

Prolonged Exposure to Industrial Noise: Cochlear Pathology does not Correlate with the Degree of Permanent Threshold Shift, but is Related to Duration of Exposure

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Abstract: Prolonged Exposure to Industrial Noise: Cochlear Pathology does not Correlate with the Degree of Permanent Threshold Shift, but is Related to Duration of Exposure: Mariola ŚLIWIŃSKA-KOWALSKA, et al. Department of Physical Hazards, The Nofer Institute of Occupational Medicine—It has been shown that the damaging effects of noise on the mammalian inner ear strongly depend upon parameters of exposure, such as the intensity of noise, the duration of exposure, etc., but the relationship between permanent hearing loss and cochlear damage still remains unclear. In this study, we were interested in the damaging effects of exposure to typical steady-state, wide-band industrial noise acting for as long as 12 weeks on guinea pigs. The aim of the study was to evaluate the relationships among the duration of exposure to industrial noise, the level of permanent hearing loss and the degree of cochlear injury and to assess the possible mechanisms of industrial noise-induced hearing loss. The results of the study indicate that 1) prolonged exposure to industrial noise causes in guinea pigs a relatively fast increase in permanent hearing threshold shift (PTS) up to 30 dB, which reaches the asymptotic level after 4 weeks of exposure; 2) the first 30 dB of PTS is caused almost exclusively by the damage to outer hair cells; 3) the degree of cochlear damage is not related to the level of permanent threshold shift; 4) the progression in cochlear pathology depends upon the duration of exposure; 5) both micromechanical and metabolic mechanisms seem to be involved in the development of industrial noise-induced hearing loss. (*J Occup Health 1998; 40: 123–131*)

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Despite several years of investigation, research on noise-induced hearing loss (NIHL) is still being conducted. One cause of this interest is, on one hand, the not fully resolved pathophysiology of NIHL, and yet another cause is the possibility of using noise as a tool to probe cochlear function^{1, 2}.

The mechanism of noise-trauma and the damaging effects of loud sounds on the mammalian inner ear strongly depend upon the parameters of exposure, such as the intensity and the frequency spectrum of noise, the duration of exposure, etc.^{3, 4}. From Witmaack's earliest work in 1907, up to today's investigations, the majority of experimental observations have used exposures of short duration (1 min to 4 hr) to very high intensity noise (105 to 138 dB SPL)^{2, 3, 5–7}. Such exposures produce rather pronounced cochlear damage and are not typical of noise in the human workplace. Fewer experiments deal with the effects of prolonged exposure to industrial (or industrial-like) noise^{8–12} which is generally of moderate intensity, so that the relationship between the duration of exposure, permanent hearing loss and pathomorphological changes in the cochlea is still not fully recognized³.

Although several investigators have shown a fairly good correlation between hearing loss and pathomorphology^{5, 10, 13, 14}, some studies have shown that individuals with permanent hearing loss did not exhibit any recognizable pathology in the cochlea^{15, 16}. In contrast to this, other studies have reported normal hearing in animals with substantial damage to hair cells¹⁷. Such discrepancies in the relation of cochlear damage to hearing loss may depend upon the duration of exposure and the timing of the evaluation of noise trauma. It also appears that there is no straight correlation between hearing loss and pathomorphology^{12, 18}.

This study was interested in the damaging effects of exposure to typical steady-state, wide-band industrial noise, acting for as long as 12 weeks. The study attempted

to answer the following questions: 1) Is there an orderly relationship among the duration of exposure to industrial noise, the level of permanent hearing loss and the degree of cochlear injury?, 2) What are the possible mechanisms of industrial NIHL?

Because most types of tape-recording and electroacoustical reproduction of noise can introduce distortion or filtering of the original sound, the animals in this study were exposed to genuine industrial noise in a weaving-mill. In assessing the pathomorphology of the cochlea, this study used both light and electron microscopy methods. The comparison between permanent hearing loss and cochlear damage was made upon reaching an asymptotic (or plateau) level of hearing loss, the most logical point from which to proceed.

Material

Two groups of three-month-old Hartley guinea pigs, weighing from 450 g to 600 g, were used in this study. Only animals with normal tympanic membrane appearance and with hearing thresholds at 45 dB SPL or better in both ears, as assessed by auditory brainstem audiometry, were included. The experimental group of 32 animals were exposed to the genuine weaving-mill noise, while the control group of 18 animals were kept in a room in the Animal Care Department, where the background noise did not exceed 60 dB(A). The room temperature and humidity did not differ significantly between groups. During the experiment, all the animals were given free access to food and water.

Methods

Noise exposure

The experimental group of animals were exposed to continuous, broad-band, steady state weaving-mill noise at an intensity of 96–98 dB(A), 16 hr a day, 5 days a week for 12 weeks. The level and frequency spectrum of the weaving-mill noise are shown in Fig. 1.

During the exposure, each animal was housed in a separate cage of a four-tiered rack. The rack of cages was placed in a room containing about 100 working machines, 5 m behind the last row of machines. Noise levels were measured with a sound level meter (type 2231 Bruel & Kjaer) and a condenser microphone (type 4155 Bruel & Kjaer). The difference in sound pressure across the cages was less than 1 dB. The animals were rotated to different rows of the rack every week to minimize any effects of differences in sound pressure. Because the sound field was uniform (i.e., SPLs varied no more than 1–2 dB throughout the work room), the animals were exposed to noise levels equivalent to those experienced by workers in the weaving-mill.

Functional assessment

Before exposure, after every 4 weeks of exposure, and

in an experimental group of animals after 8 weeks of recovery, the hearing function was assessed by auditory brainstem response (ABR) technique.

ABR measurement was performed in all guinea pigs before and after 4 weeks of experimentation. After 8 and 12 weeks of exposure the number of animals systematically decreased due to the sacrifice of some animals for the pathomorphological study. In 9 experimental guinea pigs, the hearing function was also estimated after 8 weeks of recovery. Hearing threshold and latency-intensity function for wave I were assessed. The ABR measurement was performed no sooner than 24 hr after finishing the exposure to noise.

ABR Measurement: Prior to measuring its auditory threshold, a guinea pig was anesthetized intraperitoneally with sodium pentobarbital at a dose of 40 mg per kg of body weight. Subcutaneous stainless steel electrodes were placed in the vertex, and in the ipsilateral retroauricular region, and the lower back served as the ground. The acoustic stimuli, which were 100 μ s, wide-band clicks, presented at a repetition rate of 31 per sec, were delivered through earphones (mini Sony Walkmans, type MDR-E515), closely attached to the pinna by means of surgical glue. Auditory potentials were registered and analyzed with the Amplaid mk 10 system with 50–2,500 Hz filters. The analysis time was 12 ms. The acquisition of auditory evoked potentials started at a stimulus of 100 dB SPL and was decreased in 10–5 dB steps until the threshold was approached. The detection of the threshold was defined on the basis of 2–3 repetitive measurements near the threshold. All ABR measurements were done in a soundproof room by the same investigator. During the measurement, the body temperature of the animal (37.5–38°C) was stabilized by means of a lamp. The calibration of this method of ABR measurement, which was done before starting this experiment, revealed very good test-retest reproducibility. The difference between the mean

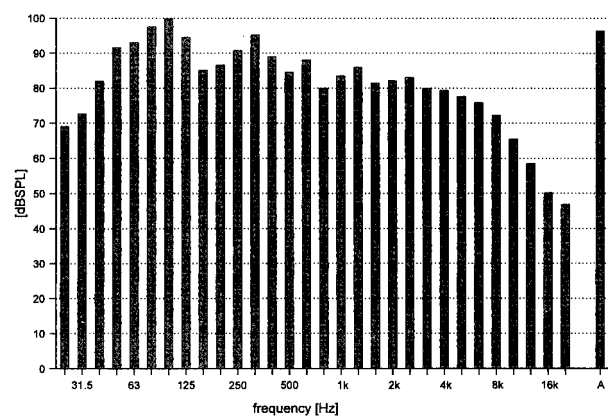


Fig. 1. The level and frequency spectrum of the weaving-mill noise.

values for two tests was 2.5 dB; this difference was not significantly altered at the 5% level (Student's t-test). The normal ABR hearing threshold ranged from 35 to 45 dB peSPL.

Pathomorphologic evaluation

Cochleae from 24 experimental guinea pigs (8 animals after every 4 weeks of exposure) and from 10 control animals were assessed. The animals were anesthetized with sodium pentobarbital and perfused through the heart with a 0.1 M cacodylate buffer followed by a cold fixative: 2% paraformaldehyde, 3% glutaraldehyde in a 0.1 M cacodylate buffer, pH 7.4. After perfusion, the bullae were removed and the cochleae were exposed and postfixed by immersion in the same fixative for three hours. The cochleae were then carefully dissected and the bony capsule and stria vascularis were removed. One cochlea from each animal was prepared for examination by light microscopy and the second by transmission electron microscopy.

Light microscopy: The cochleae were divided into segments that were mounted on slides, coverslipped with DAKO mounting medium (DAKO Corporation, Santa Barbara, CA) and viewed and photographed through a Zeiss NU microscope. Surface preparations from all cochlear turns were examined.

Electron microscopy: The cochleae were washed and postfixed for one hour in 1% OsO₄ in a 0.1 M cacodylate buffer. The cochleae were dissected into several segments, dehydrated and embedded in Poly/Bed 812 (Polysciences, Inc.). Thick sections were examined by light microscopy (Zeiss NU). Thin sections were then obtained and examined under the transmission electron microscope (JEM 100C).

Results

Impairment in hearing sensitivity

Figure 2 shows the mean hearing thresholds (h.t.) in control animals and in experimental animals before exposure, after 4, 8 and 12 weeks, and in the experimental group after 8 weeks of recovery. Before exposure, the mean hearing thresholds in both groups of animals were similar (41.7 ± 3.9 dB peSPL in control animals and 41.3 ± 3.6 dB peSPL in experimental animals). In experimental group, after 4 weeks of exposure a permanent h.t.s. of 22.8 ± 6.8 dB was observed (h.t. after 4 weeks of exposure was 64.1 ± 5.9 dB peSPL; the difference in h.t.s. was statistically significant compared to controls, $*p < 0.001$), but after subsequent weeks of exposure h.t. had not deteriorated significantly (h.t. was 67.3 ± 9.2 and 68.3 ± 11.7 dB peSPL after 8 and 12 weeks of exposure, respectively), so that the permanent h.t.s. reached an asymptotic (or plateau) level. In order to assess the reversibility in hearing sensitivity loss, h.t. in experimental group was also assessed after 8 weeks of recovery from exposure to noise. No

hearing improvement was observed.

The mean hearing threshold did not change significantly in the control animals during the subsequent weeks of the experiment (h.t. was 42.8 ± 3.0 , 44.0 ± 3.7 and 46.7 ± 4.7 dB peSPL after 4, 8 and 12 weeks of the experiment, respectively).

After the first 4 weeks of exposure, a hearing threshold shift (h.t.s.) from 20 to 30 dB was observed in a majority of ears (48/64 or 78.1%). In 13/64 ears (15.8%), h.t.s. was less than 20 dB, and in only 5/64 (7.8%) ears h.t.s. was greater than 30 dB. A similar range of h.t.s. was seen after 8 and 12 weeks of exposure (h.t.s. ranged from 20 to 30 dB in 72.2% and in 71.5% of ears after 8 and 12 weeks of exposure, respectively), but an increase in the number of ears with hearing impairment greater than 30 dB (from 6.1% to 21.4%) and a decrease in the number of ears with hearing impairment smaller than 20 dB (from 15.8% to 7.2%) was observed (Fig. 3).

Latency-intensity function

The first positive peak in the ABR pattern corresponds to the averaged synchronous activity in the auditory nerve. The analysis of latency of wave I - click intensity function did not reveal significant prolongation in the wave I latency in exposed-to-noise animals in comparison with the pre-exposure function (Student's t-test, $p > 0.05$). Only at the near-threshold intensity was the latency of wave I slightly increased, but it did not exceed the range of ± 2 std (Fig. 4).

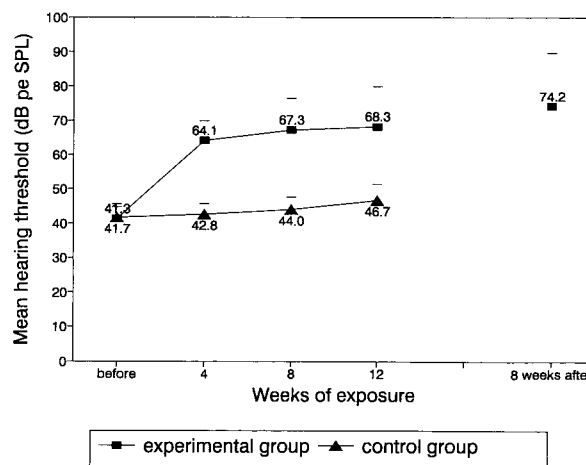


Fig. 2. The mean hearing threshold (+ 1 std) of the group of exposed-to-noise guinea pigs (squares) versus that of the control animals (triangles), after consecutive weeks of the experiment. In the exposed-to-noise animals a hearing loss of about 23 dB is observed after 4 weeks of exposure, but after an additional 8 weeks of exposure, no significant deterioration in the hearing level is seen in comparison with the control group (the asymptotic curve).

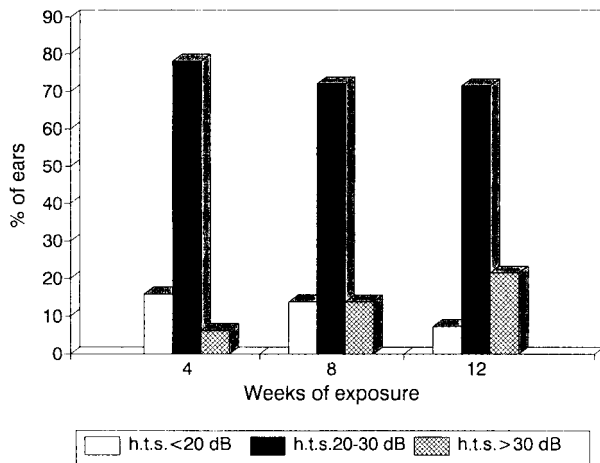


Fig. 3. Hearing threshold shifts (h.t.s.) after consecutive weeks of exposure. Note that in a majority (more than 70%) of ears, the h.t.s. ranged from 20 to 30 dB, regardless of the duration of exposure. The longer the exposure, the smaller the number of ears with a h.t.s. below 20 dB and the greater the number of ears with a h.t.s. above 30 dB.

Pathomorphology

Since h.t.s. from 20 to 30 dB was present in the majority of animals, only guinea pigs with such h.t.s. were studied for morphology, regardless of the duration of their exposure to noise. Although pathomorphology revealed a significant inter-subject variability, it was possible to register some tendencies in the development of the changes.

Light microscopy

Surface preparations: After 4 weeks of exposure, morphological changes were found mainly in the second and the third turns of the cochlea. In the second turn of the cochlea, the predominant damage was missing OHC1 and OHC3 (Fig. 5b). In the third turn of the cochlea the changes were more pronounced, as apart from missing OHC1 and OHC3, the foci of disarrangement of all rows of OHC and displacement of OHC1 toward the tunnel of Corti were also found. In two animals, mechanical distortion of the cochlea, seen as so called "waves" along the cochlea, was observed (Fig. 5c). In none of the animals were changes to inner hair cells (IHC) seen.

After 8 weeks of exposure, the damage to OHC was more pronounced and widespread, including the fourth turn of the cochlea. The changes were qualitatively similar to those observed after 4 weeks of exposure, but the disarrangement and displacement of all rows of OHC were found more often (Figs. 5d, 5e). Mechanical distortion of the organ of Corti was observed in the third and fourth turns of the cochlea. But even when the damage to OHC was severe, there was no damage to IHC (Fig. 5e), with the exception of one animal, in which a

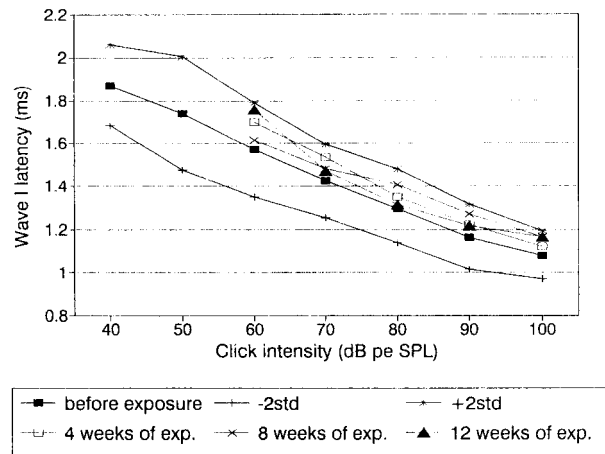


Fig. 4. Wave I latency-click intensity function in the group of exposed-to-noise guinea pigs before and after 12 weeks of exposure. No significant prolongation in the wave I latency is found.

few IHC were missing.

After 12 weeks of exposure, morphological changes in OHC did not progress and seemed to be even less widespread than after 8 weeks of exposure. Missing OHC were detected in the second and third turns of the cochlea. No changes were observed in the fourth turn, but, apart from damage to OHC, a loss of outer pillar cells was also occasionally observed (Fig. 5f).

Cross sections: Changes found in cross sections (thick sections) after 4 weeks of exposure were consistent with those observed in surface preparations. The most vulnerable to noise were OHC1 and OHC3 (Fig. 6a). Very often a disruption of reticular laminae, a broadening of Nuel spaces and an increase in lipofuscin granules were also seen (Fig. 6a). Occasionally, foci of damage were observed in all three rows of OHC (Fig. 6b). No changes in IHC were seen. In all ears, the changes in stereocilia, such as floppy and disarrayed stereocilia, were more widespread than the damage to hair cells, including both OHC and IHC.

After 8 weeks, as in the surface preparations, an increase in cochlear damage greater than that at 4 weeks of exposure was observed. Apart from missing or damaged OHC, the foci of damage to all hair cells were also observed (Fig. 6c). Also increased from the 4 week examination was the damage to OHC in all rows or complete loss of OHC and the disruption of reticular laminae (Fig. 6c). The damage to both OHC and IHC with a proliferation of supportive cells and cellular debris in the endolymph was also seen (Fig. 6d).

The progression in the pathology of stereocilia was mainly observed after 12 weeks of exposure. Floppy and disarrayed stereocilia were seen on both IHC and

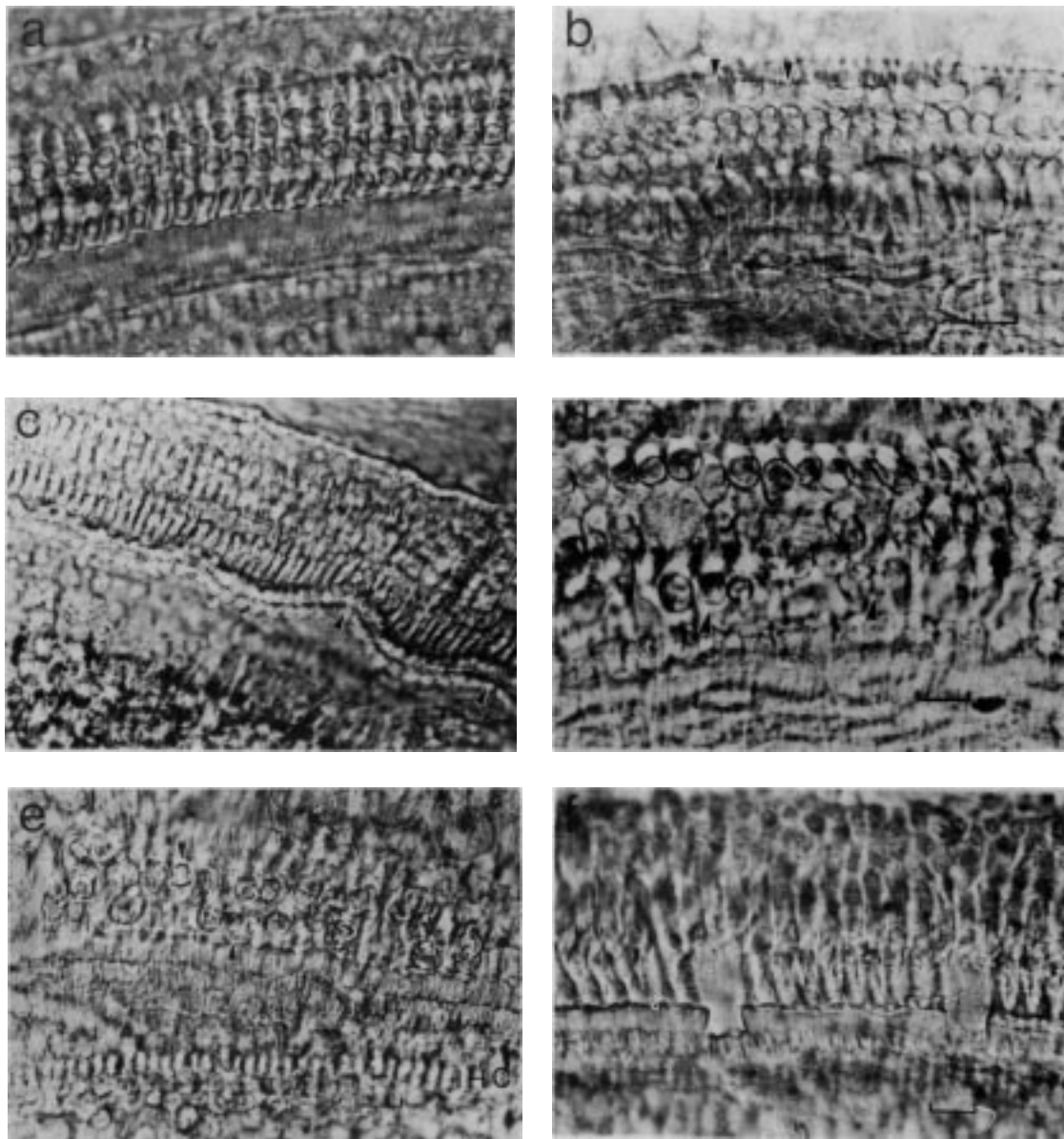


Fig. 5. Micrographs of surface preparations of the guinea pigs' organs of Corti. Bar = 20 μm . a) Control animal—three rows of undamaged outer hair cells, third turn. b) 4 weeks of exposure to noise, second turn—the arrows point to the loss of OHC of the 1st and 3rd rows. c) 4 weeks of exposure to noise, third turn—mechanical distortion of the organ of Corti (“waves”). d) 8 weeks of exposure to noise, third turn—severe damage to all rows of OHC. e) 8 weeks of exposure to noise, fourth turn—in spite of severe changes in OHC (arrows), inner hair cells (IHC) are intact. f) 12 weeks of exposure to noise, second turn—two instances of missing outer pillar cells.

OHC in all turns of the cochlea, including the first one. In one animal, no pathology was observed other than floppy stereocilia on both the OHC and IHC.

Electron microscopy

After 4 weeks of exposure, floppy or disarrayed stereocilia were seen primarily on OHC (Fig. 7), but only rarely on both OHC and IHC. No changes to the cuticular

plates were found. In the cell body, a proliferation of subsurface cisternae, an increase in the number of mitochondria and Hensen's bodies were observed (Fig. 8).

After 8 weeks of exposure, the damage to stereocilia was more severe. Apart from disarrayed stereocilia, stereocilia were found broken near their rootlets or torn off completely. The cuticular plate was softened and protruded (Fig. 9). The damage to stereocilia and the cuticular plate

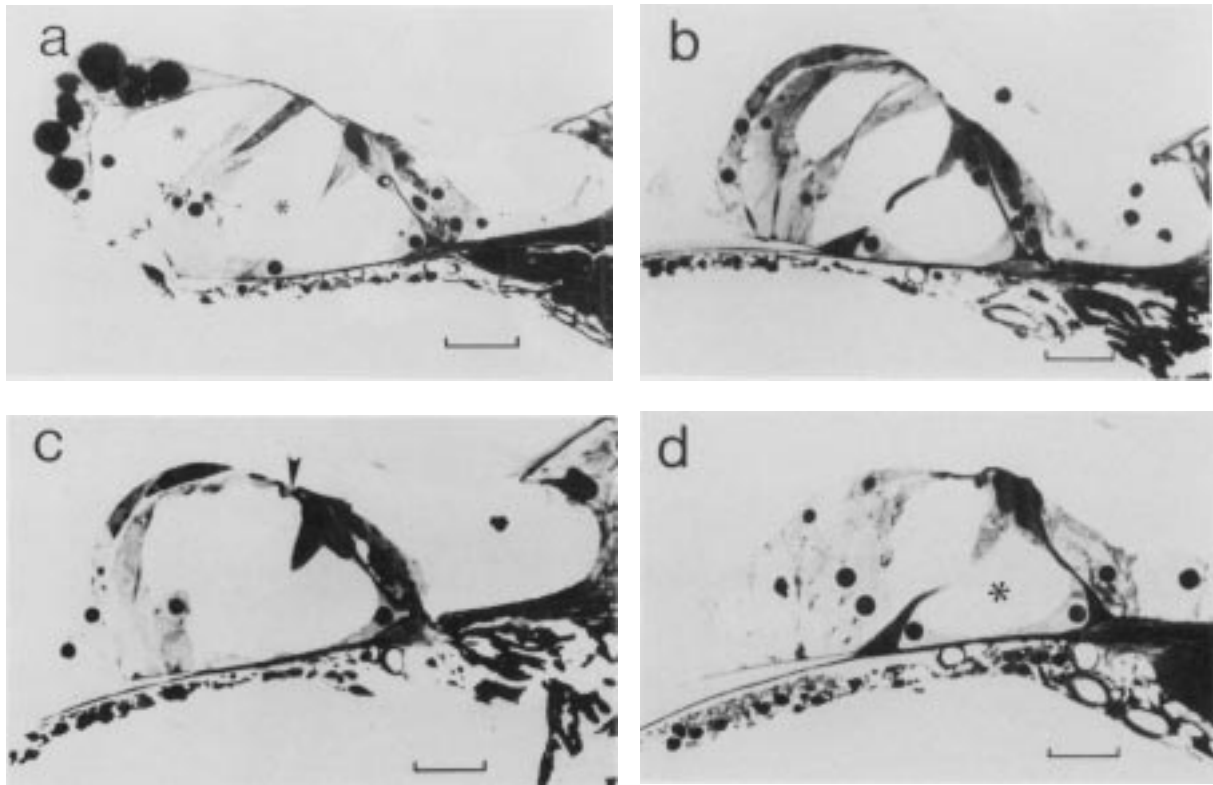


Fig. 6. Micrographs of guinea pigs' organs of Corti, cross sections. Bar = 20 μm . a) 4 weeks of exposure to noise, second turn—the loss of OHC in the 1st and 3rd rows is observed, as well as enlarged Nuel spaces (stars) and an increase in lipofuscin granules. b) 4 weeks of exposure to noise, third turn—the degeneration of all OHC. IHC unchanged. c) 8 weeks of exposure to noise, third turn—complete loss of OHC and the disruption of reticular laminae (arrow). d) 8 weeks of exposure to noise, second turn—the loss of all hair cells, a flattening of the organ of Corti and a proliferation of supportive cells.

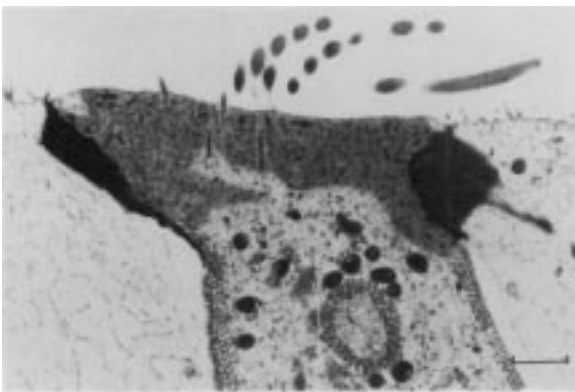


Fig. 7. Electron micrograph from the 2nd turn of the organ of Corti, 4 weeks of exposure to noise—disarrayed stereocilia. Bar=1 μm .

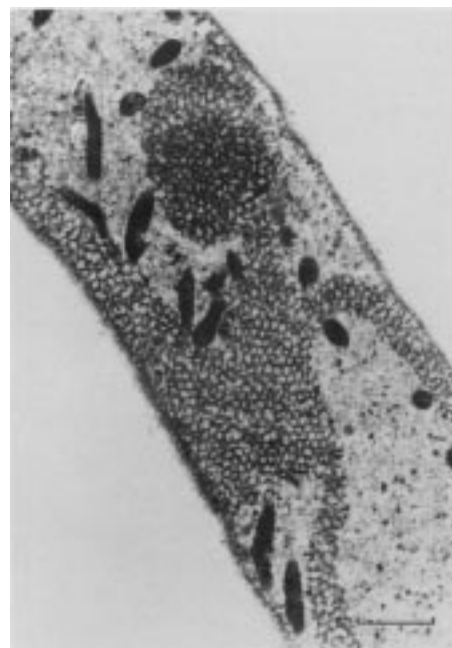


Fig. 8. Electron micrograph from the 2nd turn of an exposed-to-noise guinea pig organ of Corti, 4 weeks of exposure to noise—proliferation of subsurface cisterns. Bar=1 μm .

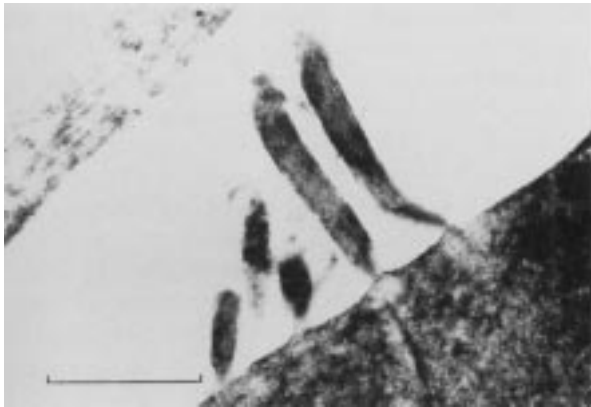


Fig. 9. Electron micrograph from the 2nd turn of the organ of Corti, 8 weeks of exposure to noise—broken stereocilium of OHC1 and softened cuticular plate around the rootlets of cilia. Bar=1 μ m.

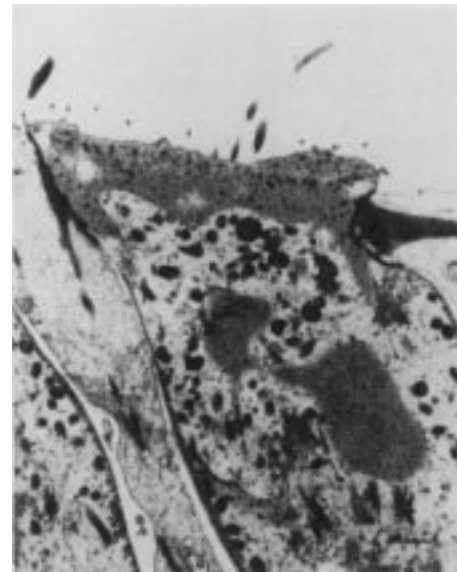


Fig. 11. Electron micrograph from the 3rd turn of the organ of Corti, 12 weeks of exposure to noise—the cuticular plate is distorted and has bulged outward into the subtektorial space. Bar=1 μ m.

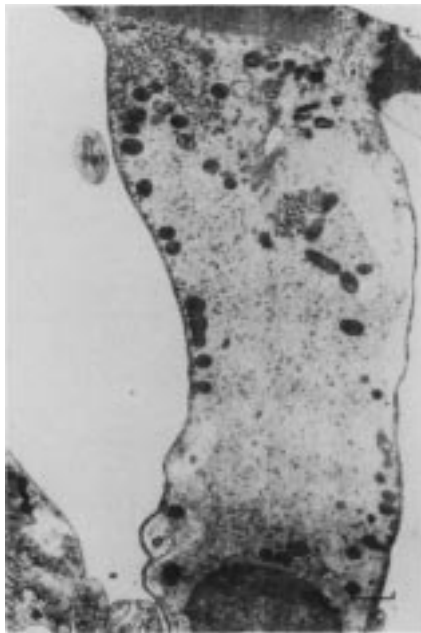


Fig. 10. Electron micrograph from the 3rd turn of the organ of Corti, 8 weeks of exposure to noise—OHC swelling. Bar=1 μ m.

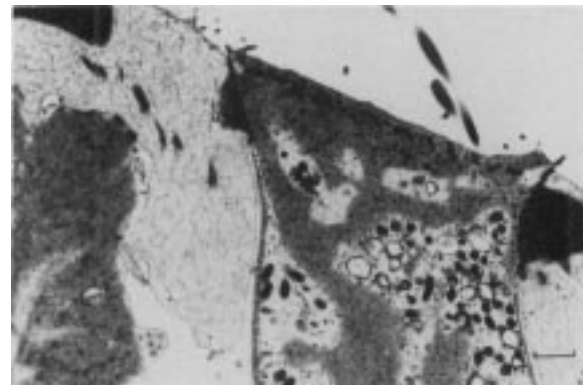


Fig. 12. Electron micrograph from the 2nd turn of the organ of Corti, 12 weeks of exposure to noise—cuticular plate is softened, distorted and has bulged outward into the subtektorial space. Increase in the number of lysosomes. Bar=1 μ m.

appeared to be permanent. Pronounced swelling of the OHC bodies was seen (Fig. 10).

After 12 weeks of exposure, the most evident pathology concerned the stereocilia and cuticular plates. Stereocilia were fractured, broken, torn off or missing. Cuticular plates were distorted and very often protruded, bulging outward into the subtektorial space (Fig. 11). In the cell body, a significant increase in the number of lysosomes and Hensen's bodies was observed (Figs. 11 and 12).

The region of nerves and synapses did not reveal any recognizable pathology.

Control group: In the control group of animals, no damage to hair cells in the organ of Corti was found after 4 weeks of the experiment (Fig. 5a). After 8 and 12 weeks of the experiment, a few missing OHC3 in the apical part of the cochlea were occasionally detected.

Discussion

It has been known that prolonged exposure to industrial

noise at moderate levels causes a relatively fast and dynamic increase in permanent hearing threshold shift (PTS), followed by a slow deterioration in hearing²⁾. In this study, the development of a PTS of about 23 dB was observed after the first 4 weeks of exposure. After a subsequent 8 weeks of exposure, the mean hearing threshold did not deteriorate significantly in comparison to the control group. A slight increase in hearing impairment, observed in both control and experimental group after 8 and 12 weeks of exposure, was similar in the two groups and may be explained by presbycusis.

Typically (in more than 70%, regardless of the duration of exposure to noise), the hearing loss varied from 20 to 30 dB. This first 30 dB of permanent hearing loss after exposure to industrial noise, as shown earlier by Hamernik *et al.*, on chinchillas¹⁹⁾ and confirmed by the results of this study, is caused primarily by damage to OHC, which are much more vulnerable to noise-trauma than IHC. In this study, however, animals were observed with a hearing threshold shift of up to 30 dB with no changes in hair cells visible through light microscopy. In such cases, the evident pathological damage on OHC and IHC stereocilia were, in principle, much more evident and widespread than the damage to OHC. A similar observation concerning acute trauma was made by Liberman^{20,21)}, so that it should be concluded that the damage to stereocilia, and not to the hair cells themselves, is responsible for hearing impairment due to noise. As shown by Gao *et al.*, permanent hearing loss is connected to irreversible changes in the stereocilia of hair cells²²⁾.

In spite of hearing threshold shift, as long as the IHC remain intact no significant impairment in the synchronization of acoustic nerve response is observed, as was shown by analyzing the latency of wave I in the ABR in this study.

The occurrence of hearing loss (temporary or permanent) is the most obvious consequence of exposure to intense sound, but the anatomical changes in the auditory system that accompany this are more subtle, and no straight correlation between hearing loss and pathomorphology was found in earlier observations¹²⁾. Boettcher *et al.*, showed that after exposure to noise at a moderate level (95 dB SPL), the threshold shift on the first days of exposure decreased as the duration of exposure increased (about 10 dB after the 15th day of exposure). But in contrast to the improvement in threshold, the damage to hair cells and stereocilia increased. The results of this study partly confirm the observations of Boettcher *et al.* We demonstrated that the damage to the cochlea caused by industrial noise progresses as the duration of exposure increases and does not correlate with permanent hearing threshold shift. This study found that after 4 weeks of exposure, only OHC1 and OHC3 were damaged, while, after longer exposure, missing IHC and outer pillar cells were also detected.

Cellular debris in the endolymph, indicative of intense cell degeneration, was seen only after longer exposure. At the subcellular level, the progression in cochlear injury in relation to the duration of exposure was even more evident, especially in the stereocilia and in the upper portions of hair cells. The longer the exposure, the more severe and profound was the damage observed to stereocilia and cuticular plates. After 4 weeks of exposure, the changes in stereocilia were reversible (mainly floppy stereocilia). After longer exposures, signs of irreversibility in the changes were seen (fractured, broken, torn out, missing). After 4 weeks of exposure, there were no changes to the cuticular plate. After longer exposures, the structure of the cuticular plate was softened, and the plate protruded, bulging outward into the subtektorial space.

In addition, cochlear damage did not depend upon the permanent threshold shift value. Despite the fact that the morphology was assessed in animals with similar (20–30 dB) levels of hearing loss, cochlear damage varied significantly from animal to animal, and progressed as the duration of exposure increased, regardless of the stable hearing level.

It is known that the mechanism of cochlear damage depends upon the level of noise. Two processes, mechanical (or micromechanical) and metabolic, have been suggested as mechanisms that cause the hair cell damage with overstimulation of the ear. Generally, mechanically induced injuries have a rapid onset, whereas those induced by metabolic exhaustion have a more gradual onset²⁻⁴⁾. It has been further suggested that for moderate exposures (up to 100 dB) the mechanism of trauma is mainly metabolic, while for higher intensity exposures the mechanical damage is more important. The signs of metabolic exhaustion have been observed in this study. After a shorter exposure (4 weeks) what was seen was mainly a depletion of subsurface cistern and an increase in the number of mitochondria, suggesting a deficiency in fuel utilization, protein synthesis and energy production²³⁾. After longer exposures, an increase in the number of lysosomes was observed, suggesting an increase in the synthesis of lipids. The disturbance of lipid synthesis due to noise trauma may impair the selective permeability of the cell membrane, leading to its lysis²³⁾.

Apart from a metabolic mechanism, microtraumata should be considered in the development of industrial noise-induced hearing loss, especially in the earlier stages of the disease. In this study, after 4 and 8 weeks of exposure, a distortion of the organ of Corti, seen as “waves” along the cochlea, and mechanical changes in the stereocilia were observed, which may confirm the effects of this mechanism. Industrial noise-induced hearing loss seems to be a dynamic process, in the development of which both micromechanical and metabolic factors play a significant role.

Conclusions

1. Prolonged exposure to industrial noise causes, in guinea pigs, a relatively fast increase in permanent hearing threshold shift (in most animals, up to 30 dB), which reaches an asymptotic level by 4 weeks of exposure.
2. The first 30 dB of permanent hearing threshold shift is caused almost exclusively by damage to the outer hair cells, particularly to their stereocilia and cuticular plates.
3. The degree of cochlear damage is not related to permanent hearing loss.
4. The progression in cochlear pathology depends upon the duration of exposure.
5. Both micromechanical and metabolic mechanisms seem to be involved in the development of industrial noise-induced hearing loss.

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