Noise-induced hearing loss among workers in textile factory

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Introduction
Noise-induced hearing loss (NIHL) is one of the most common chronic health problems, which produces gradual progressive impairment and disturbs the patient's quality of life. Industries in general and especially textile ones produce noise levels that, if intensified, can cause damage to worker's hearing. Hence, premature hearing loss is a well-known outcome of noise exposure at work in industrial workers.

Aims of the work
The aim of the study was to assess hearing threshold levels among exposed patients and to compare them with the nonexposed control group and to evaluate other variables such as outer hair cell and medial olivocochlear bundle function represented with transient evoked otoacoustic emissions (TEOAEs) testing with and without suppression and to find the relationship with duration of exposure if any.

Patients and methods
The study included 145 patients and same number of controls who were exposed to hazardous levels of noise for variable durations. All participants were subjected to audiological examination including basic audiological evaluation, otoacoustic emissions, and otoacoustic emissions with contralateral suppression (CAS).

Results
Of the 145 patients (290 ears), 214 ears showed sensorineural hearing loss (73.8%). Sensorineural hearing loss was mild in 63 (43.44%) ears, moderate in 82 (56.55%) ears, and severe in 69 (47.58%) ears. TEOAEs were found in those with mild hearing loss with significantly lower amplitudes. Intact suppression (CAS) was significantly lower for the study group than for the control group. However, there was no significant difference in level of suppression for different duration of exposure.

Conclusion
A high incidence for NIHL is present among workers in textile factories, which indicate the mandatory use of different protective measures. CAS can be used as a predictor for the susceptibility to NIHL.

Keywords:
Noise induced hearing loss, textile factories, TEOAEs, olivocochlear bundle

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Introduction

About 30 million workers are exposed to hazardous noise, with an additional nine million exposed to solvents and metals that put them at risk for hearing loss (HL). Occupational HL is one of the most common occupational diseases. In all, 49% of male miners have HL by the age of 50 years. By the age of 60 years, this number goes up to 70% [1].

This problem is faced by a large sector of the working force; worldwide, about 16% of the disabling HL in adults (over four millions) results from occupational noise [2].

HL due to chronic noise exposure or noise-induced hearing loss (NIHL) has been associated with industry for many years [3]. Most of the western countries have their own regulations and rules for the protection of workers in noise-producing factories [4].

The Occupational Safety and Health Administration (OSHA) describes standards for occupational noise exposure in articles 1910.95 and 1926.52 [5]. OSHA states that an employer must implement hearing conservation programs for employees, if the noise level of the workplace is equal to or above 85 dB(A) for an averaged 8-h time period [6].

OSHA also states that exposure to impulsive or impact noise should not exceed 140 dB sound pressure level (SPL) peak. The United States Department of Defense (DoD) instruction 605512 has some differences from the OSHA 1910.95 standard – for example, OSHA 1910.95 uses a 5 dB SPL exchange rate and DoD instruction 605512 uses a 3 dB SPL exchange rate.

Hearing conservation programs in the workplace and in the general population seek to increase compliance and effectiveness of the hearing protection protocols through audiometric screening tests and education on the dangers of noise exposure.

Employees are required to wear hearing protection when it is identified that their 8-h time weighted average is above the exposure action value of 90 dB SPL. If subsequent monitoring shows that 85 dB SPL is not surpassed for an 8-h time weighted average, the employee is no longer required to wear hearing protection [7].

Occupational health diseases generally are difficult to diagnose early because they often have a long latency period [8]. Hence, it is important to monitor worker's hearing for early diagnosing and preventing NIHL through a program of hearing conservation [9].

This study was designed to assess hearing threshold levels among exposed patients and to compare them with the nonexposed control group and to evaluate other variables such as outer hair cell and medial olivocochlear bundle function in this vulnerable group.

Patients and methods

This was a case–control study conducted among workers of Zefta textile factory (the exposed group) and among normal persons of the official clerks of Zagazig University of Medicine and relatives of the patients after their consent (the control group). Evaluation was performed in Audiology Units, Otolaryngology Department, Zagazig University Hospitals, Egypt. Occupational HL among textile workers is 30% according to Farouk *et al*. (2000) [10]; hence, the sample size required was calculated using Epi-Info program (Epi Info™ Help Desk Centers for Disease Control and Prevention, Atlanta, USA) to be 145 workers.

All 145 patients were men with age range 35–50 years (43.4 ± 10.1) to avoid HL due to aging. Interviewing questionnaire for included patients was developed by the researchers acquiring about sociodemographic characteristics, audiological symptoms, and brief occupational history.

Setting for interviewing patients and detecting level of noise was at Zefta textile factory within both textile and weaving sectors for the exposed group and at administration building of Zagazig Faculty of Medicine for the nonexposed group. Audiological examination was performed at the Audiology Unit, ENT Department, Zagazig University.

Noise level detection was performed using EXTECH noise level meter model (Omni Controls, Tampa, Florida), 407764, with available range of 30–130 dB, A and C frequency weighting. Display is with dB(A).

All patients were clinically examined (otoscopical examination). Those who had suggestive history of HL, such as diabetes, hypertension, and family history of HL or head trauma, were excluded from the study. Any pathology of middle or external ear, such as impacted wax or otitis media, was detected and treated first to avoid fallacies in audiological tests.

Tympanometry was performed in all patients using Tympanometer Ampalid 724 (Amplifon, Milano, Italy); only patients with normal middle ear pressure were involved in this study.

Audiometric assessment by standard pure-tone audiometry, using Audiometer Orbiter 922 (GN Otometrics, Taastrup, Denmark), was performed by the audiology consultants; bone and air conduction for both ears were individually performed from 250 up to 8000 Hz. HL was categorized according to Clark [11] into the following:

(1) *Mild HL*: hearing threshold between 26 and 40 dB HL.
(2) *Moderate HL*: hearing threshold between 41 and 55 dB HL.
(3) *Moderately severe HL*: hearing threshold between 56 and 70 dB HL.
(4) *Severe HL*: hearing threshold between 71 and 90 dB HL.
(5) *Profound HL*: hearing threshold more than +90 dB HL.

Otoacoustic emissions testing was performed using ILO version 6 (Otodynamics Ltd, Hertfordshire, UK). Contralateral suppression (CAS) of otoacoustic emissions was performed using Amplaid 309 audiometer (Amplifon), delivering white noise of 70 dB SPL.

Statistical analysis

Data were analyzed using SPSS version 17 (SPSS Inc., Chicago, Illinois, USA). Comparison between the study and the control group was performed using the *t*-test for two independent means. Comparison among the subgroups of the study group was carried out using one-way analysis of variance test, and comparison for nonparametric data was carried out using the Fisher exact test.

Results

Noise levels in the factory departments using the portable noise level meter taken from different spots with different number of machines in work at the same time revealed that textile (new machines) noise level equals 95–104 dB(A), textile sections (old machines)

noise level equals 98–109 dB(A), and in the weaving sections bed sheets and coverings using manual weaving machines noise level equals 80–84 dB(A).

Among 145 patients (290 ears), 214 ears showed sensorineural hearing loss (SNHL) (73.8%). SNHL was found to be nearly equal bilaterally ranging from mild to severe and mostly involving the high-frequency region in most of the affected patients. Involvement of other frequency regions was associated with longer time of exposure (Table 1).

Reviewing the degrees of severity of SNHL, SNHL was mild in 63 (43.44%) ears, moderate in 82 (56.55%) ears, and severe in 69 (47.58%) ears (Fig. 1). Pure-tone audiometry thresholds revealed statistically significant difference between the study and control groups in the high-frequency region (Table 2). Similarly, statistically significant difference was found between both the study and control groups with respect to speech discrimination scores (Table 3).

To study the factor of duration of exposure, the study group was divided into three subgroups: those exposed for less than 1 year (subgroup A) included 32 cases, those exposed for 1–5 years (subgroup B) included 60 cases, and those exposed for 5–10 years (subgroup C) included 53 cases (Figs. 2–4).

One-way analysis of variance test was performed to study the effect of duration of HL on the hearing threshold levels across different frequencies. A statistically significant difference was found among the three subgroups frequencies starting from 1 kHz and above (Table 4).

Once it is determined that differences exist among the means, post-hoc range tests and pairwise multiple comparisons can determine which means differ. Range tests can identify homogeneous subsets of means that are not different from each other. Pairwise multiple comparisons can test the difference between each pair of means and yield a matrix where asterisks indicate significantly different group means at an α-level of 0.05 (Table 5). It showed significant difference between each pair of means except between groups B and C at 1 kHz.

Regarding prevalence, 53.125% had HL in subgroup A and 70% in group B, whereas 90.567% had HL in group C (Table 6). This difference was found to be statistically significant as proved by the Fisher exact test.

Table 1 Age distribution for both the control and study groups (*n* = 145)

Groups	Age (years)			
	X	SD	*t*-value	*P*
Control	41.6	9.5	−1.563	0.119
Study group	43.4	10.1		

Table 2 Showing pure-tone audiometry thresholds in the patients and control groups across frequencies of 500–8000 Hz

Frequencies (Hz)	Control group	Study group	*t*-value	*P*
250	22.61 ± 6.31	24.71 ± 6.8	−1.4099	0.154
500	20.56 ± 5.69	23.61 ± 5.3	−1.626	0.105
1000	23.67 ± 7.69	25.35 ± 9.5	−1.655	0.099
2000	19.33 ± 5.34	49.2 ± 6.7	−41.98	<0.01*
4000	23.83 ± 4.45	71.42 ± 8.6	−59.18	<0.01*
8000	20.83 ± 5.1	80.7 ± 5.7	−94.25	<0.01*

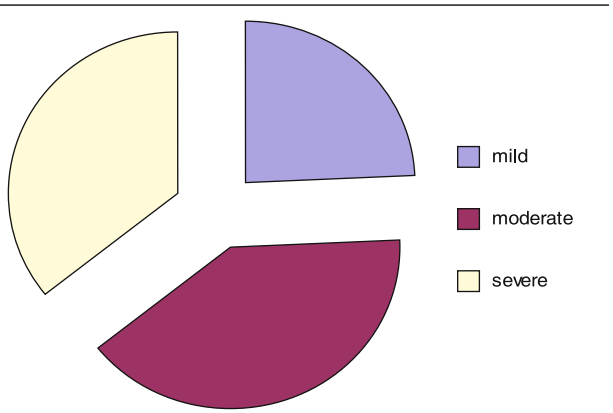
*Significant.

Table 3 SRTs and WDSs

Speech audiometry results/group	Control		Study group			
	X	SD	*X*	SD	*t*-value	*P*
SRT	20.41	2.3	66.32	20.3	−27.06	<0.001*
WDS	87.72	4.51	72.65	18.85	9.363	<0.001*

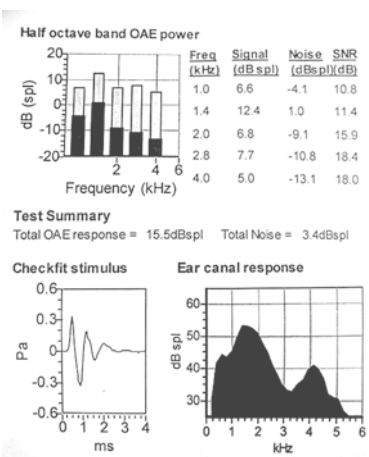
SRT, speech reception threshold; WDS, word discrimination score; *Significant.

Figure 1



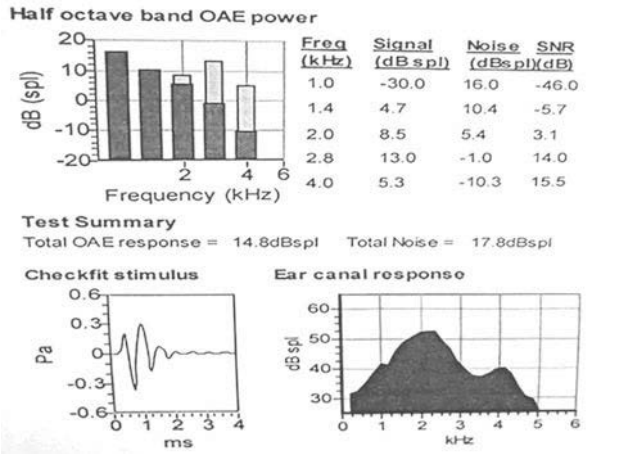
Degrees of severity of hearing loss.

Figure 2



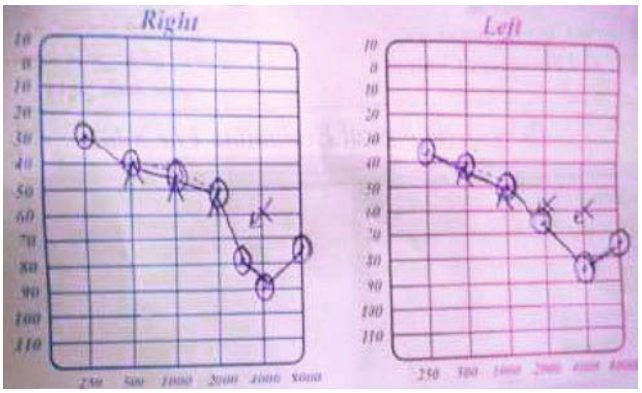
TEOAEs testing for a patient with mild high-frequency hearing loss showing preserved OAEs.

Figure 3



TEOAEs testing for the same case after contralateral suppression.

Figure 4



Audiogram of a case showing bilateral mild to severe sloping sensorineural hearing loss.

Table 4 One-way ANOVA to compare the three subgroups regarding pure-tone audiometry thresholds

Frequencies	Subgroup A	Subgroup B	Subgroup C	*F*	*P*
250 Hz	20.32 ± 4.3	22.3 ± 4.2	22.2 ± 5.3	2.16	0.119
500 Hz	23.6 ± 5.2	24.8 ± 4.3	25.3 ± 6.3	1.04	0.357
1 kHz	24.6 ± 4.4	28.7 ± 3.2	30.6 ± 6.5	15.089	<0.001**
2 kHz	34.6 ± 5.6	43.7 ± 6.1	50.8 ± 4.9	84.76	<0.001**
4 kHz	50.6 ± 7.8	55.6 ± 8.6	64.2 ± 7.6	31.59	<0.001**
8 kHz	56.67 ± 10.2	67.32 ± 8.65	76.53 ± 6.78	56.46	<0.001**

ANOVA, analysis of variance; **Highly significant.

Table 5 Tukey's HSD test for post-ANOVA pairwise comparisons in one-way ANOVA result

	$HSD_{0.5}$	A/B (*P*)	A/C (*P*)	B/C (*P*)
1 kHz	2.42	<0.01	<0.01	NS
2 kHz	2.79	<0.01	<0.01	<0.01
4 kHz	4.04	<0.01	<0.01	<0.01
8 kHz	4.21	<0.01	<0.01	<0.01

ANOVA, analysis of variance; HSD, Honestly Significance Difference.

Table 6 Prevalence of HL in the three subgroups

HL	Subgroup A [*n* (%)]	Subgroup B [*n* (%)]	Subgroup C [*n* (%)]	Total
Number of cases positive for HL	17 (53.125)	42 (70)	48 (90.567)	107
Number of cases negative for HL	15 (46.875)	18 (30)	5 (9.433)	38
Total	32	60	53	145

HL, hearing loss; $P = 0.0003$.

Table 7 χ^2-test to assess the degrees of HL among subgroups of noise exposure

	Subgroup A	Subgroup B	Subgroup C	Total (ears)
Mild HL	22	29	12	63
Moderate HL	12	30	40	82
Severe HL	0	25	44	69
Total (ears)	34	84	96	214

$\chi^2 = 41.592$; *d.f.* = 4. HL, hearing loss; $P < 0.001$.

Comparing degree of HL among subgroups of noise exposure was found to be significantly different (Table 7).

TEOAE testing was performed for patients with mild HL (126 ears) and for the control group and revealed absent emissions in three patients in the study group and in 11 patients in the control group. Those with preserved TEOAEs had significantly lower amplitudes in all frequencies (Table 8).

CAS of otoacoustic emissions as a measure for intact medial olivocochlear bundle was performed. The incidence of intact suppression was significantly higher for the study group than for the control group (Table 9). However, there was no significant difference for different duration of exposure (Table 10).

Discussion

NIHL is a growing health issue, with costly treatment and lost quality of life [12]. Exposure to high levels of noise can damage the inner ear and cause SNHL. These losses may be temporary or permanent. Some studies using animal models have suggested that these temporary and permanent HLs may be a result of different mechanisms rather than different stages of HL [13].

Table 8 TEOAEs in the study and control groups across different frequencies

Frequencies (kHz)	Control group	Study group	*t*-value	*P*
1	6.23 ± 4.35	3.78 ± 5.23	3.171	0.002
1.4	8.61 ± 4.5	5.77 ± 4.24	3.645	0.0004
2	9.3 ± 3.21	5.23 ± 5.97	4.969	<0.001*
2.8	8.3 ± 5.3	4.79 ± 3.26	4.937	<0.001*
4	10.3 ± 3.64	6.67 ± 4.68	5.329	<0.001*
Overall response	13.62 ± 2.68	7.16 ± 3.7	12.17	<0.001*

*Significant.

Table 9 Fisher's exact test to assess the prevalence of intact suppression between the control and study groups

CAS	Control group	Study group	Total
Present	115	8	123
Absent	19	52	71
Total	134	60	194

CAS, contralateral suppression; $P < 0.0001$.

Table 10 Fisher's exact test to assess the prevalence of intact suppression among the three subgroups

HL	1 year	1–5 years	5–10 years	Total
Present	5	2	1	8
Absent	20	18	14	52
Total	25	20	15	60

HL, hearing loss, $P = 0.4457$;

In this study, 214 ears showed SNHL (73.8%). This is consistent with the typical clinical picture of chronic noise exposure, which is always bilateral. High-frequency losses rarely exceed 75 dB. Loss is always greater at the frequencies 3000–6000 Hz than at 500–2000 Hz. Loss is usually greatest at 4000 Hz, with the 4000-Hz notch often preserved even in advanced stages. In stable exposure conditions, losses at 3000, 4000, and 6000 Hz usually reach a maximum level in 10–15 years [14].

There was a statistically higher pure-tone audiometry thresholds found among the subgroups of noise exposure (Table 4). Similarly, there was a significant difference between each pair of means except between groups B and C at 1 kHz (Table 5).

This is supported by the principal characteristics of chronic, occupational NIHL as specified by the American College of Occupational Medicine Noise and Hearing Conservation Committee, which described it as bilateral and symmetric SNHL, rarely produces a profound HL, does not progress once noise exposure is stopped, rate of HL decreases as the threshold increases, the 4 kHz frequency is the most severely affected, and the higher frequencies (3–6 kHz) are more affected than the lower frequencies (500 Hz to 2 kHz). Finally, maximum losses typically occur after 10–15 years of chronic exposure [15].

Jawed *et al.* (2010) [15] found a positive correlation of hearing impairment with the duration of job when analyzed by linear regression analysis and correlation coefficient.

With respect to prevalence, the Fisher exact test was performed and revealed that the longer the duration of exposure by years, the higher the incidence of HL (Table 6). Similarly, permanent NIHL is related to the SPL and frequency distribution of the noise, the time pattern and duration of exposure, and individual susceptibility as stated by Johnson [16].

Speech discrimination scores were significantly higher for the study group than the control group (Table 3). This is consistent with elevated hearing thresholds. This is also consistent with the assumption that the most affected frequencies by noise are the speech frequencies. Speech affection is explained by the general agreement that the hearing level at 3000 Hz is related to the hearing and understanding of speech, particularly in the presence of noise. In 1978, in the summary of an investigation by Suter [17], it was reported that 'Correlation tests revealed that frequency combinations that included frequencies above 2000 Hz were significantly better predictors of speech discrimination scores than the combination of 500, 1000, and 2000 Hz'.

Otoacoustic emission results revealed significantly lower amplitudes at the high-frequency regions in the study group than the control group (Table 8).

As generally known, the outer hair cells in the inner ear are thought to be responsible for transient evoked otoacoustic emissions (EOAE) generation and they are one of the first structures damaged by noise [18].

Healthy ears have emissions [19]; however, noise-damaged ears have fewer, smaller, or no emissions [20]. Thus, it seems plausible that, by monitoring EOAEs, we can indirectly monitor the health and status of the inner ear. Furthermore, in laboratory studies, temporary threshold shift (TTSs) and emission shifts after exposure to 105 dB SPL noise are negatively correlated (i.e. the increase in hearing level is correlated with a decrease in emission level) and follow the same recovery [21].

In contrast, in field settings, a number of studies have also shown that emissions may undergo temporary changes after exposure to noise. However, changes in emissions are not necessarily associated with TTSs. In addition, in some cases, we have found that emissions do not shift with hearing decrements, and in other cases the emissions do not recover with hearing

improvements [22]. Finally, they stated that they have noticed that, in people who are regularly exposed to hazardous levels of noise, their emissions may shift before their hearing shifts.

Finally, CAS of otoacoustic emission is a measure of state of olivocochlear bundle integrity. A statistically significant difference was found between the number of patients with intact suppression and the control group (Table 9). This reflects defective function of olivocochlear bundle in the noise-exposed group. When comparing the three subgroups of duration of noise exposure, there was a nonstatistically significant difference (Table 10). This reflects the absence of duration of noise exposure as a factor affecting olivocochlear bundle function among noise exposure patients, which may be a clue that defective olivocochlear bundle function is a cause for NIHL, not a result.

Other findings were stated by Mariola and Kowalska [23] who found a significant decrease of OAE in response to contralateral noise stimulation for the level of 70 dB SPL for a group of metal factory workers as compared with healthy nonexposed control group. However, efferent suppression was weaker for the metal-exposed group compared with the other group. Finally, they concluded that OAEs, particularly distortion produce otoacoustic emissions and otoacoustic emissions (DPOAEs) CAS, could be a promising method for early identification of auditory damage in workers at risk of developing industrial NIHL.

Conclusion

NIOSH recommends the use of quieter equipment, better work practices, and hearing protection devices and implementation of effective HL prevention programs to prevent NIHL in firefighters. In addition, audiological assessment including CAS should be performed pre-employment to find out vulnerable patients for NIHL.

Acknowledgements

Conflicts of interest

None declared.

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