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Assessment of Noise-induced Temporary Threshold Shift in Dentistry Students Following Oral Magnesium Intake

Avaliação da mudança temporária de limiar auditivo induzida por ruído em estudantes de odontologia, após uso oral de magnésio

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Abstract
Long-term exposure to sounds ≥85 dB causes noise-induced hearing loss, which can be temporary or permanent, and it can be improved through the use of magnesium. This study aimed to determine whether noise in the dental laboratory negatively affects dental students' auditory system and whether magnesium ion supplementation prevents hearing loss. Sound pressure levels (SPLs) were monitored during the laboratory class and expressed in logarithmic function. Students attending regular dental practical classes were randomized into two groups: the study group (received 200 mg of magnesium 1 h before the laboratory class) and the control group (received 200 mg placebo at the same time). Distortion product otoacoustic emission (DPOAE) tests were performed before and after noise exposure during the class. The mean SPL equivalent was 75.3 dB; however, SPL peaked at 97.3 dB. DPOAE parameters did not change after approximately 3 h of exposure. There were no significant differences between the groups in all the frequencies studied for the amplitude (p=0.82) and S/N ratio (p=0.08) analyses. A single magnesium supplementation did not produce any side effects; however, no benefit was observed in hearing preservation. Occupational noise and magnesium supplementation did not alter DPOAE results.

Keywords: Magnesium, Dietary Supplement, Occupational Noise, Occupational Exposure

Resumo
A exposição prolongada a sons ≥85 dB causa perda auditiva induzida por ruído, que pode ser temporária ou permanente, e pode ser melhorada com o uso de magnésio. Este estudo teve como objetivo determinar se o ruído no laboratório de prótese dentária afeta negativamente o sistema auditivo de estudantes de odontologia e se a suplementação de íons de magnésio previne a perda auditiva. Os níveis de pressão sonora (NPS) foram monitorados durante a aula prática e expressos em função logarítmica. Os alunos que frequentavam as aulas práticas de odontologia regulares foram randomizados em dois grupos: o grupo estudo (recebeu 200 mg de magnésio 1 h antes da aula em laboratório) e o grupo controle (recebeu 200 mg placebo ao mesmo tempo). Testes de emissão otoacústica por produto de distorção (EOAPD) foram realizados antes e após a exposição ao ruído durante a aula. O equivalente médio NPS foi de 75,3 dB; no entanto, o NPS atingiu um pico de 97,3 dB. Os parâmetros das EOAPD não se alteraram após aproximadamente 3 h de exposição. Não houve diferenças significativas entre os grupos em todas as frequências estudadas para as análises de amplitude (p=0,82) e relação S/R (p=0,08). Uma única suplementação de magnésio não produziu efeitos colaterais; entretanto, nenhum benefício foi observado na preservação auditiva. O ruído ocupacional e a suplementação de magnésio não alteraram os resultados das EOAPD.

Palavras-chave: *Magnésio, Suplemento Alimentar, Ruído Ocupacional, Exposição Ocupacional*

1. Introduction

Noise-induced hearing loss (NIHL) is caused by long-term exposure to sounds of 85 dB or louder. In addition, there is evidence that prolonged exposure to moderate intensity noise (60–70 dB SPL) can result in changes throughout the cochlea and central auditory pathways.[1] It refers to pure, bilateral, symmetrical sensorineural hearing loss, predominantly observed at high frequencies. Moreover, it is the fourth cause of incapacity globally (16% of disabling hearing loss in adults).[2] Approximately 104 million people in the United States are exposed to harmful noise, and 1 out of 4 adults in the United States suffers from hearing loss caused by exposure to excessive noise levels.[3]

NIHL can be temporary or permanent, depending on the intensity and duration of exposure. The term “temporary threshold shift” (TTS) is used to describe objective changes in hearing acuity immediately after an episode of exposure to loud sounds, such as attending a concert, and that revert to preexposure levels after a few days to 2 weeks. Temporary threshold shifts are characterized subjectively by decreased hearing sensitivity, a feeling of fullness in the ears, tinnitus, and a perception that sounds are muffled. Prolonged or repeated exposure to noise can cause the death of sensory hair cells and permanent hearing loss referred to as a “permanent threshold shift.”[3]

The National Institute of Occupational Safety and Health[4] has incorporated work-related hearing damage in a list of 21 essential fields of study of this century, in addition to other risk areas, such as vibration and hand neuropathy, to determine whether these problems are related to practicing dentistry. The sound sources that might affect hearing in a dental practice working area include high-speed and low-speed turbines, amalgamators, high-volume suction devices, ultrasonic instruments, vibrators, model trimmers, and compressors.[5-10] Even with the advanced technology in modern dentistry, occupational health problems also exist, and these professionals are still exposed to occupational noise.[5]

Dental students start their practical experience as soon as they are in the fourth period of training at the laboratory facilities of the Brazilian dental school and may develop NIHL during their training. Thus, they are exposed to occupational noise during college, even before they start professional practice at private or public offices. Kadanakuppe et al.[11] measured noise levels in three dental students teaching areas (preclinical, clinical, and laboratory areas) and found levels ranging from 64 to 97 dBA, approaching the risk limit for potential noise-induced hearing impairment. They also reported that the laboratory devices generated the highest noise levels compared with high-speed and low-speed handpieces. In addition, they showed that used equipment from dental laboratories generates more noise than brand new ones. When comparing the hearing thresholds of first-year dentistry students with dentists working for more than five years, a significant decrease was seen at 4 kHz.

Evoked otoacoustic emissions (EOAs) are sensitive to outer hair cell damage. Therefore, it can be used to detect cochlear alterations in the ear and allow specific assessment of functionality in the outer hair cells (OHCs).[3,6] OAE has a well-documented role in detecting early and transitory changes in hearing status in studies concerning occupational noise. The amplitude of the signal and signal-to-noise (S/N) ratio of distortion product otoacoustic emissions (DPOAE) can detect these changes before alteration in pure tone audiometry becomes apparent since EOAEs are more sensitive to OHC damage.[7,8] Hence, investigators frequently use DPOAE as an OAE test in studies related to noise exposure.[9,10]

Under normal circumstances, the human cochlea contains antioxidants that play a crucial role against oxidative compounds produced during noise exposure[12]. In cases of excessive noise exposure, the naturally occurring antioxidant systems may not render sufficient detoxifying effects.[13,14] The outer hair cells endure metabolic depletion, leading to the accumulation of reactive oxygen and nitrogen species, ultimately leading to necrosis and apoptosis. [15]

Inner ear neural impulses are elicited, maintained, and propagated by electrochemical gradients. These gradients are maintained by energy-consuming active transport mechanisms in which free extracellular magnesium plays a vital role in binding, translocation, and intracellular release processes, preserving normal cell membrane stability and permeability to calcium.[14-19] Reduced extracellular magnesium levels, induced by either excessive stimulation or due to preexisting low magnesium levels, will result in decreased magnesium concentrations at the hair cell membrane and consequently cause an overall increase in membrane permeability. The decreased electrolytic gradients induce greater transport activity, which leads to an increase in energy turnover.[16] Further increases in free intracellular calcium ions can lead to energy depletion in the cell and, through activation of calcium ion-dependent enzymes, lead to a temporary or permanent impairment of hair cells.[14]

This study aims to identify whether noise exposure during dental practical classes may cause noise-induced TTS. Given that magnesium ions play an essential role in cochlear hair cells, this research also analyzes whether oral magnesium supplementation would have a protective effect against TTS evaluated by the DPOAE.

2. Metodology

This study was carried out between March and October 2019 at the Dental Department of the University of Brasília - UnB, in cooperation with the Otolaryngology research and teaching laboratory of the University of Brasília Medical School.

The authors received no specific funding for this work. We performed a randomized, triple-blind clinical trial (registered in the Brazilian Clinical Registry - identifier: https://ensaiosclinicos.gov.br/trial/9830). This study was submitted to and approved by the Human Research and Ethics committee at the University of Brasilia Medical School (n. 1.716.731). The authors confirm that all ongoing and related trials for this drug/intervention are registered.

The registration in the clinical trials platform was performed after the participants' recruitment due to the Brazilian policy of ethics in Human research. The clinical trials must be submitted and approved previously on the Brazilian platform, and we believed that this was enough to begin the research recruitment.

The participants recruited in this study attended regular practical dentistry classes during the fourth period of the dental department. Initially, the students were oriented to the research objectives. The students were also informed about the risks and benefits of magnesium used in this study. The enrolled participants included students aged 17–30 years, presenting no self-reported comorbidities, had no previous history of using ototoxic drugs, no amplified music exposure in the past 24 hours before the investigation, no history of middle ear infection, no auditory complaints, and an average serum magnesium level evaluated at a maximum of 1 month before the investigation. All participants signed a self-informed consent form and were advised of the possible adverse gastrointestinal effects (diarrhea, nausea, and abdominal pain) and the benefits of magnesium on their hearing. All participants recruited to the study were put to hearing rest 24 hours before the intervention.

The students were randomized (factorial method) into two groups: the study group (SG) received a dosage of 200 mg of magnesium aspartate 1 hour before noise exposure during practical laboratory training. The control group (CG) received a placebo treatment at the same dose, 1 hour before exposure. Cards were distributed randomly by a blinded researcher, which contained the letters "A" and "B." There was also one unblinded researcher for group distribution who was contacted by the students in case of side effects of magnesium. All participants enrolled in this study were exposed to the same environment of practical training, which ensured that they had approximately the same noise exposure conditions. The participants, audiologists, and statisticians were unaware of the groups and the treatment administered.

To measure the sound pressure during the study, the equivalent sound pressure level -Leq, that is, a continuous level equivalent to the sound produced during a given period was measured. Equivalent sound pressure level, Leq, in dB, was calculated according to the Brazilian NBR 10151 standards for the evaluation of urban noise by equation (1):

(1)

$$L_{eq} = 10 \times log_{10}\left(\frac{1}{T}\int_0^T \frac{P(t)^2}{P_0^2}dt\right)$$

where T is the duration of the reference period; P (t) is the instantaneous sound pressure; P0 is the reference sound pressure (2,0 ´ 10^{-5} N/m^2). LAeq is measured in weighting mode A.

Parameter declaration: L90, L10, Lmin, and Lmax
L90 - sound pressure level exceeded by 90% of the effective measurement time.
L10 - sound pressure level exceeded by 10% of the effective measurement time.
Lmin - lower sound pressure level at a given time interval;
Lmax - higher sound pressure level in a given time interval.

Environmental noise measurements were recorded in 15-minute time intervals at each of the four points, all located inside the laboratory close to the students performing practical drilling activities in resin molds with a high-speed handpiece and high-volume suction equipment. The equipment used for the acoustic measurements included the sound meter level, Solo Black, from 01 dB – Stell, France (65236), equipped with a frequency filter of one-third of octaves. The acoustic calibrator (0256), from the same company, is also class 01. The equipment was calibrated before and after the measurements.

All students underwent otoscopic inspection and audiological examination using DPOAE in a quiet, acoustically isolated room before and immediately after the exposure to laboratory noise to evaluate even temporary auditory threshold changes. The MAICO EROSCAN equipment (MAICO, Berlin, Germany) was used for the DPOAE test. The students remained seated while the audiologist performed the procedures in the following order: DPOAE in the right ear and followed by the left ear. The frequencies of 2, 4, 6, 8, 10, and 12 kHz were tested. To obtain the DPOAE, two pure tones were applied in the ratio F2/F1 = 1.22 and presented with an intensity of 65 dB SPL for F1 and 55 dB SPL for F2.

DPOAEs acquired before noise exposure were considered normal when the S/N ratio response was ≥ 6 dB SPL, and a signal amplitude was ≥ -5 at the analyzed frequencies.[3] Therefore, no student recruited was excluded because of the initial DPOAE result.

The amplitude of the signal and S/N ratio were then acquired and used for statistical comparisons in all individual frequency ranges analyzed.

The sample calculation analysis was performed using G*Power 3.1 software.[19] Based on a priori analysis, we adopted a power of 0.85, a = 0.05, a correlation coefficient of 0.5, nonsphericity correction of 1, and effect size (ES) of 0.20. From these values, an N of 32 subjects was calculated. The sample size was calculated based on procedures suggested by Beck et al.[20] This a priori statistical power analysis was conducted to reduce the likelihood of committing a type II error and determine the minimum number of participants needed for this investigation. It was determined that the selected sample size was sufficient to provide a statistical power greater than 86.1%. As the sample was completed with 33 subjects, in a post hoc analysis, the power of the final effect was 87.3%. The normality of data distribution was tested by *Kolmogorov-Smirnov* (K-S) tests. They showed normal distribution for tested variables ($p > 0.05$). A chi-squared adherence test tested sample homogeneity between SG and CG regarding sex distribution.

The amplitudes of the signal and S/N ratio variation between the right and left ears in each frequency range were dependent variables that were used for statistical comparisons separately in pre- and post-noise exposure situations between the SG and CG. The null hypothesis (H0) was considered if there were no differences between the right and left ears and/or between the SG and CG in the pre- and post-noise exposure period. Thus, the H0 is that the noise to which dentistry students are exposed to during one practical class does not change DPOAE. The differences were considered to be statistically relevant if the *p*-value was less than 0.005. Subsequently, the H0 was rejected, and an alternative hypothesis (H1) was considered. In this case, the amplitude of the signal and the S/N ratio between the ears and/or SG and CG showed statistical differences. Consequently, the alternative hypothesis is that the noise to which the dentistry students are subjected alters the temporary hearing thresholds. Magnesium acts as a protector, preventing this unwanted variation.[5,15]

Statistical Package for the Social Sciences (SPSS), version 21, was used to tabulate and analyze data. Descriptive statistical analyses were performed, such as mean, standard deviation, maximum, and minimum values percentages. Multivariate analysis of variance (MANOVA) was employed to compare the means of amplitudes and S/N ratio of DPOAE of SG and CG in pre- and post-noise exposure conditions among the different frequency ranges (2, 4, 6, 8, 10, and 12 kHz), in the right and left ears separately. If there was no difference between the right and left ears, they were grouped and analyzed together.

3. Results

The present study outlines a randomized triple-blinded clinical trial and included the participation of the University of Brasília dental students, who were allocated to two groups: SG and CG. In SG, 18 subjects with ages ranging from 19 to 24 years (M = 21.38 years; SD = 1.37) were included, mostly female (77.8%). In CG, 15 subjects with ages ranging from 19 to 25 years (M = 21.33; SD = 1.58) were allocated, including mostly females (60%). Two individuals were excluded since they did not complete the auditory examination protocols. We did not observe any statistical difference between the SG and CG regarding gender (p = 0.200).

A chi-square adherence test was performed to observe if the gender distribution was homogeneous, in which it was verified that there was no homogeneity between gender [$\square^2(2)=40.36$, $p < 0.001$]. The relevant data are shown in Table 1.

Table 1. Age and gender distribution in Study group and control group

	Study group	Control group
Age	*M* = 21.38 years; *SD* = 1.37 Min = 19 years Max = 24 years	*M* = 21.33 years; *SD* = 1.58 Min = 19 years Max = 25 years p = 0.200
Gender	Female = 77,8% Male= 22,2%	*Female = 60%* *Male= 40%* p < 0.001

*Note *: M = Mean; SD = standard deviation; p = Level of significance by MANOVA; Min = Minimum and Max = Maximum.*

No side effects were observed in the students after the administration of drugs. Thus, no participant was excluded.

Environmental sound pressure evaluation

The sound pressure level (SPL) evaluations are shown in table 1. The residual SPL measured–Leq assessed in the laboratory without practical dental activities was 54.3 dB (A), which indicates that the assessed noise, shown in Table 2, is essentially due to the activities developed during the practical laboratory training.

Table 2. Sound pressure levels measured in the laboratory (dB) (A)

Indexes	L_{min}	L_{90}	L_{eq}	L_{10}	L_{max}
1	55.3	57.0	74.4	78.2	98.8
2	56.5	57.3	74.6	77.8	90.9
3	56.7	59.3	76.8	78.8	98.3
4	54.5	58.3	74.8	77.8	97.7
Logarithmic Average	55.8	58.1	75.3	78.2	97.3

Note: L90 – sound pressure level exceeded by 90% of the effective measurement time; L10 – sound pressure level exceeded by 10% of the effective measurement time; Lmin – lower sound pressure level at a given time interval; Lmax – higher sound pressure level at a given time interval.

According to the SPL measurements, we found sound peaks almost as high as 100 dB (A). The mean Lmax was up to 97.3 dB. The mean L10 was 78.2 dB, and L90 was 58.1 dB. These findings demonstrate an important sound pressure oscillation during practical activities. The Leq varied from 74.4 to 76.8 dB (A) among the four different positions at the laboratory, presenting a logarithmic average of 75.3 dB (A).

DPOAE interaural comparison (right ear and left ear)

When the distribution of S/N ratio and amplitudes of signal in SG and CG in the right and left ears in all range of frequencies during the pre-noise exposure time were compared, no statistically significant differences was found [MANOVA test Pillai's trace = 0.04; F (6.57) = 0.40; *p* = 0.870 (for S/N ratio); Pillai's trace= 0.16; F (12.50) = 0.80; *p* = 0.640 for the amplitude of signal]. Therefore, the following statistical analyses were performed for the right and left ears together.

The amplitude of the signal and S/N ratio comparisons between the SG and CG in pre-noise exposure time among all frequencies studied

There were no statistically significant differences between the groups in all the frequencies studied for the amplitude analysis. MANOVA, [Pillai's trace = 0.14; F (6.56) = 1.55; *p* = 0.170]. In the S/N ratio, the MANOVA test showed no statistically significant differences between the groups [Pillai's trace = 0.10; F (6.56) = 1.107; *p* = 0.770].

The amplitude of the signal and S/N ratio comparisons in the SG in the post-noise exposure period among the frequencies studied

Table 3 shows the comparisons of the amplitude of the signal of DPOAE in the SG between the pre- and post-noise exposure period for all the frequencies studied. There were no statistically significant differences when we compared pre- and post-noise exposure periods. MANOVA, [Pillai trace = 0.007; F (6.63) = 0.07; *p* = 0.99].

Table 3. Mean, maximum, and minimum and p values of comparisons of the amplitude of the signal of DPOAE in all frequencies studied in the study group in the pre- and post-exposure noise period.

	Pre-noise exposure	*Post-noise exposure*	*P*
2 Hz	*M = 4.14; SD = 6.16* *Min = -8* *Max = 26*	*M = 4.16; SD = 7.66* *Min = -11* *Max =17*	*p = 0.990*
4 Hz	*M = -2.44; SD = 7.87* *Min = -18* *Max =15*	*M = -2.36; SD = 7.88* *Min = -18* *Max =19*	*p = 0.960*
6 Hz	*M = -5.73; SD = 9.56* *Min = -20* *Max =10*	*M = -5.05; SD = 10.44* *Min = -20* *Max =20*	*p = 0.770*
8 Hz	*M = -5.82; SD = 9.77* *Min = -20* *Max =10*	*M = -4,55; SD = 10.65* *Min = -20* *Max =19*	*p = 0.600*
10 Hz	*M = -1.32; SD = 9.39* *Min = -20* *Max =19*	*M = -0.19; SD = 10.76* *Min = -20* *Max =19*	*p = 0.640*
12 Hz	*M = -5.11; SD = 9.39* *Min = -20* *Max =15*	*M = -5.52; SD = 9.10* *Min = -20* *Max =20*	*p = 0.850*

*Note *: M = Mean; SD = standard deviation; p = Level of significance by MANOVA; Min = Minimum and Max = Maximum.*

Regarding the S/N ratio comparisons of DPOAE (Table 4), no statistically significant differences were observed in the post-noise exposure period in the SG. MANOVA, [Pillai trace = 0.04; F (6.63) = 0.517; *p* = 0.790].

Table 4. Mean, maximum, and minimum and p values of comparisons of the S/N ratio of DPOAE in all frequencies studied in the study group in pre- and post-exposure noise period.

	Pre-noise exposure	Post-noise exposure	*P*
2 Hz	*M* = 7.73; *SD* = 7.97 Min = -11 Max = 27	*M* = 10.55; *SD* = 8.90 Min = -8 Max = 30	*p* = 0.160
4 Hz	*M* = 12.47; *SD* = 8.81 Min = -7 Max =32	*M* = 14.33; *SD* = 7.90 Min = -1 Max =27	*p* = 0.350
6 Hz	*M* = 11.08; *SD* = 8.67 Min = -7 Max =28	*M* = 12.19; *SD* = 10.12 Min = -8 Max =32	*p* = 0.620
8 Hz	*M* = 9.32; *SD* = 10.04 Min = -7 Max =32	*M* = 10.61; *SD* = 10.93 Min = -6 Max =35	*p* = 0.610
10 Hz	*M* = 12.94; *SD* = 11.27 Min = -12 Max = 31	*M* = 9.30; *SD* = 9.52 Min = -14 Max = 32	*p* = 0.350
12 Hz	*M* = 6.38; *SD* = 9.31 Min = -10 Max = 27	*M* = 5.19; *SD* = 9.13 Min = -9 Max = 24	*p* = 0.590

*Note *: M = Mean; SD = standard deviation; p = Level of significance; Min = Minimum and Max = Maximum.*

The amplitude of the signal and S/N ratio comparisons in the CG in the post-noise exposure period among the frequencies studied

Regarding the amplitude of the signal of DPOAE, there were no statistically significant differences in any frequency studied after noise exposure in CG, MANOVA test [Pillai's trace = 0.14; F (6.52) = 1.504; *p* = 0.19]. Comparing the S/N ratio of DPOAE after noise exposure showed no statistically significant differences in any of the frequencies studied, MANOVA test [Pillai's trace = 0.07; F (6.52) = 0.678; *p* = 0.660].

The amplitude of the signal and S/N ratio of DPOAE comparisons between the SG and CG after noise exposure among the frequencies studied

When the amplitude of the signal of DPOAE between the SG and CG were compared, no statistically significant differences in any of the frequencies analyzed, MANOVA test [Pillai trace = 0.04; F (6.59) = 0.478; *p* = 0.82].

Regarding the comparisons of the S/N ratio of DPOAE between SG and CG (Table 5), we found no statistically significant differences in any of the frequencies analyzed, MANOVA test [Pillai trace = 0.08; F (6.59) = 0.851; *p* = 0.080].

Table 5. Mean, maximum, and minimum and p values of comparisons of the S/N ratio of DPOAE in all frequencies studied in the study group and control group in the post-exposure noise period.

	SG post-noise exposure	CG post-noise exposure	*P*
2 Hz	*M* = 10.55; *SD* = 8.90 Min = - 11 Max = 30	*M* = 13.13; *SD* = 8.68 Min = -11 Max = 30	*p* = 0.240
4 Hz	*M* = 14.33; *SD* = 7.90 Min = - 7 Max = 27	*M* = 16.00; *SD* = 7.42 Min = - 3 Max = 32	*p* = 0.380
6 Hz	*M* = 12.19; *SD* = 10.12 Min = - 8 Max = 32	*M* = 12.06; *SD* = 7.82 Min = -7 Max = 28	*p* = 0.950
8 Hz	*M* = 10.61; *SD* = 10.93 Min = - 6 Max = 35	*M* = 10.03; *SD*=8.43 Min = -7 Max = 28	*p* = 0.810
10 Hz	*M* = 12.94; *SD* = 11.27 Min = - 12 Max = 31	*M* = 9.30; *SD* = 9.52 Min = -14 Max = 32	*p* = 0.160
12 Hz	*M* = 5.19; *SD* = 9.13 Min = -9 Max = 27	*M* = 3.53; *SD* = 6.79 Min = -10 Max = 23	*p* = 0.410

*Note *: M = Mean; SD = standard deviation; p = Level of significance; Min = Minimum and Max = Maximum.*

3. Discussion

These study results show that there was no significant change in the amplitude of the signal and S/N ratio of DPOAE in all the studied ranges of frequencies in SG and CG, after approximately 3 hours of noise exposure during the dental laboratory class. Furthermore, the results showed that the effects of magnesium intake did not differ from the effects of placebo since there were no statistically significant differences in the DPOAE variables between the SG and CG.

The role of EOAEs in studies dealing with occupational noise and their accuracy in detecting early and transitory changes of hearing status is well recognized.[7,8] Studies indicate that exposure to intense sounds for about 15 minutes is sufficient for auditory changes to occur.[23-28] The signal amplitude and S/N ratio of DPOAE can detect these changes before alteration in pure tone audiometry becomes apparent since EOAEs are more sensitive to OHC damage. For these reasons, we employed the evaluation of DPOAE in the present study. Moreover, from our previous investigation, it was revealed that young individuals[2] presented with significant alterations in the DPOAE test when frequently exposed to amplified music. Additionally, 79.9% of the study sample presented with alterations, out of which 94.0% used earphones. Nonetheless, we developed this study and attempted to recruit participants with specific inclusion criteria, such as normal DPOAE and no comorbidities.

In addition to loud noise, there are many other risk factors (modifiable and non-modifiable) that can induce the progression of hearing loss. Modifiable risk factors include smoking, diabetes, and lack of exercise, and non-modifiable risk factors include

aging, race, and genetics. These factors can overlap with noise and accelerate the occurrence of permanent threshold shifts.[29] Older individuals and those who have ever suffered from sensorineural hearing loss are more susceptible to noise. Approximately 23% of those between the ages of 65 and 75 years suffer from mild or severe hearing loss. Over the age of 75 years, about 40% of individuals have hearing impairment.[29] Again, in the present investigation, we put in efforts to select a very controlled group of subjects to test our main hypothesis.

In the last few years, the proportion of women in dentistry has been rising steadily. By 2010, the majority of dental students in North America were female.[30] Both study groups had women predominantly. Both genders usually respond equally to noise.[31] However, gender influences acoustic risk-taking behaviors, e.g., boys engage in significantly more high-risk noise activities than girls.[31] Thus, since any abnormalities in DPOAE acquisition before sound exposition were exclusion criteria, the minor differences in gender distribution between groups might not interfere with the results.
The SPL monitoring showed that the mean logarithmic sound pressure at the four measured points in the laboratory was 75.3 dB lower than the safety level of 85 dB established.[1] The mean logarithmic sound pressure at percentile 90 (Leq90) was 58.1 dB; however, some mean values were measured up to 97.3 dB. This sound pressure, by itself, is enough to damage the auditory system.[32] Kadanakuppe et al.[18] studied the laboratory noise exposure in preclinical dental students and reported SPL up to 97 dB, approaching the risk limit for potential noise-induced hearing impairment. They also reported that the laboratory devices generated the highest levels of noise as compared to high-speed and low-speed handpieces of the dental clinic. In addition, they showed that used equipment from dental laboratories generates more noise than brand new equipment at the clinic. In our study, these sound pressure levels did not change the hearing status of the dental students in the laboratory class.

No significant change in the amplitude of the signal and the S/N ratio of DPOAE occurring in SG and CG might be explained by the noise exposure time and the averaged noise in the classroom (75.3 dB). It was demonstrated that SPL indicated pressure levels higher than 85 dB (Leqmax = 97 dB) during the class; however, the exposure time might not be enough to cause even a transitory threshold shift. Such SPL was enough to cause damage in the OHC functioning, as reported by several authors.[33,34]

One of the mechanisms of NIHL is the persistence of temporary damage to the hair cells, leading to irreversible damage. Thus, if there is some protector mechanism that interferes with the variation of these temporary changes, it needs to be a very effective otoprotector. Studies on the harmful effects of noise on hearing were first reported with the onset of industrialization. In the United States and Europe, these workplaces received great incentives due to the high social and economic impact. In this sense, dental students were not investigated previously regarding the association of noise exposure during the laboratory classes and hearing loss. They could be studied as a very controlled group for this analysis since they were not submitted to noise in dental clinics and were starting their practical training.

Dental professionals have to deal with occupational noise in their general practice, and no ear protector is usually allowed since they need to keep communicating with the patients during their treatment sessions. Following this scientific reasoning, we proposed to use a single dosage of magnesium to prevent DPOAE changes and further hearing loss seen in the PTA. The literature review did not elucidate the presentation and dosage of magnesium compounds to be supplemented in the diet to increase the levels of magnesium in the blood to prevent

hearing loss. Two previous reports in humans described the potential role of magnesium supplementation on hearing protection.[15,35]

The participants enrolled in our study had normal serum magnesium levels evaluated through a previous blood sampling. Attias et al[15] reported that even though serum magnesium levels are reduced to normal values via renal excretion within several hours, a magnesium depletion from intracellular sites and through the blood-brain barrier is much slower.[12] Therefore, intracellular parameters show only the long-term effects of magnesium supplementation. Therefore, we decided not to collect blood samples after the magnesium supplementation.

The laboratory noise exposure not changing the DPOAE parameters in the dental students significantly might explain the lack of hearing protection by the magnesium supplementation as reported by previous authors. However, NIHL is influenced by individual predisposition within high-risk populations. Therefore, further studies must be conducted to establish the role of magnesium supplementation in hearing preservation in NIHL. Since our study enrolled a very controlled population in a well-monitored SPL environment, we might propose another hypothesis: magnesium somehow plays a central role in inner ear cellular metabolism and during noise exposure in predisposed subjects, and in case of intracellular magnesium depletion, the supplementation may benefit high-risk populations, such as dental students. The participants of the present investigation had normal serum magnesium levels, and the supplementation did not alter the intracellular magnesium status and had no influence on the OHC metabolism.

Several authors[10,25] have reported that the reversible nature of the temporary threshold shift indicates that the underlying mechanism associated with hearing deterioration is most probably an ionic imbalance. Noise exposure results in high-energy consumption by the hair cells, and a magnesium deficiency may increase the potential for temporary threshold shifts and subsequently, permanent threshold shifts. The exact manner by which magnesium affects the susceptibility to noise-induced hearing loss is still unknown. However, in cases of normal magnesium reserves, no effects on hearing status may be observed during noise exposure. In the present study, the dental students had normal serum magnesium levels and did not present with even a transitory shift on DPOAE parameters. To elucidate its role in NIHL, further studies should be conducted comparing the effects of noise exposure in patients with normal and abnormal magnesium levels.[16]

Searching for novel hearing protectors is increasingly emphasized in dental workplaces since the impact of noise on hearing loss is becoming increasingly apparent. It has already been proven that for loud noises (over 120 dB), ear protectors cannot prevent hearing impairment because of bone transmission. In addition, in some professions, such as dentistry, ear protectors may interfere with the work to be performed, such as at times when it is necessary to identify the change of sound tone to detect the end of the procedure. Given these problems, the need for viable hearing protector options without sound muffling becomes evident.

In this research, the noise to which the students were exposed did not cause changes in DPOAE which is a limitation to the proper evaluation of the effect of magnesium supplementation on dentistry students submitted to noise exposure during the dental laboratory class. A large sample size might minimize type II error and identify temporary threshold shifts in dentistry students. Thus, the role of magnesium supplementation could be better evaluated.

4. Conclusions

Despite exposure to high-intensity sounds, even with 97dB peaks, we found no variation in hearing thresholds, as measured by DPOAE. A single 200 mg dose of magnesium did not produce any side effects; however, no effect was observed in hearing preservation in this investigation. Further studies, preferably clinical trials with large sample sizes, should be designed to understand whether magnesium supplementation preserves hearing in dental professionals.

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