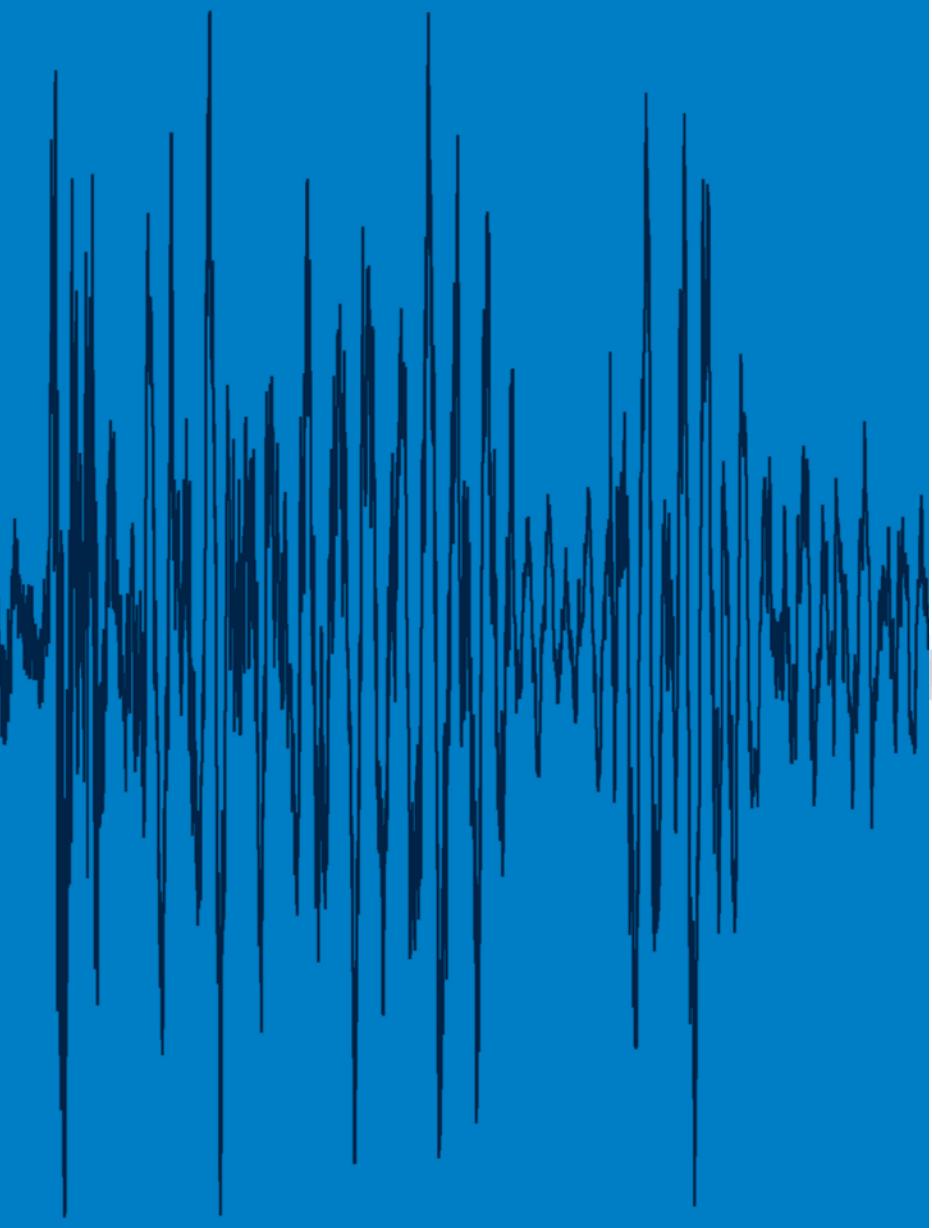
ISSN 1463-1741



Noise & Health

A Bi-monthly Inter-disciplinary International Journal

www.noiseandhealth.org

March-April 2014 | Volume 16 | Issue 69

Wolters Kluwer Health | Medknow

Evaluation of the olivocochlear efferent reflex strength in the susceptibility to temporary hearing deterioration after music exposure in young adults

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Abstract

The objective of the current study was to evaluate the predictive role of the olivocochlear efferent reflex strength in temporary hearing deterioration in young adults exposed to music. This was based on the fact that a noise-protective role of the medial olivocochlear (MOC) system was observed in animals and that efferent suppression (ES) measured using contralateral acoustic stimulation (CAS) of otoacoustic emissions (OAEs) is capable of exploring the MOC system. Knowing an individual's susceptibility to cochlear damage after noise exposure would enhance preventive strategies for noise-induced hearing loss. The hearing status of 28 young adults was evaluated using pure-tone audiometry, transient evoked OAEs (TEOAEs) and distortion product OAEs (DPOAEs) before and after listening to music using an MP3 player during 1 h at an individually determined loud listening level. CAS of TEOAEs was measured before music exposure to determine the amount of ES. Regression analysis showed a distinctive positive correlation between temporary hearing deterioration and the preferred gain setting of the MP3 player. However, no clear relationship between temporary hearing deterioration and the amount of ES was found. In conclusion, clinical measurement of ES, using CAS of TEOAEs, is not correlated with the amount of temporary hearing deterioration after 1 h music exposure in young adults. However, it is possible that the temporary hearing deterioration in the current study was insufficient to activate the MOC system. More research regarding ES might provide more insight in the olivocochlear efferent pathways and their role in auditory functioning.

Keywords: Contralateral acoustic stimulation, distortion product otoacoustic emissions, efferent suppression, transient evoked otoacoustic emissions

Introduction

It is well-known that excessive noise exposure can lead to temporary, as well as permanent hearing damage due to metabolic and/or mechanical cochlear changes.[1] Occupational noise exposure probably contributes to 5-10% of the hearing loss burden in the USA, whereas a similar impact of non-occupational noise exposure is suggested.[2] Therefore, noise-induced hearing loss (NIHL) is one of the most preventable causes of hearing loss and its early detection is crucial in hearing conservation programs. However, a large inter-subject variability in susceptibility to NIHL has been observed in the laboratory and demographic studies.[3] Identifying subjects with high vulnerability to inner-ear damage would optimize early interventions and prevent NIHL.

Access this article online	
Quick Response Code:	Website: www.noiseandhealth.org
	DOI: 10.4103/1463-1741.132094

Otoacoustic emissions (OAEs) are suggested as a promising tool in the early detection of subtle cochlear damage caused by noise exposure.[4,5] The activity of outer hair cells (OHCs), which are vulnerable to excessive noise exposure,[6,7] is directly reflected by OAEs. Therefore, low-level or absent OAEs may serve as preclinical indices of inner-ear damage.[8] Furthermore, OAEs can be used to explore non-invasively the olivocochlear efferent auditory system by applying binaural, ipsilateral or contralateral acoustic stimulation (CAS).[9,10]

Efferent cochlear innervation is provided by the olivocochlear bundle (OCB) arising from the superior

olivary complex.[11] Activation of the medial olivocochlear (MOC) bundle has an inhibitory effect on OHC motility,[12-14] suppressing the gain of the cochlear amplifier.[15] However, the exact physiological role of the MOC system is not yet completely understood. It is probably involved in low-level signal detection in noise and auditory adaptation thereby improving the signal-to-noise ratio (SNR),[16-18] and protection against noise. However, it was hypothesized that this protection is only an epiphenomenon from the inherent suppressive nature of the MOC bundle to select relevant acoustic signals from background noise.[19] Nevertheless, the olivocochlear efferent reflex strength measured using CAS of OAEs is suggested as a tool for the evaluation of susceptibility to NIHL.

Several animal studies have demonstrated that electrical or acoustical stimulation of the MOC bundle decreases the amount of temporary threshold shifts (TTS)[20-24] and MOC bundle sectioning increases the amount of permanent threshold shifts (PTS) after noise exposure.[25-29] It was found that this effect was dependent on frequency[23] and on noise exposure conditions producing the largest amount of TTS.[21] However, it must be noted that other studies do not support the role of the MOC system as protective against noise probably due to methodological considerations,[30,31] or suggest the role of slow efferent responses in the protective effect.[32,33] Nevertheless, it was found that the olivocochlear efferent reflex strength and PTS magnitude are negatively correlated and thus provide a noninvasive test to measure susceptibility to NIHL.[34]

In humans, literature regarding the protective effects of the MOC system against noise exposure is limited and more research is needed to gain insight in the ability of the olivocochlear efferent reflex strength, as measured using CAS of OAEs, to predict cochlear susceptibility to noise exposure. Therefore, the aim of the present study was to evaluate the predictive role of the olivocochlear efferent reflex strength in temporary hearing deterioration measured by transient evoked OAEs (TEOAEs) and distortion product OAEs (DPOAEs) in young adults exposed to music.

Methods

Subjects

A total of 28 young adults of which 14 were males and 14 were females participated in the current study. The subject's age ranged from 19 to 30 years (mean: 22.61 years; SD: 2.96 years). One ear per subject was selected at random: 14 right ears (6 males, 8 females) and 14 left ears (8 males, 6 females). All subjects voluntary participated in the study, which was approved by the local ethical committee and all volunteers signed the informed consent in accordance with the declaration of Helsinki.

Experimental protocol

The experimental protocol consisted of three measurement sessions. Before music exposure (pre), immittance measurements, pure-tone audiometry, TEOAEs, DPOAEs and CAS of TEOAEs were measured. After 1 h of listening to an MP3 player (post 1), pure-tone audiometry, TEOAEs and DPOAEs were evaluated and after another 30 min (post 2), these measurements were repeated. The measurements immediately after music exposure-pure-tone audiometry, TEOAEs and DPOAEs-were performed in <10 min and executed in random order. All hearing tests were conducted in a double-walled sound-attenuated booth.

Music exposure

Subjects listened to a fully charged iPod Nano® 2 GB MP3 player (model A1199, 2nd generation, Apple Inc.) with stock iPod earbuds (Apple Inc.). The music sample, genre poprock, lasted approximately 1 h (01:01:55) and was identical for all subjects. The gain setting of the MP3 player was individually determined as a loud, but still a comfortable volume. The volume bar on the MP3 player was marked with a 10% step size to accurately determine the gain setting. The output levels ($L_{Aeq,1h}$) were measured using a right ear simulator (Type 4158c, Brüel and Kjær) of a head and torso simulator (Type 4128c, Brüel and Kjær). The gain setting selected by our subjects was 60% (2 subjects), 70% (2 subjects), 80% (12 subjects) and 90% (12 subjects) which corresponded with a $L_{Aeq,1h}$ of 82.52 dBA, 87.46 dBA, 92.25 dB and 98.70 dBA respectively. Detailed information regarding the output measurement of the MP3 player is provided in Keppler *et al.* (2009).[35]

Immittance measures

Tympanometry was performed with an 85 dB SPL 226 Hz probe tone. Acoustic stapedial reflexes were registered ipsilateral and contralateral at 1.0 kHz and contralateral with broadband noise (TympStar, Grason-Stadler Inc.). Type A tympanogram and acoustic stapedial reflex thresholds at 1.0 kHz between 80 and 120 dB SPL were used as inclusion criteria in the study to ensure normal middle ear function. Further, only subjects with contralateral acoustic stapedial reflex thresholds for broadband noise ranging between 65 and 110 dB SPL were included in the study to avoid eliciting the middle ear acoustic reflex during the measurement of CAS of TEOAEs.[36]

Audiometric evaluation

Air-conduction thresholds at conventional octave frequencies 0.25-8.0 kHz, half-octave frequencies 1.5, 3.0, 6.0 kHz and broadband noise were measured using the modified Hughson-Westlake method with step size 5 dB (Orbiter 922 Clinical Audiometer, Madsen Electronics). Only subjects with hearing thresholds at all measured frequencies ≤25 dBHL were included in the study.

OAEs

All OAEs were measured using the ILO 292 USB II module, ILO V6 clinical OAE software and DPOAE probe (Otodynamics Ltd.). The probe was calibrated before each measurement session using the 1 cc calibration cavity provided by the manufacturer.

TEOAEs at 80 ± 2 dBpeSPL were registered using the non-linear differential stimulus paradigm with rectangular pulses of 80 μs at a rate of 50 clicks/s. Two-hundred and sixty accepted sweeps were obtained and a noise rejection level of 4 mPa was used. Emission and noise amplitudes were calculated by the software in half-octave frequency bands centered at 1.0, 1.5, 2.0, 3.0 and 4.0 kHz. A probe stability of at least 90% was needed and TEOAEs were considered present if the SNR was at least 0 dB at each half-octave frequency band separately. A substitution method in case of present TEOAEs before, but absent TEOAEs after music exposure, was applied to maximize available responses.[37]

DPOAEs were measured with primary tone level combination L1/L2 = 65/55 dB SPL. The f2/f1 ratio was 1.22, with f2 ranging from 0.841 to 8.0 kHz at eight points per octave. A noise artefact rejection level of 6 mPa was used. DPOAEs were considered present if the SNR at all measured frequencies was at least 0 dB. The substitution method mentioned above,[37] was also applied. Present emission and noise amplitudes were then converted to pressure levels and averaged into half-octave frequency bands with center frequencies 1.0, 1.5, 2.0, 3.0, 4.0, 6.0 and 8.0 kHz. For absent DPOAEs at all frequencies within a given half-octave band, emission and noise amplitudes in that frequency band were considered as missing data.

TEOAEs with and without CAS in alternating blocks of 10 s were measured using the linear stimulation method. Clicks, as well as contralateral continuous white noise were presented at 60 dBpeSPL. The intensity levels were chosen based on, first, that efferent suppression (ES) using CAS of TEOAEs is greatest for click intensity levels ranging between 55 and 65 dBpeSPL and second, that the suppressor intensity level is preferably of the same intensity or 5 dB higher than the intensity level of the clicks eliciting TEOAEs.[38-40] Two-hundred and sixty sweeps in the condition without CAS were obtained and a noise rejection level of 4 mPa was applied. TEOAEs with SNR of at least 3 dB in the condition without CAS were considered present. The amount of ES was calculated as the difference (in dB) in TEOAE amplitude with and without CAS at the half-octave frequency bands centered around frequencies 1.0, 1.5, 2.0, 3.0 and 4.0 kHz. Therefore, the higher the value of ES, the stronger the olivocochlear efferent reflex strength.

Data analysis

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 15 (IBM Inc.).

First, mean differences in hearing between the three sessions (pre, post 1 and post 2) were evaluated using one-way ANOVA with *post-hoc* Scheffé test when the significance level $P < 0.05$ was reached. Moreover, hearing thresholds, TEOAE and DPOAE amplitudes before music exposure (pre) were subtracted from values immediately after music exposure (post 1). This resulted in threshold or emission shifts which are considered to be significant based on the values given in Keppler *et al.* (2009).[35] Then, the percentage of significant threshold shifts (STS) or significant emissions shifts (SES) was determined. For pure-tone audiometry, STS+ and STS− reflect respectively a deterioration and an improvement in hearing thresholds in the post 1 session as compared to the session before music exposure. For TEOAEs and DPOAEs, SES− and SES+ indicate respectively a deterioration and an improvement in emission amplitudes in the post 1 session when compared to the session before music exposure.

Second, it was evaluated if the amount of ES at each half-octave frequency was significantly different than 0 using a *t*-test with significance level $P < 0.05$. Then, temporary emission shift (TES) was also calculated by subtracting the pre-measurement data from the results immediately after music exposure (post 1). Regression analysis was performed to evaluate the relationship between TES as an outcome variable on one hand and the amount of ES and gain setting of the MP3 player as predictor variables on the other hand. Three forced entry models with one of the predictors each and the combination of both predictors were applied. ES at the half-octave frequency bands 1.0, 1.5, 2.0, 3.0 and 4.0 kHz was correlated with TES of TEOAEs and TES of DPOAEs at corresponding frequencies.

Results

No significant differences in hearing thresholds, nor TEOAE and DPOAE amplitudes were found between the three sessions except for audiometric thresholds between post 1 and post 2 who were significantly different at 4 kHz ($P < 0.05$) [Table 1]. Mean hearing thresholds, TEOAE and DPOAE amplitudes at measured frequencies at the three sessions are reflected in Figure 1. Mean hearing thresholds, TEOAE and DPOAE amplitudes worsened between pre and post 1 except for audiometric thresholds at several frequencies, whereas mean values at post 2 were systematically better than at post 1.

In Table 2, the percentage of STS+ or SES−, and STS− or SES+ reflecting respectively a deterioration and an improvement in hearing, is given. The SES-amounted up to 17.4% for TEOAEs at the half-octave frequency band 4.0 kHz and 53.6% for DPOAEs at the half-octave frequency band 6.0 kHz.

ES was significantly different from 0 at all frequencies, as seen by the results from the *t*-test - $t(23) = 5.76$, $P < 0.001$,

Table 1: Results from the one-way ANOVA with dependent variables hearing thresholds, TEOAE or DPOAE amplitudes at measured frequencies and independent variables before music exposure (pre), immediately after music exposure (post 1) and 30 min after music exposure (post 2)

Frequency (kHz)	Hearing thresholds	TEOAE amplitudes	DPOAE amplitudes
0.25	$F_{2,81}$=1.53	NA	NA
0.5	$F_{2,81}$=0.36	NA	NA
1.0	$F_{2,81}$=0.38	$F_{2,76}$=0.26	$F_{2,81}$=0.02
1.5	$F_{2,81}$=0.99	$F_{2,76}$=0.28	$F_{2,81}$=0.09
2.0	$F_{2,81}$=0.98	$F_{2,78}$=0.13	$F_{2,81}$=0.16
3.0	$F_{2,81}$=1.45	$F_{2,76}$=0.46	$F_{2,81}$=0.33
4.0	$F_{2,81}$=4.88*	$F_{2,66}$=0.11	$F_{2,81}$=0.40
6.0	$F_{2,81}$=0.54	NA	$F_{2,81}$=0.46
8.0	$F_{2,81}$=0.42	NA	$F_{2,52}$=0.22

*Significance level $P < 0.05$, TEOAE = Transient evoked otoacoustic emission, DPOAE = Distortion product otoacoustic emission, NA = Not applicable, ANOVA = Analysis of variance

Table 2: Percentages of STS and SES for TEOAEs and DPOAEs at measured frequencies. A deterioration in hearing is reflected by STS+ and SES−, whereas an improvement in hearing is indicated by STS− and SES+

Frequency (kHz)	Hearing thresholds		TEOAE amplitudes		DPOAE amplitudes	
	STS− (%)	STS+ (%)	SES− (%)	SES+ (%)	SES− (%)	SES+ (%)
0.25	0.0	0.0	NA	NA	NA	NA
0.5	0.0	7.1	NA	NA	NA	NA
1.0	0.0	0.0	7.7	3.8	25.0	10.7
1.5	0.0	0.0	0.8	1.0	7.1	3.6
2.0	3.6	3.6	11.1	7.4	17.9	7.1
3.0	3.6	0.0	15.4	3.8	21.4	0.0
4.0	7.1	3.6	17.4	4.3	28.6	7.1
6.0	7.1	3.6	NA	NA	53.6	14.3
8.0	7.1	7.1	NA	NA	21.7	4.3

TEOAE = Transient evoked otoacoustic emission, DPOAE = Distortion product otoacoustic emission, SES = Significant emission shifts, NA = Not applicable, STS = Significant threshold shifts

$t(27) = 5.84$, $P < 0.001$, $t(26) = 5.97$, $P < 0.001$, $t(25) = 5.96$, $P < 0.001$ and $t(19) = 2.35$, $P < 0.05$ respectively at 1.0, 1.5, 2.0, 3.0 and 4.0 kHz [Figure 2].

Table 3 indicates that TES of TEOAEs was negatively correlated with the gain setting of the MP3 player, except at the half-octave frequency bands 1.0 and 1.5 kHz. This correlation was significant at the half-octave frequency band 3.0 and 4.0 kHz. TES of TEOAEs and ES were positively correlated only at the half-octave frequency bands 2.0 and 3.0 kHz, but this was not significant. There was no significant increase in R^2 from the model with predictor variable gain to the model with both predictors. However, at the half-octave frequency band 3.0 kHz, there was a significant R^2 change from the model with predictor variable ES to the model with both predictors. The highest variance explaining the TES of TEOAE by both predictor variables was 26% at the half-octave frequency band 3.0 kHz.

As it has shown in Table 4, for the TES of DPOAEs, a significant negative correlation was seen with the gain setting of the MP3 player at the half-octave frequency band 4.0 kHz. TES of DPOAEs and ES were positively correlated, except at the half-octave frequency band 4.0 kHz. There was only a slight increase in R^2 from the model with either one predictor to the model with both predictor variables, with the exception at the half-octave frequency band 4.0 kHz. The increase in R^2 was significant for variables ES to variables gain and ES, reaching an explained variance in TES of DPOAEs of 43% at the half-octave frequency band 4 kHz.

Discussion

In the current study, the predictive role of the olivocochlear efferent reflex strength using ES in temporary hearing deterioration in subjects exposed to music was evaluated. The rationale is based on (1) animal data suggesting

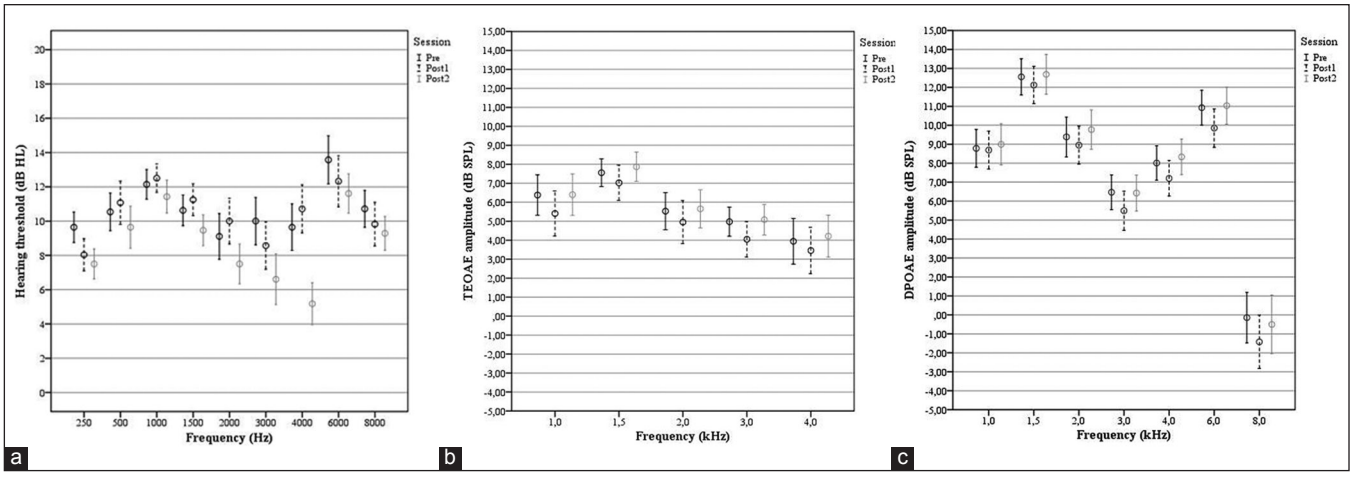

Figure 1: Mean (±1 standard error) hearing thresholds (a), transient evoked otoacoustic emissions (OAEs) amplitudes (b) and distortion product OAEs amplitudes (c) at measured frequencies before music exposure (pre), immediately after music exposure (post 1) and after another 30 min after music exposure (post 2)

a noise- protective role of the MOC system in TTS, as well as PTS experiments,[20,22-29,41] and (2) the ability to explore the MOC system by measuring OAEs with CAS.[9,10] Therefore, one would assume that subjects with a higher amount of ES sustain less cochlear damage after noise exposure. This hypothesis was supported by Maison and Liberman (2000) stating that MOC reflex strength is correlated with the amount of PTS in guinea pigs, making it possible to differentiate between tender and tough ears and thus predicting its vulnerability to permanent acoustic injury.[34] Predicting an individual's susceptibility to NIHL, would optimize preventive strategies for hearing conservation.

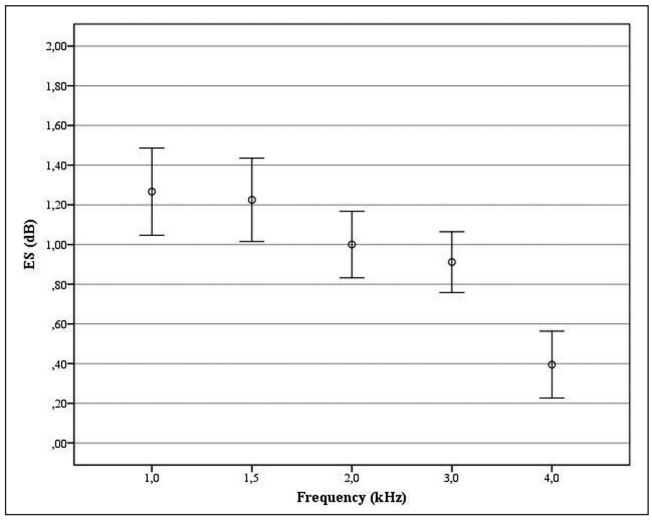

Figure 2: Mean (±1 standard error) efferent suppression measured using contralateral acoustic stimulation of transient evoked otoacoustic emissions amplitudes at half-octave frequency bands 1.0, 1.5, 2.0, 3.0 and 4.0 kHz

Although a PTS cannot be predicted from the initial TTS,[42] occurrence of a TTS indicates exposure to hazardous noise doses and potential development to a PTS. Experimental conditions with potential risk to PTSs in human subjects is, due to obvious reasons, not possible. Therefore, in the current study, subjects listened voluntarily to a commercially available MP3 player during 1 h at an individually preferred loud listening level. Temporary hearing deterioration after listening to a personal music player was found previously for pure-tone audiometry, as well as OAEs.[35,43-49] Although currently no significant differences in hearing status after music exposure were found, there was a tendency that mean hearing thresholds, but especially TEOAE and DPOAE amplitudes, worsened after the 1 h listening session. Furthermore, SES− indicating a deterioration in emission amplitudes was found in 17.4% and 53.6% for TEOAEs and DPOAEs, respectively. However, it must be emphasized that hearing recovered completely in all of our subjects.

The preferred loud listening levels varied from 60% to 90% of the gain setting of the MP3 player which corresponds to output levels between 82.52 dBA and 98.70 dBA.[35] Mostly, TES was correlated with the chosen gain setting of the MP3 player, i.e. more temporary cochlear damage with louder listening levels. The variability in shifts across

Table 3: For the TEOAE amplitudes at half-octave frequency bands from 1.0 to 4.0 kHz, the Pearson correlation coefficient, R^2 and the results from the ANOVA analysis are reflected for gain setting of the MP3 player, ES and variables gain and ES. Further, β and the change in R^2 are given for both predictor variables

Frequency (kHz)	Gain				ES				Gain × ES					
	r	R^2	*F*	β	*r*	R^2	F	*B*	R^2	*F*	β		R^2 change	
											Gain	ES	Gain	ES
1.0	0.15	0.02	$F_{1,21}$=0.46	0.15	−0.13	0.02	$F_{1,21}$=0.36	−0.13	0.03	$F_{2,20}$=0.33	−0.11	0.13	0.01	0.02
1.5	0.06	0.00	$F_{1,24}$=0.08	0.06	−0.23	0.05	$F_{1,24}$=0.33	−0.23	0.05	$F_{2,23}$=0.65	−0.24	−0.03	0.05	0.00
2.0	−0.26	0.07	$F_{1,24}$=1.69	−0.26	0.09	0.01	$F_{1,24}$=0.20	0.09	0.07	$F_{2,23}$=0.84	0.05	−0.25	0.00	0.06
3.0	−0.51**	0.26	$F_{1,24}$=8.53**	−0.51	0.06	0.00	$F_{1,24}$=0.09	0.06	0.26	$F_{2,23}$=4.10*	0.02	−0.51	0.00	0.26**
4.0	−0.44*	0.19	$F_{1,18}$=4.26	−0.44	−0.23	0.05	$F_{1,18}$=0.96	−0.23	0.21	$F_{2,17}$=2.20	−0.12	−0.41	0.01	0.16

The significance levels are indicated by asterisks, *P < 0.05, **P < 0.01. ES = Efferent suppression, TEOAE = Transient evoked otoacoustic emission, ANOVA = Analysis of variance

Table 4: For the predictor variables gain of the MP3 player, ES and variables gain and ES, the effects on the DPOAE amplitudes at half-octave frequency bands from 1.0 to 4.0 kHz are given: Pearson correlation coefficient, R^2 and the results from the ANOVA analysis, as well as β and R^2 change

Frequency (kHz)	Gain				ES				Gain × ES					
	r	R^2	*F*	β	*r*	R^2	*F*	β	R^2	*F*	β		R^2 change	
											Gain	ES	Gain	ES
1.0	−0.13	0.02	$F_{1,22}$=0.39	−0.13	0.01	0.00	$F_{1,22}$=0.00	0.01	0.02	$F_{2,21}$=0.19	−0.02	−0.13	0.00	0.02
1.5	−0.27	0.07	$F_{1,26}$=2.07	−0.27	0.17	0.03	$F_{1,26}$=0.80	0.17	0.09	$F_{2,25}$=1.15	0.11	−0.24	0.01	0.06
2.0	−0.12	0.01	$F_{1,25}$=0.35	−0.12	0.04	0.00	$F_{1,24}$=0.17	0.04	0.01	$F_{2,24}$=0.17	0.02	−0.11	0.00	0.01
3.0	−0.25	0.06	$F_{1,24}$=1.55	−0.25	0.21	0.04	$F_{1,24}$=1.09	0.21	0.10	$F_{2,23}$=1.23	0.19	−0.23	0.04	0.05
4.0	−0.64**	0.41	$F_{1,18}$=12.62**	−0.64	−0.28	0.08	$F_{1,18}$=0.1.57	−0.28	0.43	$F_{2,17}$=6.37**	−0.13	−0.61	0.02	0.35**

The significance levels are indicated by asterisks, *P < 0.05, **P < 0.01. ES = Efferent suppression, DPOAE = Distortion product otoacoustic emission, ANOVA = Analysis of variance

gain setting, however, underlines the presence of an inter-individual vulnerability to noise. The highest variance explained by gain setting of the MP3 player was 26% and 43% for TES of TEOAEs at half-octave frequency band 3.0 kHz and TES of DPOAEs at half-octave frequency band 4.0 kHz, respectively.

ES as only predictor in the regression model explained only small amounts of variance in TES of TEOAEs and TES of DPOAEs. Adding ES as second predictor in the model did only increase the R^2 significantly at some frequencies. Moreover, ES was negatively, as well as positively correlated with TES. So, from this study, it does not seem possible to predict the amount of temporary hearing deterioration from the olivocochlear efferent reflex strength using CAS of TEOAEs. This is consistent with earlier findings reporting that CAS of DPOAEs did not predict TTSs, nor TESs measured by DPOAEs after noise exposure.[50-52]

Several factors can possibly explain these findings. First, the MOC system did not evolve as a noise-protective mechanism since high-intensity natural acoustic environmental noise is uncommon.[19] However, this would not explain why the protective effect of the MOCB is observed in several mammalian species such as cats, guinea pigs and chinchillas,[20,22-29,41,53] but not in humans. Second, the MOC system cannot be measured using CAS of OAEs. However, abnormal ES using CAS of evoked OAEs was found in patients with vestibular neurectomy and thus sectioning of the OCB.[54-56] Nevertheless, it is possible that clinical measures of ES by CAS of TEOAEs using commercially available OAE-equipment are too rough to detect subtle differences in MOC system functioning, or cannot differentiate between a variety of efferent reflex strengths due to the limited range of ES[57] which could also compromise statistical measures.[58] Nevertheless, the current study indicated that ES was significantly different than 0 at all tested frequencies. Nevertheless, it was not possible to measure ES using CAS of DPOAEs with the used OAE-equipment which could result in slightly different findings. Further, using CAS of evoked OAEs requires evoked OAEs to be present with a considerable amplitude. Low-level or absent OAEs, especially in the high-frequency range, might indicate pre-existing inner ear damage,[4,5,37] but are excluded from analysis although providing valuable information regarding susceptibility to NIHL. Thus, it seems that the technique used to measure ES by CAS of TEOAEs, as well as data analysis should be optimized before ruling out the relationship between MOC activity and hearing deterioration. Third, optimal noise-exposure conditions to activate the MOC system are not easily achieved and could probably explain why others, even in guinea pigs, did not found noise-protective effect of the MOCB.[59] However, neither impulse nor occupational noise exposure previously,[51,52] nor music exposure in the current study, comprising a range of types of noise exposure were capable of detecting a negative correlation between efferent reflex strength and temporary hearing deterioration. However, it could be interesting to evaluate the predictive role of the olivocochlear efferent reflex strength in temporary hearing deterioration after other types of noise exposure, e.g. with white noise. A final explanation could be that no noise-protective effect of the MOC system was seen due to the limited temporary hearing deterioration observed in the current study. In animals, effects were mostly seen at largest TTS,[21] but this is an inherent limitation in all human TTS studies. Nevertheless, it is possible that the predictive role of MOC system in humans is only reflected in permanent hearing damage.

Conclusion

Clinical measures of ES using CAS of TEOAEs in the current study were not possible to establish a clear relationship between the protective effects of the MOC system and temporary hearing deterioration. However, it is possible that the temporary hearing deterioration in the current study was insufficient to activate the MOC system. More research including a higher amount of subjects is therefore advised. Nevertheless, ES measures using CAS of TEOAEs should be optimized to be able to differentiate more accurately between subtle differences in olivocochlear efferent reflex strengths. Further, large scale longitudinal studies are necessary to investigate the role of MOC system in the development of permanent hearing damage. Therefore, in our opinion, it is too soon to reject ES using CAS of evoked OAEs as a non-invasive tool to evaluate susceptibility in individuals to cochlear damage. More research regarding ES might provide more insight in the olivocochlear efferent pathways and their role in auditory functioning.

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How to cite this article: Hannah K, Ingeborg D, Leen M, Annelies B, Birgit P, Freya S, *et al*. Evaluation of the olivocochlear efferent reflex strength in the susceptibility to temporary hearing deterioration after music exposure in young adults. Noise Health 2014;16:108-15.

Source of Support: Hannah Keppler was funded through an Aspirant Scholarship of the Research Foundation Flanders (FWO), Belgium. **Conflict of Interest:** None declared.