

Presenting a Signal to Noise Ratio Model Based on the Combined Effect of Sound Pressure Level/frequency, Exposure Time and Oral Potassium: Experimental Study in Rats

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Abstract: Exposure to noise can lead to anatomical, nonauditory, and auditory impacts. The auditory influence of noise exposure is manifested in the form of Noise-induced hearing loss (NIHL). The current study aimed at present a signal to noise ratio model of otoacoustic emission of rats' ears in the light of the combined effect of sound pressure level, sound frequency, exposure time, and potassium concentration of the used water. In total, 36 adult male rates, whose age varied from 3 to 4 months and had a weight of 200 ± 50 g, were randomly divided into 12 groups, with each group consisting of 3 rats. The rats in both groups (case and control groups) were exposed to SPLs of 85, 95, and 105 dB, emitted from sources that generated white noise. A distortion product otoacoustic emission (DPOAE) machine (4000 I/O manufactured by Homoth of Germany) was utilized to gauge the signal to noise ratio (SNR) of otoacoustic emissions of rats' ears at various frequencies in an acoustic room. The inclusion criterion was $SNR \geq 6$ dB. The collected data were fed into the Statistical Package for Social Sciences (SPSS) version 18, followed by conducting descriptive and inferential data analysis procedures. The results of SNR analysis indicated that over 82% of all data had SNRs that were equal to or greater than 6 dB. These data were considered as acceptable response. Furthermore, SPL and sound frequency had significant associations with SNR ($P < 0.0001$). Exposure time also significantly correlated with SNR ($P = 0.008$). However, the potassium concentration of the used water had no significant correlation with SNR ($P = 0.97$). High sound pressure levels result in lower DPOAE. Furthermore, higher frequency leads to higher SNR. On the contrary, longer exposure time reduces SNR. Finally, the potassium concentration of the used water has no effect on SNR.

Keywords: Noise; sound pressure level; potassium; DPOAE; rat



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1 Introduction

Noise is the most common physical occupational factor across the world and is the main source of physical injuries that are caused by work [1]. Numerous studies have shown that harmful factors in the workplace such as noise, vibration, and shift work and so on have detrimental effects on workers' health [2-5]. As a work-related health issue, exposure to excessive noise can have a wide array of social and physiological repercussions, e.g., anatomical, nonauditory, and auditory effects [6]. More than 30 million workers, in the United States, are exposed to hazardous noises and 7.4-10.2 million industrial workers are at risk of hearing loss resulted by occupational noise. According to WHO standards, in Germany, around 4 to 5 million workers (constituting 12% to 14% of the country's population) are exposed to excessive sound pressure levels. The majority of work-related activities are along with a proportion of noise; however, some of these activities are conducted through excessive sound pressure levels [6].

Cardiovascular diseases (CVDs) have been frequently discussed in the scientific literature during the past 40 years and research has suggested that occupational noise exposure may possibly cause increased blood pressure (BP), hypertension [7]. The researchers have estimated that a 5-dB community noise reduction in the United States could lead to a decrease in prevalence of hypertension by 1.4% and coronary heart disease by 1.8%. However, research on the relationship between occupational noise exposure and cardiovascular outcomes is more ambiguous [8]. In addition, The auditory side effect of exposure to noise is manifested in the concept of noise-induced hearing loss (NIHL) [9] and tinnitus (that affected by risk factors including age, gender, various diseases, hearing loss, ototoxic drugs, caffeine, nicotine, and alcohol) [10]. These disorders, when combined with other workplace complications such as shift-work (in developing countries around 15% to 20% of the workforce and in developed countries around 25% of the workforce are engaged in shift- work) or heat and light of some occupations, have more severe effects on human health [11, 12].

NIHL causes bilateral and symmetrical hearing impairment [13, 14]. Out of the three main parts of ear (the outer ear, the middle ear, and the inner ear), the last one is mainly influenced by exposure to excessive noise [15, 16]. NIHL leads to mechanical and metabolic changes in the inner ear. In fact, high energy transfer, which is caused by high levels of noise, results in mechanical damages in delicate parts of the outer hair cells (OHCs) in the cochlea. This, in turn, has anatomical and physiological consequences because of the overstimulation of the inner ear. High energy transfer also increases metabolic stress in the endolymphatic fluids of the cochlea, resulting in the swelling and degeneration of the eighth nerve terminals attached to the inner hair cells [17, 18].

Otoacoustic emissions (OAEs) stem from microscopic biochemical activities of healthy outer hair cells (OHCs). Mechanical movements in the cochlea are caused by these activities. The movements are subsequently transferred from the tympanum to the outer ear and are resonated in the auditory canal [19].

Pre-neural phenomena create these emissions since they occur prior to the transfer of a signal to the auditory nerve [20]. Such emissions can be very helpful in examining the OHCs of the cochlea [21]. Auditory emissions consist of automotive auditory emissions and evoked otoacoustic emissions (transient otoacoustic, distortion product otoacoustic emission (DPOAE), and motive frequency otoacoustic emissions) [22].

In the current study, we used DPOAE test, which is one of the features of various types of OAEs, to examine cochlea's otoacoustic performance. In order to measure DPOAE, emissions made and reinforced in the cochlea by particular frequencies (f_1 and f_2) are measured [23]. As an objective and non-aggressive test, DPOAE exploits the properties of frequency sensitivity to assess otoacoustic damage [24].

In order to reveal the difference between the measured OAE and the background noise level, SNR is used. More precisely, positive SNR shows a measurable response over the background noise [25, 26]. In addition, similar studies for this purpose are scarce, and this study may be useful. Researchers can find a

suitable model by comparing the results of this study with other studies. As a result, based on the effect of sound pressure level (SPL) on OAEs, the current study aimed at:

1. Determining the SNR of otoacoustic emissions of rats' ears at various frequencies.
2. Comparing changes in SNR under various exposure conditions.
3. Presenting a model of the SNR of otoacoustic emissions of rats' ears based on the combined effect of SPL, sound frequency, exposure time, and potassium concentration of the used water.

2 Materials and Methods

2.1 Experimental Animals

Thirty-six adult male Sprague-Dawley rats were purchased from Pasteur Research Institute. They had an age range of 3 to 4 months and an average weight of 200 ± 50 g. Before embarking upon the study, they were kept in the animal unit of the School of Health, Tehran University of Medical Sciences. The rats were exposed to a photoperiodic cycle that consisted of 12 hours of light phase and 12 hours of dark phase while the temperature was around $23 \pm 2^\circ\text{C}$. All the rats had free access to water and food. We observed all the principles of the Declaration of Helsinki in conducting our experiment on these laboratory animals.

At the beginning of the research, the following two steps were taken to make sure that the rats' auditory system was healthy:

1. Rats' external auditory canal and eardrum was tested by the use of an otoscope (BI97150, made in the USA). The rats with defective ears (for example, excessive secretion of earwax/cerumen) were excluded from the study.
2. A particular sound was generated around the rats' ears. Rats' response to this sound indicated that their ears were healthy. In contrast, lack of any response was indicative of defective auditory system. Thus, those rats that did not have any reaction to the sound were excluded from the study.

2.2 Instruments for Noise Exposure

A highly efficient four-cell echo chamber with the dimensions of $40 \times 50 \times 60$ cm was used for conducting the experiment. In the chamber, sound energy was equally distributed in all directions, hence the amount of sound that the animals were exposed to did not depend on their location in the chamber. According to recommended conditions for taking care of animals, the room air must be replaced 12 times per hour [27]. To do this, a ventilation with a flow of 24 L/min was installed inside the chamber. It was composed of an environmental pump and a flow meter to control the flow rate. In the course of the experiment, the chamber's temperature was kept at $25 \pm 2^\circ\text{C}$, while the moisture was 50%. Three rats were put inside each chamber.

2.3 The Software and Source of Noise Generation

White noise was generated by the use of Signal. The noise files were played by the use of Coll Edit Pro (version 1-2, manufactured by Syntrillium Software Corporation in the United States in 1999–2000), while two speakers (PROBIT, manufactured in Iran) were exploited to produce the noise. The speakers had an input–output resistance of impedance: 4 (ohms) power: 5 (W) that was directly amplified through an amplifier (model ES-2000s, ES Audio Industrial Corporation) manufactured in Taiwan. The speakers were symmetrically installed in the chamber ceiling.

2.4 Preparing the Water Consumed by Rats with Various Levels of Potassium

The same water containing bicarbonate, calcium, sulfate, magnesium, sodium, fluoride, nitrate, chloride, phosphate, and ammonium was used in all groups. Potassium was the only ingredient that was manipulated in the light of the study. Based on the national standard of Iran (Standard 1053, the features of drinking water)

and the standards proposed by the World Health Organization, the maximum limit of potassium in drinking water is 10 mg/L [28, 29]. In the current study, the amount of potassium in the consumed water ranged from 0.1 mg/L to 8 mg/L. by doing this, it was possible to assess the effect of potassium level on otoacoustic emission of rats' ears. The water was accessible by rats 12 hours before the experiment, a move to ensure that they would drink the water.

2.5 Organization of the Experimental Groups

The selected rats were randomly divided into 12 groups, with each group containing 3 rats [6]. The status of rats in the control and case groups is explained below.

The rats in both groups (case and control) were exposed to SPLs of 85, 95, and 105 dB, manifested through white noise. The rats in the case group used the water that had a potassium concentration of 8 mg/L. The properties of the 12 subgroups of the control and case groups are displayed in the Tab. 1.

Table 1: Different subgroups of rats in the control group and case group

| Group name | Level of potassium in the consumed water | Sound pressure level (dBA) | Exposure time (h) |
|---------------|--|----------------------------|-------------------|
| control group | 0/1 Mg/L | 85 | 3 |
| | | | 8 |
| | | 95 | 3 |
| | | | 8 |
| | | 105 | 3 |
| | | | 8 |
| case group | 8 Mg/L | 85 | 3 |
| | | | 8 |
| | | 95 | 3 |
| | | | 8 |
| | | 105 | 3 |
| | | | 8 |

2.6 Measurement

2.6.1 Noise

A sound level meter in model CEL-440 (CEL-440, CASELLA, USA) was exploited to assess SPL in the four-cell chamber. This machine has an octave parser, hence indicating the SPL in octave band centers. Prior to the study, the machine was calibrated by the use of CEL-282 calibrator (CASELLA, USA). SPL was randomly gauged in various spots of each chamber cell.

2.6.2 Measuring DPOAEs

In order to measure their DPOAEs, rats should become unconscious. This was accomplished by the use of two types of drugs (Ketamine and Xylazine), with proportions of 60% and 40%, respectively. By the use of insulin syringes, 3 mL of the mixture was injected inside the peritoneum of each rat. A DPOAE machine (DPOAE 4000 I/O manufactured by Homoth of Germany) was utilized to gauge OAEs in animal phase in the following frequencies: 562, 1125, 2062.5, 3937.5, and 6562.5 Hz. The measurements were performed

in the acoustic room of the physical factors laboratory of the School of Public Health, Tehran University of Medical Sciences. The ratio of sounds emitted to rats' ears was $f1/f2 = 1/22$, whereas their intensity were $L1 = 65$ dB and $L2 = 55$ dB. In addition, SNRs for the three groups were calculated by the use of distortion-product otoacoustic emission - noise floor (DP-NF). Prior to administering DPOAE tests, the researchers ensured that the following prerequisites were met: (1) the external ear should not be obstructed; (2) the probe should be properly inserted in the ear canal; and (3) the probe should be appropriately positioned inside the ear canal [6].

2.7 Statistical Analysis

The collected data were fed into the Statistical Package for Social Sciences (SPSS) version 18. First, the data were summarized using descriptive statistical procedures. Subsequently, Shapiro-Wilk test was performed to assess the normal distribution of the data. Then, both within and between groups repeated measure analysis of variance (ANOVA) were conducted to examine the difference among various groups. The significance level was set at 0.05 ($P < 0.05$).

2.8 Ethical Considerations

The Ethics Committee of Tehran University of Medical Sciences (ID: 1394.5) approved this study. Additionally, we observed all the principles of the Declaration of Helsinki about conducting experiments on laboratory animals.

3 Results

3.1 The Results of SNR of DPOAEs in Rats

3.1.1 SNR Scores for Control Group

Table 2 illustrates the SNRs of otoacoustic emission of rats' ears when they were exposed to 85, 95, and 105 dBA white noise and consumed water with a potassium concentration of 0.1 mg/L. The measurements were conducted after 3 and 8 hours of exposure to sound.

Table 2: Mean and standard deviation of SNR in different times and frequencies among six control groups

| Sound pressure level (dBA) | Exposure time (h) | | Frequency (Hz) | | | | |
|----------------------------|-------------------|------|----------------|------|--------|--------|--------|
| | | | 562 | 1125 | 2062.5 | 3937.5 | 6562.5 |
| 85 | 3 | Mean | 8.70 | 10.3 | 11.42 | 17.28 | 9.33 |
| | | SD | 0.37 | 0.40 | 0.19 | 0.34 | 0.22 |
| | 8 | Mean | 7.76 | 9.5 | 10.48 | 16.53 | 8.66 |
| | | SD | 0.29 | 0.20 | 0.26 | 0.17 | 0.43 |
| 95 | 3 | Mean | 8.14 | 8.89 | 10.1 | 14.7 | 8.2 |
| | | SD | 0.18 | 0.29 | 0.27 | 0.19 | 0.34 |
| | 8 | Mean | 7.20 | 7.91 | 9.39 | 13.73 | 7.15 |
| | | SD | 0.15 | 0.44 | 0.21 | 0.35 | 0.14 |
| 105 | 3 | Mean | 7.93 | 7.8 | 9.15 | 12.74 | 7.28 |
| | | SD | 0.33 | 0.21 | 0.11 | 0.20 | 0.22 |
| | 8 | Mean | 6.44 | 7.36 | 8.41 | 11.80 | 7.08 |
| | | SD | 0.38 | 0.32 | 0.38 | 0.40 | 0.65 |

* SD: Standard deviation

3.1.2 SNR Scores for Case Group

Table 3 illustrates the SNRs of otoacoustic emission of rats' ears when they were exposed to 85, 95, and 105 dBA white noise and consumed water with a potassium concentration of 8 mg/L. The measurements were conducted after 3 and 8 hours of exposure to sound.

Table 3: Mean and Standard deviation of SNR in different times and frequencies among six case groups

| Sound pressure level (dBA) | Exposure time (h) | | Frequency (Hz) | | | | |
|----------------------------|-------------------|------|----------------|-------|--------|--------|--------|
| | | | 562 | 1125 | 2062.5 | 3937.5 | 6562.5 |
| 85 | 3 | Mean | 9.01 | 10.44 | 11.51 | 17.32 | 9.30 |
| | | SD | 0.18 | 0.51 | 0.17 | 0.51 | 0.34 |
| | 8 | Mean | 6.7 | 8.67 | 9.39 | 15.62 | 7.62 |
| | | SD | 0.43 | 0.22 | 0.34 | 0.14 | 0.53 |
| 95 | 3 | Mean | 8.24 | 8.43 | 10.20 | 14.80 | 8.43 |
| | | SD | 0.22 | 0.41 | 0.35 | 0.12 | 0.38 |
| | 8 | Mean | 6.14 | 6.73 | 8.25 | 12.57 | 6.16 |
| | | SD | 0.16 | 0.64 | 0.23 | 0.49 | 0.16 |
| 105 | 3 | Mean | 8.18 | 7.75 | 9.11 | 12.77 | 7.29 |
| | | SD | 0.21 | 0.22 | 0.11 | 0.21 | 0.21 |
| | 8 | Mean | 5.75 | 6.31 | 7.34 | 10.7 | 6.34 |
| | | SD | 0.25 | 0.44 | 0.48 | 0.61 | 1.02 |

* SD: Standard deviation

3.2 Final Model of the SNR

Table 4 contains the variables of the statistical model of the SNR of otoacoustic emissions of rats' ears.

$$\text{SNR (dB)} = 19.62 - 0.117 \text{ SPL} - 0.120 \text{ Time} - 0.001 K + 0.001 F \quad (1)$$

SPL = sound pressure level (dB)

Time = exposure time (h)

K = potassium concentration (mg/L)

F = frequency (Hz)

Table 4: The final SNR's model parameters of otoacoustic emissions of rats' ears

| Model variables | Non-standard coefficients | | Standard coefficient | P Value |
|----------------------|---------------------------|-------|----------------------|---------|
| | B | SE | BETA | |
| Fixed number | 19.62 | 0.824 | | <0.0001 |
| SPL (dB) | -0.117 | 0.010 | -0.524 | <0.0001 |
| Exposure time (hour) | -0.120 | 0.045 | -0.107 | 0.008 |
| Potassium (mg/L) | -0.001 | 0.034 | -0.001 | 0.97 |
| Frequency (Hz) | 0.001 | 0.000 | 0.381 | <0.0001 |

4 Discussion

The current study focused on assessing SNR in the frequencies of 562, 1125, 2062.5, 3937.5, and 6562.5 Hz and the SPLs of 85, 95, and 105 dBA. Comparisons were made between two different exposure times (3 and 8 hours) and two different potassium concentrations in the consumed water (0.1 and 8 mg/L). SNRs that were equal to or greater than 6 were regarded as acceptable responses. The results indicated that over 82% of the data had SNRs that were equal to or greater than 6 dB. Hence, these data were considered acceptable responses. The largest bulk of the 18% of data that was removed had to do with SNRs that were recorded in the frequency of 2000 Hz.

The results of data analysis also revealed that SPL and sound frequency are significantly related with SNR ($P < 0.0001$). More specifically, higher SPLs lead to lower SNRs, while greater frequencies result in higher SNRs. Moreover, exposure time correlated considerably with SNR ($P = 0.008$). That is, smaller SNRs were recorded for longer exposure times. Conversely, the concentration of potassium in the consumed water had no significant association with SNR ($P = 0.97$). Thus, increasing potassium concentration in water from 0.1 to 8 mg/L did not measurably influence SNR. On the other hand, the results of data analysis indicated that SPL explained 30.1% of the variations in SNR, whereas exposure time explicated 5% of such variations. Further, frequency sensitivity explained 6.6% of variations in SNR. The potassium concentration in water also explained 4% of the changes in SNR. In total, 45.7% of variations in SNR was explained by SPL, exposure time, potassium concentration, and frequency sensitivity.

Emmerich et al. conducted a long-term study on Indian guinea pigs to examine the effect of exposure to occupational noise on the otoacoustic emission of ears and destruction of hair cells. The destruction of hair cells was examined among 12 Indian guinea pigs before and 2 hours after exposure to specialized industrial noise, with the highest SPL being 105 dBA. The researchers reported that all the animals had fixed and acceptable DPOAE before the experiment. After a few hours of exposure to noise, the studied animals' DPOAE significantly reduced. Four months after the exposure, the animals' DPOAE improved up to 70%. The researchers argued that exposure to industrial noise causes both morphological and electrophysiological changes in the intermediate frequency range. They also indicated a close connection between reduced DPOAE and loss of sound resonance in the cochlea and damaged external hair cells. The researchers also reported a considerable association between decrease in DPOAE and decline in SPL [30]. Similarly, the results of this study showed that noise exposure causes decline in DPOAE thresholds.

Lund et al. studied the long-term influence of exposure time and low SPL on the variations in the threshold of hearing and DPOAE among rats. They showed that, in examining rats' threshold of hearing, DPOAE is a very sensitive test. In addition, they demonstrated that the results of assessing DPOAE drop among anesthetized rats were acceptable [31]. Similarly, the findings of the current study showed that, in sound pressure levels that are higher than 4 KHz, noise exposure leads to more significant drops in DPOAE threshold. In another study, Salehi et al. [32] investigated how well outer hair cells performed in rabbits as a result of exposure to noise. They revealed that noise exposure causes reduction in DPOAE threshold in frequencies of 4 to 10 KHz. Likewise, it was demonstrated in the current study that noise exposure causes measurable drops in DPOAE threshold in SPLs that are greater than 4 KHz.

Attias et al. [33] claimed that DPOAE test appropriately shows the changes in the case group (compared to the control group). Thus, it is a suitable test for assessing the performance of cochlea. In addition, Vinck et al. [34] concluded that noise exposure causes significant changes in DPOAE and TEOAE. Therefore, they stated that these tests can be used in order to evaluate the performance of cochlea. The above mentioned studies clearly indicate the validity of DPOAE test. As a result, this test was used in the current study.

The limitation of this study was the small number of DPOAE devices that were prepared by the researchers with much effort; and, in addition, the storage conditions of rats was another important limitation.

5 Conclusion

Based on the above mentioned findings, it is concluded that high SPLs lead to lower DPOAE thresholds. Furthermore, longer exposure time reduces SNR. Higher frequency also increases SNR. Nonetheless, potassium concentration in the used water does not have any significant impact on SNR.

Since DPOAE is sensitive to frequency range, it is recommended that the test should be used to assess the performance of cochlea (external hair cells).

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