Original Article

The Correlation Between Hair and Eye Colour and Contralateral Suppression of Otoacoustic Emissions

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Abstract

Genetics and environmental factors frequently influence individual's susceptibility to hearing loss. It is postulated that melanin in the inner ear is related to individual's susceptibility to noise induced hearing loss (NIHL). General pigmentation in turn, suspected to be related to the amount of pigmentation in the inner ear. The amount of melanin in the inner ear is said to modulate the endocochlear potential and provide an otoprotective effect. **Aim:** The study aimed to determine the relationship between the contralateral suppression of otoacoustic emissions (CSOAE) in individuals with brown eyes and hair, and blue eyes and blond hair, and temporary emission shift (TES) after short-term noise exposure. **Setting and Design:** The research was conducted using a quantitative research design with a quasi-experimental repeated within the subject design to compare the CSOAE in subjects with different hair and eyes colour with TES after short-term noise exposure. Quantitative research was used to determine the relationship between the measurable variables to predict occurrence. **Material and Method:** The hearing sensitivity of young adults was determined by using pure tone audiometry followed by CSOAE's and distortion product otoacoustic emissions (DPOAE) before listening to music for one hour individually. Pure tone audiometry and DPOAE's were repeated after music exposure to determine the amount of TES and temporary threshold shift (TTS). **Statistical Analysis used:** One-way ANOVA was used during the analysis of the data obtained during this research study, in addition to, two-tailed Wilcoxon Sign Rank test and Friedman's test. In all analyses, a 95% level of significance ($P<0.05$) was used. **Results:** No statistically significant difference between efferent suppression was measured by CSOAE's between the participant groups. A larger TTS at 4000 Hz and TES at 2000 Hz was evident in the blue eyes and blond hair group after short-term music exposure. Conclusion: CSOAE's were unable to predict which group of individuals were more susceptible to NIHL after short-term noise exposure.

Keywords: Contralateral suppressions, melanin, music exposure, noise-induced hearing loss, otoacoustic emissions

INTRODUCTION

Noise-induced hearing loss (NIHL) is an irreversible hearing impairment due to a single or repeated exposure to loud sounds in the higher frequency region from 2 kHz to 6 kHz. NIHL is becoming more prevalent in the modern society and is said to affect 16% of adults worldwide.[1] The impact of hazardous noise exposure is known to cause a temporary hearing deterioration or permanent hearing damage to the sensory cells of the cochlea.[2-5] The severity of NIHL increases as the duration and intensity level increases.[5,6] The increased occurrence of NIHL due to occupational and recreational exposure led to more social and public health complications in the society.[6] Therefore, NIHL is eventually affecting the progression of age-related hearing loss due to the early damage to the cochlea. It is estimated by the World Health Organisation (WHO) (2018)[7] that 1.1 billion young adults between the ages of 12 to 35 years are at risk for developing hearing loss due to recreational noise exposure. Young adults' main source of noise exposure can be linked to discotheque noise.[5] Recreational activities such as music concerts, nightclubs, MP3 players, gyms, shooting, and more hobbies may have a potentially damaging effect on our hearing sensitivity.[1] Discotheque intensity levels at concerts and nightclubs have been recorded between 84 to

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120 dBA.[8,9] The WHO established guidelines in which noise levels at entertainment events may not exceed 100 dBA if individuals are exposed for a maximum of four hours.[10] It is important to identify factors influencing an individual's susceptibility with the increase in prevalence of NIHL. There are multiple possible factors that may influence an individual's susceptibility to NIHL and their recovery following noise exposure. These possible factors include environmental factors or genetics.[1,2,8] Melanin, that has been recognized to be present in the inner ear, is primarily influenced by genetics. The amount of pigmentation in the inner ear has been related to general pigmentation and eye colour.[11] The stria vascularis contains melanin-producing cells that play a crucial role in the production of endocochlear potentials and provides an otoprotective effect. It is suggested that hearing loss in individuals with auditory-pigmentary syndromes, namely Waardenburg syndrome and Tietz syndrome may be influenced by the lack of melanin.[12] Waardenburg syndrome and Tietz syndrome are characterized by hypopigmentation and hearing loss. The susceptibility to NIHL can be attributed to inter-individual variability which can influence the tonic activity of the auditory pathways due to melanin levels.[2,13] The underlying mechanism of pigmentation influencing the auditory system is still unclear and not fully understood. Numerous studies have referred to work of Bonaccorsi (1965) which suggested that brown-eyed individuals had more melanin in their temporal bones in comparison to blue-eyed individuals.[14-16] Thus, studies were postulated that the brown-eyed population were less susceptible to NIHL.

Melanin is postulated to play a protective role in the auditory system and against noise trauma,[11,16-18]. Melanin is known to be involved with the calcium ion homeostasis in the cochlea.[18] Calcium homeostasis is crucial for the transduction of sound. Endocochlear electrical potential is dependent upon high calcium concentrations which are required for compound action potential leading to excitatory postsynaptic potential in the auditory nerve.[18] The amount of endocochlear melanin is said to regulate the release of cochlear calcium upon noise exposure to protect the auditory system.[16] It is postulated that melanin levels in the inner ear may modulate the auditory-neural transduction process due to the role of calcium on the cochlear hair cell functioning.[12,16] Damage to the hair cells after prolonged noise exposure can occur in the absence of melanin due to higher levels of calcium in the inner ear without modulation.[16] Lower levels of melanin can influence the efficient transduction to inner hair cells and auditory nerve. Individuals with darker skin possess higher melanin levels in the cochlea compared to individuals with lighter skin, which in turn influences hearing sensitivity.[18]

Numerous research[14-16] have focused on the association between eye colour and susceptibility to NIHL. Da Costa *et al*. and Mujica-mota *et al*. reported that light-eyed people had a greater permanent hearing loss following noise exposure in comparison to brown-eyed individuals who exhibited with better hearing thresholds after prolonged noise exposure. Thus, noise exposure has a more forceful effect on individuals with lighter pigmented iris colour. Hannula *et al*.[17] findings contradicted the previous studies with results indicating that participants with a combination of dark hair and eyes were not more protected against NIHL. Their conclusion stated that individuals with brown eyes were more susceptible to hearing loss. Driscoll *et al*.[18] study found a correlation between otoacoustic emissions (OAE) and different skin pigmentation. Hood *et al*.[15] reported a relationship between the amount of melanin and susceptibility to the temporary threshold shifts. Increased auditory thresholds after noise exposure that recover gradually are known as a temporary threshold shift (TTS).[19] It is suggested that metabolic overstimulation after noise exposure may be the cause of TTS. A difference between individuals in TTS can be attributed to individual susceptibility, the duration of noise exposure as well as the intensity that the individual is exposed to.[19] Noise exposure reduces the motility of the outer hair cells (OHC) influencing hearing sensitivity[9] A direct measure of the cochlear OHC can be provided by OAEs. OAEs are an objective measurement to record changes in amplitudes, provide insight of the cochlea's pre-neural and biomechanical aspects of acoustic information which can be influenced by melanin.[20] Damage to OHC due to overexposure to noise may lead to a reduction in OAE amplitudes.[21] A response level shift after noise exposure can be observed by utilizing OAEs. To examine the relationship between different levels of melanin and OAEs, Driscoll *et al*.[18] measured more spontaneous OAEs in darker pigmentated individuals, for example, African Americans in comparison to Caucasians and Asians. Their results supported the hypothesis that the functioning of the OHC may be modulated by melanin as estimated by skin colour. Due to the effectiveness, objective and non-invasive manner of OAEs, it may be the best method of evaluating the correlation between eye and hair colour and cochlear functioning.[19,22]

The response mechanism of the auditory system can be measured with contralateral suppression of OAE (CSOAE).[22] The medial olivocochlear (MOC) bundle is known as the efferent branch of the auditory feedback system which originates in the brainstem and terminates predominantly in the OHCs of the opposite cochlea.[23,24] The MOC reflex is the suppression effect in response to acoustic stimulation and serves as a cochlear defensive mechanism from acoustic damage.[4] The MOC reflex measurement is postulated to be valuable for screening an individual's susceptibility to acoustic trauma due to weakened MOC effect for those who have a preferential susceptibility to NIHL.[25,26] The MOC efferent system has assumed roles by inhibition of cochlear amplifier gain such as aiding listening in noise, slowing age-related hearing loss and protection against acoustic overexposure.[2,4,13,23] The strength of the MOC reflex is considered to be able to predict threshold shifts.[4] The MOC reflex is activated by

acoustic stimulation and induces an inhibitory effect on OHC motility.[4,23] The protection provided by the MOC reflex can be attributed to the reduction in the interruptions of electro-mechanical transduction of the OHC.[13] The reduction in amplitude with a contralateral masker was expected as small as 1 to 4 dB SPL reduction in amplitude.[26] A noticeable shift can be measured in the OAEs before fluctuations in behavioural pure tone thresholds may be identified.[27] A temporary emissions shift (TES) may provide a better indication of the effect of noise exposure on the cochlea than other audiometric results.[4] An emission shift after noise exposure is expected to occur at 2000 to 6000 Hz.[19]

Previous studies have investigated the correlation between the amount of melanin based on eye and hair colour and the effect that it has on OHCs or amount of melanin in the temporal bones. No studies have investigated the relationship between eye and hair colour and CSOAEs after short-term noise exposure. CSOAEs can provide information about the protective function of the efferent system while OAEs provides information on whether the protective reflex maintains over time with acoustic stimulation. Numerous studies stated that an earlier indication of cochlear damage is provided by OAEs in comparison to behavioural pure tone threshold audiometry.[9,27] The study aimed to determine the relationship between the CSOAEs in individuals with different hair and eye colour, and TES after short-term noise exposure.

SUBJECTS AND METHODS

Participants

Twenty-five normal hearing adults, between the ages of 18 and 28 years were selected for the current study. All participants volunteered to participate in the study, which was approved by an institutional review board. The study consisted of twenty females and five males (Mean age: 21.64 years; SD 1.80). Participants were non-smokers, should have no history of ear diseases and in generally good health. Additional inclusion criteria were defined. A normal bilateral otoscopic examination, Jerger type A tympanograms measured with a Y-226 Hz probe tone utilising a GSI Tympstar Middle Ear Analyzer (Grason-Stadler, Eden Prairie, Minnesota). Jerger Type A tympanograms were defined as a static compliance of 0.3 to 1.75 mmho and a peak pressure of +100 to −100 daPa.[28] Normal ipsilateral and contralateral acoustic reflexes elicited at 85 to 100 dB SPL at frequencies 0.5, 1, 2 and 4 kHz. Furthermore, pure tone thresholds were obtained using the modified Hughson-Westlake procedure[29] on a GSI 61 clinical audiometer with supra-aural TDH-39 headphones (Grason-Stadler, Eden Prairie, Minnesota). Participants were selected based on normal behavioural pure tone air conduction thresholds $\leq$15 dB HL at octave intervals from 125 to 8000 Hz and including half octave frequencies of 3000 and 6000 Hz were included. Speech detection threshold must be within normal limits of the pure tone averages (PTA) of 0.25 to 2 kHz. All testing was conducted in a double-walled soundproof booth. Participants were grouped into two categories: group A consisted of individuals with brown eyes and hair, and group B was individuals with blue eyes and blond hair. In the study, 47% of the participants had brown eyes and brown hair and 53% of the subjects had blue eyes and blond hair.

Baseline testing

Baseline testing of pure tone audiometry, CSOAE and distortion product otoacoustic emissions (DPOAE) precede a one hour by music exposure session at 90 dBA. After the exposure, DPOAE measurements and pure tone audiometry were repeated. All testing was conducted in a double-walled soundproof booth.

Otoacoustic emissions

An Otodynamics DP Echoport ILO V6 was used for both CSOAE and DPOAE measurements. Probe calibration was performed at the beginning of each session using a 1 cm^3 calibration cavity. For CSOAE measurements, a linear mode of stimulation was used with a rate of 50 clicks/sec using an 80μs rectangular electrical pulse. Clicks were evoked with an intensity of 65 dB SPL with and without masker in the contralateral ear. The contralateral stimulus consisted of a continuous broadband noise presented at a stimulus level of 60 dB SPL to elicit efferent activity.[30] The noise rejection level was set at 49.5 dB SPL.[31] To evaluate the level of suppression, transient evoked OAE (TEOAE) responses without a broadband noise were measured followed by a measurement with contralateral broadband noise stimulation. The absolute TEOAE suppression was determined by subtracting the amplitude with contralateral stimulation from the amplitude with contralateral stimulation in each ear specifically.[32] The obtained TEOAE response levels and noise amplitudes were analysed in half-octave frequency bands centred at 1, 1.4, 2, 2.8 and 4 kHz. To determine if whether a TEOAE response was present, the following factors relating to the recording parameters were taken into consideration.[25,31] Measurements were present when a stimulus stability of 90% and a reproducibility of 75% were present. Signal to noise ratio (SNR) must have been $\geq$ 6 dB.[25] Following CSOAE measurements, ipsilateral $2f_1$-f_2 DPOAEs were evoked by stimulation generation of two primary tones (f1 and f2) with an f2/f1 frequency ratio of 1.22 and f2 ranging from 1001 to 7996 Hz. The stimulus tone level combination L1/L2 was set to 65/55 dB SPL, to ensure that the optimal SNR was achieved.[33] The noise rejection level was set at 49.5 dB SPL.[21] The obtained DPOAE responses were converted and reported into half-octave frequency with the center frequencies at 1, 2, 3, 4, 6, 7.96 kHz. DPOAE results were deemed present if four or more frequencies had a DP amplitude exceeded the noise floor by 6 dB, therefore an SNR of $\geq$ 6 dB SPL.[34]

Music exposure

Following the baseline measurements, each participant was exposed to continuous discotheque music set at an intensity of 90 dBA for one hour in a double-walled soundproof booth. A sound level meter (RION NA-42) was placed at ear level of the participants to monitor noise in the room. The noise exposure LAeq dBA level over the complete duration of the music sample was measured at 90 dBA. The music was presented by using an ASUS K541U laptop that was connected to the GSI 61 Clinical Audiometer (Grason-Stadler, Eden Prairie, Minnesota) to ensure the intensity was kept consistently at 90 dBA. The music was transduced through free-field via the audiometer's two GSI speakers with the participants seated one meter from the two speakers mounted in the front corners of the booth.

Post-exposure measurements

Post-exposure measurements commenced within five minutes after the end of music exposure. Ipsilateral DPOAE measurements and pure tone audiometry were repeated after the music exposure, which served as the post-exposure measurements. Post-exposure DPOAE responses and pure tone thresholds were subtracted from the baseline data to calculate the difference between measurements. The difference between the pre and post of DPOAE amplitudes constitutes the TES and for pre- and post-pure tone thresholds, the TTS.

Statistical analysis

The Statistical Package for the Social Sciences (SPSS) version 25 was used for the statistical analysis of all data. Amplitudes, SNR and audiometric results were described using descriptive statistics to determine the mean values and standard deviations. The Shapiro Wilk test was used to evaluate normality of distribution. If the results were normally distributed, a paired sample t-test was used to compare differences in the mean values between DPOAE and CSOAE response level, and SNR as well as audiometric shift after music exposure of the two groups. One-way ANOVA was used to analyse the influence of two categorical independent variables on one dependent variable, namely the DPOAE response level between the two groups.[35] If the results were not normally distributed, the data were analysed using the two-tailed Wilcoxon Sign Rank test and Friedman's test of analysis of variance. Wilcoxon Sign Rank test was used to compare repeated measurement in a single sample, DPOAE and pure tone results before and after music exposure. Friedman's test of analysis of variance was used to determine if there was a statistically significant difference between the distribution of three or more related groups of CSOAE frequencies. All results were categorized according to individual-related factors namely gender (female's vs males), ear (left vs right) and hair and eye colour (brown eyes and hair vs blue eyes and blond hair). In all analyses, a 95% level of significance ($P < 0.05$) was used.

RESULTS

Impact of hair and eye colour on the contralateral suppression of transient evoked otoacoustic emissions

Table 1 displays the mean absolute TEOAE amplitude with noise and amount of suppression per test frequency for participants with brown eyes and hair, and for participants with blue eyes and blond hair.

The mean absolute efferent suppression decreases from the mid frequencies towards the higher frequency region in all participants. The two groups exhibited the same degree of efferent suppression with similar mean suppression at each frequency. The mean efferent suppression decreased in the higher frequencies from 2.8 kHz in comparison to the lower frequency region. No statistically significant difference in suppression could be measured between the two groups ($z = -0.30$–1.60; $P > 0.05$). However, a significant difference was observed between the degree of suppression between the individual test frequencies.

A Friedman's analysis of variance (ANOVA) with Bonferroni correction of the difference suppression values at different test frequencies in the total participants group indicated a statistically less suppression for 4000 Hz compared to 2828 Hz ($P < 0.03$) and highly significant difference between the following frequencies: 4000 and 2000 Hz ($P < 0.001$); 4000 and 1001 Hz ($P < 0.001$); 4000 and 1414 Hz ($P < 0.001$); 2828 and 2000 Hz ($P < 0.001$); 2828 and 1001 Hz ($P < 0.001$) and 2000 and 1001 Hz

Table 1: The mean efferent contralateral suppression of otoacoustic emissions (CSOAE) (dB SPL) per frequency for group with brown eyes and hair and group with blue eyes and blond hair

Frequency (Hz)	1001	1414	2000	2828	4000
Brown eyes and brown hair ($n = 24$ ears)					
Absolute TEOAE amplitude with noise(dB SPL ± SD)	8.34 ±4.04	9.67 ±3.30	6.84 ±3.13	4.26 ±4.63	0.94 ±4.84
Amount of suppression (dB)	0.46	0.53	0.54	0.57	0.38
Blue eyes and blond hair ($n = 26$ ears)					
Absolute TEOAE amplitude with noise(dB SPL ± SD)	7.50 ±5.84	7.52 ±4.35	7.57 ±3.61	4.74 ±3.64	1.23 ±5.98
Amount of suppression (dB)	0.48	0.54	0.38	0.44	0.29

CSOAE, contralateral suppression of otoacoustic emissions; dB, decibel; dB SPL, decibel sensation level; Hz, frequency; SD, standard deviation; %, percentage.

Table 2: Mean pure tone thresholds in participants with brown eyes and hair and participants with blue eyes and blond hair after music exposure at each frequency ($n = 50$ ears)

Frequency (Hz)	125	250	500	1000	2000	3000	4000	6000	8000
Brown eyes and hair (n = 24 ears)									
Baseline pure tone threshold (dB HL ±SD)	1.25±2.66	1.87 ±3.55	1.04±2.54	0.42±1.41	0.83±2.40	0.42 ±1.41	0.42 ± 1.41	1.46± 3.12	0.83 ±2.82
Pure tone threshold after music exposure (dB HL ± SD)	1.87±3.55	2.5 ±3.10	1.46±2.75	0.63±1.69	0.63±1.69	0.42±1.41	1.25±2.66	1.86 ±2.88	1.25 ±3.69
Threshold shift (dB)	**0.63**	**0.63**	**0.42**	**0.21**	**0.21**	**0**	**0.83**	**0.42**	**0.42**
Blue eyes and blond hair (n = 26 ears)									
Baseline pure tone threshold (dB HL ±SD)	1.92 ±3.49	0.77 ± 1.84	0.96 ± 2.83	0.58 ± 1.63	0.38 ±1.36	0.38 ±1.36	0.96 ±2.83	3.27 ±3.99	2,12 ±3.21
Pure tone threshold after music exposure (dB HL ± SD)	2.31 ±4.30	1.54 ±2.75	1.35 ±4.14	0.58 ±2.16	1.54 ±3.68	1.35 ±2.68	4.04 ±4.69	5.00 ±5.48	3.65 ±5.20
Threshold shift (dB)	**0.38**	**0.77**	**0.38**	**0**	**1.15**	**0.96**	**3.08 ***	**1.73**	**1.54**

dB HL, decibel hearing level; Hz, frequency; SD, standard deviation; * indicative of significance

Table 3: Mean baseline and post-exposure distortion product otoacoustic emissions (DPOAE) signal to noise ratio (SNR) in all participants ($n = 50$ ears)

Frequency (Hz)	1001	1501	2002	3003	4004	6006	7996
Mean baselineSNR (dB SPL ±SD)	16.52 ±5.67	20.9 ±5.23	20.9 ±4.72	18.05 ±4.55	21.36 ±4.32	21.70 ±6.07	11.06 ±10.8
Mean post-exposureSNR (dB SPL ±SD)	12.43 ±6.78	18.35 ±6.26	18.65 ±6.18	15.61 ±5.12	18.38 ±4.52	19.12 ±7.41	5.67 ±14.32
Mean difference betweenbaseline andpost-exposureSNR (dB SPL ±SD)	4.09 ±2.89*	2.55 ±1.80*	2.25 ±1.59*	2.44 ±1.73*	2.98 ±2,10*	2.58 ±1.82*	5.81 ±3,11*

dB SPL, decibel sensation level; DPOAE, distortion product otoacoustic emissions; Hz, frequency; SD, standard deviation; SNR, signal to noise ratio; * indicative of significance

Table 4: The mean distortion product otoacoustic emissions (DPOAE) half octave response level shift after music exposure in participants with brown eyes and hair and participants with blue eyes and blond hair ($n = 50$ ears)

Frequency (Hz)	1001	1501	2002	3003	4004	6006	7996
Brown eyes and brown hair (n = 24 ears)							
TES(dB SPL ±SD)	1.64±2.74	1.61±2.07	1.07±2.5	1.90 ±2.16	2.35 ±2.72	2.65 ±2.52	4.44 ±8.96
Blue eyes and blond hair (n = 26 ears)							
TES(dB SPL ±SD)	2.69 ±2.70	1.99 ±2.05	2.69 ±2.52*	1.85 ±2.12	3.06 ±2.72	2.23 ±2.48	4.16 ±8.80

dB SPL, decibel sensation level; Hz, frequency; SD, standard deviation; TES, temporary emission shifts; * indicative of significance

($P < 0.001$). The other frequencies did not exhibit a statistical significance ($P > 0.05$) at 2000 and 1001 Hz, 2000 and 1414 Hz and 1001 and 1414 Hz.

The mean efferent suppression was symmetrical between the left and right ears did not indicate a statistical significance ($z = -0.22$–0.66; $P > 0.05$).

Differential impact of short-term music exposure on pure tone audiometric thresholds

Table 2 provides the baseline measurements in pure tone thresholds, post-exposure pure tone thresholds, and the TTS in the brown eyes and brown hair participants and blue eyes and blond hair participants.

The mean PTA before and after noise exposure were the same in participants with brown eyes and hair, and participants with blue eyes and blond hair, namely 1.00 dB HL. The participants with blue eyes and blond hair had a larger mean pure tone threshold in the higher frequencies from 2000 to 8000 Hz after the music exposure in comparison to participants with brown eyes and hair. The blue eyes with blond hair group displayed a significant larger TTS from baseline measurement to post-exposure at 4 kHz ($z = -2.17$; $P < 0.05$). The brown eyes with brown hair group indicated a mean TTS of 0.83 dB HL which is smaller than the participants with blue eyes with blond hair group with a mean TTS of 3.00 dB HL. The mean TTS at all other frequencies between the two groups exhibited no statistically significant differences ($z = -0.07$–1.87; $P > 0.05$).

The mean TTS at 4 kHz was much larger in the left ear than in the right ear but was not statistically significant ($z = -1.46$; $P > 0.05$). The mean TTS did not indicate a significant difference between the left and right ear at frequencies 125 to 8000 Hz ($z = -0.20$–1.75; $P > 0.05$).

Differential impact of short-term noise exposure on distortion product otoacoustic emissions

Table 3 displays the mean baseline SNR measurement, post-exposure SNR measurement and reduction in SNR at each frequency in all the participants.

The mean DPOAE SNR reduction indicated a highly significant difference ($t = 3.73–6.93$; $P < 0.001$) at each frequency. The mean TES also indicated a highly significant difference at each frequency in all participants ($t = 3.46–7.08$; $P < 0.001$). The difference in the mean TES is shown in Table 4 between participants with brown eyes and hair, and participants with blue eyes and blond hair from 1001 to 7996 Hz *(* indicative of significance).*

The Wilcoxon signed rank test indicated a statistically significant difference in TES at 2 kHz between the two groups ($z = -1.05$; $P < 0.05$) with a mean shift of 2.69 dB SPL. At the other frequencies tested the TES from baseline to post-exposure did not indicate statistically significant difference ($z = -0.30–2.41$; $P > 0.05$) between the participants with brown eyes and hair, and participants with blue eyes and blond hair. A significant difference between groups was also displayed for the SNR of the DPOAEs at 2 kHz ($F = 4.77$; $P < 0.05$).

The baseline measurement and post-exposure measurement of TES indicated symmetrical shifts between the left and right ear. For each of the frequencies, there was no statistically significant difference between the left and right ears ($z = -0.14–1.55$; $P > 0.05$).

Discussion

The current study explored the relationship between CSOAEs of individuals with blue eyes and blond hair, and brown eyes and hair, and their TES after one hour of music exposure at 90 dBA. Statistically significant TTS and TES were recorded after music exposure in individuals with blue eyes and blond hair, in comparison to individuals with brown eyes and hair. No significant difference in efferent suppression was observed between participants with brown eyes and hair, and participants with blue eyes and blond hair.

Differential impact of short-term music exposure on pure tone audiometric thresholds

The present study found a mean TTS ranging from 0 to 0.83 dB HL in the brown eyes and hair, and 0 to 3.08 dB HL in the blue eyes and blond hair. Pure tone audiometry has been widely used to evaluate the correlation between iris pigmentation and NIHL in previous studies.[14,17] The current study found that participants with blue eyes and blond hair showed a statistically larger TTS, by 2.25 dB HL, at 4 kHz after music exposure than the participants with brown eyes and hair. The reduction in pure tone threshold at 4 kHz is typical of NIHL and was evident in the blue eyes and blond hair group after music exposure. The current study did not find significant differences between groups at other test frequencies other than 4000 Hz. Previous studies found significant TTS at 2000 to 6000 Hz in comparison to the current study, which only recorded a significant TTS at 4000 Hz. The results of the current study did not suggest that music exposure of one hour at 90 dBA causes a significant TTS at each frequency within the noise-sensitive region of 2000 to 6000 Hz. Bhagat and Davis[36] found that in noise-exposed individuals that the audiometric results were not indicative of noise trauma after 30 minutes of noise exposure and were rather revealed in OAE results. Significantly better PTA have been recorded in dark-eyed individuals in comparison to light-eyed individuals after noise exposure.[14,16] Dark-eyed individuals have been reported to have from 1 to 5 dB HL better hearing thresholds in the noise susceptible frequency region.[14] Previous studies based their conclusion on individuals with long-term noise exposure, ranging from two to 42 years.[14,16] Although the current study used short-term music exposure with participants with normal hearing, the same trend was observed in which the individuals with dark eyes and hair had better hearing thresholds after short-term music exposure. Therefore, it is suggested that the blue-eyed and blond-haired may be more susceptible to acoustic overstimulation even after only a short-term noise exposure.

Differential impact of short-term noise exposure on distortion product otoacoustic emissions

Due to the effectiveness of OAEs and the objectivity it provides, most studies including the present study used OAEs in addition to pure tone audiometry. Studies utilized DPOAE to measure changes in amplitudes after noise exposure.[9,37,38] The current study measured significant differences in all participants' TES and DPOAE SNR at each frequency. A reduction of 2.25 to 5.81 dB SPL was measured in the DPOAE SNR in all participant in the current study with the largest shifts at 4004 to 7996 Hz. Although pure tone audiometry did not display the same results after one-hour noise exposure at each frequency, DPOAEs were able to monitor slight changes in the amplitudes of emissions after noise exposure. The present study found a significantly larger mean TES of 2.69 dB SPL at 2000 Hz in participants with blue eyes and blond hair than in participants with brown eyes and hair. A study reporting on DPOAEs after exposure to impulse noise, reported a significant shift at 4004 to 7996 kHz while the lower frequencies remained stable.[39] Studies have described a trend of TES at 3049 to 7996 Hz after acoustical overstimulation.[3,9] Research investigating noise exposure and TES varied from an exposure intensity of 85 to 103 dB SPL for 30 minutes to five hours.[9,36,38] The degree of shift is highly dependent on the intensity and duration of exposure. Individuals have shown mean TES of 7 to 10 dB SPL after exposure to broadband noise, music in nightclubs and MP3 players.[9,15,21] The current study observed a smaller TES after music exposure in comparison to previous research although the affected frequency region correlated.[9,21] The

larger TTS and TES in the blue eyes and blond hair group were only measured at one test frequency per test, namely a TTS at 4kHz and a TES at 2 kHz.

Mujica-mota *et al*.[16] reviewed numerous studies and concluded that although differences between lighter and darker pigmented individuals were noted, it is considered only a modest risk factor. Studies have suggested that individuals with lighter pigmentation were only at risk when exposed to high noise levels over a prolonged period of time.[14,15,17] The differences in results and degree of shifts between the blue-eyed and blond-haired, and the brown-eyed and haired TTS and TES can be attributed to the following three reasons. Firstly, the intensity of the noise exposure in previous studies was greater in comparison to the present study using a 90 dBA exposure level. A louder exposure level may cause larger shifts in the same duration of exposure. Secondly, the duration of noise exposure of previous studies was five to six hours or even years in comparison to the single hour in the present study. According to the equal energy rule, when the intensity increases by three dB it leads to the doubling in sound energy and the reducing duration in half which is acceptable to be exposed to.[40] As soon as the intensity becomes too loud or duration longer, it may cause larger shifts and more permanent damage. Due to the reduced duration, despite similar exposure levels, although shifts were recorded, it was much smaller in comparison to previous studies. Lastly, the TES and TTS can be attributed to inter-individual variability.

There is an agreement between studies that intrinsic and extrinsic factors influence individual's susceptibility and auditory health.[2,3,13,14,17,36] The differences in TTS and TES between individuals with blue eyes and blond hair, and brown eyes and hair may be attributed to an intrinsic factor such as pigmentation that is influenced by genetics. The present study supports the hypothesis of Bonaccorsi (1965) that there may be a correlation between the amount of pigmentation in the iris and temporal bone due to the visible difference between the blue eyes and blond hair and brown eyes and hair in the current study. Although differences between the blue eyes and blond hair, and the brown eyes and hair groups are seen, the results are unlikely to be attributed to pigmentation alone. Factors such as other genetic and environmental factors should not be excluded when examining individuals' susceptibility to NIHL.

Impact of hair and eye colour on the contralateral suppression of transient evoked otoacoustic emissions

The current study aimed to determine whether a correlation existed between CSOAEs and TES and TTS after music exposure at 90 dBA in participants with more pigmentation in comparison to participants with less pigmentation. The current study could not identify a difference in efferent suppression as measured by CSOAEs in participants with brown eyes and hair, and participants with blue eyes and blond hair. The present study, however, did observe stronger efferent suppression from 1 to 2 kHz in all participants. This finding correlated with that of Otsuka *et al*.[4] who recorded stronger efferent suppression from 1 to 3 kHz in comparison to the higher frequency region in young adults with normal hearing sensitivity. Therefore, the results suggest that MOC efferent system may be stronger at 1 to 2 kHz and weaken in the NIHL sensitive area.[2] The current study observed the same trend of decrease in CSOAE amplitude at ≥2.8 kHz with a larger TES measured at 2000 Hz in individuals with less pigmentation compared to those with more pigmentation. The TES was only observed in the group with blue eyes and blond hair although their amount of efferent suppression during CSOAEs was equivalent between the two groups. A previous study used CSOAEs to measure the MOC reflex to assess the risk of hearing loss among orchestra musicians which indicated significant TTS at 4000 Hz as well as a decrease in efferent suppression at 4000 Hz.

Impact of hair and eye colour on the contralateral suppression of transient evoked otoacoustic emissions and distortion product otoacoustic emissions

The aim of the current study was to determine if CSOAEs will be able to predict if individuals with different hair and eye colour will be more susceptibility to short-term noise exposure and that the differences will therefore be exhibited in their TES and TTS. The results of the current study found no evidence that CSOAE's are able to predict the TES in DPOAE after short-term music exposure. However, individuals with blue eyes and blond hair did indicate larger TES and TTS after acoustical overstimulation in comparison to the brown eyes and hair. Therefore, less pigmented individuals presented to be more susceptible to NIHL than more pigmented individuals. Numerous studies have concluded the inability of the CSOAE to reliably predict TTS or TES.[3,4] It is postulated that MOC reflex may provide a protective factor in non-traumatic sound and that the role of the MOC reflex is still vague.[41] The current study was therefore unable to identify a relationship between the strength of efferent suppression, TTS and TES after music exposure in individuals with blue eyes and blond hair and individuals with brown eyes and hair. However, the present study effectively utilized OAEs and pure tone audiometry to measure differences between blue eyes and blond hair and brown eyes and hair after acoustic overstimulation.

A limitation of the current study was that pigmentation was quantified based on hair and eye colour determine the susceptibility to NIHL. Thus, limiting the ability to determine the effect of ethnicity/race on susceptibility to NIHL. The current study aimed to determine the correlation between CSOAE's in different hair and eye colour, and their TES after music exposure, therefore, purposefully excluding race/ethnicity to effectively control variables. Previous studies have investigated the effect of ethnicity/race on hearing loss by using OAEs and pure tone audiometry.[42,43] The differences between ethnicities were

seen in the noise susceptible region of 4000 to 8000 Hz, regardless of gender.[43] It is acknowledged that individuals from different races and ethnicities have a differing predisposition for NIHL. Pigmentation distribution to skin, hair, and eyes are influenced by genetics and a lack of pigmentation have been observed in genetic disorders.[44] The current study excluded race/ethnicity that is also influenced by genetics to have less variables influencing current study outcome. Pigmentation syndromes have indicated that there is a correlation between skin colour and sensorineural hearing loss.[45] Therefore, race/ethnicity should not be excluded when investigating the effect of pigmentation on hearing sensitivity after noise exposure due to its possible influence on individuals' susceptibility to NIHL.

Research regarding genetic factors influencing individual's susceptibility to NIHL has been limited due to the inability to determine cumulative recreational noise exposure across a lifetime. Multiple factors such as duration of noise exposure, intensity of exposure, and even length of ear canal and exposure to sunlight have been postulated to have an effect on the relationship between pigmentation and individuals' susceptibility to NIHL.[46] Although it was beyond the scope of the current article it should be explored in future studies using a pre- and post quasi-experimental design in simulated and real-world noise-exposed individuals. NIHL is becoming more prevalent in the modern society and is preventable. Previous studies mainly focused on acoustic overstimulation in a laboratory or occupational setting rather than a recreational setting.[13] Future research should focus on monitoring the effect of recreational noise exposure on individual's hearing sensitivity. Individuals are differently susceptible to NIHL, therefore, some individuals may have more resilient ears in comparison to others against noise exposure. The underlying mechanism of this phenomenon is not yet understood. The use of OAEs allows the objective and non-invasive monitoring of changes in OHCs when measured directly after exposure to identify individuals that are more susceptible to NIHL.[39] Inter-individual susceptibility should not be underestimated, and awareness should be created to identify possible risk factors to NIHL. The current study population included normal hearing young adults with less cumulative exposure to occupational and recreational noise. Identifying the influencing factors of inter-individual susceptibility will aid audiologists to better prevent and treat individuals susceptible to NIHL. Efforts should be made to better understand the underlying mechanisms influencing NIHL.

CONCLUSION

No clear relationship was determined between CSOAEs of individuals with blue eyes and blond hair, and brown eyes and hair, and their TES after one hour of music exposure. There was a significant TES at 2000 Hz in the individuals with blue eyes and blond hair after noise exposure in comparison to individuals with brown eyes and hair. In addition, a larger TTS was measured at 4000 Hz in the individuals with blue eyes and blond hair in comparison to the individuals with brown eyes and hair. No statistically significant difference between the two participant groups could be measured with CSOAEs. Therefore, CSOAEs were unable to predict which group of individuals were more susceptible to NIHL after music exposure. To effectively identify factors that influence individual susceptibility to NIHL, more variables must be identified and monitored over a longer exposure duration.

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Conflicts of interest

There are no conflicts of interest.

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