson University Institutional Review Board (IRB) for the Protection of Human Subjects (see Appendix A).

Screening protocol

A complete audiological evaluation took place in a double-walled sound-treated booth. The evaluation began with an otoscopic examination of both ear canals and tympanic membranes. Tympanometry was also conducted to establish middle ear admittance using the GSI TympStar. Pure tone thresholds to -10 dB HL were established for both ears within 2 dB HL accuracy using the GSI 61 audiometer. Air conduction values were collected for 250 Hz, 500 Hz, 1 kHz, 2 kHz, 4 kHz, 6 kHz, 8 kHz, and 12.5 kHz. Bone conduction values were obtained for 500 Hz, 1 kHz, 2 kHz, and 4 kHz within 2 dB HL accuracy, and White Noise (WN) thresholds were established using 1 dB steps. The threshold

of WN was established to use later as the basis for calculating minimum masking levels in dB SL.

After all measurements were performed and participants' hearing was determined to be within normal limits and negative for middle ear pathology, participants were given a 5 minute rest period outside the sound-treated booth before starting the experiment. During this time participants were made aware of the potential emergence of transient tinnitus and instructed about the purpose of the experiment, the testing procedure, and the way that they were to communicate to the examiner.

Experimental protocol

Participants then entered the sound-treated booth. They communicated (through gestures and writing with a soft pen on laminated forms) if and when they heard any changes in the environment (heard any sounds). Participants were also instructed to describe any sounds that they heard by circling or writing this information on the forms provided.

Once 20 minutes had passed, psychoacoustical characterization of any silence-induced tinnitus was performed. The written descriptions from the participants were used to determine whether the tinnitus was similar to a pure tone (i.e. ringing), narrowband noise (NBN) (i.e. ocean, seashell), or WN (i.e. electronic noise); if tinnitus was present in one or both ears or in the head; and if there was a difference between ears. If tinnitus was described to be only in one ear, then that ear was tested. Otherwise, measurements were performed on both ears. The order for the psychoacoustical protocol remained constant for all

participants and was set to ensure that tinnitus perception was not lost during the experiment. In the case that participants' perception of tinnitus was lost during the experiment, they were instructed (prior to the start of the experiment) to inform the examiner. Further testing continued based on the participants' recollection of the perceived sound.

Pitch match. Pitch matching was performed first using the GSI 61 audiometer in the double-walled booth to obtain a depiction of the perceived signal. If the tinnitus sound that the participant described was most similar to WN, pitch matching could not be done and only loudness matching could be performed. A reference sound, either pure tone or narrowband, was presented to the contralateral ear beginning with 1 kHz at 3 dB SL for the tested frequency. The participant was instructed to indicate if the sound with which they were presented was higher, lower, or similar in pitch to the sound that they heard in the sound-proof booth. The pitch assessment was narrowed down by presenting the participant with a choice of frequencies and having them pick the one that was most similar to the sound heard. Once pitch matching was completed, the participant's threshold was measured at the frequency corresponding to the tinnitus pitch with 1 dB accuracy.

Loudness match. Loudness match was then performed to determine the perceived loudness of the tinnitus. A pure tone or narrowband noise at the frequency indicated in the pitch match was presented to the contralateral ear below the participant's threshold of hearing. The tone was increased in 1 dB

steps until the participant indicated that the sound was at the same level as their tinnitus. This threshold test was then repeated for reliability.

Minimum masking level. After the loudness match the minimum masking level was determined to distinguish how well the brain could detect the tinnitus signal in the presence of competing noise (Jastreboff, 2004). Depending on the perceived location of the tinnitus (right ear, left ear, bilateral) WN was presented ipsilaterally, contralaterally, and binaurally below the participants' threshold. Participants were instructed to listen to their tinnitus and report any changes to it. WN was increased in 1 dB steps until the participant reported that the tinnitus was no longer perceived.

Threshold check. After psychoacoustical characterization was performed, pure tone thresholds to -10 dB HL were again established for both ears within 2 dB HL accuracy using the GSI 61 audiometer in the sound-treated booth. Air conduction values were collected for 250 Hz, 500 Hz, 1 kHz, 2 kHz, 4 kHz, 6 kHz, 8 kHz, and 12.5 kHz. These thresholds were compared to the participants' initial thresholds.

Anechoic chamber. Participants then exited the sound-treated booth and were placed in a sound-proof anechoic chamber. They remained there until tinnitus was perceived, but no longer than 20 minutes. Upon exiting the chamber, participants were asked to compare their experience to their experience in the sound-treated booth, specifically the amount of time that was needed to pass before tinnitus was perceived.

The ambient noise level of the booth and anechoic chamber were measured with the Bruel and Kjaer (B&K) 2203 sound level meter at ear level. Measurements were taken across the spectrum and at individual frequencies (31.5 Hz, 63 Hz, 125 Hz, 250 Hz, 500 Hz, 1000 Hz, 2000 Hz, 4000 Hz, 8000 Hz, and 16000 Hz).

CHAPTER 4

RESULTS

Ambient Noise Levels

The ambient noise levels of the booth, anechoic chamber, and an empty classroom were measured and are shown in Figure 1. Ambient noise levels were measured using 1-octave bands centered at 31.5 Hz, 63 Hz, 125 Hz, 250 Hz, 500 Hz, 1 kHz, 2 kHz, 4 kHz, 8 kHz and 16 kHz. Overall ambient noise levels were highest in the classroom and softest in the anechoic chamber. The differences in the ambient noise levels between the sound booth and anechoic chamber are displayed in Figure 2. The largest difference was approximately 11 dB and was located around 100 Hz.

Incidence of Tinnitus

The incidence of tinnitus in young-adult (18-32 years) Caucasian participants with normal hearing is shown in Table 1. Overall, tinnitus was perceived in the sound-treated booth in 8 out of 20 (40%) of the normal hearing individuals after a period of 20 minutes. This study showed that 5 of 10 females (50%) and 3 of 10 males (30%) heard tinnitus in the sound booth. There was no significant difference in tinnitus perception between genders in the sound booth ($\chi^2 = 0.833$, df = 1, N.S.). In the anechoic chamber tinnitus was perceived in 12 out of the 20 (60%) participants; eight out of 10 females (80%) and 4 out of 10 males (40%) heard tinnitus in the anechoic chamber. There was no significant

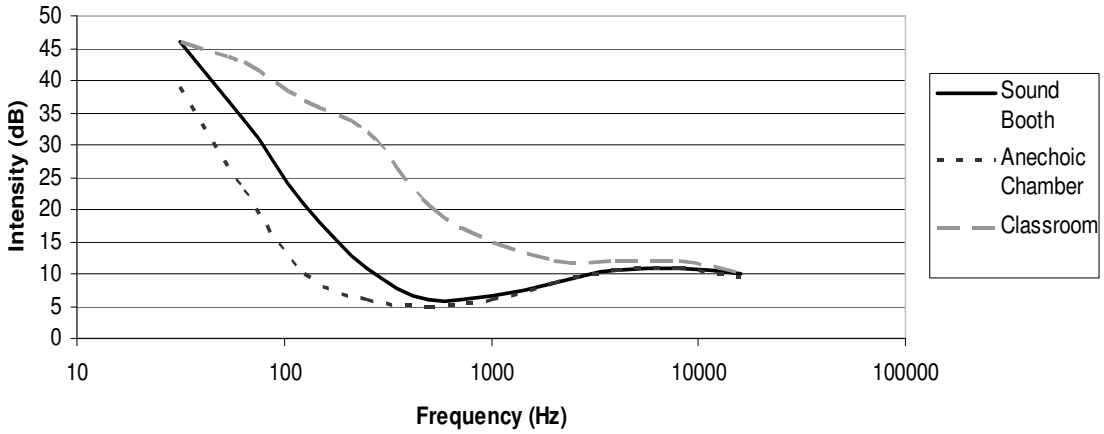

Figure 1. Ambient noise levels in the sound-treated booth, anechoic chamber, and classroom, measured in 1-octave bands in octave intervals from 31.5 Hz to 16 kHz.

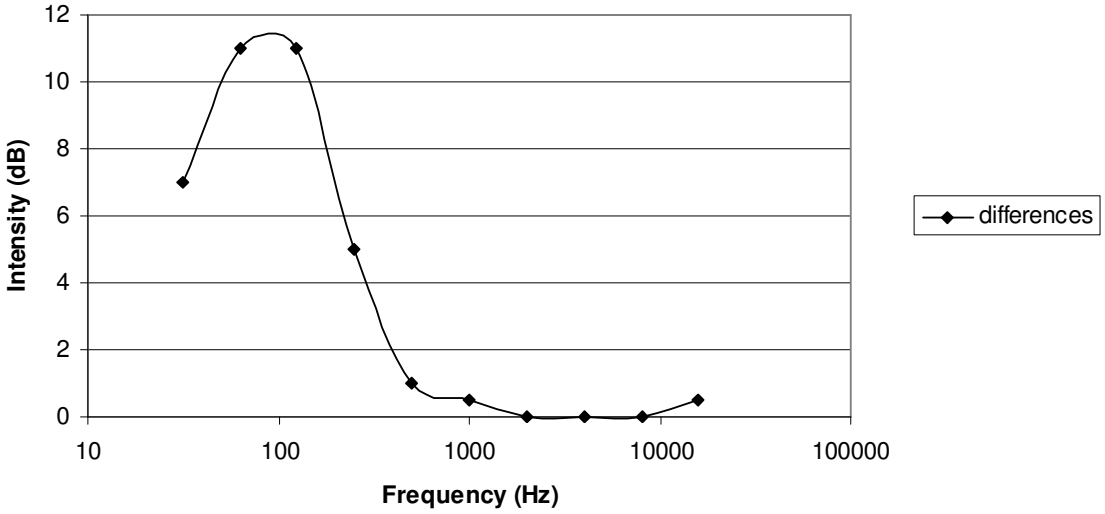

Figure 2. Differences in ambient noise levels between the sound booth and anechoic chamber measured in 1-octave bands in octave intervals from 31.5 Hz to 16 kHz.

Table 1.

Incidence of tinnitus

Incidence of tinnitus in sound booth			
Population	# of Participants hearing tinnitus	Mean # of sounds, (S.D.)	Mean time, (S.D.)
Overall	40% (8/20)	1.63 (.99)	5.25 min (5.36)
Females	50% (5/10)	1.2 (.40)	7 min (6.13)
Males	30% (3/10)	2.33 (1.25)	2.33 min (.47)

Incidence of tinnitus in anechoic chamber			
Population	# of Participants hearing tinnitus	Mean # of sounds, (S.D.)	Mean time, (S.D.)
Overall	60% (12/20)	1.17 (.37)	5.13 min (4.51)
Females	80% (8/10)	1.25 (.43)	4.89 min (4.65)
Males	40% (4/10)	1 (0)	5.83 min (3.97)

gender difference in tinnitus perception in the anechoic chamber ($\chi^2 = 3.333$, df = 1, N.S.).

All of the participants who perceived tinnitus in the sound booth also perceived tinnitus in the anechoic chamber. There was no significant difference between the ratio of the emergence of tinnitus in the sound booth versus in the anechoic chamber ($\chi^2 = 1.6$, df = 1, N.S.).

The mean time between the time participants entered the test room and the beginning of participants' perceived tinnitus in the sound-treated booth and anechoic chamber is shown in Table 1. The individual times of emergence are shown in Figure 3. Six of the 8 listeners who experienced tinnitus in the sound-treated booth reported that it was perceived early (less than 5 minutes into the 20 minute session of silence). The other two participants who experienced tinnitus in the sound booth reported that they perceived it later in the study (after 14 minutes into the 20 minute session of silence). Seven of the 8 listeners who experienced tinnitus in the sound-treated booth reported that the tinnitus lasted from the time of emergence until after they left the sound enclosure. One participant who experienced tinnitus in the sound-treated booth reported that the tinnitus stopped after approximately 2 minutes.

Half of the 12 listeners who experienced tinnitus in the anechoic chamber reported that it was perceived early (less than 5 minutes into the 20 minute session of silence). The other half who experienced tinnitus in the anechoic chamber reported that it was perceived later (after 5 minutes into the 20 minute

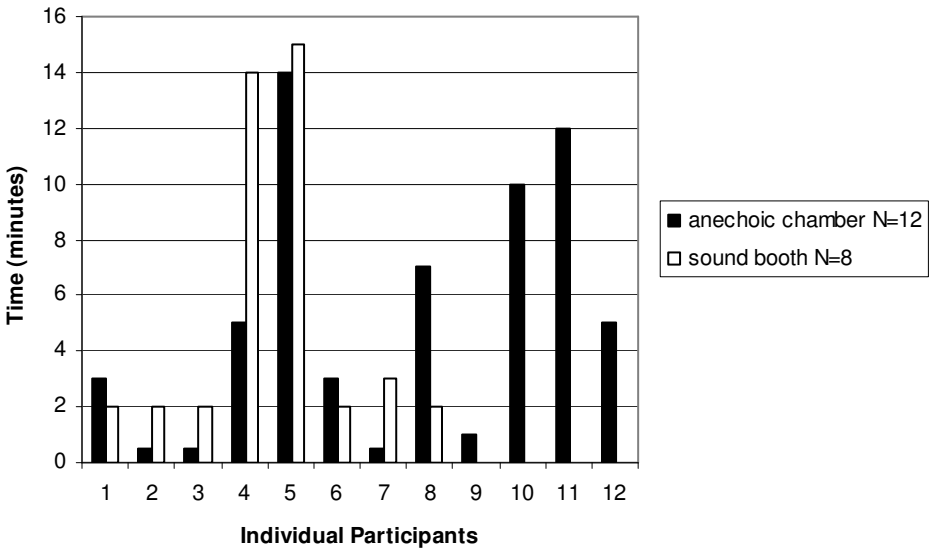

Figure 3. The time of emergence of tinnitus in the sound booth and anechoic chamber.

session of silence). Male listeners perceived tinnitus sooner than females in the sound booth and female listeners perceived tinnitus sooner in the anechoic chamber. There was no significant gender difference in the amount of time that it took for tinnitus to be perceived in the sound booth (t = -1.513, df = 4, p = 0.205) or anechoic chamber (t = 0, df = 7, p = 1).

All eight of the participants who experienced tinnitus in the sound booth also experienced tinnitus in the anechoic chamber. Figure 4 shows the differences in the time of tinnitus emergence in the sound-treated booth and in the anechoic chamber. Five listeners (63%) perceived tinnitus earlier in time when in the anechoic chamber than when in the sound booth. Three listeners (37%) perceived tinnitus sooner when in the sound-treated booth than when in the anechoic chamber. There was no significant difference between the time of tinnitus emergence in the sound booth versus the time of emergence in the anechoic chamber (t = 0.756, df = 7, p = 0.475).

Psychoacoustical characterization of tinnitus

Types of tinnitus sounds. The types of sounds perceived by normal hearing individuals in the sound booth and anechoic chamber are shown in Table 2. Ring (75%), hum (25%), and cricket (25%) were the most common sounds perceived in the sound booth. Ring (67%) and hum (17%) were also the most common sounds heard in the anechoic chamber. Overall, ring was the most common tinnitus sound reported. When the number of participants reporting ring, hum, and others were compared, there was no significant difference between the sound booth and the anechoic chamber (χ^2 = 1.16, df = 3, N.S.).

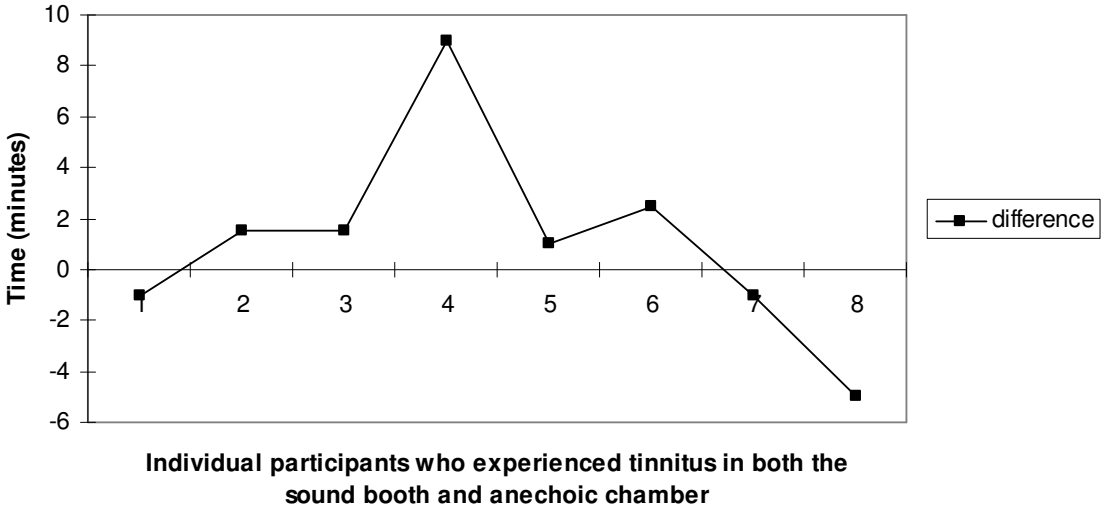

Figure 4. The time differences between the emergence of tinnitus in the sound-treated booth compared to the anechoic chamber.

Table 2.

Types of tinnitus sounds

Types of tinnitus sounds heard in sound-treated booth

Sound	Females	Males	Total
Ring	4	2	6 (75%)
Hum	2	0	2 (25%)
Hiss	0	1	1 (13%)
Ocean	0	1	1 (13%)
Beep	0	1	1 (13%)
Cricket	0	2	2 (25%)
Buzz	0	1	1 (13%)
No sound	5/10	7/10	12/20

A total of 8 participants heard tinnitus emerge in the sound booth. Percentages are based on this number.

Anechoic chamber

Sound	Females	Males	Total
Ring	7	1	8 (67%)
Hum	2	0	2 (17%)
Hiss	0	1	1 (8%)
Ocean	1	0	1 (8%)
Beep	0	1	1 (8%)
Cricket	0	1	1 (8%)
No sound	2/10	6/10	8/20

A total of 12 participants heard tinnitus emerge in the anechoic chamber. Percentages are based on this number.

Only 3 participants reported cricket, so a statistical comparison was not completed. Females most commonly heard ring and hum in both the sound-treated booth and anechoic chamber, whereas males most commonly heard ring and cricket in the sound booth. All of the eight participants who perceived tinnitus in the sound booth reported hearing the same types of tinnitus sounds in the anechoic chamber. Two of those participants heard multiple sounds in the sound booth and only heard one sound in the anechoic chamber.

Perceptual location. Table 3 shows the perceptual location of tinnitus in the sound booth and anechoic chamber. Out of the 8 participants who perceived tinnitus in the sound booth, five reported bilateral tinnitus (63%) and three reported unilateral tinnitus (37%, 2-right ear, 1-left ear). Out of the 12 participants who perceived tinnitus in the anechoic chamber, four reported bilateral tinnitus (33%) and eight reported unilateral tinnitus (67%, 5-right ear, 3-left ear). There was no significant difference in the perceptual location of tinnitus in the sound booth compared with the anechoic chamber ($\chi^2 = 1.65$, df = 1, N.S.).

Pitch. Of the eight participants who perceived tinnitus in the sound booth, six (9 ears total) were able to match the pitch of their tinnitus. Figure 5 shows the frequencies where tinnitus was perceived in the sound booth. One participant was unable to match the pitch because they no longer perceived the tinnitus. The other participant perceived their tinnitus was higher than 12.5 kHz, which exceeded the limit of the established test protocol. The pitch of tinnitus was perceived to be at 500 Hz in one out of the 9 ears (11%). Tinnitus was perceived to be at 1000 Hz in two ears (22%) and at 8000 Hz in two ears (22%). Tinnitus

Table 3.

Location of tinnitus

Perceptual location of tinnitus in the sound booth

Participants	Right ear	Left ear	Bilateral
Overall	2	1	5
Females	2	1	2
Males	0	0	3

Perceptual location of tinnitus in the anechoic chamber

Participants	Right ear	Left ear	Bilateral
Overall	5	3	4
Females	4	2	2
Males	1	1	2

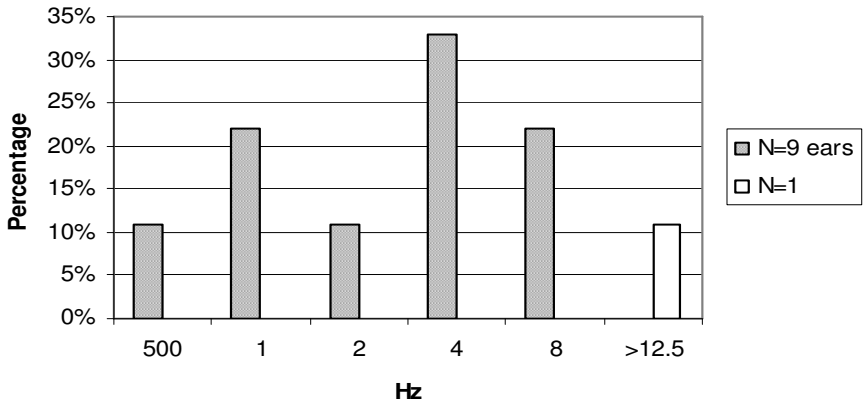

Figure 5. Tinnitus pitch match of the 6 participants (9 ears total) that were able to match their tinnitus pitch. One participant could not match due to limitations of the testing protocol, but noted that it was higher than 12.5 kHz.

was perceived to be at 2000 Hz in one ear (11%) and the largest percentage (33%) perceived their tinnitus to be at 4000 Hz.

Loudness. The 6 participants who were able to match the pitch of their tinnitus were also able to match the loudness of their tinnitus. Figure 6 displays the results of the loudness matching. Loudness measures were calculated based on the sensation level in decibels (dB SL) of the signal above threshold at the tinnitus frequency. Four out of the 9 ears (44%) perceived their tinnitus to be between 0-2 dB SL. Three out of 9 (33%) perceived their tinnitus to be between 3-5 dB SL, and 2 out of 9 (22%) perceived their tinnitus to be between 6-8 dB SL.

Masking. Four (5 ears) out of the 6 participants (9 ears) who were able to match pitch and loudness were able to mask their tinnitus. Two participants (4 ears) did not clearly understand the instructions, and therefore responded inaccurately, signaling when they first heard the white noise, not when their tinnitus stopped or changed. Even after they were reinstructed, the participants failed to complete the task. Figure 7 shows the amount of white noise masking that was needed, when presented to the tinnitus ear, to change or stop the perception of tinnitus. Masking levels were calculated in dB SL based on the participants white noise threshold. One participant (20%) reported that their tinnitus stopped or changed when white noise was presented to their tinnitus ear with an intensity of 0-3 dB SL, another (20%) at 4-6 dB SL, and another (20%) at 7-9 dB SL. Two participants (40%) reported that their tinnitus stopped or changed when more than 10 dB SL (14 dB SL) of white noise was presented to their tinnitus ear.

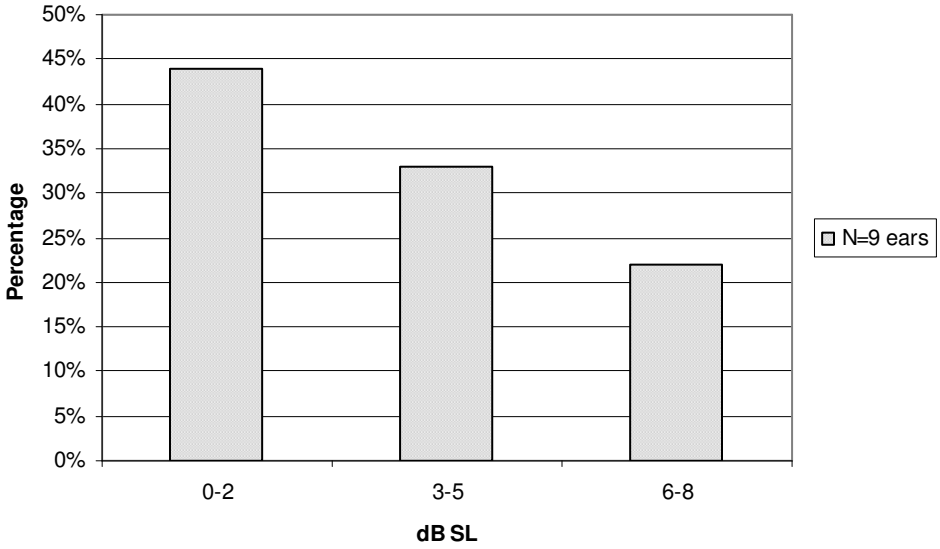

Figure 6. Loudness match at the tinnitus frequency. Six participants (9 ears total) were able to match the loudness of their tinnitus.

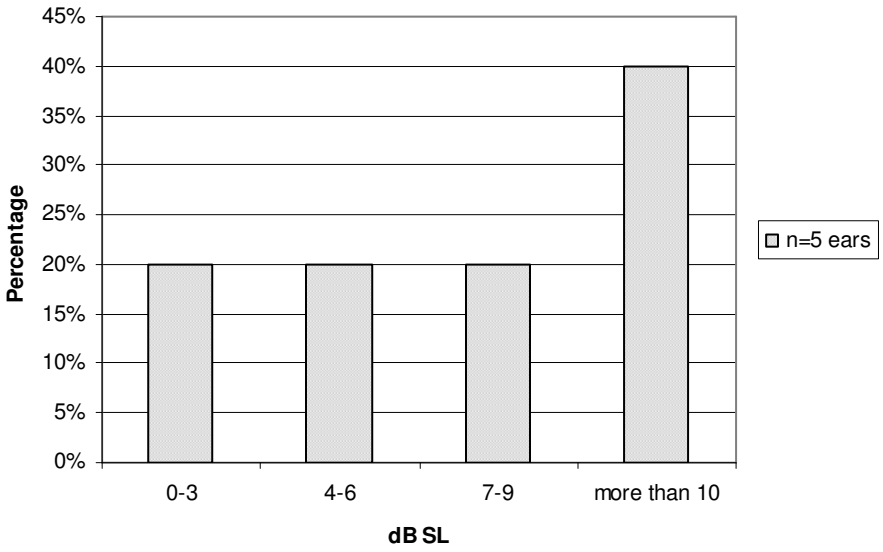

Figure 7. The minimum masking levels of the 4 participants (5 ears) who were able to mask their tinnitus.

Figure 8 shows the minimum masking levels for the 4 participants (5 ears) in relationship to their loudness matches. One ear (20%) with a loudness match of 0 dB SL needed 9 dB SL of white noise presented to their tinnitus ear in order to stop or change their tinnitus. Two ears (40%), one with a loudness match of 1 dB SL and one with a loudness match of 2 dB SL needed 14 dB SL of white noise in order to stop or change their tinnitus. One ear (20%) with a loudness match of 4 dB SL needed 5 dB SL of white noise and one ear (20%) with a loudness match of 7 dB SL needed 3 dB SL of white noise in order to stop or change their tinnitus. There was no significant correlation between the participant's loudness match and their minimum masking levels ($r = 0.219$, df = 3, N.S.).

Of the 8 participants who perceived tinnitus in the sound booth, 7 thresholds were re-checked after the psychoacoustical characterization of tinnitus was completed. One of the participant's thresholds was not re-checked because their perception of tinnitus did not last the entire time in the sound booth. Therefore, they could not participate in the psychoacoustical characterization of their tinnitus. The threshold differences between test and re-test of the right and left ears at each frequency tested (250-12.5 Hz) are shown in Table 4. A 2 x 2 x 8 repeated measures ANOVA was calculated (test, ear, frequency). A significant effect was found for frequency ($F(7,42) = 3.80$, $p < 0.001$), but not for test repeatability ($F(1,6) = 3.88$, N.S.) or ears ($F(1,6) = 2.54$, N.S.). A 2-way interaction between test and frequency is illustrated in Figure 9 by the crossing of test 1 and test 2.

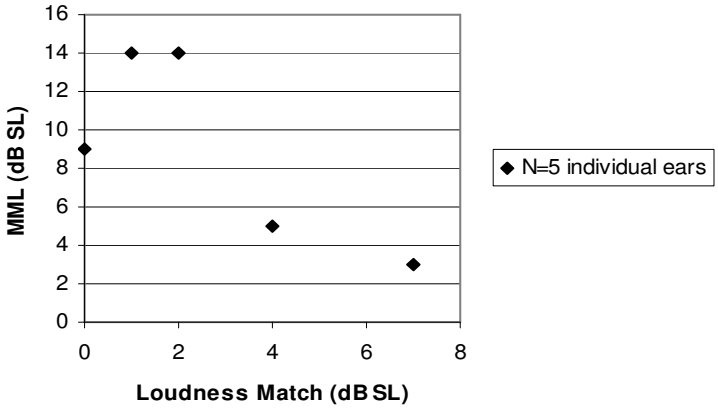

Figure 8. Loudness match and MML relationship between the 5 ears.

Table 4.

Difference in thresholds

	Right Ear								Left Ear							
	250	500	1k	2k	4k	8k	10k	12.5k	250	500	1k	2k	4k	8k	10k	12.5k
1	-2	-4	0	-2	2	-4	-2	-2	2	-6	0	-4	-4	0	-4	0
2	-6	-8	-4	4	-2	0	0	-6	-2	-2	8	-4	0	-2	-2	0
3	-2	-2	2	0	2	0	0	4	-2	0	0	0	0	2	4	4
4	0	-4	-2	-2	0	0	2	-2	-2	0	2	-2	2	0	-2	0
5	-2	-10	-2	-8	0	10	-12	-8	-2	2	2	2	4	-4	-2	8
6	-2	0	-2	0	2	0	0	4	-2	-2	0	0	2	2	0	0
7	0	-6	-4	0	6	2	2	-2	2	-6	-10	-4	2	-2	-4	2
Mean	-2	-4.9	-1.7	-1.1	1.4	1.1	-1.4	-1.7	-0.9	-2	0.3	-1.7	0.9	-0.6	-1.4	2
SD	1.9	3.2	2	3.4	2.3	4	4.5	4.2	1.8	2.8	4.9	2.2	2.4	2.1	.6	2.8

Note: Threshold differences before and after the 20 minute silent period in the sound booth from the 7 participants that perceived and participated in the psychoacoustical characterization of their tinnitus. Negative numbers indicate a decrease in threshold and positive numbers indicate an increase in threshold from the initial check.

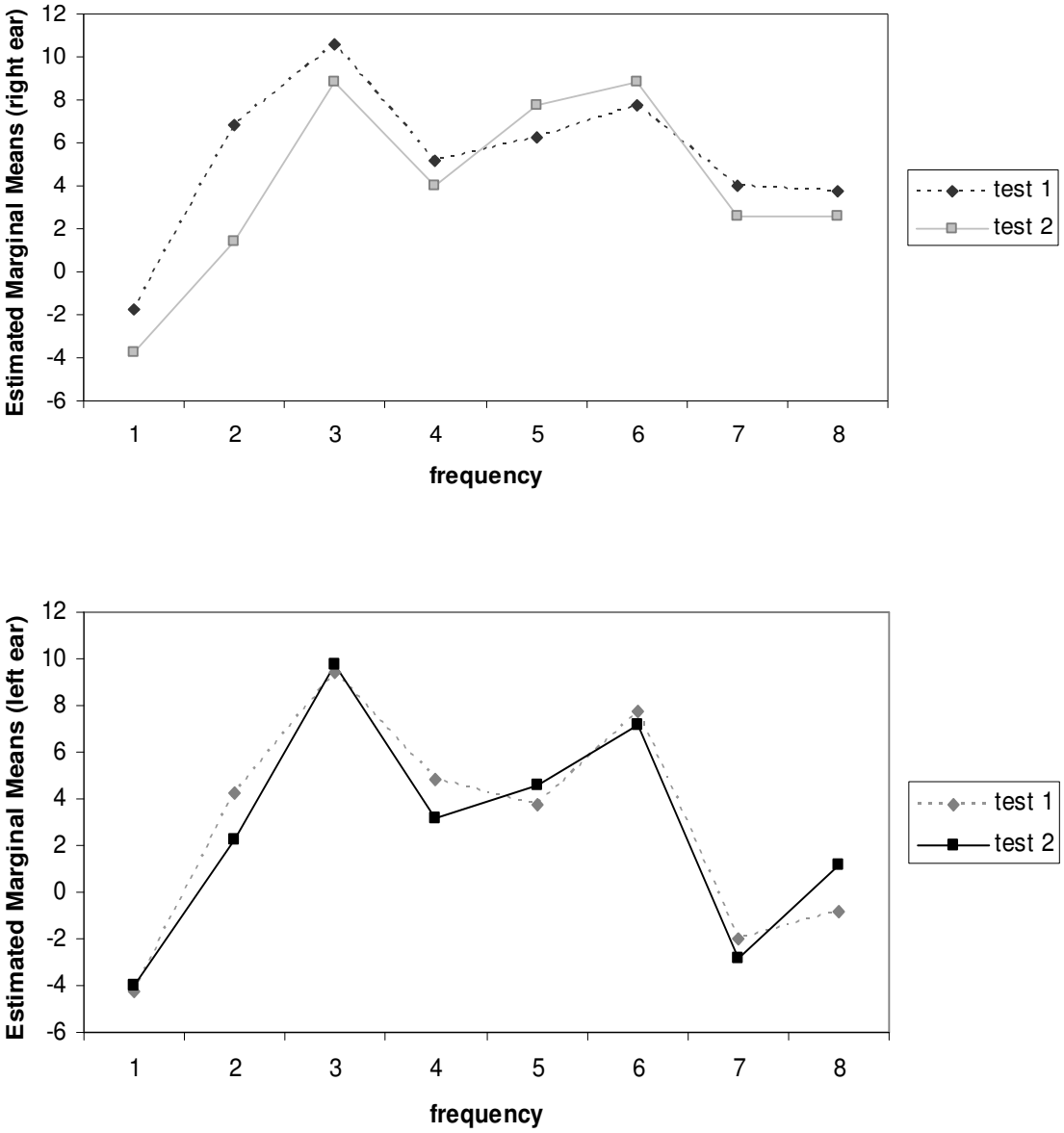

Figure 9. Estimated marginal means for the right and left ears.

CHAPTER 5

DISCUSSION

Comparison of tinnitus perception

Incidence. The ambient noise levels of the sound booth in the Tucker et al. (2005) study (24 dB at 125 Hz, 15 dB at 250 Hz, 6 dB at 500 Hz, 12 dB at 1000 Hz, 7 dB at 2000 Hz, 7 dB at 4000 Hz, and 6 dB at 8000 Hz) were higher than those reported for the sound booth in the current study and for the anechoic chamber in the lower frequencies up to 1 kHz. For the higher frequencies (2 kHz, 4 kHz, and 8 kHz), Tucker et al. (2005) reported lower ambient noise levels than those from the current study in both the sound booth and anechoic chamber above 2000 Hz. The ambient noise levels reported in Tucker et al. (2005) and those from the present study (sound booth and anechoic chamber) are shown in Figure 6. Tucker et al. (2005) did not report the ambient noise levels of frequencies lower than 125 Hz and greater than 8000 Hz. The higher levels of ambient noise in the higher frequencies in the present study may be a reason for the lower prevalence of tinnitus.

The overall perception of tinnitus in the present study (40% in the sound-treated booth and 60% in the anechoic chamber) was lower than the results from Heller and Bergman (1953) and Tucker et al. (2005), which were 94% and 64% respectively. This difference was not expected because the current study only included Caucasian adults previously found to have a higher prevalence of

tinnitus. When race was included as a factor in the study Tucker et al. found significant differences in tinnitus perception based on race. They found that 38% of African American participants experienced tinnitus compared to 78% (62 out of 80) of Caucasian participants. These results are still larger when compared to the current study, however not significant when compared to the results from the sound booth ($\chi^2 = 10.71$, df = 1, N.S.) or anechoic chamber ($\chi^2 = 2.55$, df = 1, N.S.). This difference in emergence may be attributed to the current study's small sample size (20 participants).

Tucker et al. (2005) also found that 31 out of 40 (78%) Caucasian females and 31 out of 40 (78%) Caucasian males experienced tinnitus. In the present study, 50% of the females and 30% of the males experienced tinnitus in the sound booth and 80% of the females and 40% of the males perceived tinnitus in the anechoic chamber. Although females had a higher incidence of tinnitus in both environments, there was no significant difference between the percentage of males and females who experienced tinnitus in either the Tucker et al. (2005) study or the current study in either the sound booth ($\chi^2 = .44$, df = 1, N.S.) or the anechoic chamber ($\chi^2 = 1.12$, df = 1, N.S.).

The current study found a difference in the percentage of participants experiencing tinnitus in the sound booth (40%) compared to the anechoic chamber (60%). Although this difference was not a significant factor with the current sample size, it is possible this difference may be attributed to the lower ambient noise levels in the anechoic chamber, especially in the frequencies surrounding 100 Hz (e.g. measured differences of 11 dB at both 63 Hz and 125

Hz). Also, the order in which the study was conducted (sound-treated booth first, followed by the anechoic chamber) may have attributed to the difference. Due to the design of this study and the location of the equipment, however, the sequence could not be altered. It was also revealed that 63% of the participants in the current study perceived tinnitus sooner in the anechoic chamber compared with the sound booth. Again, although not significant, this difference may be attributed to the lower ambient noise levels in the anechoic chamber and the order that the study was conducted. A larger sample size may have resolved the issue of the significance of this observation.

If subsequent research with a larger sample size indicates that the difference seen here is not random, it is possible this difference in low frequency background noise may contribute to the reason why some completely in the canal (CIC) and in the ear (ITE) hearing aid wearers complain of tinnitus. Because CIC and ITE hearing aids generally occlude or reduce low frequency sounds, wearers are losing some of the everyday background noises which may be masking the presence of tinnitus.

Sounds perceived. The mean number of sounds perceived by Caucasian participants in the Tucker et al. (2005) study (overall = 1.44, females = 1.39, males = 1.55) was similar to the mean number of sounds perceived by participants in the current study in the sound booth (overall = 1.63, females = 1.2, males = 2.33) and in the anechoic chamber (overall = 1.17, females = 1.25, males = 1). The most common sounds heard by Caucasian participants in the

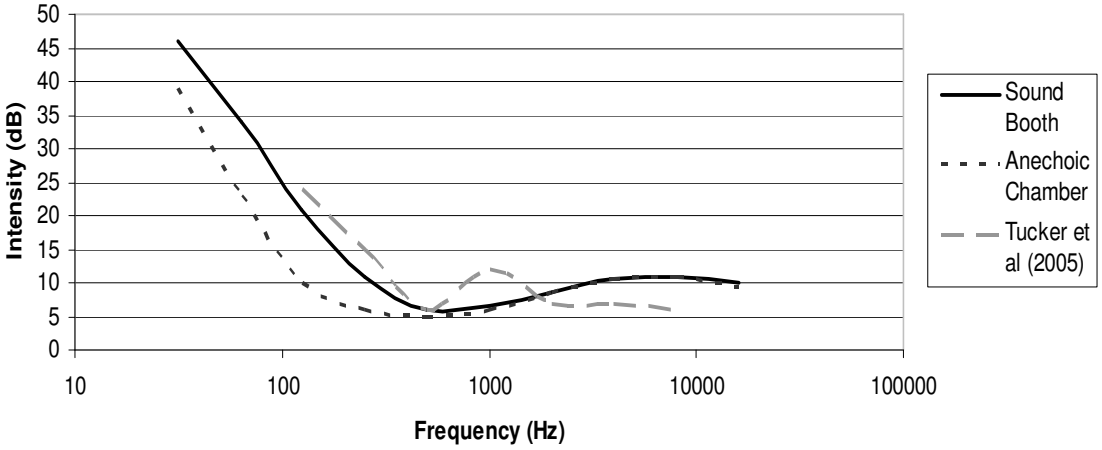

Figure 10. Comparison of ambient noise levels to Tucker et al. (2005)

Tucker et al. study were ring, hum, and heartbeat and the ring sensation (75% in the sound booth, 67% in the anechoic chamber) was also the most common sound heard in the present study.

The mean time of emergence of tinnitus in Caucasian participants in the Tucker et al. study (overall = 2.19, females = 3.19, males = 1.19) was shorter than the current study's mean time in the sound booth (overall = 5.25, females = 7, males = 2.33) and in the anechoic chamber (overall = 5.13, females = 4.89, males = 5.83).

Comparison to tinnitus patients

There are no known studies that analyze the psychoacoustic parameters of silence-induced tinnitus in normal hearing individuals. Therefore, there are no results to compare with the present study's findings. There are, however, psychoacoustic reports from patients who do have tinnitus. These reports will be used to compare the perceptions of tinnitus patients to the present study's findings.

Types of sounds. Stouffer and Tyler (1990) reported that the most common patient description of tinnitus was ringing (37.5%). The most common participant description in this study was also ringing (75% in sound booth, 67% in anechoic chamber), however it was not a significant finding in the sound booth or the anechoic chamber.

Location. The current study found that 63% of participants in the sound booth and 33% in the anechoic chamber experienced bilateral tinnitus. The Oregon Health and Science University's tinnitus data registry from 1981-1994

reported that 1026 out of 1629 (63%) tinnitus patients experienced tinnitus bilaterally. This finding is not significant for either the sound booth ($\chi^2 = 1.91$, df = 1, N.S.) nor anechoic chamber ($\chi^2 = 0.14$, df = 1, N.S.). The present study also found that 37% of participants in the sound booth and 67% in the anechoic chamber perceived tinnitus unilaterally. The Oregon Health and Science University reported that out of 1629 participants, 388 (23.8%) perceived tinnitus unilaterally. These reports from the current study were not statistically significant when compared to the sound booth, but were significant for participants' perception of location (e.g. right, left, bilateral) in the anechoic chamber.

Pitch match. The present study reported that the largest percentage of participants (33%) perceived their tinnitus to be at 4000 Hz. When compared to patients with tinnitus, Meikle (1995) reported that 33% of participants matched their tinnitus between 3500 Hz and 6499 Hz. Penner (2000) and Reed (1960) reported that the most commonly matched frequency was 3 kHz and Vernon (1987) reported that 83% matched tinnitus greater than 3 kHz. The Oregon Health and Science University also reported that 19.9% matched their tinnitus between 4 kHz and 5.9 kHz. Therefore, the present study's results are in general agreement with the literature.

Loudness match. The current study found that the largest percentage of participants perceived their tinnitus to be between 0-2 dB SL. This is in agreement with the Oregon Health and Science University that reported 40.2% of the tinnitus patients matched their tinnitus within 0-3 dB SL.

Minimum masking level. The present study found that the largest percentage (40%) of normal hearing individuals with silence-induced tinnitus were able to mask their tinnitus when white noise was presented higher than 10 dB SL (14 dB SL) to their tinnitus ear. This percentage is higher than the 7.5% reported from the Oregon Health and Science University and was a statistically significant difference. The reason for such a significant difference is not clear, but could be related to the difference in participants' thresholds or the length of time that participants experienced their tinnitus prior to the masking task.

Mitchell, Vernon, and Creedon (1993) reported a relationship between the patient's tinnitus loudness match and their minimum masking level. As the loudness match increased, the amount of masking needed to stop or change the tinnitus also increased. This relationship was not seen in the present study, and actually, the opposite was seen. Overall, as the loudness match increased the amount of masking needed to stop or change the tinnitus decreased. It is not clear why this relationship appeared.

Future research

It is clear from the results of this study and 2 previous studies that many normal-hearing listeners experience the emergence of tinnitus in silence and that the psychoacoustic characteristics of their tinnitus are similar to patients with tinnitus. However, the incidence of this phenomenon may not be as prevalent as the earlier studies suggested (Heller & Bergman; Tucker et al.). Therefore, more research is needed to further explore this phenomenon in a larger sample of normal hearing individuals, including factors such as age, race, and varying

degrees of hearing loss. Prior to the current study there were no known data on the psychoacoustic characterization of silence-induced tinnitus in normal hearing individuals. Additional data could potentially provide an insight as to the exact mechanisms of tinnitus and therefore allow audiologists to successfully help their clients and practice tinnitus management effectively.

APPENDICES

Appendix A

TOWSON UNIVERSITY

APPROVAL NUMBER: 06-A060

To: Jamie Walck

From: Institutional Review Board for the Proctection of Human Subjects Patricia Alt, Chair

Date: Wednesday, March 15, 2006

RE: Application for Approval of Research Involving the Use of Human Participants

Office of University Research Services

Towson University
8000 York Road
Towson, MD 21252-0001

t. 410 704-2236
f. 410 704-4494

Thank you for submitting an Application for Approval of Research Involving the Use of Human Participants to the Institutional Review Board for the Protection of Human Participants (IRB) at Towson University. The IRB hereby approves your proposal titled:

Transient Tinnitus in People with Normal Hearing Induced by Sound Deprivation

If you should encounter any new risks, reactions, or injuries while conducting your research, please notify the IRB. Should your research extend beyond one year in duration, or should there be substantive changes in your research protocol, you will need to submit another application for approval at that time.

We wish you every success in your research project. If you have any questions, please call me at (410) 704-2236.

CC: M. Jastreboff
File

REFERENCES

Bauer, C., & Brozoski, T. (2001). Assessing tinnitus and prospective tinnitus therapeutics using a psychophysical animal model. *Journal of the Association for Research in Otolaryngology, 2,* 54-64.

Bauer, C. (2003). Animal models of tinnitus. *Otolaryngologic Clinics of North America, 36*, 267-285.

Davis, A. & Rafaie, E. (2000). Epidemiology of tinnitus. In R. Tyler (Eds.), *Tinnitus Handbook* (pp. 1-23). San Diego, CA: Singular Press.

Dobie, R. (1999). A review of randomized clinical trials in tinnitus. *The Laryngoscope, 109,* 1202-1211.

Dobie, R. (2004). Clinical trials and drug therapy for tinnitus. In J. Snow, (Ed.), *Tinnitus: Theory and Management* (pp. 266-277). Hamilton, ON: BC Decker.

Formby, C. & Gold, S. (2002). Modification of loudness discomfort level: Evidence for adaptive chronic auditory gain and its clinical relevance. *Seminars in Hearing,* 1,21-34.

Formby, C., Sherlock, L., & Gold, S. (2003). Adaptive plasticity of loudness induced by chronic attenuation and enhancement of the acoustic background. *The Journal of the Acoustical Society of America, 114*, 55-58.

Goldreich, D. & Kanics, I. (2003). Tactile acuity is enhanced in blindness. *The*

Journal of Neuroscience, 23, 34-39.

Heller, A. (2003). Classification and epidemiology of tinnitus. *Otolaryngologic Clinics of North America, 36*, 239-248.

Heller, M. & Bergman, M. (1953). Tinnitus aurium in normally hearing persons. *Annals of Otology, Rhinology and Laryngology, 62*, 73-83.

Hoffman, H. & Reed, G. (2004). Epidemiology of tinnitus. In J. Snow, (Ed.), *Tinnitus: Theory and Management* (pp. 16-41). Hamilton, ON: BC Decker.

Jastreboff, P., Brennan, J., & Sasaki, C. (1988). An animal model for tinnitus. *Laryngoscope, 98, 280-6.*

Jastreboff, P. (1990). Phantom auditory perception (tinnitus); Mechanisms of generation and perception. *Neuroscience Research, 8*, 221-254.

Jastreboff, P. & Hazell, J. (1993). A neurophysiological approach to tinnitus: Clinical implications [Review]. *British Journal of Audiology, 27*, 7-17.

Jastreboff, P. & Hazell, J. (2004). *Tinnitus retraining therapy: Implementing the neurophysiological model.* (pp. 276). Cambridge University Press.

Jastreboff, P. (1995). Tinnitus as a phantom perception: Theories and clinical implications. In J. Vernon, & A. Moller (Eds.), *Mechanisms of tinnitus* (pp. 73-87). Massachusetts: Allyn & Bacon.

Jastreboff, P. (1996). Clinical implication of the neurophysiological model of tinnitus. In J. Vernon & G. Reich (Eds.), *Proceedings of the Fifth International Tinnitus Seminar,1995* (pp. 500-507). Portland, OR: American Tinnitus Association.

Jastreboff, P., Gray, W., & Gold, S. (1996). Neurophysiological approach to

tinnitus patients. *The American Journal of Otology, 17,* 236-240.

Jastreboff, P. & Jastreboff, M. (2003a). Decreased sound tolerance. In J. Snow, (Ed.), *Tinnitus: Theory and Management* (pp. 8-15). Hamilton, ON: BC Decker.

Jastreboff, P. & Jastreboff, M. (2003b). Tinnitus and hyperacusis. In J. Snow, & J. Ballenger (Eds.), *Ballenger's otorhinolaryngology head and neck surgery, 16th Edition* (pp.456-475). Hamilton, ON: BC Decker.

Jastreboff, P. & Jastreboff, M. (2003c). Tinnitus retraining therapy for patients with tinnitus and decreased sound tolerance. *Otolaryngologic Clinics of North America, 36*, 321-336.

Johnson, R., Brummett, R. & Schleuning, A. (1993). Use of alprazolam for relief of tinnitus: A double-blind study. *Archives of Otolaryngology Head and Neck Surgery, 119,* 842-845.

Meikle, M. (1995). The interaction of central and peripheral mechanisms in tinnitus. In J. Vernon & A. Moller (Eds.), *Mechanisms of Tinnitus* (pp. 181-206). Needham Heights, MA: Allyn & Bacon.

Mitchell, C., Vernon, J. & Creedon, T. (1993). Measuring tinnitus parameters: Loudness, pitch, and maskability. *Journal of the American Academy of Audiology, 4,* 139-151.

Moller, A., Moller, M., & Yokota, M. (1992). Some forms of tinnitus may involve the extralemniscal auditory pathway. *Laryngoscope, 102,* 1165-71.

Moller, A. (2000). Similarities between severe tinnitus and chronic pain. *Journal of the American Academy of Audiology, 11*, 115-124.

Moller, A. (2003). Pathophysiology of tinnitus. *Otolaryngologic Clinics of North America, 36*, 249-266.

Nelson, J. & Chen, K. (2004). The relationship of tinnitus, hyperacusis, and hearing loss. *Ear, Nose & Throat Journal, 83*, 472-476.

Oregon Health & Science University, (1995-2004). Tinnitus archive, second edition. Retrieved April, 27, 2006 from http://www.tinnitus archive.org/datasets/set-1/.

Penner, M. (2000). Spontaneous otoacoustic emissions and tinnitus. In R. Tyler (Eds.), *Tinnitus Handbook* (pp. 1-23). San Diego, CA: Singular Press.

Reed, G. (1960). An audiometric study of 200 cases of subjective tinnitus. *Archives of Otolaryngology, 71,* 84-94.

Rubinstein, J., Tyler, R., Johnson, A., & Brown, C. (2003). Electrical suppression of tinnitus with high-rate pulse trains. *Otology and Neurotology, 24,* 478-485.

Ruckenstein, M., Hedgepeth, C., Rafter, K., Montes, M., & Bigelow, D. (2001). Tinnitus suppression in patients with cochlear implants. *Otology and Neurotology. 22,* 200-204.

Simpson, J., Davies, W. (1999). Recent advances in the pharmacological treatment of tinnitus. *Trends in Pharmacological Sciences, 20,* 12-18.

Salvi, R., Saunders, S., Gratton, M., Arehole, S., & Powers, N. (1990). Enhanced evoked response amplitudes in the inferior colliculus of the chinchilla following acoustic trauma. *Hearing Research, 50*, 245-258.

Stouffer, J. & Tyler, R. (1990). Characterization of tinnitus by tinnitus patients.

Journal of Speech and Hearing Disorders, 55, 439-453.

Tonndorf, J. (1981). Stereociliary dysfunction, a cause of sensory hearing loss, recruitment, poor speech discrimination and tinnitus. *Acta Otolaryngologica (Stockholm.), 91*, 469-479.

Tondorf, J. (1987). The analogy between tinnitus and pain: A suggestion for a physiological basis of chronic tinnitus. *Hearing Research, 28*, 271-275.

Tucker, D., Phillips, S., Ruth, R., Clayton, W., Royster, E., & Todd, A. (2005). The effect of silence on tinnitus perception. *Otolaryngology Head And Neck Surgery, 132*, 20-24.

Tyler, R. (2000). The psychoacoustical measurement of tinnitus. In R. Tyler (Eds.), *Tinnitus Handbook* (pp. 149-179). San Diego, CA: Singular Press.

Vernon, J. (1987). Assessment of the tinnitus patient. In J. Hazell (Ed.), *Tinnitus* (pp. 71-95). Edinburgh: Churchill Livingstone.

Vernon, J. & Meikle, M. (2000). Tinnitus Masking. In R. Tyler (Eds.), *Tinnitus Handbook* (pp. 313-355). San Diego, CA: Singular Press.

CURRICULUM VITA

NAME: Jamie Walck

PERMANENT ADDRESS: 11800 Old Georgetown Rd.
Unit 1111
North Bethesda, MD 20852

PROGRAM OF STUDY: Audiology

DEGREE AND DATE TO BE CONFERRED: Doctor of Audiology, 2007

Secondary education: Lehighton Area High School, Lehighton, PA 18235

Collegiate institutions attended	Dates	Degree	Date of Degree
Pennsylvania State University	1999-2003	B.S.	May 2003
Towson University	2003-2007	Au.D.	May 2007

Major: Audiology