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MAIN ARTICLE

Effects of chronic noise exposure on speech-in-noise perception in the presence of normal audiometry

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Abstract

Objective: To assess auditory processing in noise-exposed subjects with normal audiograms and compare the findings with those of non-noise-exposed normal controls.

Methods: Ten noise-exposed Royal Air Force aircrew pilots were compared with 10 Royal Air Force administrators who had no history of noise exposure. Participants were matched in terms of age and sex. The subjects were assessed in terms of: pure tone audiometry, transient evoked otoacoustic emissions, suppression of transient evoked otoacoustic emissions in contralateral noise and auditory processing task performance (i.e. masking, frequency discrimination, auditory attention and speech-in-noise).

Results: All subjects had normal pure tone audiometry and transient evoked otoacoustic emissions amplitudes in both ears. The noise-exposed aircrew had similar pure tone audiometry thresholds to controls, but right ear transient evoked otoacoustic emissions were larger and speech-in-noise thresholds were elevated in the noise-exposed subjects compared to controls.

Conclusion: The finding of poorer speech-in-noise perception may reflect noise-related impairment of auditory processing in retrocochlear pathways. Audiometry may not detect early, significant noise-induced hearing impairment.

Key words: Hearing Loss, Noise Induced; Trauma; Central Auditory Processing Disorder; Speech Perception

Introduction

Noise-induced hearing loss is a common occupational disorder worldwide. In the UK, around one million workers are exposed to damaging levels of noise.[1] It has been estimated that 153 000 men and 26 000 women aged 35–64 years may have severe hearing difficulties attributable to noise exposure at work.[2] Noise-induced hearing loss is more prevalent in heavy industry, manufacturing and the military. The National Institutes of Health considers levels above 80 dB SPL to be hazardous.[3] Noise exposure above this level may, in theory, cause temporary hearing deficits or even permanent threshold shifts. The sound pressure level required to cause permanent threshold shifts varies amongst individuals. Only 5 per cent of those exposed daily to an equivalent average noise exposure level of 85 dB(A) throughout an 8-hour day, over a 30-year period, developed a significant hearing loss.[4] The UK Control of Noise at Work Regulations (2005) have limited the average noise exposure level over an 8-hour work day to 80 dB(A); specific actions are to be taken if noise exceeds this level, with an exposure maximum set at 87 dB(A) over an 8-hour work day.[5]

The most prominent histopathological feature of permanent threshold shifts in noise-induced hearing loss is progressive damage to hair cells.[6] In addition, intense noise exposure leads to structural and functional changes of the tectorial membrane, sensory hair bundles, tip links, and intracellular organelles of the cochlear hair cells.[7] It also leads to the loss of the cell bodies of the cochlear afferent neurons within the spiral ganglion that are in contact with the damaged hair cells.[8] These changes may impact on the structure and function of the central auditory nervous system.[7] A reduced neural output from the cochlea in response to noise, reduces nuclear density in the central auditory nervous system,[9] while functionally, after denervation, the redundant central auditory nervous system begins to respond to neighbouring frequencies.[10] The effects of noise on the cochlea and hearing nerve after a permanent threshold shift, and the resulting changes in the central auditory nervous system are well documented.

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However, the effects of noise exposure on the retrocochlear pathways that do not lead to permanent threshold shifts are less well studied.

Royal Air Force (RAF) aircrew are exposed to significant levels of noise when flying, both from the airframe and in-ear communication devices. Research indicates that the aircrew are regularly exposed to noise levels greater than 85 dB(A).[11] The RAF pilots undergo six-monthly medicals; the examinations include pure tone audiograms, which are conducted to ensure normal hearing and determine whether the pilots remain fit to fly. The RAF pilots therefore represent a unique study group of noise-exposed subjects with normal audiometry.

This study assessed auditory processing in RAF Chinook helicopter aircrew using a psychoacoustic test battery. This population, who had both a quantifiable history of noise exposure (data obtained from a flying log book) and normal audiometry, was compared with RAF administration staff (matched in terms of sex and age). The RAF administrators had normal hearing and no history of noise exposure. The study aimed to examine the potential effects of noise exposure on the central auditory pathway.

Materials and methods

Ethical considerations

This study was approved by the Ministry of Defence Ethics Committee. Informed consent was obtained from all study participants. Testing was conducted at the RAF Odiham Medical Centre, UK.

Subjects

Case subjects. The case subjects comprised 10 male, otologically normal RAF pilots who were attending their biannual aircrew medical (mean age was 31.2 years, standard deviation (SD) 5.1, median 28.4, range 24.1–38.3 years). Aircrew were included if they had a minimum of 500 flying hours and normal hearing thresholds in their last audiogram, (i.e. no threshold greater than 20 dBHL in either ear between 500 Hz and 4000 Hz). The aircrew subjects had a mean average of 1438 flying hours (median 1650, SD 627, range 680–2200 hours).

Control subjects. Ten male, otologically normal RAF administrators (mean age 30.4, SD 5.6, median 32.0, range 24.8–39.1 years) were recruited as controls. These subjects had no history of noise exposure and all had normal hearing thresholds.

Females were excluded from the study in order to decrease experimental variation. Age was not significantly different between the two groups ($p = 0.796$).

Test procedures

Baseline tests. These included otoscopy followed by wax removal if required, and pure tone audiometry, which was conducted as per standard guidelines using a GSI 61 audiometer with TDH-49 earphones (Guymark UK, Brierley Hill, UK) in a sound-attenuated room. The average hearing level for four frequencies (0.5, 1, 2 and 4 KHz) was calculated (in dBHL) for each ear.

Transient evoked otoacoustic emissions (TEOAEs) were used to assess outer hair cell and inner ear function. This test was conducted in both ears using a dual-channel Otodynamic ILO88/92 Analyser (Otodynamics, Hatfield, UK). A standard default setup was used,[12] with an 80-μs click stimulus of 80 dB; the response amplitude (in dB) was averaged over 260, 20-ms sweeps. Normal TEOAEs in the 2.5 to 20 ms post-stimulus period (across 500–4000 Hz) were defined as an overall response amplitude signal-to-noise ratio of at least 6 dB and waveform reproducibility of more than 70 per cent in at least 3 adjacent octave bands.[13] The overall TEOAE amplitude and reproducibility was recorded for each ear.

Suppression of otoacoustic emissions was tested using contralateral noise (TEOAE plus suppression). This was done in both ears using the same dual channel analyser as for TEOAE. The amplitudes of TEOAE are reduced with contralateral ear sound stimulation.[14] This is mediated by the efferent medial olivocochlear bundle that is excited at the brainstem level via the afferent auditory pathways,[15] which may enhance speech intelligibility in background noise.[16] The TEOAEs suppression test was carried out using one channel for ipsilateral and the other for contralateral acoustic stimulation. A linear click of 60 dB SPL was applied for ipsilateral stimulation and a broad band noise (0.50–6 kHz) of 40 dB SPL sensation level was used for contralateral stimulation. Average responses over 600 sweeps were computed. Suppression was determined by subtracting the TEOAE with noise average amplitude from the TEOAE without noise average amplitude; values greater than or equal to 1 dB were considered normal.[16]

Auditory processing tests. The Institute of Hearing Research Multicentre study of Auditory Processing (‘IMAP’) test battery[17] includes tests of: temporal processing (backward masking tests with no gap or with a 50 ms gap), spectral processing (simultaneous masking tests with a delay or with a delayed notch), frequency discrimination, auditory attention, and recognition of speech-in-noise (vowel-consonant-vowel test in International Collegium for Rehabilitative Audiology (ICRA) noise).

All tests were conducted binaurally and presented as a computer game. The outcome measures for the backward and simultaneous masking tasks were threshold measurements, calculated as the mean of the last three trials of each track (expressed as dB SPL). The outcomes for the frequency discrimination tests (expressed as percentage difference) were established using an adaptive, three-interval, three alternative (odd one out) forced-choice paradigm. Specifically,

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three auditory stimuli were presented to the subject via headphones and reinforced with three corresponding visual choices on the computer screen; subjects were required to identify the target or 'odd one out' using a purpose-built button box. Threshold measurements were also attained (in dB SPL) for the adaptive staircase vowel-consonant-vowel test in International Collegium for Rehabilitative Audiology noise, which required subjects to repeat the vowel-consonant-vowel. The outcome measure for the auditory attention test was reaction time (for the cued and non-cued conditions, and the difference between the two).

Analysis

The test results were summarised using the mean, SD, mean difference and confidence intervals of the difference. Taking into consideration the small sample size, Mann–Whitney U tests were conducted to explore the statistical significance of differences in test results between the two groups. A *p*-value less than 0.05 was considered to be indicative of statistical significance.

Results

Baseline tests

Pure tone averages demonstrated normal hearing thresholds in both groups, with no statistically significant difference between the two groups for any frequency in either ear. The pure tone average tended to be slightly better in both ears (by 2 dB) for the control group, but this was not statistically significant (Table I).

All noise-exposed subjects and non-noise-exposed controls had normal transient evoked otoacoustic emissions (TEOAEs). The aircrew subjects tended to have larger TEOAE amplitudes than controls in both ears, but only the right ear differences (of 4 dB) were statistically significant ($p = 0.043$, Table II). Suppression values did not differ between the two groups (Table II).

Auditory processing tests

Auditory processing was assessed using the Institute of Hearing Research Multicentre study of Auditory Processing test battery. The results revealed that noise-exposed aircrew subjects had worse thresholds than controls in the vowel-consonant-vowel test by 3.9 dB (mean 49.7 dB SPL in subjects *vs* 45.8 dB SPL in controls; $p = 0.019$). Backward and simultaneous masking tests were associated with similar thresholds in the two groups (Table III). In the frequency discrimination test, the results tended to be worse for aircrew subjects (mean 4.62 per cent, SD 9.53) than for controls (mean 1.5 per cent, SD 1.55), but the difference was not significant (Table III). The aircrew subjects had six times more variability in performance. The auditory attention test indicated better reaction times for the aircrew versus the controls, but there was no significant difference (Table IV).

TABLE I
PURE TONE AUDIOGRAM AVERAGES*

Ear	Subjects (mean (SD); dB)	Controls (mean (SD); dB)	Mean difference (95% CI)	*p*
Right	5.6 (3.5)	3.1 (3.0)	2.4 (−0.6–5.5)	0.143
Left	5.9 (4.1)	3.7 (3.8)	2.2 (−1.4–5.9)	0.218

*Average hearing level for four frequencies (0.5, 1, 2 and 4 KHz) in noise-exposed subjects and non-noise-exposed controls. SD = standard deviation; CI = confidence interval

Discussion

This study compared cochlear function and auditory processing in a noise-exposed versus a non-noise-exposed, age-matched male population. The most prominent difference between the two groups was the speech-in-noise test performance, which was almost 4 dB worse in the noise-exposed RAF aircrew compared with the non-noise-exposed RAF administrators ($p = 0.019$). By selecting controls who worked for the RAF, the effect of potentially confounding factors such as higher order effects of intelligence on speech recognition was minimised. Furthermore, the auditory attention test did not identify any significant differences between the two groups that could account for these findings.

The two groups had similar pure tone audiometry results, indicating that the worse speech-in-noise performance for the aircrew could not be accounted for by a difference in hearing levels. Transient evoked otoacoustic emissions (TEOAEs) may be lost before audiometric thresholds change in up to 56 per cent of noise-exposed subjects.[18] However, TEOAE average responses were normal in both study groups, with a criterion of normal responses in at least three adjacent frequency bands between 1 and 4 kHz. In addition, TEOAE overall amplitude was significantly larger in the noise-exposed aircrew versus the controls in the right ear ($p = 0.043$), indicating enhanced cochlear sensitivity across the three frequency bands within the speech frequency range.

Long-term moderate noise exposure in guinea pigs can increase distortion product otoacoustic emission amplitudes at low frequencies (1.0 to 3.0 kHz),[19] which is probably a result of conditioning. This is consistent with the results of the present study. That study also found decreased olivocochlear efferent suppression at the same frequencies. In our study, overall suppression values were not different between the two groups, but we cannot exclude the possibility that reduced suppression in specific frequency bands adversely affected speech-in-noise perception, as reported by Mukari and Mamat (2008).[20] Other studies have reported reduced suppression in noise-exposed humans with normal hearing thresholds.[21] However, these findings may not be directly comparable to ours, as their noise-exposed study group had significantly higher (albeit within the normal range)

TABLE II
MEAN TEOAE AND TEOAE SUPPRESSION RESPONSES*

Parameter	Subjects (mean (SD); dB)	Controls (mean (SD); dB)	Mean difference (95% CI)	p
R ear TEOAE	15.9 (3.2)	11.9 (3.9)	4 (0.7–7.4)	0.043
L ear TEOAE	14.3 (2.3)	12.6 (4)	1.7 (−1.4–4.7)	0.529
R ear TEOAE suppression	1.04 (0.7)	1.09 (0.6)	−0.05 (−0.7–0.6)	0.739
L ear TEOAE suppression	0.39 (2.7)	1.33 (0.6)	−0.94 (−2.9–1)	0.579

*For noise-exposed subjects and non-noise-exposed controls. SD = standard deviation; CI = confidence interval; R = right; TEOAE = transient evoked otoacoustic emissions; L = left

high frequency thresholds, and there was a tendency for TEOAE amplitude to be lower than in controls, which is in contrast to the findings of Mukari and Mamat's study wherein suppression was reduced in high frequencies.

Animal studies indicate that noise exposure leads to damage and reorganisation of the auditory pathways from the level of the auditory nerve up to the cortex, in the presence of normal or abnormal audiometric thresholds. Extensive permanent noise was shown to provoke the loss of afferent nerve terminals, and the delayed degeneration of the cochlear nerve was reported following exposure to noise that caused a temporary threshold shift, with complete threshold recovery and normal hair cell function.[22] The authors of that study suggested that the consequences of primary neuronal loss on the auditory processing of suprathreshold sounds are likely to be dramatic, despite a threshold recovery.

Reduced speech-in-noise perception is common in patients with auditory neuropathy, some of whom may have normal audiometric thresholds.[23] We did not assess auditory nerve function using auditory brainstem evoked responses; however, the Institute of Hearing Research Multicentre study of Auditory Processing test battery results may offer some insight. Patients with auditory neuropathy are reported to have reduced frequency discrimination at frequencies below 4 kHz compared with normal controls.[24] This is to some degree consistent with our finding of more variable frequency discrimination performance in noise-exposed aircrew pilots. However, auditory neuropathy patients also show prominent deficits in temporal tasks, including masking.[24] This was not the case for the aircrew subjects in our study, whose performance in the masking tests was similar to that of normal controls. An auditory nerve lesion in the aircrew pilots is thus unlikely, but cannot be excluded altogether.

Previous studies have reported the effects of noise exposure on auditory cortex function in humans. For instance, chronic low level background noise exposure in otherwise healthy individuals seems to alter the normal left hemisphere dominant speech-induced activity to right hemisphere dominance for speech processing.[25] Kujala *et al.* assessed speech sound discrimination in noise-exposed shipyard workers (aged less than 35 years old) with normal pure tone audiometry.[26] They compared the results of behavioural and electrophysiological tests (the auditory-evoked N1/P2 and mismatch negativity components) with non-exposed normal controls. The authors found there was impaired speech discrimination in the noise-exposed subjects, consistent with the findings of the present study, which was attributed to an early cortical sound discrimination dysfunction. Novel sounds presented in noise distracted the noise-exposed subjects significantly more than controls, indicating reduced attention control. There was no effect on attention in our study; however, this may be due to the use of reaction times as an outcome measure.

Our study found no further evidence for impaired auditory cortical processing on the basis of the non-speech test results. However, the frequency

TABLE III
MEAN AUDITORY PROCESSING TASK THRESHOLDS*

Test (units)	Subjects mean threshold (SD)	Controls mean threshold (SD)	Mean difference (95% CI)	p
Speech-in-noise (dB SPL)	49.7 (3.3)	45.8 (3.65)	3.8 (0.6–7.12)	0.019
Simultaneous masking (dB SPL)				
– Delay	64.3 (5.5)	66.5 (2.2)	−2.2 (−6.2–9.1)	0.393
– Delayed notch	42.3 (3.8)	42.6 (4.4)	−0.3 (−4.1–3.6)	0.853
Backward masking (dB SPL)				
– No gap	35.4 (6.2)	35.6 (12.5)	−0.2 (−9.7–9.3)	0.436
– 50 ms gap	28.53 (3.7)	28.57 (5.5)	−0.04 (−4.4–4.3)	0.912
Frequency discrimination (% difference)	4.62 (9.53)	1.5 (1.55)	3.1 (−3.8–9.9)	0.436

*For noise-exposed subjects and non-noise-exposed controls, using the Institute of Hearing Research Multicentre study of Auditory Processing test battery. SD = standard deviation; CI = confidence interval

TABLE IV
AUDITORY ATTENTION TASK MEAN RT*

Condition	Subjects (mean (SD); ms)†	Controls (mean (SD); ms)†	Mean difference (95% CI)	*p*
Cued	346 (67)	381 (101)	−35 (−114–45)	0.393
Non-cued	495 (102)	530 (102)	−35 (−130–61)	0.529
Difference‡	148 (66)	149 (89)	1 (−73–73)	0.853

*For noise-exposed subjects and non-noise-exposed controls. †Mean RT = reaction time. ‡Difference = Non-cued minus cued. SD = standard deviation; CI = confidence interval

discrimination results were better for controls (at 1.52 per cent) and worse for subjects (at 4.62 per cent) compared with adult normative data of 2.5 per cent.[27] In addition, performance was more variable in the noise-exposed group; the lack of a significant difference between the noise-exposed subjects and non-noise-exposed controls may have been due to the low power of the study. This potential difference needs to be explored further using a larger sample, as the primary auditory cortex and surrounding region play a critical role in perceptual pitch discrimination.[28]

- **This study assessed auditory processing in noise-exposed versus non-noise-exposed subjects; all had normal audiograms and cochlear function**
- **Noise-exposed subjects showed impaired speech-in-noise perception**
- **This effect may be due to abnormal processing of sound within retrocochlear pathways**
- **Audiometry may not detect early, significant noise-induced hearing impairment**

In conclusion, this study found a clinically important and significant ($p = 0.019$) reduction of speech-in-noise perception in noise-exposed pilots versus non-exposed controls, despite both groups having normal TEOAE amplitudes and hearing thresholds. The region responsible for this impairment was likely to be posterior to the cochlea at the auditory cortex level, as described in other studies. The risk of auditory symptoms has been shown to increase with the number of years of occupational noise exposure, and the use of hearing aids rises as the symptoms become more severe.[2] However, average thresholds may not correlate with the degree of reported auditory symptoms.[29] Our findings, together with those of other authors, indicate that audiometry, which forms the cornerstone of detection in occupational hearing conservation schemes, is not sufficient to detect significant noise-induced hearing impairment. Such impairments include reduced speech-in-noise recognition, which may have significant effects on performance or productivity, and on safety, which is particularly important in military professions. Further research is required to elucidate the anatomical level(s) of lesions that underpin these findings, and to investigate auditory processing in this population in more detail.

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References

1 Noise at work. In: http://www.hse.gov.uk/noise/ [14 December 2012]
2 Palmer KT, Griffin MJ, Syddall HE, Davis A, Pannett B, Coggon D. Occupational exposure to noise and the attributable burden of hearing difficulties in Great Britain. *Occup Environ Med* 2002;**59**:634–9
3 Noise. In: http://www.nlm.nih.gov/medlineplus/noise.html [14 December 2012]
4 Dobie RA. Noise-induced hearing loss. In: Bailey BJ, ed. *Head and Neck Surgery-Otolaryngology*, 2nd edn. Philadelphia: Lippincott-Raven, 1998;1782–92
5 The Control of Noise at Work Regulations 2005. In: http://www.legislation.gov.uk/uksi/2005/1643/contents/made [13 December 2012]
6 Saunders JC, Dear SP, Schneider ME. The anatomical consequences of acoustic injury: a review and tutorial. *J Acoust Soc Am* 1985;**78**:833–60
7 Saunders JC, Cohen YE, Szymko YM. The structural and functional consequences of acoustic injury in the cochlea and peripheral auditory system: a five year update. *J Acoust Soc Am* 1991;**90**:136–46
8 Kujawa SG, Liberman MC. Acceleration of age-related hearing loss by early noise exposure: evidence of a misspent youth. *J Neurosci* 2006;**26**:2115–23
9 Basta D, Tzschentke B, Ernst A. Noise-induced cell death in the mouse medial geniculate body and primary auditory cortex. *Neurosci Lett* 2005;**381**:199–204
10 Wang J, Salvi RJ, Powers N. Plasticity of response properties of inferior colliculus neurons following acute cochlea damage. *J Neurophysiol* 1996;**75**:171–83
11 Küpper TE, Steffgen J, Jansing P. Noise exposure during alpine helicopter rescue. *Ann Occup Hyg* 2004;**48**:475–81
12 Kemp DT, Ryan S, Bray P. A guide to the effective use of otoacoustic emissions. *Ear Hear* 1990;**11**:93–105
13 Hurley RM, Musiek FE. Effectiveness of transient-evoked otoacoustic emissions (TEOAEs) in predicting hearing level. *J Am Acad Audiol* 1994;**5**:195–203
14 Collet L, Kemp DT, Veuillet E, Duclaux R, Moulin A, Morgon A. Effect of contralateral auditory stimuli on active cochlear micro-mechanical properties in human subjects. *Hear Res* 1990;**43**:251–62
15 Brown GJ, Ferry RT, Meddis R. A computer model of auditory efferent suppression: implications for the recognition of speech in noise. *J Acoust Soc Am* 2010;**127**:943–54
16 Ceranic BJ, Prasher DK, Raglan E, Luxon LM. Tinnitus after head injury: evidence from otoacoustic emissions. *J Neurol Neurosurg Psychiatry* 1998;**65**:523–9

https://doi.org/10.1017/S002221511200299X Published online by Cambridge University Press

17 Moore DR, Ferguson MA, Edmondson-Jones AM, Ratib S, Riley A. Nature of auditory processing disorder in children. *Pediatrics* 2010;**126**:e382–90
18 Desai A, Reed D, Richards S, Prasher D. Absence of otoacoustic emissions in subjects with normal audiometric thresholds implies exposure to noise. *Noise Health* 1999;**1**:58–65
19 Peng JH, Tao ZZ, Huang ZW. Long-term sound conditioning increases distortion product otoacoustic emission amplitudes and decreases olivocochlear efferent reflex strength. *Neuroreport* 2007;**18**:1167–70
20 Mukari SZS, Mamat WHW. Medial olivocochlear functioning and speech perception in noise in older adults. *Audiol Neurootol* 2008;**13**:328–34
21 Sliwinska-Kowalska M, Kotylo P. Occupational exposure to noise decreases otoacoustic emission efferent suppression. *Int J Audiol* 2002;**41**:113–19
22 Kujawa SG, Liberman MC. Adding insult to injury: cochlear nerve degeneration after "temporary" noise-induced hearing loss. *J Neurosci* 2009;**29**:14077–85
23 Kraus N, Bradlow AR, Cheatham MA, Cunningham J, King CD, Koch DB *et al*. Consequences of neural asynchrony: a case of auditory neuropathy. *J Assoc Res Otolaryngol* 2000;**1**: 33–45
24 Zeng FG, Kong YY, Michalewski HJ, Starr A. Perceptual consequences of disrupted auditory nerve activity. *J Neurophysiol* 2005;**93**:3050–63
25 Brattico E, Kujala T, Tervaniemi M, Alku P, Ambrosi L, Monitillo V. Long-term noise exposure altered the strength and the hemispheric organization of speech-sound discrimination and decreased the speed of sound-change processing. *Clin Neurophysiol* 2005;**116**:190–203
26 Kujala T, Shtyrov Y, Winkler I, Saher M, Tervaniemi M, Sallinen M *et al*. Long-term exposure to noise impairs cortical sound processing and attention control. *Psychophysiology* 2004;**41**:875–81
27 Moore DR, Ferguson M, Halliday F, Riley A. Frequency discrimination in children; perception, learning and attention. *Hear Res* 2008;**238**:147–54
28 Tramo MJ, Cariani PA, Koh CK, Makris N, Braida LD. Neurophysiology and neuroanatomy of pitch perception: auditory cortex. *Ann N Y Acad Sci* 2005;**1060**:148–74
29 Schmuziger N, Patscheke J, Probst R. Hearing in nonprofessional pop/rock musicians. *Ear Hear* 2006;**27**:321–30

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