Noise-Induced Hearing Loss in Working Environment and its Background

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Abstract: Noise-Induced Hearing Loss in Working Environment and its Background: Ikuharu MORIOKA, et al. Department of Hygiene, School of Medicine, Wakayama Medical University—The excessive exposure to noise results in temporary and/or permanent changes in hearing ability in both human and animal subjects. The noise-induced hearing loss in workers was previously known as industrial deafness and it is still an important problem in occupational health. The purpose of this review is to describe the major findings obtained in epidemiological and experimental studies on the effects of noise in the working environment on man. The discussion begins with a classification of noise-induced hearing loss and the background of hearing impairment in workers. The degree of noise-induced hearing loss depends on both the characteristics of noise and the individual sensitivity to noise. Factors related to noise-induced hearing loss are reviewed. When the hearing ability is evaluated among aged workers, presbycusis cannot be ignored. Important new evidence is reviewed on hearing impairment caused by both aging and noise-induced hearing loss. Exposure to intensive noise changes the structure and function of the basilar membrane, sensory hair cells, the tectorial membrane and tip links, and the cochlear blood flow. The pathology and pathophysiology of noise-induced hearing loss are also explored. Relation between exposure to noise and hearing impairment is discussed to predict the effects of long-term exposure. Finally, the diagnosis and prevention of noise-induced hearing loss are reviewed.

Key words: Noise-induced hearing loss, Occupational noise, Auditory perception, Cochlea, Risk factors, Work, Auditory threshold, Deafness

In the field of human ecology, a sound which is unpleasant and disturbs daily life activity, is called noise, despite the physical characteristics of the sound. Noise often occurs in daily life, and brings feeling of unpleasantness, interference with communication, sleep disturbance, and various physiological dysfunctions including noise-induced hearing loss. In this review, the subjective noise is limited to noise in the working environment. We survey the recent trend of studies about its effects on man.

Effects of noise in the working environment less amenable to standardization at this time, such as effects on the extra-auditory system, namely, stress actions and physiological responses (systemic circulation, autonomic nervous system and so on), effects of interference with speech on safety, effects on working efficiency and annoying effects, are summarized in the annex A in ISO/DIS 9612: Guidelines for the measurement and assessment of exposure to noise in the working environment. Yamamoto systematically reported relationship between the effects of noise and working environment control, focusing on noise-induced hearing loss that is a characteristic influence of noise in the working environment and is repeatedly studied to establish the quantitative causal relation to noise in the working environment. Takeda also recently discussed working environment control, work control and health management related to noise in the working environment. There is a valuable review by Shusterman and Sheedy about occupational hazards to vision, hearing, balance, olfaction and taste, of which treatments are limited to a small fraction of special medical practices in ophthalmology and otolaryngology.

Since various existing reviews deal with the general effects of noise in the working environment, we decided to limit the scope of this review to noise-induced hearing loss.

1. A classification of noise-induced hearing loss

Hearing impairment induced by exposure to intensive noise is generally classified and explained from the standpoint of origin as follows:

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1.1. Traumatic hearing loss

(1) Acute acoustic trauma

This is well-known acoustic injury in the narrow sense. It results from exposure to unexpected intensive noise. If the sound level is more than 125–130 dBA, even very short-time exposure often causes hearing impairment. If the sound level exceeds 140–150 dBA, such as an explosive noise, the noise instantly causes severe hearing impairment.

(2) Acute acoustic sensorineural hearing loss

This includes so-called disco hearing loss, and so on. It often results from expected short-time exposure to intensive noise, but it happens after the longer time exposure than acute acoustic trauma. The sound level is regarded as 100–120 dBA. This level is not so intense as to introduce hearing loss in all persons who hear it.

It is possible to recover from the traumatic hearing loss by receiving an early treatment.

Noise-induced sudden deafness unexpectedly occurs due to occupational long-term exposure to noise. This is not traumatic. It is closely related to an individual vulnerability to noise in the peripheral hearing organ. Many patients suffering from noise-induced sudden deafness have not complained of deafness although they have already been exposed to the same kind of intensive noise frequently or for a long time. Noise-induced sudden deafness often occurs in only one ear.

1.2. Noise-induced hearing loss

This, in general, is an irreversible hearing impairment caused by long-term exposure to noise exceeding 85 dBA. Noise-induced hearing loss occurs typically at high frequencies, usually with a maximum hearing level at around 4 kHz (4 kHz dip) as shown in Fig. 1. It occurs gradually, usually over a period of 1–5 years. At the time, it is often not recognized because it does not impair speech communication. As the duration or sound level of the exposure increases, the hearing loss progresses and spreads to lower and higher frequencies. The person affected usually is aware of this change after the noise exposure has been of sufficient severity and duration to affect the speech frequency ranges (0.5, 1 and 2 kHz, as tested on an audiometer), and of these frequencies, the hearing level at 2 kHz shows the first involvement. The audiogram shows a typical curve of the sensorineural hearing loss. The bone conductive hearing level also falls, and recruitment of hearing occurs. This leads to a decreased threshold of discrimination of sound level and decreased speech discrimination to less than 75%. Efficacy of a hearing aid cannot be expected. Such hearing loss progresses remarkably for the first 10–15 years of exposure to noise, and then slowly.

The degree of hearing loss varies with frequency composition, the noise state and the exposure time, in addition to the sound level. It also depends on the sensitivity to noise, which differs considerably among individuals.

2. Background of hearing impairment in workers

The prevalence rates for hearing loss, defined as 25 dB averaged over 0.5, 1 and 2 kHz, are assumed to be 77 males and 70 females for every 1,000 adults in a certain western industrialized country. The prevalence estimates of causes are shown in Fig. 2. Clearly, among males, noise-induced hearing loss is the most prevalent of all causes of hearing loss.

An etiologic paradigm of hearing loss in industrial workers is shown in Table 1. Attention has
focused in recent years on ototoxic organic solvents and noise. The adjusted relative risk estimates were 11 times greater for the group exposed to noise and toluene\textsuperscript{14}). Styrene is also probably a human neurotoxicant\textsuperscript{15, 16}). For this reason, the influence of industrial ototoxic factors as well as noise in the working environment on presbycusis will become impossible to ignore in the future. Future research is necessary on the combined effect of organic solvents and noise.

Extra-occupational factors, including age, noise exposure outside the working environment, ear disease, ototoxic drugs and trauma, are shown in Table 1. Figure 3 shows the range of maximum sound levels for common nonoccupational noise\textsuperscript{17}). Although all activities have the potential for dangerous levels of exposure to noise, the most serious threat to hearing comes from recreational activities. Exposure to gunfire noise is high enough to cause severe hearing impairment if effective hearing protectors are not worn at all times when shooting\textsuperscript{18}).

3. Factors related to noise-induced hearing loss

Those who live in non-industrialized areas (relatively noise-free environments) have been found to have more acute hearing than the urban population in corresponding age groups. It is not clear whether such audiometric differences are due to the lack of exposure to noise. Differences in the time of onset and magnitude of noise-induced hearing loss may result from genetics, connective tissues changes, atherosclerosis, metabolism, nutrition, climate or stress\textsuperscript{19}). Recent studies report that noise-induced hearing loss is related to noise in the living environment, nutrition, general condition of health, blood flow and melanin in the inner ear.

3.1. Living environmental noise

Some studies show that there is a temporary threshold shift (TTS) after using headphones on a portable cassette player (Walkman and so on)\textsuperscript{20}). The ratio of those with hearing loss exceeding a certain limit is higher among users of headphones than among non-users\textsuperscript{21}). Some users are found to have a 4 kHz dip on their audiograms\textsuperscript{22}). Some reports therefore warn that using portable cassette players with headphones causes noise-induced hearing loss\textsuperscript{23}). A limit of 90 dBA is recommended for personal cassette players\textsuperscript{24}). Practical research, however, shows that the risk of permanent noise-induced hearing loss is not so high under usual listening conditions because the sound is appropriately adjusted by each person to relatively safe sound levels\textsuperscript{20}). Nevertheless, we must remember that occupational noise and additional living environmental noise prolong the total time of exposure to noise and increase the total amount of sound energy.

3.2. Nutrition

Vitamin B\textsubscript{1} deficiency was once widely accepted as one of the causes of hearing impairment. The effect of vitamin A deficiency in guinea pig on noise-induced TTS was evaluated after 15-min
acoustic overstimulation with 90 dB white noise\textsuperscript{25). Statistical analysis of compound action potential data showed that vitamin A deficiency increased the sensitivity of the inner ear to noise, namely the probability of noise-induced hearing loss. Many (47\%) army personnel exposed to military noise with chronic tinnitus and noise-induced hearing loss, exhibited vitamin B\textsubscript{12} deficiency (vitamin B\textsubscript{12} serum level was less than 250 pg/ml\textsuperscript{26}). Iron deficiency can provide a pathological basis for noise-induced hearing loss, because the TTS induced by steady state white noise was significantly greater in iron-deficient rats than in normal rats, and because most iron-deficient rats with a greater threshold shift showed varying degrees of permanent shift at 11 days after exposure\textsuperscript{27}.

3.3. General condition of health

Meyer-Falcke et al.\textsuperscript{28) examined the prognostic value of numerous parameters for the development of noise-induced hearing loss to devise a test for noise sensitivity. The most sensitive parameters were diastolic blood pressure, on and off finger pulse reaction and TTS between 3 and 6 kHz.

The relationship between hypertension and noise-induced hearing loss is often reported\textsuperscript{29-32). Talbott et al.\textsuperscript{29) examined 245 retired metal assembly workers aged 56 to 68 with chronic noise exposure of 30 years or more at greater than or equal to 89 dBA. They found that there was a significantly increased prevalence of hypertension (greater than or equal to 90 mmHg diastolic and/or currently taking hypertensive medicine) among older workers (ages 64 to 68) with severe noise-induced hearing loss (greater than or equal to 65 dBA loss at 3, 4 or 6 kHz). Gates et al.\textsuperscript{30}) determined the hearing status of a cohort of 1,662 elderly men and women, and compared it with their 30-year prevalence of cardiovascular disease. They found that a low-frequency hearing level (arithmetic average of 0.25, 0.5 and 1 kHz) was related to cardiovascular disease in both sexes but more in women. In women, the odds ratio for having any cardiovascular disease for a 40 dB low-frequency hearing level was 3.06. In men with a 40 dB low-frequency hearing level, the odds ratio for stroke was 3.46 and for coronary heart disease it was 1.68.

3.4. Other factors related to the inner ear

Regarding the blood flow in the cochlea, Okada et al.\textsuperscript{31) found that the spiral vessels in the guinea pig dilated after exposure to rock music (110-120 dB SPL, 1-3 hr) and then constricted. They point out that this finding—that early dilation of the spiral vessels is followed by constriction—may be one of the causes of noise-induced TTS.

Barrenas and Lindgren\textsuperscript{32) examined the relationship between skin pigmentation and noise-induced TTS to investigate the function of inner ear melatonin. The subjects were Caucasians divided into three groups according to their sun sensitivity. The noise exposure consisted of a 1/3-octave band-filtered noise with a center frequency of 2 kHz at 105 dBB SPL for 10 min. As a result, the most pigmented subjects developed the least TTS in the frequency range 2–8 kHz, and the least pigmented subjects the greatest TTS.

4. Hearing impairment caused by aging and noise-induced hearing loss

Hearing ability decreases with age (presbycusis). Corrections for aging should therefore be considered when examining data on hearing impairment caused by exposure to noise. Noise-induced hearing loss is the quantity of hearing loss attributable to noise alone, after values for presbycusis have been subtracted. These values may differ slightly according to where and how the presbycusis data are collected. The general pattern of progression of presbycusis has been established, and data are available in reference sources\textsuperscript{33, 34). ISO 1999\textsuperscript{35) provides the determination of occupational exposure to noise in the working environment and the estimation of noise-induced hearing loss. It adopts the fundamental hypotheses that the sound exposure level is desirably estimated by the equivalent sound pressure level for a nominal 8 hr working day and that the measured hearing level is the algebraic sum of hearing loss only by aging and hearing loss only by the noise. In other words, the following empirical formula is used as a related expression of both.

\[
\text{HTLAN} = \text{HTLA} + \text{HTLN} - (\text{HTLA} \times \text{HTLN}) / K
\]

where, HTLAN is the dB value for the measured hearing level associated with age and noise, HTLA the hearing level associated with age (presbycusis) and HTLN the actual or potential noise-induced permanent threshold shift (PTS). K is a constant, and was adopted as 120 in ISO 1999. If K becomes infinity, (HTLA + HTLN)/K becomes 0. Thus, HTLAN is a value simply including HTLN and HTLA.

The hearing level of a noise-unexposed population associated with age (HTLA) depends on these fundamental hypotheses. The International Standard permits two data bases (data bases A and B) to be used for HTLA. Data base A (Table 2) derives from otologically normal persons, i.e. persons in a normal state of health who are free all signs or
symptoms of ear disease and from obstructing wax in the ear canal and who have no history of undue exposure to noise. The statistical distribution of the hearing level is also shown in ISO 7029\(^{36}\). In data base B, a set of data were collected from a population not occupationally exposed to noise. Data base B in Table 3 is for an industrialized country. Which data base, A or B, is more suitable as a control group for hearing handicapped persons in a work place, depends on the working environment.

On the other hand, Bies and Hansen\(^{38}\) mathematically examined the former formula (1) that is the elementary hypothesis of ISO 1999. They then proposed that the HTLA and HTLN should be additive on an antilogarithmic basis, as shown in formula (2).

\[\text{HTLAN} = 10 \log(10^{\text{HTLA}/10} + 10^{\text{HTLA}/10} - 1) \quad (2)\]

To examine the fitness of both formulas, Macrae\(^{39}\) examined deterioration in the hearing level at 4 kHz due to aging (average 11.3 years of age) in 240 male war veterans (30-75 years of age at the initial test) with high-frequency sensorineural hearing loss by acoustic trauma or other war-time noise exposure. His data did not support the formula (2) but provided support for the formula (1).

Clark and Boh\(^{40}\) conducted a recalculation of the values contained in data bases A and B of ISO 1999, utilizing procedures specified in the standard and its supporting documents. Three simple errors in calculation, which occurred in the supporting documents and were perseverated into the standard, were identified. A corrected version of data base B (Table 3) is shown. In other words, a correction was needed at the 10th percentile at 3 kHz for 30-year-old males (20 should be 30), the 10th percentile at 6 kHz for 30-year-old males (32 should be 48), and the 90th percentile at 1 kHz for 50-year-old females (+4 should be −4).

ISO 1999\(^{37}\) provides statistical models for hearing threshold shifts associated with aging and exposure to noise, as mentioned above. When the intensity and duration of exposure to noise are known or can be estimated, a method has already been proposed for the allocation of hearing loss between aging and noise in individual cases\(^{41}\). When an individual's hearing level exceeds the sum of the median levels expected given that individual's age, gender, exposure level and duration, the appropriate allocation depends on the correlation between age-related and noise-induced changes. Software that is capable of calculating hearing loss from audiometric data and the proportion of hearing loss due to presbycusis, has been developed\(^{42}\). Four patients illustrate the utility of this type of computer analysis. There is, however, a report indicating that the additivity is no longer valid at higher ages and that the term "age correction" is inadequate\(^{43}\).

Rosenhall et al.\(^{44}\) studied the influence of aging and occupational exposure to noise on hearing ability.

### Table 2. Hearing threshold level, in decibels, from data base A (from Ref. 37)

<table>
<thead>
<tr>
<th>Frequency kHz</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
</tr>
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<tbody>
<tr>
<td>Age, years</td>
<td>90</td>
<td>50</td>
<td>10</td>
<td>90</td>
</tr>
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<td>Males</td>
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<tr>
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<td>−6</td>
<td>1</td>
<td>9</td>
<td>−5</td>
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<td>6</td>
<td>−8</td>
<td>3</td>
<td>16</td>
<td>−5</td>
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<tr>
<td>Females</td>
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<td></td>
<td></td>
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<tr>
<td>0.5</td>
<td>−6</td>
<td>1</td>
<td>9</td>
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<tr>
<td>4</td>
<td>−7</td>
<td>1</td>
<td>12</td>
<td>−6</td>
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<tr>
<td>6</td>
<td>−8</td>
<td>2</td>
<td>14</td>
<td>−6</td>
</tr>
</tbody>
</table>

Data base A is auditory threshold values from otologically normal persons with no history of undue exposure to noise.

### Table 3. Hearing threshold level, in decibels, of an unscreened population from data base B (from Ref. 37)

<table>
<thead>
<tr>
<th>Frequency kHz</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>90</td>
<td>50</td>
<td>10</td>
<td>90</td>
</tr>
<tr>
<td>Males</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>0.5</td>
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<td>7</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>−5</td>
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<td>13</td>
<td>−3</td>
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<td>2</td>
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<tr>
<td>4</td>
<td>−1</td>
<td>10</td>
<td>38</td>
<td>4</td>
</tr>
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<td>6</td>
<td>8</td>
<td>18</td>
<td>32</td>
<td>11</td>
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<tr>
<td>Females</td>
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<td></td>
</tr>
<tr>
<td>0.5</td>
<td>−6</td>
<td>1</td>
<td>15</td>
<td>6</td>
</tr>
<tr>
<td>1</td>
<td>−6</td>
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<tr>
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<td>−4</td>
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<tr>
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<td>−5</td>
<td>4</td>
<td>16</td>
<td>−4</td>
</tr>
<tr>
<td>6</td>
<td>3</td>
<td>12</td>
<td>25</td>
<td>5</td>
</tr>
</tbody>
</table>

Data base B is auditory threshold values from a population not occupationally exposed to noise.
ty in a longitudinal and an age cohort comparing studies of the participants 70, 75 and 79 years of age. As a result, hearing ability was found to decrease with age. Seventy-year-old men exposed to occupational noise had 10 to 15 dB poorer hearing in the high frequency range than unexposed men. The difference in hearing ability decreased with age. The difference between exposed and unexposed older persons was no longer significant at age 79. In women there was no difference in hearing ability between those exposed to noise and those unexposed. Men unexposed to noise had 10 to 15 dB poorer hearing at 4 kHz than women of the same age also unexposed to noise. A gender difference concerning susceptibility to noise could be one possible explanation. Another possible explanation is that the sound levels in factories where women worked were lower than in factories where men worked.

On the other hand, Takeda et al.\textsuperscript{45)} have defined the upper limit of hearing as the maximum audible frequency measured with fixed intensity and changing frequency. They measured the upper limit of hearing in 6105 otologically normal ears of subjects ranging in age from 5 to 89 years and have established the standard upper limit aging curves from the age variation (Fig. 4). To clarify the effects of noise in the working environment on the upper limit of hearing, Morioka et al.\textsuperscript{46)} measured it in 239 healthy male workers exposed to noise. Compared with the standard upper limit aging curves, the age variation deteriorated significantly. Even if ears that had normal hearing levels (35 dB or less) were selected, the deterioration in the upper limit of hearing was noticeable (Table 4). They therefore suggest that the upper limit of hearing may serve as clinically useful information on the hearing loss that precedes noticeable hearing loss in conventional audiometry for workers exposed to noise in the working environment. Recently the extended high frequency threshold is often used to detect noise-induced hearing loss\textsuperscript{47)}. The upper limit of hearing is a very sensitive method among several audiometric tests for noise-induced TTS, although it depends on the help and experience of the subjects tested\textsuperscript{48).}

Table 4. Variation in the upper limit of hearing in workers exposed to noise in the working environment whose hearing level was less than 35 dB at each frequency in conventional audiometry, by noise and by working carrier (from Ref. 46)

| Sound level (dBA) | Working career (years) | Upper limit of hearing (
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<tbody>
<tr>
<td></td>
<td>-25th percentile (n)</td>
<td>25-50th percentile (n)</td>
</tr>
<tr>
<td>≥ 85</td>
<td>0-19</td>
<td>2</td>
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<tr>
<td>30-</td>
<td>0-19</td>
<td>0</td>
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<tr>
<td>&lt; 85</td>
<td>20-29</td>
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<td>7</td>
<td>10</td>
</tr>
<tr>
<td>0-9</td>
<td>8</td>
<td>19</td>
</tr>
</tbody>
</table>

* * p<0.01.

5. Pathology and pathophysiology of noise-induced hearing loss

Cochlear anatomy related to cochlear micro-mechanics has already been reviewed\textsuperscript{49)}. The TTS is related to the ciliary rigidity in the hair cells of cochleas. Intensive noise causes a loss of ciliary stiffness. The floppy cilia become erect following recovery from TTS. Repetition of TTS brings degeneration of the hair cells.

In the Journal of the Acoustical Society of America in 1991, there were a series of reviews on the pathology and pathophysiology of noise-induced hearing loss. Among them, three reviews titled "acoustic injury in the cochlea and peripheral auditory system\textsuperscript{50)}," "human temporary threshold shift (TTS) and damage risk\textsuperscript{51)}," and "TTS and permanent threshold shift (PTS) in animals\textsuperscript{52)}," agree with the focus of this head. In particular, the relationship between the minute construction and function of the peripheral auditory system has been elucidated by means of the scanning electron microscope and cell biology rapidly in the past 10 years. This has made it possible for the micromechanics of the organ of Corti to explain the process of sound reception\textsuperscript{53}). With this advance, pathophysiological studies on noise-induced hearing loss have also rapidly advanced.

![Fig. 4. Standard upper limit age curves for males (redrawn from Ref. 45).](image-url)
5.1. Hair cell transduction
The hair cell converts vibrational energy into intracellular electrochemical reactions, and eventually neurotransmitter release. The essential element of this process is the transduction channel at the tip of the stereocilia\(^{49}\). This channel is activated by a mechanically controlled gating process associated with movements of the hair bundle.

During an excitatory deflection of the hair bundle, shear forces between adjacent hairs cause the tip links to stretch, opening a channel and thus allowing entry of charged (K\(^+\)) ions into the cell. The resulting transduction current depolarizes the plasma membrane\(^{49}\). This depolarization spreads to the base of the cell and becomes a trigger which releases the neurotransmitter.

5.2. Basilar membrane mechanics
The basilar membrane was considered to undergo simple passive linear vibration. Recent measurements have completely altered this notion. The basilar membrane is highly non linear and very sharply tuned\(^{54}\). The non linearity appears to be part of an active motility of the outer hair cells. The sharp tuning depends on its communication with outer hair cells, the tectorial membrane and the cochlear active process associated with the outer hair cells.

5.3. Roles of the hair cells and K ion in the endolymph related to the cochlear active process
Active motility of the outer hair cells in the organ of Corti was proved and shown to contribute to active mechanical control of the basilar membrane motion (the cochlear active process). ‘Tuning curves’ for motile response were obtained from the isolated outer hair cells\(^{55}\). Thus it was made clear that highly selective frequency discrimination in the inner ear is achieved by the active motile responses of the outer hair cells. More recent research has focused on motility in the isolated cochlea. Morioka et al.\(^{60}\) showed tuned displacement responses in the plane of the organ of Corti with an isolated temporal bone preparation. The effect of intensive noise on such an active reception mechanism will be discussed in further studies.

Cody and Russell\(^{57}\) made intracellular recordings from both inner and outer hair cells in the basal turn of the guinea pig cochlea. Receptor potentials exhibit the largest decrease at the characteristic or best frequency. This frequency selective loss of sensitivity resulted in a decrease in the tuning of the hair cells. They suggest that the outer hair cells are the target organ of overstimulation based on evidence that cochlear active processes associated with the outer hair cells are responsible for sharp tuning in the inner ear.

One hypothesis concerning the role of outer hair cell motility during overstimulation has been developed\(^{57}\). This idea is based on the observation that slow outer hair cell motility occurs when the hair cells are exposed to high K medium\(^{58}\). The K utilization by the outer hair cells may be expected to produce changes in the endolymph concentration of the K ion during and after overstimulation. Then, the effects of acoustic overstimulation on the K ion concentration in the endolymph were examined in the guinea pig. The results showed that there was little change in the K ion concentration 30 min after 30-min exposure to a 2 kHz pure tone at 120 dB SPL\(^{59}\).

5.4. Tectorial membrane
Changes in the tectorial membrane were proved after a PTS (1 kHz at 105 dBA for 72 hr). The tectorial membrane showed waviness, fiber clumping and a discontinuous or curved Hensen’s stripe in the middle zone of the membrane\(^{60}\). The thickness and stiffness of the membrane was decreased. It is important to note that the middle zone of the membrane lies directly above the inner and outer hair cells, and these structural changes may be expected to affect stereocilia stimulation.

5.5. Structural damage to stereocilia, in particular, tip links
Figure 5 shows a number of potential injury sites in stereocilia induced by acoustic overstimulation\(^{61}\). The tip links may be damaged by intensive noise stimulation (Fig. 5a). The distortion of the tip links between the stereocilia gives rise to sensory transduction\(^{62}\). The disruption of the tip links may in this way severely impair the important function of the hair cells\(^{63}\). The status of the lower part of the stereocilia and the continuity of the cuticular plate play an important role in determining the reversibility of the threshold shifts\(^{64}\).

Other studies show changes in intracellular organelles in the hair cells and a synapse to the nerve ending after exposure to noise\(^{65,66}\).

5.6. Relation between hair cell loss and permanent threshold shift
Many studies of noise-induced hearing loss concentrate on the relationship between hair cell loss and PTS. Hamernik et al.\(^{57}\) reported on the basis of experimental data obtained from 420 noise-exposed chinchilla that the amount of hair cell loss was quantitatively related to the amount of PTS at 8 audiometric test frequencies between 0.125 and 16
kHz. Their results for the 4 kHz octave band are shown in Fig. 6. The first approximately 30 dB of PTS was established by loss of primarily outer hair cells, and subsequently the higher level of PTS depended on loss of inner hair cells. The three rows of outer hair cells showed the same degree of loss for a given PTS.

On the other hand, Kaltenbach et al. examined the damage to hair cells and their stereocilia in the Syrian golden hamster with exposure to a 10 kHz pure tone. The exposure conditions were varied over an intensity range of 90-129 dB SPL; the exposure periods were varied over a range of 30 min to 4 hr. The lesions in animals with mild damage were commonly restricted to either the inner hair cells and/or the first row of outer hair cells. The order of damage susceptibility was inner hair cells, and the first row, second row, and third row of outer hair cells. Damage to the second and third rows of outer hair cells was found only in animals with the severest lesions.

5.7. Regeneration of hair cells in the chick cochlea

The sensory epithelium of a bird is quite similar to the organ of Corti of a mammal. Surprisingly, the hair cells in the bird cochlea are regenerated even after damage induced by exposure to noise. Quantitative data indicate that an exposure caused a 35% loss of hair cells. The new hair cells are found in four or five days after the loss. After 14 days' recovery, no hair cell loss could be detected. The hearing ability also returns to preexposure levels over the course of 100-600 hr. Even if new hair cells are produced, it is not yet clear if or how the neural connections of nerve fibers reestablish themselves.

5.8. Changes in the cochlear blood flow after intensive noise exposure

Laser Doppler techniques and microsphere methods have been used to study cochlear blood flow after noise exposure. Blood flow reduction, by as much as 70%, was found in the cochlea of the guinea pig after a 1-hour exposure to 110 dB SPL high-pass noise (10-40 kHz). Similarly, exposure to industrial noise for three months significantly decreased the blood flow in the cochlea of rats.
Okamoto et al.\(^{(73)}\) examined the effects of the frequency and intensity of sound on blood flow in the cochlea of the guinea pig. No change in blood flow was found in the basal turn at lower frequencies (6 and 7 kHz) up to 120 dB SPL. The 120 dB SPL sound of at higher frequency (10 kHz) induced a decrease in blood flow only in the basal turn. These results suggested that there was a corresponding blood flow area sensitive to specific frequency in the cochlea, but it still has not been confirmed whether the such decrease in blood flow in the cochlea is related to the decrease in the cochlear function and loss of outer hair cells.

Chronic exposure to intensive noise induces chronically decreased blood flow in the cochlea, and this results in ischemia in the area controlled by the blood flow\(^{(74)}\). The basilar membrane of the human cochlea sensitive to a sound in the 3 to 6 kHz frequency band, corresponds to the anatomical position of ending anastomosis of the cochlear branch of the cochlea propria artery. In this ending anastomosis a decrease in blood flow readily occurs, and it does not recover sufficiently. This may be the cause of the high frequency dip (4 kHz dip, etc.) which is the initial finding in noise-induced hearing loss.

6. Relation between exposure to noise and hearing impairment

6.1. Combined effects of intensity and duration of exposure to noise

Most data on long-term noise hazard are related to occupational exposure. In order to predict the effects of long-term exposure, the results of epidemiological and experimental studies must be used. It is difficult to establish limits for safe noise exposure, since predictions by various methods conflict with each other. To correct for intermittency and to extrapolate to 24 hr, two hypotheses are necessary\(^{(75)}\). The following is a brief review of two hypotheses used to integrate the combined effects of intensity and duration.

The equal temporary effects rule is the hypothesis that the PTS due to long-term, daily, steady-state noise exposure is equal to the average TTS produced by the same daily noise in healthy young ears. Later studies support the observation that for a given length of exposure, frequently interrupted noise is less harmful than continuous steady-state noise at the same level\(^{(76)}\). An extension of this hypothesis is that PTS is unlikely if there is complete recovery from the TTS before the beginning of exposure the next day. An early occupational noise criterion was based on this hypothesis\(^{(76)}\).

The equal energy rule is the theory that the hazard to hearing is determined by the total sound energy (the integrated product of sound intensity and duration) entering the ear each day. This rule has natural appeal, since the exposure dose is quite simple to assess and, according to epidemiological studies, is reasonably well correlated with the accumulated physical damage. It should be noted that the range of sound duration covered by this rule may be limited by the need for protection against possible damage by high level, short duration, impulsive sounds\(^{(1)}\).

To simplify different damage risk criteria, noise exposure histories are frequently expressed as equivalent sound levels. The equivalent sound level expresses the sound level of the steady state noise having the energy (average squared sound pressure level) during a certain time equal to that of the noise changing with time. The equivalent sound level in the working environment is usually shown as noise exposure level $L_{eq}$ standardized for 8 hr. Many studies agree that this equivalent sound level closely corresponds to the human physiological and psychological reaction to non-steady state noise. It is therefore widely used to evaluate non-steady state noise. There is, however, an argument against the equal energy rule\(^{(8)}\), and some studies show it is not always suitable in the case of an impact or impulse noise\(^{(77-79)}\).

It is important to notice that a limit for safe exposure to noise in the working environment is set merely to prevent human noise-induced PTS. Even in the working environment where a limit of 85 dBA is strictly enforced, the noise in the working environment is often poorly matched to workers, as pointed out by Hetu\(^{(80)}\). Careful attention should be paid to the auditory sensitivity to noise of an individual worker.

6.2. Temporary threshold shift and permanent threshold shift

Available data show that there is considerable variation in human sensitivity with regard to PTS. The hazardous nature of a noisy environment is therefore described in terms of "damage risk." It is now accepted that this risk is not negligible at noise exposure levels of more than 85 dBA \(L_{eq}\)\(^{(1)}\).

The damage risk contours published by Chaba\(^{(26)}\) were recognized as a milestone in predicting the hazard of noise to hearing. Melnick\(^{(25)}\) reviewed information on the relation of human TTS to properties of steady-state and intermittent noise. These data indicated that 2 of 3 major postulates on which the original contours were based were not valid.

1. Recovery from TTS is not independent of
the conditions that produced the TTS as was formerly assumed.

(2) The assumption that all exposures that produce equal TTS 2 min after the end of the exposure (TTS₂) are equally hazardous is not substantiated.

(3) The postulate that noise-induced PTS produced by 10 years of daily exposure is approximately equal to the TTS₂ produced by the same noise after 8-hr exposure can be valid.

On the other hand, Clark53) reviewed the major findings from laboratory studies on experimentally induced hearing loss, both temporary and permanent, resulting from exposure to noise in animal subjects which had been published since the report by Kryter et al.76) The reviewed data supported the following general statements:

(1) The chinchilla is the most widely used and most suitable animal model for studies of noise-induced hearing loss;

(2) With continuous exposure to moderate-level noise, thresholds reach asymptotic levels within 18–24 hr;

(3) PTS, however, depends on the level, frequency and the duration of exposure;

(4) Below a "critical level" of about 115 dB, PTS and cell loss are generally related to the total energy in continuous exposures;

(5) Periodic rest periods inserted in an exposure schedule are protective and result in less hearing loss and cochlear damage than equal energy continuous exposures;

(6) Under some schedules of periodic exposure, threshold shifts increase over the first few days of exposure, then recover as much as 30 dB as the exposure continues.

7. Conclusion—Diagnosis and prevention of noise-induced hearing loss

The foregoing is an introduction to recent studies on the background, pathology and pathophysiology of noise-induced hearing loss. Many points are, however, still indistinct. Noise-induced hearing loss often observed in the workplace is irreversible hearing impairment. Even if its pathology is elucidated, it is impossible to devise drastic treatment. The studies introduced in this report are therefore very interesting as physiological studies on hearing. The results provide an industrial physician with a theoretical attitude of mind to evaluate noise, but are rarely applied directly to the workplace.

The revised Industrial Safety and Health Ordinance has recently induced an important new era into the workplace in Japan. The Japanese Ministry of Labour revised a part of the Industrial Safety and Health Ordinance (Article 590, Article 591) in 1992 to promote the prevention of noise hazards. It also adopted Guidelines for the Prevention of Noise Hazards in October, 199281). The guidelines systematically describe measures based on the Industrial Safety and Health Ordinance and steps for preventing noise hazards which an enterprises are asked to provide independently. In the guidelines, the control level of noise has been established, based on the equivalent sound level described in ISO/DIS 961225).

The guidelines have newly set the management classification of the working environment to prevent hearing impairment, and they attach importance to the hearing level at 4 kHz to detect noise-induced hearing loss earlier. They have greatly improved health management for hearing conservation. We have pointed out for a long time that we should consider the hearing level at frequencies higher than 2–3 kHz for preventing noise-induced hearing loss, and Shida and Yoshida75) also pointed out the high frequency dip. The hearing level at higher frequencies should be discussed further.

Evaluating the degree of hindrance in daily life due to noise-induced hearing loss is often discussed, related to compensation for industrial accidents and occupational diseases. In particular, the relationship between the average hearing level and speech discrimination needs to be discussed further82, 83). Enterprises need to predict PTS due to exposure to noise in workers with preexisting sensorineural hearing loss.

There are some studies on the prevention of noise-induced hearing loss. Some of them indicate the prospects for future strategies in predicting susceptibility to noise-induced hearing loss85). Some show that oral magnesium supplementation is effective as a prophylaxis for noise-induced hearing loss86, 87).

Finally, we want to show the another view: that of Shida7) discussing the problem of diagnosis and compensation for noise-induced hearing loss.

"The conception that all sensorineural hearing loss in workers exposed to noise is noise-induced hearing loss, is a misconception of industrial physicians who are lacking in knowledge of the recognition of hearing. The otologist who has sufficient knowledge of the physiology and pathology of the ear should positively deal with the problem of noise-induced hearing loss, make a diagnosis and agree about compensation."

It is not an important matter who deals with the problem of noise-induced hearing loss. Industrial physician or otologists who have sufficient knowledge of noise-induced hearing loss should manages it.
References


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