A Comparison of Multiple Measures of Quantifying Sensorineural Hearing Loss

A COMPARISON OF MULTIPLE MEASURES OF QUANTIFYING SENSORINEURAL HEARING LOSS

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Abstract

This master's thesis presents results from a study evaluating several different methods of assessing hearing loss. Twenty-seven participants with sensorineural hearing loss (SNHL) completed a combination of clinical, psychoacoustic, and objective tests. Each test chosen in this study yielded a measure of either inner hair cell loss (IHC) loss or outer hair cell (OHC) loss. The goal of this thesis was to evaluate whether variance in audiometric thresholds in patients with SNHL could be predicted based on different tests of specific hair cell loss. The tests chosen were included because each addresses specific deficits audiograms do not measure, such as holes in hearing and loudness growth. The tasks were the psychophysical tuning curve (PTC) task, psychoacoustic loudness growth, and an auditory brainstem response (ABR) loudness growth task. All tests were completed at 1000 Hz and at 4000 Hz and used an octave-band chirp stimulus. The chirp stimulus simultaneous increases signal to noise ratio, and decreases testing time.

It was hypothesized that audiometric thresholds could be predicted through some combination of tests that measure IHC and OHC loss. Since the ABR loudness growth task is an objective test, it was hypothesized that it would be a key predictor at both frequencies. We hypothesized that the subjective tests would only be predictors of audiometric thresholds at 1000 Hz.

We were able to significantly predict audiometric thresholds at 1000 Hz from 2 different regressions: 1) tip frequency (from the PTC task) and loudness ranking (from the psychoacoustic loudnesss growth task), as well as from 2) tip frequency and wave V amplitude (from the ABR loudness growth task). None of the predictors were able to explain audiometric threshold shifts significantly at 4000 Hz. In addition, ABR Wave V amplitude correlated with loudness ranking at 1000 Hz but not at 4000 Hz. However, ABR wave V amplitude correlated with stimulus intensity at both frequencies. This thesis provides evidence that ABR loudness growth tasks provide more information about patients compared to more subjective tasks.

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"If you wish to make an apple pie from scratch, you must first invent the universe." - Carl Sagan. I guess I need to thank the universe, without you, this thesis wouldn't have been possible.

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Declaration of Authorship

I, Ranya AMIRTHAMANOHARAN, declare that this thesis titled, “A Comparison of Multiple Measures of Quantifying Sensorineural Hearing Loss” and the work presented in it are my own. I confirm that I:

- Wrote each chapter.
- Conducted all experiments.

Chapter 1

Sound, Hearing, and Hearing Loss

1.1 Introduction

The main purpose of this master's thesis was to assess multiple measures of sensorineural hearing loss (SNHL) and to compare them to traditional methods of quantifying SNHL. As the number of people affected by hearing loss increases, there is an increased need to compare different methods to assess hearing loss. This study evaluated whether clinically common tests of hearing loss, air conduction audiograms, correlated with tests that are supposed to determine specific hair cell loss, the psychophysical tuning curve. We predicted that our specific tasks of inner hair cell and outer hair cell loss could be used to predict audiometric thresholds. The audiogram only conveys information on the total amount of hearing loss, but does not attempt to pinpoint whether the loss originates from inner hair cell or outer hair cell damage, or some combination of the two. Air and bone conduction audiograms attempt to differentiate between SNHL and conductive hearing loss, instead of delineating the different causes of SNHL. Since SNHL is much more common than conductive hearing loss, it is necessary to find methods to properly diagnose where in the pathway the deficits occur. Many of the methods used in this study are primarily used only in a research capacity and not in a clinical setting. Since SNHL is a complex deficit in hearing loss, multiple measures should be used in an attempt to better characterize the loss.

1.2 Sound

At birth, the human ear is fully developed and newborns are able to respond to both sounds that are faint and sounds that are loud. This perception of sound, or hearing, is a critical component of day to day life. Hearing is the process of detecting sound and assigning a meaning to it. It is important for communication, social relationships, and interacting with our world.

All sounds have a source. Sources can be many things, such as a baby crying, a loudspeaker or a tuning fork. From this source there is a displacement of particles in whichever medium the sound travels through. If the source is in water, it is a displacement of water particles; if it is in air, then it is a displacement of air particles. Then there is a series of condensations and rarefractions of air particles, which results in periodic changes of density in the medium. Through this mechanism, sound waves propagate through a medium such as air. Since sound is a physical property, it can be analyzed in terms of its physical features, primarily its frequency and amplitude. Frequency refers to the rate of air displacement, or how often the sound source vibrates. Frequency is measured in Hertz (Hz) which is cycles per second. Amplitude, which refers to how much air is displaced, can be measured in several different ways. Pascal is the unit of pressure fluctation from a sound wave and it is the SI unit of sound pressure. The pascal scale ranges from 1:10,000,000 and is too large to be practical with respect to human hearing. Thus a logarithmic scale, called the decibel scale (dB), is also used. The decibel scale relates the effective pressure of a sound to some reference value. For example, the dB sound pressure level (SPL) scale relates the measured sound pressure level of a sound to the lowest hearing threshold of a healthy ear. Frequency and amplitude are physical qualities that can be measured, and they relate to perceptual qualities that can only be measured subjectively as pitch and loudness. Frequency and amplitude are considered lower level features of sound.

1.3 General Hearing

The auditory system can be divided into two components: the peripheral auditory system and the central auditory system. The central auditory system primarily refers to the brain regions involved in auditory processing. The peripheral auditory system includes the ear and the auditory nerve. Lower level features, like frequency and amplitude, are encoded by the auditory periphery.

The ear in turn is divided into three parts: the outer ear, the middle ear, and the inner ear. Sound waves initially reach the outer ear and are funnelled by the auricle (pinna) to the auditory canal. As the sound waves travel down the auditory canal in the outer ear, sound waves in the range from 2000 – 7000 Hz are amplified by 15-20 dB (Yost, 1994). The tympanic membrane (ear drum) and the oval window are membranes that separate the outer ear from the inner ear. After sound waves travel through the auditory canal, the sound waves push against the tympanic membrane and cause it to vibrate. The middle ear, which is located behind the eardrum, contains three tiny ossicles, the incus, malleus and stapes, which vibrate in response to the eardrum's vibrations.

All structures discussed up to now are filled with air. However, past the ossicles is the inner ear, which is fluid filled. Since the density of the fluid is higher than the density of air, there is an impedance mismatch. This mismatch could result in 30 dB loss (Killion and Dallos, 1979). Thus, more pressure is needed to propagate the signal in the inner ear compared to the middle ear. The effective area of the eardrum is approximately 20 times larger than the effective area of the stapes' footplate (Poritsky, 2003). This large mismatch in size results in the sound pressure force acting on the tympanic membrane being greatly concentrated against the oval window. Thus, the impedance mismatch is overcome in the middle ear.

Both systems that have been described so far (the outer ear and middle ear) are linear systems. This means that they follow the scaling principle: if the input is scaled by a factor, the output must also be scaled by the same factor. The next section concerns the inner ear, or the cochlea, which is predominantly a nonlinear system.

1.4 The Inner Ear

The cochlea is an organ that is divided into 3 fluid filled chambers: the scala media, scala tympani, and the scala vestibuli. Reissner's membrane separates the scala media from the scala vestibuli, and the basilar membrane separates the scala media from the scala tympani.

When the stapes from the middle ear oscillates against the oval window, the fluid from the scala vestibuli flows to the scala tympani. This movement of fluid eventually leads to the the round window, another cochlear opening, being pushed outward. The oval window and round window movements lead a structural element inside the cochlea, called the basilar membrane, to vibrate.

The basilar membrane is narrower and more taut at the base compared to its apex, which is wider and less taut. These elastic properties allow high frequencies to resonate more easily at the apex and low frequencies to resonate more easily at the base. Thus, the basilar membrane is tonotopically arranged, with each position on the length responding preferentially to a different frequency (its characteristic frequency). A travelling wave moves along the basilar membrane starting from the base of the cochlea to the apex. The point of greatest excitation is the spot along the basilar membrane with the largest amplitude, and this spot is different for each frequency. After the point of maximal displacement, the amplitude of basilar membrane vibration greatly drops off.

On the basilar membrane is the organ of Corti, which is bound by the basilar membrane and the tectorial membrane. The organ of Corti contains tonotopically arranged hair cells and nerve fibers, meaning that they are arranged according to frequency. There are two types of hair cells, and they are structurally and functionally different from each other. Inner hair cells (IHC) innervate afferent nerve fibers, which transmit electrical signals to higher auditory centres. IHCs are involved in directly transmitting signals at their preferred frequency along the auditory pathway to the brain.

Outer hair cells (OHC) are innervated by the efferent nerve fibers. OHCs are attached to stereocilia imbedded in the tectorial membrane. OHCs are motile, and change in both length and stiffness in response to sound. The OHCs' changing physical properties affect the basilar membrane's response, thus creating an active mechanism. OHCs modulate the response of the auditory nerve fiber in a nonlinear manner, sharpening the tuning curve to provide contrast enhancement between neighboring frequencies, and compressing the loudness function. The IHCs are arranged in one row. There are 3500 IHCs, and 12000 OHCs divided into 3 rows. On top of both OHCs and IHCS are tiny stereocilia.

As the basilar membrane vibrates, the fluid in the scala media also moves, and the tectorial membrane moves in a back and forth (shearing) motion. The tectorial membrane shearing motion causes the stereocilia on the OHCs to move. The stereocilia on the IHCs move when the basilar membrane moves perpendicularly. The movements of the hair cells cause a transmission of electrical charge, and result in an electrical signal sent to the brain.

1.5 Hearing Loss

The range of frequencies that young adults can hear is between 20-20000 Hz (Yost, 1994). With age, adults typically lose sensitivity for higher frequencies. This age related hearing loss is known as presbycusis and it is the most common type of hearing loss. Hearing loss has very detrimental effects on an individual's standard of living. Hearing loss is implicated in social isolation, depression, anxiety and dementia (Weinstein and Ventry, 1982; Tambs, 2004; Lin, 2011) Older adults have a much higher risk for these conditions accompanying hearing loss.

There are two major classifications of hearing loss: conductive and sensorineural. Conductive hearing loss is defined as a problem with the conduction of sound waves to the inner ear from the outer ear and middle ear. Examples of conductive hearing loss include a major earwax buildup, or malfunctioning ossicles in the middle ear. This thesis is primarily concerned with sensorineural hearing loss (SNHL).

Sensorineural hearing loss is the more prevalent type of hearing loss and can be defined by damage to hair cells and/or to auditory nerve fibers. SNHL affects many adults, with its current prevalence at about 20%, with the amount affected only predicted to increase (Statistics Canada, 2014).

Discrimination between the two types of hearing loss can be done through comparisons of bone conduction and air conduction audiometry. Audiometry is the classic way clinicians test for hearing loss. The patient sits in a sound booth and the clinician plays different pure tones to the participant. The intensity level of the tone is increased or decreased until the threshold for each frequency is found. If this is done through headphones over the ear, it is air conduction audiometry, and if it is done through an oscillator placed behind the ear, it is bone conduction audiometry. Conductive hearing loss is characterized by the thresholds of bone conduction audiometry being significantly lower than the thresholds from air conduction. Since SNHL is typically caused by malfunctioning or dead IHCs and/or OHCs, but a relatively healthy middle and outer ear, this difference between audiometric thresholds should not occur if the hearing loss is pure SNHL. This thesis only deals with participants with pure SNHL.

1.6 Classical Ways to Measure SNHL

To some degree, SNHL can be treated with hearing aids. Hearing aids are fit to a prescription based on the patient's hearing loss profile, known as the hearing aid fit. The profile is made based on audiogram results. However, many participants who wear hearing aids complain that low level sounds are too quiet, and high level sounds are too loud. This is called loudness recruitment. In addition, patients with hearing aids often report they are unsatisfied, with many reports of patients not wearing them at all. While one possible explanation for the lack of satisfaction with hearing aids is that the technology is lacking. Another possible explanation is that the way we measure and fit the hearing aid is lacking.

Current methods for fitting hearing aids involve the patient going to a clinic to see an audiologist. The audiologist seats the participant in a sound booth, and an audiogram is administered. The audiologist plays pure tone signals, and the participant is asked to press a button when they can hear the signal. If they cannot hear the signal, the audiologist increases the level of the signal by 5 dB HL. If the participant can hear the signal, the level of the audiogram is decreased by 10 dB HL. This method is repeated for every frequency until the threshold for each frequency is obtained. Frequencies typically range from 250 Hz to 8000 Hz and signals are played to one ear at a time. As stated earlier, this can either be done in air or through bone.

Chapter 2

Shortcomings of Audiograms

2.1 Introduction

There are several problems with using audiograms to measure hearing loss. First, two patients with identical audiograms can have very different hearing aid correction requirements. Second, audiograms are very attentionally draining, and participants with cognitive deficits may not be able to keep sustained attention for the duration it takes to administer the task.

As mentioned earlier, SNHL is due to a combination of OHC and IHC damage. OHC damage can solely be responsible for hearing loss up to 55 dB HL, however any hearing loss above this involves both IHC and OHC damage (Moore, 2004). From an audiogram alone, an audiologist is unable to determine how much of the damage comes from IHC versus OHC loss. A physiological study on chinchillas showed that extreme degradation of IHCs has only a minimal effect on audiometric threshold shifts, and thus audiograms provide little information about hearing impairment versus IHC loss (Lobarinas, Salvi, and Ding, 2013).

Based on the shortcomings of the audiogram, we chose several different tests in an attempt to overcome the issues and to deduce whether the loss can be explained by various objective and subjective estimates of IHC and OHC damage. The next sections will further discuss specific deficits with audiograms, and briefly review the rationale for the experiments chosen.

2.2 Loudness Growth

Loudness is a psychological property that is related to sound intensity. Loudness is the perceived value of sound intensity, rather than the absolute value. Since loudness is a perceptual value, it varies from person to person. In participants with normal hearing, there is a non-linear relationship between loudness and sound intensity. However, this relationship depends on the frequency of the stimuli.

The measurements of loudness are sone and phon. The phon scale was created to directly compare the loudness of sounds that have different frequencies. The phon scale is a relative loudness matching scale, where sounds are compared and matched in loudness to a 1000 Hz pure tone. In contrast, a sone is the perceived loudness of a 1000 Hz pure tone that is presented at 40 dB SPL. For frequencies other than 1000 Hz, the sone scale is calibrated according to the response of individuals at that frequency. A doubling of the sone value equates to a doubling of the loudness, however the phon scale is not proportional in this manner.

Loudness growth is the relationship between stimulus intensity and perceived loudness. As mentioned earlier, hearing aids are typically fit with audiograms. However, audiograms only address one aspect of stimulus perception, the threshold of audibility. The audiogram does not assess perception of very loud sounds. Hearing impaired people may have very altered perceptions of very loud sounds. Loudness growth is an important characteristic to look at regarding hearing loss because humans do not only interact with sounds that are at threshold, instead the sounds vary from threshold to painfully loud. Thus, a hearing impaired patient's perception of loudness should be key in helping to restore their hearing ability back to normal.

Various psychoacoustic tests have been developed to try to assess how individuals perceive loudness. These tasks yield a loudness growth curve, a curve that plots perceived loudness versus stimulus intensity level for a given frequency. Individuals with normal hearing typically have a curve that is characterized by a linear relationship between noise intensity and perceived loudness, whereas those with hearing impairment sometimes have an abnormal loudness growth curve (Allen, Hall, and Jeng, 1990). An abnormal growth of loudness is characterized by low level sounds as being ranked as inaudible, and high intensity sounds being ranked as too loud, when compared to a person with normal hearing. However, it is important to distinguish abnormal loudness growth with hyperacusis. Hyperacusis is a condition where patients have a heightened sensititivy to certain frequencies and intensities of sound. Abnormal loudness growth is thought to reflect OHC damage, as OHCs are responsible for the compressive ability of the healthy ear (Burkard, Don, and Eggermont, 2006). Thus, both the psychoacoustic loudness growth task and the objective loudness growth task described in the next sections are a measure of OHC function.

2.3 Psychoacoustic Loudness Growth

There are many different procedures to measure loudness growth. Methods vary but typically require participants to listen to some stimulus and either rank it on how loud it is perceived to be, compare it to some other stimulus, or to quantify the loudness via some other metric (e.g. cut a piece of string for how long the stimulus is). With the lack of consensus of which method is best, we chose the most straight forward task to measure loudness growth. The method we chose presents participants different stimuli and they are asked to place the stimuli in one of several perceptual categories. Typical clinical studies use just three categories: threshold, most comfortable, and uncomfortable (Smeds and Leijon, 2011). However, we have chosen to include more perceptual categories so that we can infer a better loudness growth curve.

As stated earlier, cognitive deficits can affect behavioural measures of hearing loss like the audiogram, or the psychoacoustic loudness growth task. Since the majority of SNHL patients are older in age, and elderly patients often suffer from cognitive deficits such as reduced attention, it was necessary to include a objective measure of loudness growth, discussed further in the next section.

2.4 Auditory Brainstem Response

It has been suggested that psychoaccoustic tests of loudness growth are too time consuming and require too much effort on the part of participants. The results of these loudness growth curves rely heavily on the participant's ability to maintain sustained attention. Patients with SNHL are typically older in age, and often have some cognitive impairments that are related to old age. Thus, in addition to the behavioural loudness growth curve, we have chosen to employ an objective assessment of the loudness growth curve based on the auditory brainstem response (ABR).

The ABR is an objective electrophysiological measure that is both easy to record and represents the most important features of speech sounds. It is both a reliable and valid measure of auditory brainstem function. The ABR is a noninvasive measure, which makes it clinically feasible. To record the ABR, electrode stickers are placed on the participant. One electrode is placed on the vertex of participant's scalp (at location Cz), and this potential is compared to the signal from electrodes that are on the earlobe (reference) and forehead (ground).

The ABR measures a population neural response. The output of the ABR can be decomposed temporally into five identifiable wavefronts (waves I through V). Each wave is theorized to capture a different auditory process. The ABR potential is from the brainstem nuclei (Roeser, 2000). Waves I and II result from the auditory nerve, whereas wave III reflects cochlear nucleus activity, and wave IV originates from the superior olivary complex. Wave V results from the lateral lemniscus and terminates in the inferior colliculus (Roeser, 2000) or upper colliculous in the upper midbrain (McCandless, 2011). Each waveform can be identified based on the time at the which it appears relative to the stimulus onset, and its morphology. Our waveform of interest is the most commonly analyzed waveform, waveform V. Waveform V results from the inferior colliculus of the upper midbrain. Wave V is the most commonly analyzed wave and it provides the largest amplitude in response to stimuli. In addition, wave V amplitude and latency change predictably as the stimulus intensity and latency are changed.

2.4.1 Diagnosing Hearing Impairments with ABR

Currently, ABR is used as a screening measure of hearing loss in babies (Chalak et al., 2013). The reported sensitivity and specificity are 100% and 97% respectively, regarding detecting hearing loss in infants (Hall, 1987). In contrast, the false positive rate is 3.5% with one screening, and 0.2% with 2 screenings (Mason and Herrmann, 1998).

A major advantage to using ABR versus psychoacoustical tests is that it is passively evoked, and thus participants do not have to strain themselves or concentrate during recording. Instead, participants are encouraged to relax. Thus, the ABR serves as an accurate offline audiometric test of hearing ability that is importantly not subject to attention. In regards to ABR's reliability, there are strong correlations between test-retest with respect to peak latency, stimulus to response latency, frequency representation and response magnitude. This suggests that the ABR produces reliable responses to the same stimulus, making it an ideal tool for research into methods for auditory screening in the elderly and other special populations (Song, Nicol, and Kraus, 2011; Hornickel, Knowles, and Kraus, 2012).

In addition, it appears that the ABR can reliably predict speech impairments. For example, ABR analysis was able to discriminate participants who had poor perception of speech in noise from those who did not. Those with poor speech-in-noise perception had attenuated ABR responses (Anderson et al., 2011) compared to those with normal normal speech-in-noise perception.

The ABR is affected minimally by the participant's level of conscious awareness, thus making it a great tool for those with attention impairments. The ABR is a powerful tool for diagnosing hearing impairments, and does not require many active cognitive processes from the participant. Since we are using the same stimuli for the behavioural and objective loudness growth task, if we are able to find a strong correlation between the two methods of attaining a loudness growth curve, then perhaps this will eliminate the need to administer the cognitively laborious behavioural task.

2.4.2 Estimating Loudness Growth with ABR

Several features of the ABR make it an ideal tool to study auditory functioning. The ABR changes in predictable ways to changing the stimulus. The amplitude of the ABR is related to stimulus intensity. The latency of the response is also related to stimulus intensity, with a larger latency for lower level stimuli. The ABR appears to have all the characteristics that would allow it to be an objective method to assess loudness growth.

However, in practice the results are inconsistent. Some studies have found a relationship between ABR wave amplitude and perceived loudness if there is a flat hearing loss, but no relationship if the hearing loss is sloping (Serpanos, O'Malley, and Gravel, 1997).

A study comparing broad-band frequency click evoked ABRs with psychoacoustical loudness growth tests used wave V amplitude as a physiological estimate of loudness

(Davidson, Wall, and Goodman, 1990). The clicks varied in intensity from 10-100 dB SPL, with 8000 trials per session, and 4 sessions in total. They found a significant correlation between the click ABR estimate of loudness and psychoacoustic loudness growth task only when collapsing all trials together, but not within single sessions (Davidson, Wall, and Goodman, 1990). However, this correlation was less than desirable because it did not give us any frequency specific information. Patients with SNHL typically have high frequency hearing loss, thus I would expect an abnormal loudness growth curve at high frequencies, but not low frequencies. Since the click stimuli are broad band, we cannot test this hypothesis. For this reason, we did not use click stimuli in this thesis.

In a study by Gallego et al., (1999), participants with cochlear implants had ABR recorded while listening to 75 Hz pulse trains. Participants also completed a loudness scaling task. The goal of the study was to compare the electrophysiological measure of loudness to the behavioural measure, using the same stimulus. Loudness growth functions were obtained by averaging responses corresponding to each stimulus intensity. The results were that there was a significant effect of loudness on the amplitude of waves II and V. This study confirmed that there was a statistical relationship between ABR and perceptual thresholds at 75 Hz. However, the 75 Hz pulse train is not as important clinically because it is below the fundamental frequency of most voices and is on the lower threshold of frequencies young humans can hear. Furthermore, since hearing loss is associated with a high frequency loss, 75 Hz is perhaps too low in frequency to observe any differences in the hearing impaired ear compared to a healthy ear. Finally, the middle ear alters in-coming sounds that are in between 500-5000 Hz (Aibara et al., 2001). Sounds that are between 500-5000 Hz are the ones that are most efficiently transitted, and have a better ecological value. Thus, stimuli tested should reflect this range.

Silva, (2009) compared tone-burst ABR with various behavioural loudness growth curves. In their investigation, they used 1000 Hz and 4000 Hz stimuli, which are among the most clinically relevant frequencies. They used 5 dB peSPL steps, with the maximum intensity being 100 dB peSPL. They compared various physiological features (Wave V amplitude and latency) with the behavioural tasks. They found a low mean-square-error (MSE) between the two measures. However, this study took too long to conduct to be clinically feasible. The study used blocks of 1000 trials, with each block being repeated 8 times, for a total of 8000 trials. Each trial was 41.7 ms long, and with 18 different levels, resulting in the task taking 100 minutes per frequency per ear, not including set up time. It was not clinically feasible to devote over 200 minutes to recording just the ABR, and due to time constraints, in this thesis we chose 10 dB steps instead of 5, effectively halving the testing time.

The next section briefly describes current problems with the ABR stimuli mentioned earlier (clicks and tonebursts), and the improvements with chirp stimuli.

2.4.3 ABR Stimuli

When choosing ABR stimuli, there is a tradeoff between frequency specificity and signal to noise ratio (SNR). A very common stimulus used in ABR research is the click stimulus. Click stimuli are made of many different frequencies, and thus stimulate a large area of the basilar membrane. Due to this, click stimuli elicit large ABR responses. However, since they are broad-spectrum, they provide little information about frequency specific phenomena, like loudness growth. To contrast this, toneburst stimuli are frequency specific, but in turn excite a small portion of the basilar membrane, and thus yield a smaller ABR. Toneburst stimuli are known for having a low signal to noise ratio, and many trial repetitions must be administered to get a clean ABR reading (response).

The chirp stimulus addresses the issue of frequency specificity while attempting to improve the SNR, and thus yield a more robust ABR. To understand how, first we must recall a property of the cochlea that stems from its physiological organization: the basal regions of the cochlea are excited before the apical regions. Chirp stimuli attempt to make cochlear activity more synchronous to achieve a larger SNR. The chirp stimulus is created by time shifting the different frequency components (Elberling et al., 2007). The key being that the time shift is in opposite order of the time delay along the basilar membrane, with high frequencies being delayed more compared to lower frequencies (Elberling et al., 2007). The result is that many neural units are excited simultaneously, thus giving a stronger response. In addition, the chirp stimulus is octave band filtered, which provides frequency specificity.

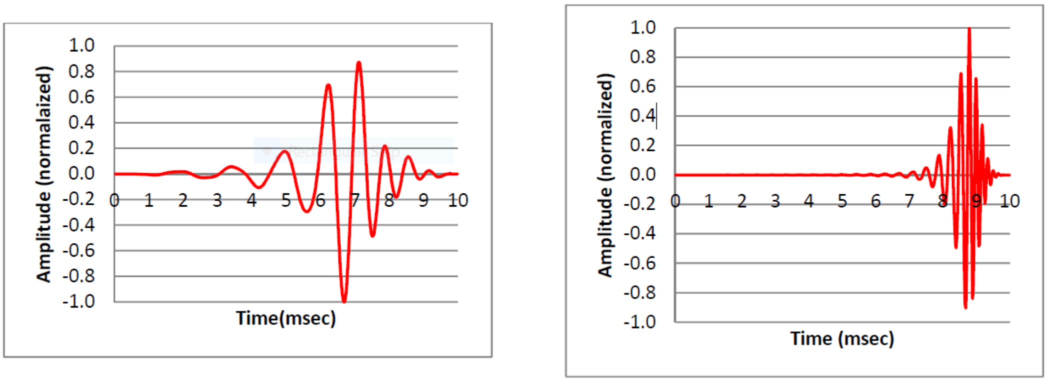


Figure 2.1: 1000 Hz center frequency octave band chirp stimulus (left) and 4000 Hz center frequency octave band chirp stimulus (right). Figures reproduced from Hoseingholizade, (2015).

Thus, the ABR is a reliable and objective measure of hearing loss and can be used to measure loudness growth. The results of this ABR loudness growth function reflect OHC loss. In addition, using chirp stimuli can improve SNR and decrease testing time, which is an improvement over the more common toneburst and click stimuli.

2.5 Holes in Hearing

Audiograms do not diagnose holes in hearing (Aazh and Moore, 2007; Moore and Alcántara, 2001; Halpin, 1994). Holes in hearing, also known as lucane, or cochlear dead regions, are regions of the cochlea where inner hair cells that correspond to a certain frequency of noise are damaged or dead (Moore and Alcántara, 2001; Moore, Glasberg, and Baer, 1997). Damage to IHCs results in the auditory nerve being less sensitive to stimulation, thus a more intense stimulus is needed to cause the basilar membrane to vibrate to threshold compared to normal. These dead regions can be compensated by off-frequency listening, where distal inner hair cells respond to the stimulation in lieu of IHCs at the dead region (Moore, 2004). This spread of response can be towards either the apical or basal region of the basilar membrane (Florentine and Houtsma, 1983; Moore, Glasberg, and Baer, 1997; Moore and Alcántara, 2001). Off-frequency listening is displayed when participants respond to hearing stimuli of certain frequencies, even though they have major cochlear damage at points that correspond to those frequencies. Off-frequency listening may result in hearing thresholds being over-estimated.

Holes in hearing are important to consider in patients with SNHL because they can affect the fit of hearing aids. Little benefit is received when gain is applied at dead regions (Moore and Alcántara, 2001; Vickers, Moore, and Baer, 2001). In addition, amplification of hearing aids in dead regions can cause major distortions which could explain the dissatisfaction many users report with their hearing aids (Amos and Humes, 2001; Ching, Dillon, and Byrne, 1998). An estimated 54-58% of patients with SNHL have dead regions, making them an important consideration when calibrating hearing aids (Moore, 2007). Since distal portions along the basilar membrane can compensate for dead regions, the audiogram can underestimate the extent of hearing loss in these regions. In addition, an individual with a normal looking audiogram can have dead regions. The current gold standard for detecting dead regions is using the psychophysical tuning curve, described in detail in detail below (Summers et al., 2003).

2.5.1 Psychophysical Tuning Curve

As mentioned previously, cochlear dead regions cannot be diagnosed from just an audiogram. Psychophysical tuning curves are currently the gold standard for diagnosing and identifying cochlear dead regions (Summers et al., 2003).

The sweeping psychophysical tuning curve (PTC) is a task where participants hear a sinusoidal signal over a narrowband masker. The general procedure for the PTC involves participants listening to a signal while a narrow band noise (masker) adaptively changes in intensity while sweeping the frequency region. When the participant reports that they can hear the signal, the level of the masker increases, when they report they cannot hear the signal, the level of the masker decreases. The signal stays stationary in both level and frequency, but cycles between on and off phases (beeps) to make it easier to detect. The level that the noise has to be to mask the signal is recorded for every centre frequency of the noise.

To obtain an accurate estimate of frequency specificity, forward and backward sweeps must be done, with an average of the two being taken. Taking the average of the forward and backward sweeps also diminishes the hysteresis effect, where a participant's response to change in perception will always lag behind their actual perception. Doing the PTC in only one direction will bias the responses to that one direction, so it is essential to do both forwards an backwards sweeps.

There are several different measures that can be obtained from the PTC, but the main properties are the the tip frequency, whether the frequency has been shifted, and the sharpness of the PTC. Before calculating the PTC's statistics, first the PTC curve must be calculated based on the two-point moving average. The more points in this average, the smoother the curve. The curve can be approximated by a quadratic function, or using double linear regression. The minimum of the quadratic fit or the intersection of the two lines in the double linear regression is the tip frequency.

It is expected that the masker is most efficient at the same frequency as the signal. Where the masker is most efficient refers to the frequency where the noise is best able to block the signal (the frequency with the lowest level noise) which is the the tip of the PTC. However, if the masker is most efficient at a distal region from the signal frequency, this implies that there is a dead region at that signal frequency. This can be viewed in the PTC by the tip-shift, a shift in the minima of the PTC from the signal frequency. The tip-shift is a measure of IHC function.

In addition to detecting IHC damage the PTC can also diagnose OHC damage. Hearing loss that is associated with damage to the OHCs can be shown physiologically with a nonlinear input/output (I/O) basilar membrane function. Perceptually, the patient will have a lack of frequency selectivity and sensitivity. Behaviourly, this can be seen with a psychophysical tuning curve that is broader than expected. Furthermore, OHC damage can be perceptually identified with difficulties hearing in noise, which is the basis of the PTC task.

The Q10 is a measure of tuning accuracy. The Q10 or bandwidth reflects the degree of OHC damage, with more damage resulting in broader tuning curves and lower Q10 values. The Q10 value is calculated by dividing the centre frequency by the bandwidth measured 10 dB above the lowest point on the tuning curve. A higher Q10 value suggests sharper tuning, and a lower Q10 value suggests a loss of frequency specificity. For a full review, please see Sęk and Moore, 2011.

In conclusion, audiograms are not an ideal measurement of hearing loss. While audiograms provide information about the minimum audible threshold, they provide no information about perception or comfort levels at high intensities. The audiogram reflects damage from both IHC and OHC degradation, but it is impossible to determine how much each contributes to the total loss. Based on the literature, the psychoacoustic and ABR loudness growth functions, and the PTC's Q10 estimate are supposed to reflect OHC loss, and the PTC's tip frequency reflects IHC loss. One of the goals of the present study was to investigate whether these subjective and objective measures of OHC and IHC loss could jointly predict threshold shifts in the audiogram, and if they could, which ones were the best predictors of audiometric thresholds. Another goal was to investigate whether there are sufficiently accurate objective measures, obtainable from the ABR, that can automatically diagnose IHC and OHC loss and could be applied in elderly people.

Based on evidence from the literature, we predicted that the loudness growth functions and the PTC's Q10 estimate will reflect purely OHC loss. In addition, we predicted that the tip frequency in the PTC will reflect IHC loss. Furthermore, we predicted that audiometric thresholds reflected an unknown combination of IHC and OHC loss, and thus combining a measure of OHC loss with a measure of IHC loss would separately predict some of the audiogram threshold but jointly would do a better job. Moreover, we predicted that ABR measures should be more accurate at reflecting hair cell loss compared to the subjective tasks.

Chapter 3

Methodology and Experiments

The purpose of this study was to compare several tests that measure IHC and OHC loss and to assess whether they can predict audiometric thresholds. The hypotheses were that we could predict audiometric thresholds from some combination of tests that measure IHC and OHC loss. In addition, we hypothesized that there would be a strong correlation between objective loudness growth (ABR) and loudness ranking (psychoacoustic loudness growth) for the low frequency case but not in the high frequency case. Finally, we predicted that ABR Wave V amplitude would correlate with the corresponding stimulus intensity at both the high and low frequencies, as it is an objective measure.

3.1 Participants

Forty-two adults participated in this study. The participants varied in age from 59-74 (mean=68, SD=4.53). Participants were recruited from the McMaster University Retiree Association (MURA) and with flyers throughout the Hamilton and Dundas community. Participants were prescreened through email to ensure that they did not have a cold, vertigo, tinnitus, any known middle ear pathologies, and had never had an ear surgery. This study was approved by the McMaster University Research Ethics Board.

3.2 General Procedure

First, the experimenter explained all procedures, protocols and inherent risks involved in the experiment. Participants then signed a consent form giving their consent to participate in the experiment. Next, participants all underwent screening protocols to ensure they had moderate sensorineural hearing loss. Participants were paid for their participation, offered light refreshments, and were encouraged to take breaks whenever needed. All tasks were conducted individually in sound attenuating rooms. The entire experiment took between 2.5 to 3 hours to administer.

3.2.1 Air Conduction Audiogram

All participants underwent pure-tone audiometry by completing an audiogram. The audiogram was completed according to the standard of the British Society of Audiology Audiology, 2008. The participants were in a sound booth with headphones on and heard pure-tones directed to one ear at a time. They were instructed to press and hold a button when they heard the tone. If the participants indicated that they heard the stimulus, the intensity of the stimulus was decreased by 10 dB (HL), however if the participants indicated that they could not hear the stimulus, the intensity was increased by 5 dB (HL). This procedure was repeated for each frequency (125, 250, 500, 1000, 2000, 3000, 4000, 5000, 6000, 8000 Hz), until the minimum audible threshold was found for each frequency. The air conduction audiogram took approximately 20 minutes to complete.

3.2.2 Bone Conduction Audiogram

To ensure that the ossicles in the middle ear were functioning normally, and to ensure that hearing loss was sensorineural in nature, bone conduction audiometry was conducted. A headphone-like device was placed behind the ear of participants. The device vibrated behind each individual ear. The same procedure as the air pure-tone audiometry was conducted. If the participant could hear the stimulus, the intensity was decreased by 10 dB HL. If they could not hear the stimulus, it was increased by 5 dB HL. If the thresholds from the bone-conduction and air conduction audiometry differed by more than 15 dB (HL) then, conductive hearing loss was assumed for the participant. The bone conduction audiogram took approximately 10 minutes to complete.

3.2.3 Tympanometry

To ensure the participants had normal middle-ear funtion, a tympanogram was performed according to the Jerger classification. A probe was presented to the right ear with a microphone recording the response produced by the ear. The machine produced a graph of the results, and only those participants with a normal type A tympanogram reading were included in the analysis. The tympanogram took approximately 5 minutes to administer.

3.2.4 Montreal Cognitive Assessment (MoCA)

Permission to use the Montreal cognitive assessment (MoCA) was granted by the creators. The MoCA is designed to detect cognitive impairments. The MoCA tests memory, attention, delayed recall, spatial orientation, language, and visuospatial abilities (Nasreddine et al., 2005). It has a high sensitivity and specificity, has been studied extensively, and has normative data. It is scored out of 30 and a score equal to or greater than 26 is considered to be within the normal range. The test was given to ensure participants did not have any detectable cognitive impairments.

A trained experimenter administered the full MoCA in English to all participants following the guidelines provided as closely as possible. The MoCA was conducted in an empty room to ensure the participant did not feel uncomfortable. The task took approximately 10 minutes to complete.

3.3 Behavioural Loudness Growth

Before the task began, the experimenter gave each participant a sheet of paper with 7 options: "TOO LOUD", "LOUD", "OKAY", "SOFT", "TOO SOFT", and "NO RESPONSE", with a brief description given for each categorical ranking. The participants were asked to read the paper to learn the different categorical rankings and were told to refer to the paper before ranking the stimuli.

Participants sat alone in a sound attenuated room and octave band chirp stimuli were presented through ear inserts to the right ear. The intensity of the chirps varied from 10 dB (nHL) to 70 dB (nHL) in 10 dB (nHL) steps, for a total of 7 different intensities. Each intensity was played 3 times to each participant, at each intensity. Each chirp stimulus was 50 ms in duration (10 ms chirp stimulus and 40 ms of silent interstimulus interval).

Stimuli were presented in two counter-balanced blocks: one 1000 Hz frequency block and one 4000 Hz frequency block, starting with a practice condition. Each stimulus intensity level was picked and presented randomly to each participant 3 times. After each stimulus presentation, participants were asked to rank the loudness of the stimulus using a touchscreen computer interface.

Participants took a 5 minute break between blocks. The task took approximately 25 minutes in total.

3.4 Recording Paradigm of the ABR Experiment

Participants heard the same chirp stimuli from the behavioural experiment during the ABR tests. All stimuli were presented to the right ear through ear inserts. The experiment was presented in two blocks: either 1000 Hz or 4000 Hz in frequency, with a 10 minute break in between. Participants watched "To Kill a Mockingbird" on mute with subtitles during the course of the experiment in a dimly lit room.

Each stimulus was presented for 4000 trials with a repetition rate of 20 stimuli/s, and each trial being a total of 50 ms in duration (10 ms chirp stimulus and 40 ms of silent interstimulus interval). Similar to the behavioural experiment, the stimulus intensity varied from 10 dB (nHL) to 70 dB (nHL) in 10 dB (nHL) steps, and was presented randomly.

Three electrodes were placed on each participant, one directly between the eyes, as the reference electrode, one behind the right ear, as the ground electrode, and the recording elctrode (Cz) at the mid-point of the head. Each electrode target spot was cleaned first with Nu-prep to clean the area of dirt and oil, and then covered in Sigma gel to make the area more conductive. The impedance was checked before recording, and recording only proceeded if the participant's impedance was less than 50 kilo-ohms.

Participants were instructed to watch the movie and to relax, but keep as still as possible. Recording took approximately 25 minutes per frequency and 15 minutes to set up.

3.5 Psychophysical tuning curve (PTC)

This task was programmed according to the instructions of Sęk and Moore, (2011). All stimuli were presented to the participant's right ear through Sennheiser HDA 250 headphones. The signal frequencies were the same as the loudness growth experiments: 1000 Hz and 4000 Hz. The signal pulsed on and off at 0.2 sec on/off. The level of the signal was 10 dB (SPL) higher than the participant's audiometric threshold for the corresponding frequency. The bandwidth of the masker was 250 Hz.

Participants heard the instructions for the task from the experimenter, in addition to having them displayed on the computer screen. Participants were instructed to press and hold the spacebar key for as long as they heard the signal and to let go of the spacebar key if they did not hear the signal. The level of the masker increased or decreased at a rate of 2 dB/sec. This continued until the marker's centre frequency spanned the whole frequency region being tested.

Both forward and backward sweeps were conducted for each frequency to diminish the hysteresis effect mentioned earlier. The experiment took approximately 5 minutes per frequency per direction, which was approximately 20 minutes for all cases.

Chapter 4

Results

4.1 Prescreening Measures

Of the 42 participants recruited for this study a total of 27 passed all prescreening measures: 5 participants had conductive hearing loss instead of SNHL according to their air and bone conduction thresholds, 4 participants had abnormal tympanograms, and 6 participants were excluded from the statistical analysis for having abnormally low MoCA scores.

4.2 Psychoacoustic Loudness Growth

All 27 participants completed the psychoacoustic loudness growth task.

4.2.1 1000 Hz

The group-averaged behavioural loudness growth curve at 1000 Hz for all 27 participants who passed the prescreening procedure can be seen in Figure 4.1. The geometric means from the psychoacoustic loudness growth task are plotted against the stimulus intensity level. The correlation was very large and highly significant, (Rho=0.964, p=0.002778).

The individual behaviour loudness growth curves for each individual participant are in Figure 4.2. All participants had a significant correlation between their loudness ranking and the stimulus intensity except participants 3, 4, 6, 9, 13, 14, 18, 20, and 21. For all individual psychoacoustic loudness growth curves, see Figure 4.2 and Table 4.1 for their respective correlation coefficients and p-values.

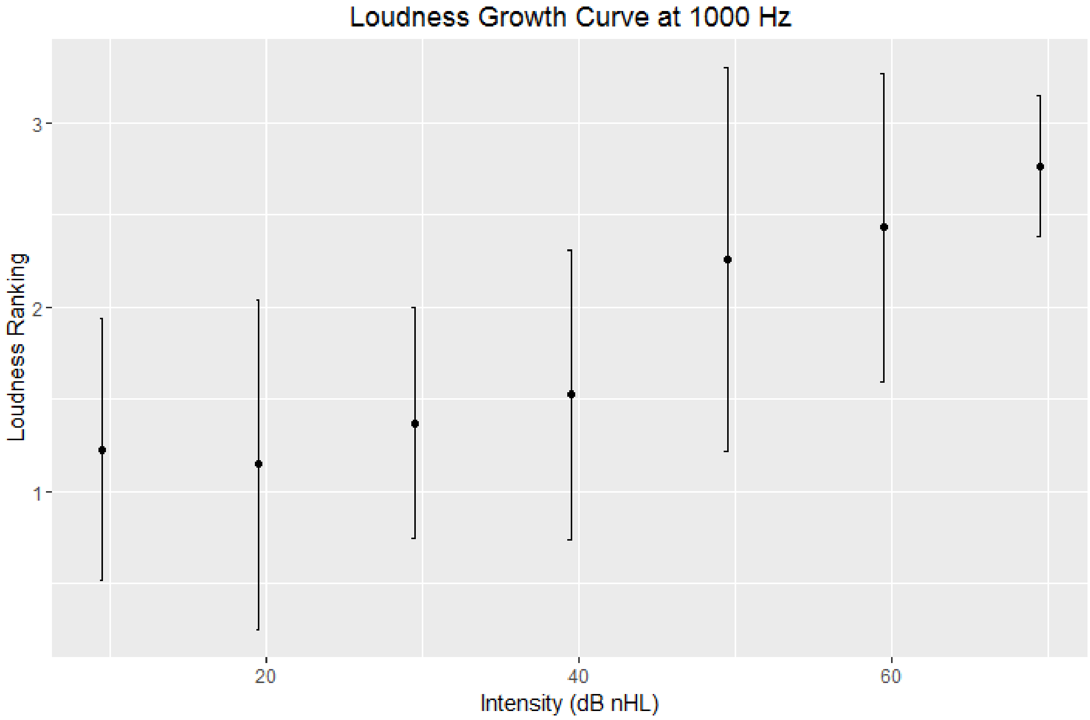
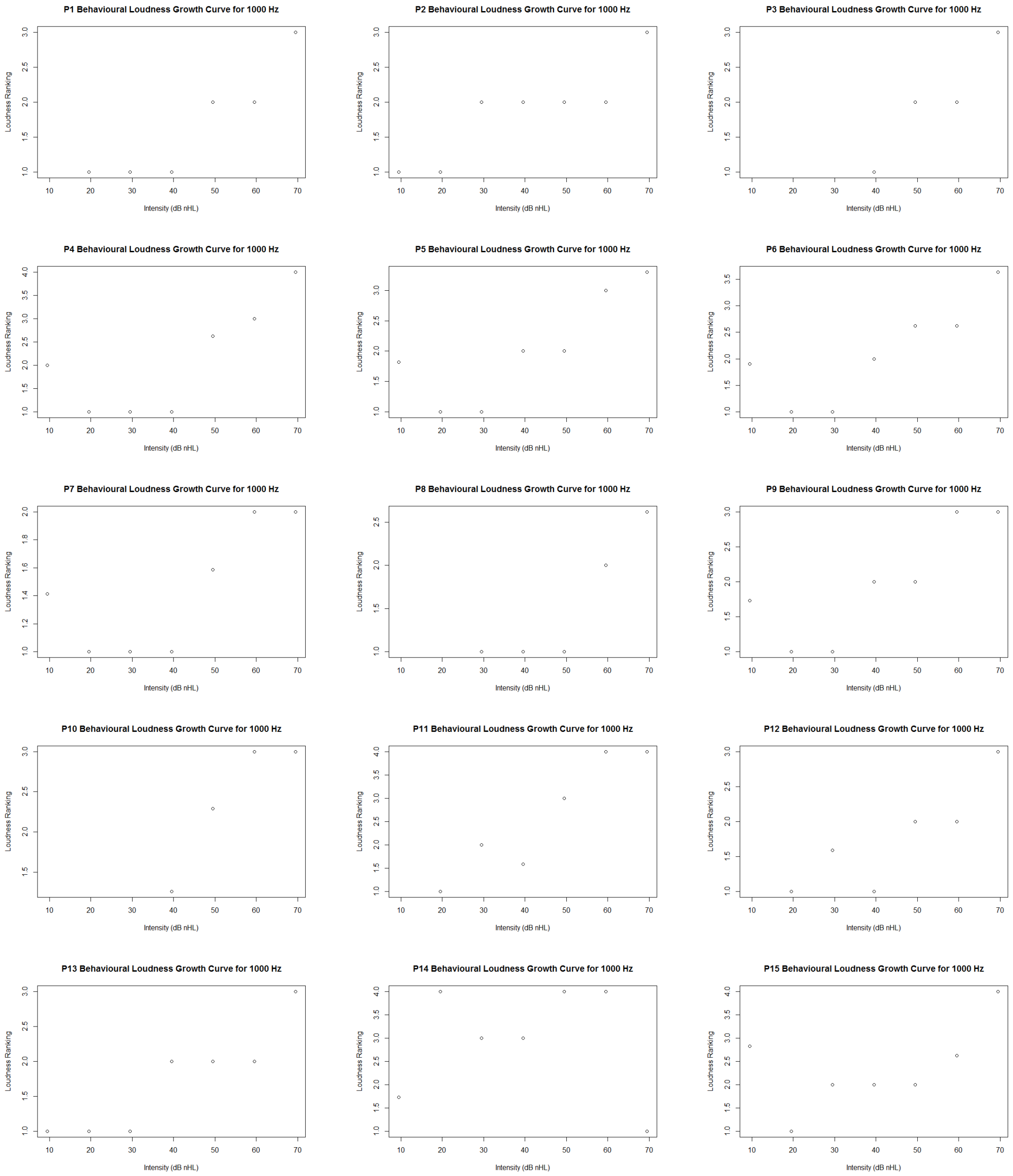


Figure 4.1: The averaged behaviour loudness growth curve for all participants at 1000 Hz. The geometric mean of loudness scaling was calculated across all participants for each stimulus presentation level, (Rho=0.964, p=0.002778). Error bars are ±1 stdev.



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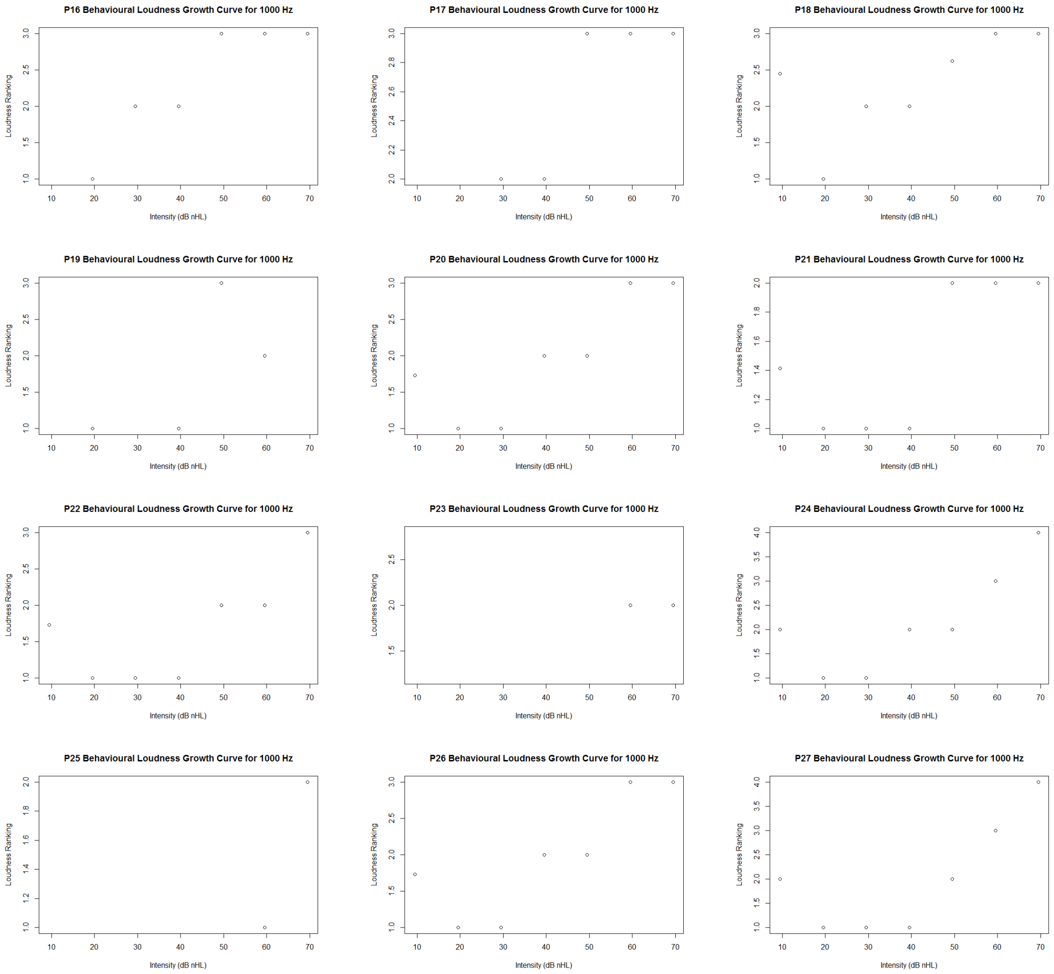


P16 Behavioural Loudness Growth Curve for 1000 Hz

P17 Behavioural Loudness Growth Curve for 1000 Hz

P18 Behavioural Loudness Growth Curve for 1000 Hz

P19 Behavioural Loudness Growth Curve for 1000 Hz

P20 Behavioural Loudness Growth Curve for 1000 Hz

P21 Behavioural Loudness Growth Curve for 1000 Hz

P22 Behavioural Loudness Growth Curve for 1000 Hz

P23 Behavioural Loudness Growth Curve for 1000 Hz

P24 Behavioural Loudness Growth Curve for 1000 Hz

P25 Behavioural Loudness Growth Curve for 1000 Hz

P26 Behavioural Loudness Growth Curve for 1000 Hz

P27 Behavioural Loudness Growth Curve for 1000 Hz

Figure 4.2: Behaviour loudness growth curves for each individual participant at 1000 Hz. The geometric mean of the participant's loudness ranking was calculated for each stimulus presentation intensity. The correlation coefficients and p-values for each respective participant can be found in Table 4.1. For full size figures, please refer to the Appendix 6.

Participant	Rho	p-value
1	0.9258201	0.00805
2	0.896	0.006267
3	0.94868	0.05132*
4	0.741249	0.05658*
5	0.87287	0.01032
6	0.87287	0.06264*
7	0.729769	0.04052
8	0.8627	0.01244
9	0.94868	0.05132*
10	0.9276	0.007666
11	0.8827	0.01982
12	0.9258	0.002765
13	-0.018712	0.9682*
14	0.4076871	0.3639*
15	0.9258201	0.00805
16	0.8660254	0.05767*
17	0.76376	0.04566
18	0.73786	0.2621*
19	0.86271	0.01244
20	0.6943651	0.08344*
21	0.72976	0.06264*
22	0.7307	0.04264
23	0.7671932	0.04411
24	0.7671932	0.04411
25	0.862712	0.01244
26	0.6736331	0.0971*
27	0.6736331	0.0171

Table 4.1: The correlation coefficients and corresponding p-values for each participant's behaviour loudness growth curve at 1000 Hz. Rho is Spearman's correlation coefficient between loudness scaling and stimulus intensity. Asterisk indicates the the p-value was not significant. Each participant's curve can be see in Figure 4.2 .

4.2.2 4000 Hz

The behaviour loudness growth curve at 4000 Hz for all 27 participants who passed the prescreening procedure can be seen in Figure 4.3. The geometric means from the psychoacoustic loudness growth task are plotted against the stimulus intensity level. The correlation was not significant, (Rho=0.214, p=0.6615).

The individual behaviour loudness growth curve for each individual participant are in Figure 4.4. All participants had a significant correlation between their loudness ranking and the stimulus intensity except participants 3, 4, 6, 9, 13, 14, 18, 20, and 21. For each participant's respective correlation coefficient and p-value, see Table 4.2.

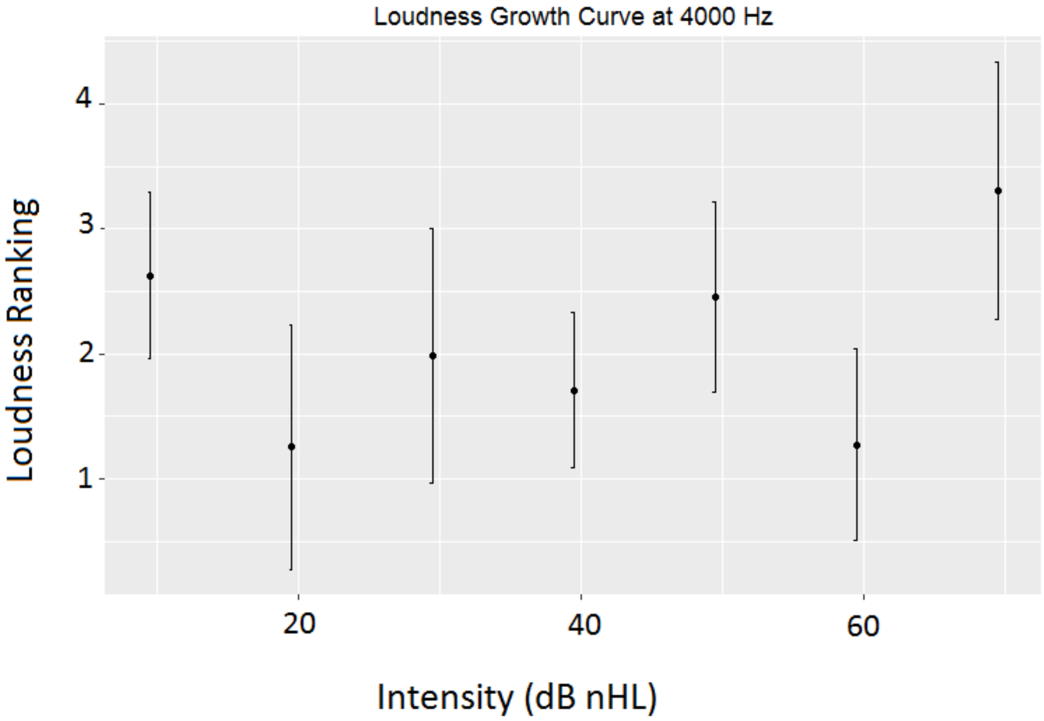
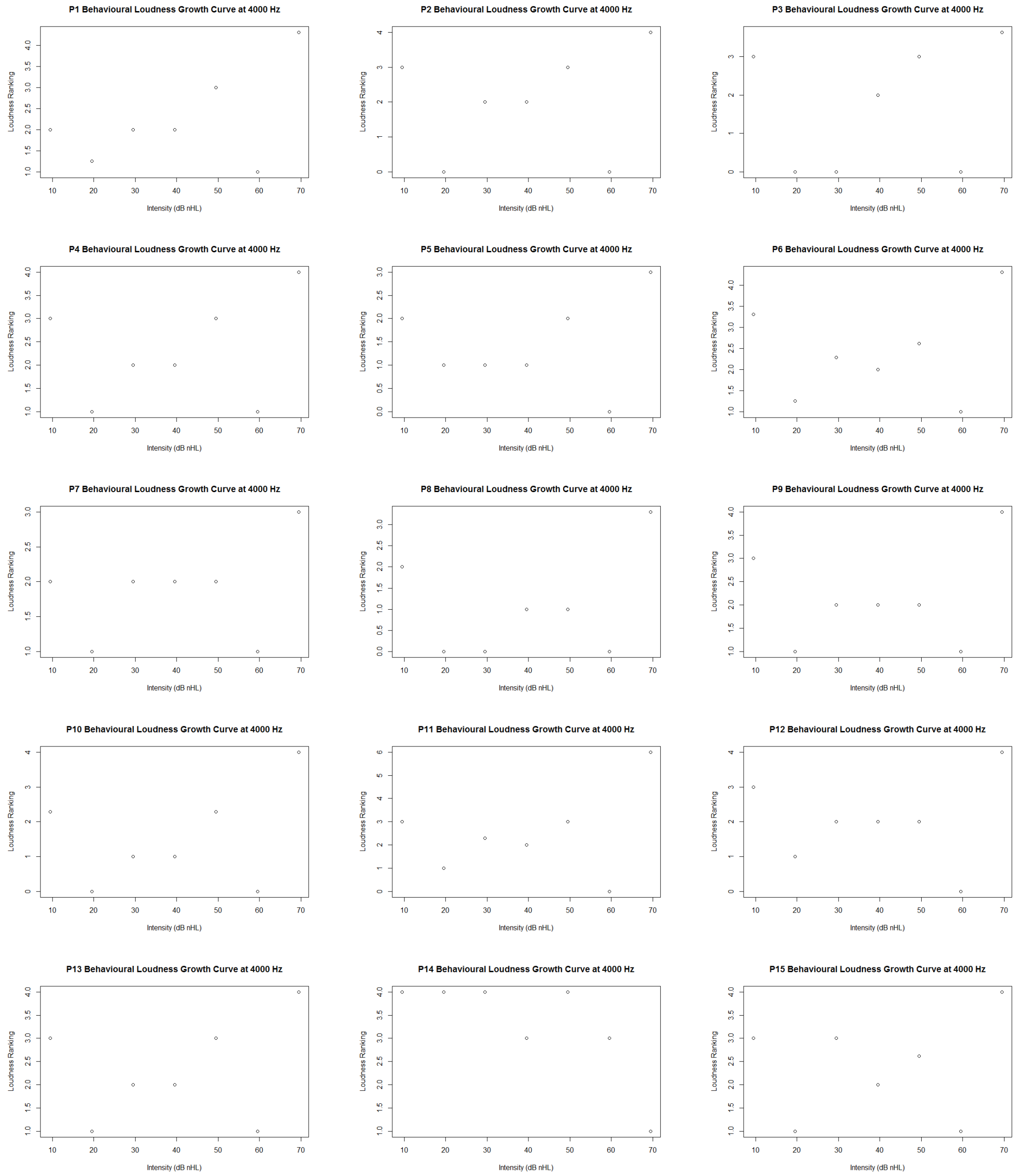


Figure 4.3: The averaged behaviour loudness growth curve for all participants at 4000 Hz. The geometric mean of loudness scaling was calculated across all participants for each stimulus presentation level, (Rho=0.214, p=0.6615). Error bars are ±1 stdev.



P1 Behavioural Loudness Growth Curve at 4000 Hz

P2 Behavioural Loudness Growth Curve at 4000 Hz

P3 Behavioural Loudness Growth Curve at 4000 Hz

P4 Behavioural Loudness Growth Curve at 4000 Hz

P5 Behavioural Loudness Growth Curve at 4000 Hz

P6 Behavioural Loudness Growth Curve at 4000 Hz

P7 Behavioural Loudness Growth Curve at 4000 Hz

P8 Behavioural Loudness Growth Curve at 4000 Hz

P9 Behavioural Loudness Growth Curve at 4000 Hz

P10 Behavioural Loudness Growth Curve at 4000 Hz

P11 Behavioural Loudness Growth Curve at 4000 Hz

P12 Behavioural Loudness Growth Curve at 4000 Hz

P13 Behavioural Loudness Growth Curve at 4000 Hz

P14 Behavioural Loudness Growth Curve at 4000 Hz

P15 Behavioural Loudness Growth Curve at 4000 Hz

Figure continued on next page.

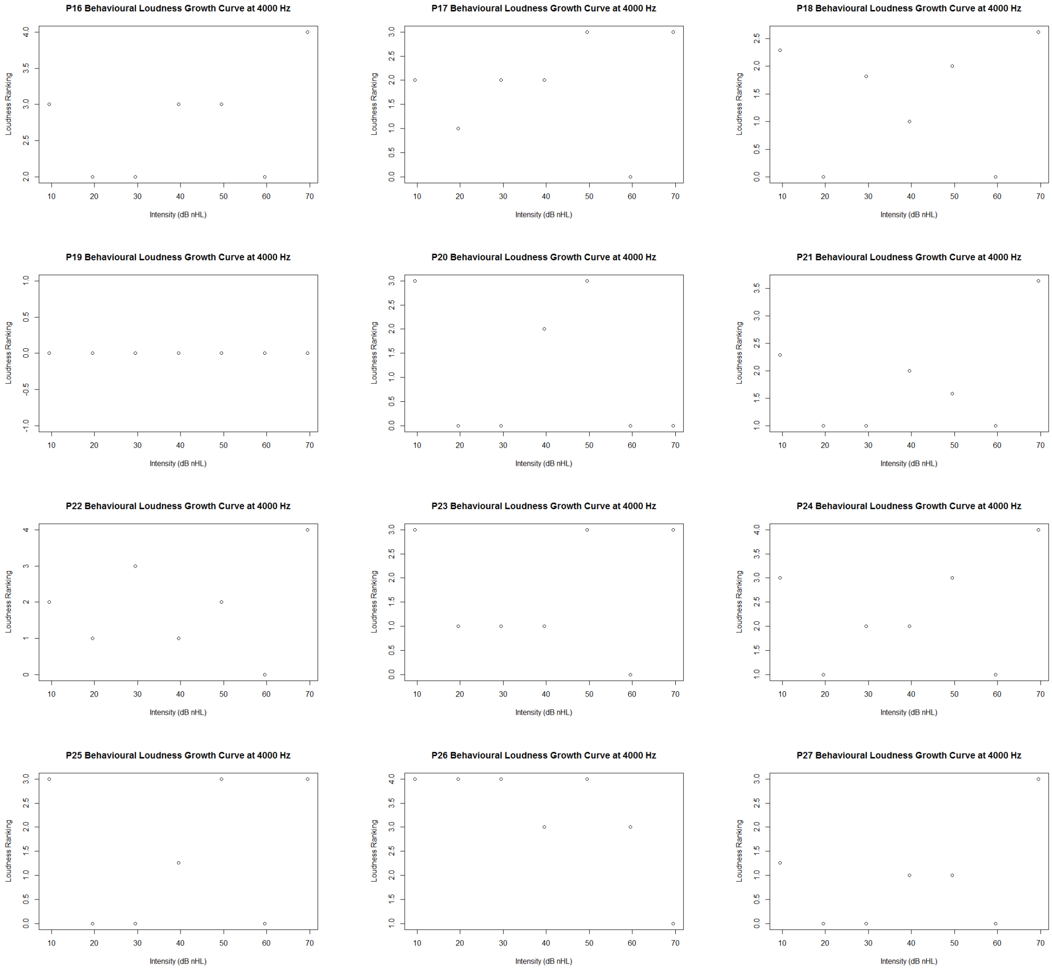


Figure 4.4: Behaviour loudness growth curves for each individual participant at 4000 Hz. The geometric mean of the participant's loudness ranking was calculated for each stimulus presentation intensity. The correlation coefficients and p-values for each respective participant can be found in Table 4.2. To see full sized figures for each participant, please refer to Appendix 6.

Participant	Rho	p-value
1	0.3335622	0.4647
2	0.2386225	0.6063
3	0.2993925	0.5142
4	0.2386225	0.6063
5	0.1122722	0.8106
6	0.071428	0.9063
7	0.2988	0.5151
8	0.2058323	0.6579
9	0.1122722	0.8106
10	0.2386225	0.6063
11	0.14415	0.7578
12	0.03706	0.9371
13	0.2386225	0.6063
14	-0.7768986	0.03988*
15	0.1091	0.8159
16	0.34718	0.4455
17	0.29939925	0.5142
18	0.14415	0.7578
19	-0.31872	0.486
20	0.1853123	0.6908
21	0.1091089	0.8159
22	-0.0385784	0.9346
23	0.2886225	0.6063
24	NA	NA
25	-0.7768986	0.03988*
26	0.2058323	0.6569
27	0.358323	0.3569

TABLE 4.2: The correlation coefficients and corresponding p-values for each participant's behaviour loudness growth curve at 4000 Hz. Rho is Spearman's correlation coefficient between loudness scaling and stimulus intensity. Asterisk indicates significant p-values. Each participant's curve can be see in Figure 4.4. Participant 24 did not complete the experiment.

4.3 ABR Loudness Growth

Of the 27 participants who satisfied the prescreening criteria, only 17 completed the ABR: 4 participants left because they were uncomfortable with an ABR recording, 2 participants had impedances that were too high and could not be rectified, and 4 participants could not be included in the statistical analysis because of too much movement during the recording.

4.3.1 1000 Hz

The ABR loudness growth curve averaged across all 17 participants can be seen in Figure 4.5. This figure plots the mean of all participant Wave V amplitude against the corresponding stimulus intensity level. The correlation was significant (Rho=0.9285714, p=0.006746). The individual ABR loudness growth curves for all participants are plotted in Figure 4.6. The corresponding correlation coefficients, p-values, and power fit exponent can be found in Table 4.3. Power exponent, x, is found for the function y = $ab^x + c$, where y is Wave V amplitude, and b is stimulus intensity.

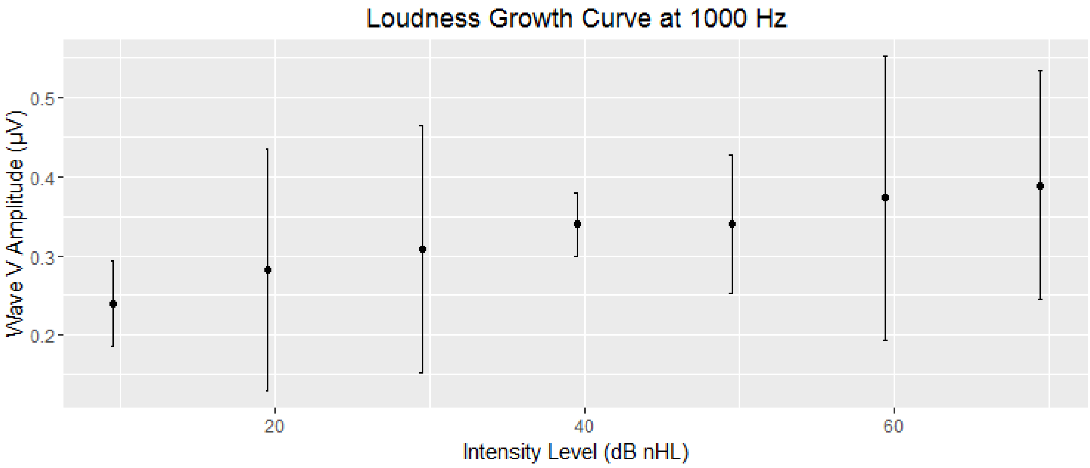
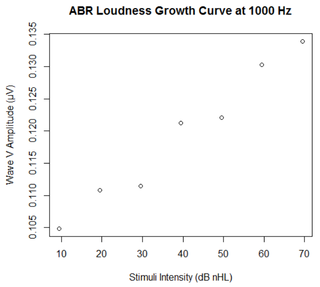
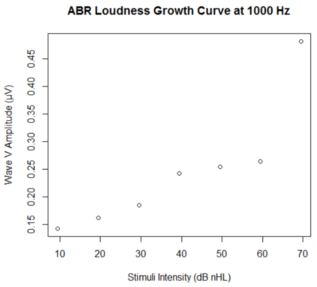
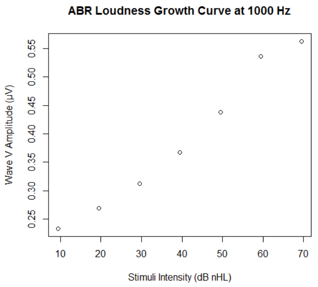
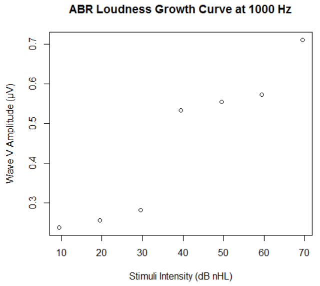


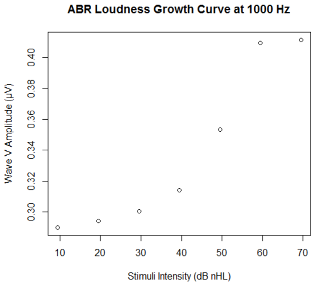
FIGURE 4.5: The averaged ABR loudness growth curve for all participants at 1000 Hz. The geometric mean of Wave V amplitude was calculated across all participants for each stimulus presentation level, (Rho=0.9285714, p=0.006746). Error bars are ±1 stdev.

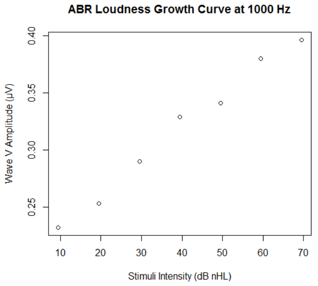

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.105
0.110
0.115
0.120
0.125
0.130
0.135
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

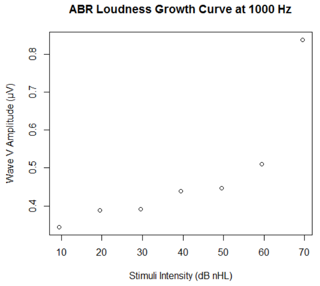

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.15
0.20
0.25
0.30
0.35
0.40
0.45
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

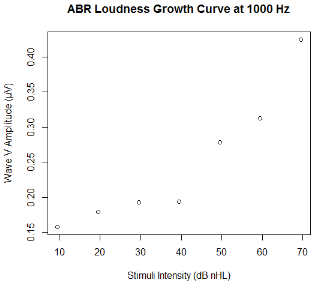

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
0.45
0.50
0.55
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

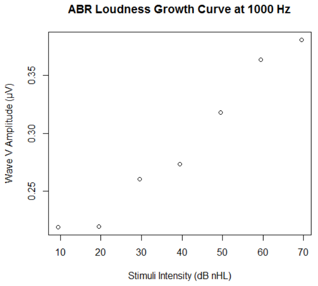

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.3
0.4
0.5
0.6
0.7
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

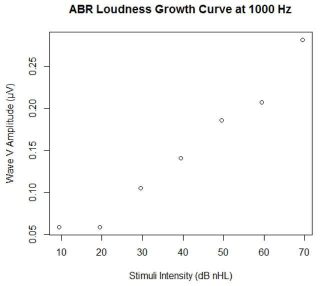

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.30
0.32
0.34
0.36
0.38
0.40
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

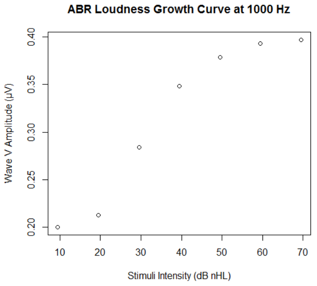

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

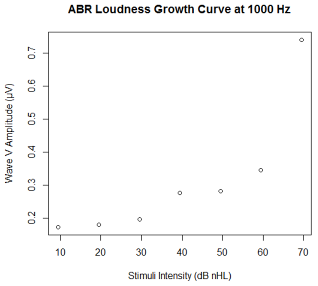

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.4
0.5
0.6
0.7
0.8
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

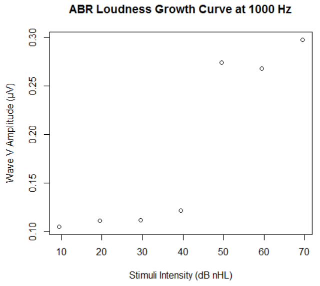

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.15
0.20
0.25
0.30
0.35
0.40
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

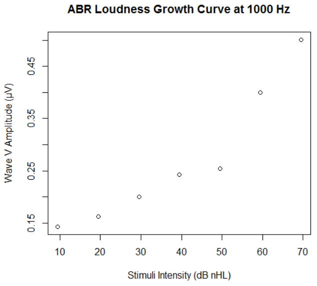

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

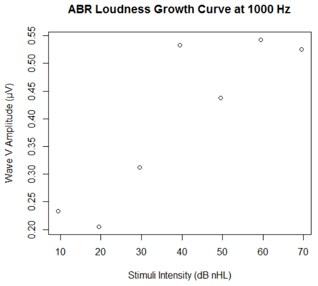

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.05
0.10
0.15
0.20
0.25
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.20
0.25
0.30
0.35
0.40
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.2
0.3
0.4
0.5
0.6
0.7
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.10
0.15
0.20
0.25
0.30
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.15
0.25
0.35
0.45
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.20
0.25
0.30
0.35
0.40
0.45
0.50
0.55
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

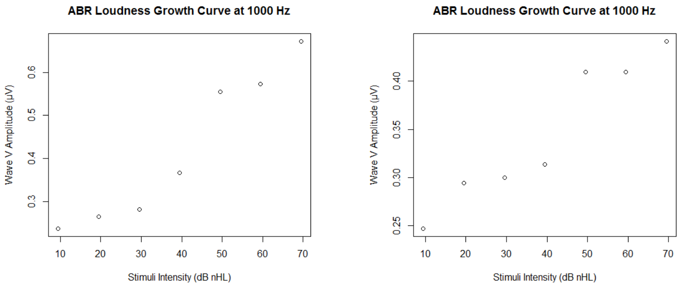


Figure 4.6: ABR loudness growth curves for each individual participant at 1000 Hz. The geometric mean of the participant's Wave V amplitude was calculated for each stimulus presentation intensity. For fill sized figures, please refer to Appendix 6.

Participant	Rho	P-value	Power exponent
1	0.94	0.0334	0.65802
2	0.34435	0.676*	0.40742
3	0.8435	0.0452	0.27069
4	0.704	0.0679*	0.22403
5	0.9343	0.00343	0.315
6	0.734	0.0932*	0.33559
7	0.96742	0.00243	0.20864
8	0.92	0.053*	0.41159
9	0.9342	0.00344	0.36458
10	0.7034	0.0454	0.6128
11	0.9327	0.0434	0.33773
12	0.9543	0.00232	0.33613
13	0.342	0.343*	0.66569
14	0.702	0.2014*	0.41042
15	0.743	0.384*	0.2722
16	0.93	0.0074	0.315
17	0.7566	0.0823*	0.22504

Table 4.3: The correlation coefficients, corresponding p-values and power function exponent for each participant's ABR loudness growth curve at 1000 Hz. Rho is Spearman's correlation coefficient between loudness scaling and stimulus intensity. Power exponent is found for the function y = ab^x + c, where y is Wave V amplitude, and b is stimulus intensity. Asterisk denotes nonsignificant p-values. Each participant's curve can be see in Figure 4.6.

4.3.2 4000 Hz

The ABR loudness growth curve averaged across all 17 participants at 4000 Hz can be seen in Figure 4.7. This figure plots the mean of all participant's Wave V amplitude against the corresponding stimulus intensity level. The correlation was significant (Rho=0.414, p=0.046119). Individual ABR loudness growth curves for each participant are plotted in Figure 4.8. The corresponding correlation coefficients, p-values, and power fit exponent can be found in Table 4.4. Power exponent, x, is found for the function y = ab^x + c, where y is Wave V amplitude, and b is stimulus intensity. In contrast to the behavioural loudness growth curves at 4000 Hz, many of the participants' ABR loudness curves have a significant correlation between response and stimulus intensity.

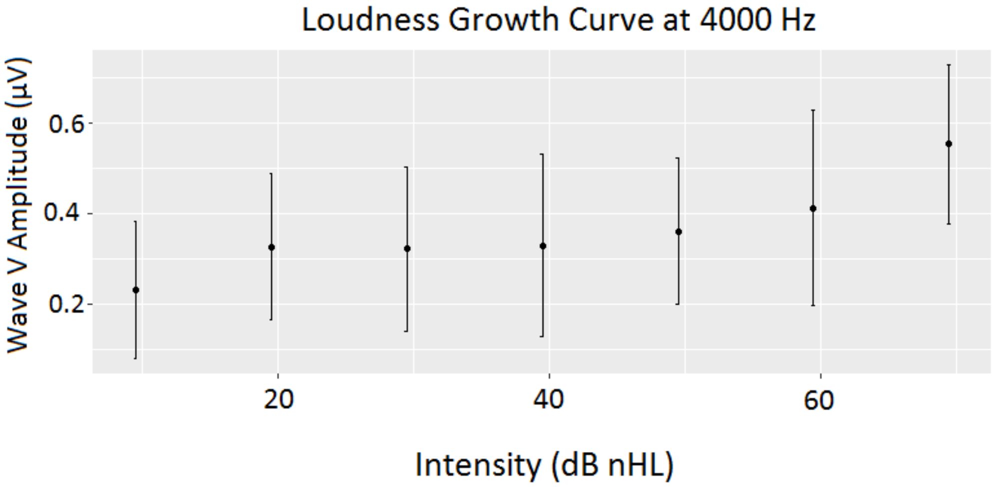
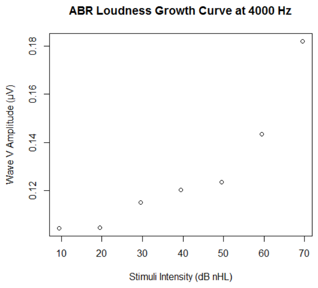
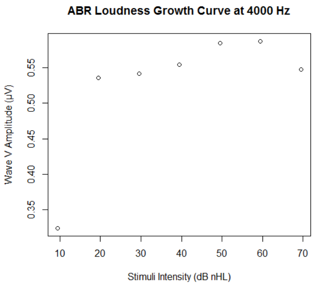
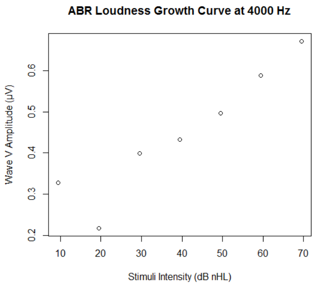
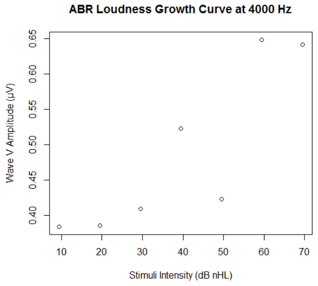


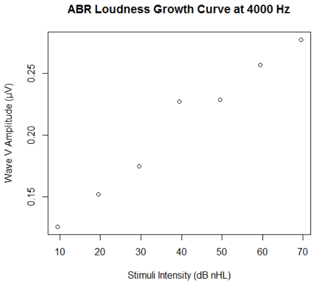
Figure 4.7: The averaged ABR loudness growth curve for all participants at 4000 Hz. The geometric mean of Wave V amplitude was calculated across all participants for each stimulus presentation level, (Rho=0.414, p=0.046119). Error bars are ±1 stdev.

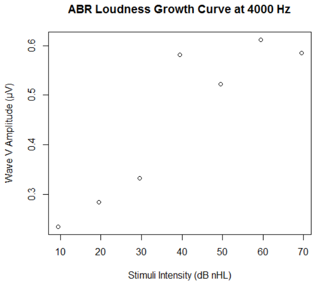

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.12
0.14
0.16
0.18
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

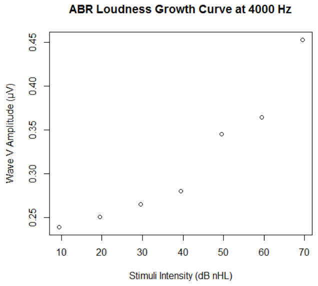

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.35
0.40
0.45
0.50
0.55
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

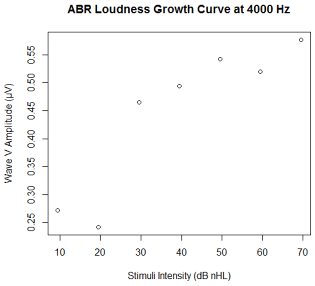

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.2
0.3
0.4
0.5
0.6
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

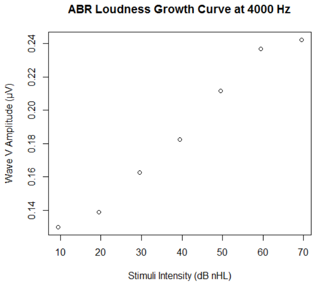

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.40
0.45
0.50
0.55
0.60
0.65
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

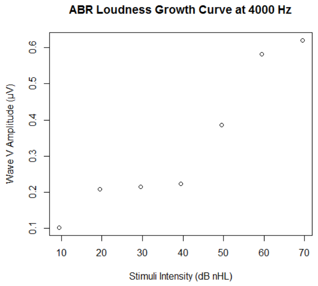

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.15
0.20
0.25
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

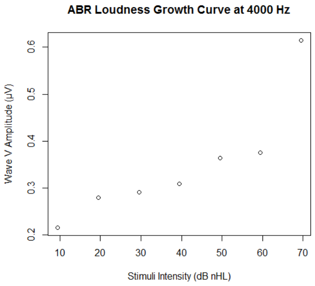

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.3
0.4
0.5
0.6
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

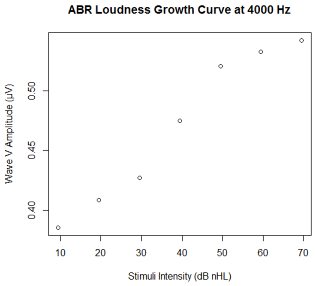

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
0.45
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

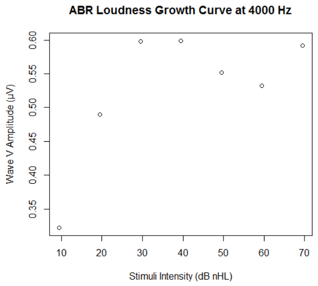

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
0.45
0.50
0.55
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

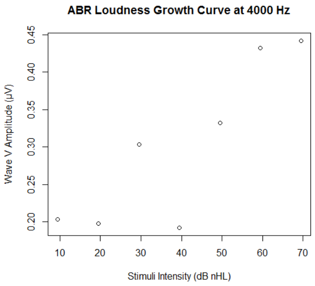

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.14
0.16
0.18
0.20
0.22
0.24
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

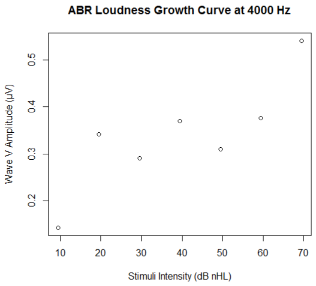

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.1
0.2
0.3
0.4
0.5
0.6
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.2
0.3
0.4
0.5
0.6
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.40
0.45
0.50
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.35
0.40
0.45
0.50
0.55
0.60
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.20
0.25
0.30
0.35
0.40
0.45
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.2
0.3
0.4
0.5
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

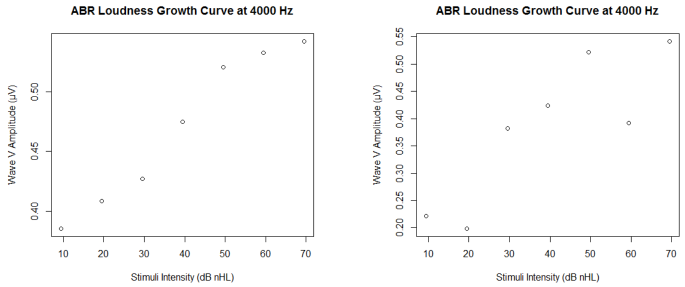


Figure 4.8: ABR loudness growth curves for each individual participant at 4000 Hz. The geometric mean of the participant's Wave V amplitude was calculated for each stimulus presentation intensity. For full sized figures, please refer to Appendix 6.

Participant	Rho	p-value	Power exponent
1	0.9445	0.0043	0.64065
2	0.5654	0.343*	0.15858
3	0.9243	0.08*	0.23568
4	0.8354	0.0654*	0.17318
5	0.9245	0.0435	0.46867
6	0.84574	0.04983	0.2048
7	0.9375	0.000754	0.3382
8	0.642	0.354*	0.20457
9	0.9683	0.0134	0.49852
10	0.7934	0.04387	0.31936
11	0.8542	0.04523	0.303
12	0.9234	0.00341	0.216432
13	0.343	0.453*	0.15877
14	0.732	0.283*	0.34083
15	0.6324	0.09*	0.303
16	0.9335	0.0023	0.216432
17	0.732	0.07*	0.21747

TABLE 4.4: The correlation coefficients, p-values, and power exponents corresponding to each participant's individual plot of ABR loudness growth (Wave V amplitude) against stimulus intensity at 4000 Hz. Asterisk signifies nonsignificant p-values. To see the individual plots, please refer to Figure 4.8.

4.4 Relation Between Psychoacoustic and ABR Loudness Growth

4.4.1 1000 Hz

The relationship between the psychoacoustic loudness ranking and ABR Wave V amplitude was significant at 1000 Hz, (Figure 4.9, rho=0.9224, p=0.03668). For each individual participant (see Figure 4.10), the relation was also significant except for participant 14. For all participants' correlation coefficients, see Table 4.5.

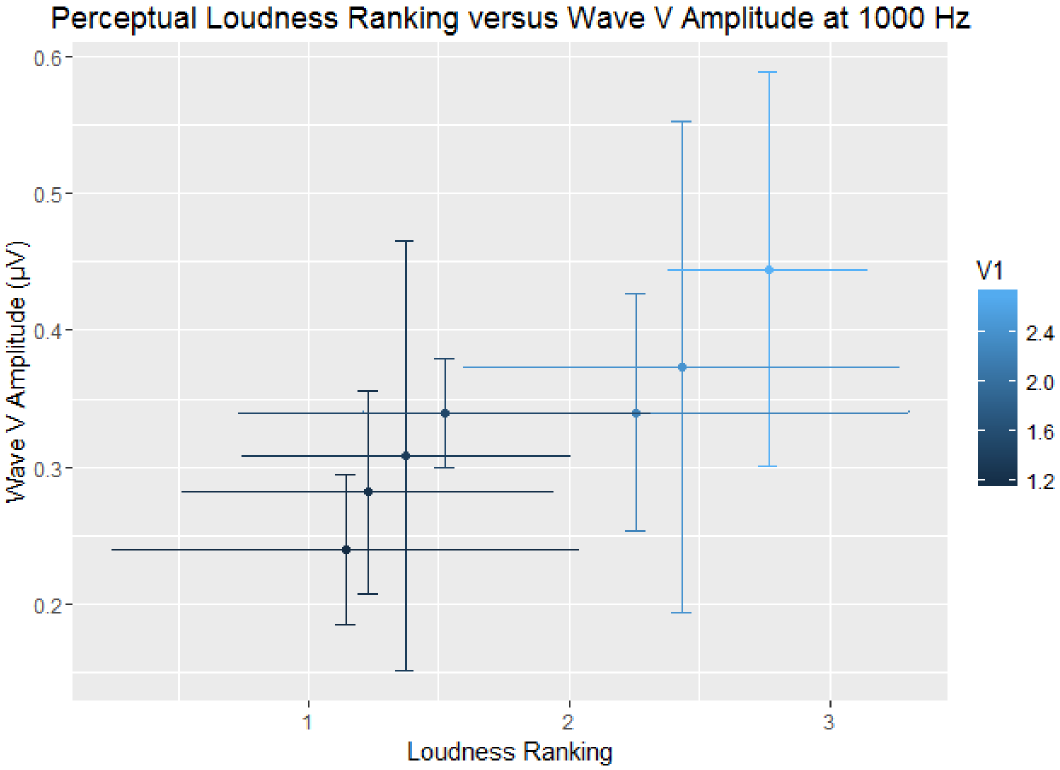
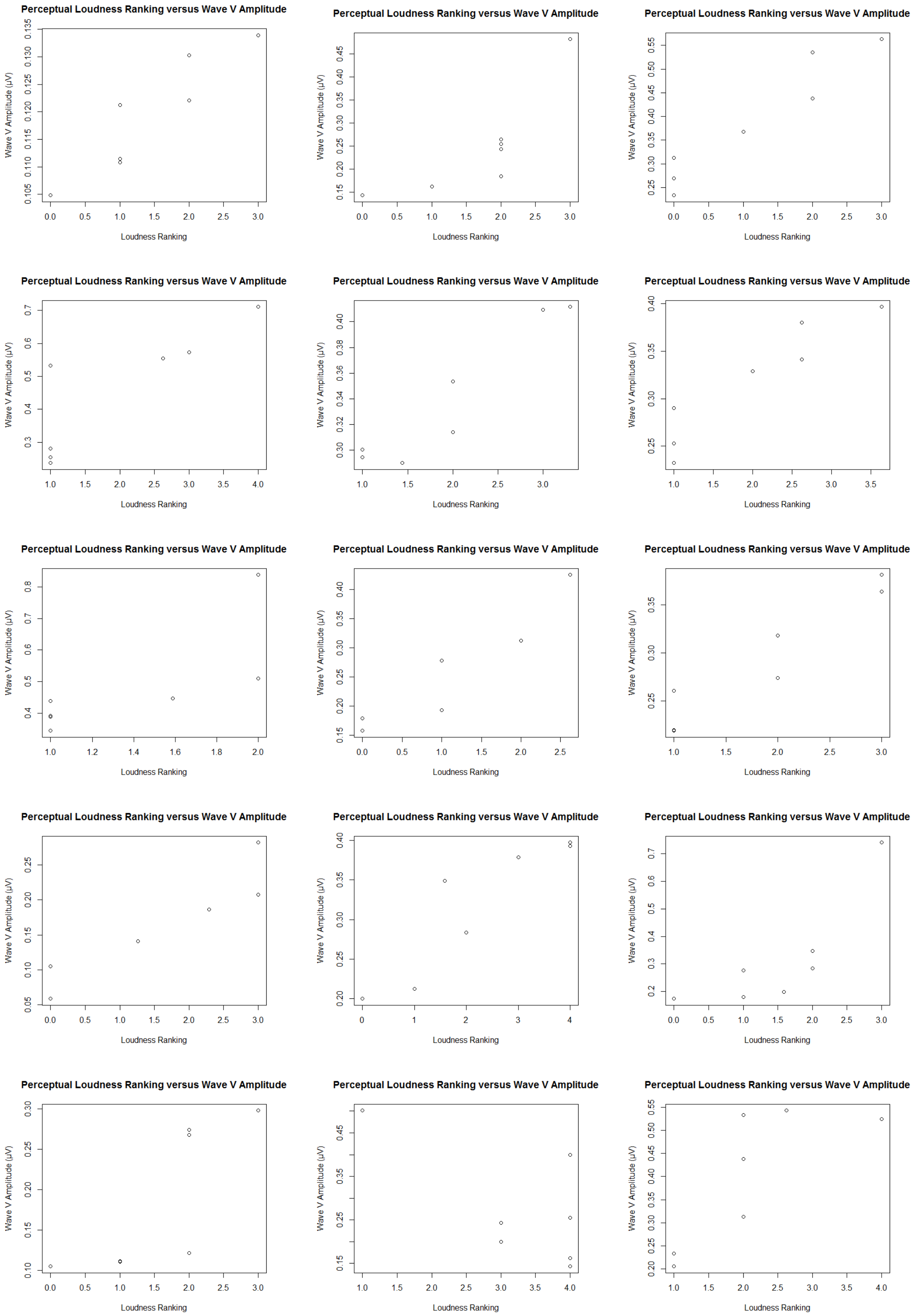


Figure 4.9: Average relation between psyachoacoustic and ABR loudness growth at 1000 Hz. Averaged Wave V amplitude plotted against averaged loudness ranking, for each individual stimulus intensity, (Rho=0.9224, p=0.03668). Error bars are ±1 stdev.


Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.105 0.110 0.115 0.120 0.125 0.130 0.135
0.0 0.5 1.0 1.5 2.0 2.5 3.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.15 0.20 0.25 0.30 0.35 0.40 0.45
0.0 0.5 1.0 1.5 2.0 2.5 3.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.25 0.30 0.35 0.40 0.45 0.50 0.55
0.0 0.5 1.0 1.5 2.0 2.5 3.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.3 0.4 0.5 0.6 0.7
1.0 1.5 2.0 2.5 3.0 3.5 4.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.30 0.32 0.34 0.36 0.38 0.40
1.0 1.5 2.0 2.5 3.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.25 0.30 0.35 0.40
1.0 1.5 2.0 2.5 3.0 3.5
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.4 0.5 0.6 0.7 0.8
1.0 1.2 1.4 1.6 1.8 2.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.15 0.20 0.25 0.30 0.35 0.40
0.0 0.5 1.0 1.5 2.0 2.5
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.25 0.30 0.35
1.0 1.5 2.0 2.5 3.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.05 0.10 0.15 0.20 0.25
0.0 0.5 1.0 1.5 2.0 2.5 3.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.20 0.25 0.30 0.35 0.40
0 1 2 3 4
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.2 0.3 0.4 0.5 0.6 0.7
0.0 0.5 1.0 1.5 2.0 2.5 3.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.10 0.15 0.20 0.25 0.30
0.0 0.5 1.0 1.5 2.0 2.5 3.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.15 0.25 0.35 0.45
1.0 1.5 2.0 2.5 3.0 3.5 4.0
Loudness Ranking
Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.20 0.25 0.30 0.35 0.40 0.45 0.50 0.55
1.0 1.5 2.0 2.5 3.0 3.5 4.0
Loudness Ranking

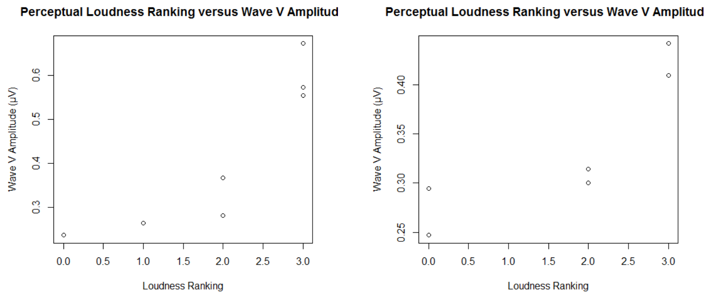


Figure 4.10: Relation between psyachoacoustic and ABR loudness growth at 1000 Hz for each individual participant. Averaged Wave V amplitude plotted against averaged loudness ranking, for each individual stimulus intensity. For each participant's correlation coefficient and power fit exponent, please refer to Table 4.5. For full sized figures, please refer to Appendix 6.

Participant	Rho	p-value	Power exponent
1	0.9543	0.000836	0.8934
2	0.906327	0.004902	0.7845
3	0.9543	0.000836	0.8435
4	0.906327	0.004902	0.9435
5	0.87287	0.01032	0.543
6	0.9543135	0.000836	0.8935
7	0.89642	0.006267	0.7236
8	0.95431	0.000836	0.746238
9	0.9449112	0.001328	0.6802
10	0.95431	0.000836	0.9234
11	0.95499	0.0008055	0.82374
12	0.927426	0.00262	0.5849
13	0.95499	0.000836	0.6623
14	-0.4183	0.3503*	0.93423
15	0.8046	0.02908	0.8452
16	0.9543	0.000836	0.77732
17	0.9449112	0.001328	0.7543

TABLE 4.5: Correlation coefficients, p-values and power exponents corresponding to each participant's individual plot of psychoacoustic loudness growth (loudness ranking) to ABR loudness growth (Wave V amplitude) at 1000 Hz. Asterisk signifies nonsignificant p-values. To see the individual plots, please refer to Figure 4.10.

4.4.2 4000 Hz

The relationship between the participant-averaged psychoacoustic loudness ranking and ABR Wave V amplitude was not significant at 4000 Hz, (rho = 0.17857, p = 0.7131). For each individual participant see Figure 4.12. None of the individual relationships were significant except for participant 7 (Rho = 0.96428, p = 0.002778). For all participants' correlation coefficients power fit exponent, see Table 4.6.

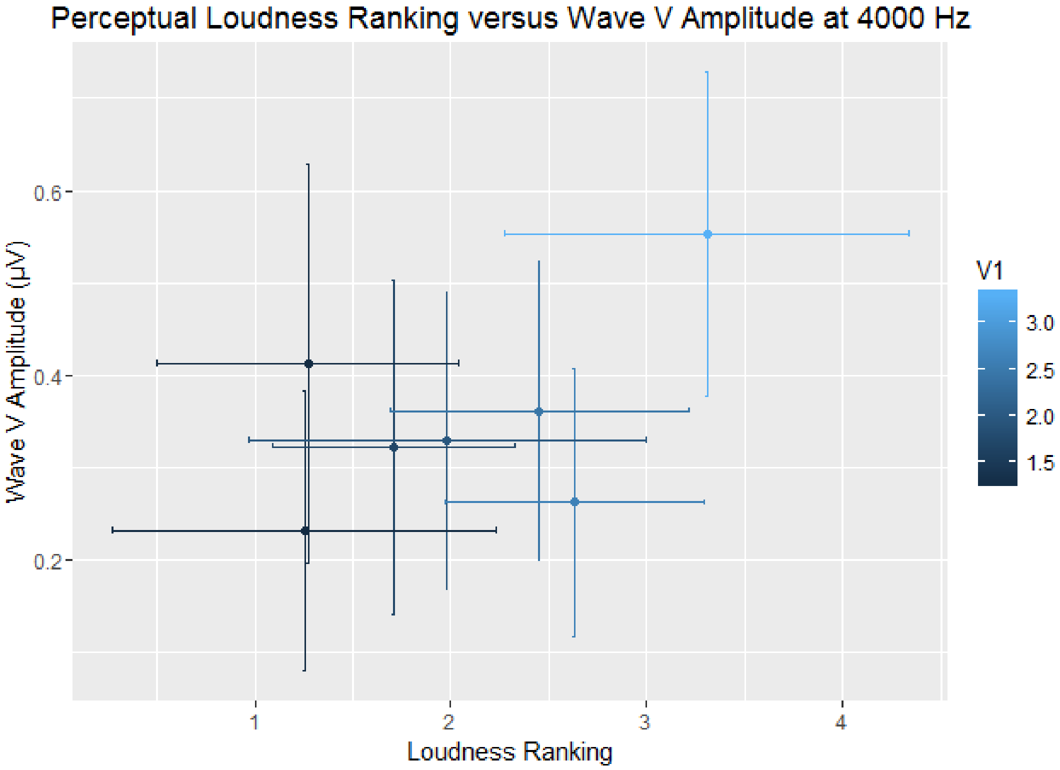


FIGURE 4.11: Average relation between psyachoacoustic and ABR loudnss growth at 4000 Hz. Averaged Wave V amplitude plotted against averaged loudness ranking, for each individual stimuli intensity, (Rho=0.17857, p=0.7131). Error bars are ±1 stdev.

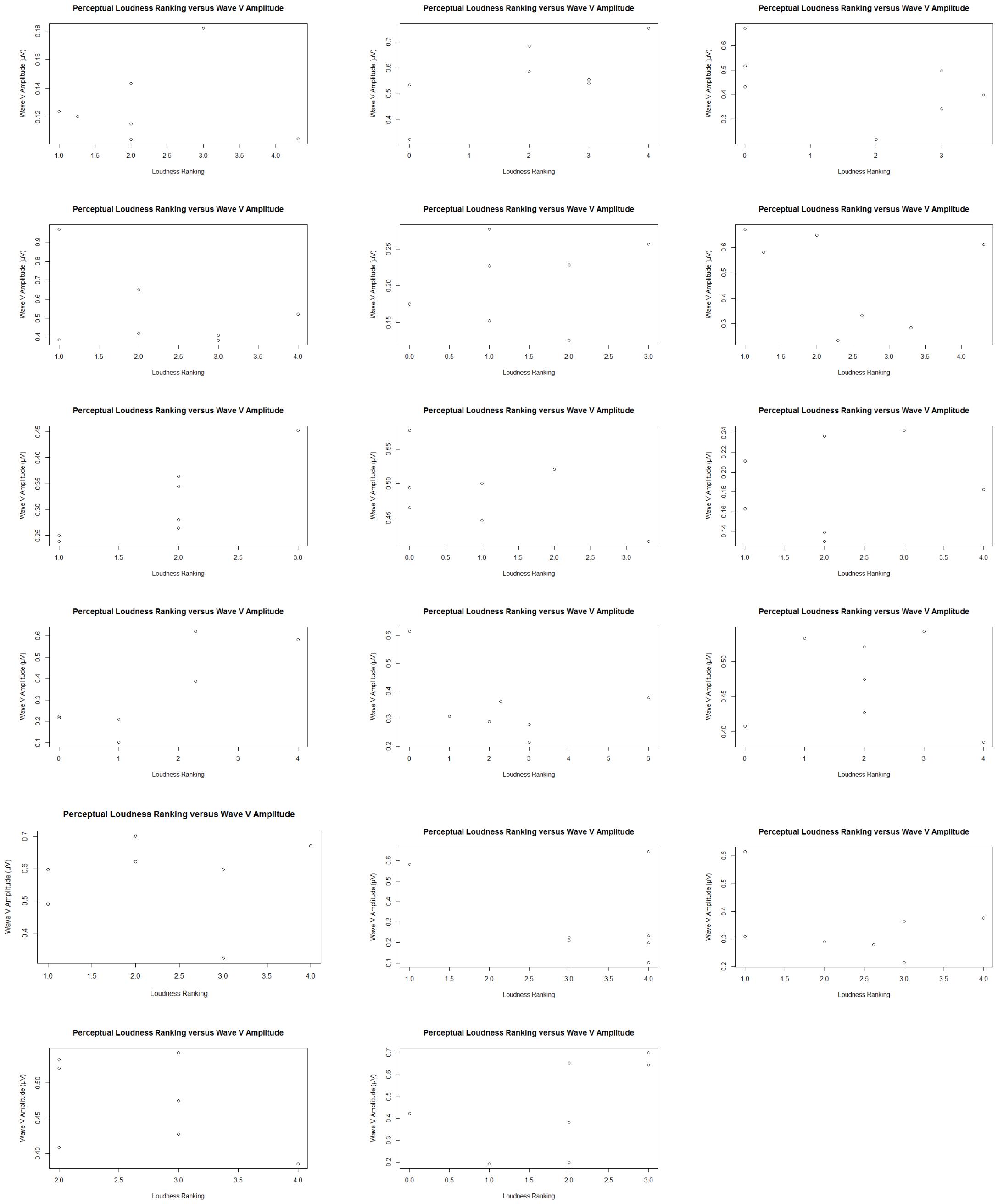


FIGURE 4.12: Relation between psyachoacoustic and ABR loudnss growth at 4000 Hz for each individual participant. Averaged Wave V amplitude plotted against averaged loudness ranking, for each individual stimuli intensity. For each participant's correlation coefficient and power fit exponent, please refer to Table 4.6. For full sized figures, please refer to Appendix 6.

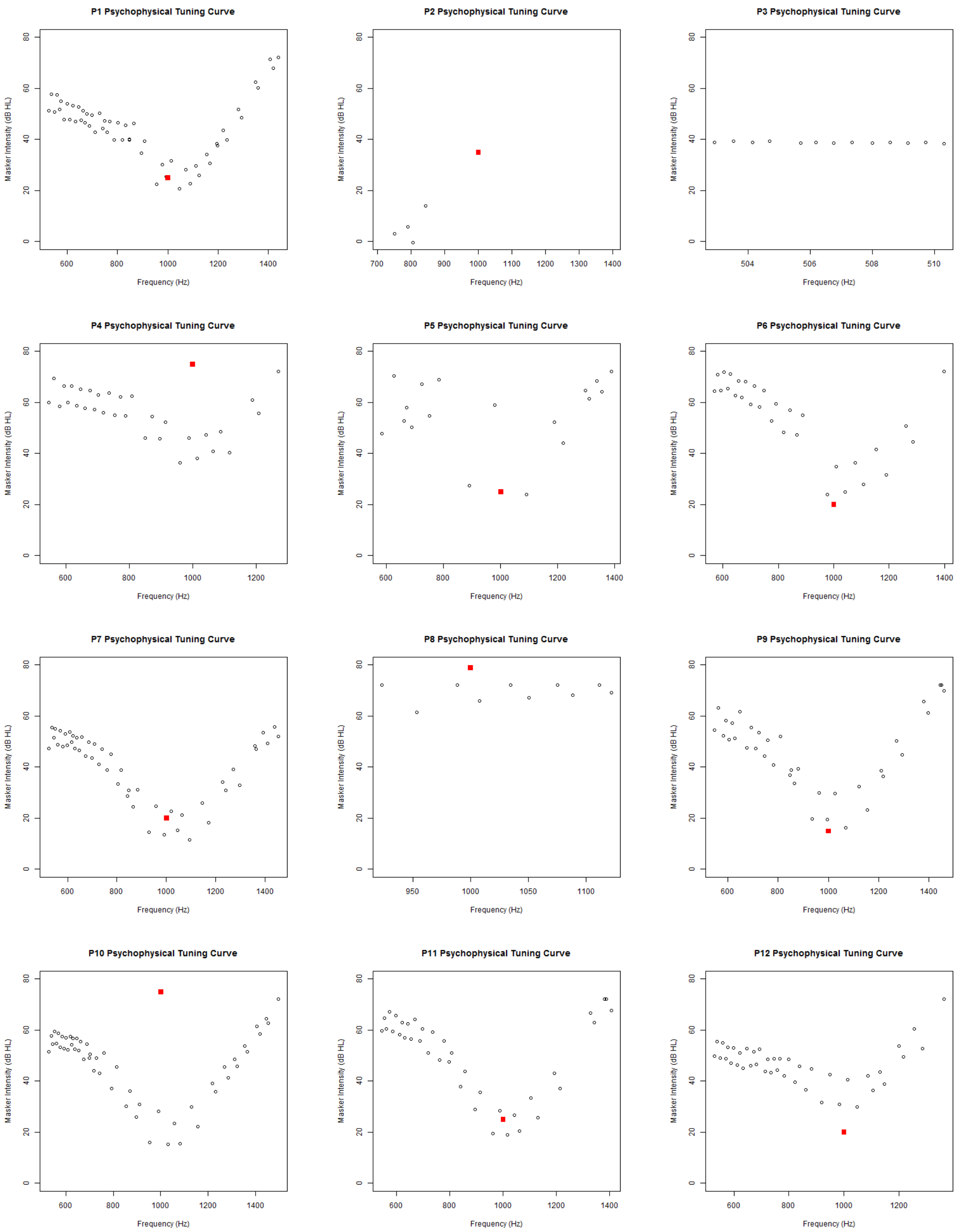
Participant	Rho	p-value	Power exponent
1	-0.35714	0.4444	-0.2328
2	0.60714	0.1667	0.343
3	-0.1428571	0.7825	-0.3234
4	-0.14285	0.7825	-0.2329
5	0.32142	0.4976	0.11243
6	-0.35714	0.44	0.2312
7	0.96428	0.002778*	0.23423
8	-0.34285	0.3956	0.273642
9	0.14285	0.7825	0.332
10	0.5714	0.2	0.2214
11	0.25	0.5948	0.1323
12	-0.214857	0.6615	0.2432
13	0.3214286	0.4976	0.2878
14	0.64285	0.1389	0.3923
15	-0.25	0.5148	0.434
16	-0.214285	0.6615	0.3244
17	0.4642857	0.3024	0.454

Table 4.6: Correlation coefficients, p-values, and power exponents corresponding to each participant's individual plot of psychoacoustic loudness growth (loudness ranking) to ABR loudness growth (Wave V amplitude) at 4000 Hz. Asterisk notes significant p-values. To see the individual plots, please refer to Figure 4.12.

4.5 Psychophysical Tuning Curve

4.5.1 1000 Hz

Of the 27 participants who passed the prescreening measures, only 24 completed the PTC at 1000 Hz. Each participant's individual PTC can be seen in Figure 4.13. The PTC plots the intensity level required of the noise to completely mask the signal's detection. The PTC plots shown are the average of forward and backward sweeps (Figure 4.13). The corresponding Q10 and tip frequency estimates for each participant are in Table 4.7. The Q10 and tip frequency estimates were obtained by first fitting a quadratic function to each curve. The minimum of the quadratic function is the tip frequency.



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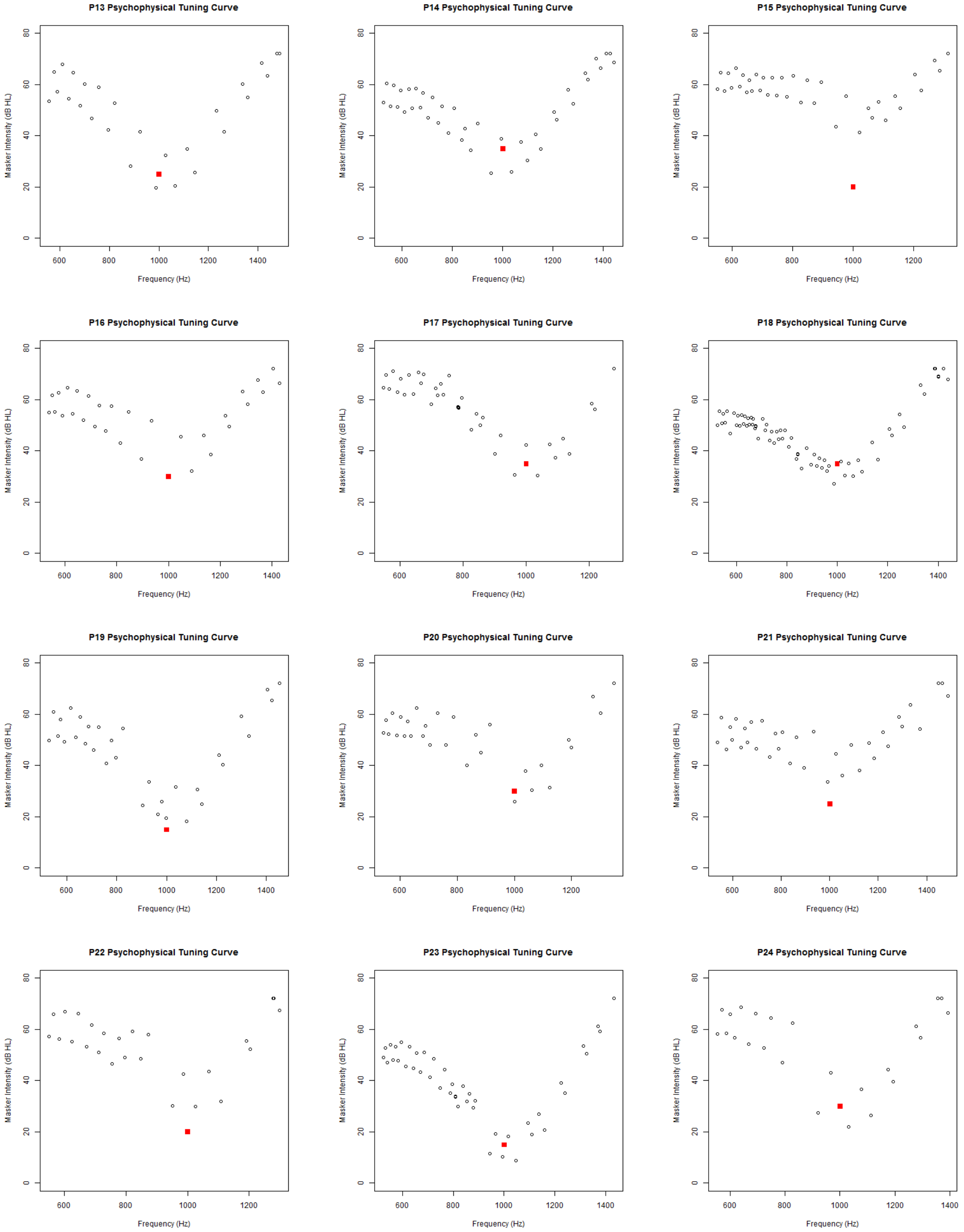


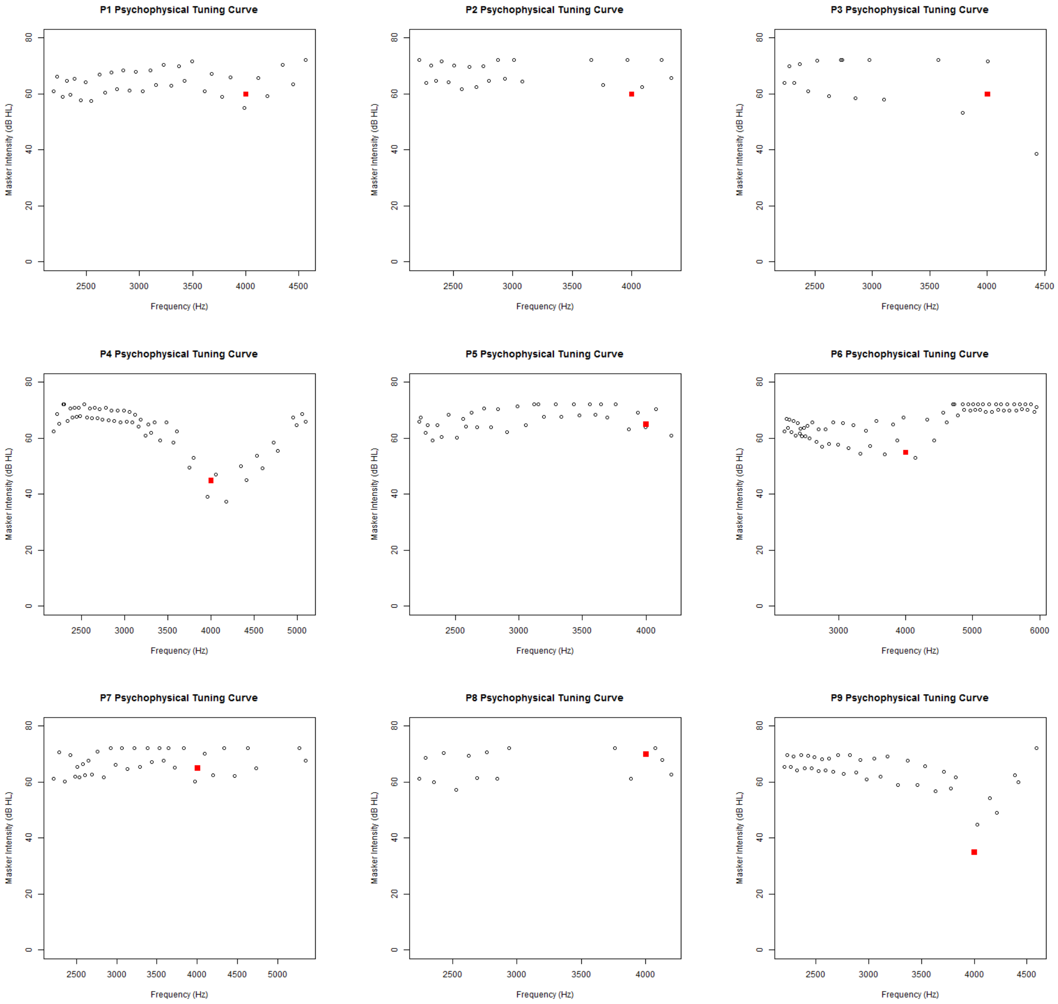
Figure 4.13: Individual PTCs for all participants at 1000 Hz. The red dot represents the signal. Black open circles plot the change in noise intensity level required to mask the signal.

Participant	Tip Frequency	Q10 Estimate
1	1054.5	3.4
2	800	NA
3	500	NA
4	960	1.1
5	1100	0.2
6	1000.5	2.3
7	1152	5.6
8	1020	0.4
9	1100	4
10	1027	3.35
11	1011.5	4
12	1100	0.9
13	1100	3.75
14	1000.5	2.6
15	1053.5	1.2
16	1033.5	1.8
17	1020	3.1
18	1004	2
19	1098	4.9
20	997	3.43
21	1000.5	2.8
22	1004	2.05
23	1018.5	4.33
24	980	4.24

Table 4.7: The corresponding Q10 and tip frequency estimates from each individual participant's PTC at 1000 Hz. Estimates are based on the quadratic fit of the PTC function. For the PTC plots, see Figure 4.13.

4.5.2 4000 Hz

Of the 27 participants who passed the prescreening measures, only 18 completed the PTC at 4000 Hz. Each participant's individual PTC can be seen in Figure 4.14. PTC plots the intensity level the noise had to be to mask the signal's perception. PTC plots shown are the average of forward and backward sweeps. The corresponding Q10 and tip frequency estimates for each participant are in Table 4.8. The Q10 and tip frequency estimates were obtained by first fitting a quadratic function to each curve. The minimum of the quadratic function is the tip frequency.



P1 Psychophysical Tuning Curve

P2 Psychophysical Tuning Curve

P3 Psychophysical Tuning Curve

P4 Psychophysical Tuning Curve

P5 Psychophysical Tuning Curve

P6 Psychophysical Tuning Curve

P7 Psychophysical Tuning Curve

P8 Psychophysical Tuning Curve

P9 Psychophysical Tuning Curve

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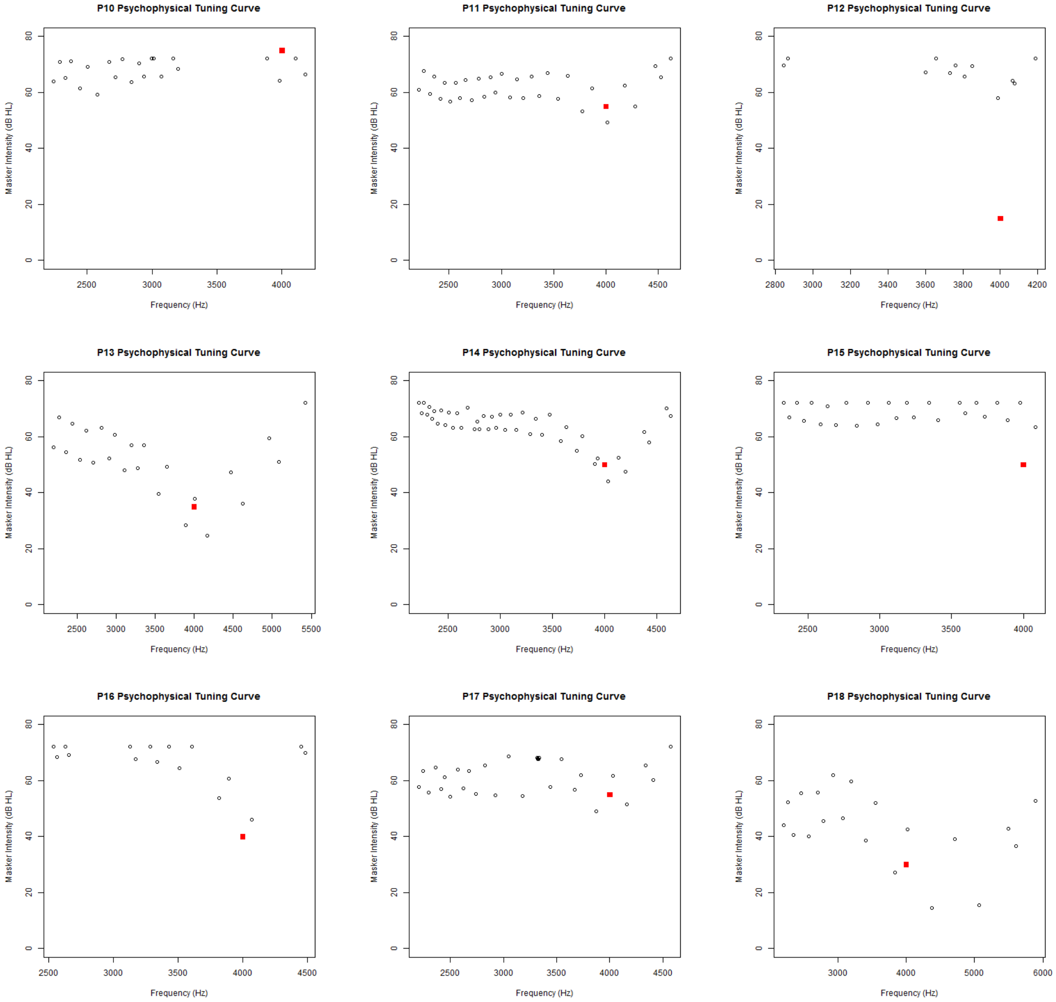


Figure 4.14: Individual PTCs for all participants at 4000 Hz. The red dot represents the signal. Black open circles plot the change in noise intensity level required to mask the signal.

Participant	Tip Frequency	Q10 Estimate
1	2700	0.3
2	2650	0.7
3	4480	0.5
4	4295.5	4.1
5	2200	0.456
6	4338	0.543
7	3980	0.63
8	2600	0.8
9	4002	2
10	2604	0.53
11	4030	0.98
12	3980	1.3
13	4200	3
14	4100	3.8
15	2840	0.634
16	4100	2.6
17	3800	1.4
18	4800	0.3

TABLE 4.8: The corresponding Q10 and tip frequency estimates from each individual participant's PTC at 4000 Hz. Estimates are based on the quadratic fit method. For the PTC plots, see Figure 4.14.

4.6 PTC versus audiogram

4.6.1 1000 Hz

Participants' audiogram thresholds at 1000 Hz did not significantly correlate with their Q10 estimate at 1000 Hz, see Figure 4.15 (Rho=-0.2882462, p = 0.172). Audiogram thresholds at 1000 Hz did not correlate with tip frequency, see Figure 4.18 (Rho=-0.3828442 , p=0.07865).

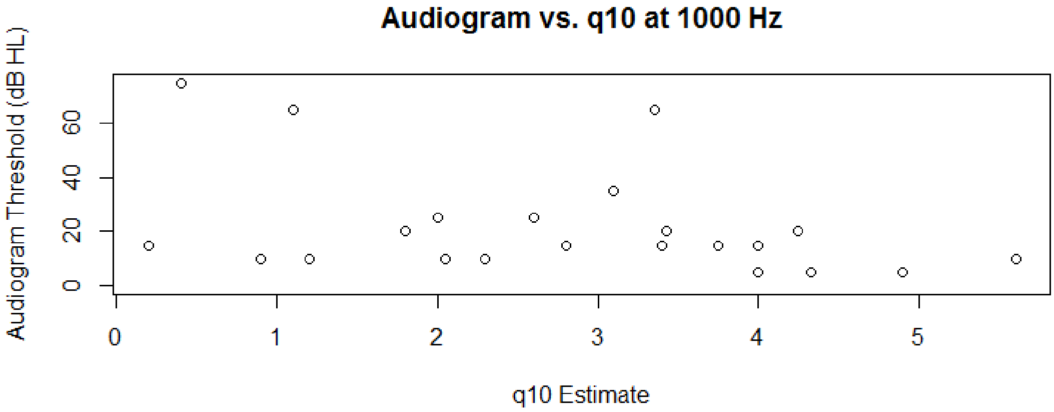


FIGURE 4.15: The relation of audiogram thresholds compared to Q10 estimates at 1000 Hz for all participants, (Rho=-0.3873197, p = 0.07493).

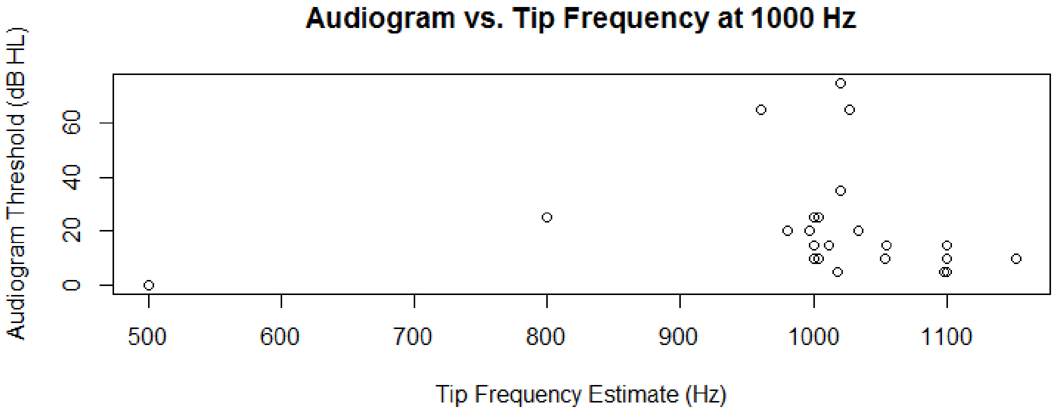


FIGURE 4.16: The relation of audiogram thresholds compared to PTC tip frequency at 1000 Hz for all participants, (Rho=-0.2882462, p=0.172).

4.6.2 4000 Hz

Participants' audiogram thresholds at 4000 Hz did not significantly correlate with their Q10 estimate at 4000 Hz, see Figure 4.17 (Rho = 0.2748676, p = 0.2696). Audiogram thresholds at 4000 Hz did not correlate with tip frequency, see Figure 4.18 (Rho = -0.09953405 , p = 0.6944).

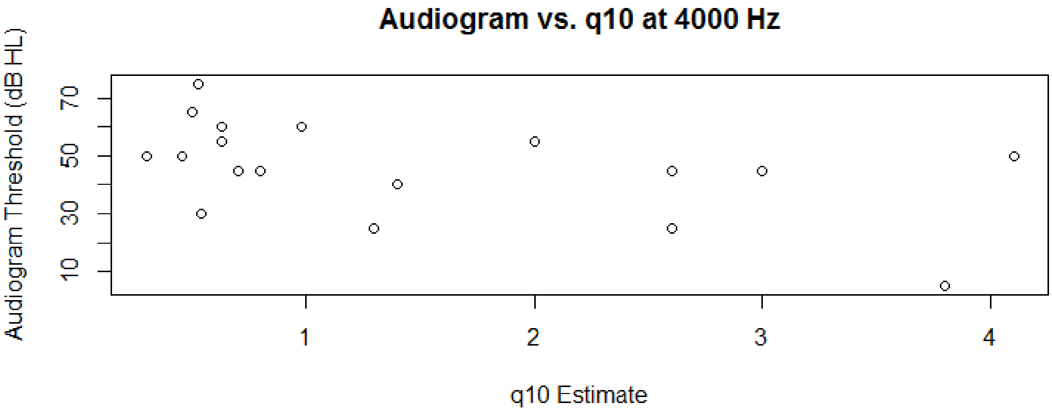


Figure 4.17: The relation of audiogram thresholds compared to Q10 estimates at 4000 Hz for all participants, (Rho = -0.4864752, p = 0.07809).

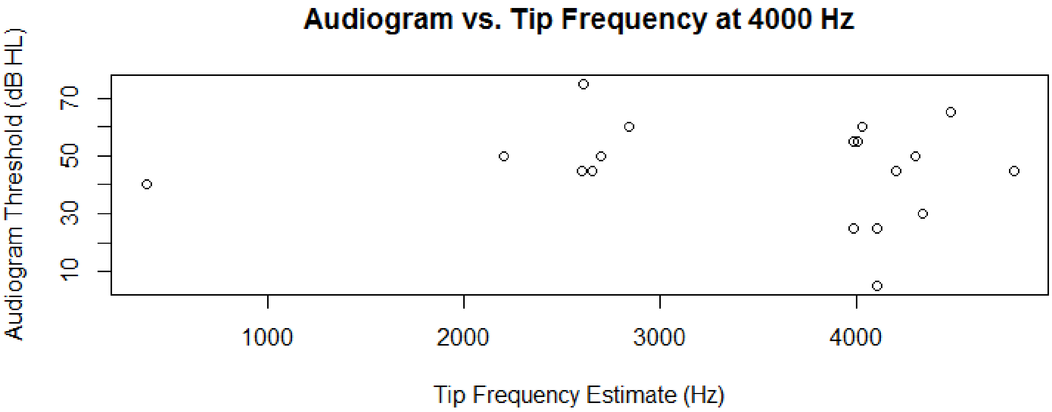


Figure 4.18: The relation of audiogram thresholds compared to PTC tip frequency at 4000 Hz for all participants, (Rho = -0.09953405 , p = 0.6944).

4.7 Predicting Audiometric Thresholds

Multiple regression analyses were conducted to test if audiometric thresholds can be predicted from a combination of IHC and OHC loss. For each regression, tip frequency from the PTC was used as a measure of IHC loss, and one measure of OHC was used (either power exponent from the ABR loudness growth function, power exponent from the behaviour and ABR loudness growth function, Q10 estimate from the PTC, Wave V amplitude, or loudness ranking).

4.7.1 1000 Hz

Regression 1: tip frequency obtained from the PTC was used as a measure of IHC loss, and the power fit exponent from the ABR loudness growth function was used as a measure of OHC loss. In this model, neither the tip frequency nor ABR power fit was significant in predicting audiometric thresholds (F(2,14)=0.1, p = 0.8537).

Regression 2: tip frequency and the power exponent from the behaviour and ABR loudness growth function. In this model, neither the tip frequency nor the power exponent from the behaviour and ABR loudness growth function significantly predicted audiometric thresholds (F(2,14)=1.141, p = 0.3476).

Regression 3: tip frequency and Q10 estimate. Neither significantly predicted audiometric thresholds (F(2,19)=2.499, p=0.1088) however tip frequency approached significance (t=2.044, p=0.0551).

Regression 4: tip frequency and ABR wave V amplitude. The results indicated that the two predictors explained 50.63% of the variance (R^2=0.5063, F(2,14)=7.178, p=0.007153). It was found that ABR Wave V amplitude significantly predicted audiometric thresholds (t=3.771, p=0.00207).

Regression 5: audiometric thresholds were predicted from tip frequency and loudness ranking values. The predictors explained 50.24% of the variance in audiometric threshold (R^2=0.4551, F(2,21)=10.6, p=0.0006559). Loudness ranking was a significant predictor in audiometric thresholds (t=3.03012, p=0.000155).

4.7.2 4000 Hz

Regression 1: tip frequency and the power exponent from the ABR loudness growth function. In this model, neither the tip frequency nor ABR power fit was significant in predicting audiometric thresholds (F(2,14)=0.4189, p = 0.6658).

Regression 2: tip frequency and the power exponent from the behaviour and ABR loudness growth function. In this model, neither the tip frequency nor the power exponent from the behaviour and ABR loudness growth function significantly predicted audiometric thresholds (F(2,14)=1.805, p = 0.2007).

Regression 3: tip frequency and Q10 estimate. Neither significantly predicted variance in audiometric thresholds (F(2,14)=0.2826, p=0.758).

Regression 4: tip frequency and ABR wave V amplitude. The results indicated that the two predictors did not significantly predict audiometric thresholds F(2,14)=1.242, p=0.3187).

Regression 5: audiometric thresholds were predicted from tip frequency and loudness ranking values. Loudness ranking values were the geometric means from the psychoacoustic loudness growth function. The predictors did not significantly explain the variance in audiometric threshold (F(2,14)=0.5974, p=0.5637).

Chapter 5

Discussion

5.1 Psychoacoustic and ABR Loudness Growth

There was a significant correlation in the behavioural loudness growth test between stimulus intensity and loudness ranking at 1000 Hz, but not for the 4000 Hz case. This is not unexpected, as all participants had normal or near-normal levels of hearing at 1000 Hz, and participants predominantly had hearing loss at 4000 Hz, according to their audiogram thresholds. When looking at individual behavioural loudness growth curves, participants 1 and 3 had a significant linear relationship between loudness ranking and stimuli intensity at 4000 Hz, and both of these participants had normal audiogram thresholds at 4000 Hz. This suggests that participants with hearing loss at particular frequencies have non-significant correlations between behaviour loudness ranking and stimulus intensity at those corresponding frequencies, but normal behaviour loudness growth curves at frequencies where their hearing threshold is normal.

This thesis makes an important contribution in validating loudness growth functions obtained from the ABR. Unlike the behaviour loudness growth curve, the ABR loudness growth task's Wave V amplitude significantly correlated with stimulus intensity at both 1000 Hz and 4000 Hz. This implies two things: 1) the ABR loudness growth task is more reliable at predicting loudness growth curves compared to the behavioural task at high frequencies, or 2) Wave V amplitude reflects loudness perception in participants with hearing loss at low frequencies, but not at high frequencies.

In addition, the error bars for both the behavioural and ABR loudness growth function are large, implying a difference in stimulus intensity of 10 dB nHL does not yield a significant difference in wave V amplitude or behavioural response. As predicted, there was a significant linear relationship between loudness ranking (behavioural loudness growth task) and wave V amplitude (ABR loudness growth task) at 1000 Hz, but not at 4000 Hz. While wave V amplitude increases with stimulus intensity, wave V amplitude does not predictably increase with participants' loudness rankings of the same stimuli. This implies there is some sort of discord between their ABR responses for a stimulus, and their perception of the same stimulus at 4000 Hz, but not at 1000 Hz.

5.2 Psychophysical Tuning Curve

For the PTC task at 1000 Hz, all but 3 participants produced normal looking PTCs. Participants 2, 3, and 8 had abnormal curves. All 3 of those participants had normal audiogram thresholds at 1000 Hz. Participant 2 responded they could hear the signal regardless of the intensity level of the masker once the masker passed 700 Hz in centre frequency. We predicted the masker would be most efficient at 1000 Hz, but instead it was at a distal frequency, suggesting that participant 2 has a cochlear dead region at 1000 Hz. Participant 3 responded that they could hear the stimuli if the noise had a centre frequency higher than 510 Hz regardless of the level of the noise. This suggests participant 3 also has a dead region around 1000 Hz. Participant 8 is characterized by not having a curve at all. The noise-masker was almost equally as efficient at the stimuli frequency, as well as frequencies within ±100 Hz from the signal. This lack of frequency specificity, or sharpness of the curve reflects OHC loss.

As predicted, the PTC plots at 4000 Hz are overall a lot more abnormal, scattered and less sharp compared to the 1000 Hz plots. This could be because most participants had hearing loss at 4000 Hz, but not at 1000 Hz. Participants 1 and 3 have normal audiogram thresholds at 4000 Hz, and thus we predicted they would have normal PTCs, however, they did not. Only participants 4, 9, 13 and 14, have normal curves. Participants 1, 5, 7, 11, 15 have extremely broad PTCs, which suggests that there is a major lack of frequency specificity. The narrow band noise almost equally masks the stimulus across a wide frequency region, and almost has no efficiency increase at the signal frequency. This suggests a loss of IHC and OHC and, because OHC health reflects tuning curve broadness.

Participants 2, 3, 8, 10, 12 and 16 have major gaps in their PTCs. These gaps correspond to the participant responding that they can hear the stimuli regardless of the intensity level of the noise. This suggests that the participant has little to no hearing ability in these regions, as providing gain to the masker could not block the signal. Due to the physiology of the basilar membrane, if a participant has normal hearing, then the masker should be able to mask the signal most efficiently at the signal frequency. However, in the participants mentioned, at frequencies near the signal, no matter the level we raise the masker to, the participant can still hear the signal, which implies there is some damage. It is possible that the haircells in this region are damaged, or there is some other physiological problem in this area. Participants with these gaps have a moderate amount of hearing loss at 4000 Hz according to their audiograms.

Finally, for participant 18, the masker was most efficient at 4300 Hz, which again implies there is some cochlear damage at 4000 Hz. Based on auditory physiology, we predicted that the masker would always be more efficient at the stimuli frequency or a lower frequency, but not a a higher frequency. One possible explanation is that the participant had trouble maintaining sustained attention and their responses were delayed.

According to Shabana et al., (2014), normal listeners who heard a 1000 Hz stimulus at 35 dB intensity had a mean tip frequency of 1007 Hz, $\sigma = 35$ dB, and an average Q10 estimate of 3.1, $\sigma = 0.5$. At 50 dB, the average tip frequency was 1006 Hz and $\sigma = 21$ Hz, and a Q10 estimate of 3, $\sigma = 0.3$, and at 65 dB, the average tip frequency was 1000 Hz, $\sigma = 36$ Hz, and an average Q10 estimate of 2.5, $\sigma = 0.6$.

For the 1000 Hz stimuli, participants 4, 5, 8, 12, 15, 16, 18 and 22 have Q10 values that were at least 2 standard deviations smaller than the mean Q10 values for their corresponding stimulus intensity level. This smaller Q10 value suggests participants have a much less sharp tuning curve, with some hearing impairments in this frequency region, whereas their audiogram thresholds are within normal ranges. All other participants have Q10 values that are normal or better than expected, for their corresponding stimulus intensity level.

At 1000 Hz, participants 2, 3, 5, 7, 9, 12, 13, 19 had tip frequencies that were greater than 2 standard deviations from the mean expected tip frequency. Participants 5 and 12 had both abnormal Q10 values and larger tip shifts than expected. Participants 2, 3, 7, 9, 13, 19 had normal Q10 values, but abnormal tip frequencies, suggesting they have some IHC deficits but not OHC. Whereas participants 4, 8, 15, 16, 18, and 22 had abnormal Q10 estimates, but normal tip frequencies, suggesting the opposite pattern of deficits: normal IHC and abnormal OHC.

According to Shabana et al., (2014), normal listeners who heard a 4000 Hz stimulus at 35 dB intensity had a mean tip frequency of 4021 Hz, $\sigma = 160$ Hz, and an average Q10 estimate of 4, $\sigma = 1.2$. At 50 dB the average tip frequency was 3957, $\sigma = 172$ Hz, and average Q10 estimate of 3.8, $\sigma = 1.6$. At 65 dB, the average tip frequency was 3925 Hz, $\sigma = 239$ Hz, and a Q10 estimate of 3, $\sigma = 1.1$.

For the 4000 Hz stimuli, participants 1, 2, 3, 5, 6, 7, 8, 10, 11, 12, 15, and 17 have Q10 values that were at least 2 standard deviations smaller than the mean Q10 values for their corresponding stimulus intensity level. This smaller Q10 value reflects their less sharp tuning curve, and reduced frequency specificity. All other participants have Q10 values that are normal or better than expected, for their corresponding stimulus intensity level.

At 4000 Hz, participants 1, 2, 3, 5, 6, 8, 10 and 15 had tip frequencies that were greater than 2 standard deviations from the mean expected tip frequency. Unlike for the 1000 Hz case, all participants with abnormal tip frequencies also had abnormal Q10 estimates. Participants 7 and 17 had abnormal Q10 estimates, but normal tip frequencies.

Finally, there was no correlation found for Q10 estimates or tip frequencies when compared to audiogram thresholds, for both 1000 Hz and 4000 Hz. This suggests that audiogram deficits do not predict any measure from the PTC, and that audiograms do not reflect whether a participant has a dead region.

5.3 Predicting Audiometric Thresholds

Of all the different predictors we used to try to estimate audimetric thresholds at 1000 Hz, only 3 were significant correlates: tip frequency, ABR wave V amplitude, and loudness ranking.

At 1000 Hz, both loudness growth functions were a significant predictor of audiometric thresholds. This provides evidence that loudness growth is an important characteristic to consider when patients have SNHL, as it can be used to predict audiometric thresholds. Since ABR wave V amplitude is easier for participants to complete compared to the to the psychoacoustic loudness growth function, and both explain the same amount of variance in audiometric thresholds, we recommend future studies using only ABR wave V as a predictor in lieu of the psychoacoustic task. The combination of both loudness ranking and ABR Wave V amplitude being significant correlates reinforces the hypothesis that loudness growth is an important descriptor of hearing loss, specifically in lower frequencies, where participants have relatively normal loudness growth functions.

At 1000 Hz, the regression with tip frequency and Q10 estimates approached significance at p=0.0551. Both of these measures come directly from the PTC task, validating using it as a test in patients with SNHL.

However, at 4000 Hz no predictor significantly predicted audiometric thresholds. This could be for many reasons. All of the participants had much more severe hearing loss at 4000 Hz. It is possible that at these frequencies, the psychoacoustic tests are too straining. In addition, loudness growth functions are typically abnormal in participants with SNHL at high frequencies, but not at low frequencies, so it is expected that there is a correlation between their loudness growth function at low frequencies, but not at high frequencies. The variance in hearing loss at 4000 Hz was also much larger than the variance at 1000 Hz, with most participants having normal hearing at 1000 Hz, whereas at 4000 Hz some participants had moderate hearing loss and others had severe hearing loss. These discrepancies could possibly explain audiometric thresholds being predicted by our tasks of IHC and OHC loss at 1000 Hz but not at 4000 Hz.

5.4 Limitations

For each participant, the intensity level of the stimuli were the same, between 10 dB nHL to 70 dB nHL. While this was sufficient at 1000 Hz, where most participants had normal hearing, this was not sufficient at 4000 Hz. Some participants had very high hearing thresholds at 4000 Hz, which could explain the abnormal loudness growth pattern we see at 4000 Hz. Participants with poor hearing at 4000 Hz likely could not hear th weaker stimuli (e.g. the 10 dB nHL chirp). A better method would be to present stimuli only in the participant's dynamic hearing range, and for each participant pick 7 equidistant intensity levels within this range. This would result in a smoother and more accurate loudness growth curve.

A better psychoacoustic loudness growth function could have been inferred if the chirp stimulus was presented more times and if the bins between stimulus intensity were smaller. In this thesis, the stimulus intensity went up in 10 dB nHL steps, but a 5 dB step increase could create a smoother curve. However, there is a trade off between the number of presentations and length of time of the experiment. Decreasing the intensity steps by a half would double the testing time. In addition, the chirp stimulus is callibrated based on the average time delay of participants. However, the time delay for participants with hearing loss is perhaps different from participants with normal hearing. Thus, a chirp stimulus callibrated on people with hearing loss would give more accurate results as it would elicit a more synchronous response.

Finally, a major weakness of the ABR task is that it was hard to record in individuals. Of the 27 participants, only 17 completed the ABR tasks for both frequencies. The ABR may not be clinically feasible in elderly populations because of the low success rate.

While the PTC task is generally a good measure of hearing loss, there are some limitations with it. For one, the participant may respond that they are hearing the signal, but instead they may be hearing the interaction of the signal and noise (Moore, 2004). It is possible that the signal and masker are in a dead region, but the combination tone (e.g. 2f1 - f2, or f2-f1) falls in a region of normal hearing. Thus, the PTC could underestimate the extent of dead regions, due to response from these combination tones. This will affect participants with relatively intact low frequency hearing, but poor high frequency hearing. This type of hearing loss is stereotypical for SNHL patients. This issue could have been overcome if low pass noise was played with the masker, to mask the perception of the difference tone (Moore, 2004).

The detection of beats can also affect PTC results (Alcántara and Moore, 2002). Beats are amplitude fluctuations that occur when two tones, in this case, the signal and masker, are presented simultaneously and are close in frequency. When the signal and masker frequency are close together, the perception of beats might occur, and the participant might respond to hearing the stimulus when instead they hear this beat. Again, this may underestimate the extent of the dead region, as the beat provides a cue to the participant. Furthermore, if the masker and signal are at the exact same frequency, then beats do not occur. Thus, when the masker is at the signal frequency, the participant does not have a cue to aid their signal detection, but they do when the masker's frequency is near the signal. This could result in a false tip in the PTC, and lead to the false negative diagnosis of no dead region. There is a tradeoff between beat detection and masker bandwidth. If the bandwidth of the masker is increased, the likelihood of hearing beats is greatly diminished. However, this lowers the frequency specificity of the PTC task. As this experiment used a relatively small bandwidth masker, participants could have been hearing beats as a cue.

Another limitation to this thesis is that it only included one measure of IHC loss. Including other measures of IHC loss could have explained more of the variance in audiometric threshold. Future studies should include another measure of IHC loss.

Finally, future studies should aim to include an age-matched control group with normal hearing. While this study tried to find such group, we were unable to.

Chapter 6

Summary and Conclusions

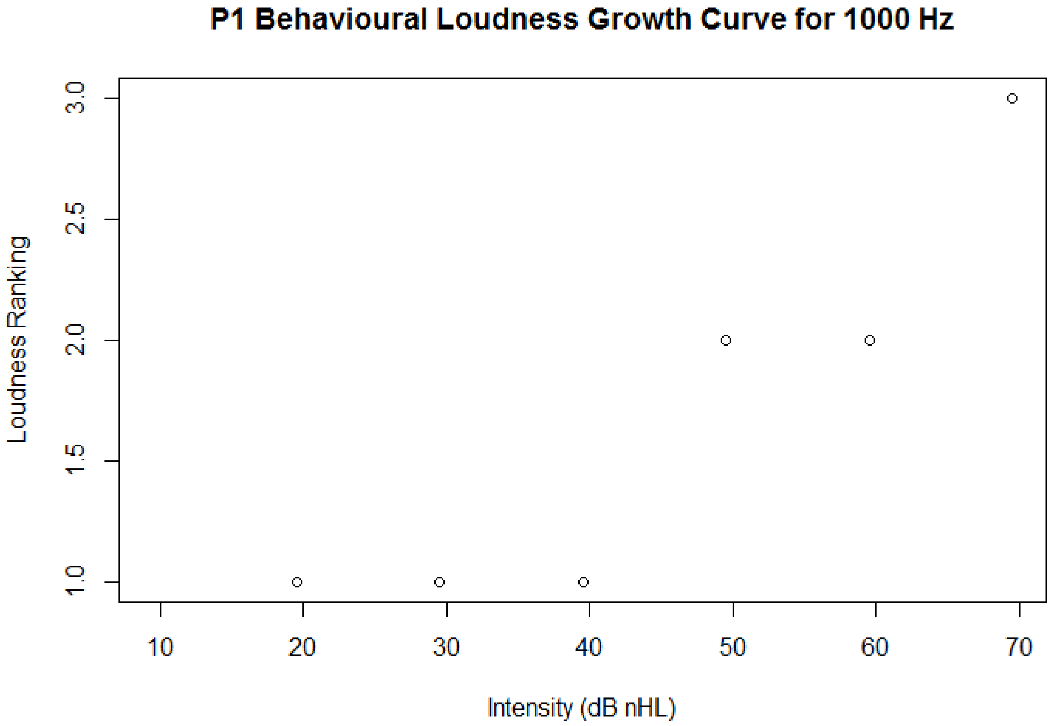
This thesis describes an experiment in which we recruited and tested 27 participants with SNHL. Participants went through extensive pre-screening to ensure they had no cognitive deficits, middle ear problems, or tinnitus. All participants completed an ABR assessment, behaviour loudness growth task, and forward and backward PTC at 1000 and 4000 Hz. We found a significant correlation between behaviour loudness ranking and stimuli intensity for 1000 Hz, but not 4000 Hz. In addition, we found a significant linear correlation between Wave V amplitude and stimulus intensity at both 1000 Hz and 4000 Hz. Wave V amplitude corresponding to stimulus intensity level correlated with loudness ranking for the same stimulus intensity at 1000 Hz, but not at 4000 Hz. In addition, we established Q10 and tip frequency norms for participants with SNHL at 1000 Hz and 4000 Hz.

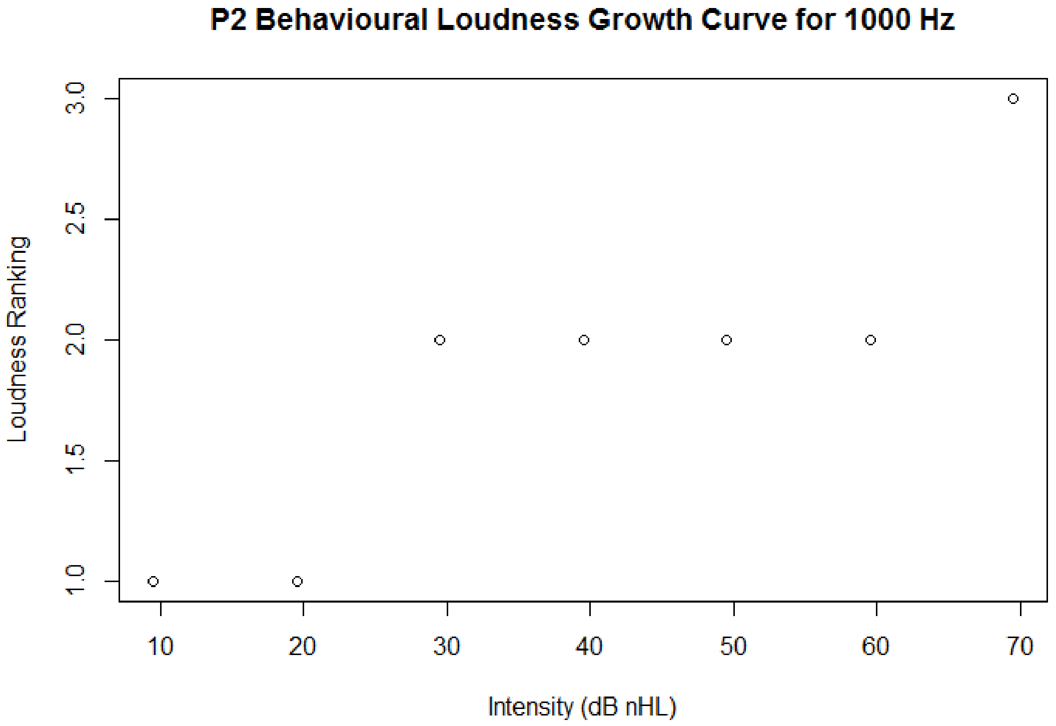
This thesis has contributed to research by helping establish norms for octave band chirp ABR wave V amplitude and behaviour loudness growth task, for participants with SNHL. In addition, we were able to demonstrate that the ABR based loudness growth function was highly reliable and correlated strongly with the behavioural one. Finally, this thesis makes an important contribution in validating LGF obtained from the ABR.

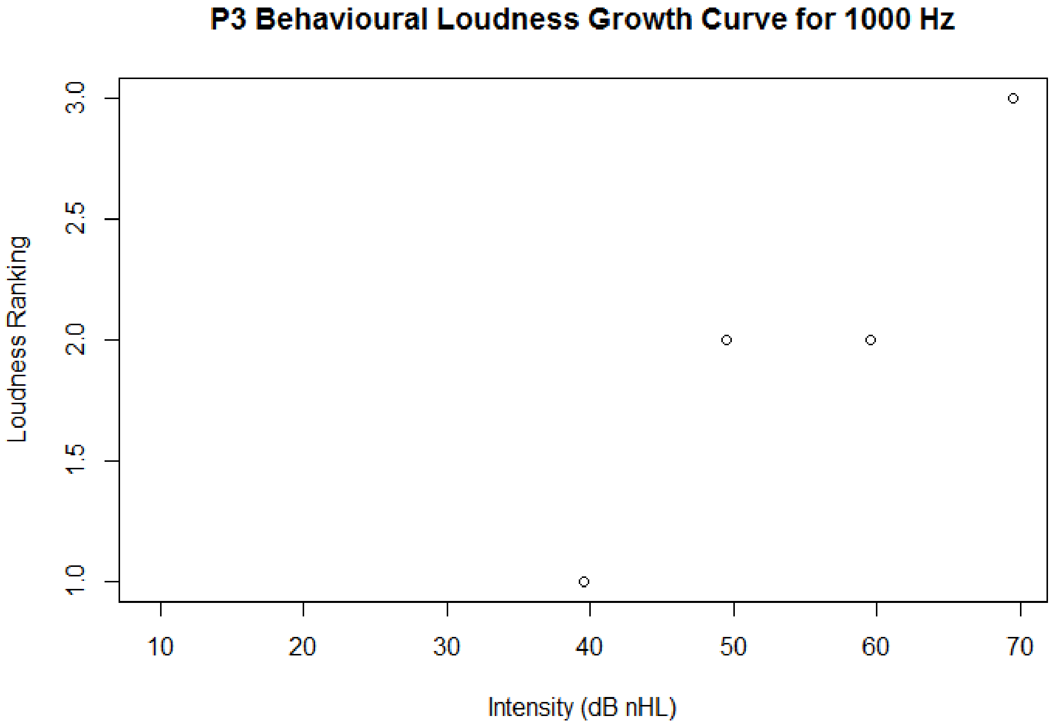
Future studies should be done with the bandwidth of the masker and the presence of low pass noise systematically changed for each PTC for each participant. This way, the true extent of beat perception and combination tone perception on the PTC can be evaluated. In addition, more frequencies should be tested. Most participants in this study begin having hearing loss around 3000-4000 Hz. Including a higher frequency case would provide much more information. Finally, a continuous rating scale for the loudness growth task could have provided a better fit between both the ABR and the ranking. Future studies should include more trials for ABR to improve the signal to noise ration and lower the error. Although with the increase of trials, the amount of time spent increases, but the 1000 Hz stimuli could perhaps be dropped.

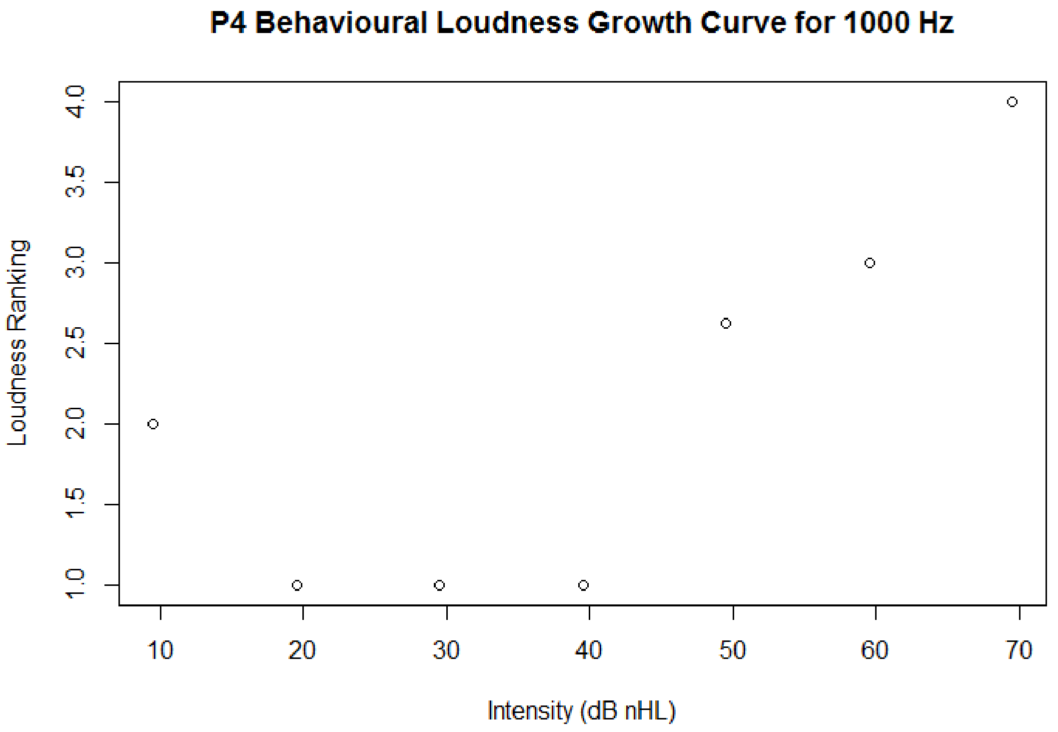
Appendix

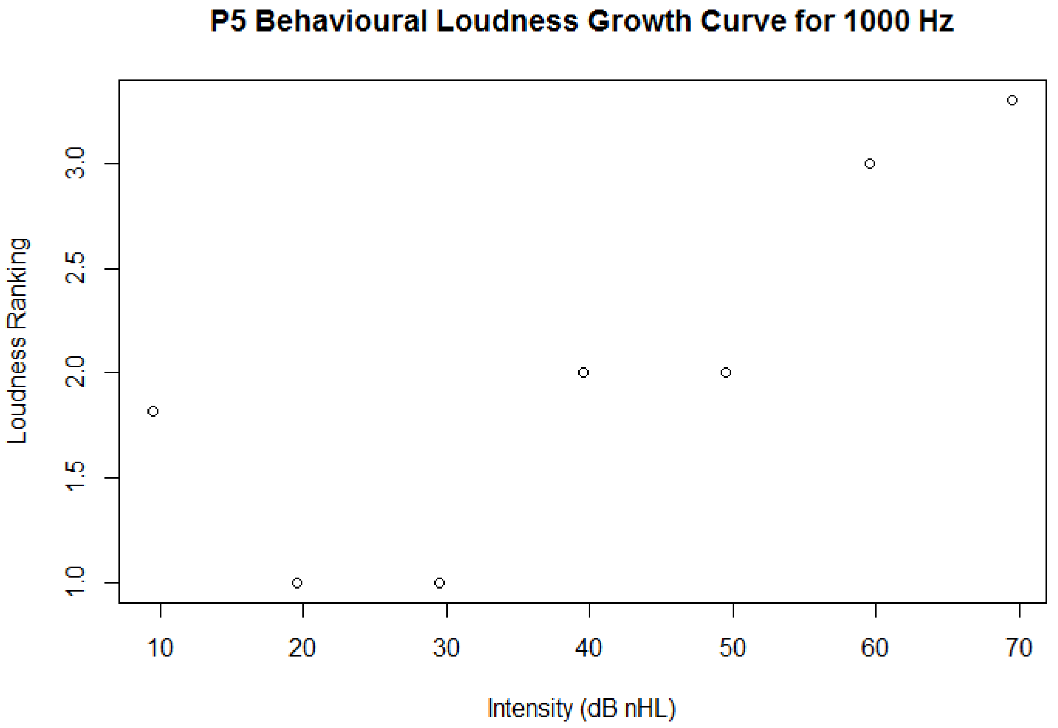
Behaviour Loudness Growth Curves for all Participants at 1000 Hz

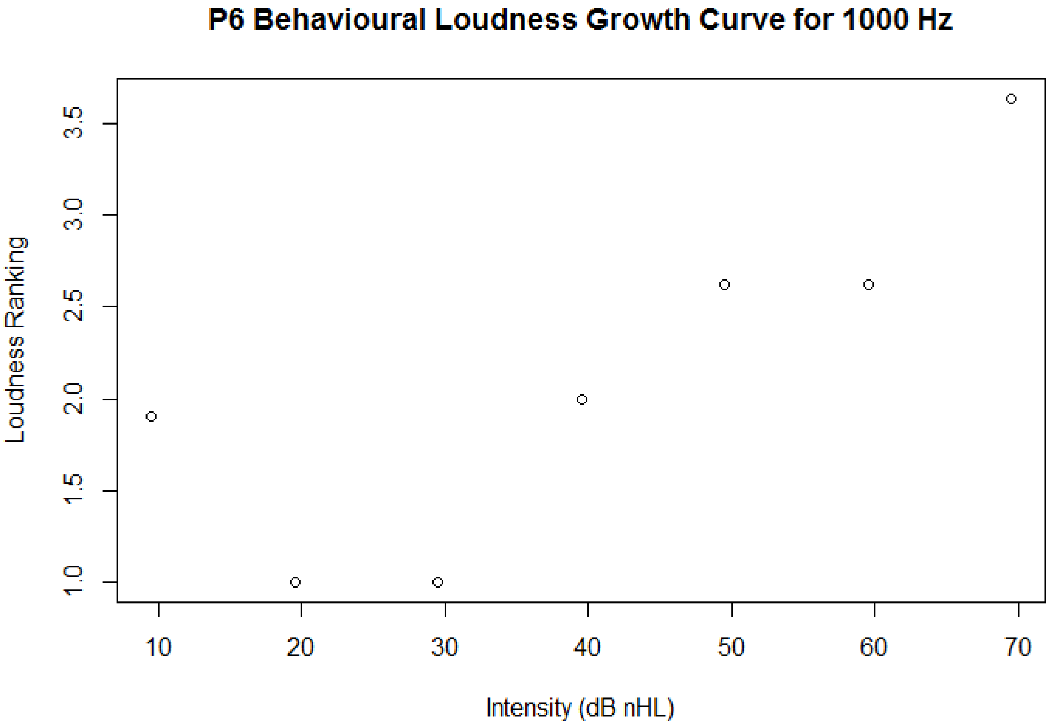


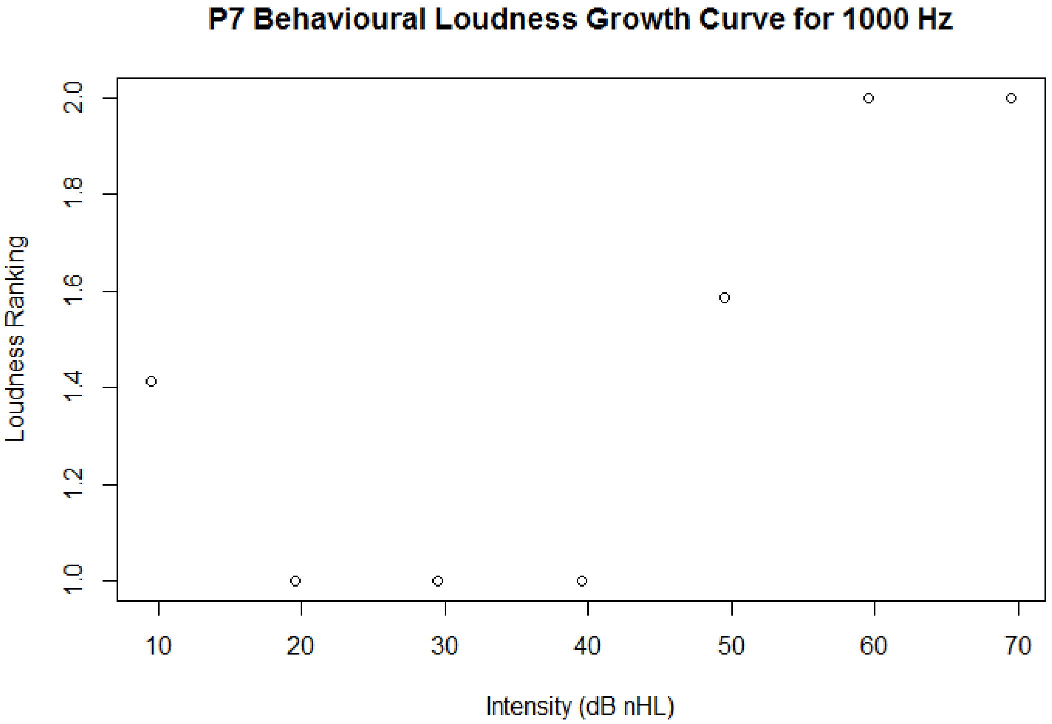

P2 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

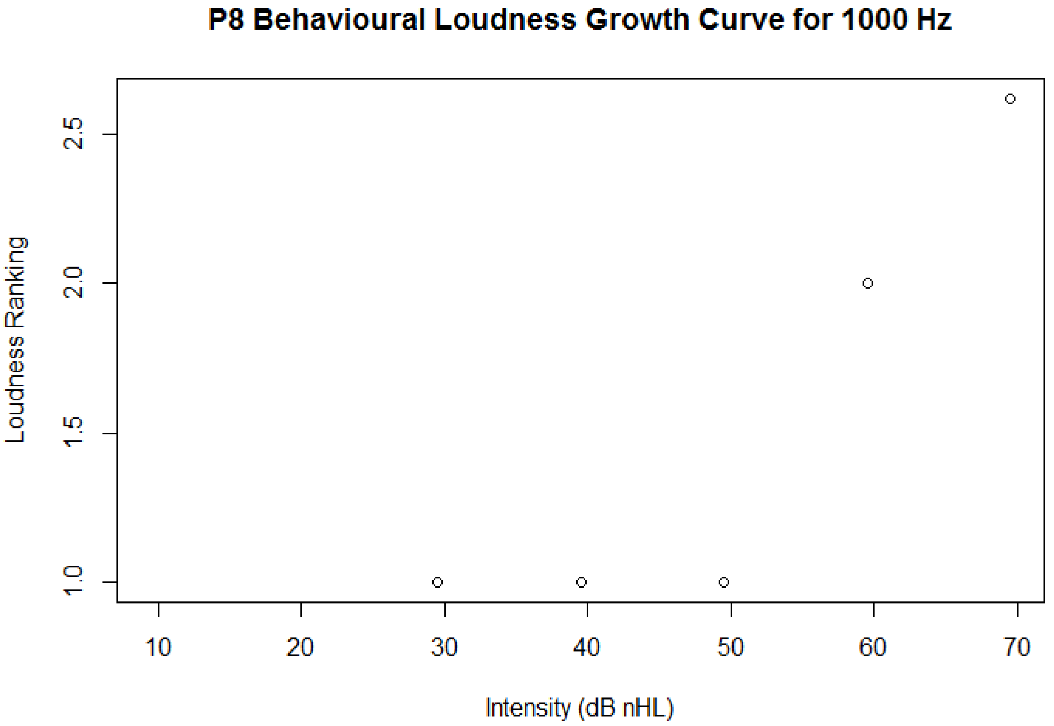

P3 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

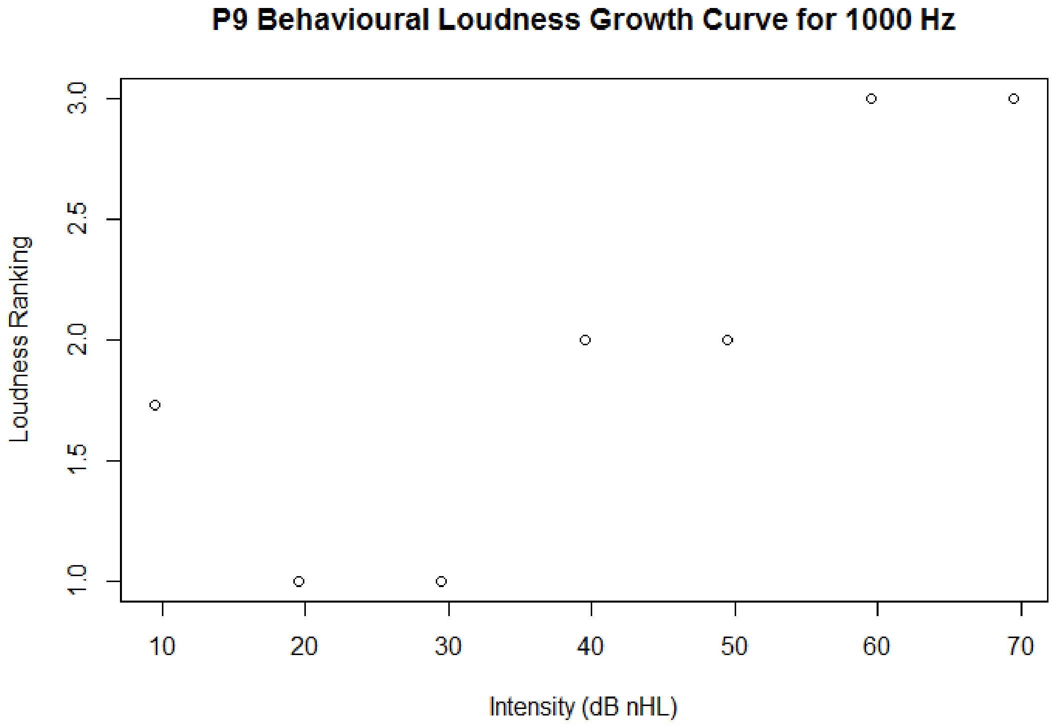

P4 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

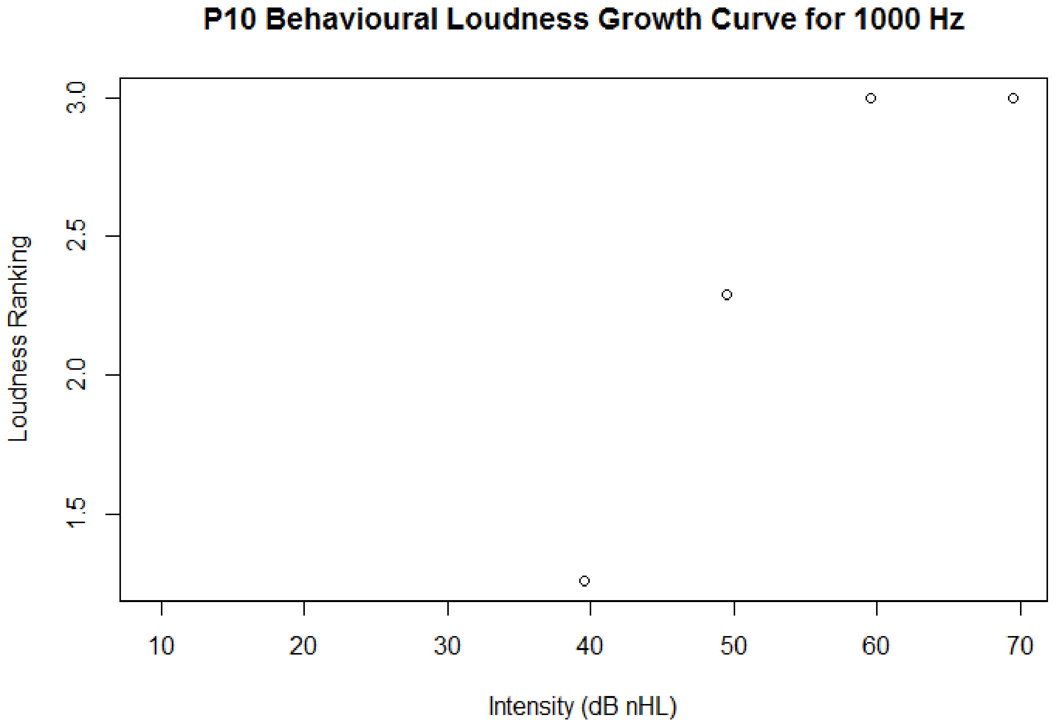

P5 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

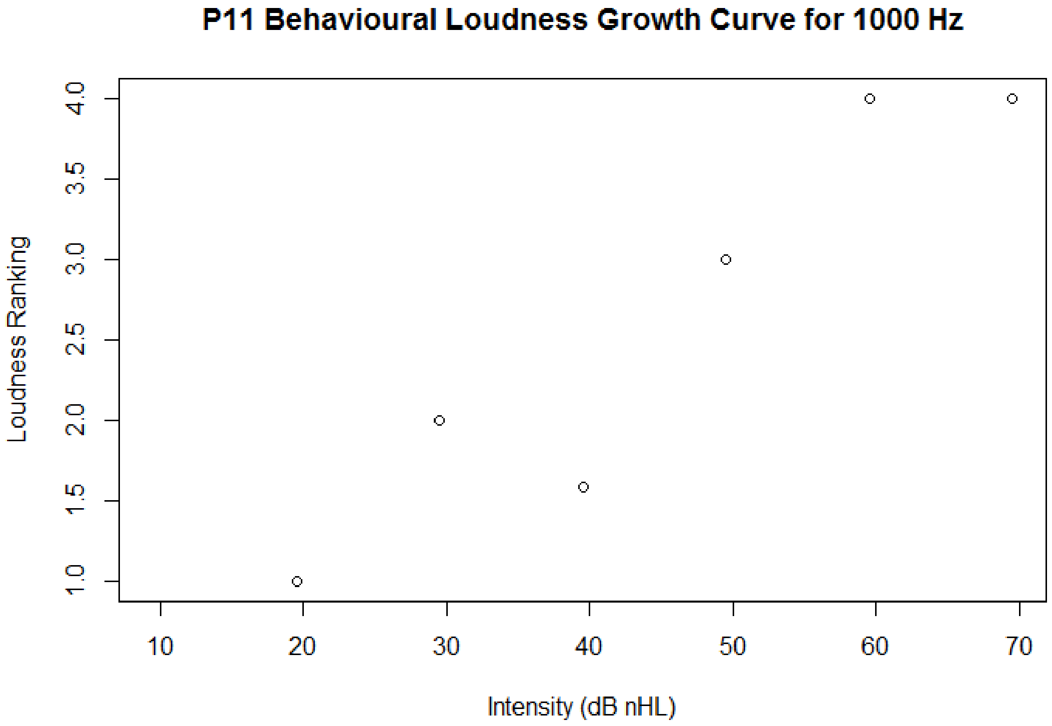

P6 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
10
20
30
40
50
60
70
Intensity (dB nHL)

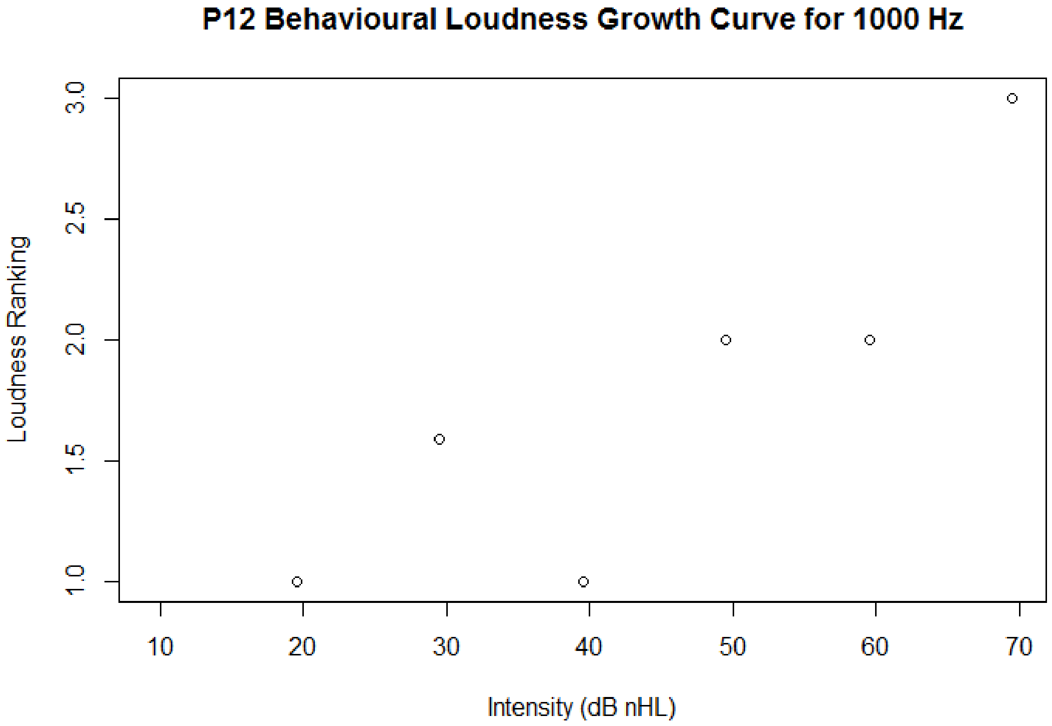

P7 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.2
1.4
1.6
1.8
2.0
10
20
30
40
50
60
70
Intensity (dB nHL)

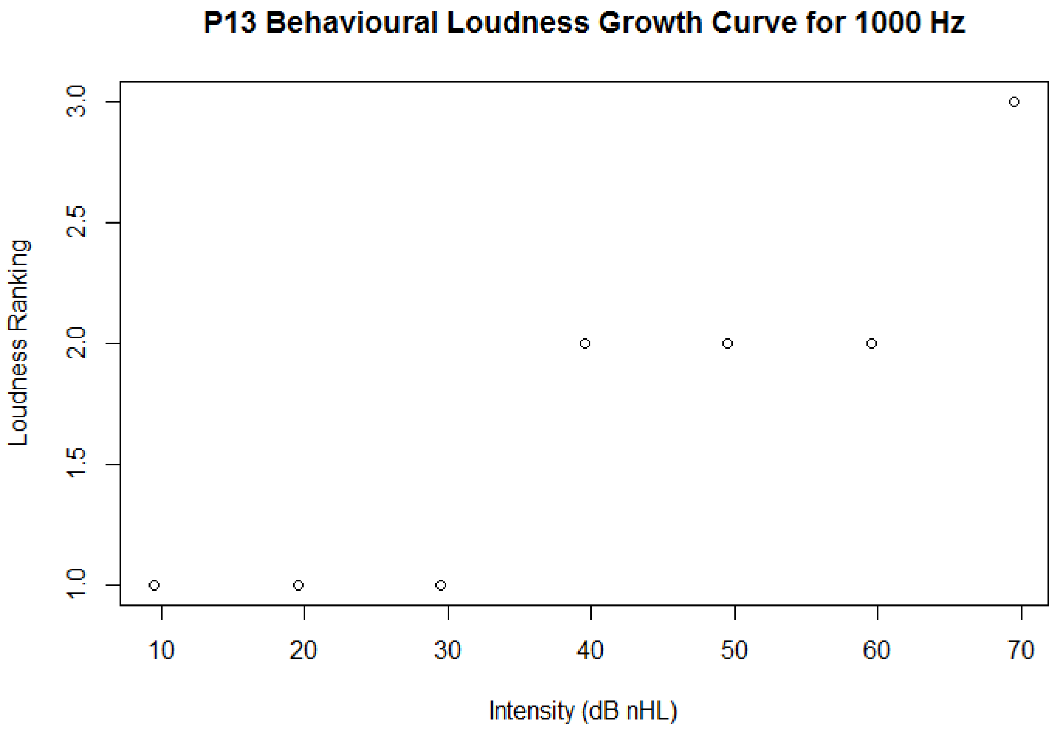

P8 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
10
20
30
40
50
60
70
Intensity (dB nHL)

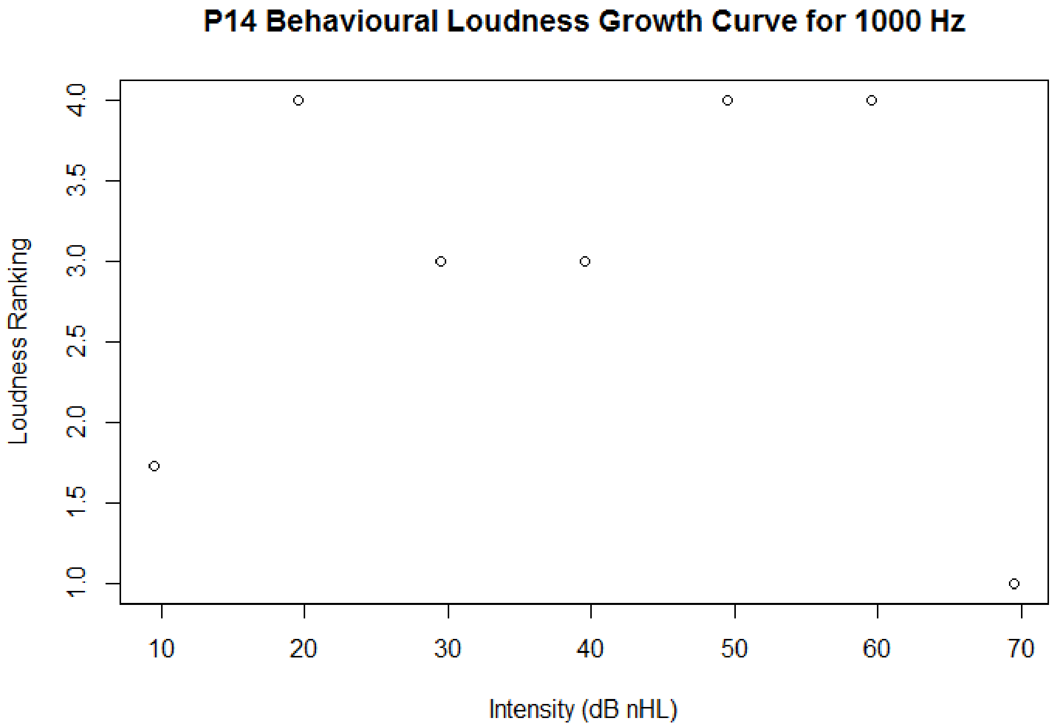

P9 Behavioural Loudness Growth Curve for 1000 Hz

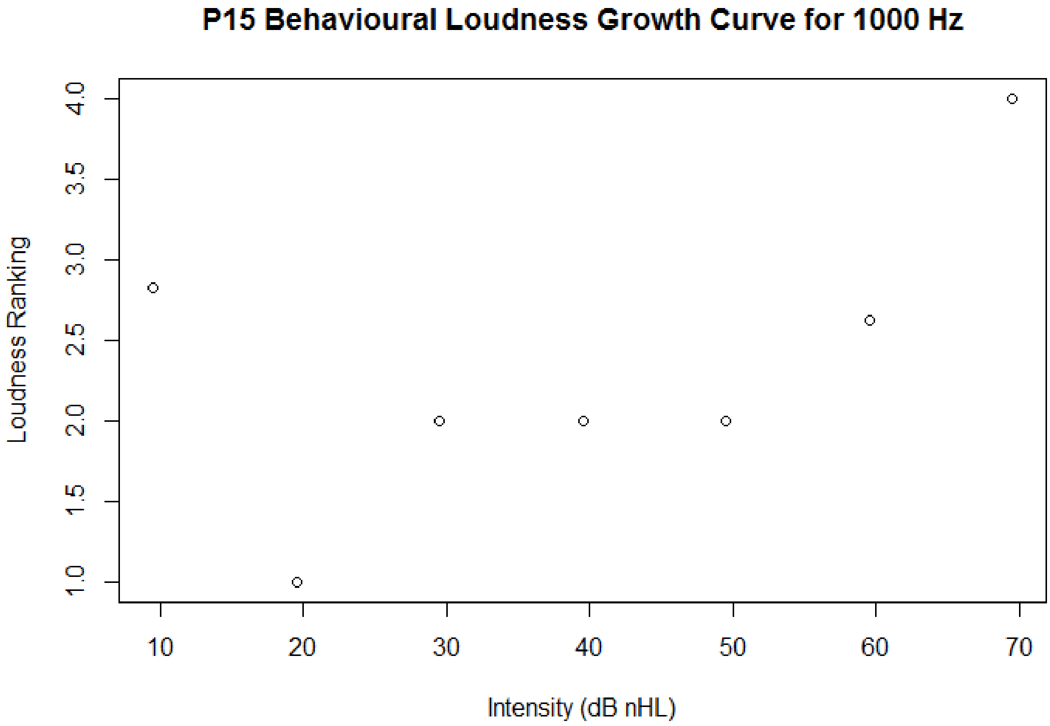

P10 Behavioural Loudness Growth Curve for 1000 Hz

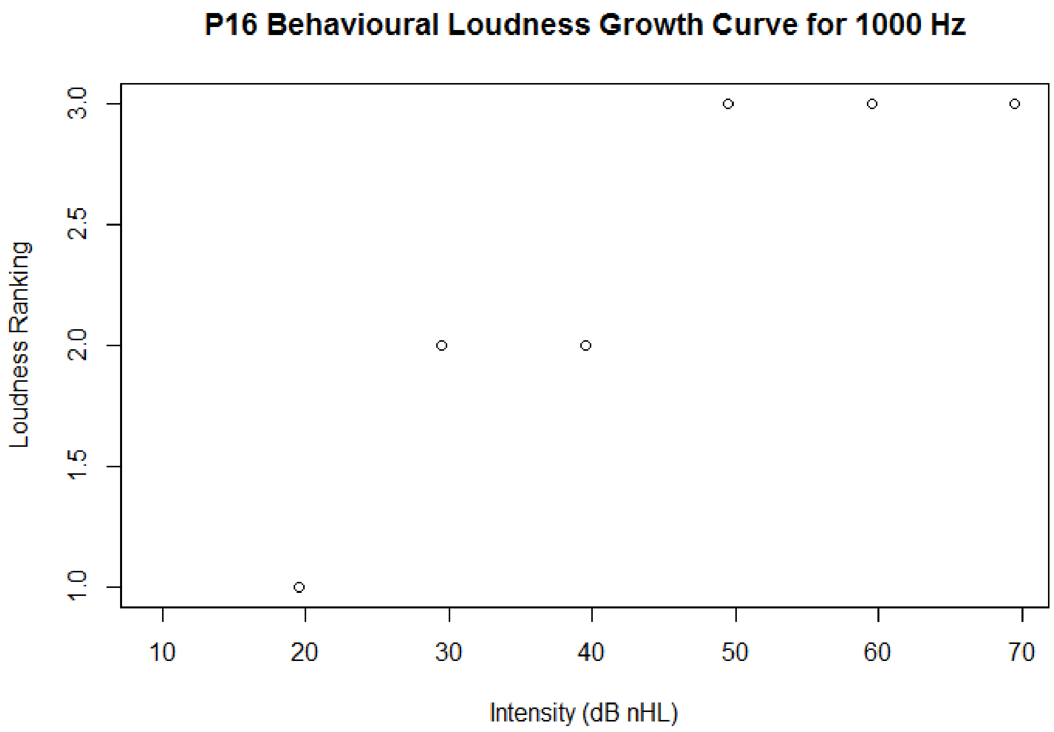

P11 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

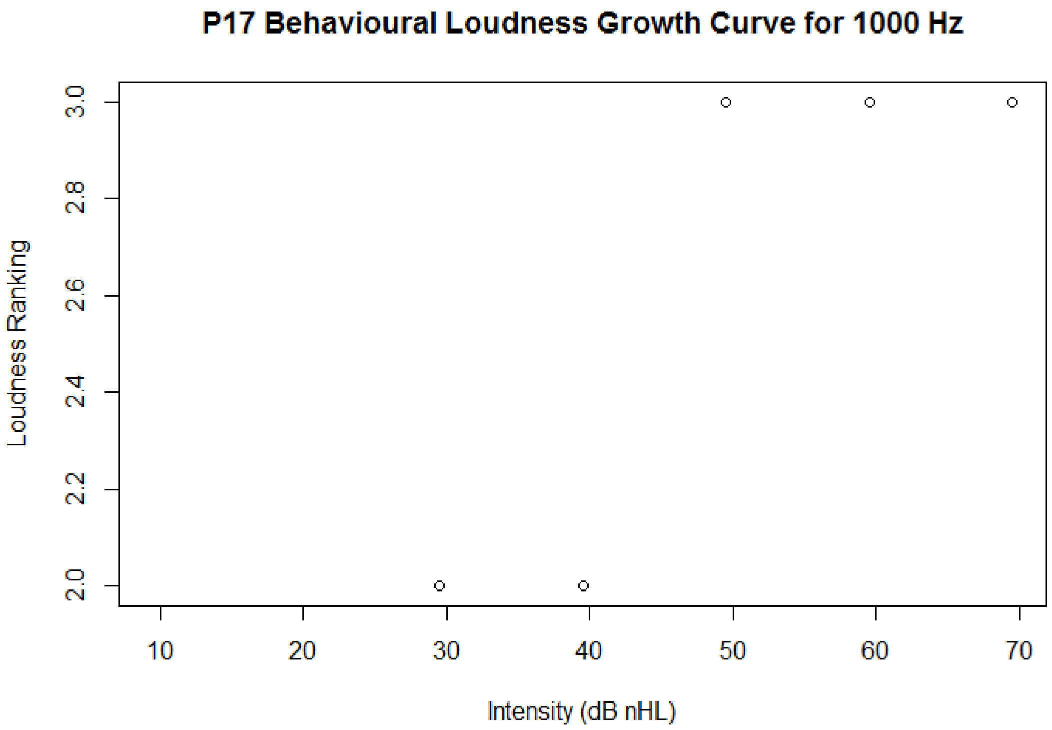

P12 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

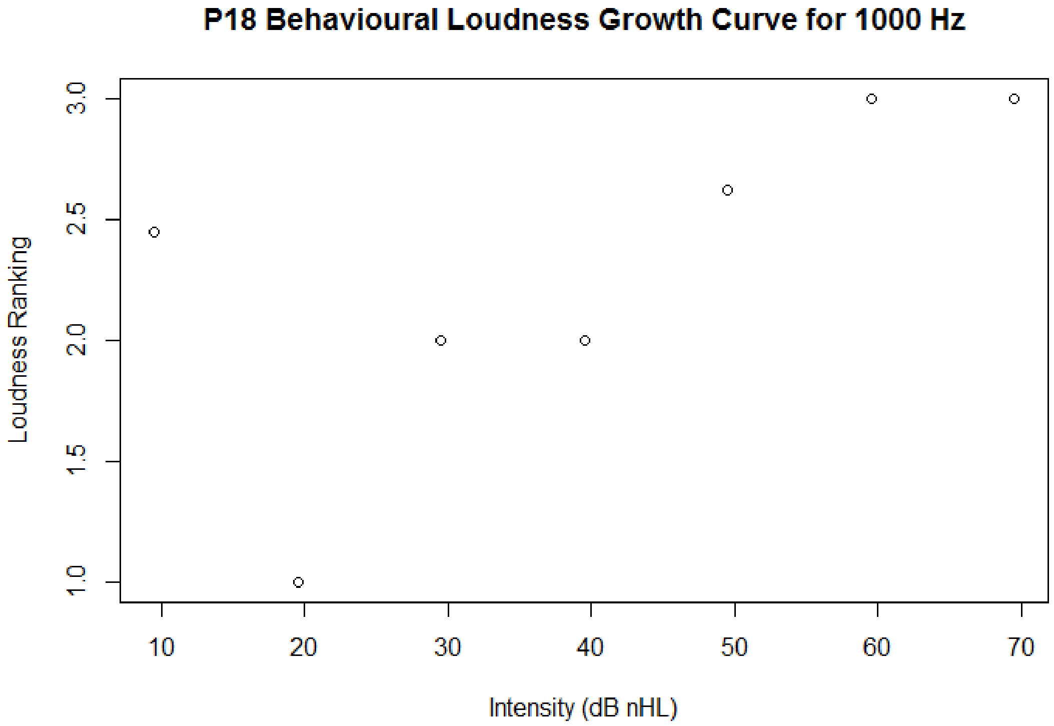

P13 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

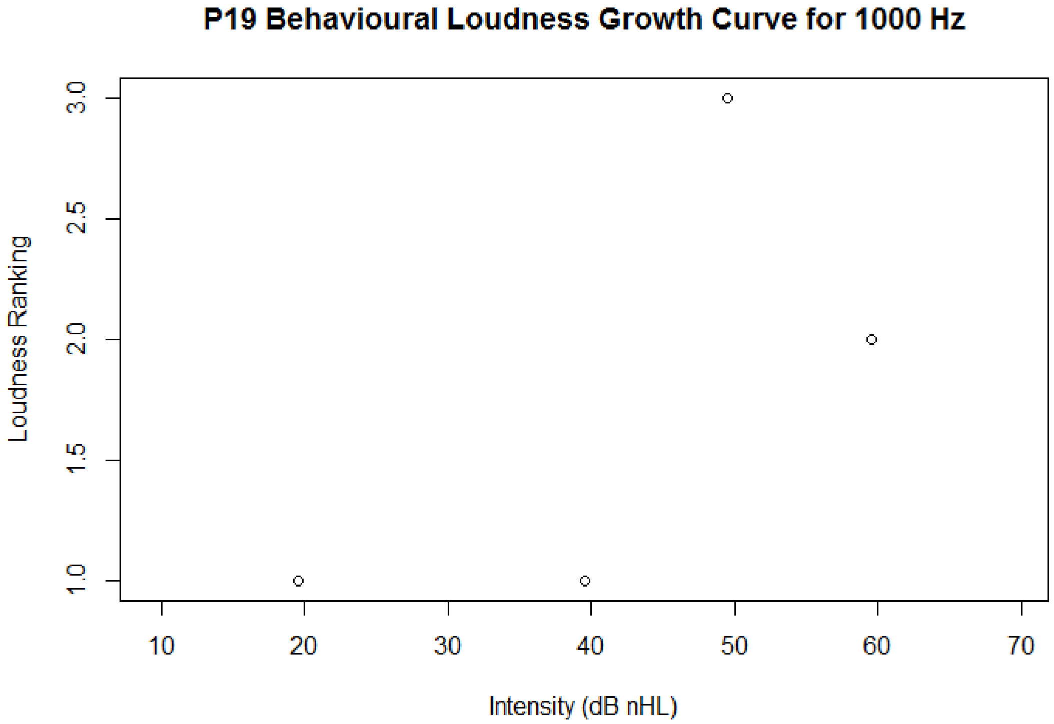

P14 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

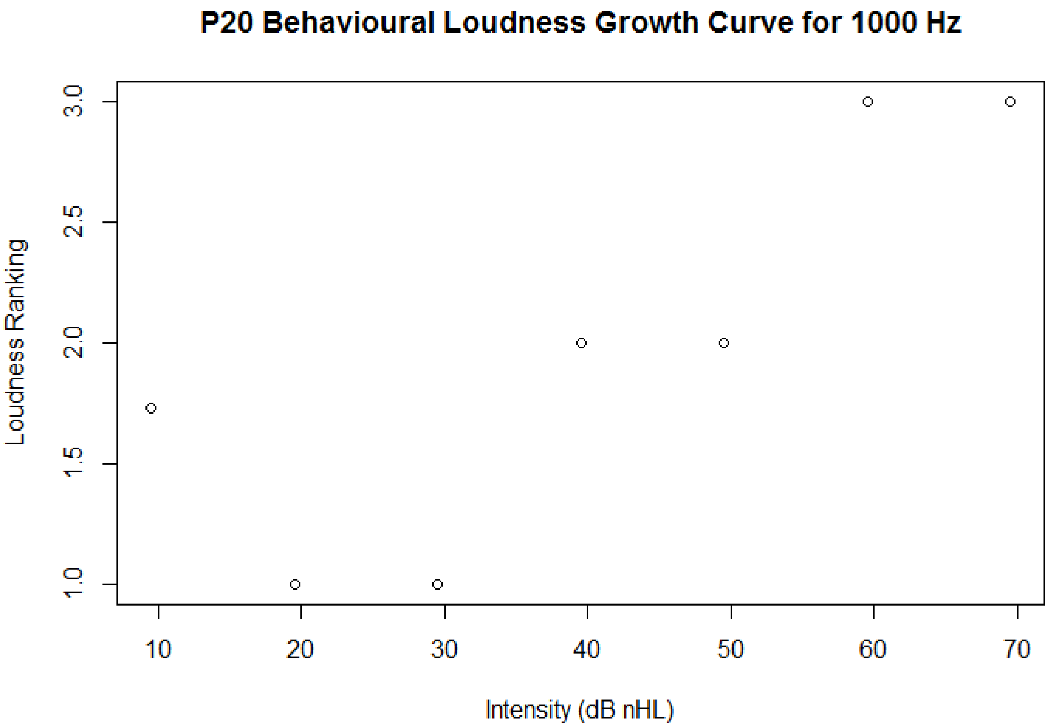

P15 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

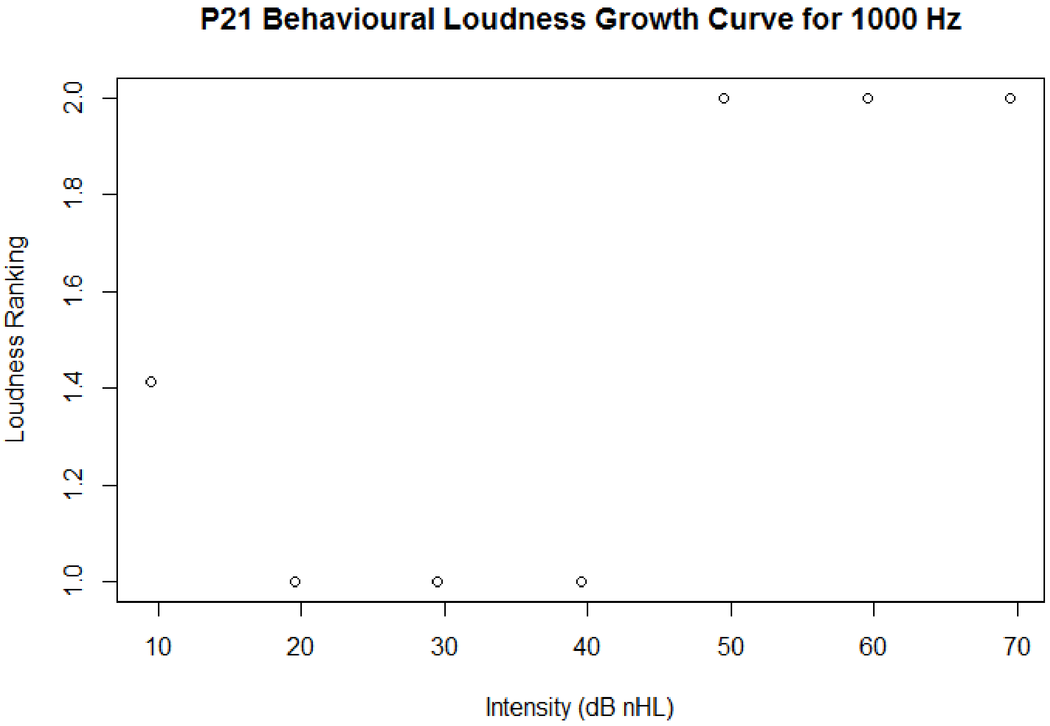

P16 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

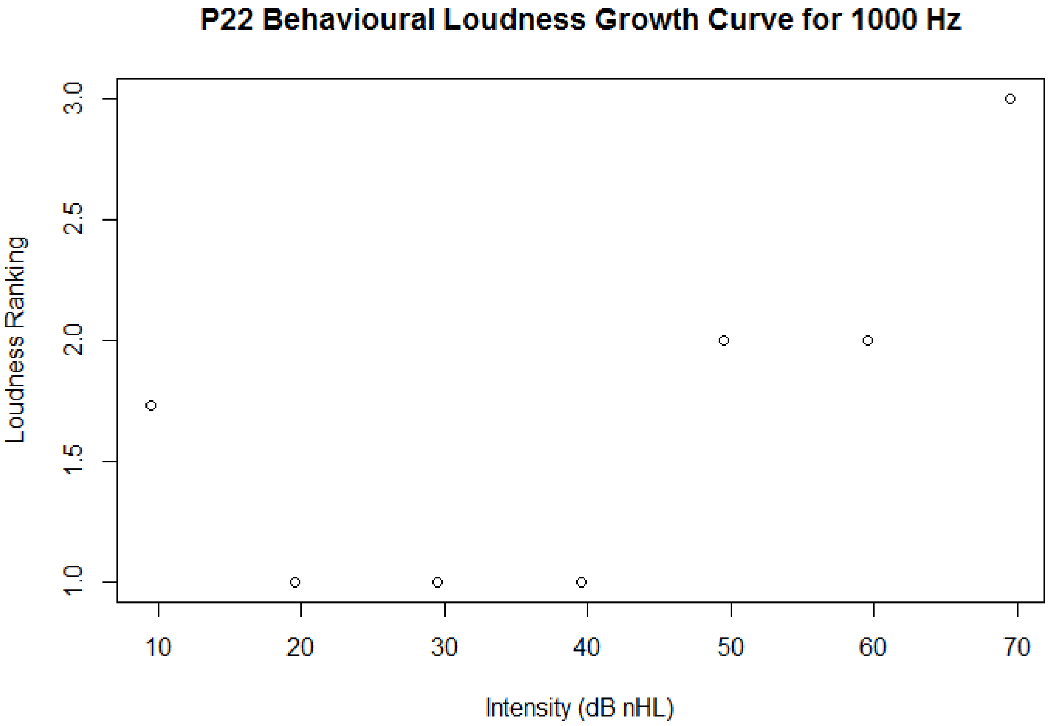

P17 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
2.0
2.2
2.4
2.6
2.8
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

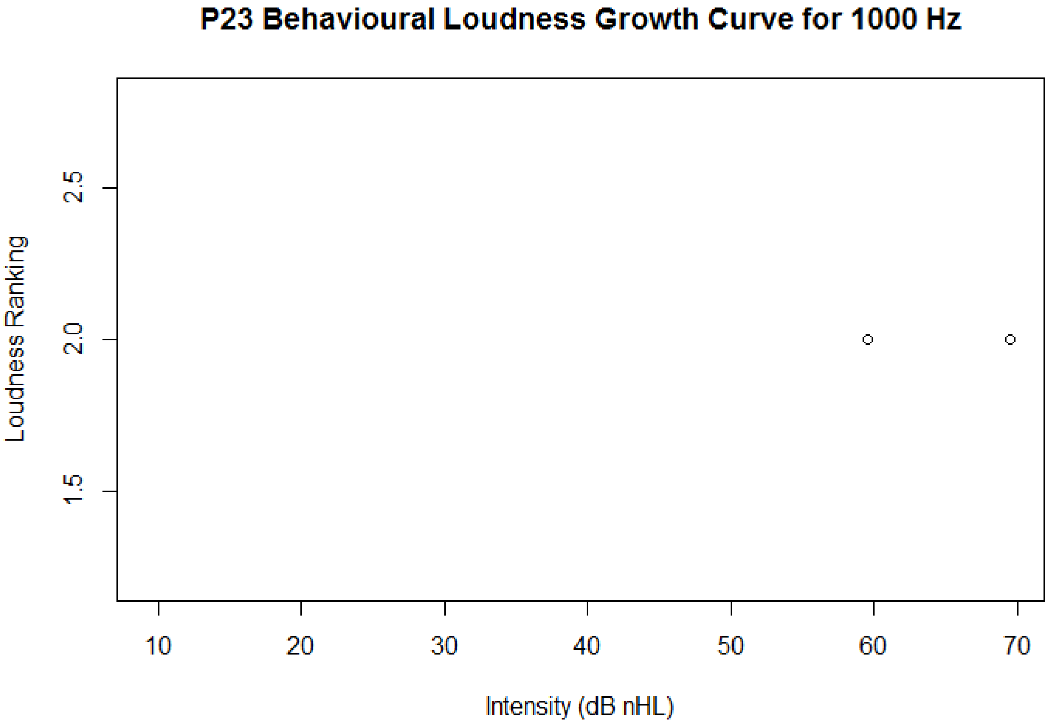

P18 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

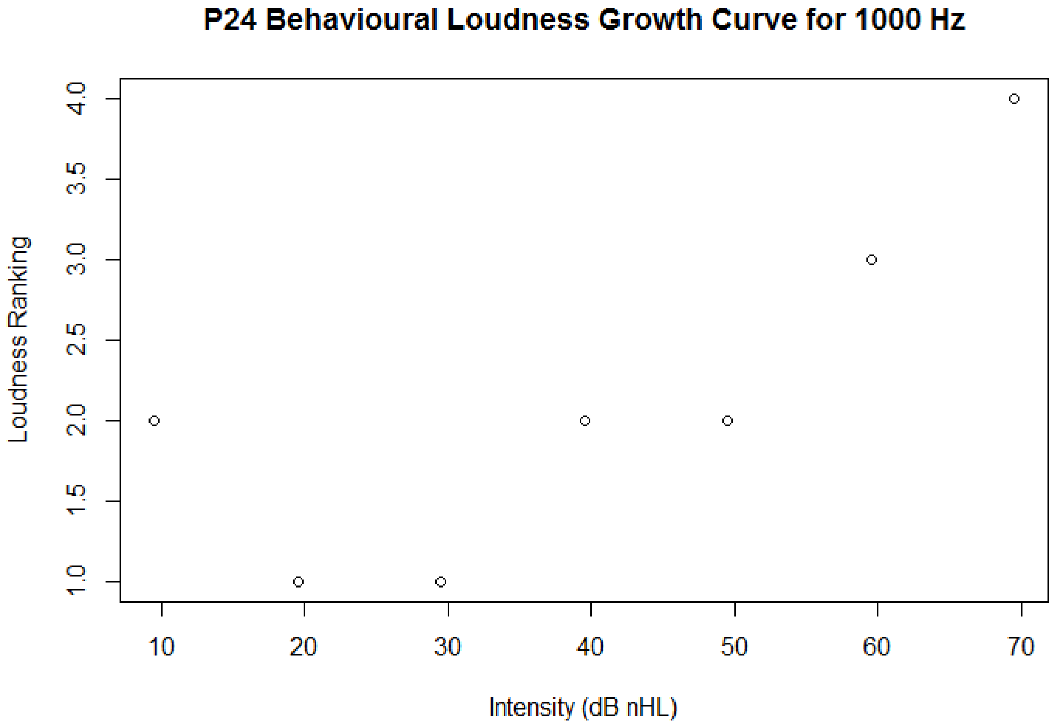

P19 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

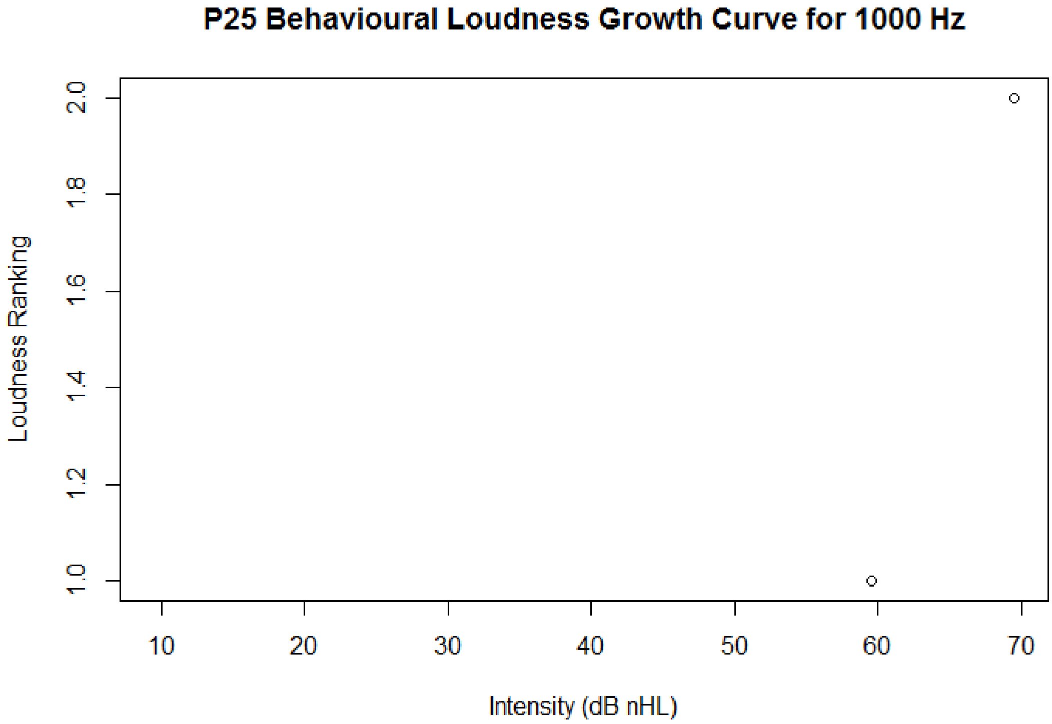

P20 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

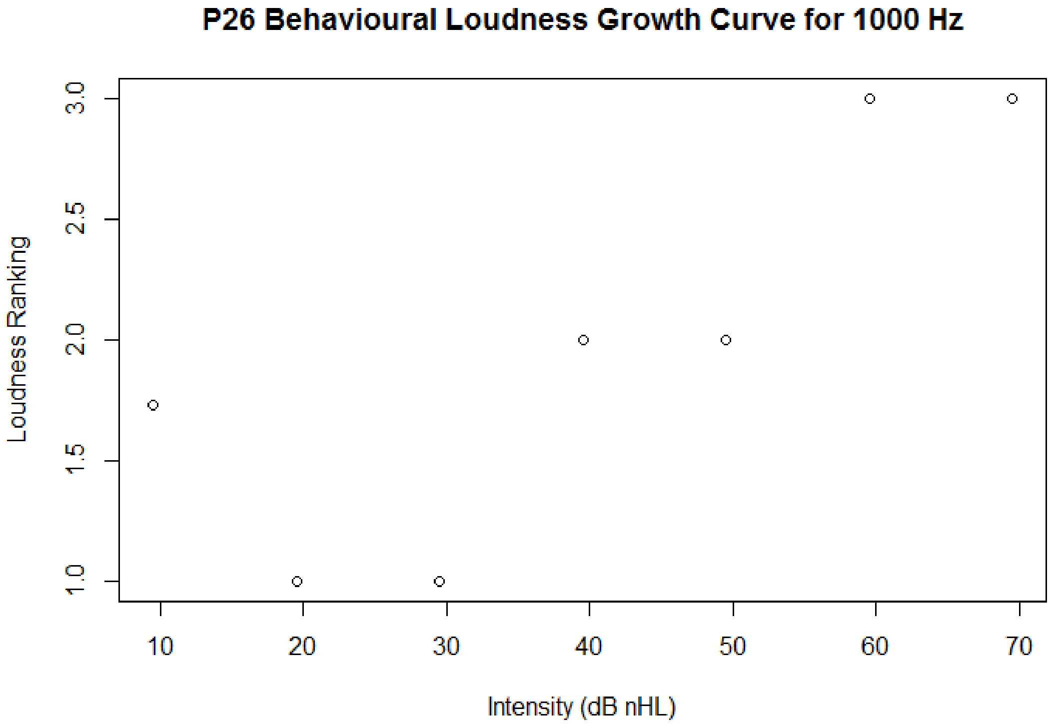

P21 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.2
1.4
1.6
1.8
2.0
10
20
30
40
50
60
70
Intensity (dB nHL)

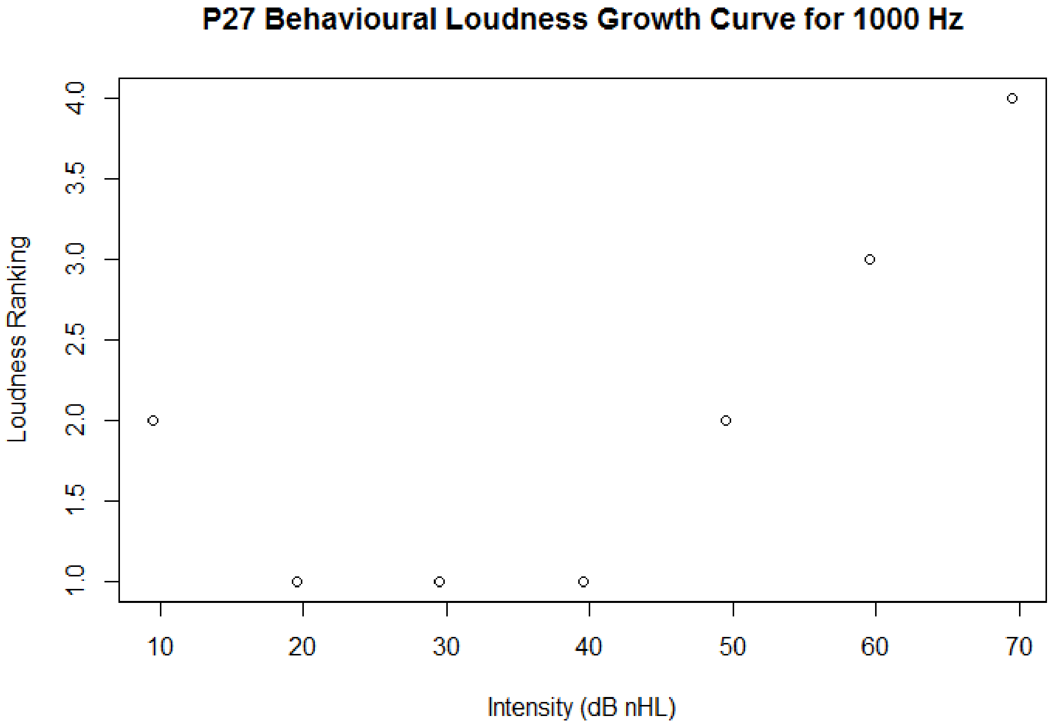

P22 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)


P23 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.5
2.0
2.5
10
20
30
40
50
60
70
Intensity (dB nHL)

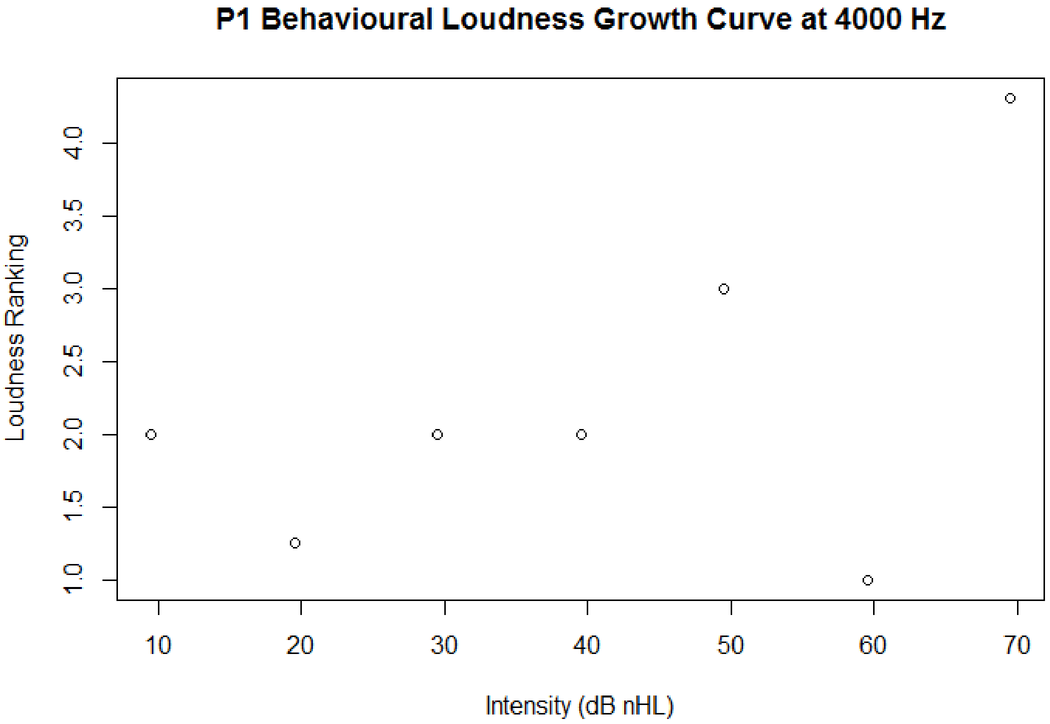

P24 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

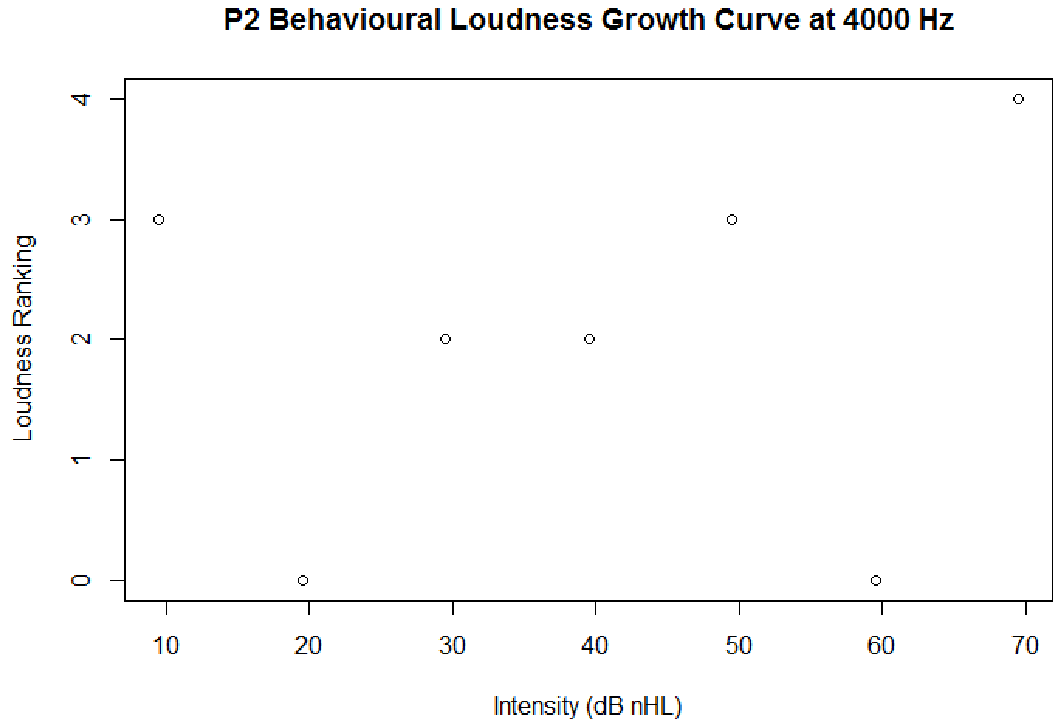

P25 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.2
1.4
1.6
1.8
2.0
10
20
30
40
50
60
70
Intensity (dB nHL)

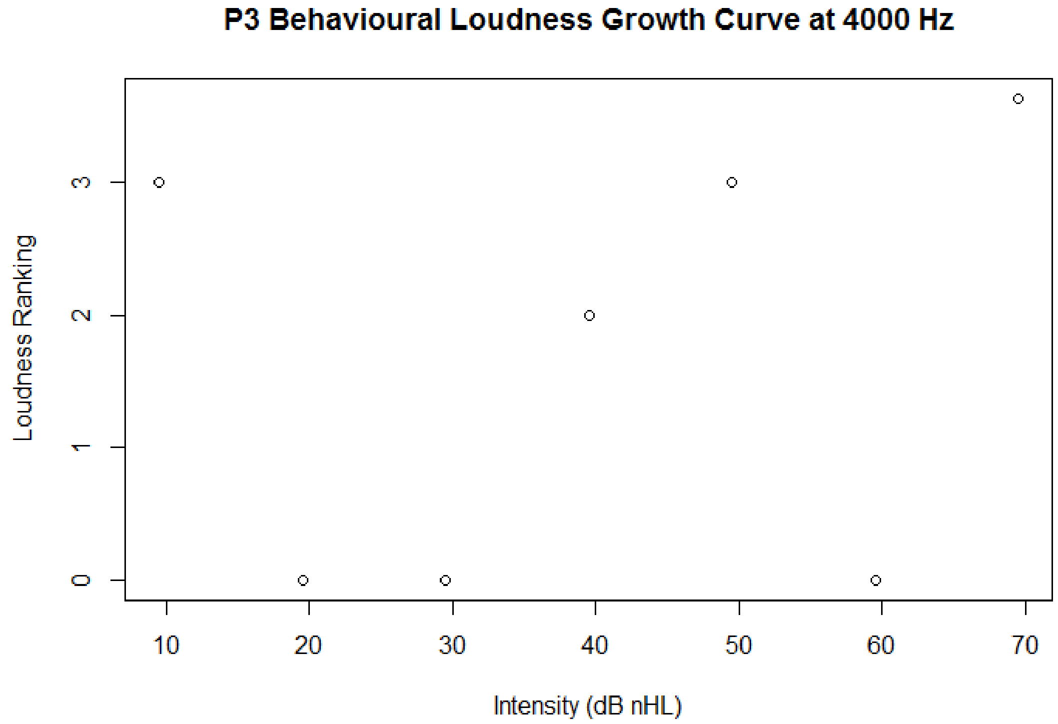

P26 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

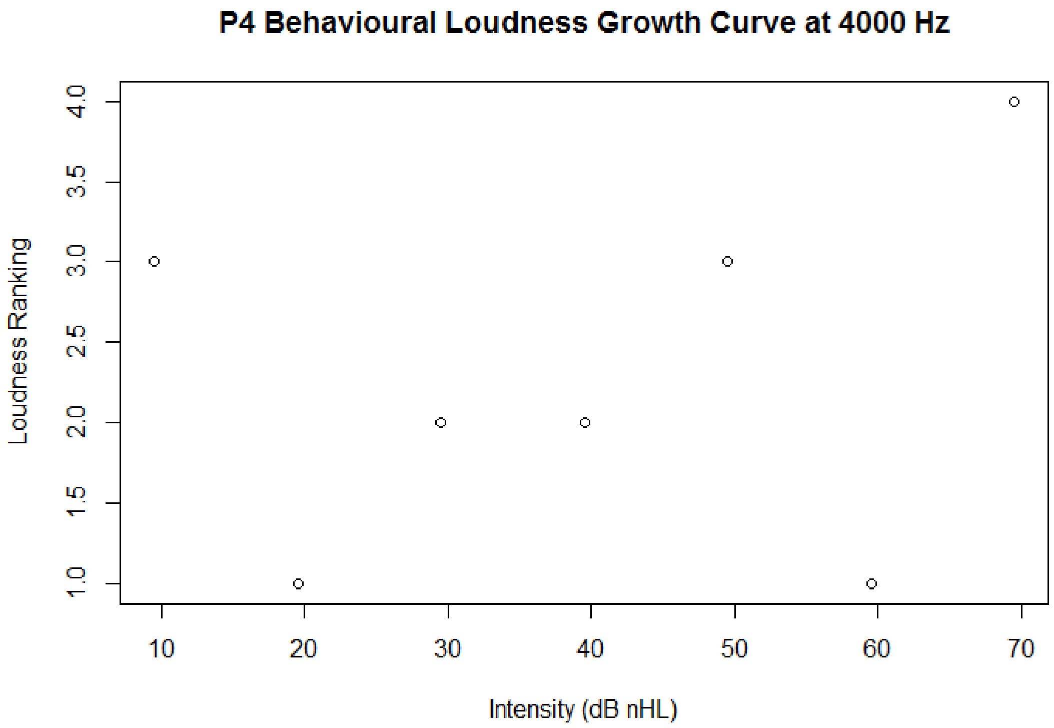

P27 Behavioural Loudness Growth Curve for 1000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

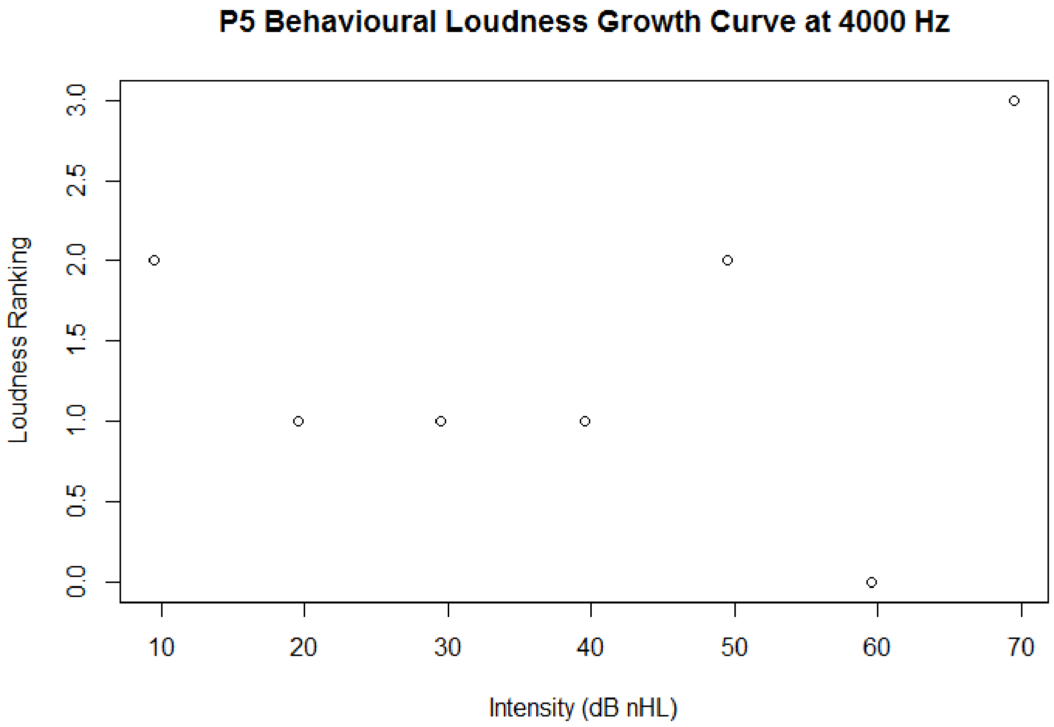
Behaviour Loudness Growth Curves for all Participants at 4000 Hz

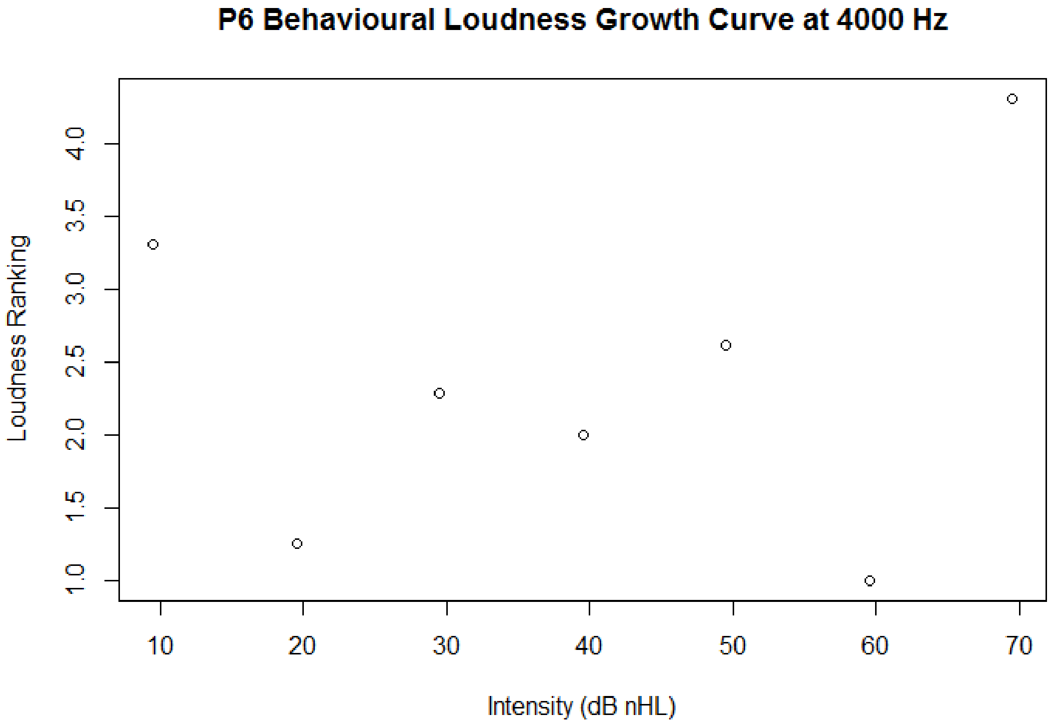


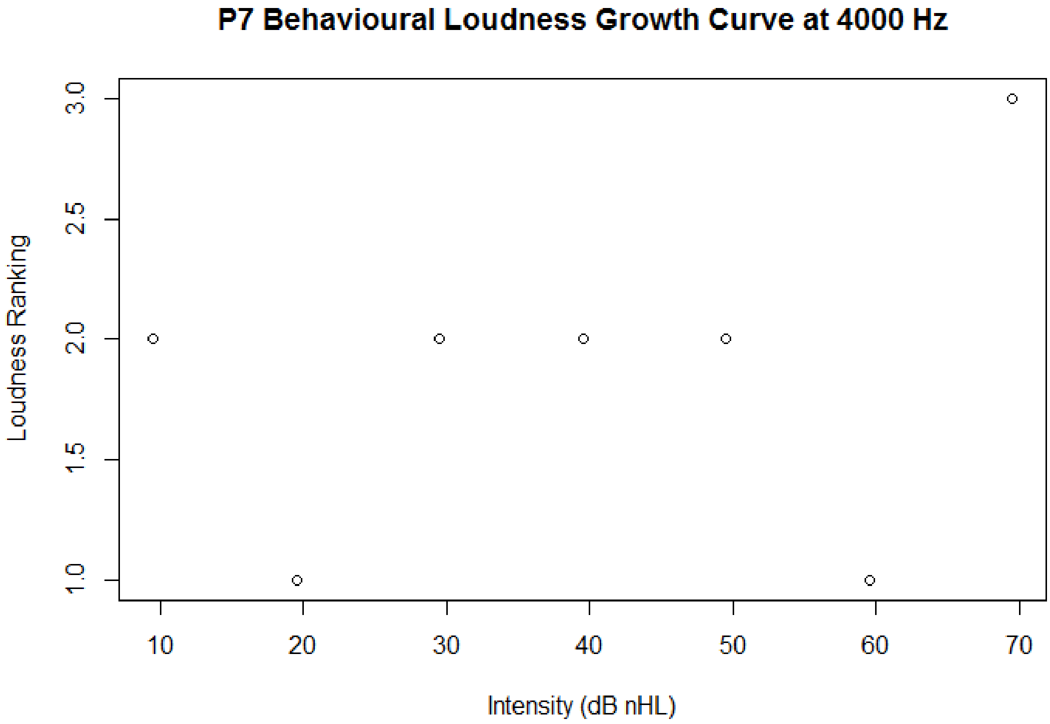

P2 Behavioural Loudness Growth Curve at 4000 Hz

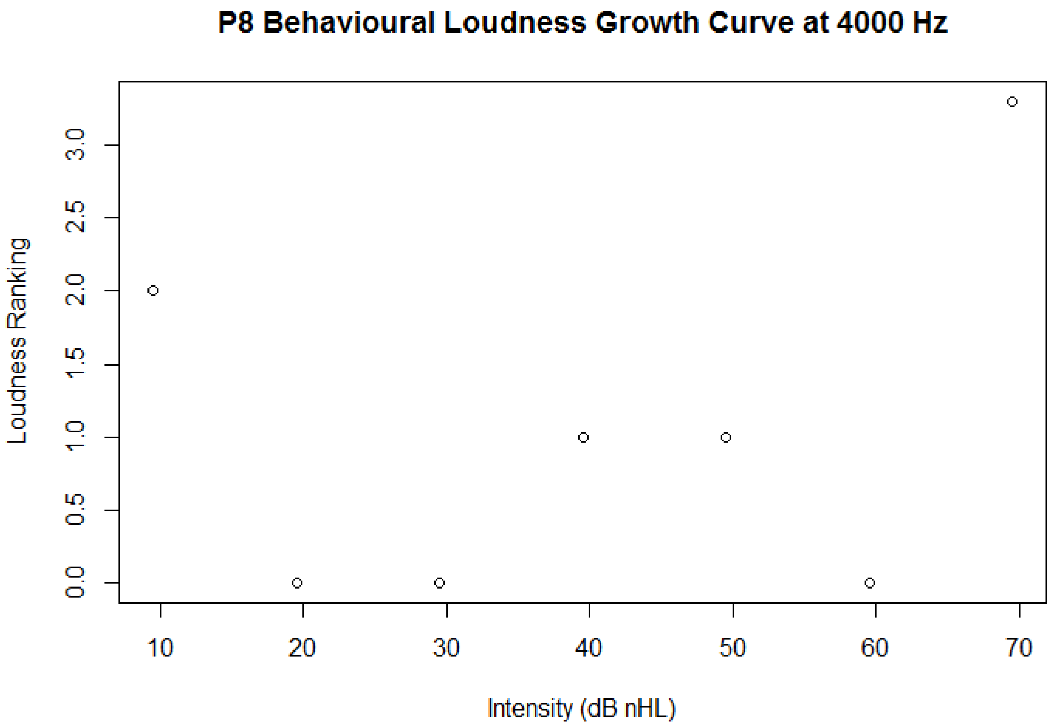

P3 Behavioural Loudness Growth Curve at 4000 Hz

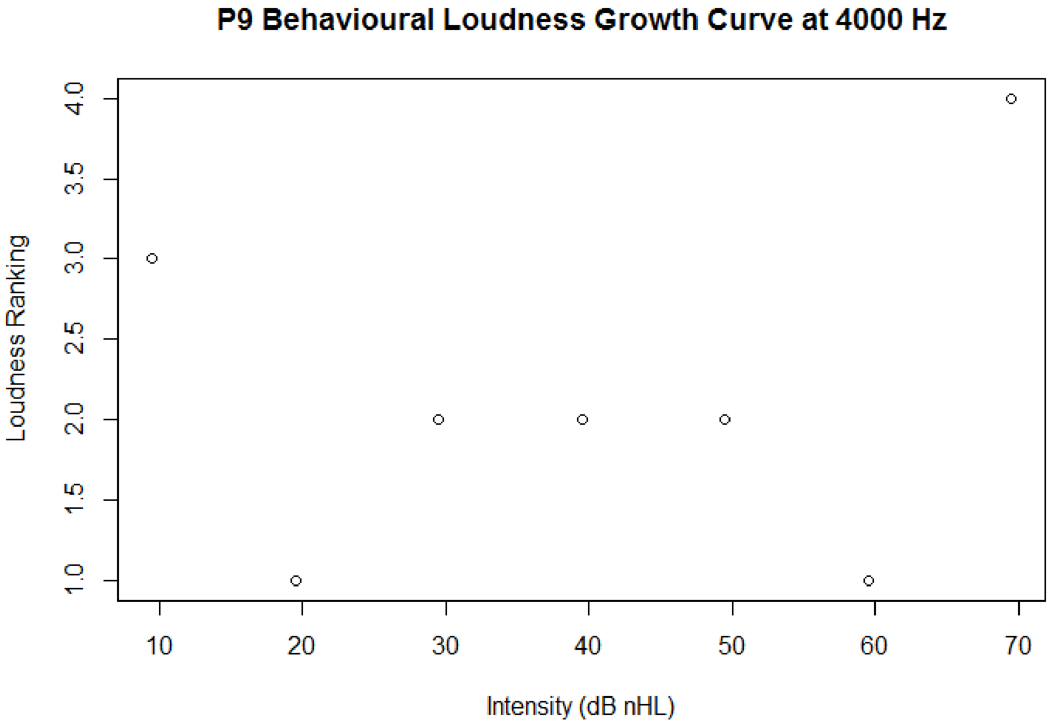

P4 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

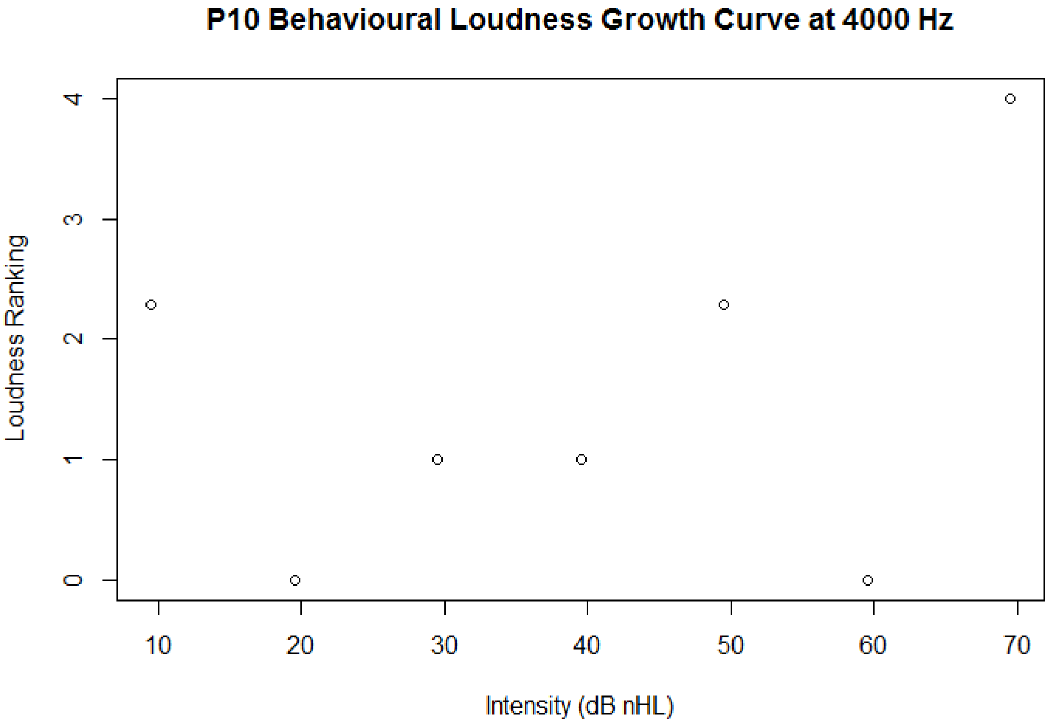

P5 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0.0
0.5
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

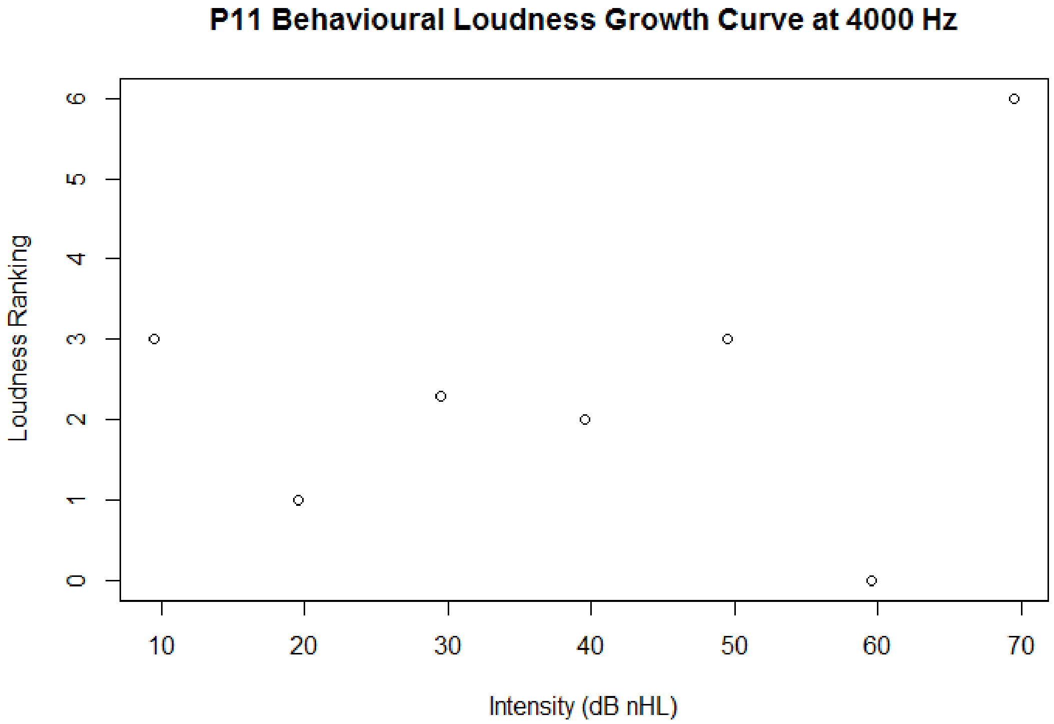

P6 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

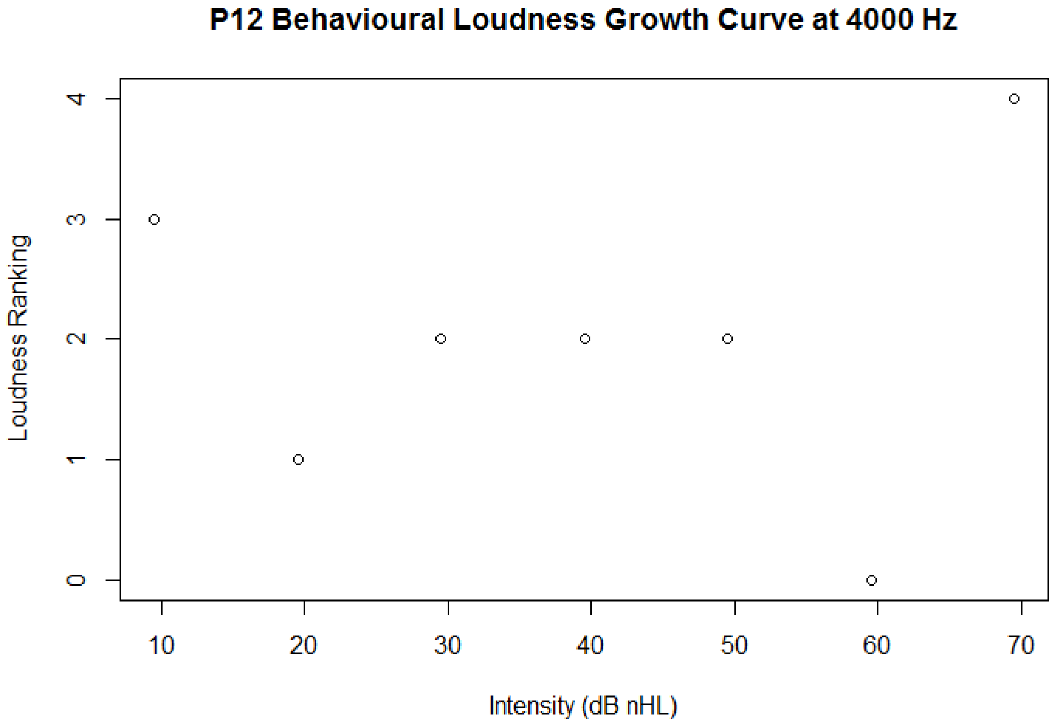

P7 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

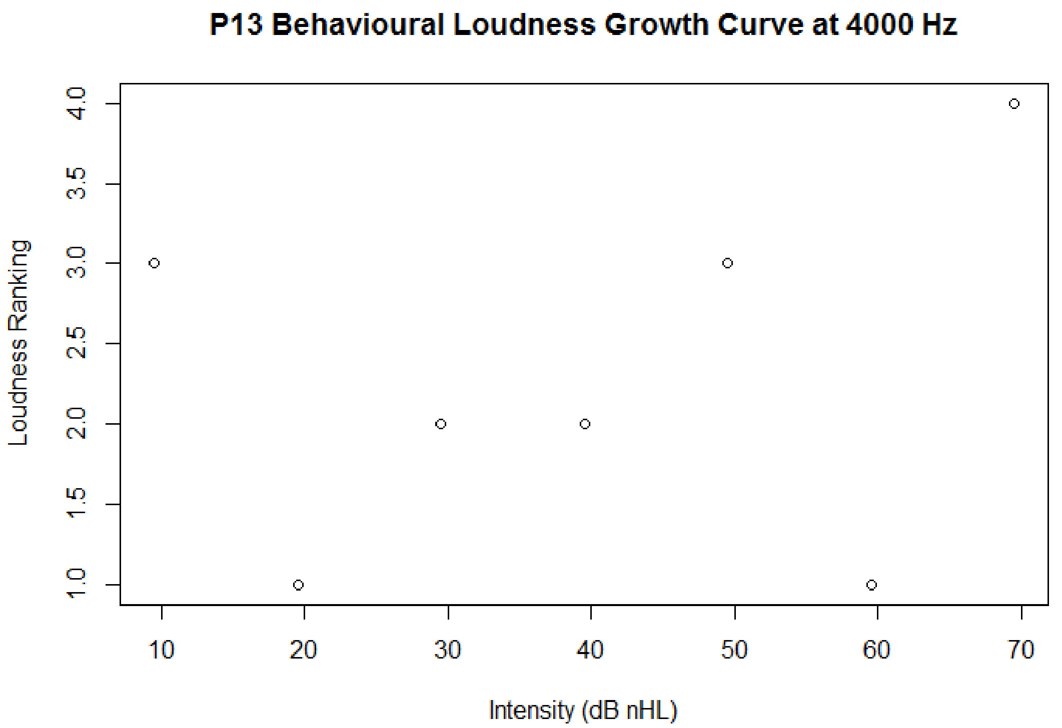

P8 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0.0
0.5
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

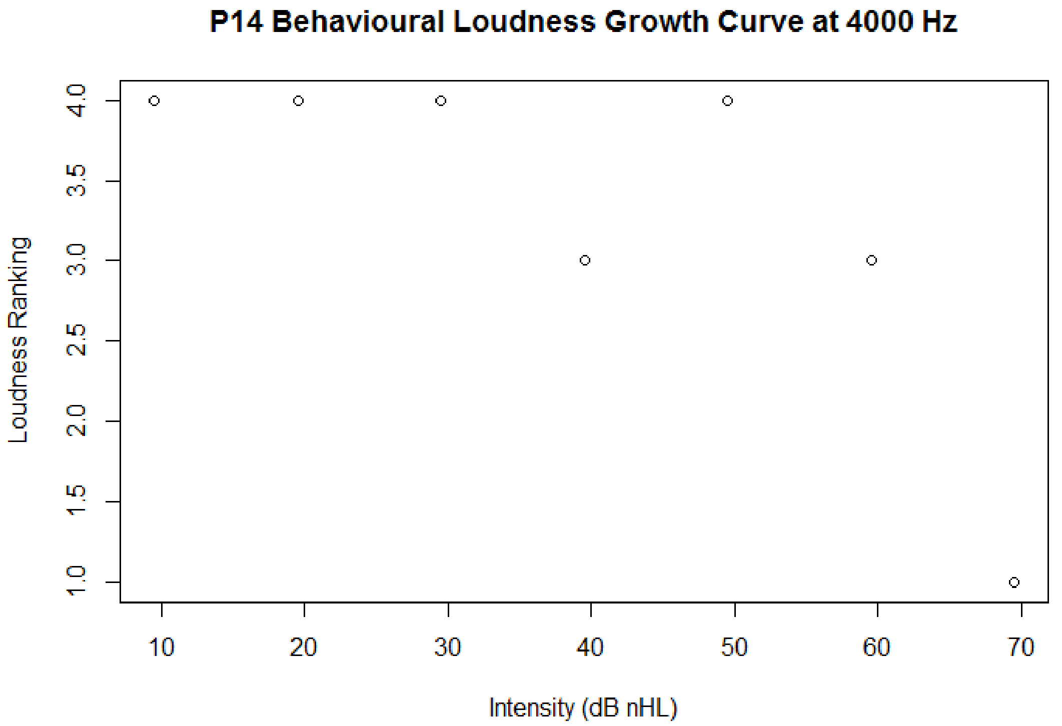

P9 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

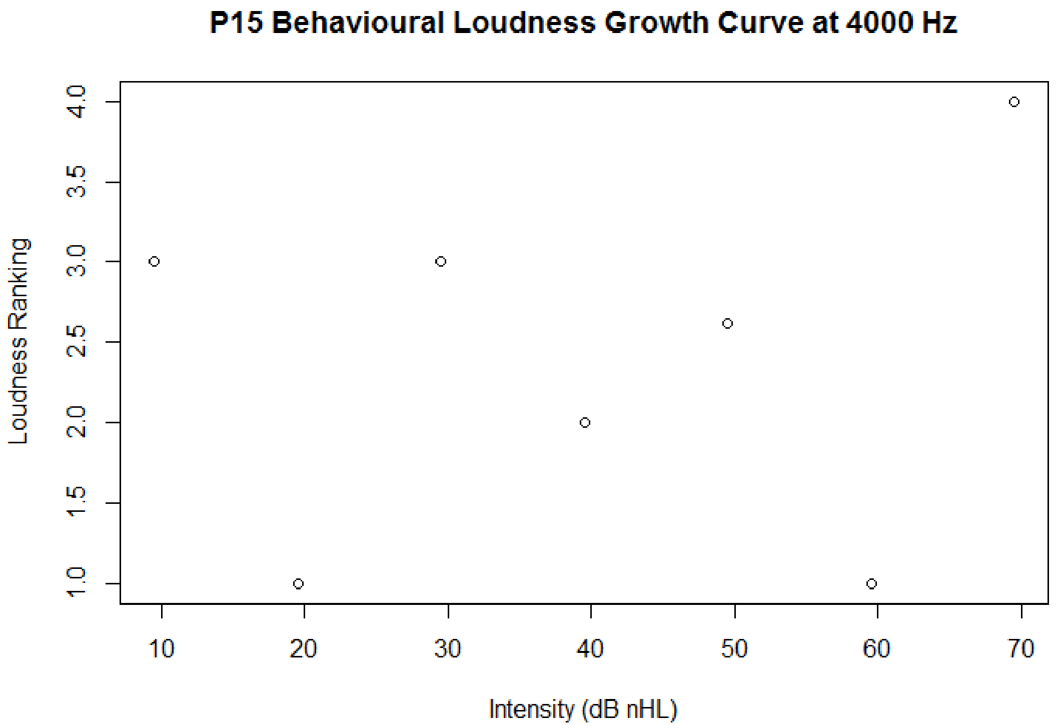

P10 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0
1
2
3
4
10
20
30
40
50
60
70
Intensity (dB nHL)

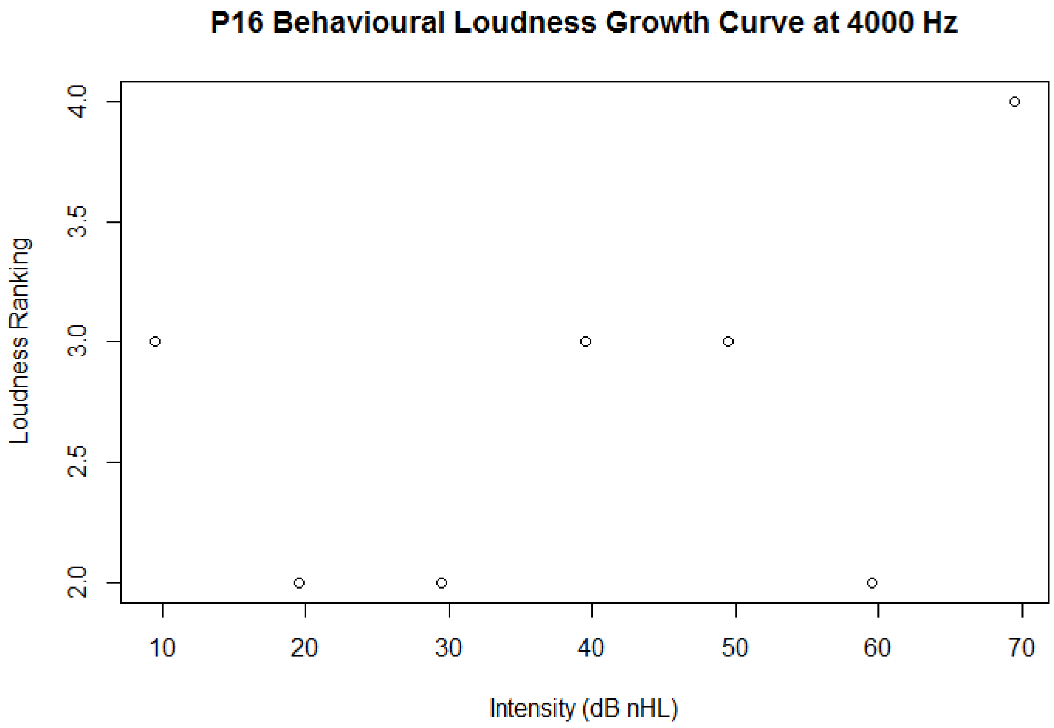

P11 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0
1
2
3
4
5
6
10
20
30
40
50
60
70
Intensity (dB nHL)

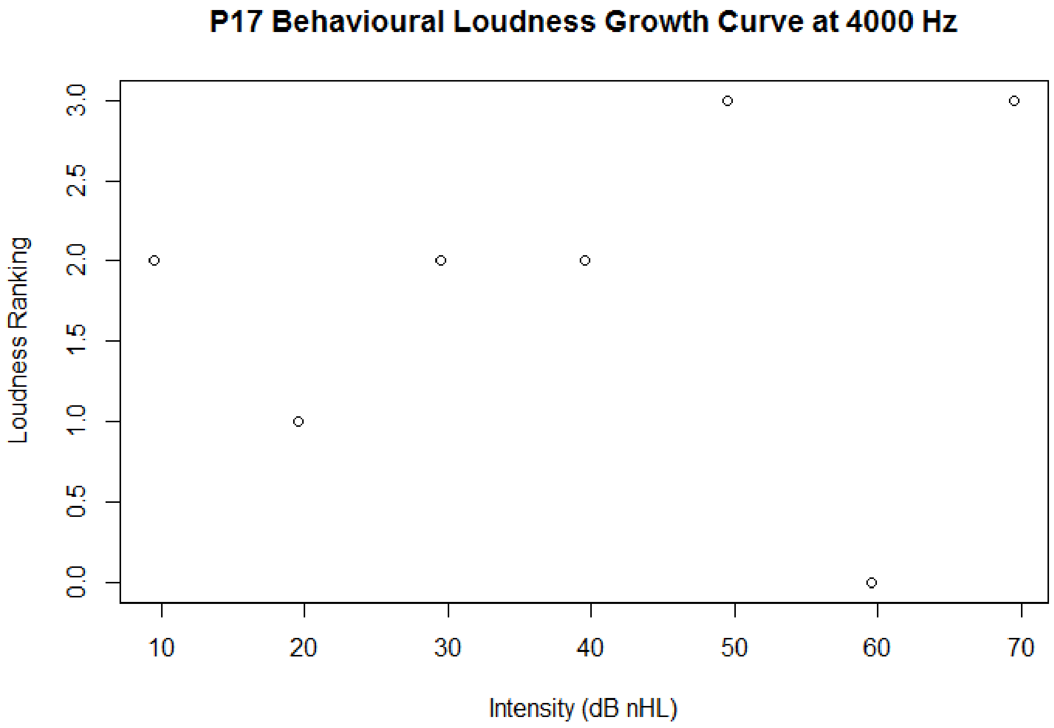

P12 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0
1
2
3
4
10
20
30
40
50
60
70
Intensity (dB nHL)

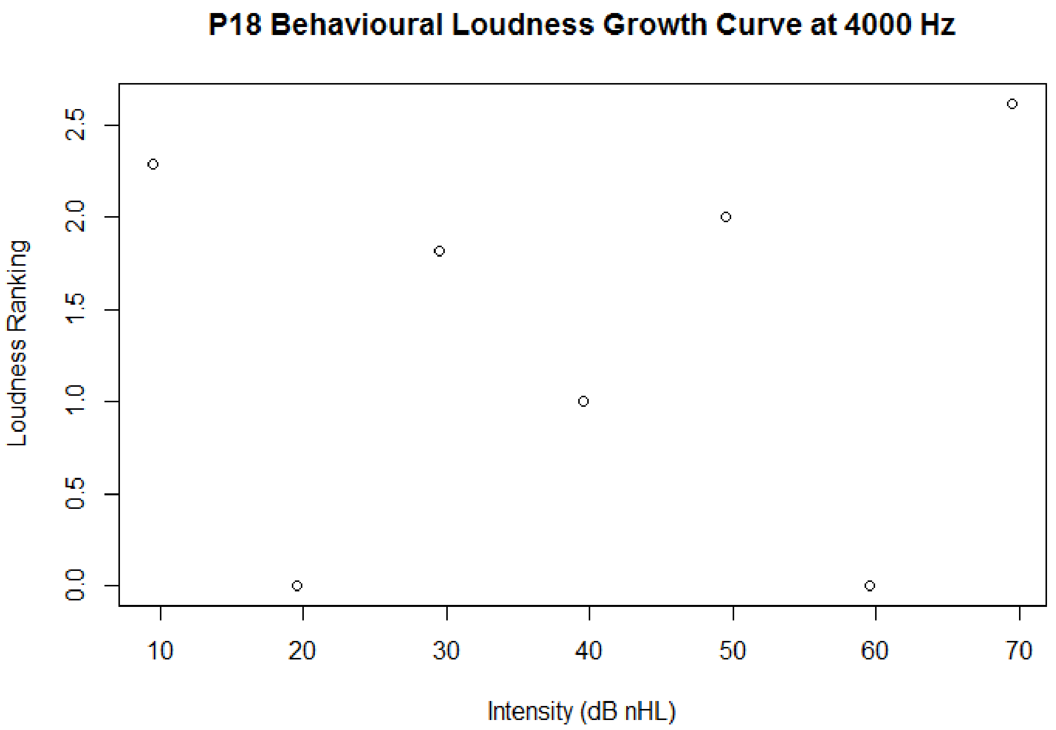

P13 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

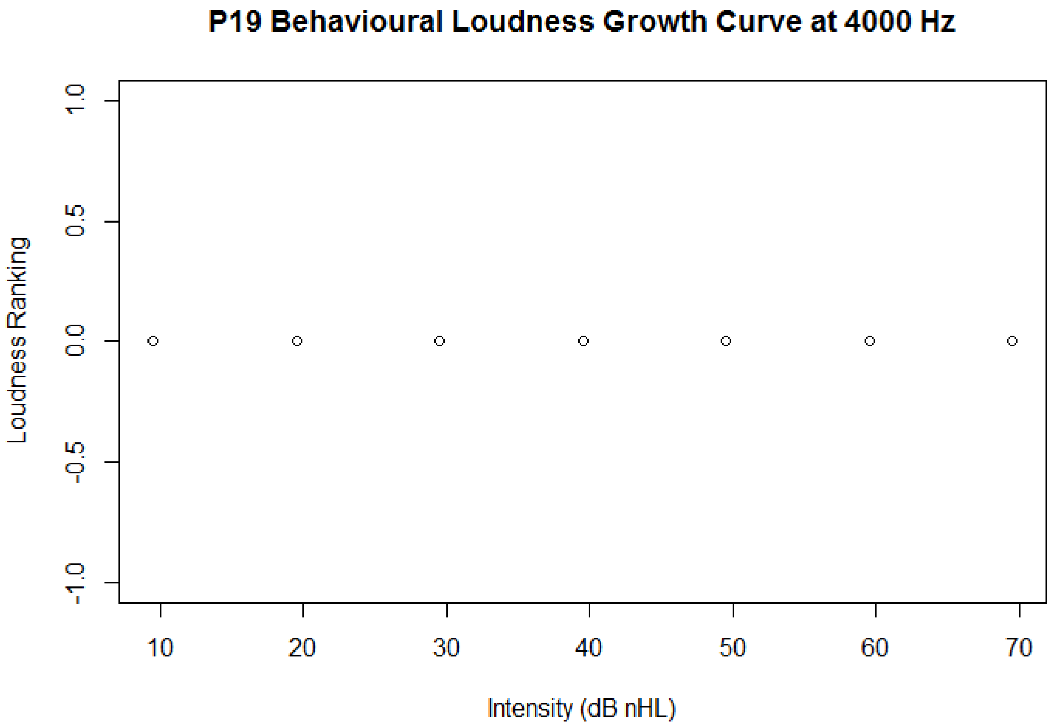

P14 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

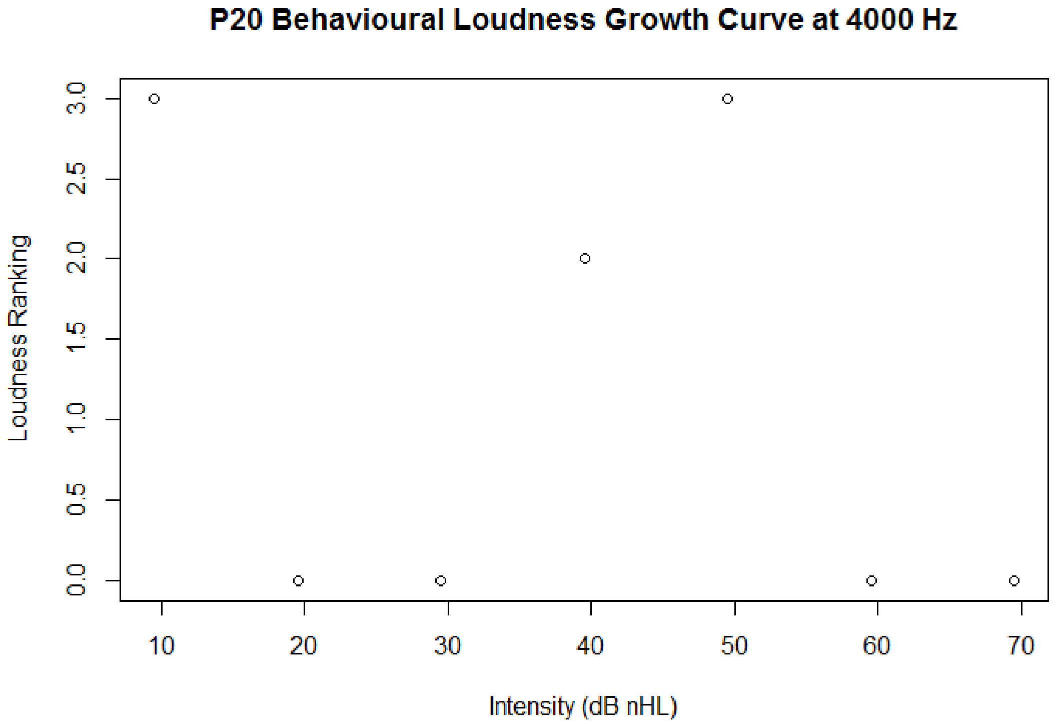

P15 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

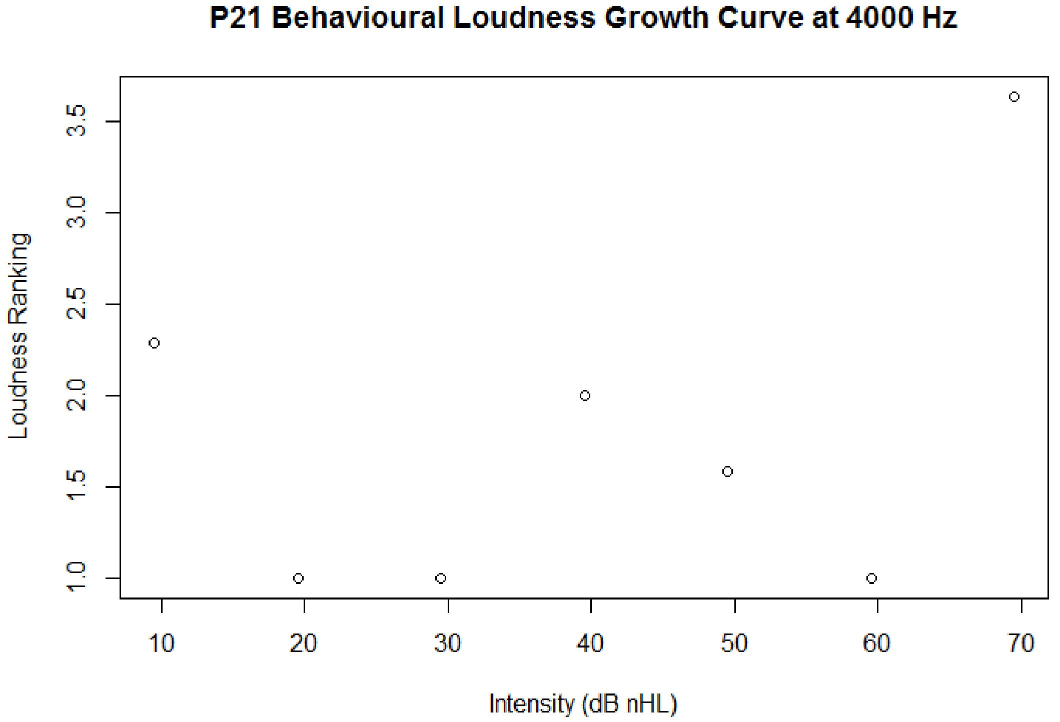

P16 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

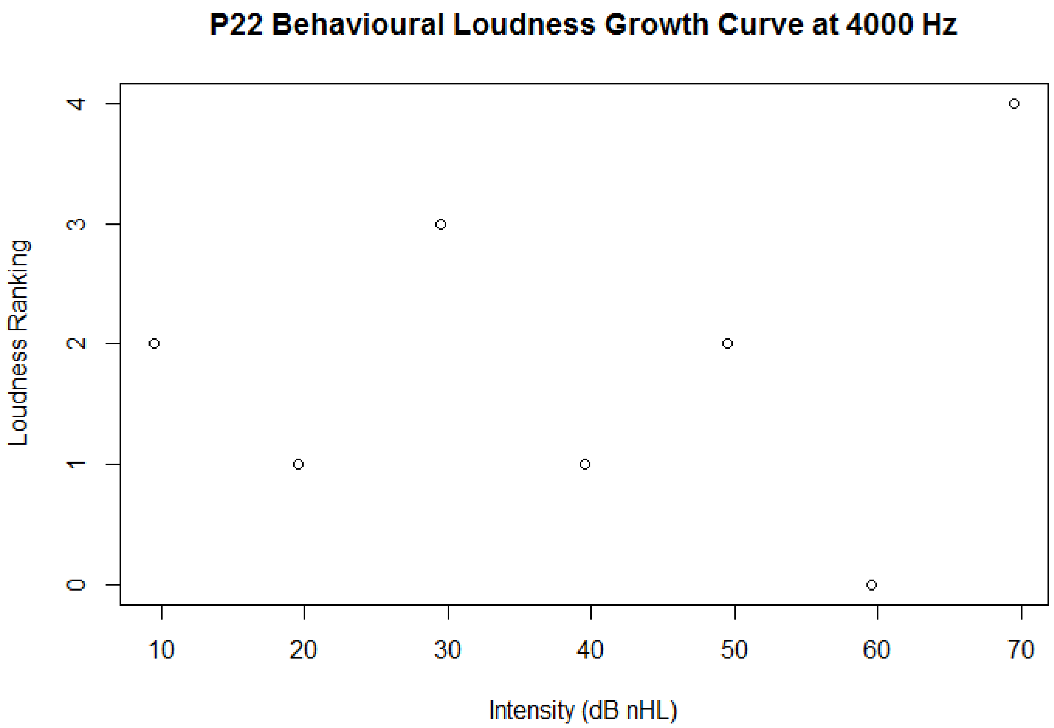

P17 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0.0
0.5
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

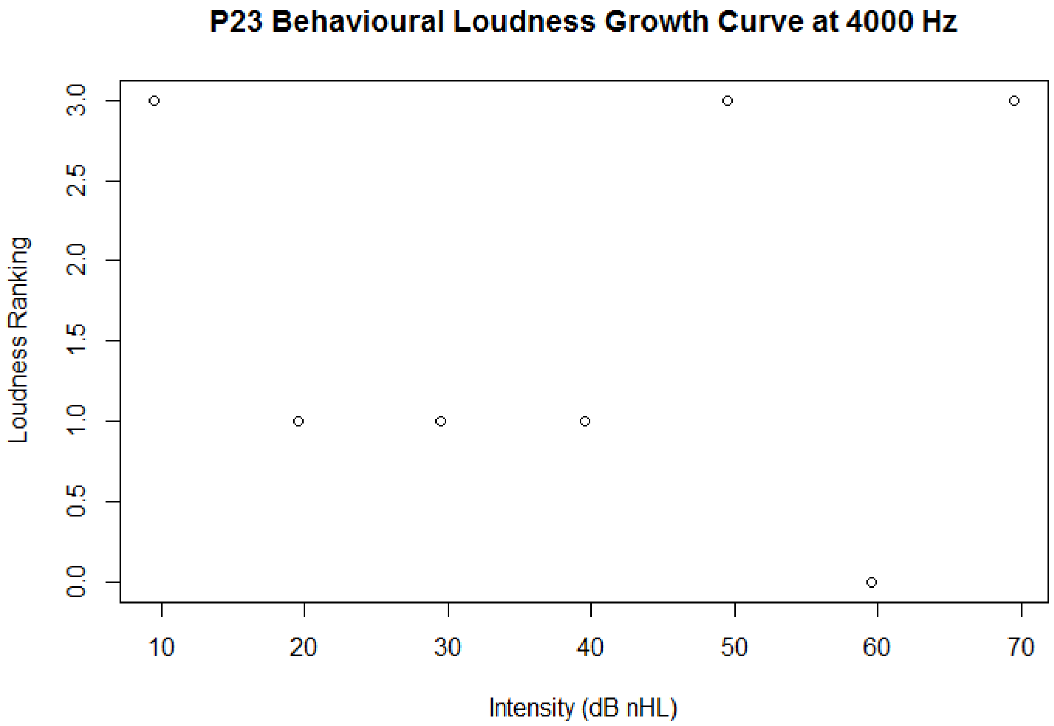

P18 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0.0
0.5
1.0
1.5
2.0
2.5
10
20
30
40
50
60
70
Intensity (dB nHL)

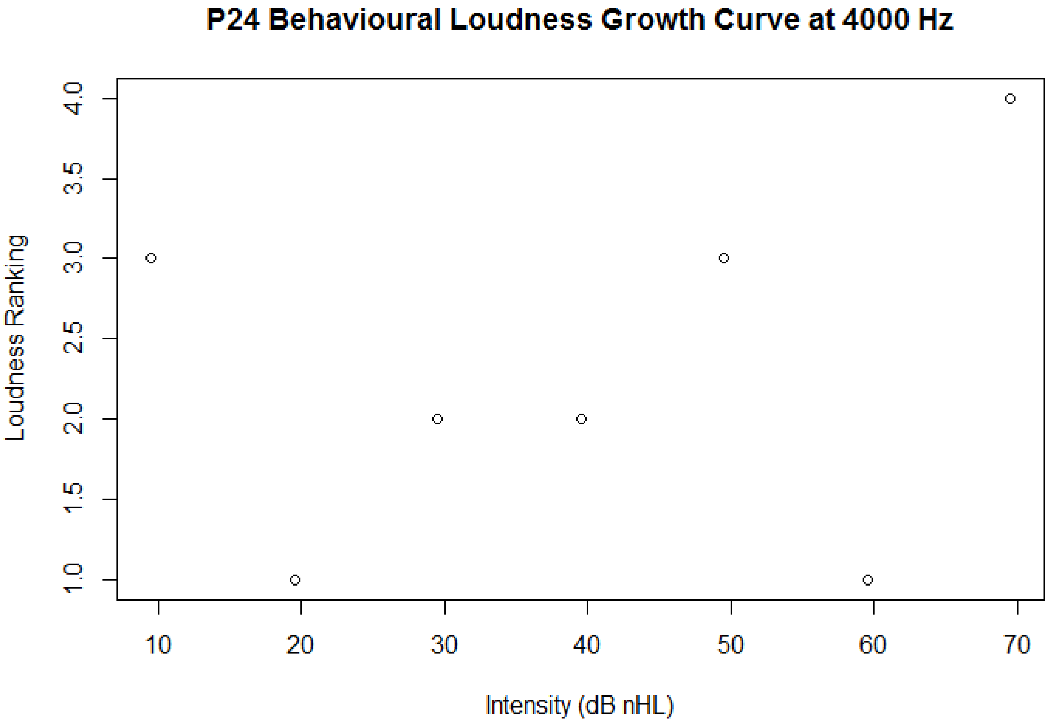

P19 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
-1.0
-0.5
0.0
0.5
1.0
10
20
30
40
50
60
70
Intensity (dB nHL)


P20 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0.0
0.5
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

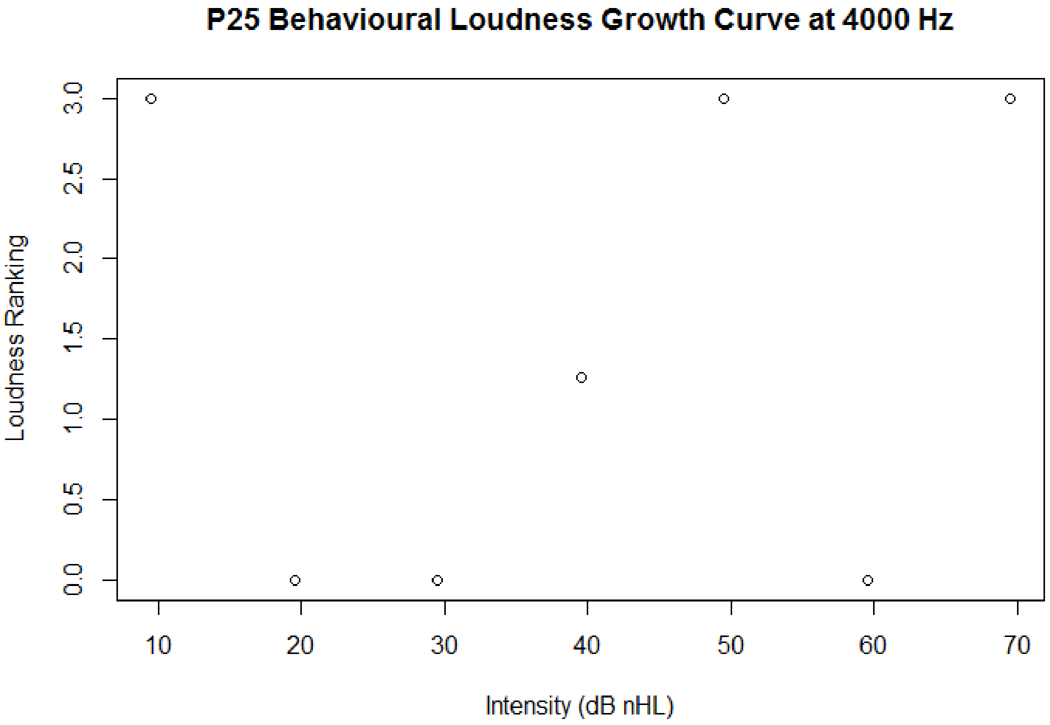

P21 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
10
20
30
40
50
60
70
Intensity (dB nHL)


P22 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0
1
2
3
4
10
20
30
40
50
60
70
Intensity (dB nHL)

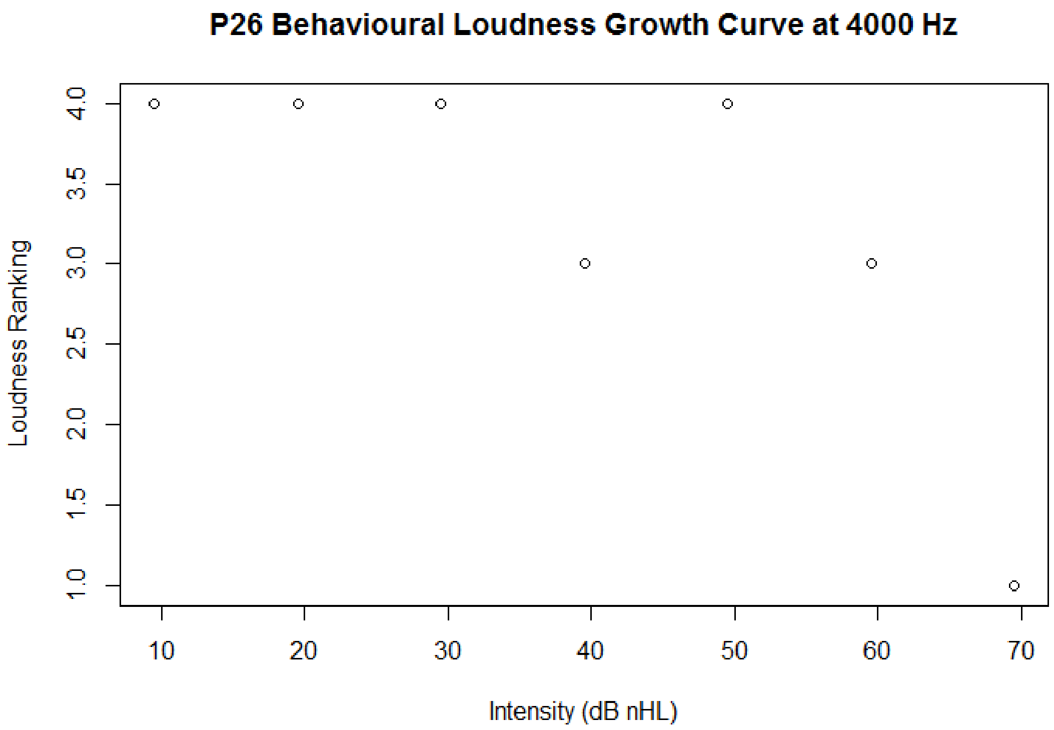

P23 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0.0
0.5
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

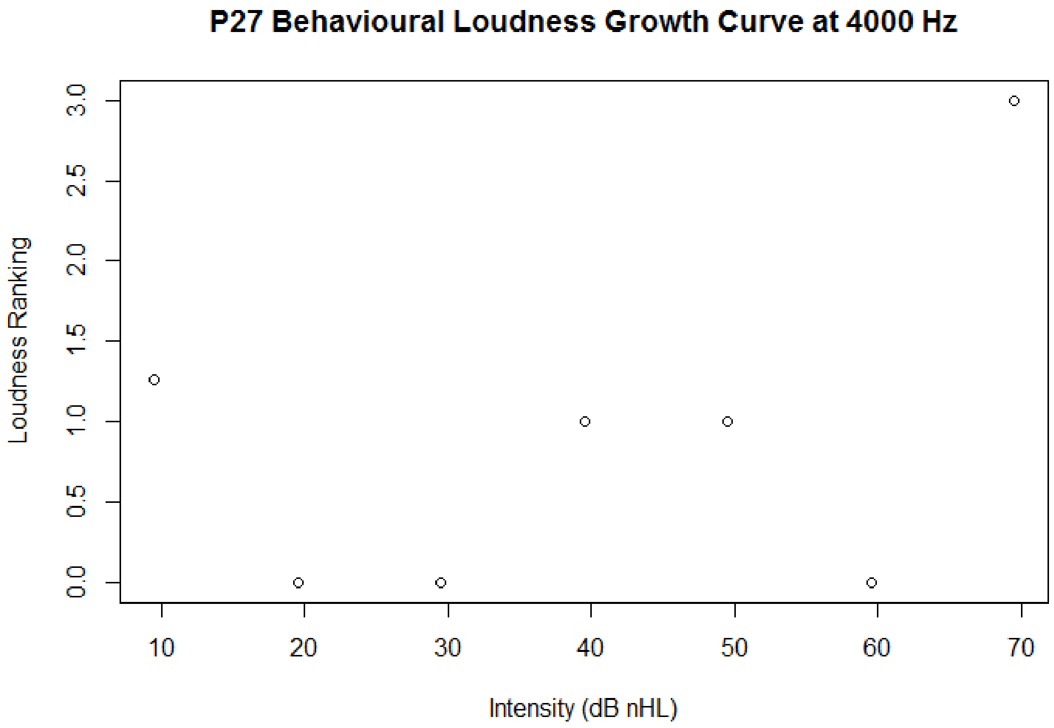


P24 Behavioural Loudness Growth Curve at 4000 Hz

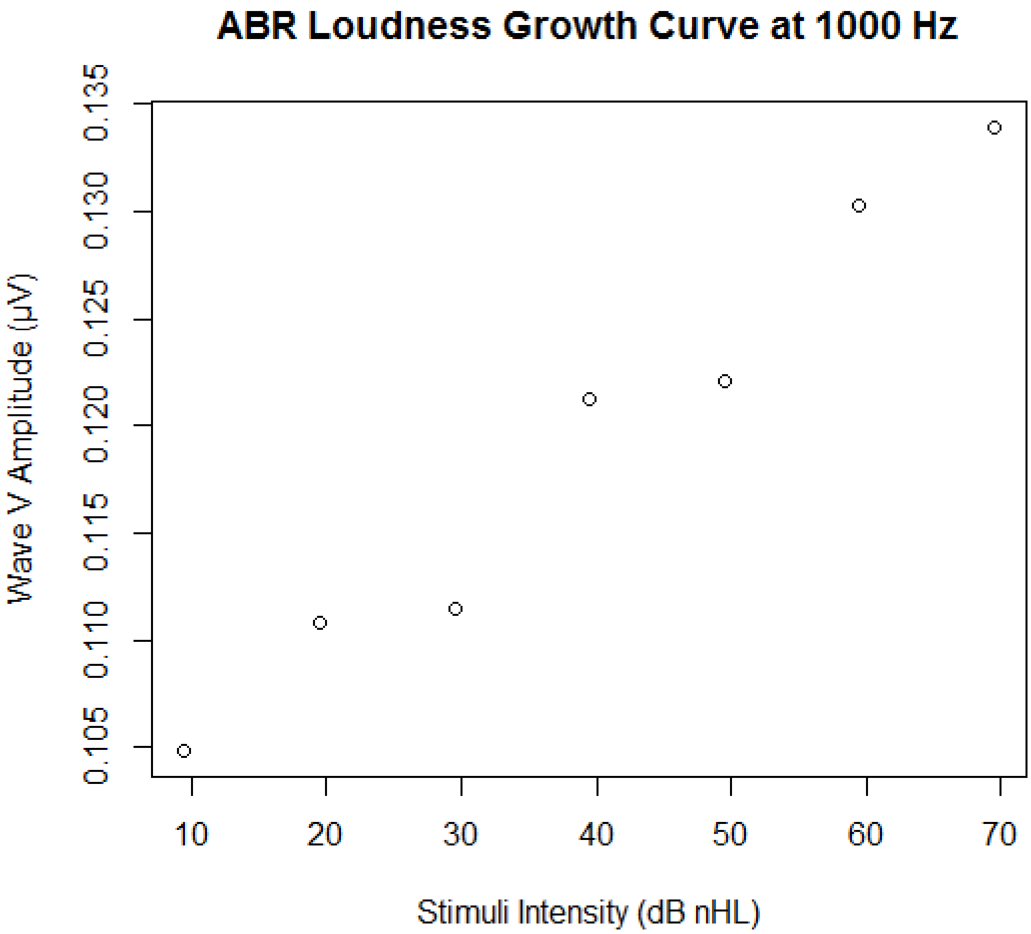


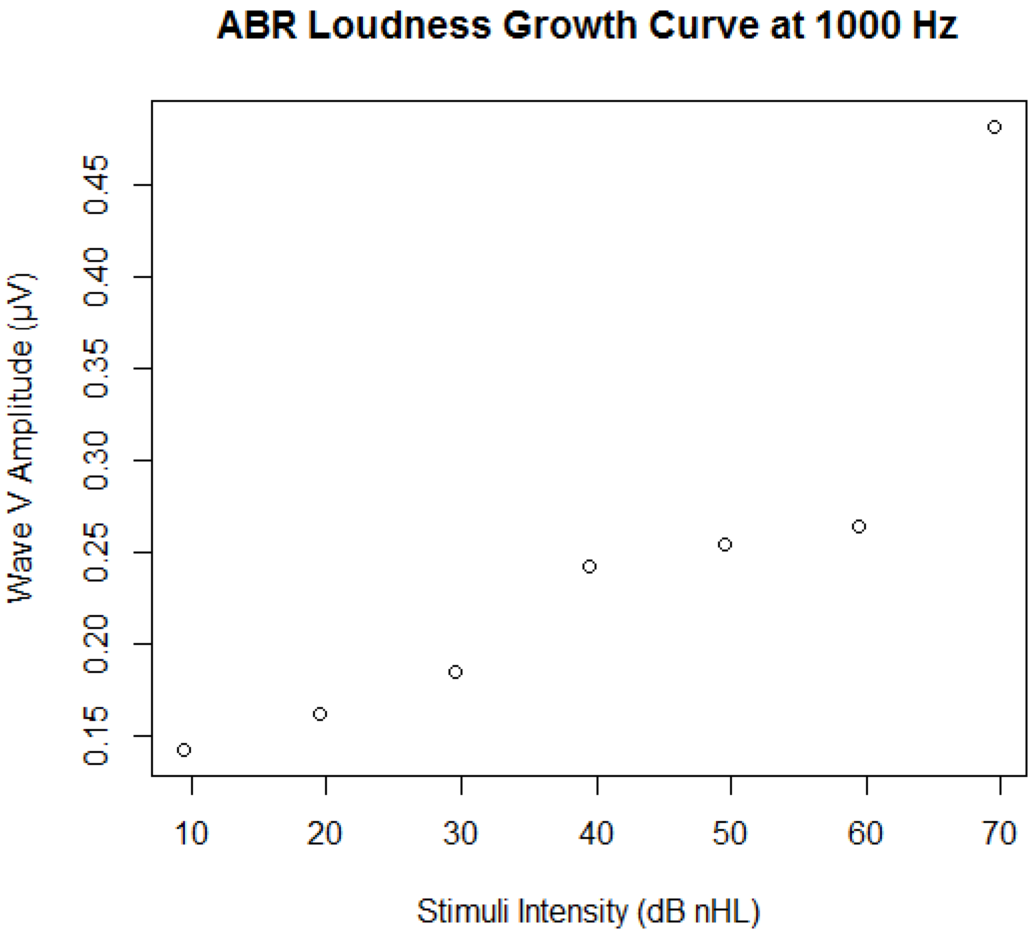
P25 Behavioural Loudness Growth Curve at 4000 Hz

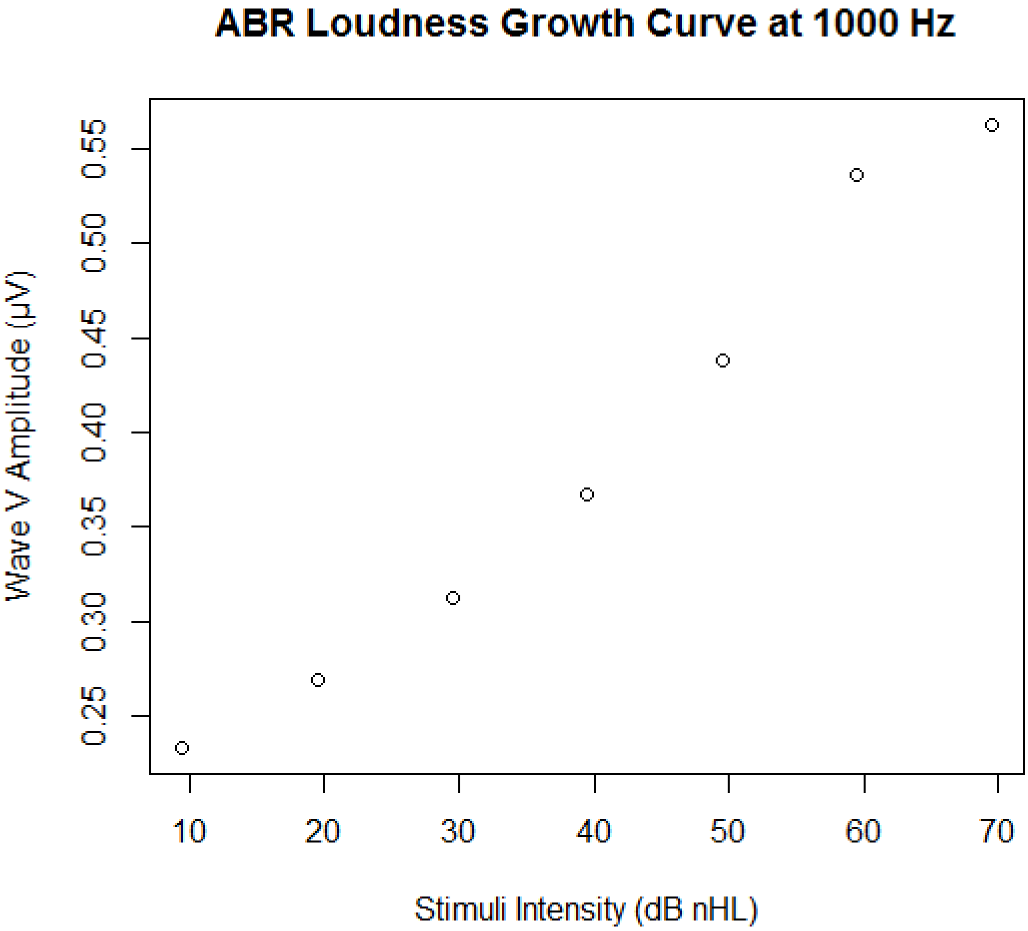

P26 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
1.0
1.5
2.0
2.5
3.0
3.5
4.0
10
20
30
40
50
60
70
Intensity (dB nHL)

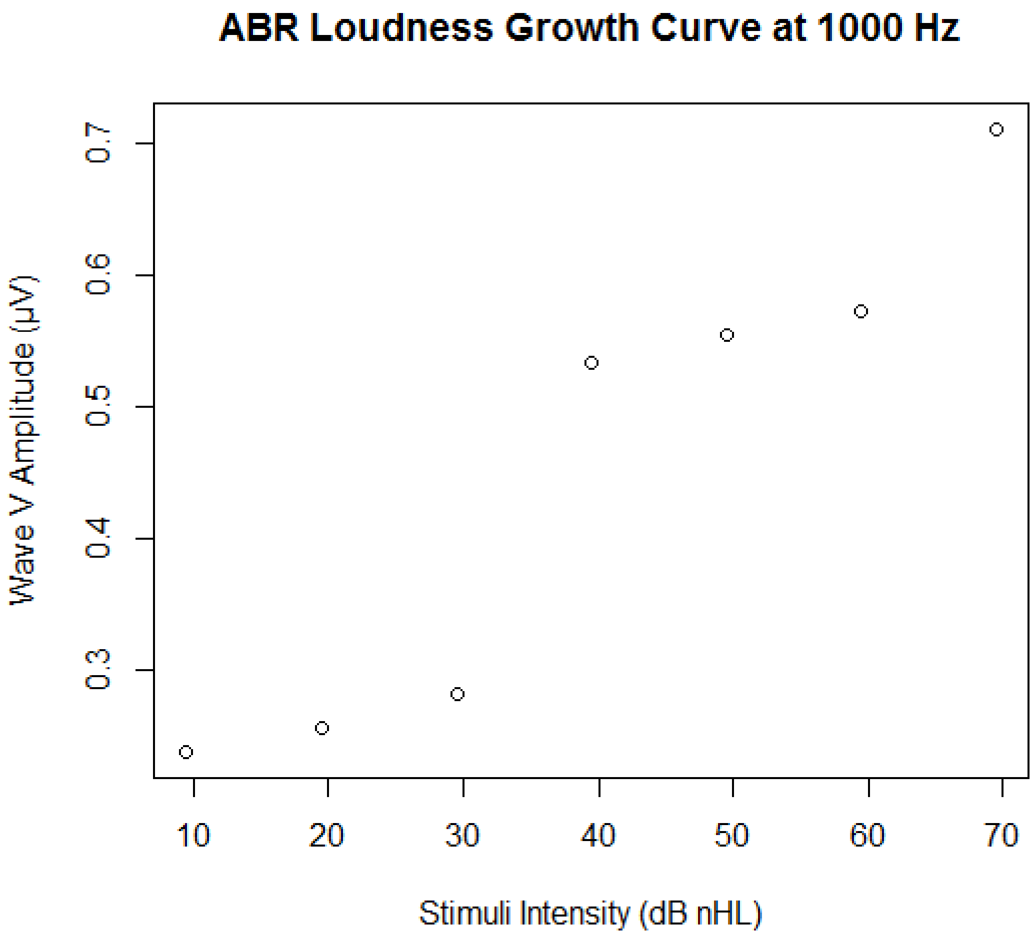

P27 Behavioural Loudness Growth Curve at 4000 Hz
Loudness Ranking
0.0
0.5
1.0
1.5
2.0
2.5
3.0
10
20
30
40
50
60
70
Intensity (dB nHL)

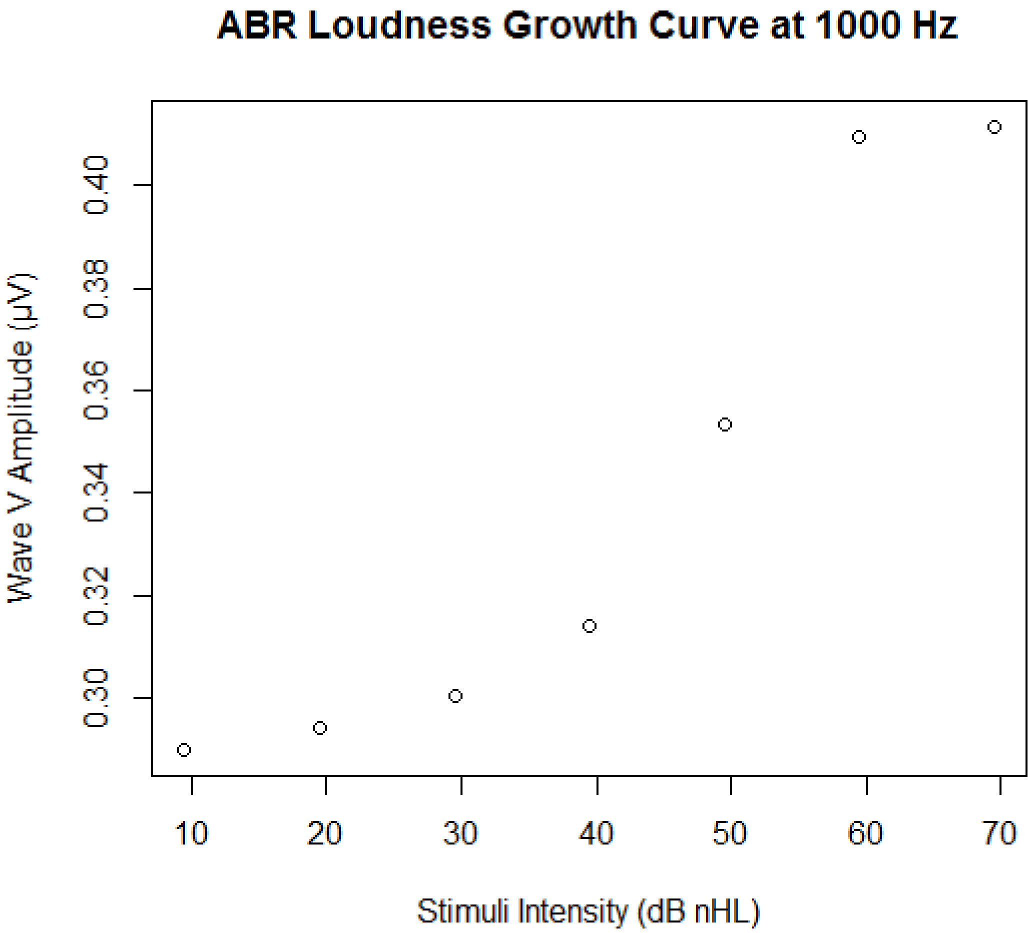
Individual ABR Loudness Growth Curves at 1000 Hz

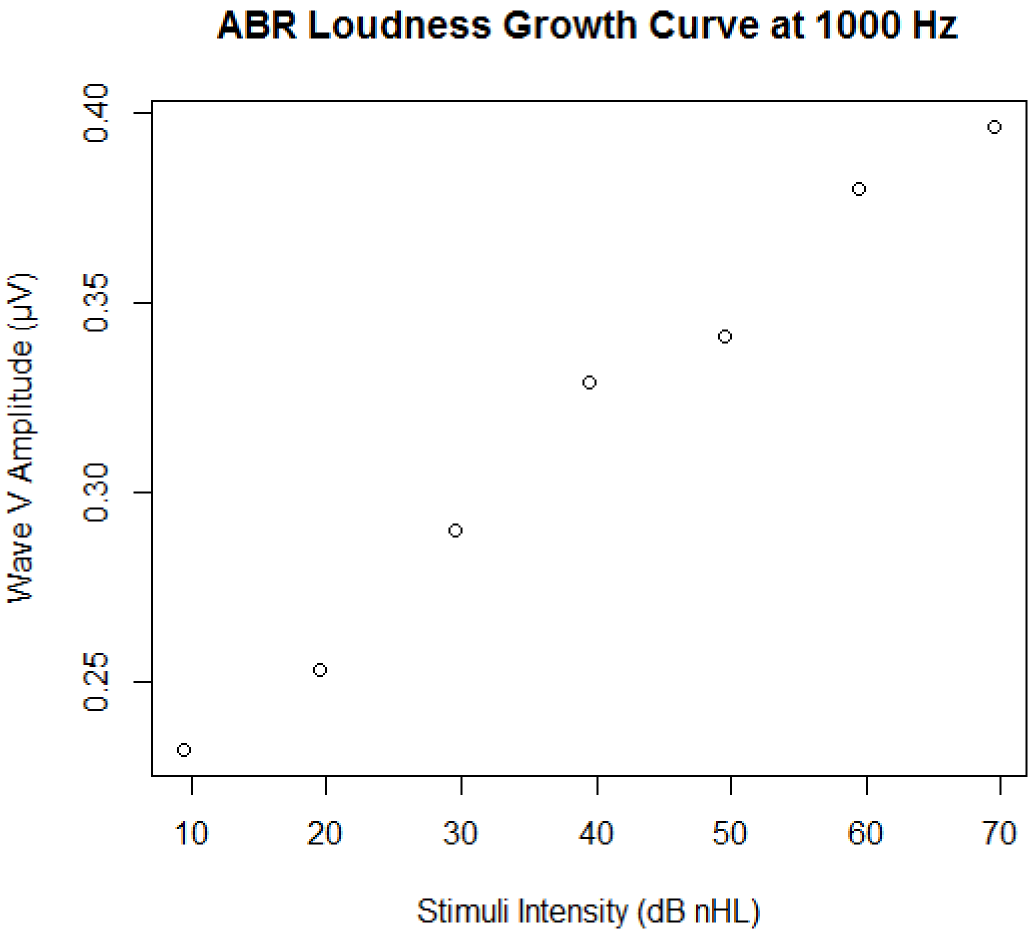


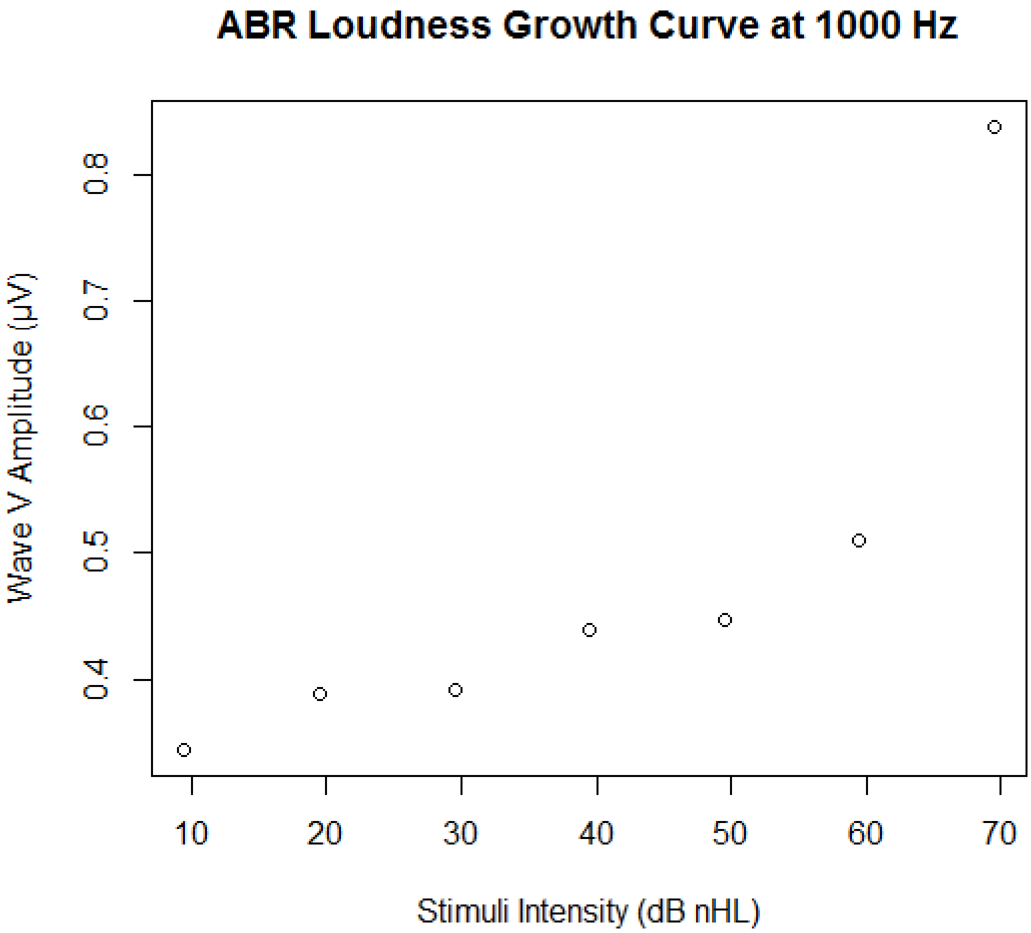

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.15
0.20
0.25
0.30
0.35
0.40
0.45
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

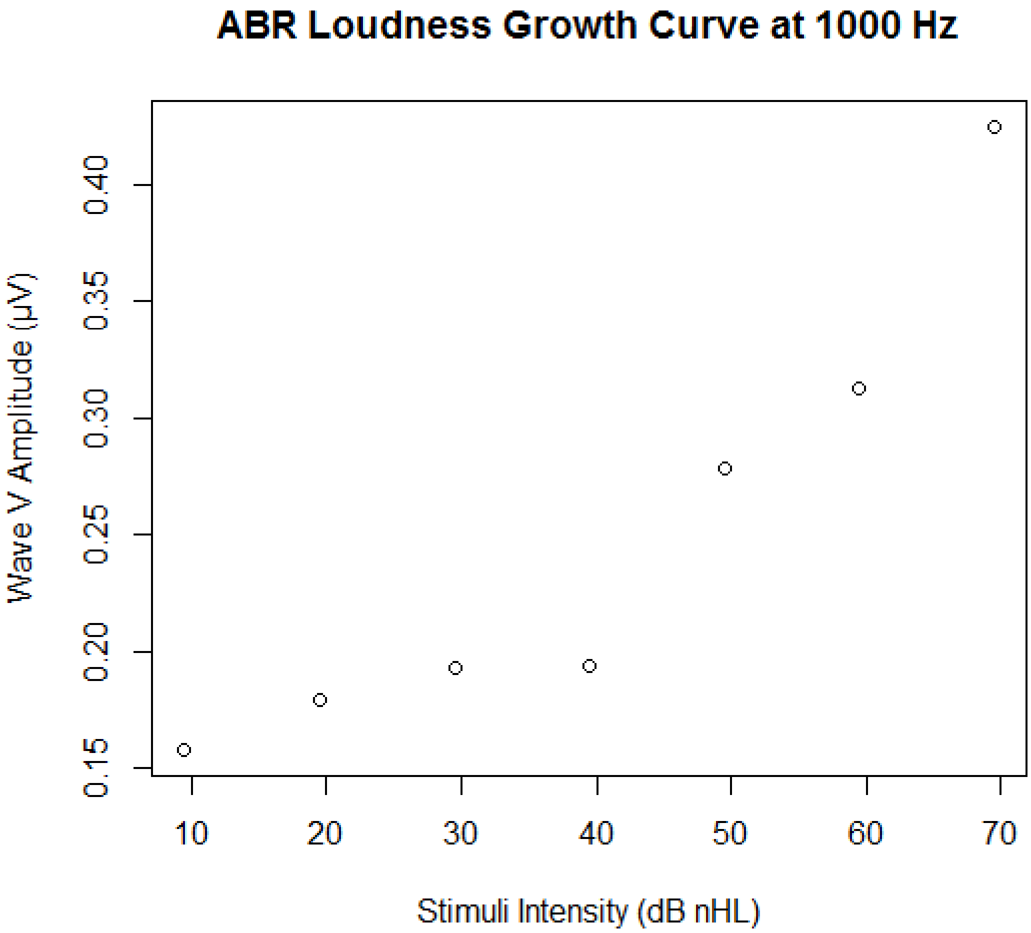

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
0.45
0.50
0.55
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

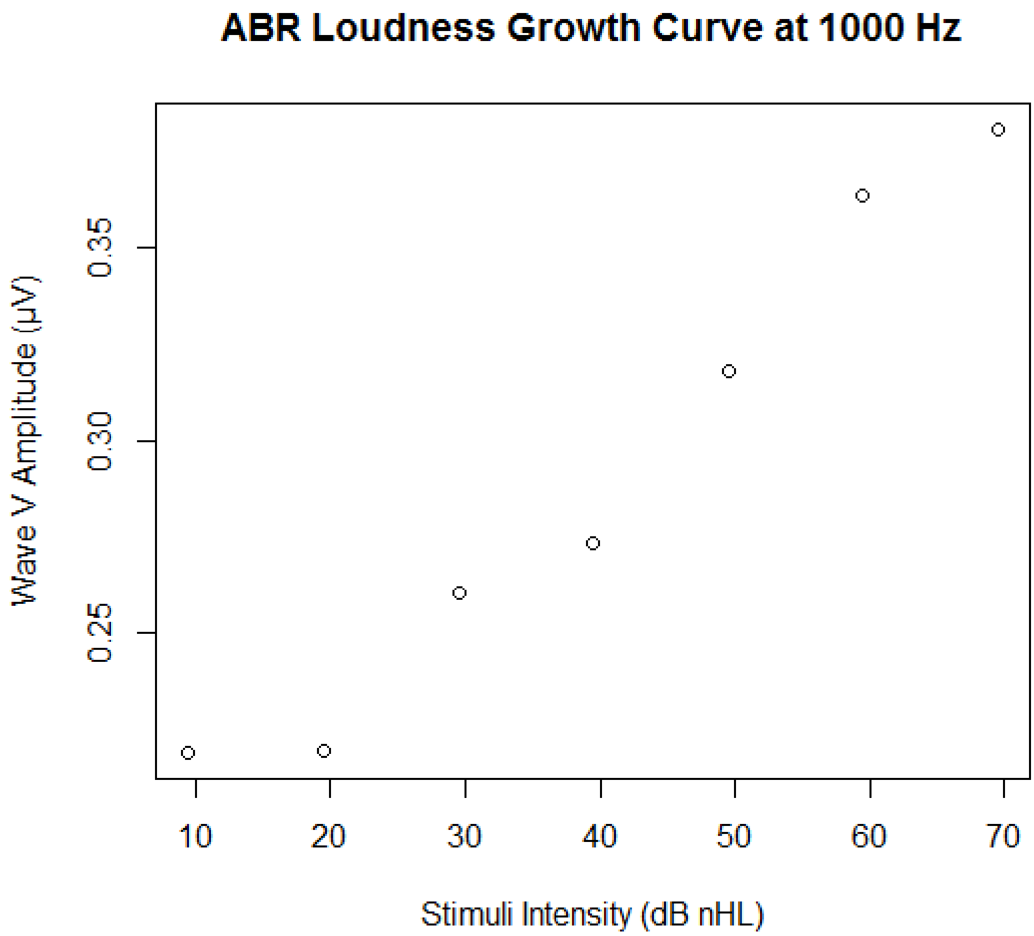

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.3
0.4
0.5
0.6
0.7
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

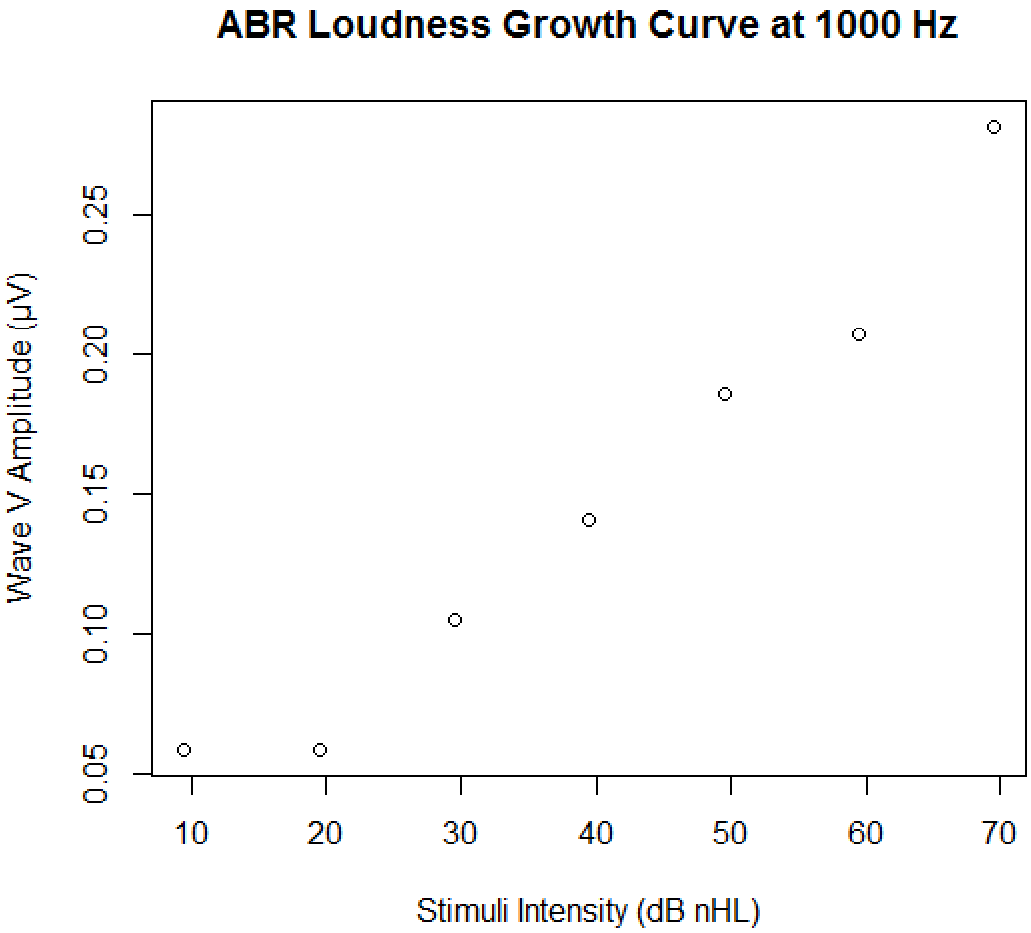

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.30
0.32
0.34
0.36
0.38
0.40
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

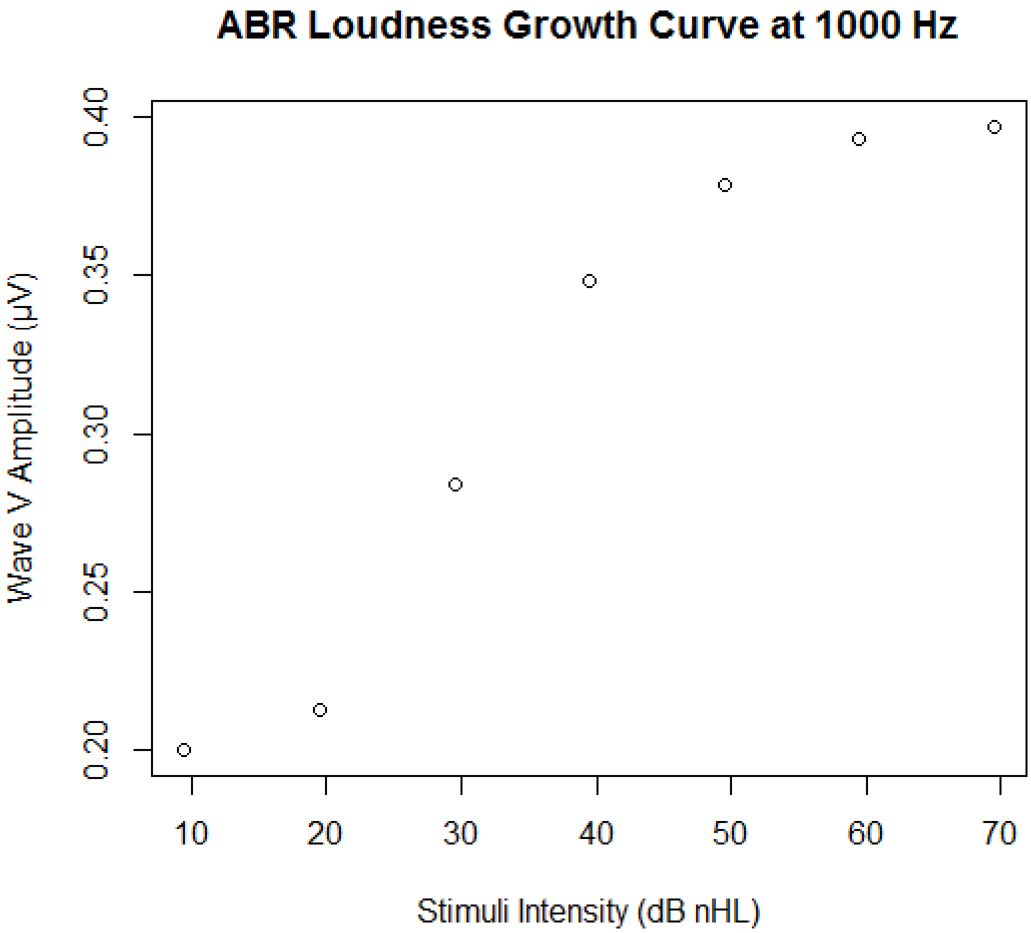

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

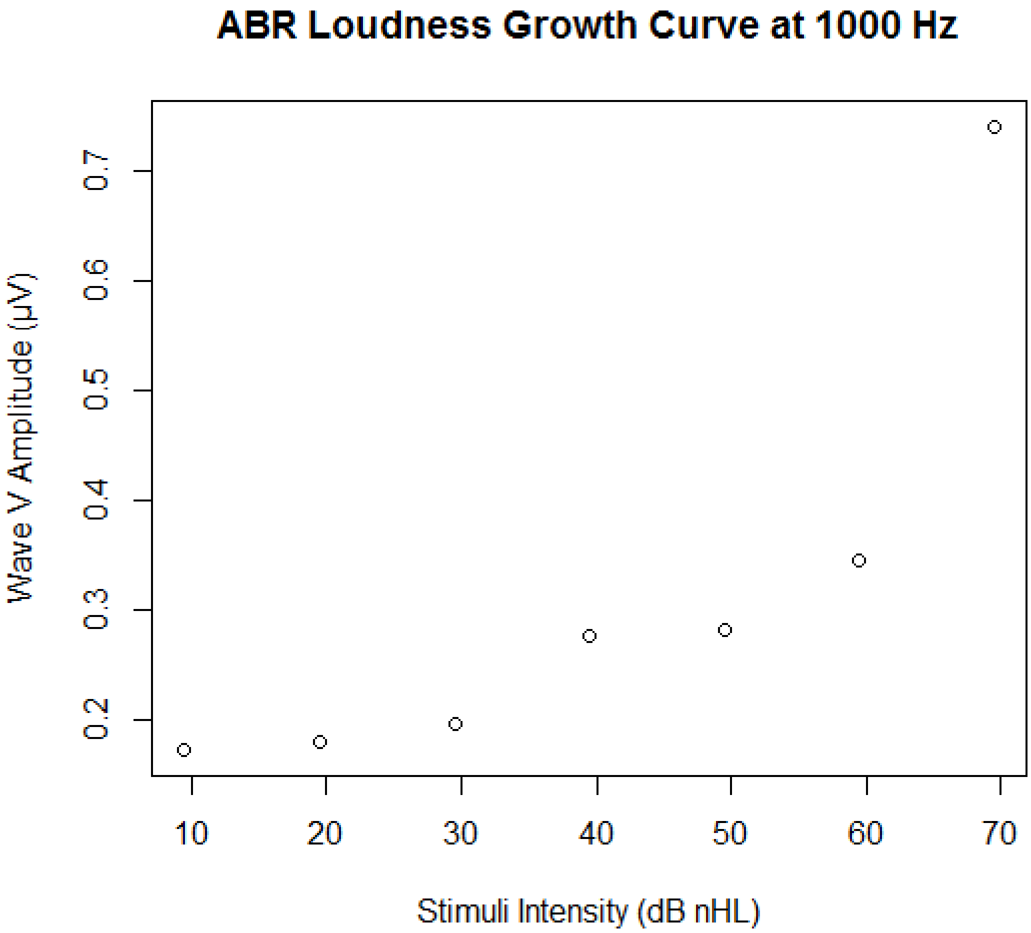

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (μV)
0.4
0.5
0.6
0.7
0.8
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

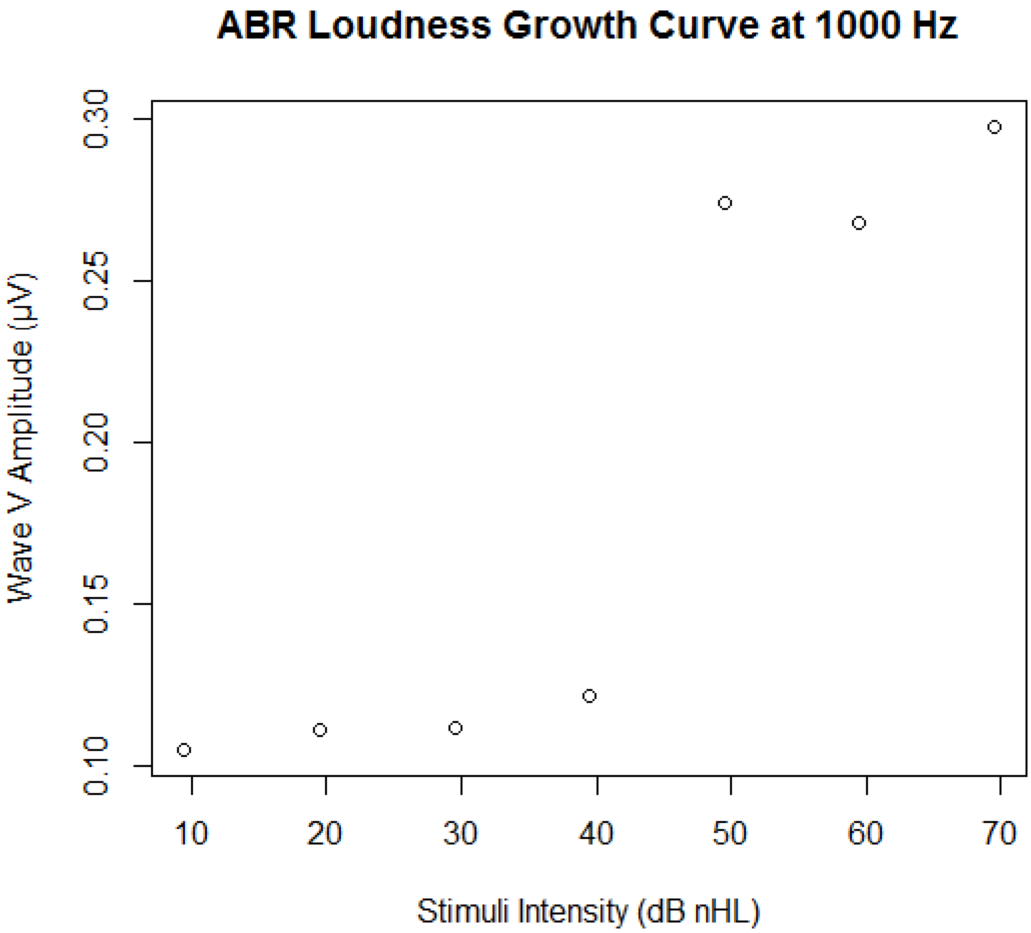

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (μV)
0.15
0.20
0.25
0.30
0.35
0.40
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

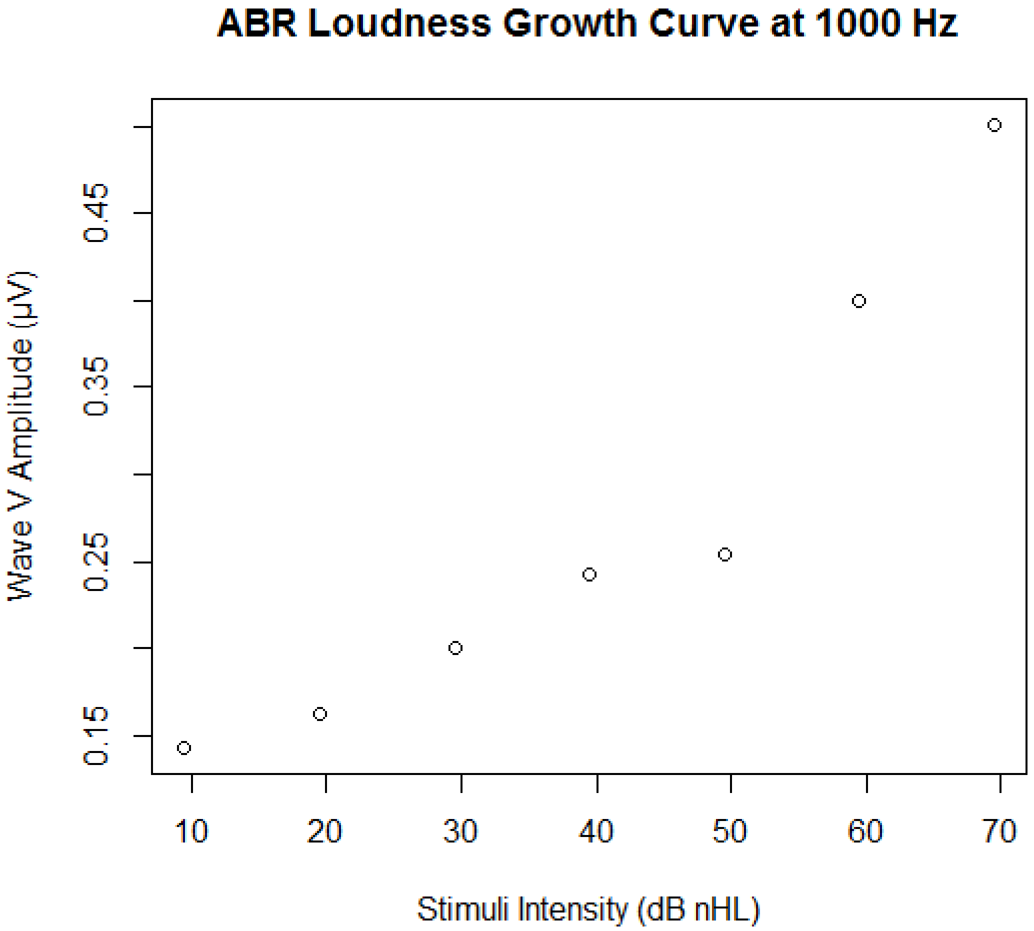

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (μV)
0.25
0.30
0.35
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

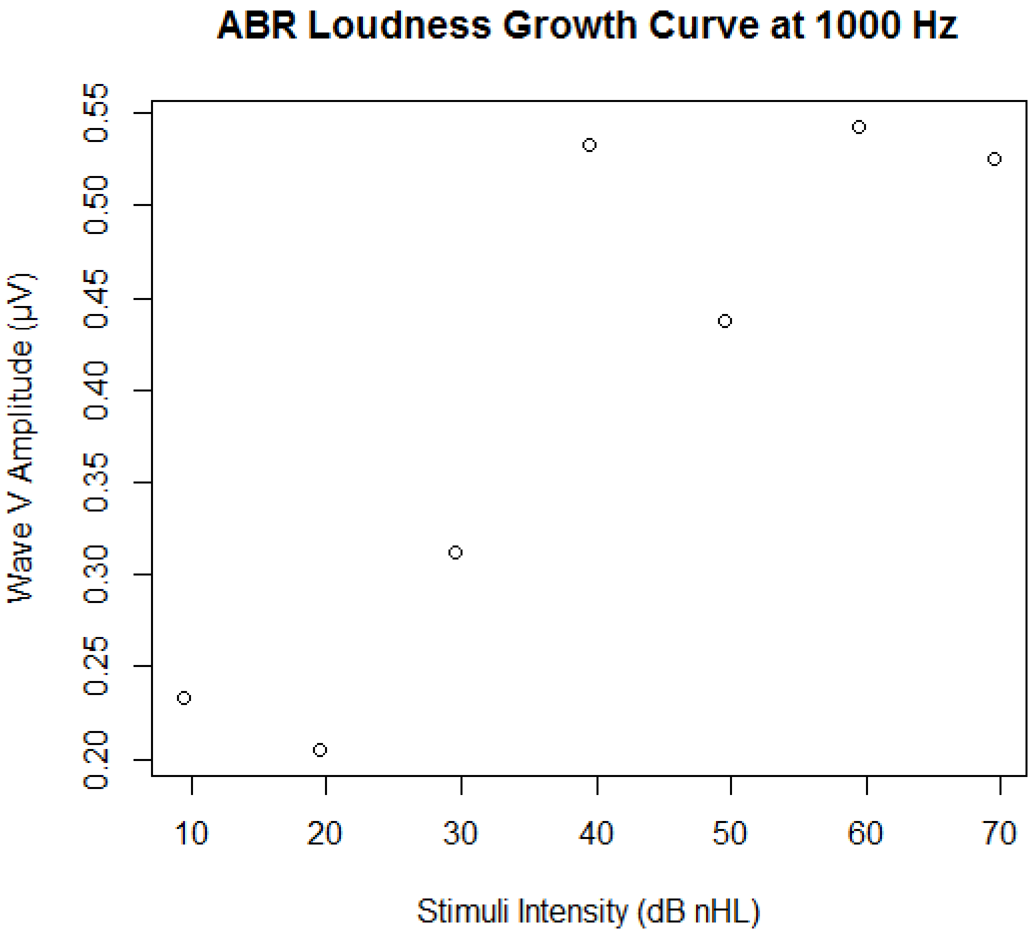

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (μV)
0.05
0.10
0.15
0.20
0.25
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

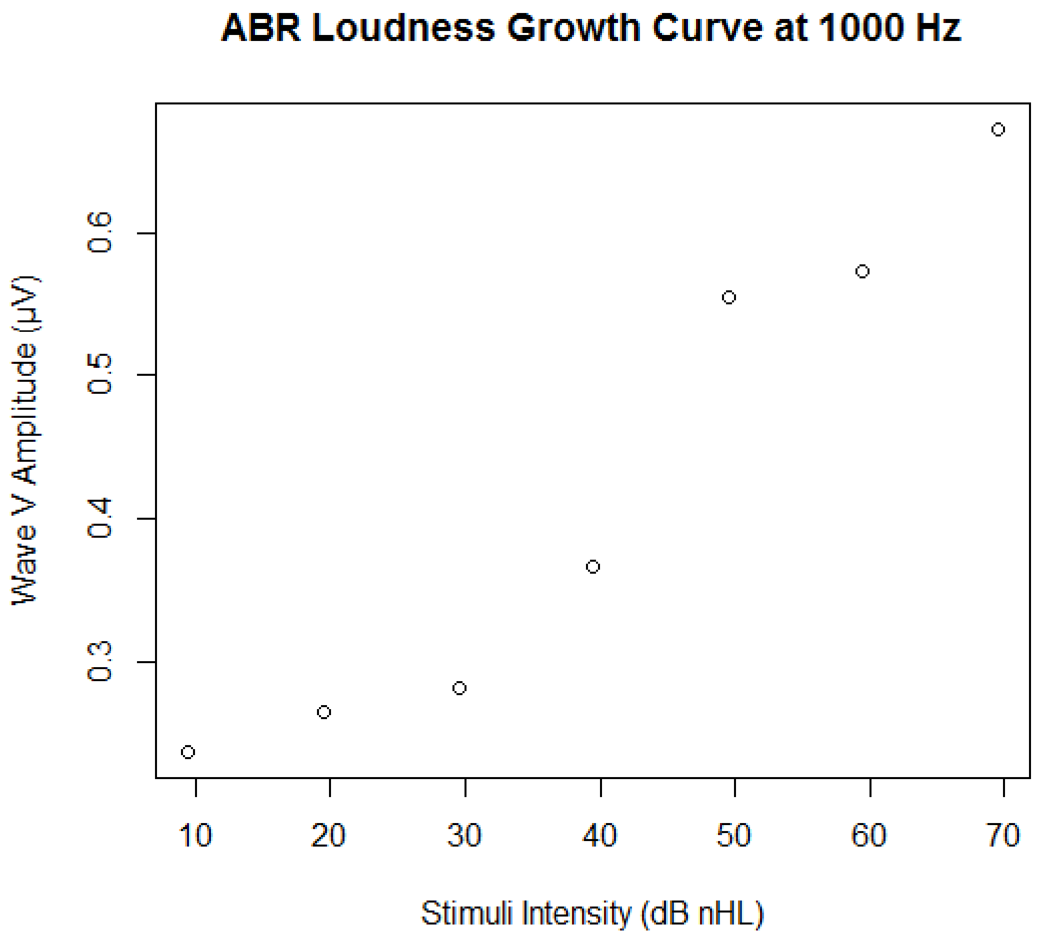

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.20
0.25
0.30
0.35
0.40
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

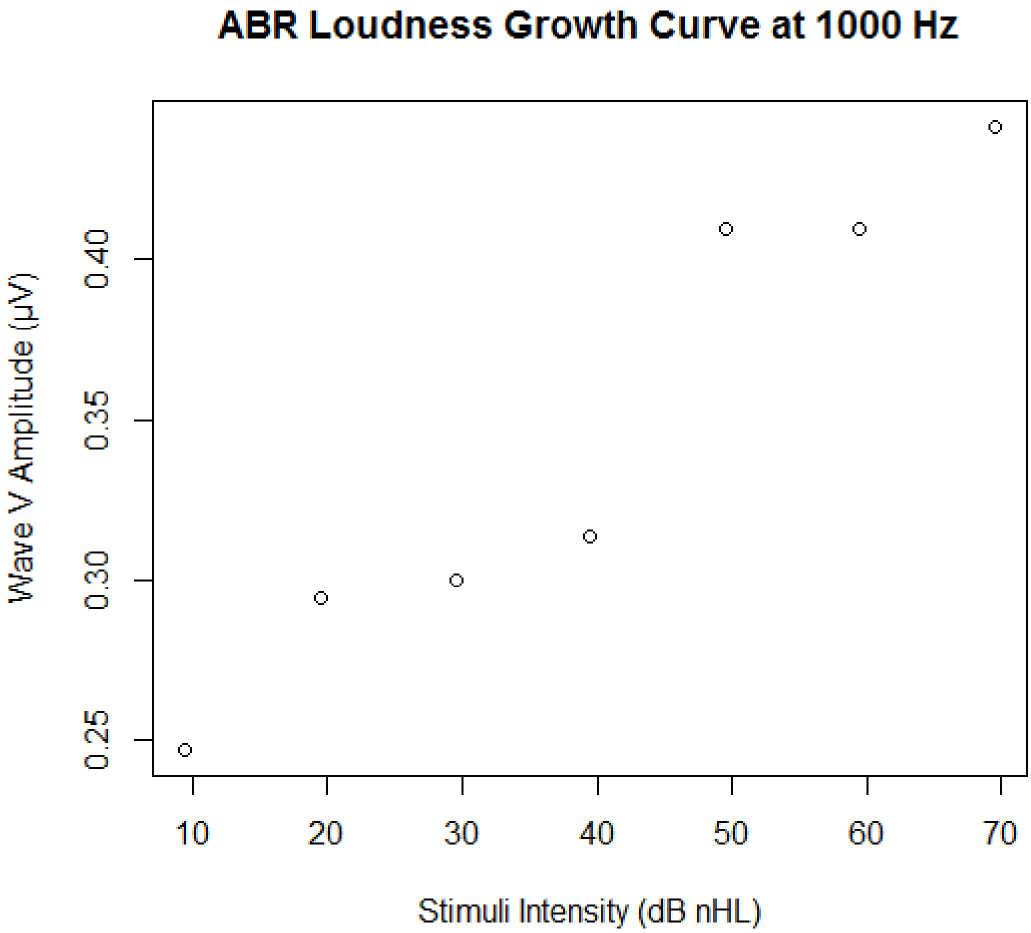

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.2
0.3
0.4
0.5
0.6
0.7
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.10
0.15
0.20
0.25
0.30
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

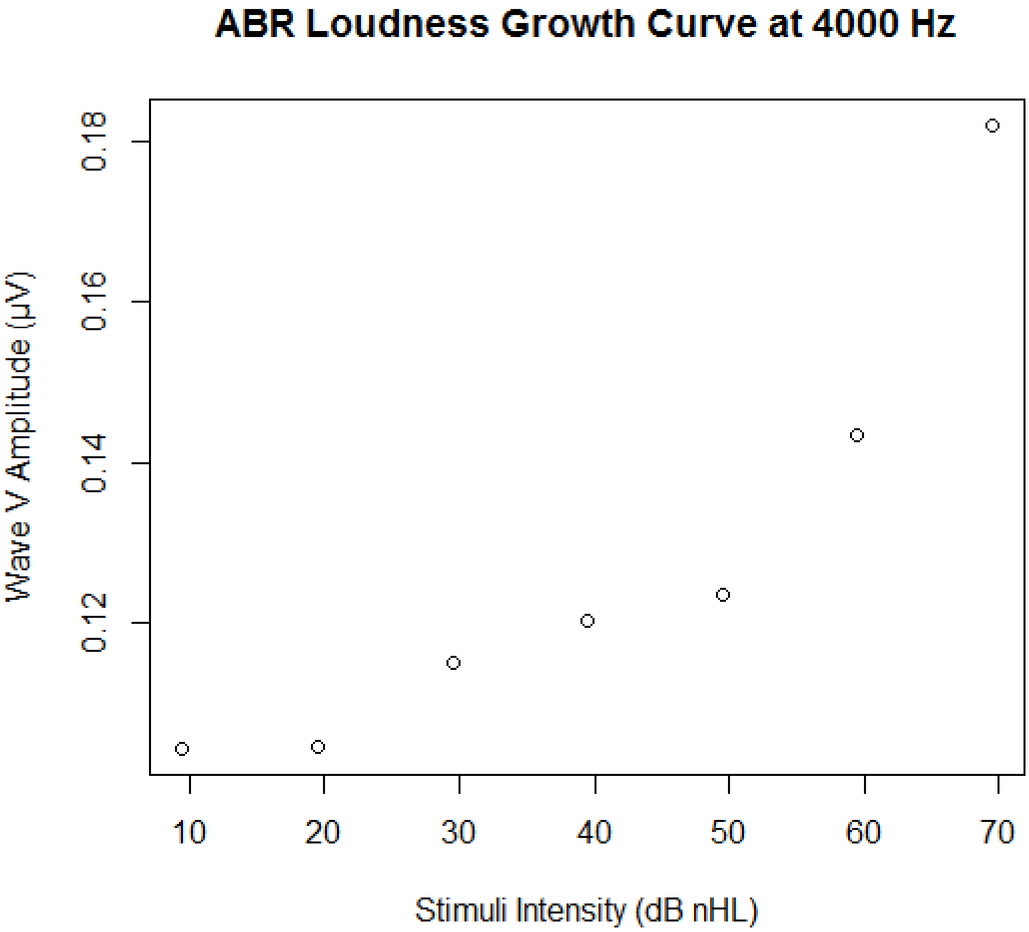

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.15
0.25
0.35
0.45
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

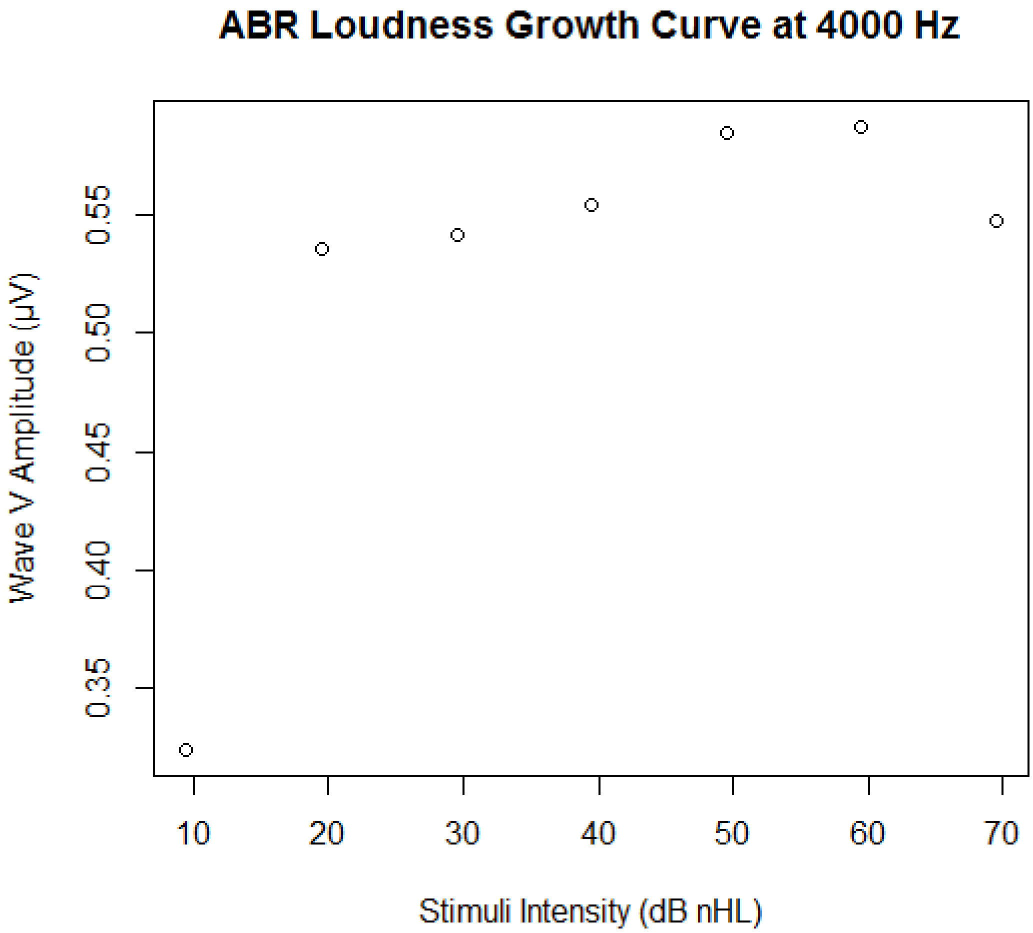

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (μV)
0.20
0.25
0.30
0.35
0.40
0.45
0.50
0.55
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

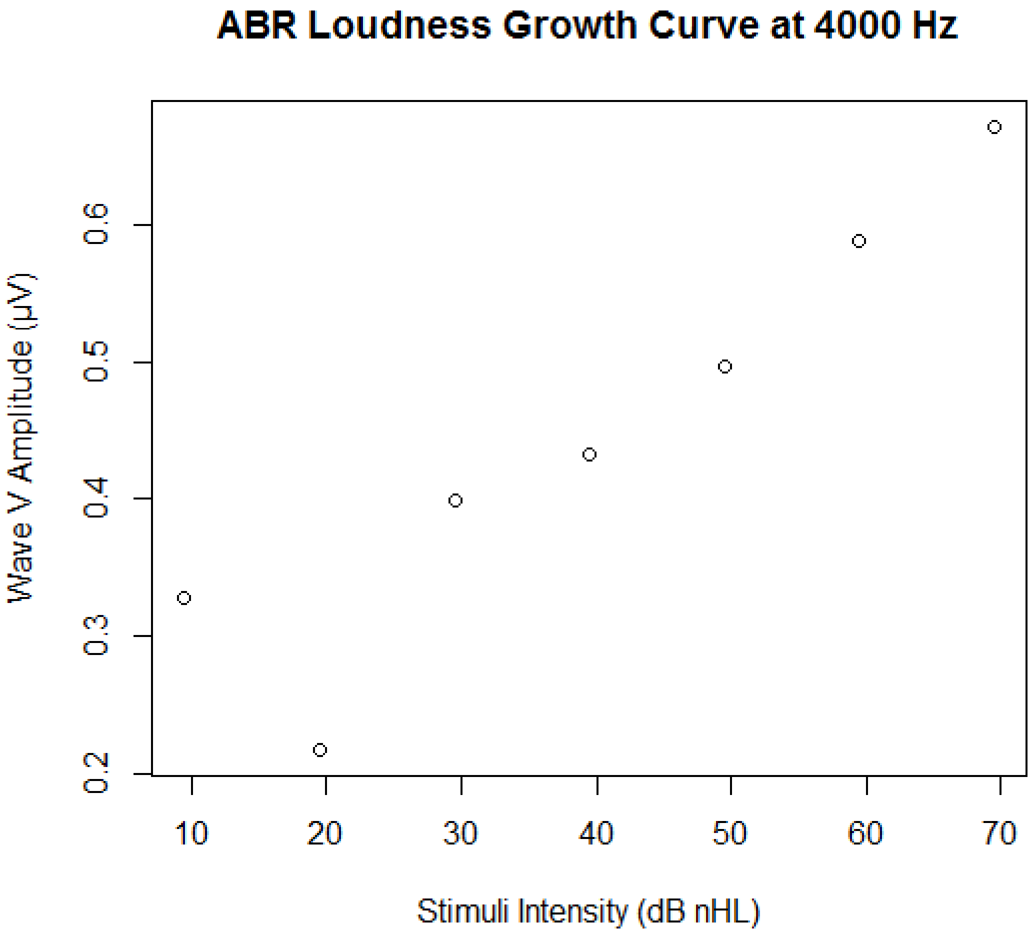

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.3
0.4
0.5
0.6
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

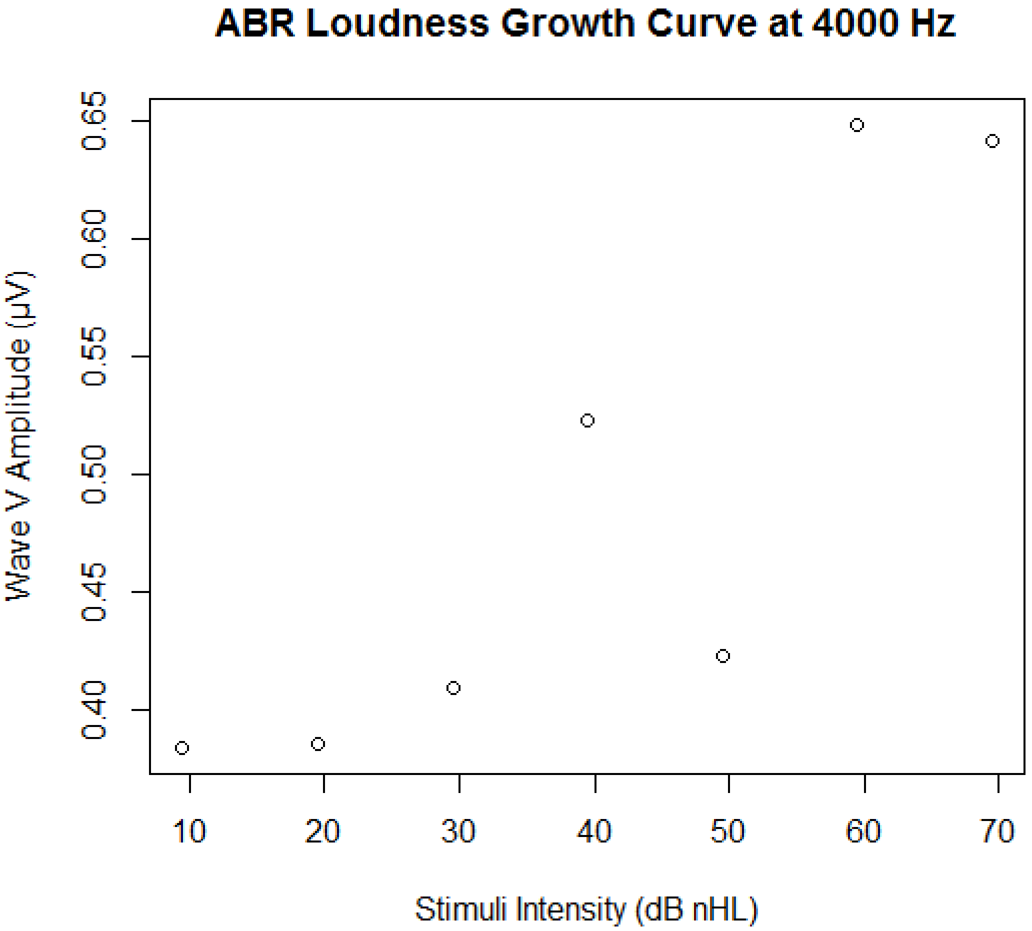

ABR Loudness Growth Curve at 1000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

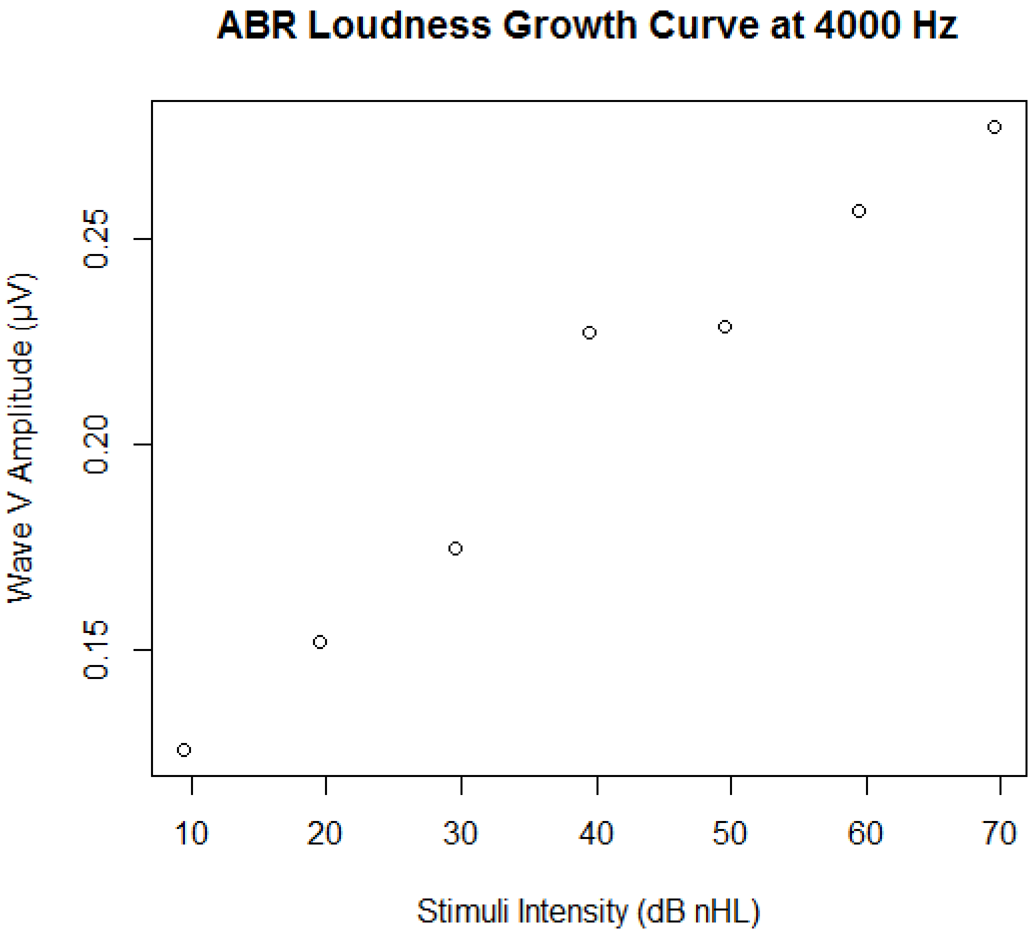
Individual ABR Loudness Growth Curves at 4000 Hz

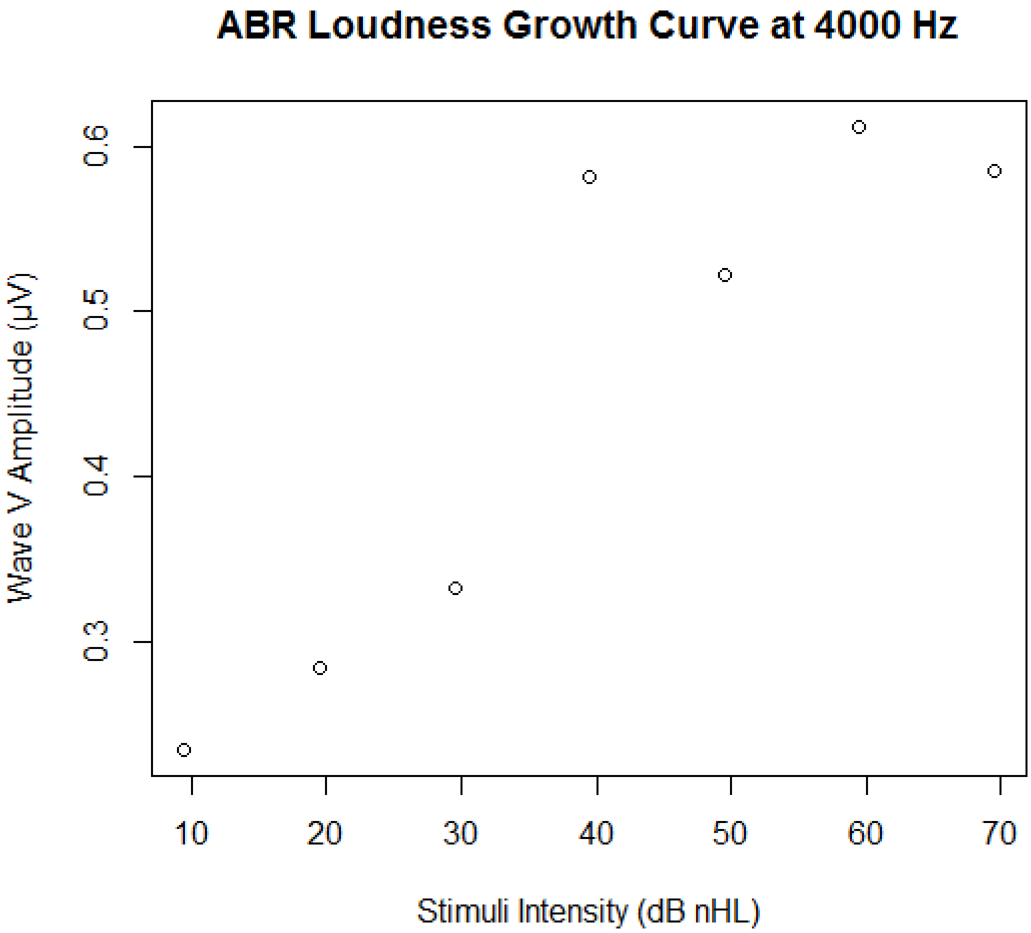


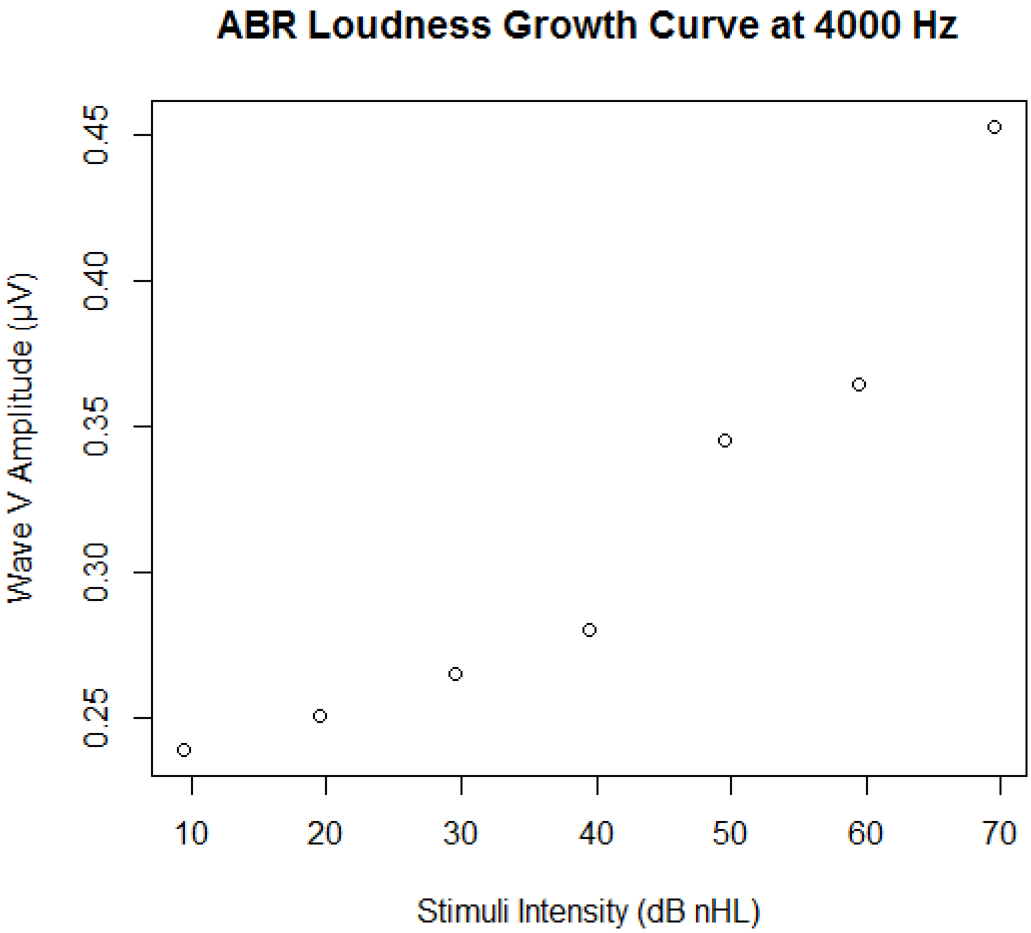

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.35
0.40
0.45
0.50
0.55
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

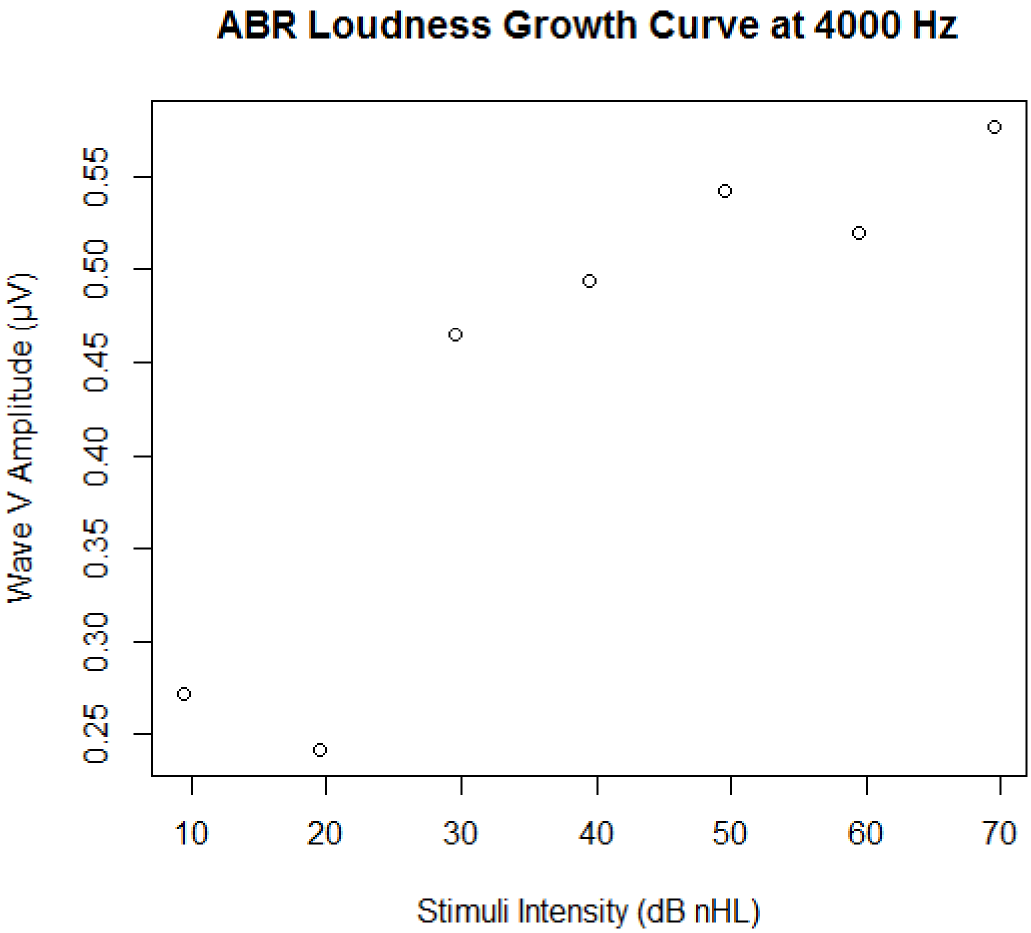

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (μV)
0.2
0.3
0.4
0.5
0.6
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

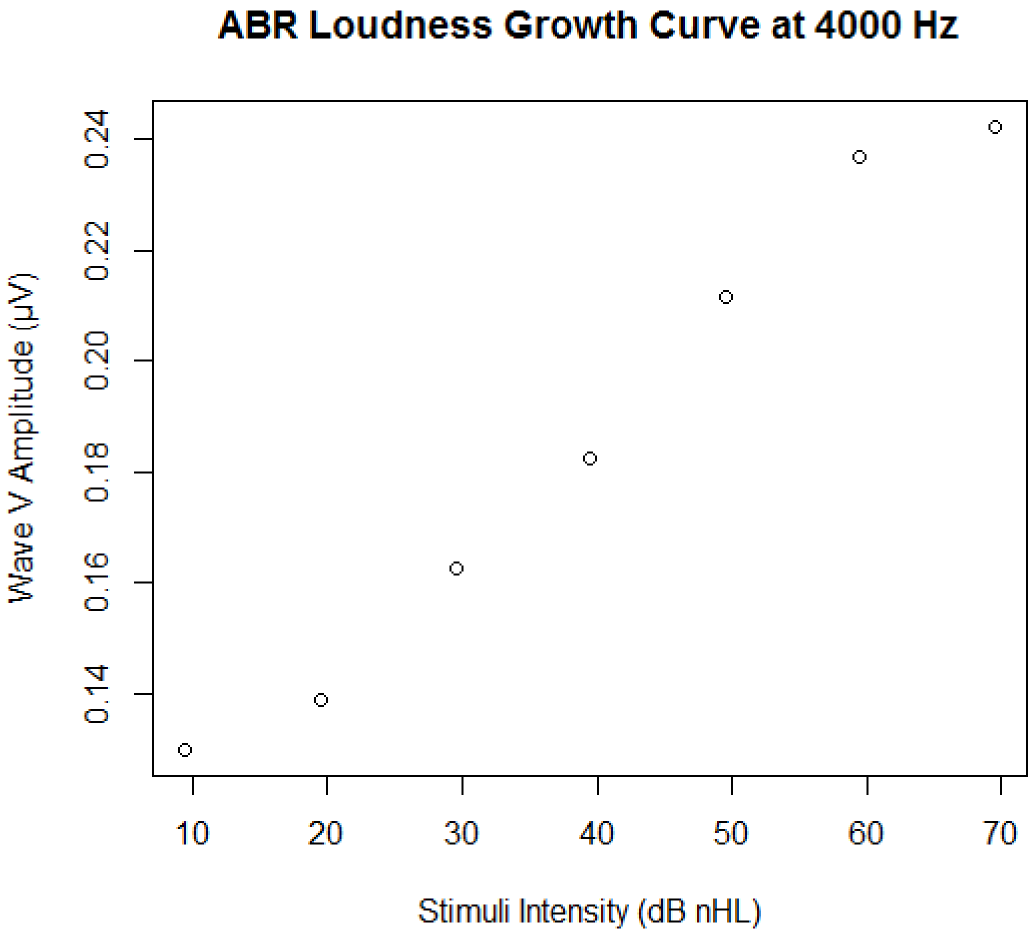

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.40
0.45
0.50
0.55
0.60
0.65
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

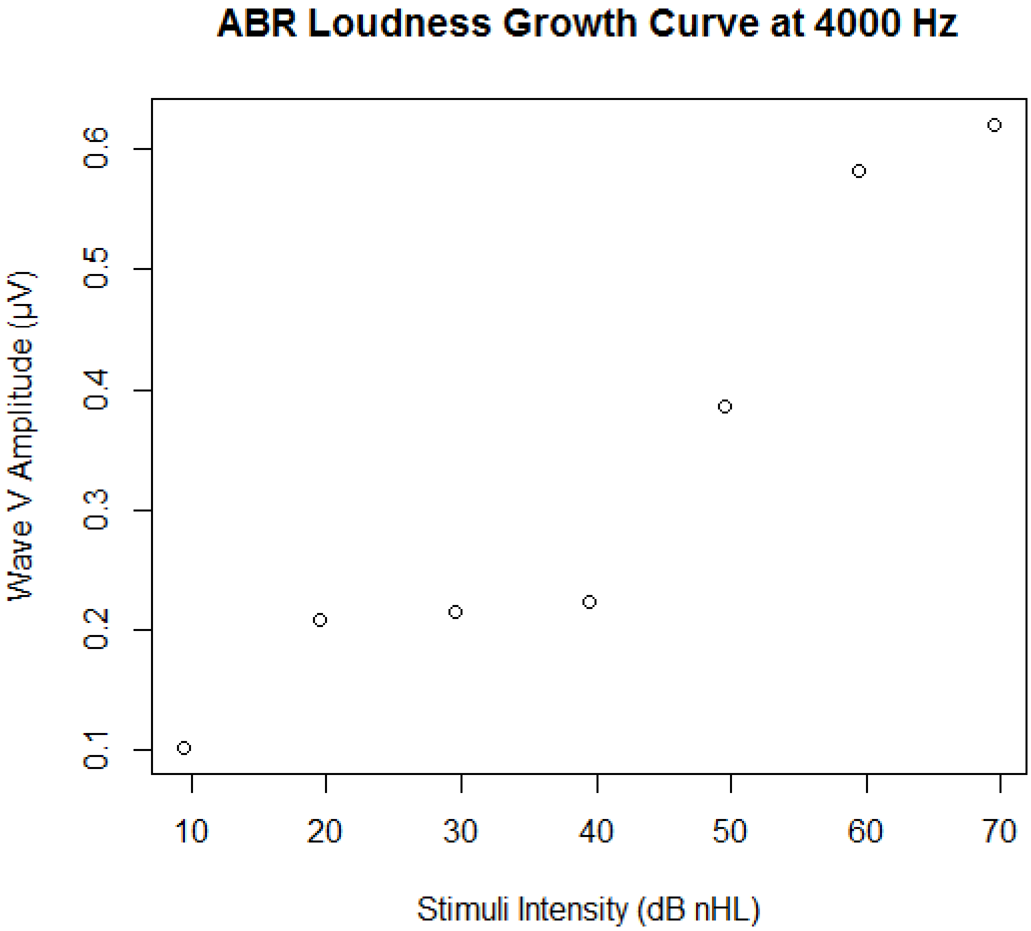

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.15
0.20
0.25
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

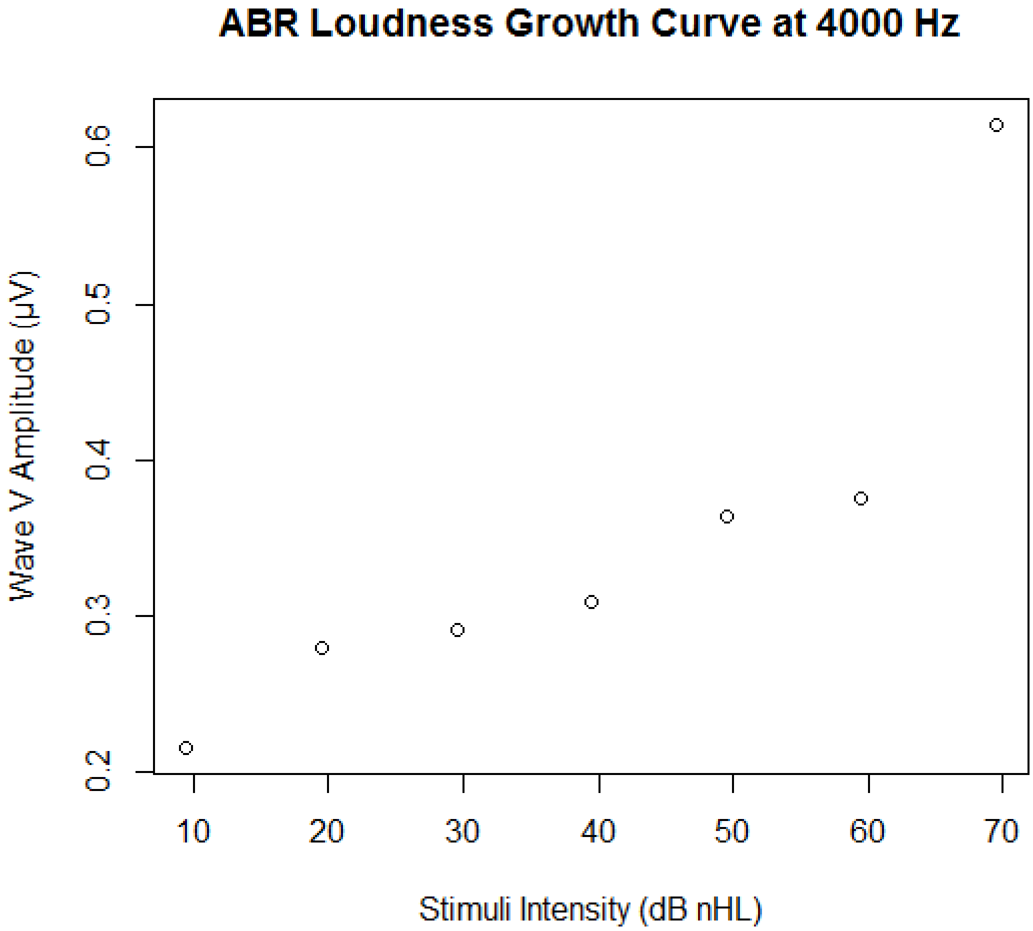

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.3
0.4
0.5
0.6
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

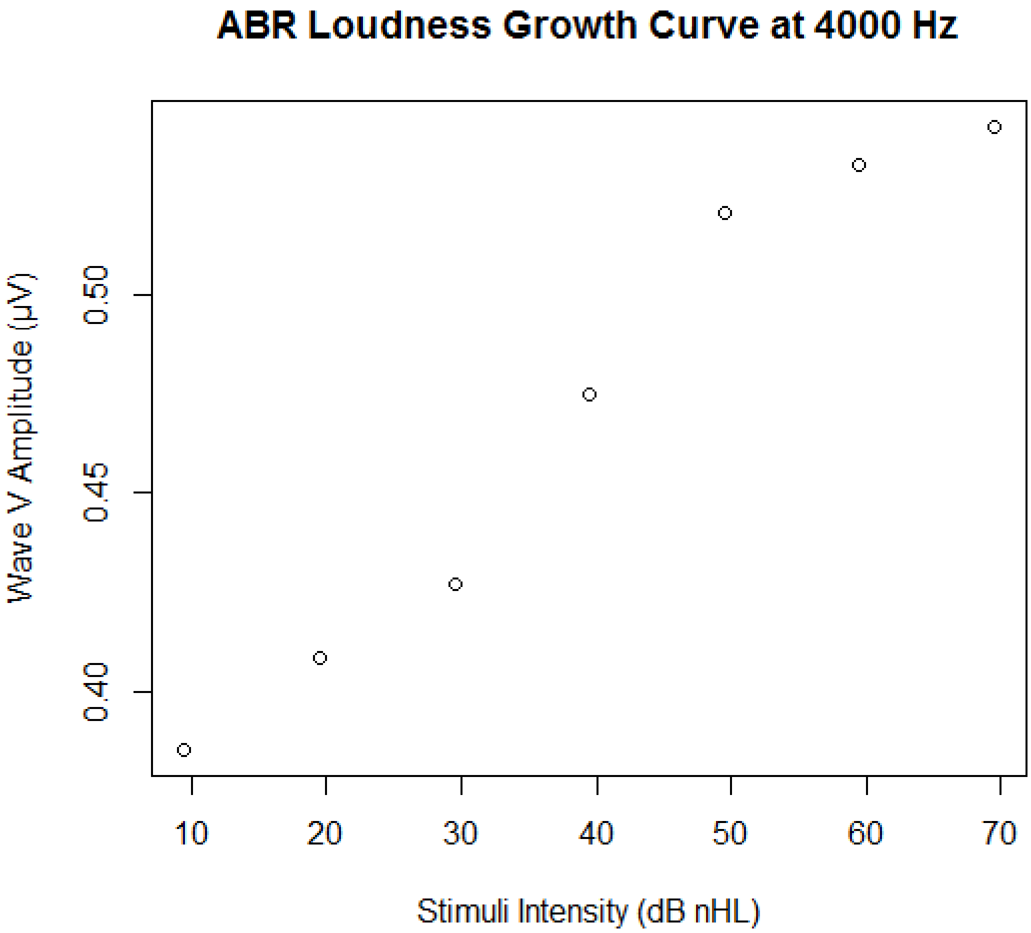

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
0.45
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

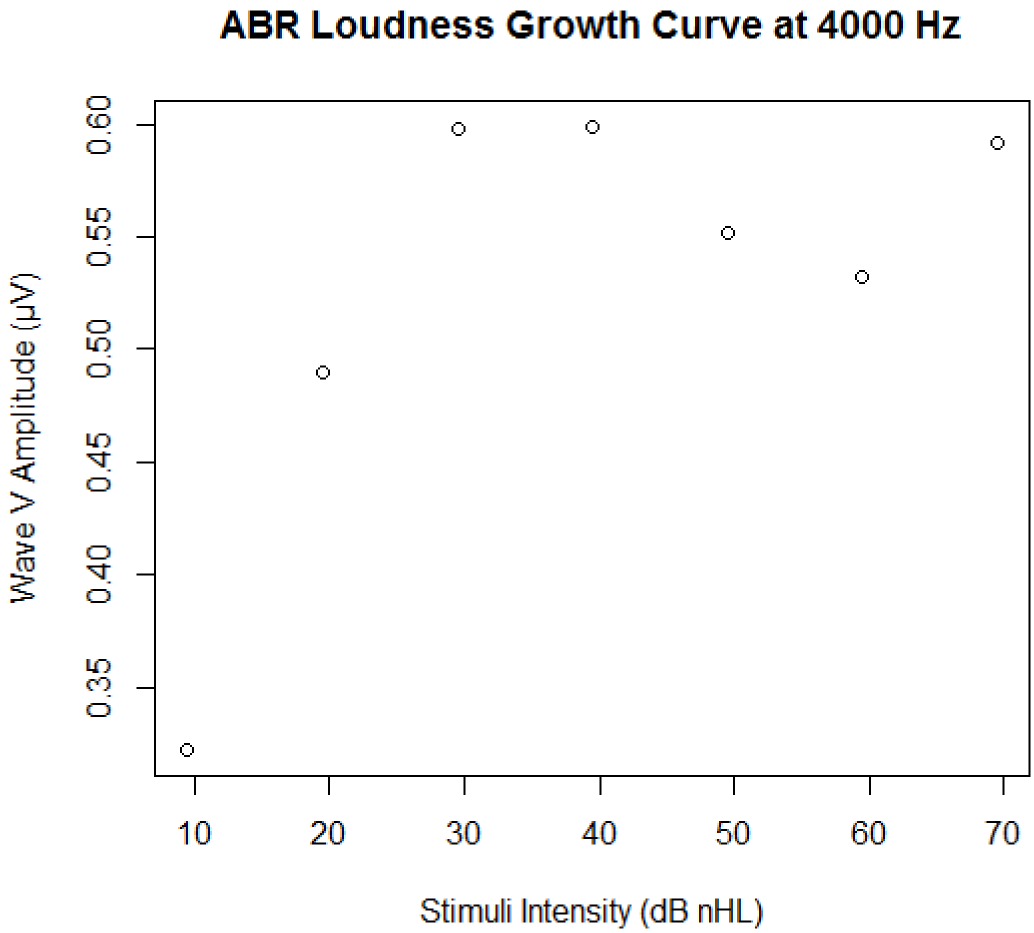

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
0.45
0.50
0.55
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

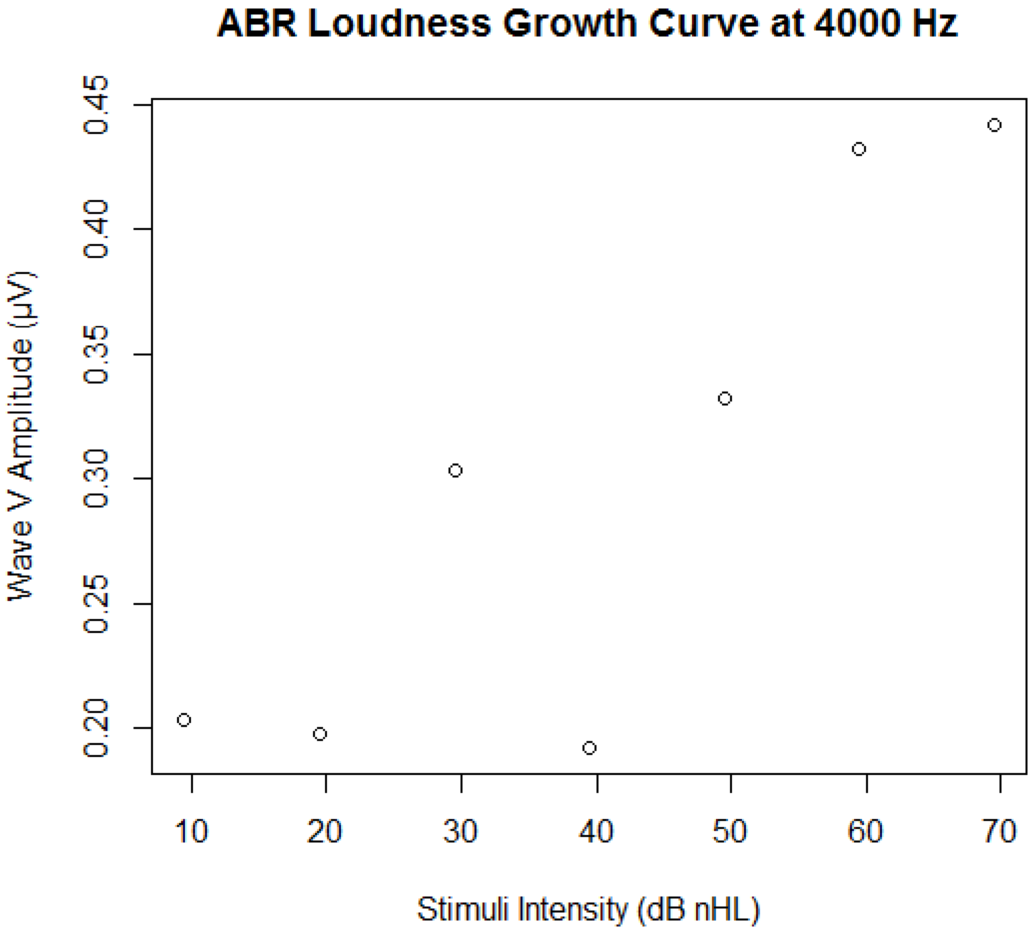

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.14
0.16
0.18
0.20
0.22
0.24
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

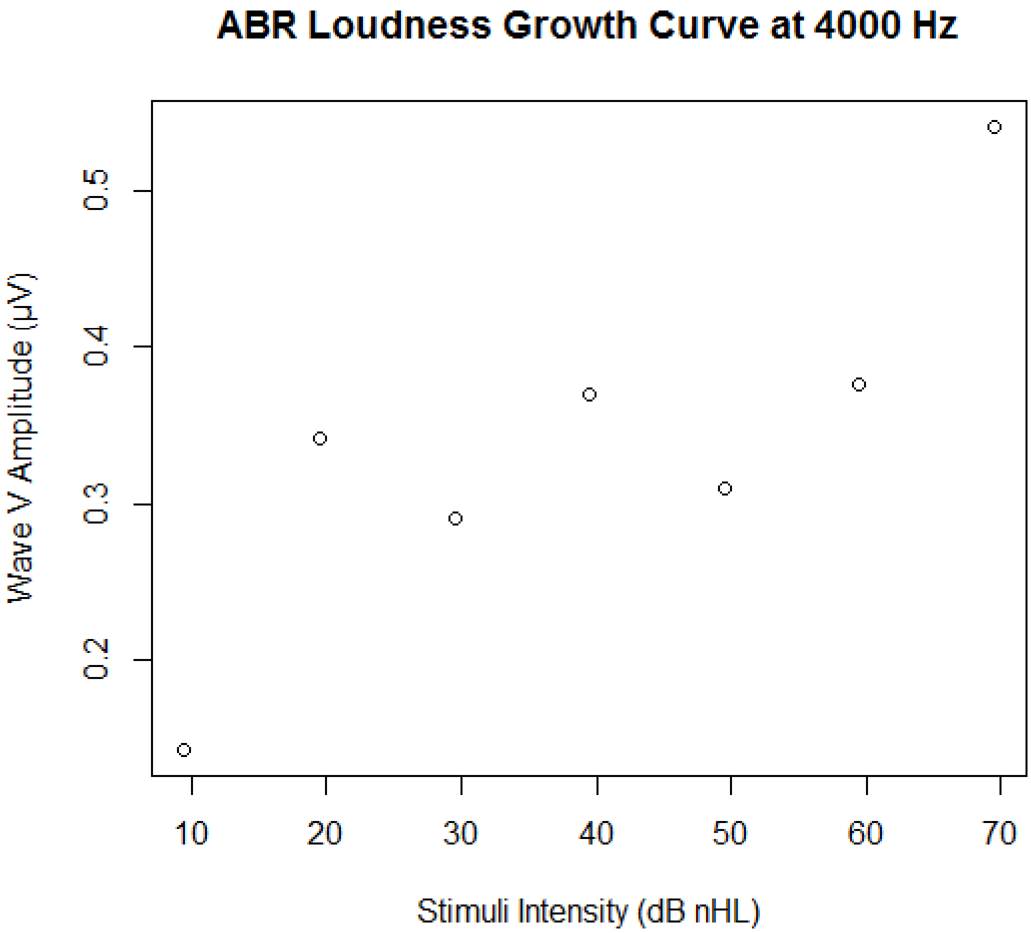

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.1
0.2
0.3
0.4
0.5
0.6
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

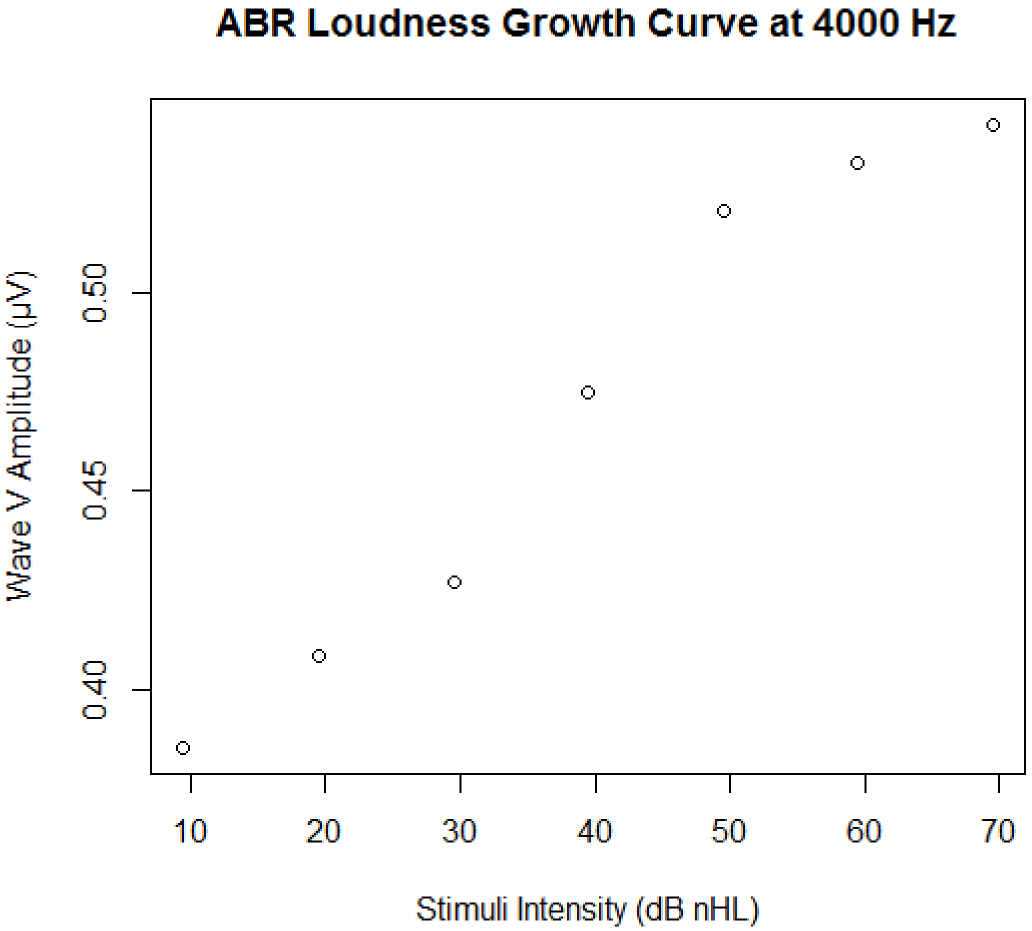

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.2
0.3
0.4
0.5
0.6
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

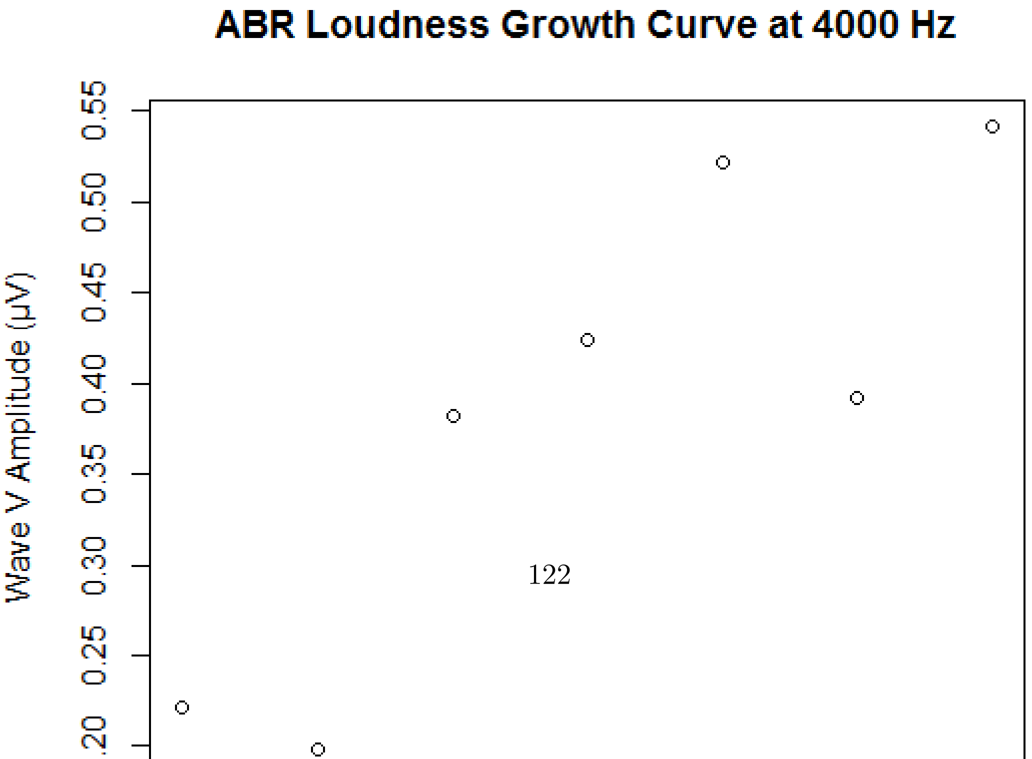

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.40
0.45
0.50
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)


ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (μV)
0.35
0.40
0.45
0.50
0.55
0.60
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

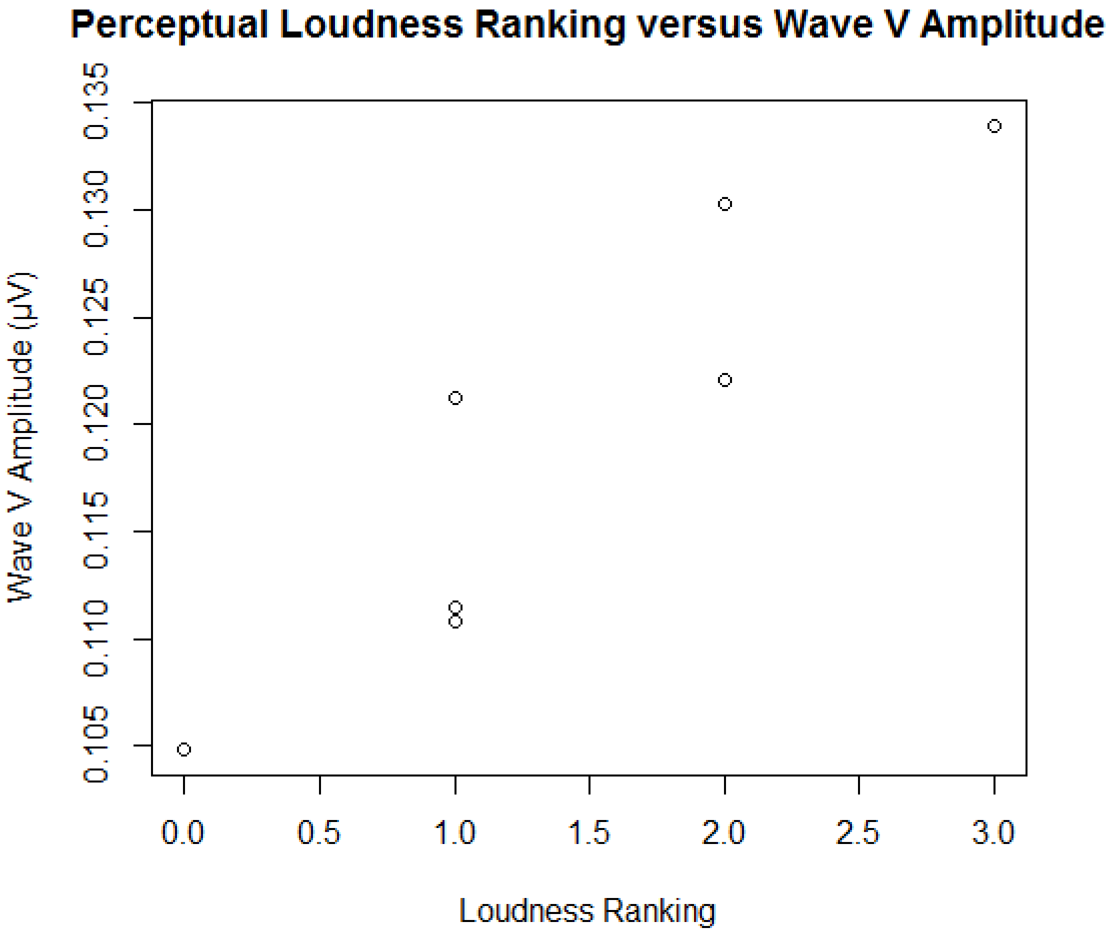

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.20
0.25
0.30
0.35
0.40
0.45
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

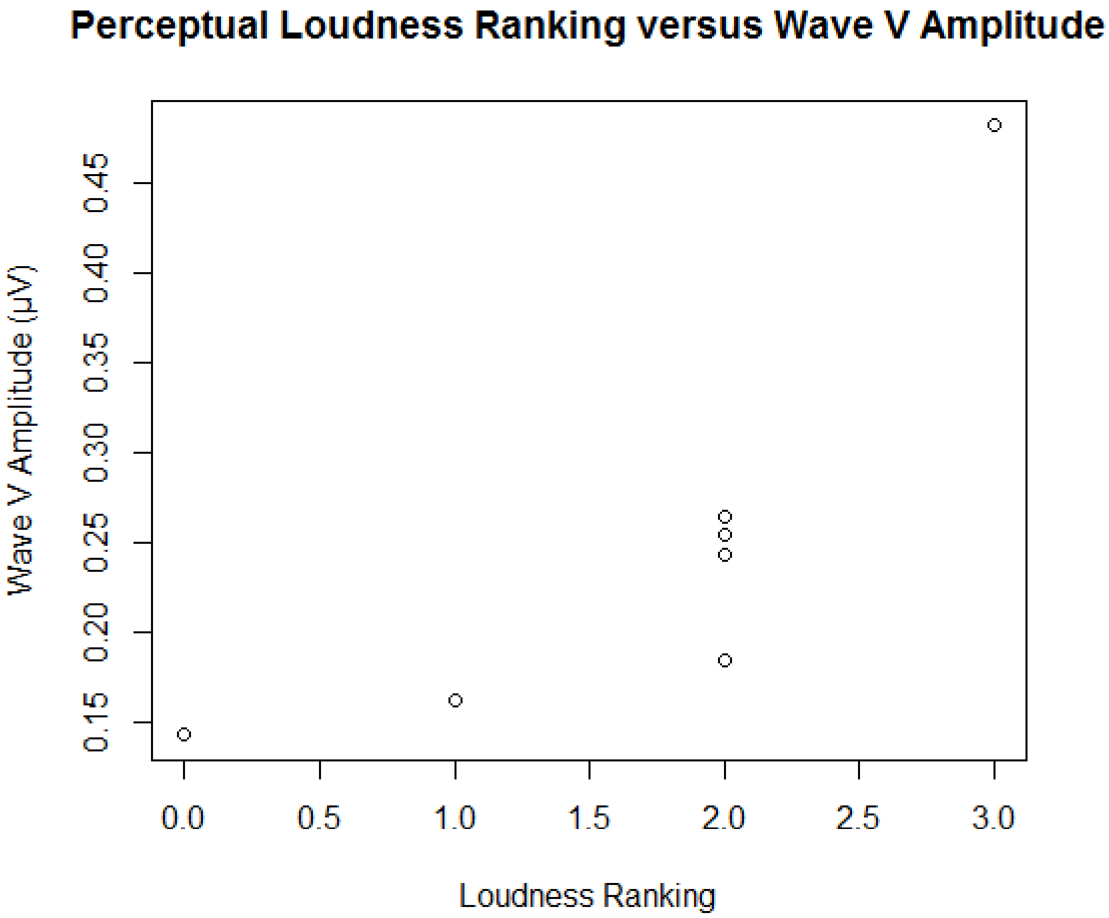

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.2
0.3
0.4
0.5
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

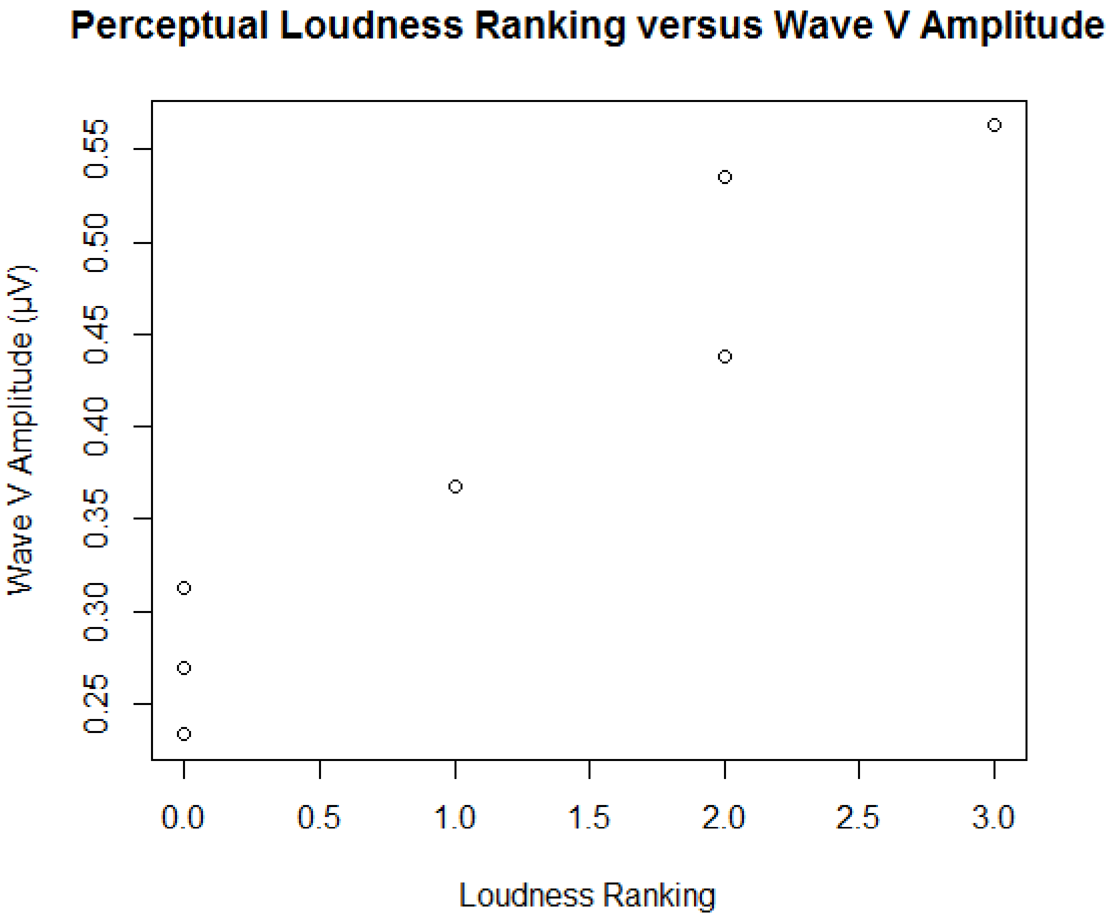

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
0.40
0.45
0.50
10
20
30
40
50
60
70
Stimuli Intensity (dB nHL)

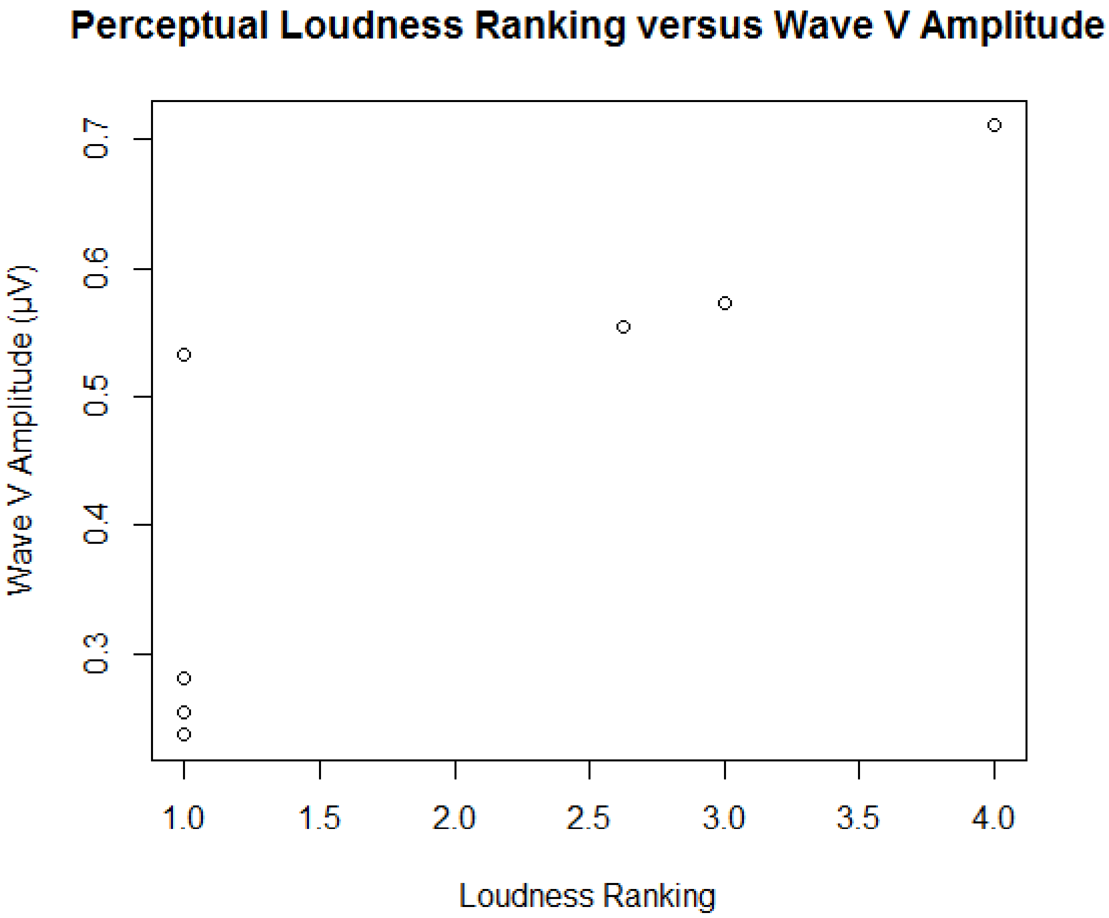

ABR Loudness Growth Curve at 4000 Hz
Wave V Amplitude (µV)
.20
0.25
0.30
0.35
0.40
0.45
0.50
0.55

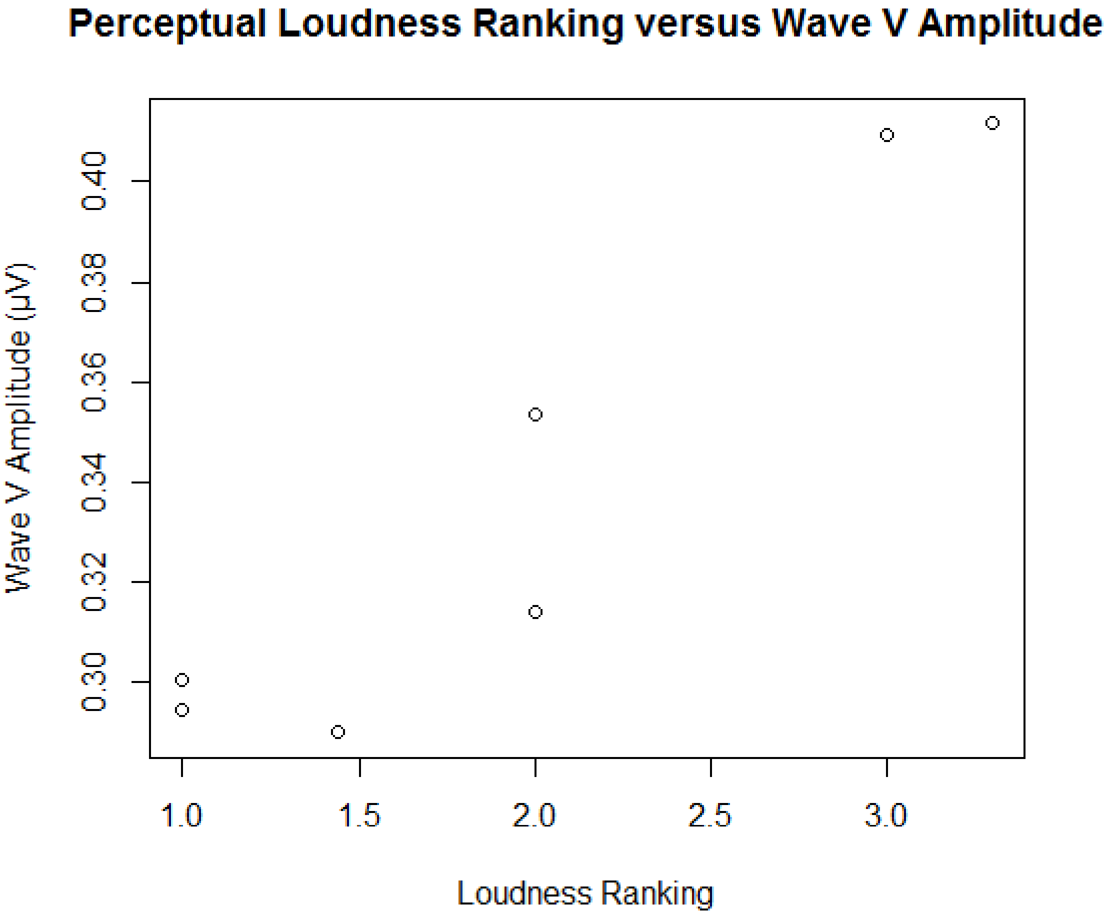
Individual Relation between Psychoacoustic and ABR Loudness Growth at 1000 Hz

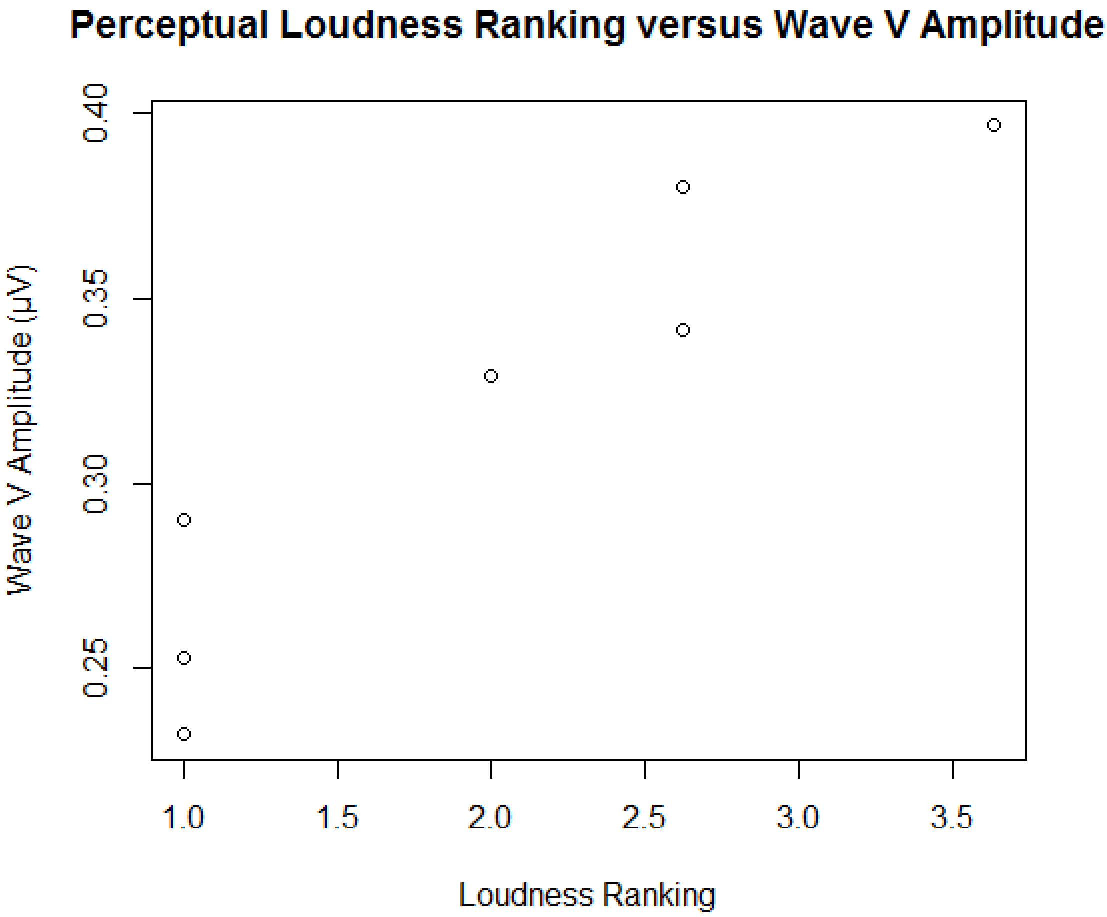


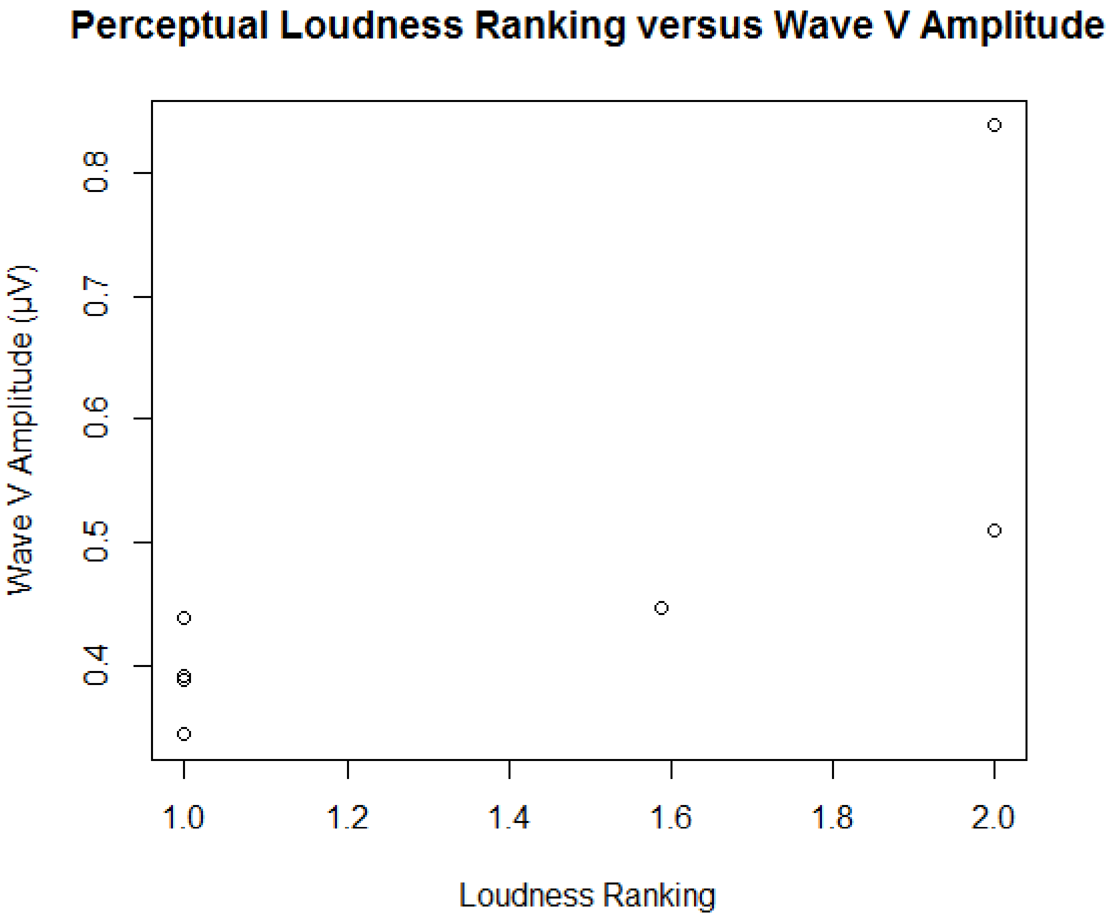

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.15
0.20
0.25
0.30
0.35
0.40
0.45
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

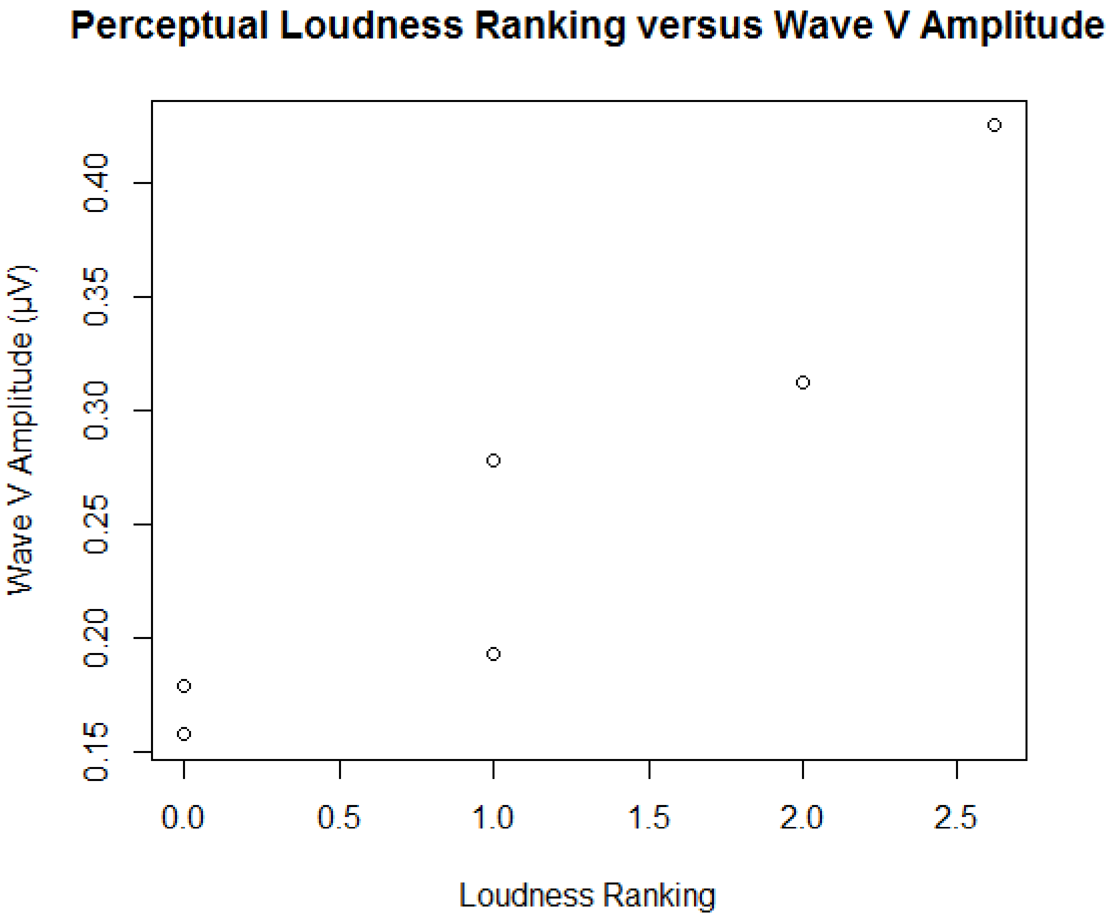

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.25
0.30
0.35
0.40
0.45
0.50
0.55
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

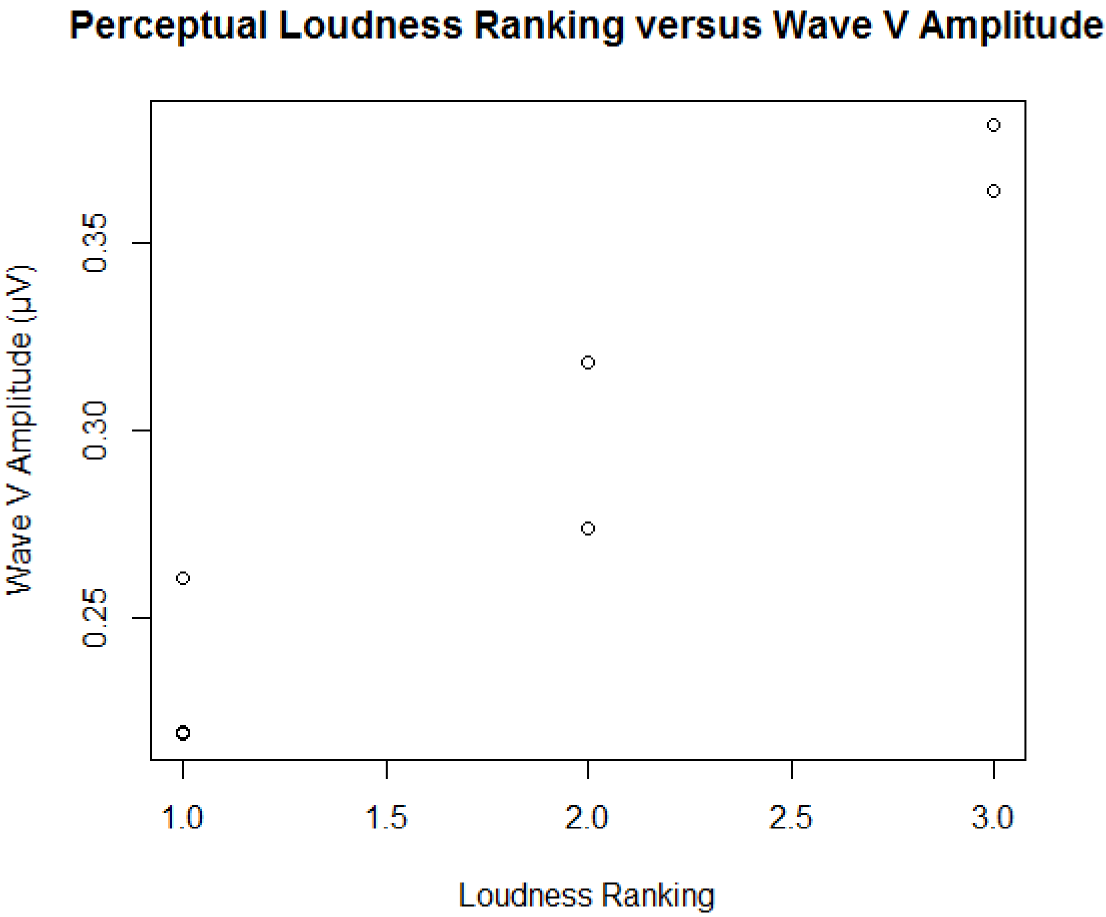

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.3
0.4
0.5
0.6
0.7
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Loudness Ranking

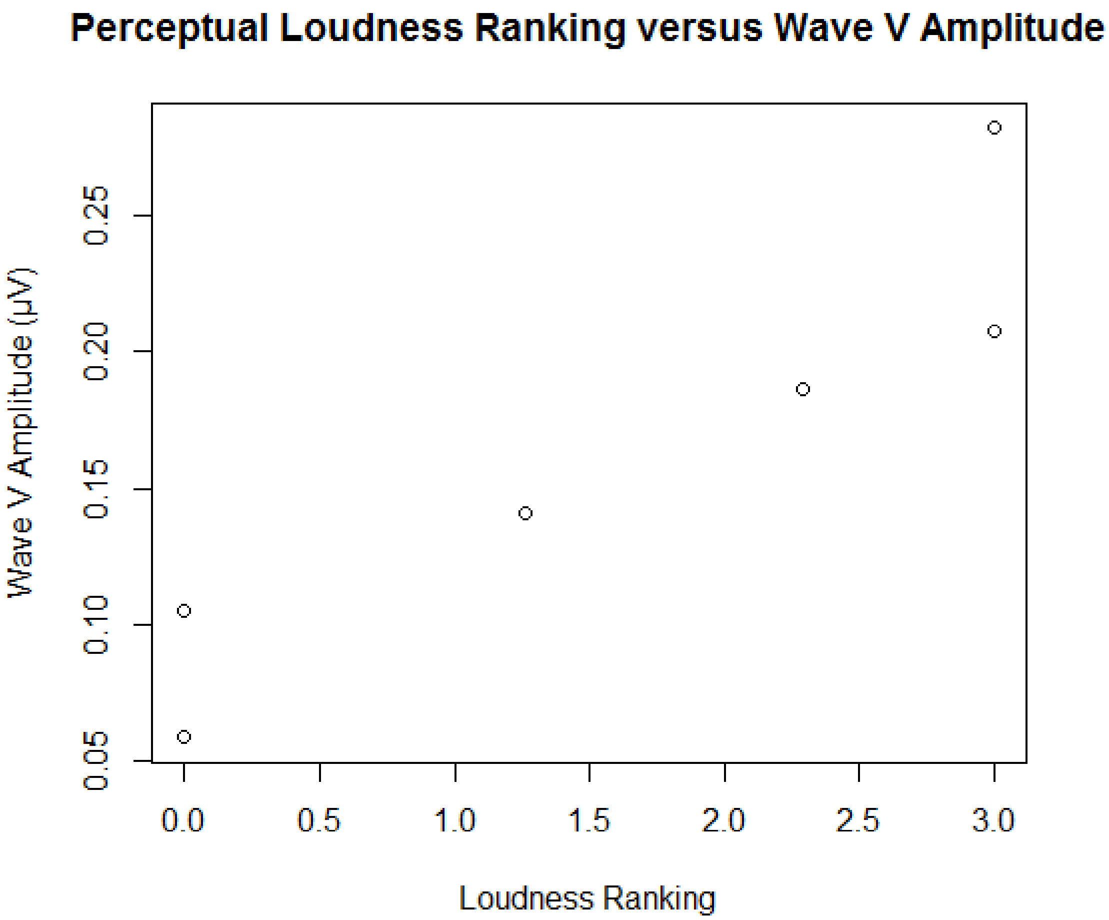

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.30
0.32
0.34
0.36
0.38
0.40
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

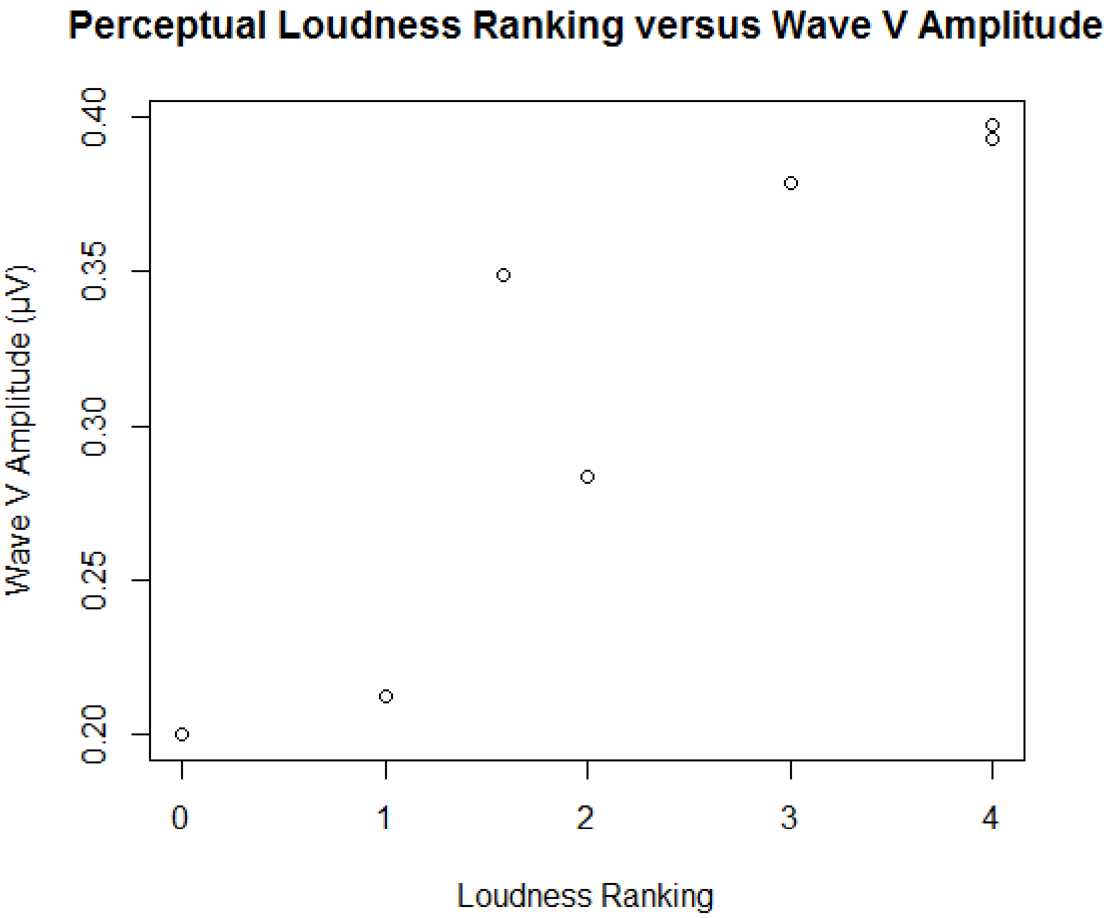

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.25
0.30
0.35
0.40
1.0
1.5
2.0
2.5
3.0
3.5
Loudness Ranking

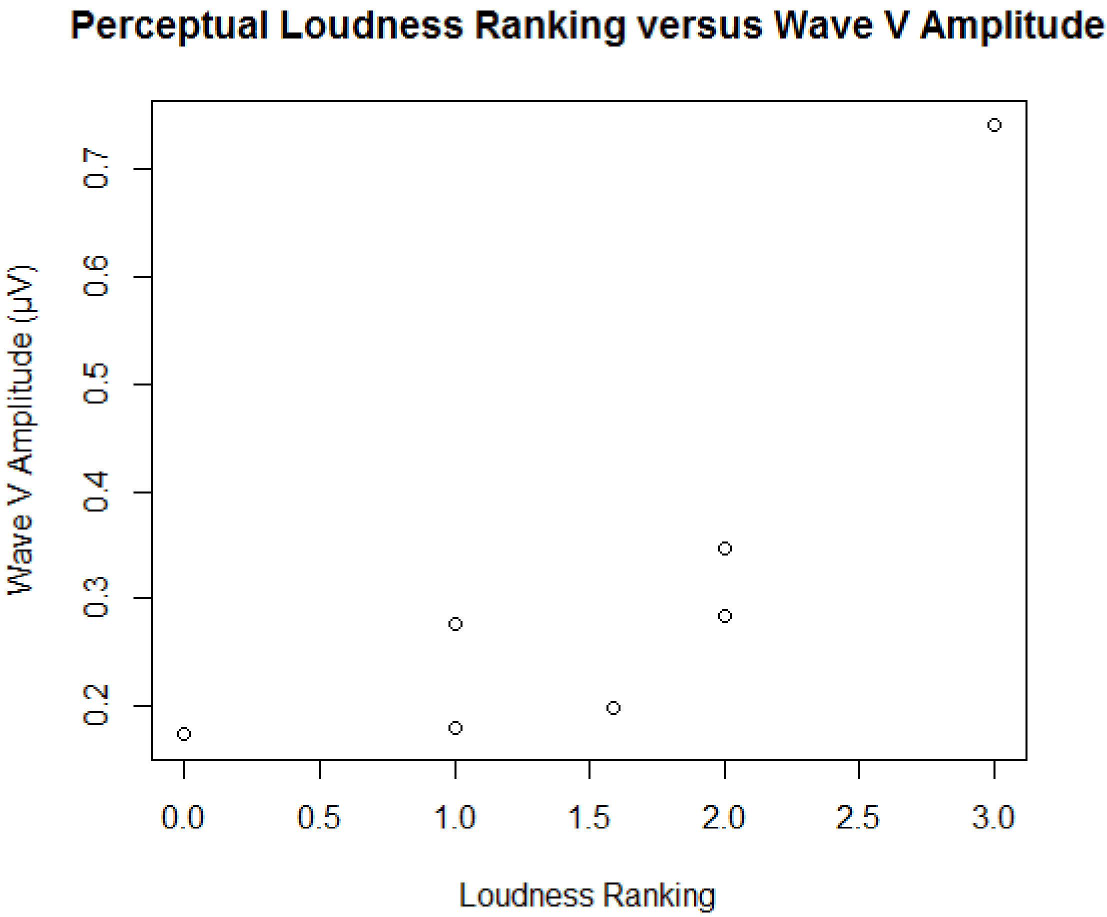

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.4
0.5
0.6
0.7
0.8
1.0
1.2
1.4
1.6
1.8
2.0
Loudness Ranking

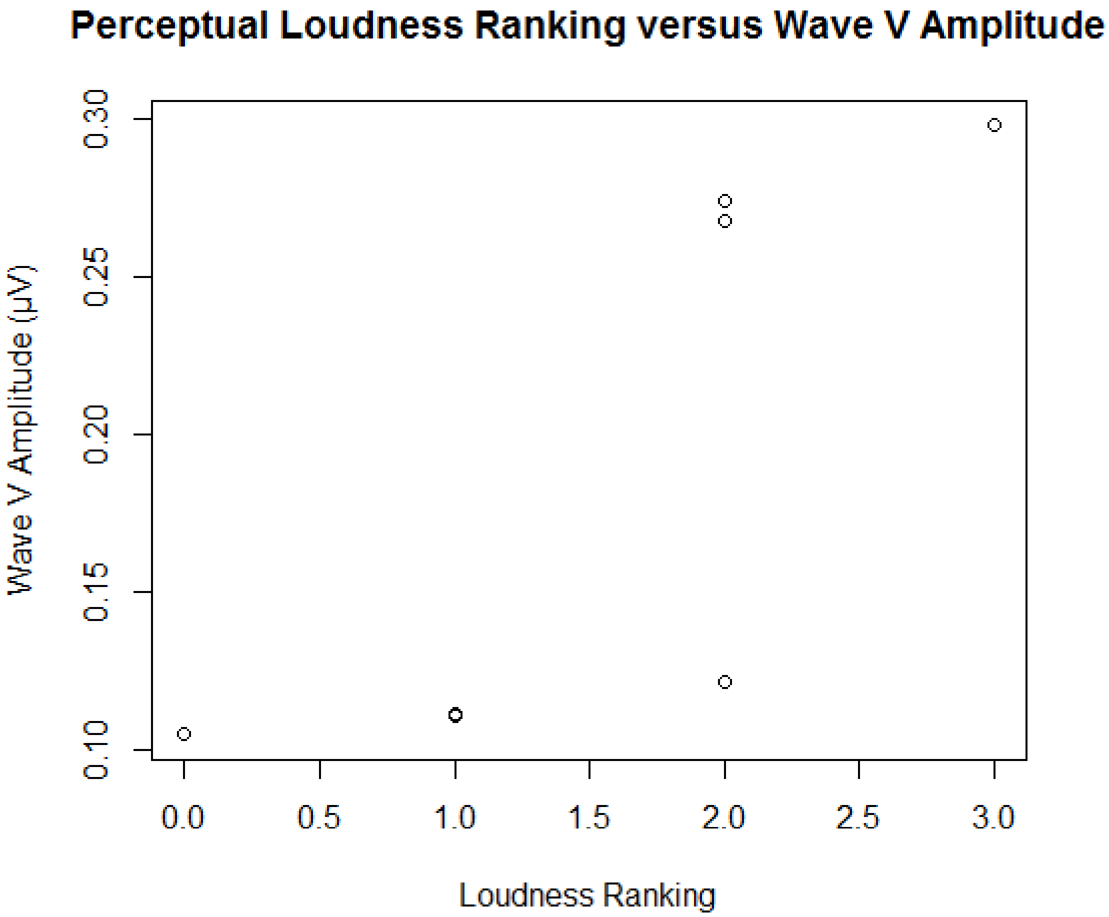

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.15
0.20
0.25
0.30
0.35
0.40
0.0
0.5
1.0
1.5
2.0
2.5
Loudness Ranking

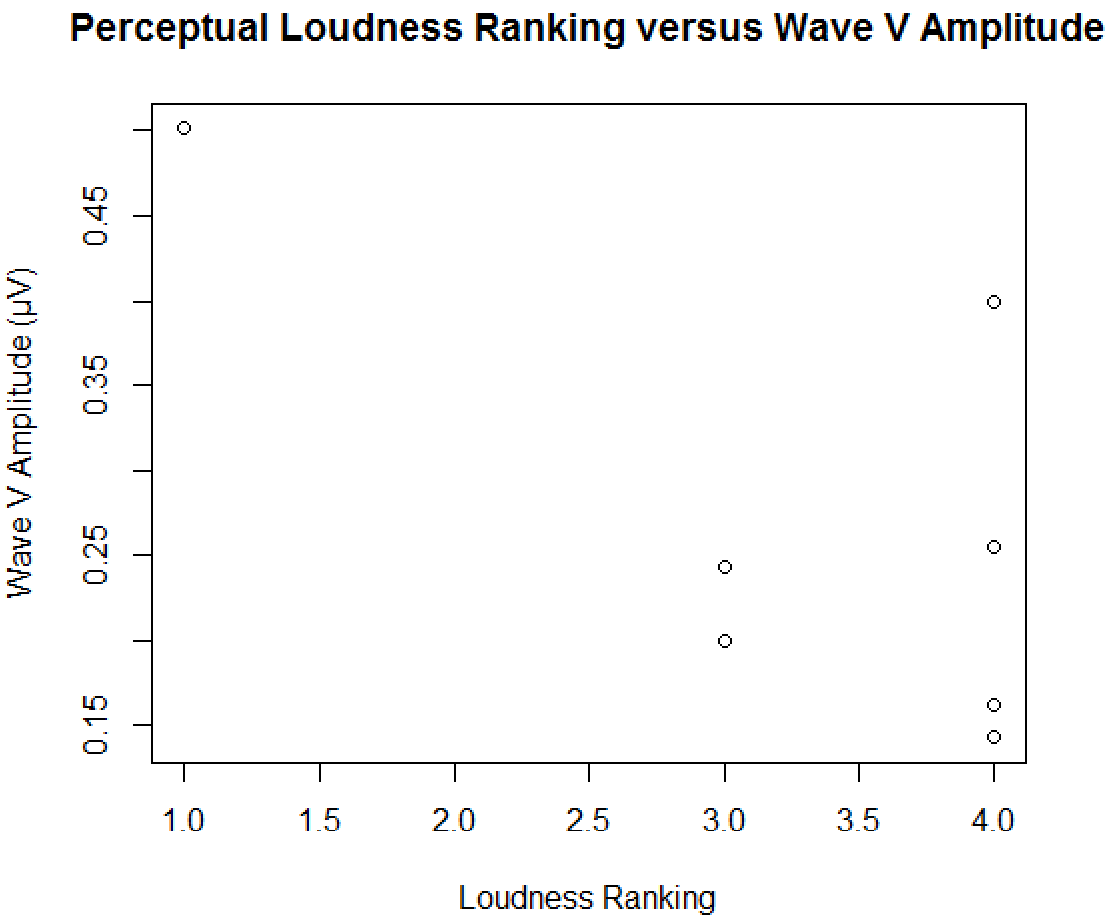

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.25
0.30
0.35
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

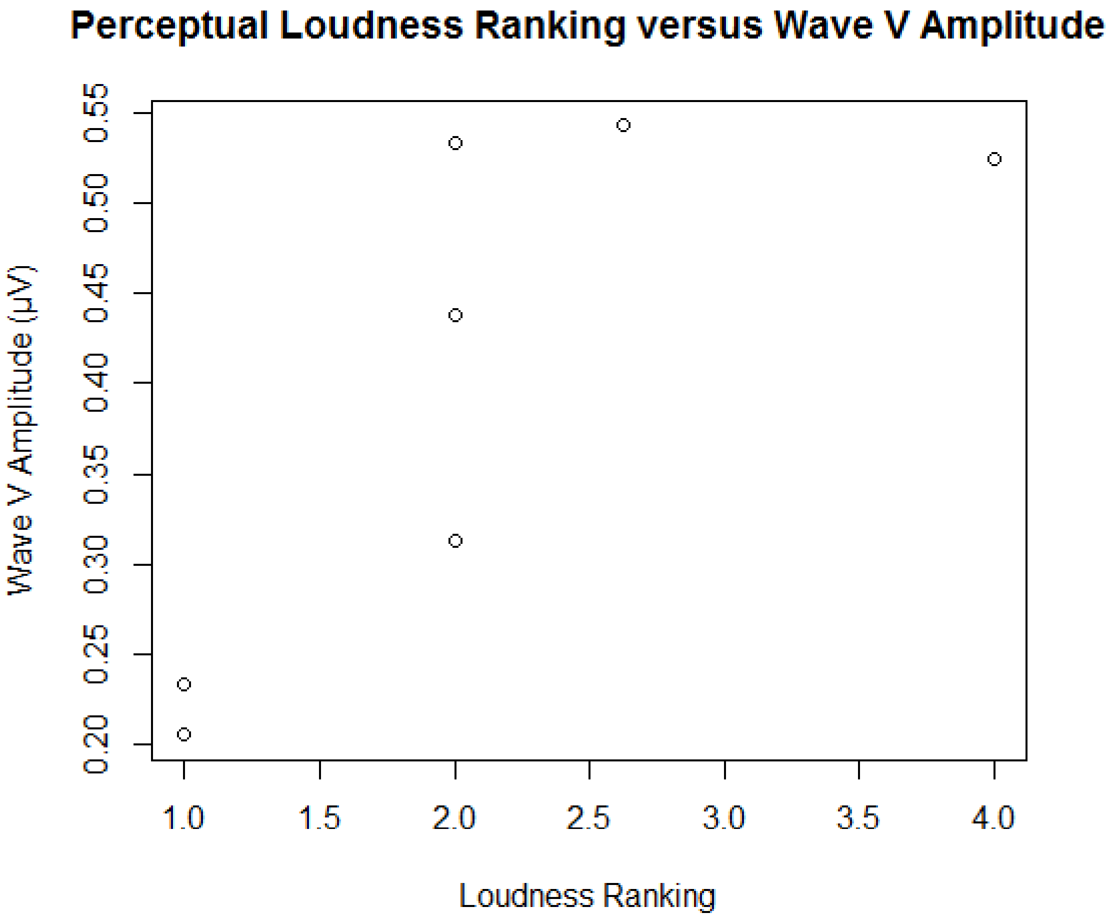

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.05
0.10
0.15
0.20
0.25
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

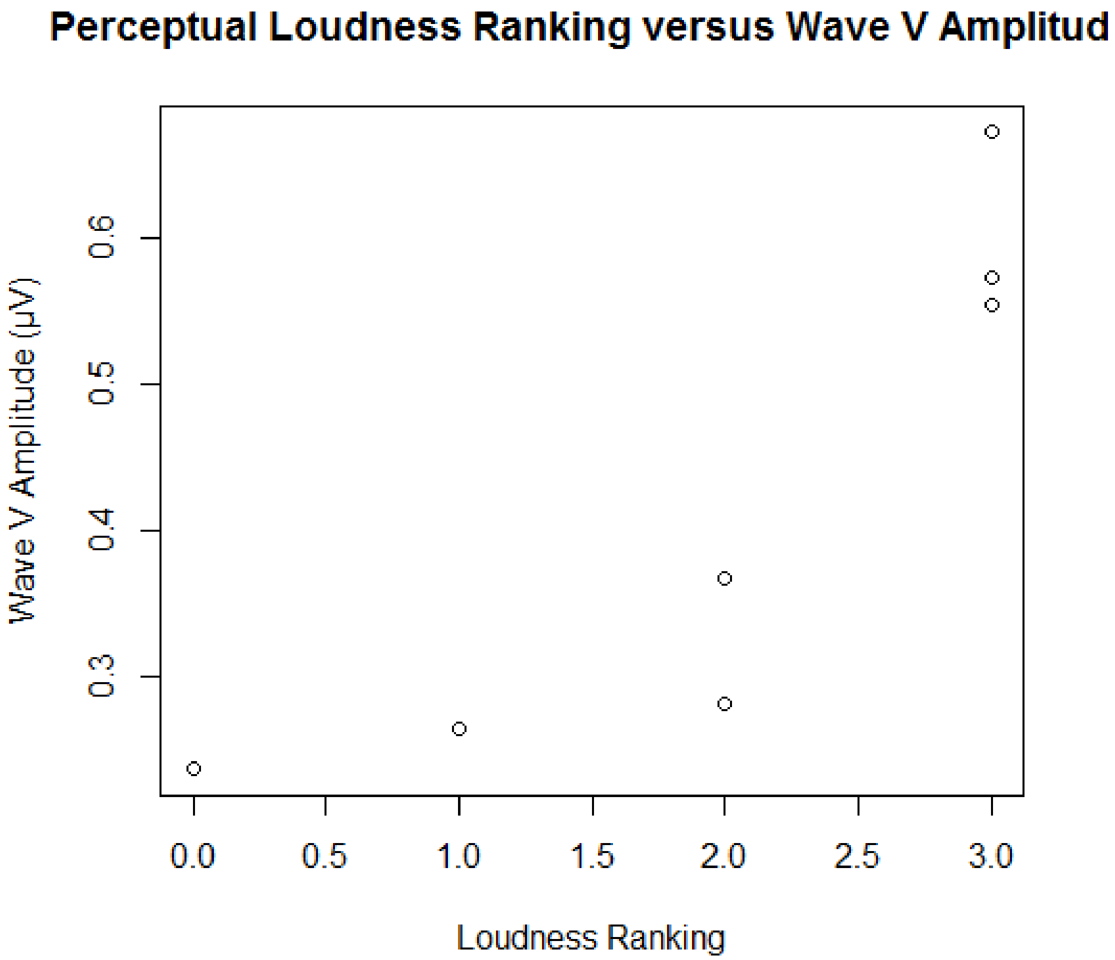

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.20
0.25
0.30
0.35
0.40
0
1
2
3
4
Loudness Ranking

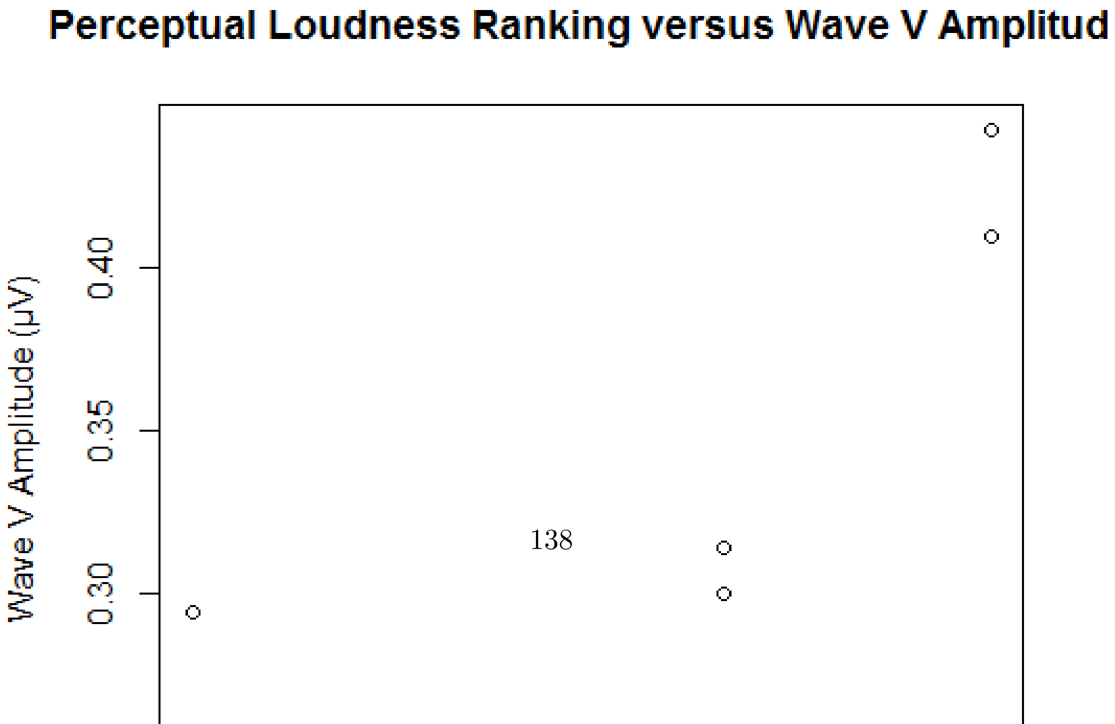

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.2
0.3
0.4
0.5
0.6
0.7
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Loudness Ranking


Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.10
0.15
0.20
0.25
0.30
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

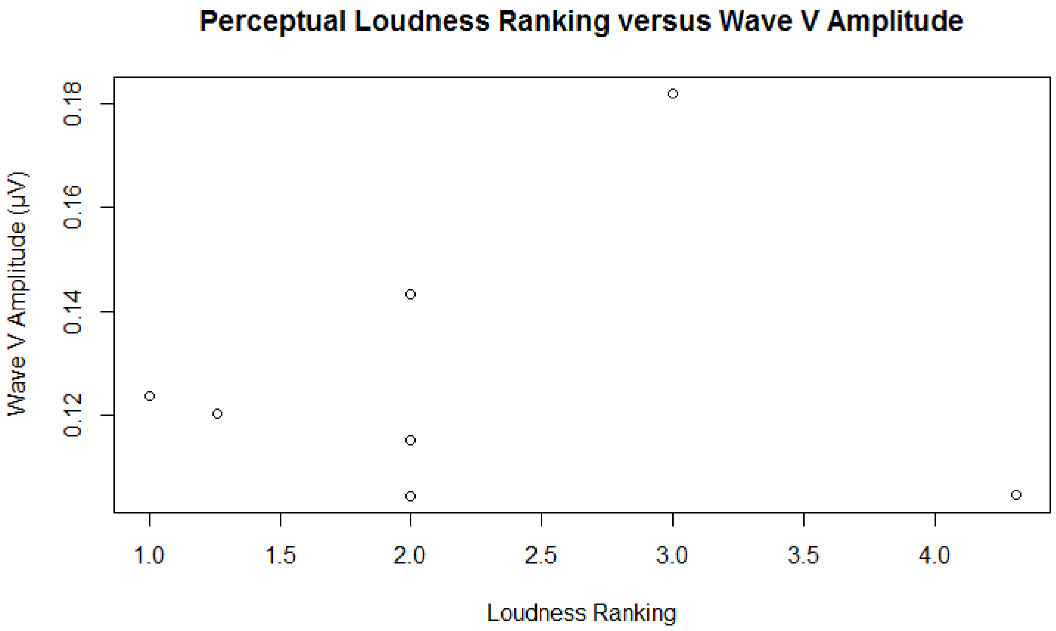

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.15
0.25
0.35
0.45
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Loudness Ranking

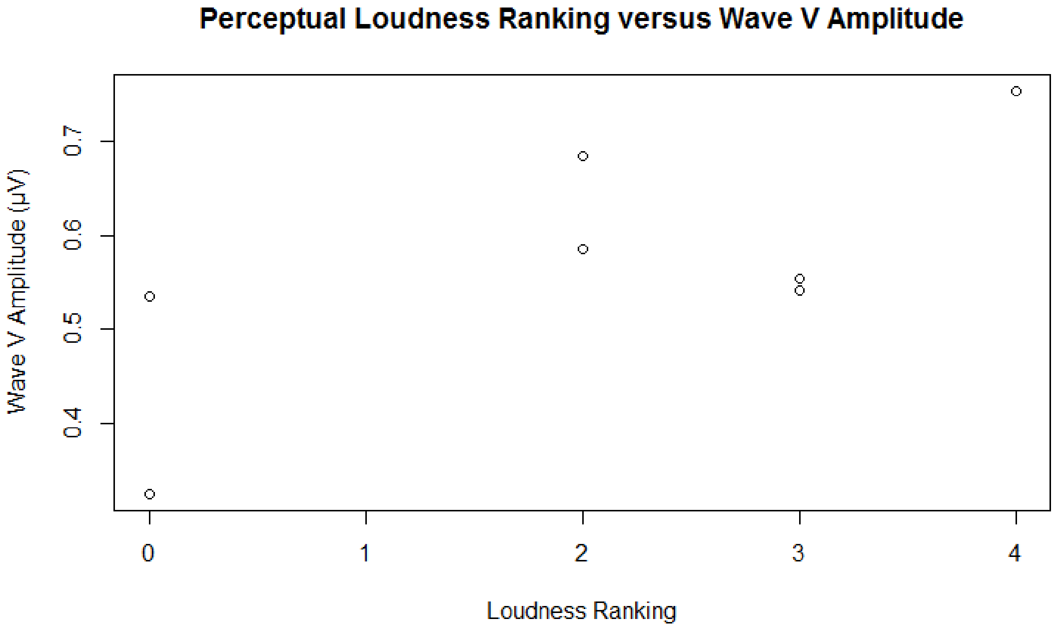

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.20
0.25
0.30
0.35
0.40
0.45
0.50
0.55
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Loudness Ranking

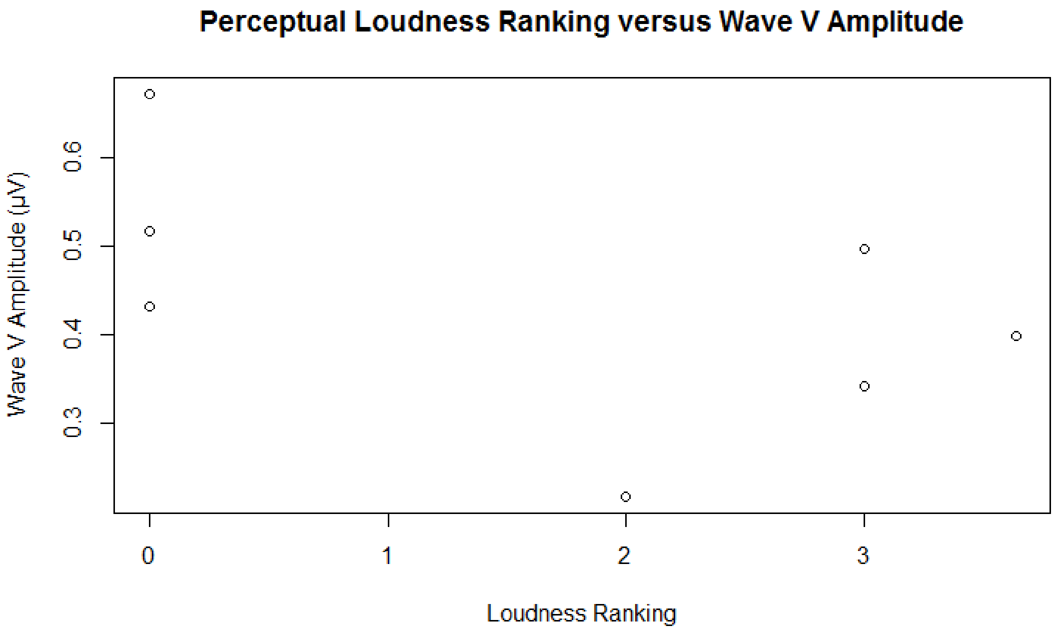

Perceptual Loudness Ranking versus Wave V Amplitud
Wave V Amplitude (µV)
0.3
0.4
0.5
0.6
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

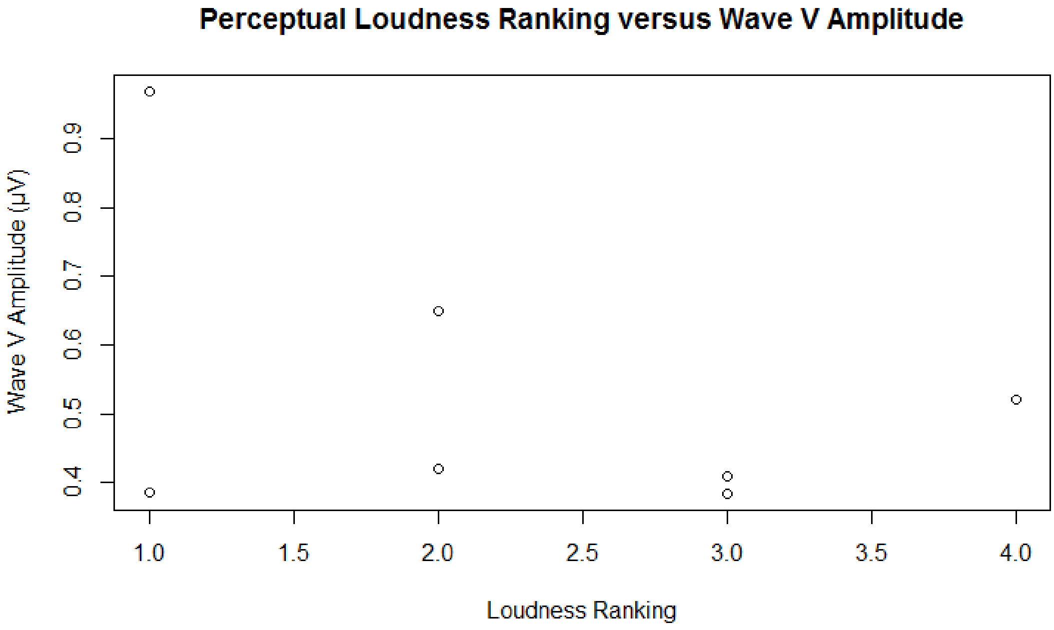

Perceptual Loudness Ranking versus Wave V Amplitud
Wave V Amplitude (µV)
0.30
0.35
0.40

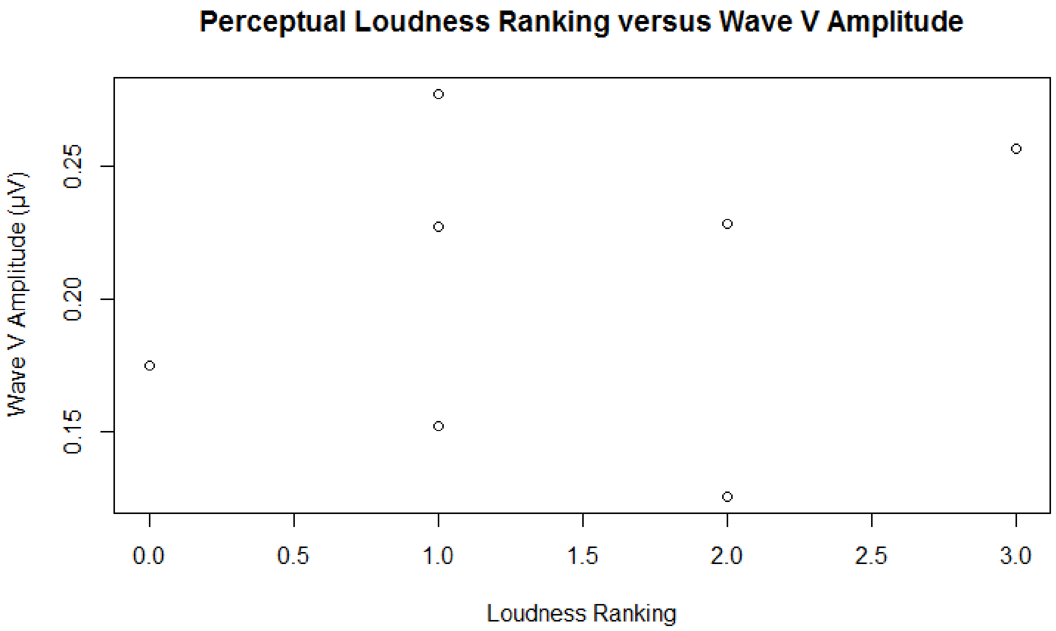
Individual Relation between Psychoacoustic and ABR Loudness Growth at 4000 Hz

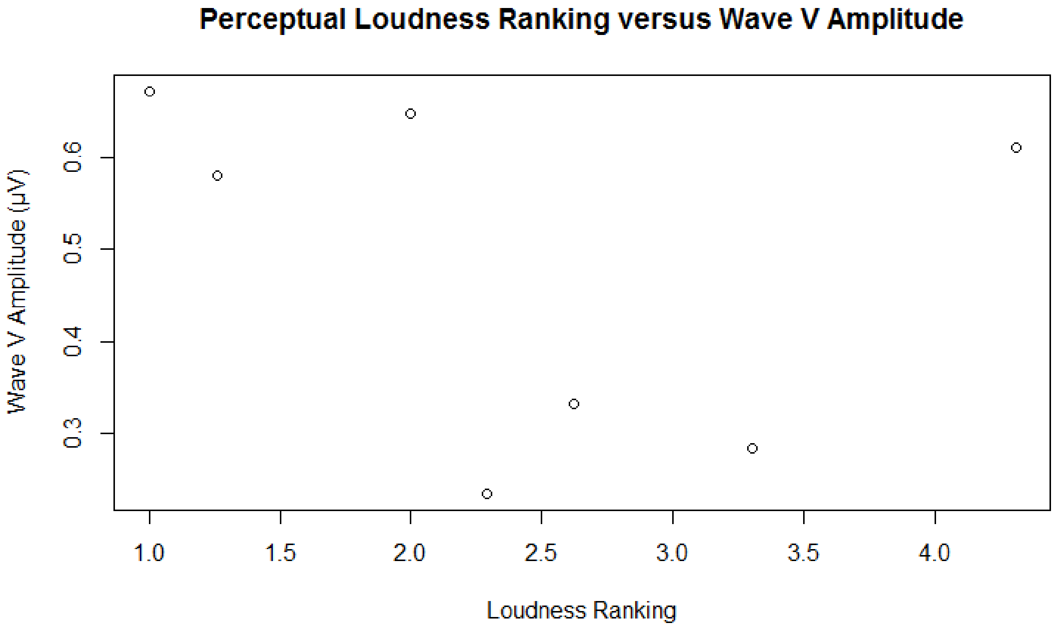


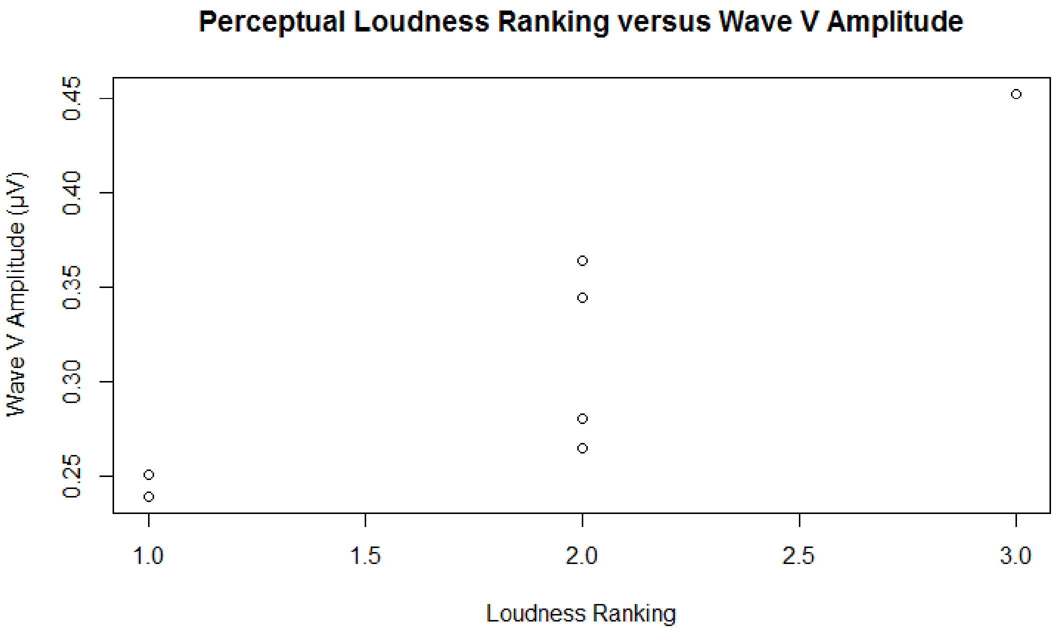


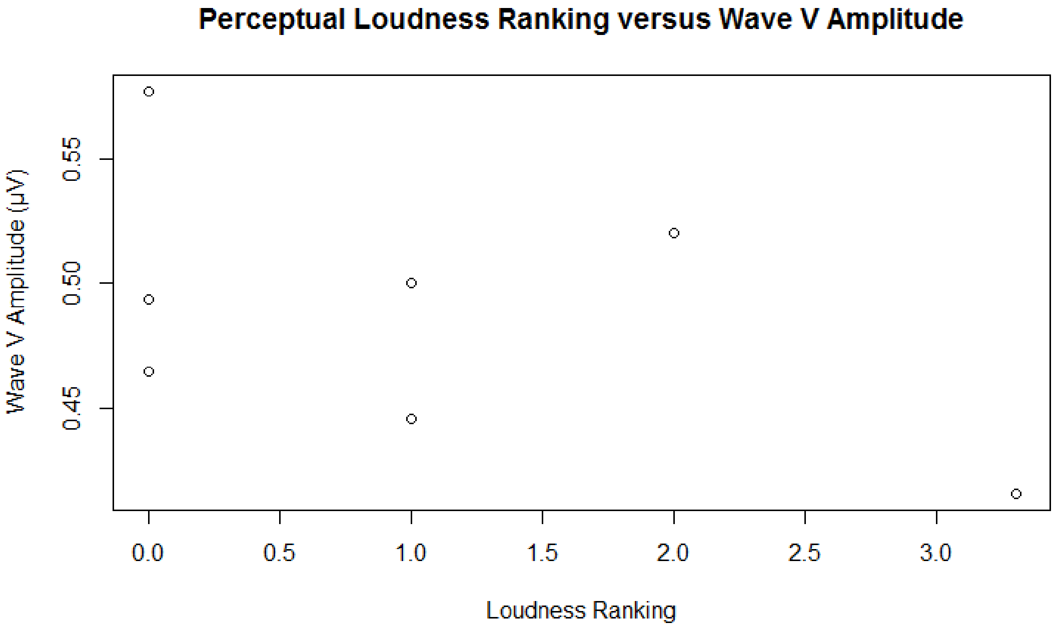

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.3
0.4
0.5
0.6
0
1
2
3
Loudness Ranking

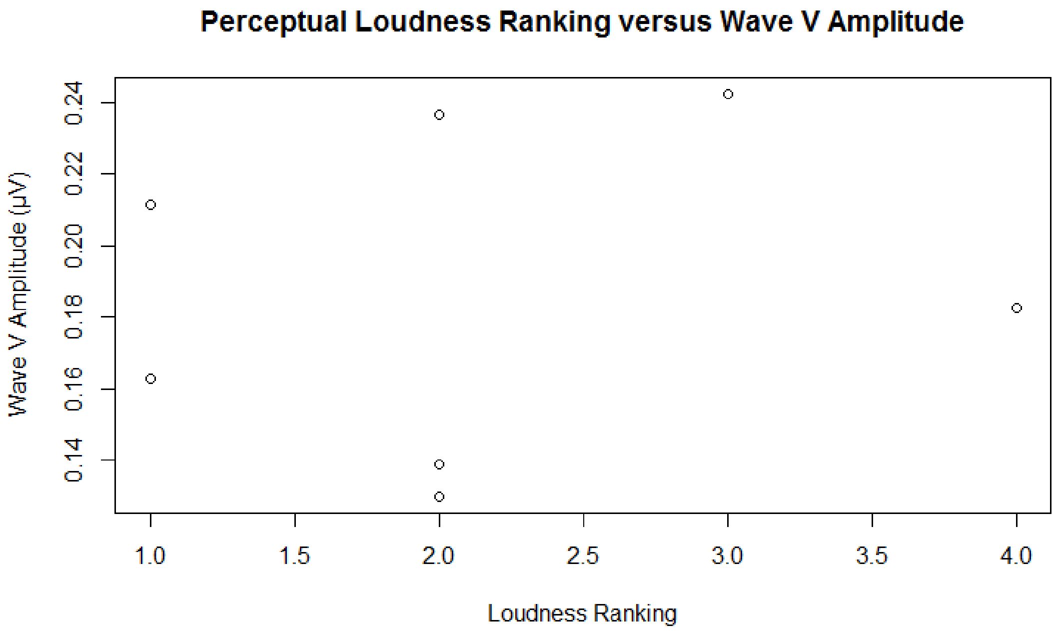

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.4
0.5
0.6
0.7
0.8
0.9
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Loudness Ranking

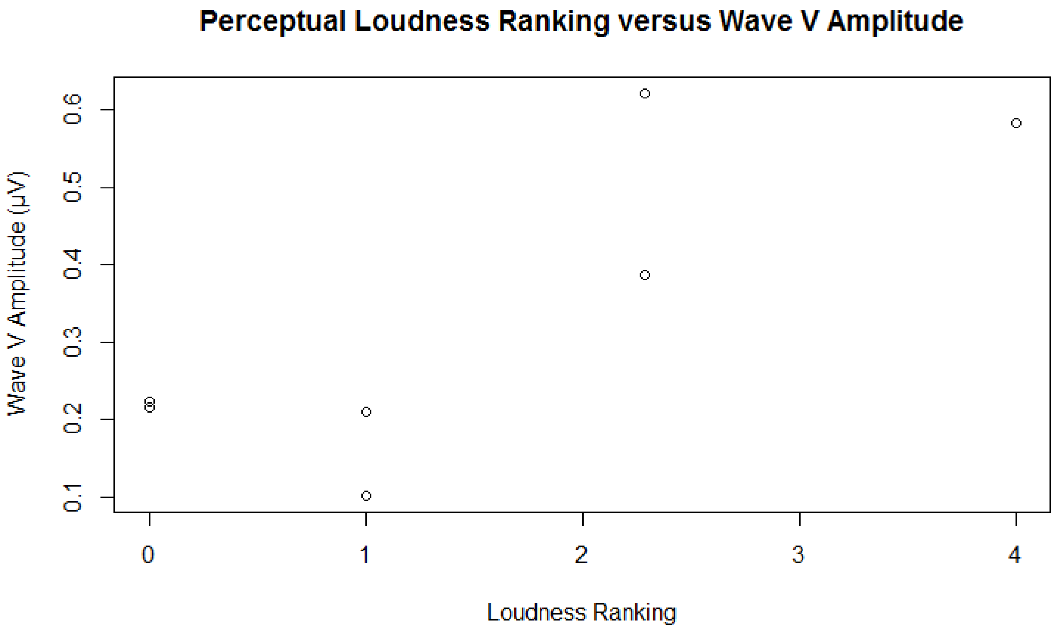

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.15
0.20
0.25
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

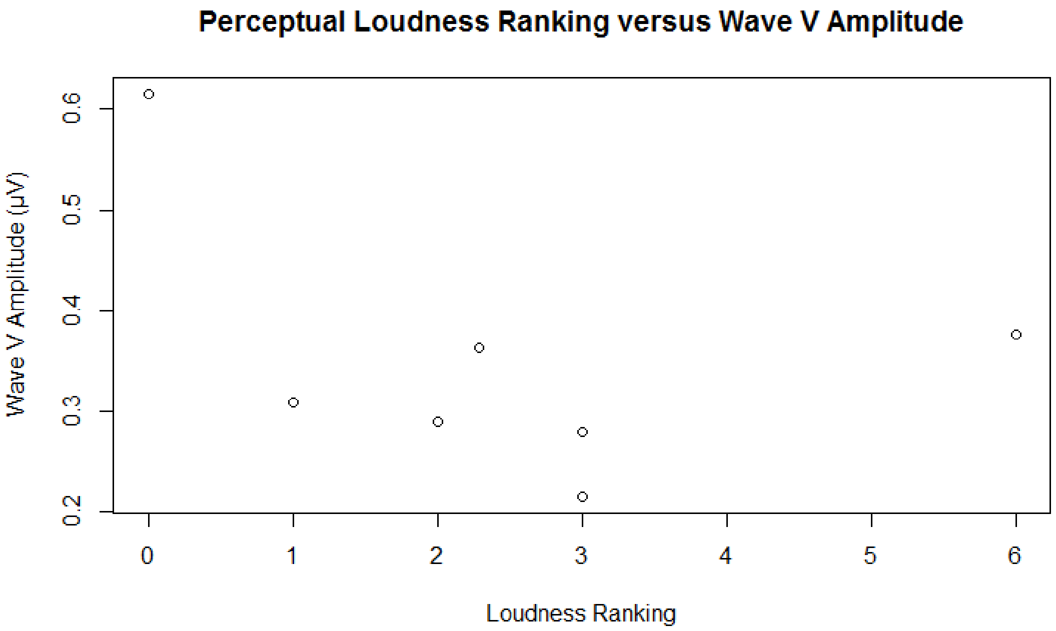

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.3
0.4
0.5
0.6
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Loudness Ranking

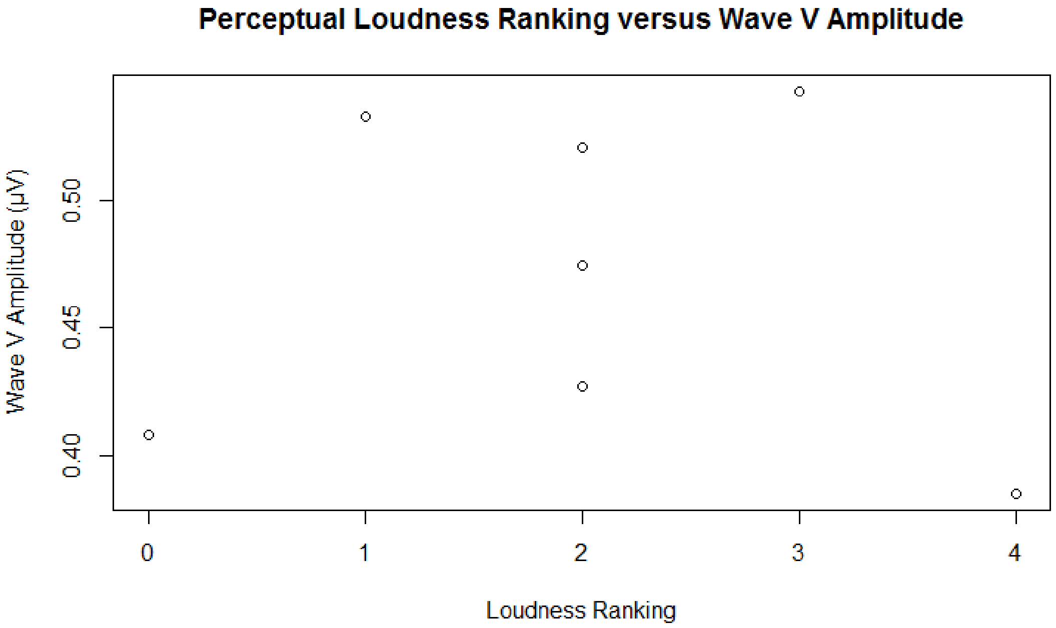

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.25
0.30
0.35
0.40
0.45
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

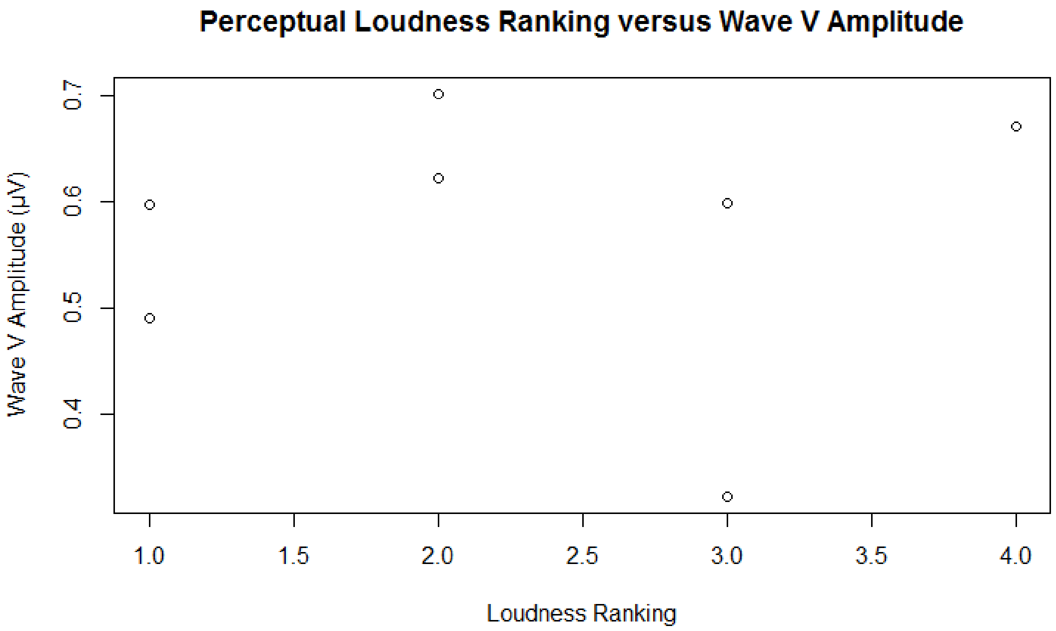

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (µV)
0.45
0.50
0.55
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

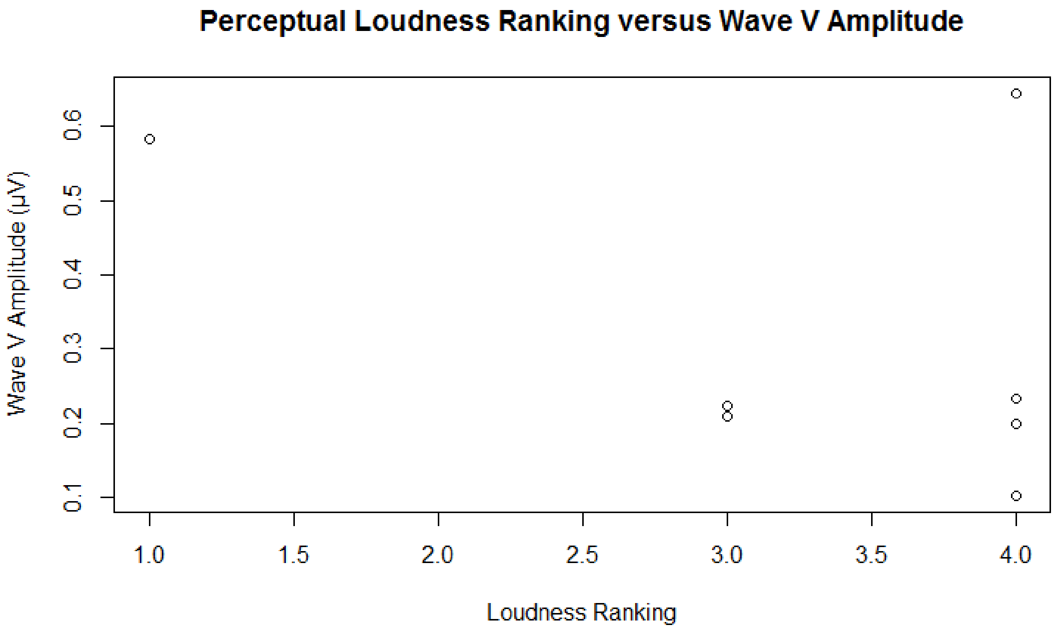

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.14
0.16
0.18
0.20
0.22
0.24
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Loudness Ranking

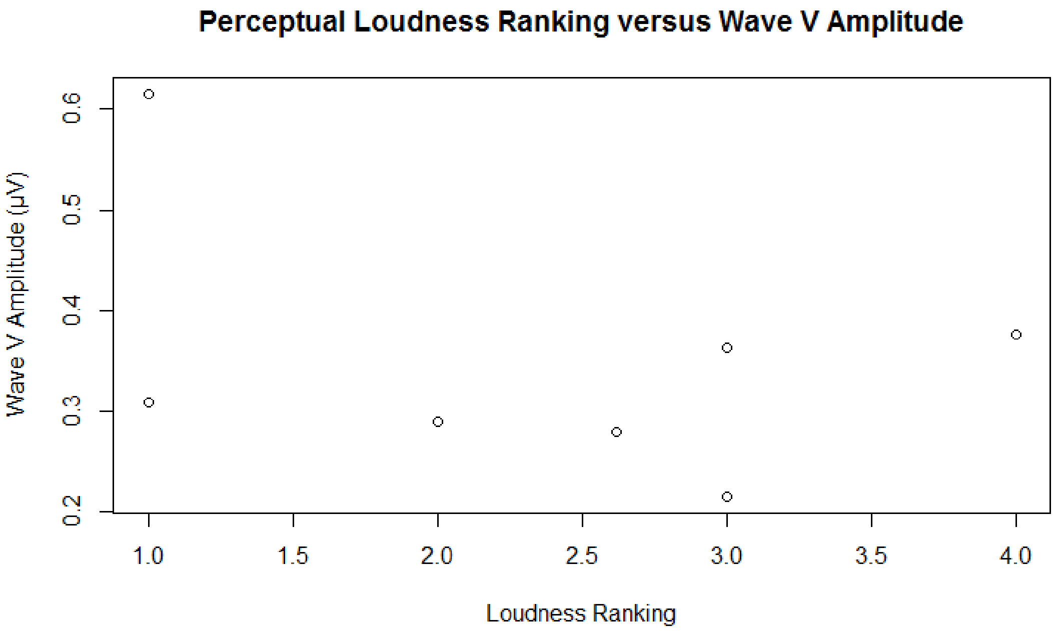

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.1
0.2
0.3
0.4
0.5
0.6
0
1
2
3
4
Loudness Ranking

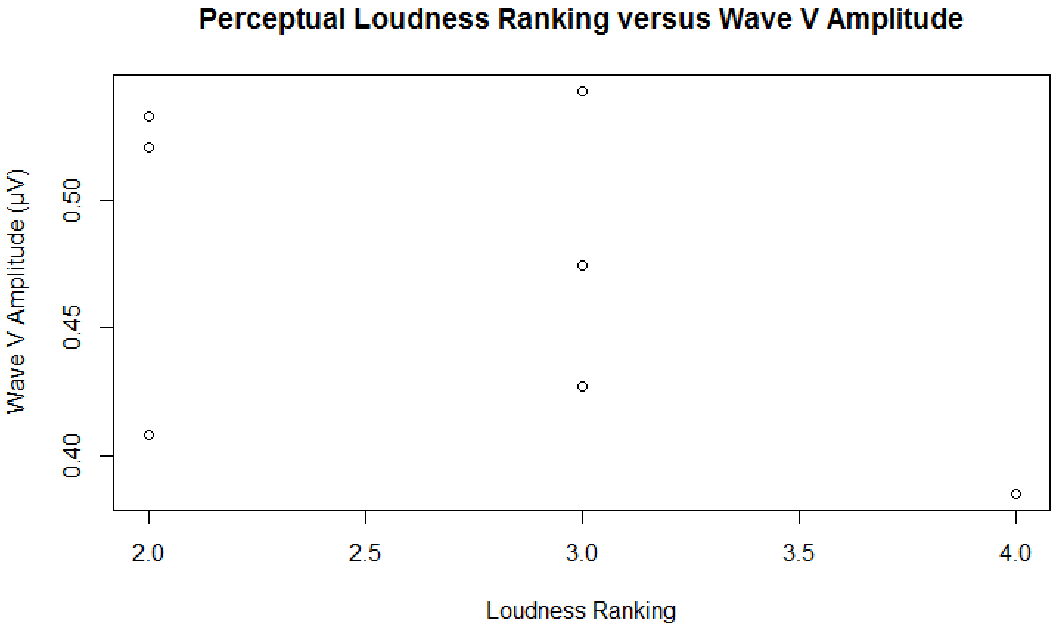

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.2
0.3
0.4
0.5
0.6
0
1
2
3
4
5
6
Loudness Ranking

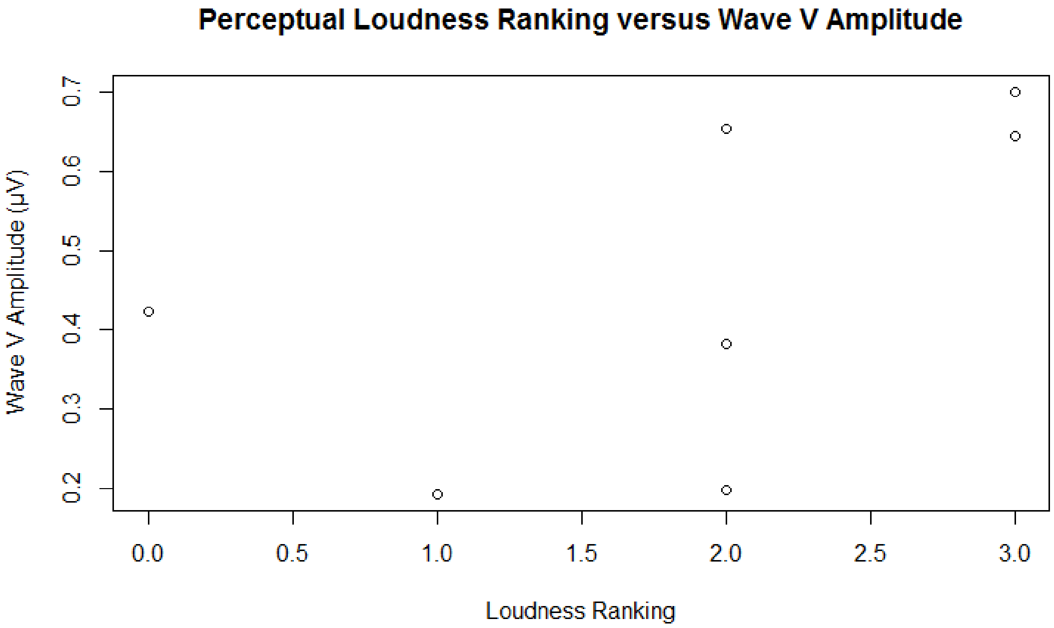

Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.40
0.45
0.50
0
1
2
3
4
Loudness Ranking


Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.4
0.5
0.6
0.7
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Loudness Ranking


Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.1
0.2
0.3
0.4
0.5
0.6
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Loudness Ranking


Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.2
0.3
0.4
0.5
0.6
1.0
1.5
2.0
2.5
3.0
3.5
4.0
Loudness Ranking


Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.40
0.45
0.50
2.0
2.5
3.0
3.5
4.0
Loudness Ranking


Perceptual Loudness Ranking versus Wave V Amplitude
Wave V Amplitude (μV)
0.2
0.3
0.4
0.5
0.6
0.7
0.0
0.5
1.0
1.5
2.0
2.5
3.0
Loudness Ranking

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