Hearing Research 317 (2014) 50–62

Contents lists available at ScienceDirect

Hearing Research

journal homepage: www.elsevier.com/locate/heares

Research paper

Amplitude-modulation detection by recreational-noise-exposed humans with near-normal hearing thresholds and its medium-term progression

Michael A. Stone [a, b, *], Brian C.J. Moore [b]

[a] Audiology and Deafness Group, School of Psychological Sciences, University of Manchester, Manchester, M13 9PL, UK
[b] Auditory Perception Group, Department of Experimental Psychology, University of Cambridge, Downing Street, Cambridge CB2 3EB, UK

ARTICLE INFO

Article history:
Received 14 June 2014
Received in revised form
22 August 2014
Accepted 11 September 2014
Available online 27 September 2014

ABSTRACT

Noise exposure can affect the functioning of cochlear inner and outer hair cells (IHC/OHC), leading to multiple perceptual changes. This work explored possible changes in detection of amplitude modulation (AM) at three Sensation Levels (SL) for carrier frequencies of 3, 4 and 6 kHz. There were two groups of participants, aged 19 to 24 (Young) and 26 to 35 (Older) years. All had near-normal audiometric thresholds. Participants self-assessed exposure to high-level noise in recreational settings. Each group was sub-grouped into low-noise (LN) or high-noise (HN) exposure. AM detection thresholds were worse for the HN than for the LN sub-group at the lowest SL, for the males only of the Young group and for both genders for the Older group, despite no significant difference in absolute threshold at 3 and 4 kHz between sub-groups. AM detection at the lowest SL, at both 3 and 4 kHz, generally improved with increasing age and increasing absolute threshold, consistent with a recruitment-like process. However, poorer AM detection was correlated with increasing exposure at 3 kHz in the Older group. It is suggested that high-level noise exposure produces both IHC- and OHC-related damage, the balance between the two varying across frequency. However, the use of AM detection offers poor sensitivity as a measure of the effects.

1. Introduction

Noise-induced hearing damage in humans is associated with an increase in absolute threshold in the frequency range 3–6 kHz, which can later spread to between 2 and 8 kHz (Smoorenburg, 1990). Above 8 kHz, audiometric thresholds may remain near-normal, but with prolonged exposure, the region between 12 and 20 kHz is also affected (Fausti et al., 1981; Hallmo et al., 1995). The earliest sign of damage often takes the form of a notch in the audiogram, centered between 3 and 6 kHz (Fowler, 1929; Coles et al., 2000). The notch can be quite narrow, and can be missed if audiometry is performed only at octave frequencies (West and Evans, 1990).

The audiogram is recognized as being an insensitive measure for quantifying hearing damage, since there may be changes in hearing even when audiometric thresholds are within the "normal" range. Such changes include tinnitus (Davis et al., 1950; Moore, 2012), broadening of the auditory filters (West and Evans, 1990), and reduced otoacoustic emissions (Attias et al., 1998; Hall and Lutman, 1999; Lucertini et al., 2002). Also, substantial loss of synapses and degeneration of neurons in the auditory nerve may occur without any marked effect on the audiometric threshold (Schuknecht, 1993; Kujawa and Liberman, 2009; Lin et al., 2011; Liu et al., 2012). Early detection of "sub-clinical" or "hidden" hearing losses has therefore attracted increasing attention, to allow identification of individuals who are at risk and to take steps to avoid further damage (Fausti et al., 1981; Lucertini et al., 2002; Stone et al., 2008; Schaette and McAlpine, 2011). The present paper describes a study using a perceptual measure, namely the detection of amplitude modulation (AM) applied to a low-level sinusoidal carrier, which might be useful in early identification of one manifestation of noise-induced hearing damage.

With increased regulation to limit noise levels and exposures arising in the workplace, the focus has moved to possible damage from noise exposure in "recreational" settings, which attract little

Abbreviations: AM, amplitude modulation; DPOAE, distortion-product otoacoustic emission; HN, high-noise exposure; IHC, inner hair cell; LN, low-noise exposure; OHC, outer hair cell; PLL, preferred listening level; PMP, personal music player; psd, power spectral density; SL, sensation level; SOAE, spontaneous otoacoustic emission

* Corresponding author. Present address: Audiology and Deafness Group, School of Psychological Sciences, University of Manchester, Manchester, M13 9PL, UK.
E-mail address: michael.stone@manchester.ac.uk (M.A. Stone).

http://dx.doi.org/10.1016/j.heares.2014.09.005

or no regulation. Of particular concern are the risks from personal music players (PMPs) and amplified music events. In the UK, the prevalence of these contributions to noise exposure was estimated to have tripled between 1980 and 1994 (Smith et al., 2000). Although combinations of PMPs and headphones/earphones are capable of producing sound levels in the range 90–120 dBA (Fligor and Cox, 2004), preferred listening levels (PLL) are generally much lower than this (Kuras and Findlay, 1974; Bradley et al., 1987; Williams, 2005; Torre, 2008; Worthington et al., 2009; Shimokura and Soeta, 2012). The PLL found in such studies indicate that the exposures would be regarded as potentially injurious only with prolonged listening, as defined by the methods used in industrial regulation (ISO 1999, 1990), although PLLs and exposure durations can vary significantly with factors such as gender and ethnicity (Torre, 2008; Vogel et al., 2008). Exposures of shorter duration, but with the potential to cause damage, have been have been found to occur in night clubs or at rock concerts, where levels in excess of 95 dBA are regularly encountered (Sadhra et al., 2002; Bray et al., 2004; Santos et al., 2007; Stone et al., 2008; Potier et al., 2009). In some of these reports, mean exposures of around 110 dBA were not uncommon, but with durations not exceeding a few hours.

The pattern of sound-induced damage and its progression in humans is not easy to determine. The "equal energy" hypothesis, which forms the basis of workplace regulation, assumes that physiological damage is proportional to energy received (Ward et al., 1981). However, it has long been recognized that additional factors play a role, such as the frequency and duration of "recovery" periods and the degree of impulsiveness of the sound (Ward, 1970; Ward et al., 1981; Davis et al., 2009). Ward et al. (1981) also noted that, above a certain "critical" sound level, more damage was observed than predicted by the equal-energy hypothesis. Caution is needed when comparing animal to human data, since noise damage in animals is usually produced by narrowband continuous steady signals, while the sounds experienced by humans are usually broadband, and vary markedly in spectrum and in temporal pattern. Borg et al. (1995) proposed that, for humans, OHC damage resulted from prolonged exposures to moderately high intensities, whereas loss of IHC function was associated more with impact or very high-intensity sounds, implicitly implying that there was a critical level for humans.

The perceptual consequences of damage to the IHCs, loss of synapses, and degeneration of primary auditory neurons are likely to be similar, in that all would result in a reduced fidelity of coding of information in the auditory nerve. In what follows, dysfunction in IHCs/synapses/neurons is referred to as IHC dysfunction for brevity. Such dysfunction would be expected to lead to impaired performance in discrimination tasks, but to have little or no effect on absolute thresholds, since only a few functioning neurons are required for detection of a signal. Conversely, OHC damage would be expected to produce reduced gain of the cochlear amplifier (Robles and Ruggero, 2001), thereby elevating absolute thresholds, and to loss of cochlear compression, perhaps affecting the perception of loudness (Moore and Glasberg, 2004) and of envelope fluctuations (Moore et al., 1996).

A few studies have employed psychoacoustical measures in addition to the audiogram in attempts to detect early hearing damage in humans. Studies with animals (Kujawa and Liberman, 2009; Furman et al., 2013) suggest that noise exposure initially leads to neuropathy mainly for neurons with low spontaneous rates and medium to high thresholds. This leads to the prediction that perceptual deficits should be apparent at medium to high presentation levels. Consistent with this, Kumar et al. (2012) performed all of their testing in humans at 80 dB SPL, and identified deficits in some psychoacoustic and speech perception tasks for their noise-exposed group.

A disadvantage of presentation at high levels is that the stimuli produce excitation of the cochlea over a considerable portion of its length, and hence performance is based on the integrated outputs of many neurons. Evidence from humans (Stone et al., 2008; Vinay and Moore, 2010) suggests that some forms of sub-clinical damage may be localized on the cochlea, and their observable effects may therefore be diluted by use of signals producing widespread excitation. An alternative approach is to use narrowband stimuli presented at low sensation levels (SLs), so as to restrict stimulus-evoked excitation to a small region of the cochlea. Any perceptual effects measured with such stimuli are likely to reflect mechanisms separate from that identified by Kujawa and Liberman (2009) and Furman et al. (2013).

Vinay and Moore (2010) investigated the perceptual effects of PMP use for male participants who habitually listened at self-judged high replay levels (not measured) for at least 2 h per day. Male participants were used because of their tendency to use higher levels than females (Torre, 2008). Vinay and Moore reported that PMP users had better AM detection thresholds but poorer frequency discrimination thresholds than a control group, even though both groups had audiometric thresholds within the normal range. Vinay and Moore proposed that the better AM detection thresholds might be a result of OHC dysfunction, which leads to steeper input–output functions on the basilar membrane (Robles et al., 1986) and increases the perceived magnitude of amplitude fluctuations (Moore et al., 1996). They suggested that the poorer frequency discrimination thresholds could result from reduced frequency selectivity associated with OHC dysfunction, or reduced sensitivity to temporal fine structure, perhaps associated with IHC dysfunction (Moore, 2014), or both.

Stone et al. (2008) tested an experimental group comprising rock musicians and attendees at night clubs, who were regularly exposed to sound levels above 100 dBA (verified by a dosimeter), but only for a few hours per week. The task was to discriminate a Gaussian noise from a low-noise noise (Pumplin, 1985) with reduced envelope fluctuations. The experimental group performed more poorly than a non-exposed control group, even though both groups had audiometric thresholds within the normal range. This pattern was attributed to IHC dysfunction in the experimental group, which would have led to a "noisier" neural representation of the signal envelope.

Vinay and Moore (2010) found better AM detection for their exposed group, while Stone et al. (2008) found poorer envelope discrimination for their exposed group. The apparent discrepancy may have occurred because PMP use involves sub-critical levels, while attending rock/club events involves at least some super-critical levels, leading to IHC damage. However, the discrepancy may also be related to the difference in the tasks used (AM detection versus envelope discrimination) and in the center frequencies used (0.5, 3, 4, and 6 kHz for Vinay and Moore; 2, 3 and 4 kHz for Stone et al.). Also, both studies had the limitation that the control group had a 6–7 year greater mean age than the experimental group, and there was a large range of ages within all groups.

The present study was intended to explore the origin of the discrepancy between the results of the two studies discussed above, while controlling more precisely for the effects of age. Since the task used by Stone et al. (2008) required rather a lot of training to achieve stable results, the present study used an AM-detection task similar to that employed by Vinay and Moore (2010). The specific questions addressed were:

(1) Does AM-detection performance for groups who are probably exposed to super-critical levels depend on the amount of the exposure? To address this, two separate experimental groups were enrolled, with ages 19 to 24 and 26–35 years.