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Association between occupational noise-induced hearing loss and genotoxicity among textile factory workers

Ahmed Mahmoud Zein-Elabedein[1], Hossam Sanyelbhaa Talaat[1], Sobhy Elsayed Hassab El-Nabi[2], Aya Sobhy Hassab El-Nabi[3*] and Asmaa Salah Moaty[1]

Abstract

Background/aim Hearing loss caused by exposure to noise is still among the most prevalent health risks for industrial workers. This study aims to evaluate the relationship between Shebien El-kom textile factory workers' occupational noise exposure, genotoxicity, and noise-induced hearing loss.

Methods This cross-sectional case–control study was performed in a textile industry in Shebin Elkom, Egypt. The participants of this work were 36 exposed male workers from the spinning section of the textile factory and 36 subjects as the control male group from administrative staff in the same factory, in the age range of 25–45 years. A pure-tone audiometer and portable sound level meter were utilized for the measurement of hearing threshold and noise level, respectively. Genotoxicity was assessed using the Comet assay technique.

Results There was no significant difference between both groups regarding age and the mean duration of work was 18.94 ± 4.88 for exposed workers. The average level of noise was 95–105 dB (A). The exposed workers' mean hearing thresholds for the left and right ears at frequencies between 2000 and 8000 Hz were substantially greater than those of the control group ($P < 0.05$). In the exposed workers, there was not a marked variation between the hearing thresholds of the left and right ears ($p > 0.05$). The exposed workers' percentage of DNA damage was substantially greater than that of the control group ($p < 0.001$). Among exposed workers, a positive correlation between DNA damage, the degree of hearing loss, and the duration of time exposed to noise was demonstrated.

Conclusion The majority of exposed workers suffered from occupational noise-induced hearing loss. A positive correlation was found between the percentage of DNA damage, duration of exposure to noise, and hearing threshold in exposed workers.

Keywords Noise exposure, Noise-induced hearing loss, Genotoxicity, Comet assay, Oxidative stress, DNA damage

Background

As stated by the Occupational Safety and Health Administration (OSHA) [1], excessive noise exposure over the allowed limit (90 dBA) is considered a main environmental health issue [2]. It is among the most prevalent work-associated injuries worldwide [3], resulting in numerous auditory effects [4] including temporary noise-induced threshold shift, permanent noise-induced threshold shift, acoustic trauma, and tinnitus [1]. Also, non-auditory health problems [4] result from noise exposure including

*Correspondence:
Aya Sobhy Hassab El-Nabi
ayasobhy9876@gmail.com
[1] Department of ENT, Audiovestibular Medicine Unit, Faculty of Medicine, Menoufia University, Shibin El Kom, Egypt
[2] Department of Zoology, Faculty of Science, Menoufia University, Shibin El Kom, Egypt
[3] Department of ENT, Audiovestibular Medicine Unit, Shebien El-Kom Teaching Hospital, Shibin El Kom, Egypt

cardiovascular disorders, disturbed sleep, and disturbed perception [5, 6]. It has been noted that occupational noise-induced hearing loss (ONIHL) was 16% in adults with hearing loss and also estimated that the disease burden was 21% in developing countries and 7% in developed countries [7].

Metabolic exhaustion, oxidative stress, direct mechanical trauma, ischemia, ionic imbalance in the fluids of the inner ear [8], DNA damage, further activating the FAS gene [9], intracellular calcium overload, and ATP depletion [10–12] were all considered factors leading to NIHL. Oxidative stress leads to damage to essential components in the body, including cell membranes and DNA [13]. Genotoxicity was described as the destructive effects, affecting the integrity of the DNA [14]. Increased genotoxicity is a marker of increased oxidative stress in the body [13].

Several animal field studies recorded the impact of noise exposure on oxidative stress and DNA [15]. In newborn rats, Ceylan et al. reported significantly higher damage of DNA among the group exposed to noise in comparison to the control group [16]. The impact of noise exposure on oxidative stress and DNA has been reported, as oxidative damage of DNA results from chronic noise exposure [17]. According to Hosseinabadi et al., occupational exposure to noise can cause damage to DNA in peripheral mononuclear blood cells [18]. Promoting a healthy workplace for millions of workers requires understanding and recognizing the health implications of noise exposure. Therefore, this study aims to evaluate the relationship between occupational noise exposure, genotoxicity, and NIHL in workers at the Shebien El-kom textile factory.

Methods

Subjects

This cross-sectional, case–control study was conducted at the audiology unit, Faculty of Medicine, Menoufia University, Egypt. The exposed group comprised 36 male workers in the age range from 25 to 45 years, recruited from Shebien El-Kom Textile Factory, Menoufia, Egypt, with at least 5 years of exposure to work-related noise during the period between February 2022 and April 2023. Workers were selected from the spinning stage of the textile factory, where the maximum level of noise was detected by a sound level meter. The workers who were exposed to noise in complete 8-h shift were included in the study subjects. Workers with abnormal middle ear function, tobacco smokers, and subjects with chronic diseases like hypertension, diabetes mellitus, and autoimmune disorders were excluded. The control group recruited from the administrative staff of the same factory comprised 36 males, between the ages of 25 and 45, with no history of noise exposure or otological symptoms as well as the same exclusion criteria of exposed workers.

Material and methods

The level of noise at the workplace was performed utilizing a portable sound level meter standard (Testo 815 sound level meter; Quest technologies) at all the work sections; the maximum level of noise was detected in the spinning section of textile factory. The workers in this section were recruited for the current study. Multiple noise level measurements at different times of working shifts were taken in our investigation and the average measurement was reported.

All subjects in the study (cases and control) were subjected to the following: full history taking (hearing loss, exposure data as duration of noise exposure, use of hearing protection devices), otoscopic examination, basic audiological evaluation, genotoxicity testing utilizing the comet assay for evaluation of damage of DNA.

Audiological evaluation: pure tone audiometry (model, Madsen; Orbitter 922), test was done for a frequency range of 250 to 8000 Hz. The test was performed in sound treated room. Hearing deterioration was assessed utilizing the pure tone average frequencies of 2000, 3000, 4000, 6000, and 8000 kHz. According to the American Speech-Language-Hearing Association (ASHA), hearing impairment was classified [19]; normal hearing = − 10–15 dB hearing threshold level (HL), slight hearing loss = 16–25dBHL, mild hearing loss = 26–40 dB HL, moderate hearing loss = 41–55 dB HL, moderately severe hearing loss = 56–70 dB HL, sever hearing loss = 71–90 dBHL and profound hearing loss = + 91 dBHL. Tympanometry (model, GSI 38) at pressure range from + 200 to − 400 mm H_2O.

Comet assay technique was applied to identify damage to DNA. This technique was conducted based on Singh et al. [20]. An amount of 2 ml of whole peripheral venous blood was incubated with erythrocyte lysing buffer (ELB), then centrifuged for 5 min. The leucocyte pellet was rinsed twice with RPMI 1640 medium which is supplemented with 10% fetal bovine serum. The leucocyte pellets are then suspended in 100 µl 0.7% low melting agarose (BRL) and added to slides which are coated by a layer of 100 µl of 0.5% ultrapure agarose. The slides then were suspended in a jar containing cold lysing solution (100-mM Tris, pH10, 2.5-M NaCl, 10%DMSOand 1% Triton X-100). Incubation of slides for 20 min in fresh alkaline buffer (1-mM EDTA, pH 13, and 300-mM NaOH) in the electrophoresis box; then, an electric current of 25 V (0.86 V/cm) and 300 mA was used for 20 min to unwind DNA and express alkali-labile sites. After electrophoresis, to neutralize the excess alkali, the slides were kept in tris buffer (400 mM Tris, pH = 7.5).

Finally, to stain the slides, a 100-µl ethidium bromide was added to every slide, covered with a coverslip, and stored for 4 days at 4 °C in a moist environment.

According to Hassab El-Nabi, the first scoring technique was applied [21]. A fluorescence microscope with 510-nm excitation and 590-nm barrier filters was used for the examination. DNA damage percentage is calculated as the proportion of damaged DNA spots among 500 randomly chosen locations per sample. Another scoring method was applied using comet score tail moment image analysis software by analysis of fifty comet nuclei. The following measurements were utilized to assess the degree of DNA damage: tail moment is estimated as follows: (tail moment = tail length × percent of DNA in tail/100). Tail length is applied to assess the extent of damage of DNA away from the nucleus and is expressed in Micro m; percentage of DNA in tail: all tail pixels intensity divided by the total intensity of all pixels in the comet. As important indications of DNA damage, the comet metrics tail length, DNA% in tail, and tail moment were altered.

Statistical analysis

SPSS version 22 (Armonk, NY: IBM Corp, 2013) and an IBM personal computer were used to compile, organize, and statistically analyze the data. Percentage, standard deviation (SD), mean, and range were utilized in descriptive statistics. Two qualitative variables were analyzed for association utilizing the chi-square test (χ^2). The Student *T*-test was employed to compare two groups that each had a quantitative variable.

The Mann–Whitney test was used to compare two groups with quantitative data that were not normally distributed. To compare several readings of normally distributed data within the same group, the paired *t* test was utilized. In order to compare more than two groups with quantitative parameters, the ANOVA test was applied. For the correlation between two continuously distributed normally distributed variables, Pearson correlation was utilized. If *P*-value *f* > 0.05, statistical significance was regarded.

Results

The current study was conducted on 36 exposed male workers and 36 control subjects. None of the exposed workers were using the hearing protection devices. The age and duration of work are displayed in Table 1 with no obvious variations between both groups considering age.

Noise measurements were taken at different times of working shifts at 7 a.m., 9 a.m., 11 a.m., 1 p.m., and 3 p.m., and the range of noise level at different sections was 95–105 dB for the spinning section, 90–95 dBA for the weaving section, 85–92 dBA for the wet processing, and 85–95 dBA for the fabrication section; the highest level of noise was reported in the spinning section.

Table 1 The mean age of the studied groups and duration of work of the exposed group

Variables	Controls No = 36	Exposed workers No = 36	*t*-test	*p*-value
Age (years):				
Mean ± SD	39.00 ± 3.36	39.19 ± 3.25	0.25	0.804
Range	34–45	34–45		
Duration of work (years):				
Mean ± SD		18.94 ± 4.88		
Range		12–30		

No Number, *SD* Standard deviation, *t-test* Student's *t* test, *p* value probability value

All exposed workers and the control group had normal tympanometry findings. There were no marked variations between both groups considering hearing thresholds of the left and right ears at frequencies 250, 500, and 1000 Hz with *p* value > 0.05. On the other hand, exposed workers had significantly higher mean values of hearing thresholds of the left and right ears at higher frequencies from 2000 to 8000 Hz, compared to the control group and *p* value < 0.05 (Figs. 1 and 2).

There were no marked variations in the thresholds of hearing of the left and the right ears at each frequency among exposed workers, *P* value > 0.05%, as shown in Fig. 3.

In comparison to the control group, exposed employees had a greater prevalence of hearing loss in both the left and right ear, which ranged from mild to moderately severe high-frequency sensory neural hearing loss (Table 2). In exposed workers, acoustic notch was found at 4 kHz in 47.22% of right ears, 50% of left ears, and 33.33% bilaterally.

In the exposed workers, there were significantly higher mean values of DNA damage parameters as shown in DNA damage spot, DNA damage percent, and tail moment in the exposed workers than in controls, as illustrated in Table 3. The significant increase in DNA damage parameters was linked to elevation in the severity of hearing loss, *p* value < 0.05, as revealed in Table 4 and Fig. 4.

Figures 5 and 6 show a positive correlation between hearing threshold and both DNA damage percent and tail moment, respectively, in the exposed workers.

Figure 7 shows single-strand breaks of DNA assessed by comet assay among the studied groups, in which A represents the control group, B represents the exposed workers with mild sensory neural hearing loss, C represents the exposed workers with moderate sensory neural

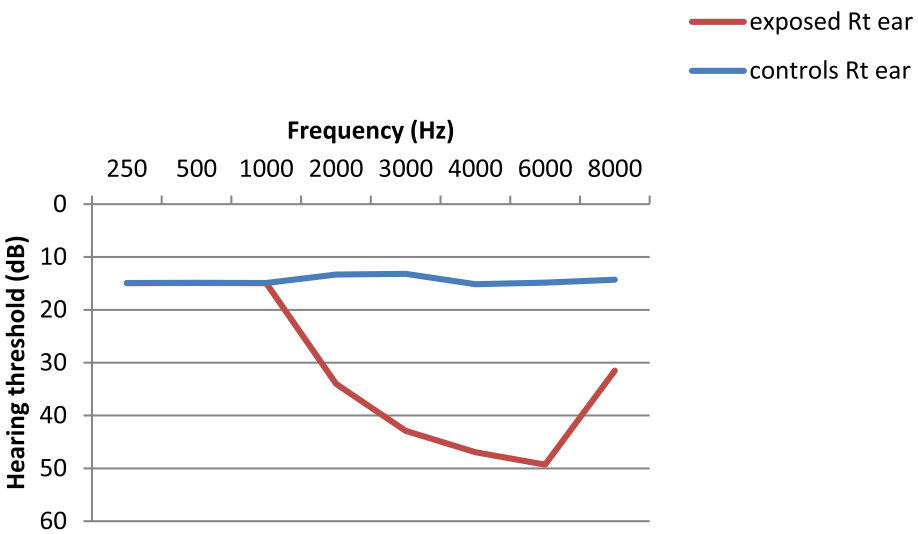

Fig. 1 Hearing threshold of the right ears at all frequencies in the studied groups

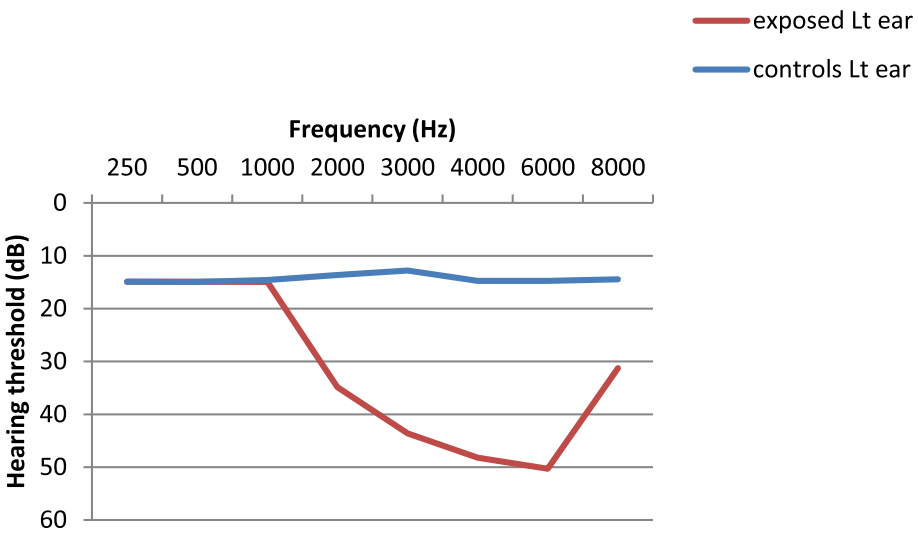

Fig. 2 Hearing threshold of the left ears at all frequencies in the studied groups

hearing loss, and D represents the exposed workers with moderate-severe sensory neural hearing loss.

Table 5 shows a significant positive association between age ($r=0.35$, p-value 0.034), duration of noise exposure ($r=0.38$, p-value $=0.022$), DNA damage % ($r=0.89$, p-value <0.001), and tail moment ($r=0.89$, p-value <0.001) and hearing threshold in the exposed workers.

Discussion

Exposure to hazardous noise levels above the permissible levels may result in auditory and non-auditory harmful impacts [18]. After presbycusis, NIHL is still the second and most common cause of acquired hearing loss in many nations [22]. Depending on the World Health Organization's 2017 estimate, 1.1 billion individuals between the ages of 12 and 35 and 360 million individuals globally suffer from loss of hearing caused by noise exposure [22].

Extreme noise exposure causes the cochlea to produce excessive quantities of reactive nitrogen species (RNS) and reactive oxygen species (ROS) [23], which can stimulate oxidative stress [24–27] and oxidative damage of DNA [23], both of which can harm sensory hair cells [12]. Therefore, the aim of the current work is to evaluate the relationship between occupational noise exposure,

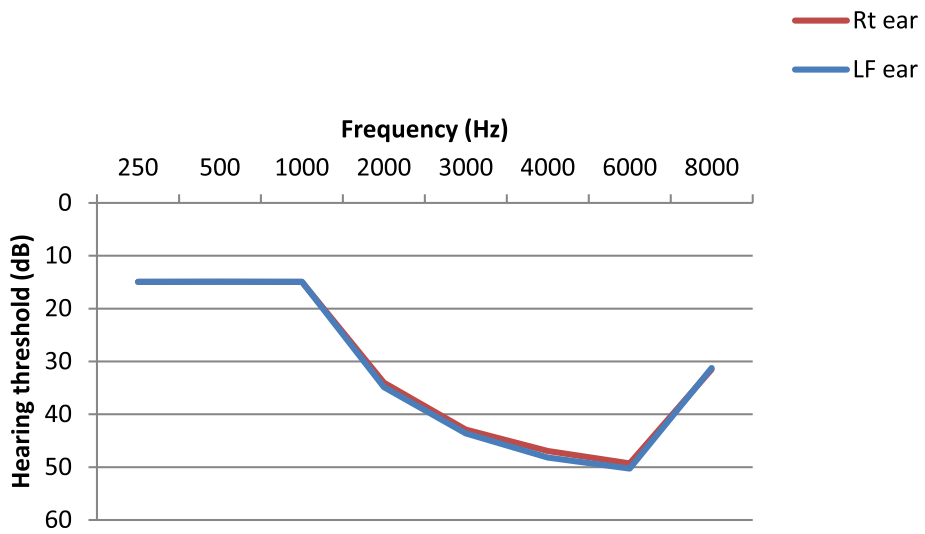

Fig. 3 Hearing threshold of the right and left ears at all frequencies in the exposed workers

Table 2 Comparison between the studied group regarding the prevalence and degree of hearing loss of the right and left ears

	Right ears		Left ears	
	Control group	**Exposed workers**	**Control group**	**Exposed workers**
Hearing loss prevalence	6 (16.7)	33 (91.7)	6 (16.7)	33 (91.7)
	$\chi^2 = 40.78$, p-value < 0.001		$\chi^2 = 40.78$, p-value < 0.001	
Degree of hearing loss				
Slight	4 (11.1)	0	4 (11.1)	0
Mild	2 (5.6)	17 (47.2)	2 (5.6)	13 (36.1)
Moderate	0	11 (30.6)	0	15 (41.7)
Moderate sever	0	5 (13.9)	0	5 (13.9)
	$\chi^2 = 25.25$, p-value < 0.001		$\chi^2 = 25.68$, p-value < 0.001	

No Number, % percentage, χ^2 chi-square, *p value* probability value

Table 3 Comparison between the studied groups regarding comet assay indices of DNA damage parameters

Comet assay indices	**Controls (No = 36) Mean ± SD**		**Exposed workers (No = 36) Mean ± SD**	***t*-test**	***p*-value**
Microscopic analysis					
Normal DNA spot	467.25 ± 2.71	412.25 ± 34.41		9.56	< 0.001
Total DNA damage spot	32.75 ± 2.71		90.53 ± 25.97	13.28	**< 0.001**
DNA damage %	6.55 ± 0.54		18.15 ± 5.15	13.44	**< 0.001**
Electronic analysis					
Tail moment	0.24 ± 0.05		4.88 ± 3.39	8.21	**< 0.001**

No Number, % percentage, *SD* Standard deviation, *t*-test Student's *t* tes, *p* value probability value

genotoxicity, and NIHL among Shebien El-kom textile factory workers.

The current study revealed no statistically significant difference between the exposed workers and the control group concerning age. All the exposed workers were taken from the spinning section, where the highest level of noise was detected by SLM. Noise level at the spinning section ranged between 95 and 105 dBA,

Table 4 Relationship between degrees of hearing loss with DNA damage parameters in the exposed group

DNA damage parameters	Degree of hearing loss in the exposed workers (No = 36)				ANOVA test	*p*-value
	Normal (No = 3) Mean ± SD	Mild (No = 13) Mean ± SD	Moderate (No = 15) Mean ± S D	Moderately severe (No = 5) Mean ± SD		
Total DNA damage spot	46.33 ± 4.04	71.62 ± 10.94	103.07 ± 9.18	128.60 ± 14.52	61.41	< 0.001 P1 = 0.001 P2 < 0.001 P3 < 0.001 P4 < 0.001 P5 < 0.001 P6 < 0.001
DNA damage %	9.27 ± 0.81	14.46 ± 2.17	20.60 ± 1.83	25.72 ± 2.90	61.03	< 0.001 P1 = 0.001 P2 < 0.001 P3 < 0.001 P4 < 0.001 P5 < 0.001 P6 < 0.001
Tail moment	0.47 ± 0.13	2.57 ± 1.21	5.67 ± 1.84	11.17 ± 0.89	52.27	< 0.001 P1 = 0.031 P2 < 0.001 P3 < 0.001 P4 < 0.001 P5 < 0.001 P6 < 0.001

No Number, *%* Percentage, *ANOVA test* Analysis of variance test, *p-value* probability value for post hoc multiple comparisons, *P1* Normal vs mild degree, *P2* Normal vs moderate degree, *P3* Normal vs moderately severe degree, *P4* Mild vs moderate degree, *P5* Mild vs moderately severe degree, *P6* Moderate vs moderately severe degree

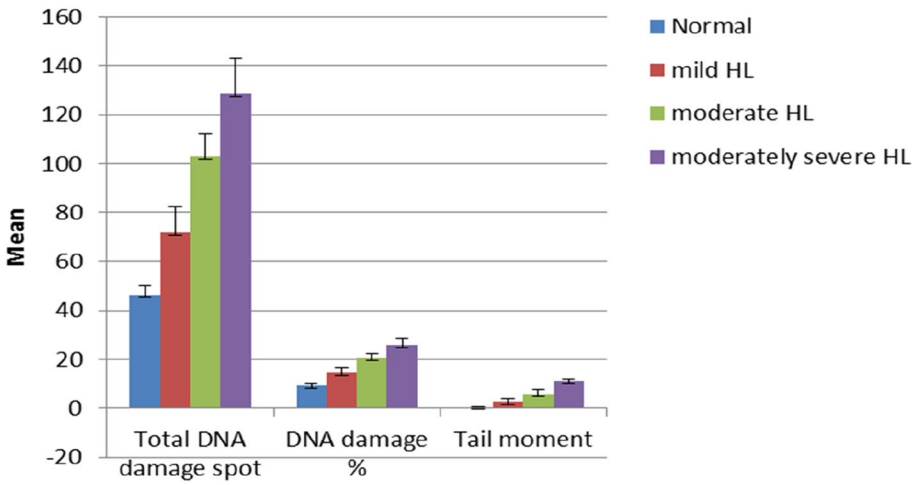

Fig. 4 Relationship between degrees of hearing loss with DNA damage parameters in the exposed workers

with a mean of 100 dB (A), exceeding the level recommended by Egyptian Environmental Law No.4 (1994) (90 dB), as well as exceeding the highest permissible level of occupational noise recommended by the international standards organization which is 85–90 dBA for 8 h/day [28]. These findings were in agreement with a study conducted in the textile industry that reported the highest level of noise in the spinning and weaving sectors ranging from 65 to 103 dB [29].

The finding of the present study reported that the mean duration of work was 18.94 ± 4.88 years. None of the exposed workers used protective devices in work shifts which may be interpreted by the lack of

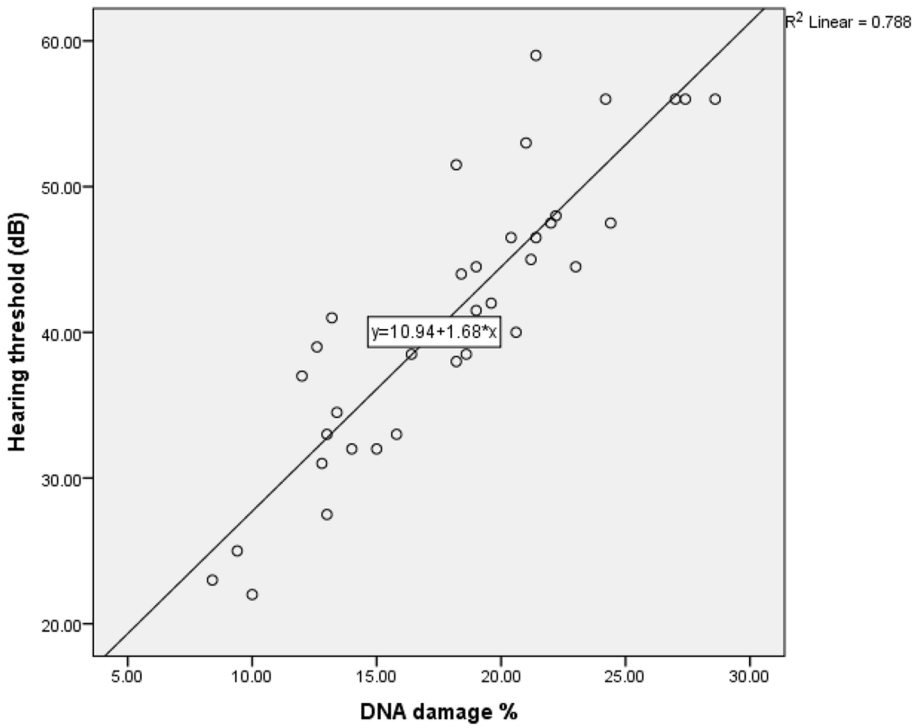

Fig. 5 Correlation between hearing threshold and DNA damage percent in the exposed workers

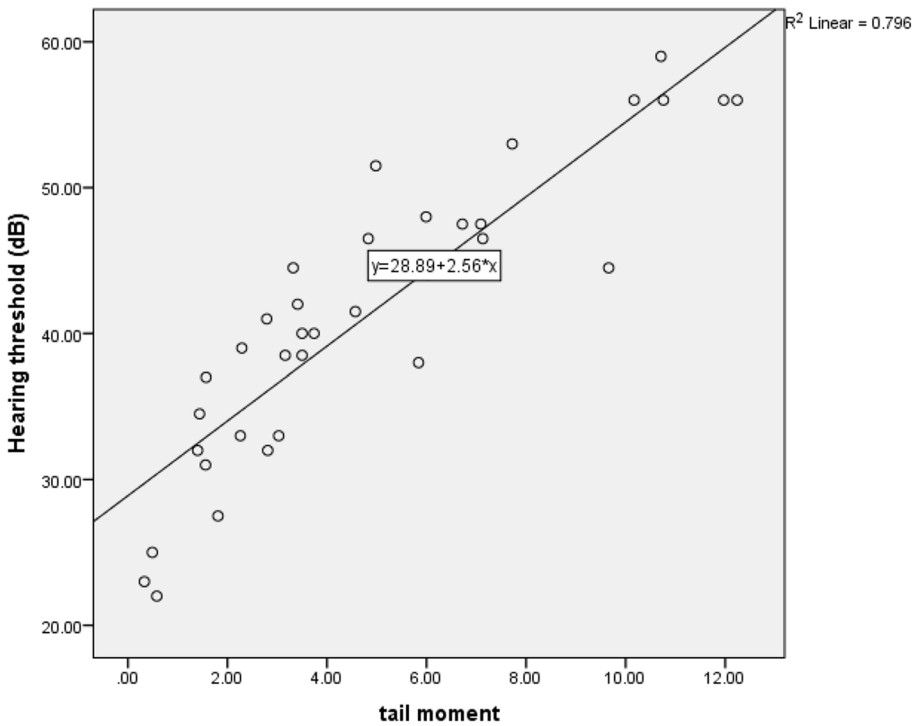

Fig. 6 Correlation between hearing threshold and tail moment in the exposed workers

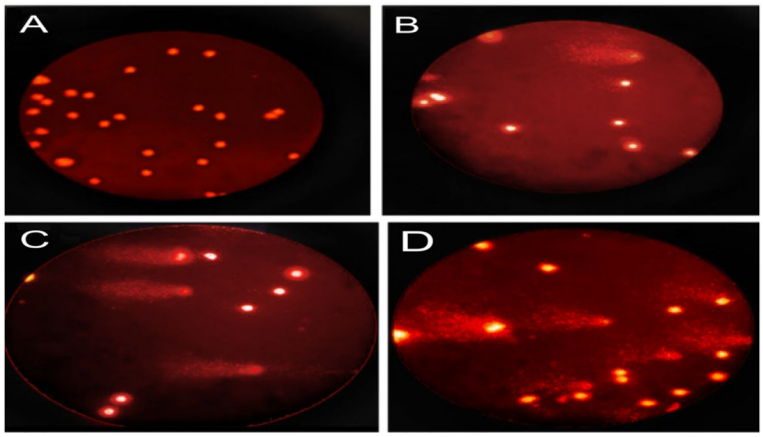

Fig. 7 Single-strand breaks of DNA illustrated by comet assay comet in the studied groups. **A** Control group. **B** Exposed workers with mild sensory neural hearing loss. **C** Exposed workers with moderate sensory neural hearing loss. **D** Exposed workers with moderate-severe sensory neural hearing loss

Table 5 Correlation between hearing threshold with age, work duration, and comet assay indices of DNA damage in exposed workers

Variables	Hearing threshold in exposed workers (No = 36)	
	r	*p*-value
Age (years):	0.35	**0.034**
Duration of work (years)	0.38	**0.022**
DNA damage %	0.89	**< 0.001**
Tail moment	0.89	**< 0.001**

No Number, *r* Spearman's correlation coefficient, *p-value* Probability value, *%* Percentage

awareness of the noise hazards and the importance of the protective devices' use.

In the current research, there were no marked variations between either group as regards thresholds of hearing of the left and right ears at frequencies 250, 500, and 1000 Hz. On the other hand, exposed workers had significantly higher mean values of hearing thresholds of the left and right ears at high frequencies from 2000 to 8000 Hz in comparison to controls. Mirza et al. stated that in NIHL, average hearing thresholds at lower frequencies of 500, 1000, and 2000 Hz are higher than average thresholds at 3000, 4000, and 6000 Hz [30].

Due to the fact that most noise exposures symmetrically impact both ears, ONIHL is typically bilateral symmetrical sensorineural hearing loss [30]. In the current work, there were no obvious variations between the thresholds of hearing of the left and the right ears at each frequency in the assessed groups, which were constant with other studies [31, 32].

The 4 kHz notch may be explained by the proven fact that the human ear is more sensitive to the frequencies 1–5 kHz and that the acoustic stapedial reflex minimizes loud sounds below 2 kHz; another possible explanation may be the resonant frequency of the external ear canal lies in the region of 2000–5000 Hz [33, 34].

In the current study, the frequency of loss of hearing in the left and right ears was higher among the exposed group as 91.7% had SNHL which ranged from mild to moderately severe high-frequency SNHL. These results were in congruence with work performed at a textile factory in Bhavnagar city of Gujarat, India, by Solanki et al. [35]. They reported that the prevalence of SNHL among workers was 84% with the majority of the exposed group suffering from mild to moderate degree of hearing loss [35]. Such differences in prevalence among studies may result from variation in the characteristics of the factors such as levels of noise exposure, noise sources, the duration of work shift, and other sources of noise exposure and socio-demographic factors [36].

In the current study, the acoustic notch at 4 kHz was noted in 47.22%, 50%, and 33.33% of the right, left, and both ears respectively. These results were similar to those of research conducted by Amer et al. in a weaving and spinning mill in Kafr Hakeem in the Giza governorate, Egypt, where they found that audiometric notches at 4 kHz were present in 62.3% of the right ear and 52% of the left ear, respectively (45.9%) [37].

One of the main causative factors in NIHL is oxidative stress which inflicts damage on sensory hair cells [12]. High ROS amounts [10, 11, 38], beyond the maximal cellular antioxidative ability [39], result in oxidative stress [24–27], which result in oxidative DNA damage [23] and then, caspase-mediated cellular apoptosis [40].

The current study used comet assay that indicates genotoxicity due to oxidative stress, to assess the effect of noise exposure on DNA in the peripheral mononuclear blood cells of exposed workers. The current findings revealed significantly higher mean values for total DNA damage spot, DNA damage percent, and tail moment in the exposed workers in comparison to the control group. In human studies investigating the impact of noise exposure on DNA, Hosseinabadi et al., who conducted a study on workers exposed to noise at a food factory in Shahroud, Iran, found the tail length and damage index were markedly greater in the exposed group [18]. In addition, another study conducted on textile factory workers by Havlioglu et al. found significant DNA damage among exposed workers [2].

Moreover, Nawaz and Hasnain had shown significantly higher levels of 8-oxodG in the serum of groups exposed to noise [41], as 8-oxodG is an oxidative DNA damage biomarker [15].

In the current research, there was a significant positive relationship between the duration of work and damage % of DNA in the exposed workers. The amount of time of worker exposure and noise intensity were the main risk variables for the severity of noise-induced hearing loss [41].

This research correlated the degree of hearing loss with DNA damage percent among exposed workers and a marked elevation in DNA damage parameters with the increase in severity of hearing loss was found. These results supported the hypothesis that noise exposure causes oxidative stress with DNA damage which is associated with the severity and degree of hearing loss.

To reduce levels of noise exposure, effective hearing conservation programs, improved workplace safety laws as recommended by National Institute for Occupational Safety & Health (NOISH), and education about occupational dangers should be implemented. For the early detection of any shift in hearing threshold, routine audiometry tests should be performed. Small sample size may represent a limitation of the current study. Further large-scale studies are recommended. Also, it is recommended to conduct more research on the genetics of noise-induced hearing loss.

Conclusion

The present work revealed high levels of noise especially in the spinning section of the textile industry; the majority of exposed workers had some degree of hearing loss which shows the typical pattern of noise-induced hearing loss with acoustic notch mainly at 4 kHz. There was an obvious association between DNA damage percent and occupational noise exposure, with elevation of damage of DNA as the period of exposure increased; in addition, there was a positive association between DNA damage percent and severity of hearing loss.

Abbreviations

ONIHL	Occupational noise-induced hearing loss
OSHA	Occupational Safety and Health Administration
DNA	Deoxyribonucleic acid
ATP	Adenosine triphosphate
NIHL	Noise-induced hearing loss
RNS	Reactive nitrogen species
ROS	Reactive oxygen species
Hz	Hertz
kHz	Kilohertz
ELB	Erythrocyte lysing buffer
FAS	Fas cell surface death receptor
SNHL	Sensory neural hearing loss
ASHA	American Speech-Language-Hearing Association

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Authors' contributions
HST and AMZE designed and performed the study; SEHE and ASHE collected and analyzed the data; ASM and AMZE contributed to the interpretation of data and interpretation of the analysis; ASM and ASHE wrote and edited the article, and HST, AMZE, and ASM revised it; all authors discussed the results and gave approval to the final manuscript.

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Availability of data and materials
The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
This study was carried out according to the guidelines and roles prescribed by the Research Ethics Committee, Menoufia University, Faculty of Medicine. Ethical approval was obtained from the ethical committee with approval number (n: 2/2022 ENT 41). The permission to collect the data was approved by authorities of Shebin Elkom textile industry, Menoufia, Egypt. Written consent was also obtained from all subjects participating in the study.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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