

Threshold shifts and enhancement of cortical evoked responses after noise exposure in rats

Josef Syka *, Natalia Rybalko

Institute of Experimental Medicine, Academy of Sciences of the Czech Republic, Vídeňská 1083, 142 20 Prague, Czech Republic

Received 17 February 1999; received in revised form 30 May 1999; accepted 9 September 1999

Abstract

The effect of exposure to various types of noise (broadband, high-frequency or low-frequency) was studied in adult pigmented rats. Thresholds and amplitudes of middle latency responses (MLR) recorded from electrodes implanted on the surface of the auditory cortex were analyzed before and after noise exposure. Exposure to noise with intensities ranging from 105 to 120 dB for 1 h produced only temporary threshold shifts (TTS). Exposure to broadband noise produced TTS throughout the whole frequency range of the rat's hearing, mostly expressed at frequencies of maximal hearing sensitivity (16–32 kHz). Hearing loss produced by high- or low-frequency noise exposure was related to the spectral characteristics of the noise. The exposure to high-intensity noise may also result in amplitude enhancement of the MLR. This phenomenon was seen mainly after broadband noise exposure and occurred in response to both low-frequency and high-frequency test stimuli. High-frequency and low-frequency noise produced amplitude enhancement mainly at frequencies which corresponded to the maximum exposure energy. In contrast to the relatively similar values of TTS obtained in different rats under the same conditions of noise exposure, great inter-individual variability was found in the MLR amplitude enhancement. In all rats the dynamics of recovery functions for amplitude enhancement were different from those for MLR thresholds. The data indicate that whereas post-exposure TTS are related to peripheral changes, the post-exposure MLR amplitude enhancement is most probably connected with a change in the processing of auditory information in the central nervous system. © 2000 Elsevier Science B.V. All rights reserved.

Key words: Noise exposure; Threshold shift; Evoked response; Enhancement; Rat

1. Introduction

Changes in auditory function after noise exposure are traditionally interpreted as the result of mechanical damage to the cochlear structures (for review see e.g. Syka, 1989; Axelsson et al., 1996). However, accumulated evidence exists that changes produced by acoustic trauma also comprise changes in the higher levels of the auditory system. It was demonstrated that partial or complete inner ear injury may induce functional and morphological reorganization of auditory nuclei (Taniguchi and Saito, 1978; Reale et al., 1987; Robertson and Irvine, 1989; Møller, 1990; Popelář et al., 1994). Acoustic overstimulation, as an example of partial and

temporary deafferentation, may result in enhanced spontaneous and evoked-unit activity in the cochlear nucleus or inferior colliculus (Henderson and Møller, 1975; Lonsbury-Martin and Martin, 1981; Willott and Lu, 1982; Salvi et al., 1982, 1992; Kaltenbach et al., 1998; Zhang and Kaltenbach, 1998), in the enhancement of evoked response amplitudes in the inferior colliculus (Willott and Henry, 1974; Bock and Saunders, 1976; Salvi et al., 1990; Szczepaniak and Møller, 1996) or increased amplitudes of middle latency responses (MLR) recorded at the auditory cortex (Popelář et al., 1987; Syka et al., 1994).

In our previous works (Popelář et al., 1987; Syka et al., 1994), MLR amplitude enhancement was studied in guinea pigs. Guinea pigs may be characterized as animals with a relatively broad frequency hearing range, which also includes low frequencies (even below 100 Hz). Their best sensitivity of hearing spans from 8 to

* Corresponding author. Tel.: +420 (2) 475 22 30;
Fax: +420 (2) 475 27 87; E-mail: syka@biomed.cas.cz

12 kHz. These studies showed that MLR amplitude enhancement in guinea pigs after noise exposure was found only when low-frequency tones were used as test stimuli. Therefore it would be of interest to study the phenomenon of MLR amplitude enhancement under similar experimental conditions in animals with hearing range shifted to high frequencies. Rats represent such a species, with the frequency hearing range spanning from approximately 800 Hz to 65 kHz, with the best sensitivity between 8 and 32 kHz.

The aim of the present study was to evaluate the effects of exposure to various types of noise on the MLR recorded in the auditory cortex in the rat. The rat was selected as an experimental animal also because it is a widely used animal model for the study of the structure and function of the central nervous system. However, in contrast to the guinea pig, chinchilla or gerbil, data concerning the influence of noise exposure on hearing function in rat are very scarce (Lenoir et al., 1979; Borg, 1982; Szczepaniak and Møller, 1996).

2. Materials and methods

Experiments were performed on 28 adult (3–5-month-old) female pigmented rats (strain Long-Evans) weighing 250–300 g. The MLR were recorded with chronically implanted teflon-coated platinum iridium ball electrodes (ball diameter 0.5 mm) fixed on the surface of the primary auditory cortex. The reference electrode was placed in the neck muscles. All electrodes were soldered to pins of a connector socket mounted on the skull with stainless steel screws and acrylic resin. During the implantation, the animals were anesthetized with an intraperitoneal injection with a mixture of ketamine (Narkamon 5%, Spofa) and xylazine (Rompun 2%, Bayer) (ratio 3:1, dose 0.1 ml/100 g b.w.).

The control pre-exposure measurement of the MLR was performed in lightly sedated animals (xylazine 0.03 ml/100 g b.w. injected intramuscularly) after 1 week recovery from surgery. The next day awake animals were exposed for 1 h to one of three types of noise: (i) broadband noise; frequency range 0.8–20 kHz, intensity either 120 dB SPL (5 rats) or 110 dB SPL (4 rats); (ii) high-frequency narrow-band noise (1/3 octave band) centered at 16 kHz, intensity 120 dB SPL (5 rats) or 105 dB SPL (4 rats); (iii) low-frequency noise; frequency range 0.1–7 kHz with maximum energy around 1 kHz, intensity 120 dB SPL (6 rats) or 115 dB SPL (4 rats). Post-exposure recording of MLR was also conducted in lightly sedated rats during the next 4 weeks (1 h, 1 day, 3 days and 1–4 weeks after noise exposure). Potentials were amplified by a differential AC amplifier (gain 80 dB, filters 10 Hz–3 kHz) and averaged with the aid of a computer combined interface CED 1401 plus

(12 bit A/D converter, sampling rate 5 kHz) with Sig-AVG software.

Free-field stimulation was carried out in an anechoic sound-attenuated room. Test stimuli were tone bursts (duration 5 ms with a 2 ms rise-fall time) of different intensities in a frequency range from 0.5 to 64 kHz or condensation clicks (duration of the electrical pulse 100 μ s). Stimulus intensity was increased in 5 or 10 dB increments from 0 dB SPL to 110 dB SPL. The stimulus repetition rate was 2 Hz. Sound stimuli were presented from a loudspeaker (Tesla ARS 824 or Motorola KSN 6005) which was placed 30 cm in front of the animal's head. Calibration of the sound field was performed with a B&K microphone (type 4134) and B&K amplifier (type 2603). The microphone was placed in the position where the animal's head was placed during the experiment.

Thresholds of MLR to tone bursts at 0.5, 1, 2, 4, 8, 16, 24, 32 and 64 kHz were determined from the screen of a computer using a criterion of a just noticeable deflection of the averaged electrical activity around the baseline. For click, low- and high-frequency test stimuli (tones 1 and 24 kHz), amplitude-intensity functions were constructed. Data before and after noise exposure were compared. In addition, changes in MLR amplitudes, recorded in response to test stimuli of the same intensity level, i.e. 80 dB above pre-exposure threshold, were analyzed. In these cases the real values of the intensity level of test stimuli ranged in different rats between 85 and 90 dB SPL for click and 24 kHz tone burst and between 105 and 110 dB SPL for 1 kHz tone burst. Peak-to-peak amplitude of the positive component with a peak latency of about 20 ms and the negative component with a peak latency of about 25 ms were measured. Relative MLR amplitudes were compared before and after exposure. The pre-exposure MLR amplitude in response to stimuli with an intensity 80 dB above pre-exposure threshold was taken as 100%. The intensity of 80 dB above threshold (SL) was chosen because in most rats under normal conditions, stimuli of this intensity evoked stable MLR with the highest amplitude. In some animals the increase of sound intensity above 80 dB SL was associated with no increase of amplitude or even a decrease.

The care and use of animals reported on in this study were approved by the Ethics Committee of the Institute of Experimental Medicine and followed the guidelines of the Declaration of Helsinki.

3. Results

In all investigated rats noise exposure produced temporary threshold shifts (TTS). The magnitude and pattern of hearing loss were dependent on the spectral

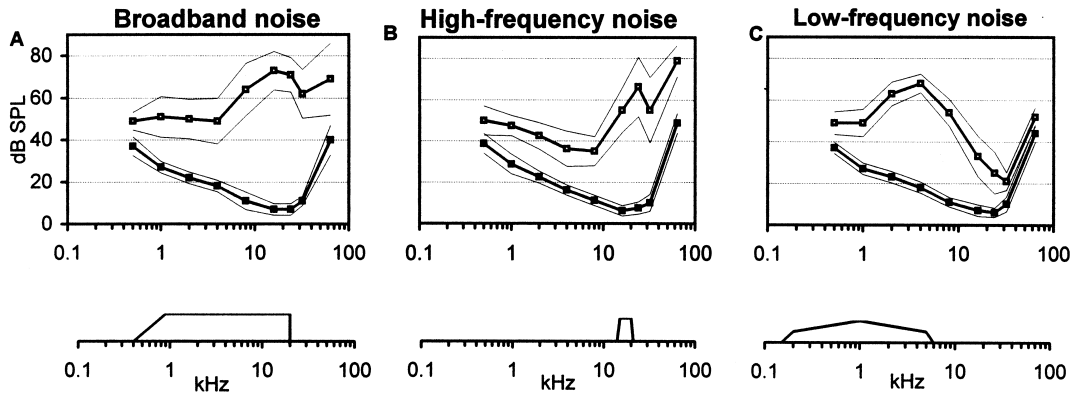


Fig. 1. Average MLR thresholds measured in rats before (full squares) and 1 h after 120 dB SPL exposure (open squares) to (A) broadband noise; (B) high-frequency noise; (C) low-frequency noise. The frequency spectrum of the noise is indicated schematically in the bottom part of the graph. Lines above and below average values represent standard deviation.

characteristics of the noise and its intensity, and were also related to hearing sensitivity at different frequencies. Exposure to 120 dB SPL broadband noise produced TTS throughout the whole frequency range of the rat's hearing, mostly expressed at frequencies of the rat's maximal hearing sensitivity (8–32 kHz). The maximal TTS value of 66 ± 8.2 (S.D.) dB was found at 16 kHz 1 h after exposure (Figs. 1A and 2A). Exposure to 110 dB SPL broadband noise evoked a similar pattern of hearing loss as did the 120 dB SPL exposure, however, of smaller magnitude. Maximal TTS, recorded 1 h after noise exposure, in this case reached 43.5 ± 5.7 dB at 16 kHz (Fig. 2B). The results of narrow-band noise exposure were dependent on the spectral characteristics of the noise: the largest TTS were observed in a

frequency range corresponding to the frequency of the maximum exposure energy shifted to higher frequencies by about 0.5–2 octaves. Exposure to 120 dB SPL high-frequency noise produced the most pronounced TTS in the frequency range of 16–32 kHz with a maximal value of 60 ± 14.1 dB at 24 kHz 1 h after exposure (Figs. 1B and 2C). 105 dB SPL high-frequency noise exposure evoked smaller TTS in comparison with the 120 dB exposure amounting to 30 ± 10.9 dB at 24 kHz 1 h after noise exposure (Fig. 2D). Exposure to low-frequency noise produced the most pronounced TTS in the range of 2–8 kHz. The maximal threshold shift was recorded 1 h after noise exposure at 4 kHz (Figs. 1C and 2E). The magnitude of the hearing loss at the maximum TTS produced by low-frequency noise was smaller than the

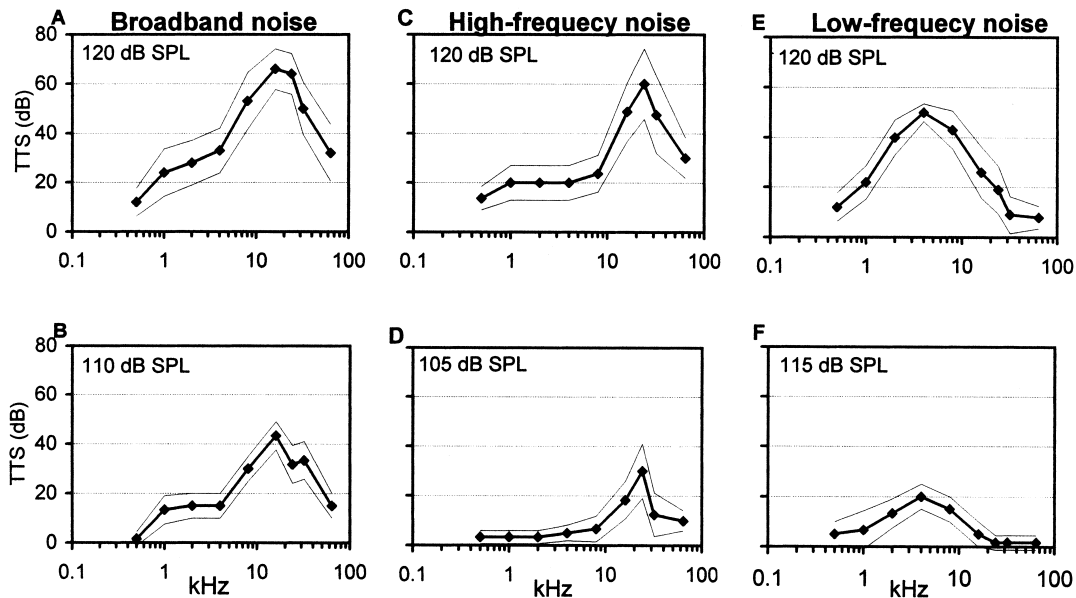


Fig. 2. Average threshold shifts (with standard deviations) as a function of test frequency determined 1 h after broadband (A, B), high-frequency (C, D) and low-frequency (E, F) noise exposure with different intensities (indicated in the upper left corner).

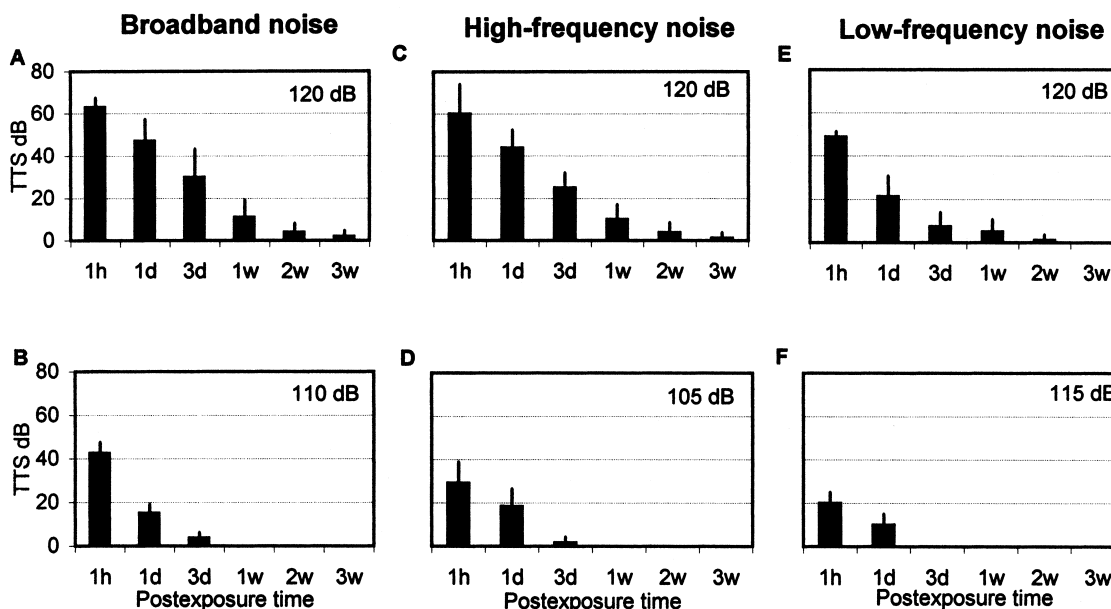


Fig. 3. Recovery of mean maximal threshold shifts after broadband (A, B), high-frequency (C, D) and low-frequency (E, F) noise exposure with different intensities as a function of the post-exposure time. Vertical bars represent standard deviation.

corresponding TTS maximum produced by broadband or high-frequency noise exposure and reached 50 ± 3.5 dB and 20 ± 5 dB respectively, when 120 dB SPL and 115 dB SPL noise were used (Figs. 1C and 2E,F).

Fig. 3 illustrates the recovery of the mean TTS after broadband, high-frequency and low-frequency noise exposure as a function of the post-exposure time. The maximal threshold shift was found immediately after the noise exposure for all exposure conditions (i.e. within 1 h after noise exposure when the measurements were performed). Hearing thresholds returned to pre-exposure levels in 2 weeks, when 120 dB noise exposure was used (Fig. 3A,C,E). The recovery period of TTS following noise exposure of lower intensities was shorter and lasted up to 3 days (Fig. 3B,D,F). No permanent threshold shift was found in any of our experimental animals.

The acoustic trauma caused by the noise exposure resulted also in an enhancement of MLR amplitudes (Figs. 4–8). This phenomenon was mainly observed after broadband noise exposure. 120 dB SPL broadband noise exposure produced an enhanced MLR amplitude, which was recorded in all rats in response to clicks and tone bursts of 24 kHz (testing in the range of maximal TTS). Fig. 4 demonstrates a typical case of the development of MLR amplitude enhancement after 120 dB SPL broad-band noise exposure. MLR to a 80 dB SL tone of 24 kHz (which represents 90 dB SPL) recorded before and after noise exposure are shown. Fig. 5 shows MLR amplitude-intensity functions to tones of 24 kHz (A) and 1 kHz (B), measured in the same rat before and at different times after noise exposure. For a

24 kHz test tone, 1 h after exposure TTS reaches 70 dB SL and then slowly returns to normal pre-exposure values in approximately 2 weeks. At 1 h after exposure the MLR amplitudes are lower or do not exceed the control level; the amplitude-intensity function can be investigated only in a very limited range (10 dB) due to the large TTS. Later, the hearing range for intensity broadened and the amplitudes of MLR increased, reaching their maximum in 1 week. During the second week after exposure, the amplitude-intensity function returns to its pre-exposure value. Fig. 5B demonstrates that the TTS at a frequency of 1 kHz amounts to 40 dB SL 1 h after exposure. The amplitude enhancement at this frequency is less pronounced than at 24 kHz, however, the time course of amplitude changes is similar except for a difference with regard to the maximal increase in the MLR amplitude, which was observed 1 day after noise exposure and which remained at this level for 1 week.

The amplitude enhancement was different in different rats under the same exposure conditions and also its time course was variable. Fig. 6 shows values of MLR amplitudes for tones of 24 kHz and 1 kHz at the same intensity level (80 dB above pre-exposure threshold) before and after 120 dB SPL broadband noise exposure in five rats (individual data). The absolute values of the intensity of test stimuli for different rats were in the range of 85–90 dB SPL for the 24 kHz stimuli and 105–110 dB SPL for the 1 kHz stimuli. The amplitudes are represented as values relative to the pre-exposure MLR amplitude at 80 dB SL taken as 100%. One hour after noise exposure, when TTS was maximal, the amplitude of the MLR in most cases was noticeably

MLR to 24kHz, 90dB SPL

Broadband noise exposure, 120dB SPL

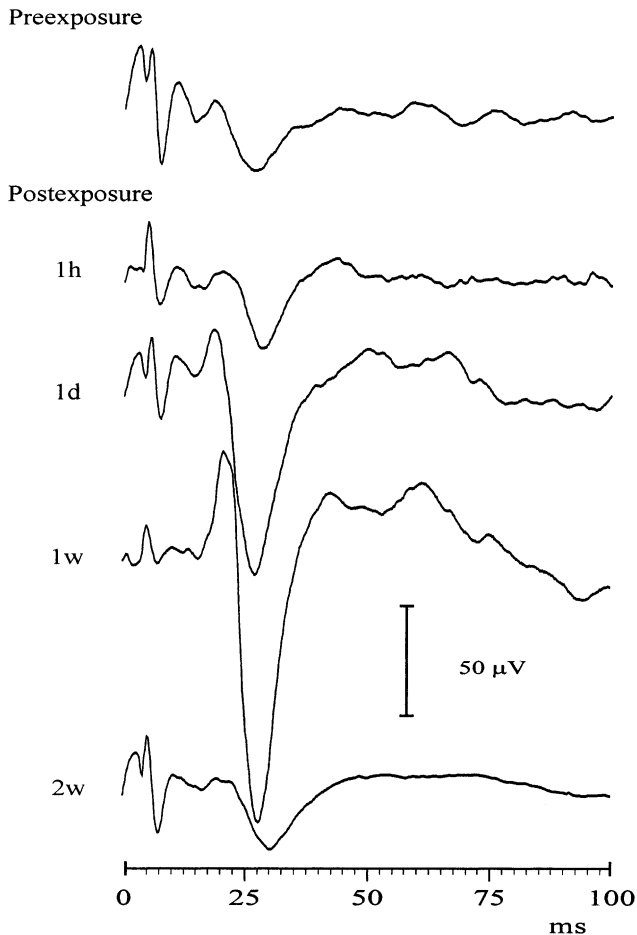


Fig. 4. Middle latency responses to a 90 dB SPL tone at 24 kHz before and after 120 dB SPL broadband noise exposure. Negative polarity is downward. Vertical bar indicates 50 μ V.

smaller than the pre-exposure amplitude for a 24 kHz test tone and did not exceed the pre-exposure value for 1 kHz. The increase in the MLR amplitude appeared the next day after exposure and was present for 1 week. The amplitude enhancement was observed in all five rats but varied from 138 to 600% of the pre-exposure level for 24 kHz. An enhanced MLR amplitude for a tone of 1 kHz (testing in the frequency range of minimal TTS) was found in only three of five rats, in those which had a greater MLR amplitude increase at 24 kHz (Fig. 6B).

Recovery functions of the amplitude enhancement and threshold shift had different time courses in individual animals. Fig. 7A–D demonstrates the recovery function for threshold and amplitude of MLR to

high-frequency (24 kHz) and low-frequency (1 kHz) tones, recorded during 4 weeks following 120 dB SPL broadband noise exposure (individual data for four rats). MLR amplitudes at an intensity of 80 dB above pre-exposure thresholds were compared. The length of the recovery period for the MLR amplitude was similar to that for the hearing threshold and in most cases amounted to 1–2 weeks. However, the dynamics of the recovery of both parameters were quite different: while TTS decreased continuously during the whole recovery period, MLR amplitude increased during the first 1–7 days after noise exposure and then declined. Fig. 7 demonstrates the independence of the processes of threshold shift and amplitude enhancement. In different rats a similar TTS at a certain frequency can be connected with a different amplitude enhancement (Fig. 7A–D). On the other hand, in the same rat both a large TTS at 24 kHz and a relatively small TTS at 1 kHz can be accompanied by a significant amplitude enhancement (Fig. 7A,B). Large interindividual variability of amplitude enhancement was observed together with relatively similar TTS in different rats under the same conditions of noise exposure.

The amplitude enhancement was observed only rarely when the intensities of the noise exposure were lower than 120 dB SPL. From four rats exposed to 110 dB SPL, only in one animal, and only for high frequency test stimuli (24 kHz), did an amplitude enhancement amounting to 240% of the pre-exposure level appear.

The amplitude enhancement was also less pronounced in cases when high-frequency and low-frequency noise exposure were used. After 120 dB SPL high-frequency noise exposure, MLR amplitude enhancement was observed in only one of five noise-exposed rats and only at high-frequency tones (24 kHz). The largest magnitude of the amplitude enhancement amounted to 130% of the pre-exposure level (Fig. 8A). Amplitude enhancement for low-frequency tone (1 kHz) after high-frequency noise exposure was not recorded (Fig. 8B). In contrast to this, for low-frequency noise exposure, amplitude enhancement was seen predominantly at low-frequency tones (1 kHz). The enhancement was observed in three of six rats, and amounted to 164–244% of the pre-exposure values. Only in one rat was amplitude enhancement recorded both for tones of 1 kHz and for tones of 24 kHz (Fig. 8C,D). Narrow-band noise exposure of an intensity less than 120 dB SPL did not evoke MLR amplitude enhancement. The recovery period for MLR amplitude under conditions of narrow-band noise exposure usually did not exceed 1 week.

In all cases in which the amplitude enhancement was present to stimulation with tone bursts, stimulation with clicks also produced amplitude enhancement. No systematic changes in the latencies of individual MLR

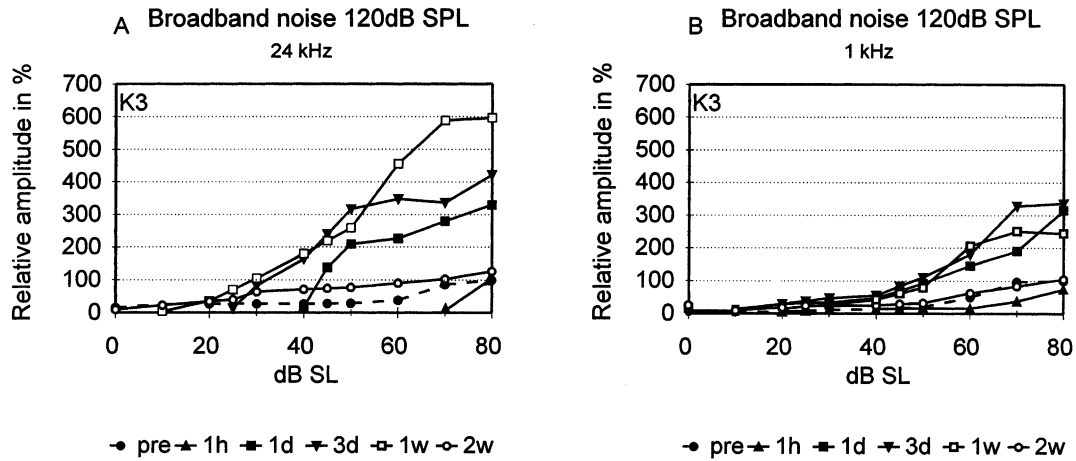


Fig. 5. Relative amplitude-intensity functions for MLR measured at 24 kHz (A) and 1 kHz (B) before and after 120 dB SPL broadband noise exposure. Ordinate: amplitude in % relative to pre-exposure MLR amplitude at 80 dB pre-exposure SL taken as 100%. Abscissa: stimulus intensity in dB above pre-exposure threshold.

waves were found when compared before and after noise exposure.

4. Discussion

The above results demonstrate that 1 h noise exposure with intensities of 105–120 dB SPL produces in adult rats temporary threshold shifts which last for about 2 weeks. Similar effects were observed by Lenoir et al. (1979), who exposed rats to 120 dB SPL white noise for 30 min. In our study it was demonstrated that the magnitude and pattern of hearing loss were dependent on the spectral characteristics of the noise and its intensity and were also related to the hearing sensitivity

of the rat at different frequencies. For broadband noise, the largest threshold elevation appeared in the range of maximum hearing sensitivity. In the case of narrowband noise exposure, the largest TTS was observed in a frequency range corresponding to the frequency of the maximum exposure energy displaced to higher frequencies. Further, it was shown that in rats, high-frequency noise exposure evoked more elevated hearing thresholds than did low-frequency noise exposure. For example, 120 dB SPL high-frequency noise exposure produced TTS of about 20 dB more in comparison with low-frequency noise exposure; the threshold elevation caused by 105 dB SPL high-frequency noise exposure was larger than that produced by 115 dB SPL low-frequency noise exposure. Our data agree with the results of other

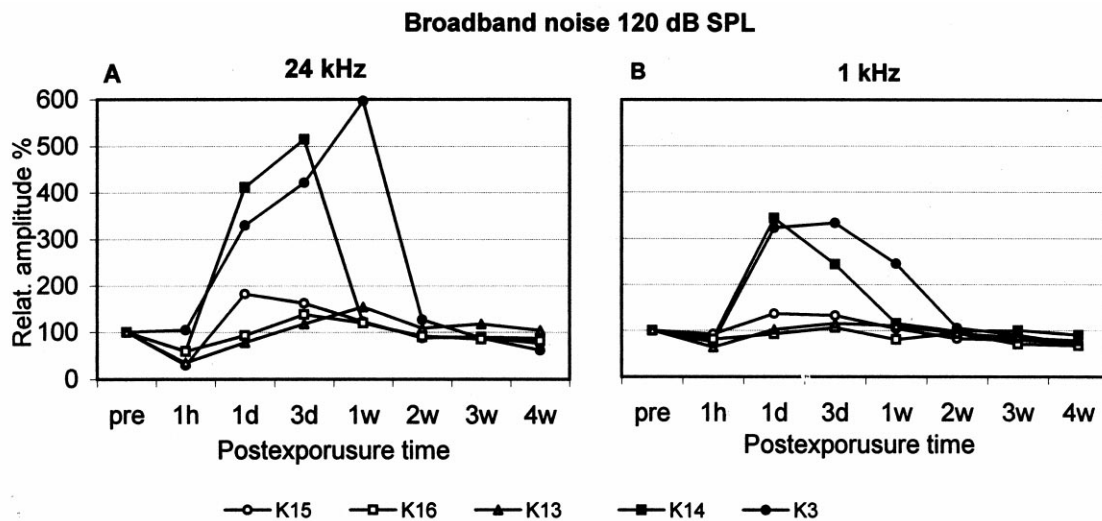


Fig. 6. Relative amplitudes of MLR recorded after stimulation with tones at 1 kHz and 24 kHz before and after 120 dB SPL broadband noise exposure in five rats (individual data); intensity of the stimulus: - 80 dB above pre-exposure threshold. Ordinate: amplitude in % relative to pre-exposure MLR amplitude at 80 dB pre-exposure SL taken as 100%.

Broadband noise 120 dB SPL

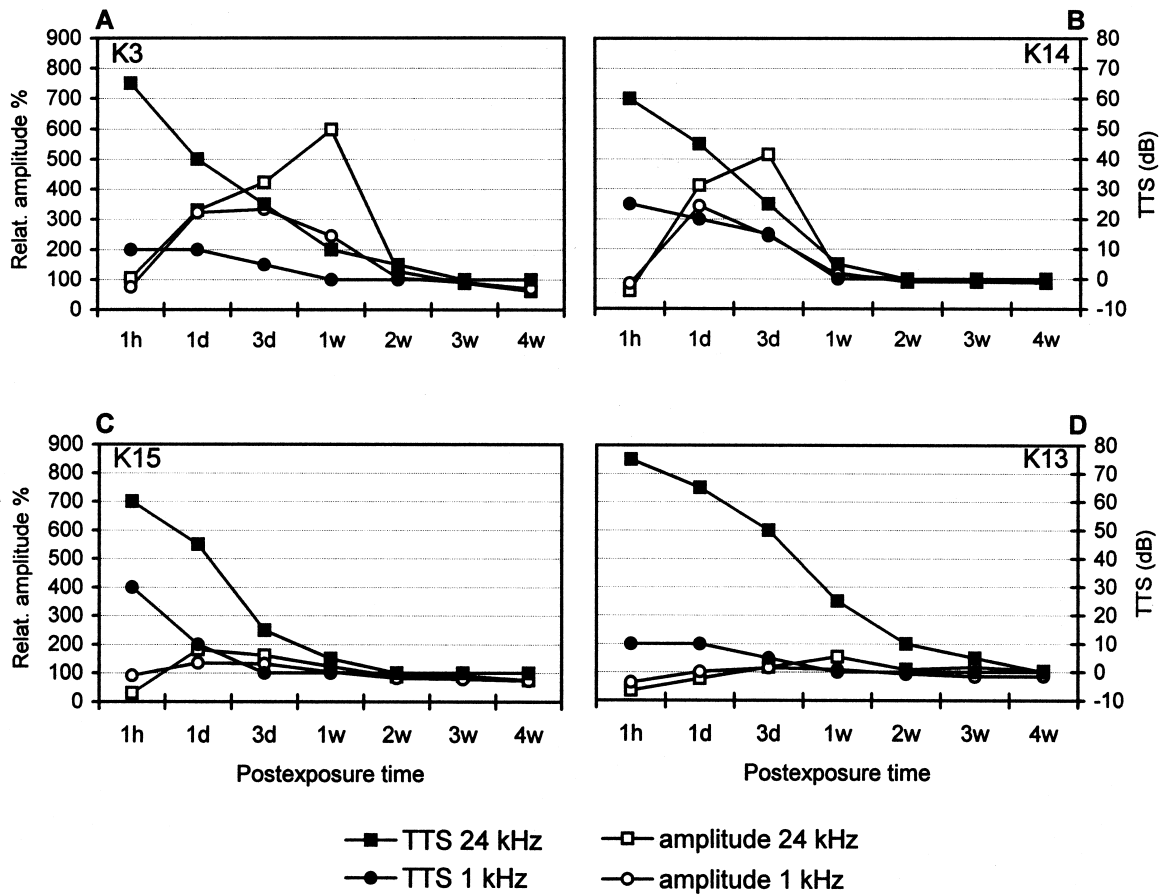


Fig. 7. Comparison of changes of TTS and MLR amplitudes (relative data) as a function of time after broadband noise exposure (120 dB SPL for 1 h). MLR were recorded to tones of 1 kHz and 24 kHz in five rats; intensity of the stimulus for MLR amplitude: 80 dB above pre-exposure threshold. Left ordinate: amplitude in % relative to pre-exposure MLR amplitude at 80 dB pre-exposure SL taken as 100%; right ordinate: TTS in dB; abscissa: post-exposure time.

authors (Lenoir et al., 1979; Borg, 1982), who observed that broadband noise exposure evoked maximal threshold elevation in the high-frequency range. With respect to the effect of low-frequency noise exposure on the rat's hearing, our data agree with the results of Ryan et al. (1996) who showed that 110 dB low-frequency (1.4–5.6 kHz) noise exposure resulted in a threshold shift, indicated by brainstem responses, with a maximum of about 25 dB at 4 kHz. The results of noise exposure on the hearing thresholds of rats differed from previous results found in guinea pigs (animals with low-frequency hearing), which have demonstrated that exposure to low-frequency noise (with the same spectral content as in the present study) evokes flat elevated thresholds over a wide range of frequencies (Popelář et al., 1987; Syka et al., 1994).

In agreement with our previous data obtained in guinea pigs, this study shows that acoustic trauma produced by exposure to high-level noise may result in an

amplitude enhancement of evoked responses recorded in the auditory cortex (Popelář et al., 1987; Syka et al., 1994). In all rats after noise exposure, an abnormally steep slope of the amplitude-intensity functions was observed. The values of the post-exposure MLR amplitudes at near threshold intensities were similar to the pre-exposure values. In contrast to this, the post-exposure amplitudes at high stimulus intensities reached or exceeded the pre-exposure values. In some animals the MLR amplitude exceeded the control level several times. A comparison of results of the present study conducted in rats and previous studies in guinea pigs (Popelář et al., 1987; Syka et al., 1994) shows that under the same condition of exposure, when low-frequency noise (frequency range 0.1–8 kHz) was used, the amplitude enhancement was more pronounced in guinea pigs. Under these conditions the amplitude enhancement in guinea pigs reached 150–500% of the pre-exposure level whereas in rats the amplitude enhance-

ment was observed only in some cases and reached 150–230% of the original amplitude value. In both guinea pigs and rats, the increased MLR amplitudes after low-frequency noise exposure were observed mainly with low-frequency test stimuli. The larger effect in guinea pigs corresponds to their better hearing sensitivity in the low-frequency range. The second reason for the lower susceptibility of the rats in our study with respect to MLR enhancement in comparison with the guinea pigs may be the fact that in contrast to the guinea pigs, which were awake throughout the experiments, the rats were tested in a slight narcosis with xylazine. This measure was necessary to avoid complications arising from the movements of the rat. According to data of Gummit and Grossman (1961) and Gerken et al. (1986) enhancement can disappear in anesthetized animals. Even with broadband noise it was necessary in rats to use an intensity of 120 dB SPL to reliably elicit a MLR enhancement. It is possible that in rats without xylazine administration, the enhancement could be elicited with noise intensities lower than 120 dB. In rats the amplitude enhancement was more pronounced after broadband noise exposure and was more generalized, being present after both high-frequency and low-frequency

test stimuli. The enhancement was observed in only one rat out of five exposed to high-frequency noise. The reason for this might be the fact that for high-frequency exposure a narrow-band (third octave) noise was used whereas in the case of low-frequency noise the spectrum spanned over five octaves.

In contrast to guinea pigs (Popelář et al., 1987; Syka et al., 1994), the maximal amplitude of MLR in rats was reached the next day or within several days after the end of noise exposure. One of the reasons why the maximum enhancement was present after a delay of many hours following noise exposure is the fact that our intensity range was very limited because of the large threshold shifts. However, even with low-frequency stimuli (where the threshold shift was very low) it was not possible to detect an amplitude enhancement immediately after exposure. This fact speaks in favor of intrinsic biological reasons for the delayed maximum of amplitude enhancement in rats and not technical reasons.

The wide variation in amplitude enhancement among our experimental animals was accompanied by almost identical temporary threshold shifts. This fact, as well as the fact that the recovery functions for the amplitude and thresholds of the MLR were also different, suggests

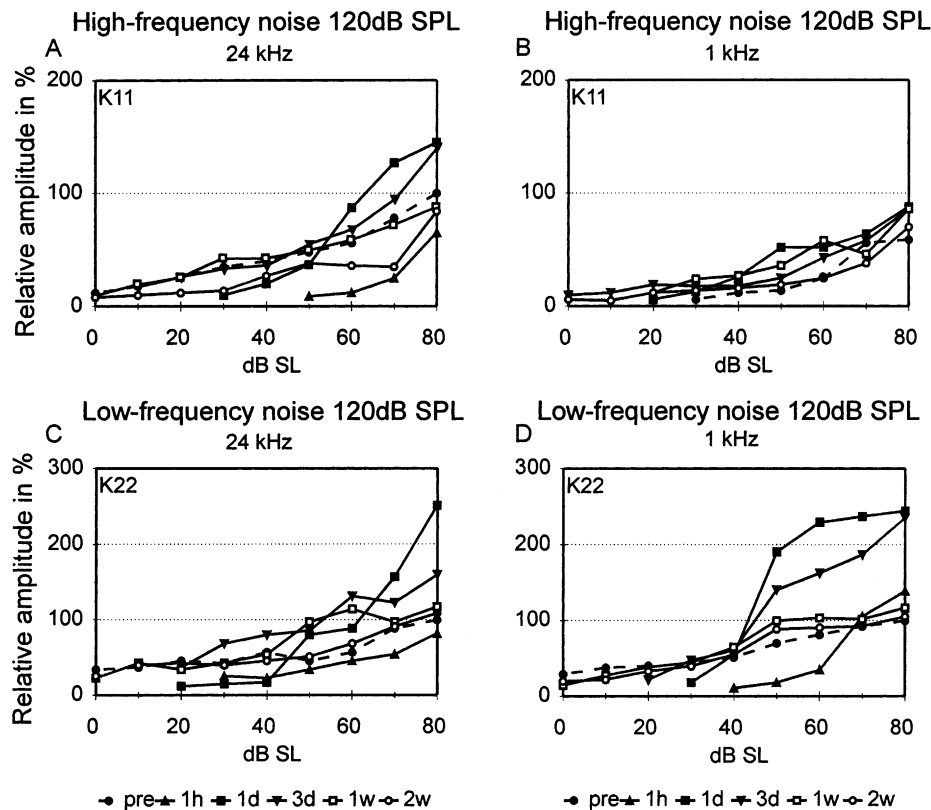


Fig. 8. Relative amplitude-intensity functions for MLR before and after 120 dB SPL high-frequency (A, B) and low-frequency (C, D) noise exposure measured at 24 kHz and 1 kHz. Ordinate: amplitude in % relative to pre-exposure MLR amplitude; MLR amplitude at 80 dB pre-exposure SL taken as 100%. Abscissa: stimulus intensity in dB above pre-exposure threshold.

that these parameters reflect post-exposure changes occurring at different levels of the auditory system. There exists accumulated evidence that threshold elevations are of cochlear origin (for review see e.g. Syka, 1989). As mentioned above, some authors consider the amplitude enhancement of evoked responses recorded at higher levels of the auditory system to be a reflection of a central impairment (Chaloupka, 1968; Bock and Saunders, 1976; Salvi et al., 1990, 1992; Popelář et al., 1987; Gerken, 1993; Syka et al., 1994; Szczepaniak and Møller, 1996). For example, when temporary hearing loss is induced in certain strains of mice, they become susceptible to audiogenic seizures several days later (Willott and Henry, 1974; Urban and Willott, 1979). This is accompanied by increases in evoked potentials and unit activity in the inferior colliculus. One aspect of the priming-induced changes is a steepening of intensity functions and/or a decrease in the number of nonmonotonic intensity functions as indicators of reduced inhibition. Our finding of the post-exposure increase in the amplitude of cortical evoked responses in rats also indicates that central disorders may play a significant role in the pathology of noise-induced hearing loss. It may be supposed that the amplitude enhancement of MLR reflects changes in the balance between inhibition and excitation in the auditory cortex and results, for example, in disinhibition produced by depletion of inhibitory transmitters during noise exposure. Intriguing with respect to mechanisms of MLR amplitude enhancement is the observation by Salvi (1999) that in chinchillas, after treatment with carboplatin, the local cortical potentials were larger than normal. Cortical amplitude initially increased during the first 2–3 weeks following carboplatin treatment and then declined slightly between 3 and 5 weeks. Since carboplatin treatment in chinchillas results in the partial destruction of inner hair cells, this represents another model in which the central response is facilitated under a condition of peripheral suppression. The results of this study and similarly our results suggest that in the auditory system there may exist an automatic gain control mechanism that compensates for a reduced peripheral input. Further studies examining changes in transmitter activities in the auditory cortex during the post-exposure period will be necessary to explain the mechanisms of this feedback control.

Acknowledgements

This research was supported by grants from the Ministry of Health 4747-3 and from the Grant Agency of the Czech Republic 309/97/0830. The authors wish to thank Dr Jiří Popelář for his assistance and comments in different stages of the experiments.

References

- Axelsson, A., Borchgrevink, H., Hamernik, R.P., Hellström, P.-A., Henderson, D., Salvi, R.J., 1996. *Scientific Basis of Noise-Induced Hearing Loss*. Thieme, New York.
- Bock, G.R., Saunders, J.C., 1976. Effects of low and high frequency noise bands in producing a physiologic correlate of loudness recruitment in mice. *Trans. Am. Acad. Ophthalm. Otol.* 82, 338–342.
- Borg, E., 1982. Noise-induced hearing loss in normotensive and spontaneously hypertensive rats. *Hear. Res.* 8, 117–130.
- Chaloupka, Z., 1968. Effects of noise on cortical evoked potentials in rats. *Act. Nerv. Super.* 10, 207–208.
- Gerken, G.M., 1993. Alteration of central auditory processing of brief stimuli; A review and a neural model. *J. Acoust. Soc. Am.* 93, 2038–2049.
- Gerken, G.M., Simhadri-Sumithra, R., Bhat, V.K.H., 1986. Increase in central auditory responsiveness during tone stimulation or following loss. In: Salvi, R.J., Henderson, D., Hamernik, R.P., Colletti, V. (Eds.), *Basic and Applied Aspects of Noise-Induced Hearing Loss*. Plenum Press, New York, pp. 195–211.
- Gummit, R.J., Grossman, R.G., 1961. Potentials evoked by sound in the auditory cortex of the cat. *Am. J. Physiol.* 200, 1219–1225.
- Henderson, D., Møller, A.R., 1975. Effect of asymptotic threshold shift in neuronal firing patterns of the rat cochlear nucleus (abstract). *J. Acoust. Soc. Am.* 57, 53.
- Kaltenbach, J.A., Godfrey, D.A., Neumann, J.B., McCaslin, D.L., Afman, C.E., Zhang, J., 1998. Changes in spontaneous neural activity in the dorsal cochlear nucleus following exposure to intense sound: relation to threshold shift. *Hear. Res.* 124, 78–84.
- Lenoir, M., Bock, G.R., Pujol, R., 1979. Supra-normal susceptibility to acoustic trauma of the rat pup cochlea. *J. Physiol. (Paris)* 75, 521–524.
- Lonsbury-Martin, B.L., Martin, G.K., 1981. Effects of moderately intense sound on auditory sensitivity in Rhesus monkeys: Behavioral and neural observations. *J. Neurophysiol.* 46, 563–586.
- Møller, D.R., 1990. Auditory brainstem of the ferret: Early cessation of developmental sensitivity of neurons in the cochlear nucleus to removal of the cochlea. *J. Comp. Neurol.* 302, 810–823.
- Popelář, J., Syka, J., Berndt, H., 1987. Effect of noise on auditory evoked responses in awake guinea pigs. *Hear. Res.* 26, 239–247.
- Popelář, J., Erre, J.-P., Aran, J.-M., Cazals, Y., 1994. Plastic changes in ipsi-contra lateral differences of auditory cortex and inferior colliculus evoked potentials after injury to one ear in the adult guinea pig. *Hear. Res.* 72, 125–134.
- Reale, R.A., Brugge, J.F., Chan, C.K., 1987. Maps of auditory cortex in cats reared after unilateral cochlear ablation in the neonatal period. *Dev. Brain Res.* 34, 281–290.
- Robertson, D., Irvine, D.R.F., 1989. Plasticity of frequency organization in auditory cortex of guinea pigs with partial unilateral deafness. *J. Comp. Neurol.* 282, 456–471.
- Ryan, A.F., Luo, L., Bennett, T., 1996. Changes in gene expression following temporary noise-induced threshold shift. In: Axelsson, A., Borchgrevink, H., Hamernik, R.P., Hellstrom, P.-A., Henderson, D., Salvi, R.J. (Eds.), *Scientific Basis of Noise-Induced Hearing Loss*. Thieme, New York, pp. 50–55.
- Salvi, R.J., 1999. Evidence of hyperactivity in the central auditory pathway following cochlear damage. *Proceedings of the 2nd Symposium on Molecular Mechanisms in Central Auditory Function and Plasticity*, Park City, UT, p. 52.
- Salvi, R.J., Saunders, S.S., Gratton, M.A., Arehole, S., Powers, N., Willott, J.F., Lu, S.-M., 1982. Noise-induced hearing loss can alter neural coding and increase excitability in the central nervous system. *Science* 216, 1331–1332.
- Salvi, R.J., Saunders, S.S., Gratton, M.A., Arehole, S., Powers, N., 1990. Enhanced evoked response amplitudes in the inferior colli-

- culus of the chinchilla following acoustic trauma. *Hear. Res.* 50, 245–258.
- Salvi, R.J., Powers, N., Saunders, S.S., Boettcher, F.A., Clock, A.E., 1992. Enhancement of evoked response amplitude and single unit activity after noise exposure. In: Dancer, A., Henderson, D., Salvi, R., Hamernik, R. (Eds.), *Effects of Noise on the Auditory System*. Mosby, Philadelphia, PA, pp. 156–174.
- Syka, J., 1989. Experimental models of sensorineural hearing loss – effects of noise and ototoxic drugs on hearing. *Prog. Sens. Physiol.* 9, 97–170.
- Syka, J., Rybalko, N., Popelář, J., 1994. Enhancement of the auditory cortex evoked responses in awake guinea pigs after noise exposure. *Hear. Res.* 78, 158–164.
- Szczepaniak, W.S., Møller, A.R., 1996. Evidence of neuronal plasticity within the colliculus after noise exposure: a study of evoked potentials in the rat. *Electroenceph. Clin. Neurophysiol.* 100, 158–164.
- Taniguchi, I., Saito, N., 1978. Plastic reorganization in the inferior colliculus of the immature mouse studied by [14C] deoxyglucose method. *Proc. Jpn. Acad.* 54, 496–499.
- Urban, G.P., Willott, J.F., 1979. Response properties of neurons in the inferior colliculi of mice made susceptible to audiogenetic seizures by acoustic priming. *Exp. Neurol.* 63, 229–243.
- Willott, J.F., Henry, K.R., 1974. Auditory evoked potentials: Developmental changes of threshold and amplitude following early acoustic trauma. *J. Comp. Physiol. Psychol.* 86, 1–7.
- Willott, J.F., Lu, S.-M., 1982. Noise-induced hearing loss can alter neural coding and increase excitability in the central nervous system. *Science* 216, 1331–1332.
- Zhang, J.S., Kaltenbach, J.A., 1998. Increases in spontaneous activity in dorsal cochlear nucleus of the rat following exposure to high-intensity sound. *Neurosci. Lett.* 250, 197–200.