

HEARES 01627

# Development of resistance to hearing loss from high frequency noise

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The effect of interrupted exposure on the development of progressive resistance to hearing loss from exposure to high frequency noise was studied using monaural chinchillas. The animals were exposed to an octave band noise centered at 4 kHz at 85 dB SPL for 6 h a day for ten consecutive days. Hearing thresholds were measured using evoked potential recording before and after each exposure. Results indicated a reduction in threshold shift with repeated exposures. This reduction in threshold shift or 'toughening' was more rapid for the high frequency exposures than the 'toughening' from similar low frequency exposures.

'Toughening'; Resistance; Frequency of exposure

## Introduction

Several studies have demonstrated that repeated exposures to a low frequency noise results in a progressive reduction in the threshold shift or 'toughening' towards further exposures to the same noise (Clark et al., 1987; Canlon et al., 1988; Henderson et al., 1991). Limited data exists regarding the auditory system's response to repeated exposures to high frequency noise (Clark and Bohne, 1991). There are a number of systematic differences between the basal (high frequency) and the apical (low frequency) parts of the cochlea in terms of basic anatomy, tonotopic organization and in the protection from and reaction to noise (Altschuler et al., 1991). Therefore, this study was designed to determine the role of exposure frequency on the development of progressive resistance to noise induced hearing loss. The results would contribute to our knowledge of the acoustic parameters governing the 'toughening' phenomenon. Such information would be crucial to the development of prophylactic exposure schedules as well as to provide directions for future research and to understand the underlying physiological mechanisms of the 'toughening' process.

## Methods

### Subjects

Six adult chinchillas (500 to 700 g) served as subjects. Each animal was anesthetized with a subcuta-

neous injection of acepromazine (0.56 mg/kg) and ketamine (36 mg/kg) and made monaural by surgical destruction of the left cochlea. A chronic recording electrode was then stereotaxically implanted into the left inferior colliculus and a ground electrode was implanted just below the dura mater (Henderson, et al. 1973). Following surgery, the animals were given antibiotics (chloramphenicol palmitate twice a day for four days) and allowed to recover for at least two weeks prior to testing.

### Audiometry

After the two week recovery period hearing thresholds were measured using evoked potential recording at octave frequencies from 0.5 to 16 kHz and at the mid-octave frequency of 5.6 kHz. Each animal was tested five times. The thresholds remained stable across the five tests. The average of these five measures constituted the pre-exposure threshold. In addition, the animals were also tested each day just before and just after the exposure. Each animal was tested separately in a sound treated booth. A yoke-like harness kept the animal's head in a fixed position within the calibrated sound field.

Test stimuli consisted of tone pips (5 ms Blackman rise/fall ramp, constant starting phase) in the frequency range of 0.5 to 16 kHz at octave intervals including the mid-octave frequency of 5.6 kHz. For details regarding stimulus generation and analysis of responses refer to Subramaniam et al. (1991).

### Noise exposure

The noise (Gaussian) was generated by a D/A converter on a signal processing board (Loughborough TMS 32020) in a personal computer (IBM compatible).

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It was routed through an attenuator (HP 350 D), a filter (Krohn-Hite 3550 R) and a power amplifier (NAD 2200) to an acoustic horn (JBL 2360).

All the animals were exposed to an octave band noise centered at 4 kHz for 6 h a day for ten consecutive days. The animals were exposed in groups of two or three. The animals were placed in the exposure booth at intervals of 20 min to enable threshold measurements just before and just after each noise exposure. Each animal was housed in a separate cage (8 inches  $\times$  8 1/2 inches  $\times$  8 inches) and given free access to food and water. The cages were placed just below the loud speaker such that the difference in the sound pressure across the cages was less than 1 dB. The animals were rotated to different cages to minimize the effects of differences in sound pressure, if any.

**Results**

*Pre-exposure thresholds*

The mean pre-exposure thresholds (Fig. 1) were consistent with the laboratory reference norms and with behavioral thresholds reported by other investigators (Miller, 1970). The inter-subject variability was small with standard deviations of  $\pm 5$  dB or less across the test frequencies.

*Post-exposure threshold shift*

Each animal was tested immediately after the daily noise exposure in an ascending order of frequency and intensity. A given frequency was thus tested at approximately the same time every day. The total test duration was about 15 min. The average audiogram (mean of six animals) obtained at the end of the first and the last

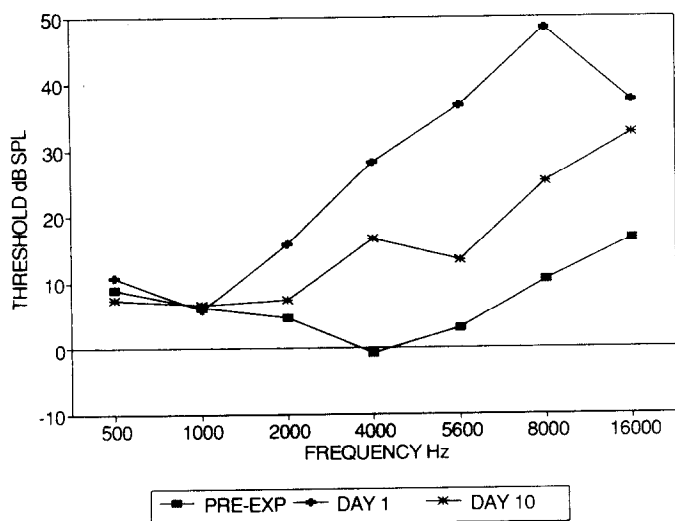


Fig. 1. A comparison of the mean thresholds at the end of first and the last exposures. The mean pre-exposure thresholds are also plotted for comparison.

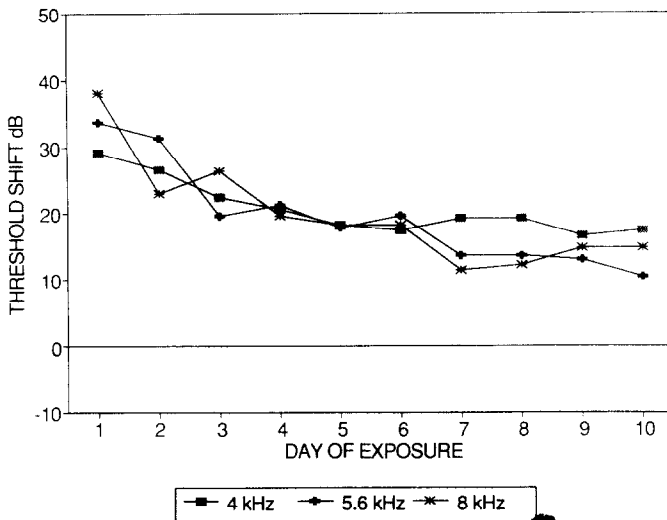


Fig. 2. Mean post-exposure threshold shifts at 4, 5.6 and 8 kHz on the ten days of exposures.

days of the exposure are plotted along with the mean pre-exposure thresholds in Fig. 1. It may be seen that there were no significant threshold shifts at 0.5 and 1 kHz. At 2 kHz an average shift of 10 dB was observed on the first day which dropped to less than 5 dB by day 10. At higher frequencies (4, 5.6 and 8 kHz) there was a 30 to 40 dB shift at the end of the first day of exposure, but by the end of the tenth day the shift had reduced by 12 dB at 4 kHz and by 24 dB at 5.6 and 8 kHz. A small decrease (5 dB) was seen at 16 kHz.

The time course of decrease in the threshold shift at 4, 5.6 and 8 kHz is presented in Fig. 2. The post-exposure threshold shift was defined as the difference between the subject's threshold obtained at the end of each day's exposure and its mean pre-exposure threshold. It may be seen that the threshold shift is greatest

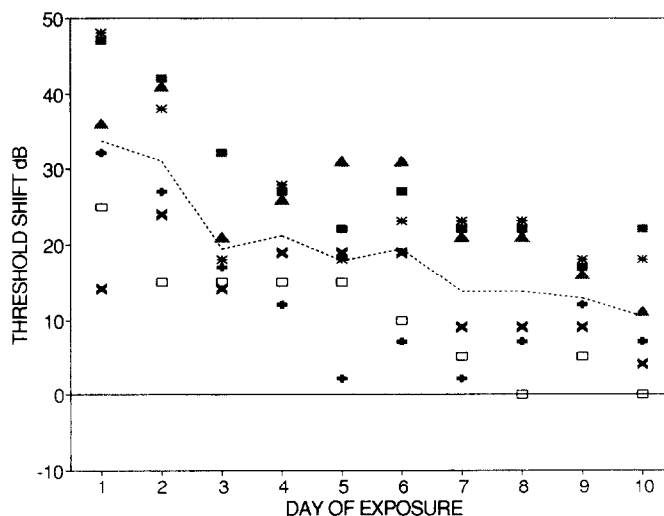


Fig. 3. Post-exposure threshold shifts at 5.6 kHz. Symbols represent data from individual animals. Dotted line represents mean.

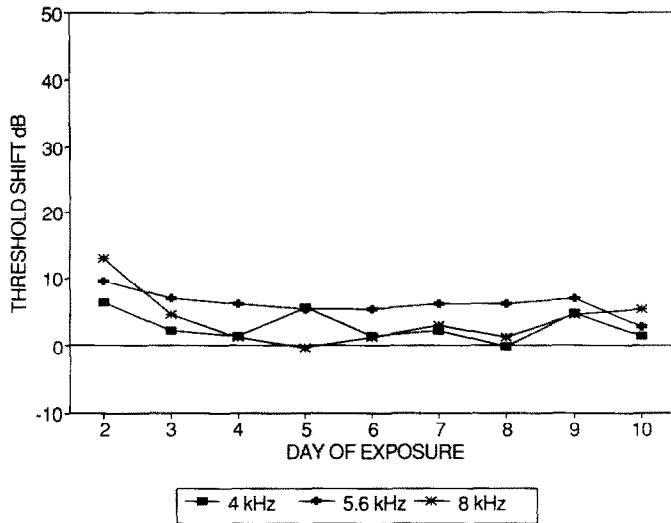


Fig. 4. Mean residual hearing loss at 18 h post-exposure, on the ten days of exposure.

on day 1 at all the frequencies. The graphs show that the threshold shift decreased considerably from day 1 to day 10 at all the frequencies, although the magnitude of reduction appeared to depend on the frequency. The mean post-exposure threshold shift showed a 12 dB decrease at 4 kHz and a decrease of 24 dB each at 5.6 and 8 kHz.

Although there were some differences in the initial threshold shift across the animals at various frequencies, all the animals showed decreased threshold shifts with repeated exposures (Fig. 3). The variability seen at 5.6 kHz is representative of the test frequencies where a significant shift was seen.

Further analysis of the data revealed no consistent relation between the magnitude of threshold shift on the first day and the decrease in the threshold shift over the ten days. While the correlation was high at 5.6 and 16 kHz ( $r = 0.8$ ) the correlation was less than 0.5 at 4 and 8 kHz.

#### Residual hearing loss

Threshold shifts recorded just before the second day of exposure (after about 18 h of recovery) indicated that the animals had an average residual hearing loss of about 10 dB across the different frequencies (Fig. 4). After the last exposure the thresholds returned to the baseline between 48 to 72 h after the last exposure.

#### Discussion

The results of the present study are in general agreement with those of Clark and Bohne (1991) in that both the studies demonstrate a decrease in threshold shifts with repeated exposures to a high frequency noise. There are also certain differences in the results

of the two studies. For instance, Clark and Bohne reported of initial shifts of 45 to 55 dB (at test frequencies from 4 to 5.7 kHz) from 6 h exposures at 86 dB SPL. This is considerably higher than the initial shifts recorded in the current study. The 1 dB difference in the exposure level could explain a difference of 1.6 to 1.9 dB in the threshold shift (Mills, 1973). The duration of the test stimulus may be a second and possibly more important contributor to the difference in the threshold shifts reported in the two studies. In the present study thresholds were measured in response to short duration tone bursts which elicited an onset response, thus, the threshold shifts primarily reflect changes in sensitivity. Clark and Bohne (1991) used long duration stimuli and hence their post-exposure threshold shifts would be reflective of both diminished hearing sensitivity as well as decreased temporal integration function. Furthermore, Clark and Bohne employed behavioral audiometry whereas in the current study evoked potential recordings were used. Though highly reliable, results of behavioral tests may be influenced by the animal's physiological and/or psychological state such as fatigue, tinnitus, attention etc. following the exposure and this could have resulted in higher thresholds. In spite of these differences it is important to note that both the studies concur in that the 'toughening' can be seen even with high frequency exposures. However, the results of both the investigations are in contrast with those of Saunders et al. (1977). While the reason for this discrepancy is unclear, Clark et al (1987) do suggest that the use of an extremely narrow aspect ratio employed in the data presentation might have de-emphasized the small but significant changes in the thresholds over the days of exposure.

While the high frequency exposures produced a 'toughening' effect, there are certain differences between the pattern of 'toughening' with high and low frequency exposures (Subramaniam et al., 1991). When the results of the 4 kHz and 0.5 kHz OBN exposures are compared for the frequencies one octave above the exposure frequency (Fig. 5), it may be seen that the high frequency exposure produces a more rapidly progressing resistance to noise. For instance, for the high frequency exposure, at one octave above the exposure frequency (8 kHz) the mean threshold shift decreased from 38 dB to 22 dB by the end of the second day of exposure (Fig. 5). Contrarily, the 0.5 kHz exposure resulted in a more progressive reduction beginning on day 6. While the threshold shift appears to have plateaued with less than 5 dB variation from day 7 to day 10 with the high frequency exposure, no such plateau is seen with the low frequency exposure.

The differences in the results of the low and high frequency exposures were also seen in the residual hearing loss. A comparison of the residual hearing loss at one octave above the exposure frequency indicates

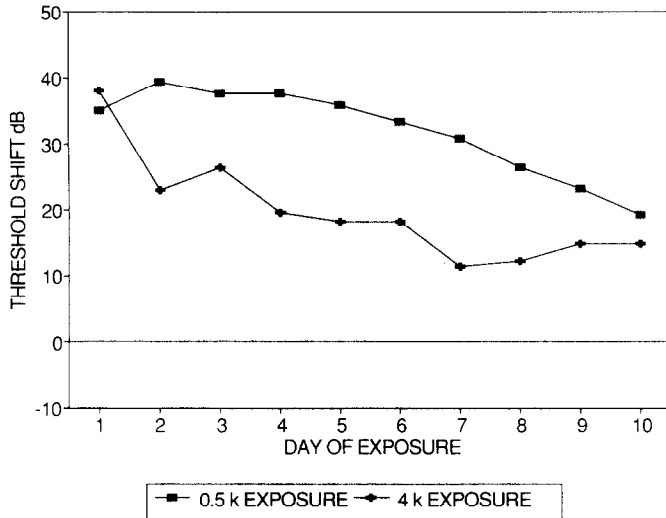


Fig. 5. A comparison of the post-exposure threshold shift at one octave above the exposure frequency.

that the high frequency exposure gave rise to little or no residual hearing loss (Fig. 6). On the other hand, a considerable residual hearing loss persisted in the case of the low frequency exposure, so that just before the tenth day of exposure it was about 10 dB greater than the residual hearing loss from the high frequency exposure.

The results thus emphasize the differences in the base and the apex of the cochlea in their responses to noise exposure (Bohne et al., 1987; Altschuler et al., 1991). While the reasons for the differences in 'toughening' are not clear, they may hold some clues to the differences in the mechanisms. A pilot study in our laboratory on the effect of the COCB on 'toughening' produced by low frequency exposures indicated that a sectioning of the COCB results in a loss of 'toughening' at the high frequencies while 'toughening' is pre-

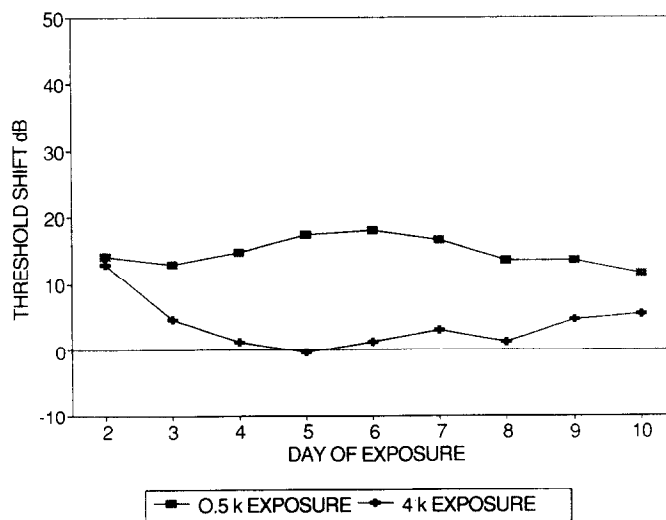


Fig. 6. A comparison of the residual hearing loss at one octave above the exposure frequency.

served at the low frequencies (Fiorino et al., 1989). The persistence of 'toughening' at low frequencies might be indicative of the acoustic reflex being the moderator of 'toughening' at low frequencies. However, detailed investigations are warranted for a better understanding of the mechanisms underlying 'toughening'.

## Conclusions

The results of this study demonstrate that considerable 'toughening' may be observed even at high frequencies. A comparison of the results with those of identical exposures at a low frequency suggests that the mechanisms underlying 'toughening' may be different at low and high frequencies and calls for more detailed studies to understand these mechanisms.

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