# INFLUENCE OF SMOKING ON HUMAN HEARING – LITERATURE REVIEW

**Karol Myszel[1], Piotr Henryk Skarżyński[2, 3, 4]**

[1]Centre of Hearing and Speech, Kajetany, Nadarzyn, Poland

[2]Department of Tele-audiology and Screening, World Hearing Centre, Institute of Physiology and Pathology of Hearing, Warsaw, Poland

[3]Institute of Sensory Organs, Kajetany, Nadarzyn, Poland

[4]Heart Failure and Cardiac Rehabilitation Department, Faculty of Dental Medicine, Medical University of Warsaw, Warsaw, Poland

**SUMMARY**

*Objectives:* Smoking is a strong addiction, that affects a huge number of people worldwide, including the young ones. Due to composition of cigarette smoke, which contains nicotine and other chemical substances, lots of harmful effects on human health were described. Apart from the influence on other organs smoking is associated with hearing loss.

*Methods:* The literature review was conducted using PubMed and the combination of the following words: smoking, hearing impairment and hearing loss.

*Results:* The total number of 585 articles published in the recent 10 years were analysed. The review results show a strong association of hearing loss with smoking, both active and passive. As the main reason for hearing loss, a damage to outer hair cells was identified. Hearing loss in such cases is basically sensorineural and usually affects high frequencies. It was also observed that the risk of hearing loss increases with time of smoking. Smoking cessation reduces the risk of hearing loss associated with smoking. This article is a review of the literature that summarizes the results of studies aiming to analyse the influence of smoking on human hearing.

*Conclusions:* As smoking causes serious health problems, public health policies in societies should promote primary prevention as well as smoking cessation (secondary prevention) to diminish the total burden of healthcare systems.

*Key words:* smoking, hearing impairment, hearing loss, cochlear dysfunction, otoacoustic emissions

**Address for correspondence:** K. Myszel, Centre of Hearing and Speech, Mokra 7 Street, 05-830 Kajetany, Warsaw, Poland. E-mail: karol@myszel.pl

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## INTRODUCTION

According to numerous research projects conducted in different areas of medicine worldwide, smoking has been identified as a reason for many diseases. The population of smokers in the world reached around 1.2 billion (1, 2). Therefore, by causing health dysfunctions, smoking is a serious problem in our civilization. Its role in generating oncological, respiratory and cardiovascular diseases has been scientifically proven. As multi-discipline research continues, more and more evidence is collected that shows the negative influence of smoking on human body functions, which were earlier not directly associated with smoking (3–7).

As a very common addiction, smoking causes a lot of health problems and generates increasing costs and burdens to healthcare systems. Tobacco contains nicotine; however, there are many other components in the smoke like carbon dioxide, tartar, phenol, cresol, polycyclic aromatic hydrocarbons, benzopyrene, β-naphthylamine, nitrosamine, hydrogen cyanide, indole, carbazole, heavy metals (mercury, arsenic), and many others. The substances present in the smoke, such as particulates and gases, act as toxins, causing irritation and damage to cells, often leading to chronic inflammation or oncogenic transformation (8).

The purpose of this study was to review the literature describing the association of smoking with hearing impairment.

## MATERIALS AND METHODS

The literature review was conducted in PubMed using the connection of key phrases: smoking, hearing impairment and hearing loss. We found 1,049 targeted publications. Following this, we narrowed our research using the publication date browsing filter and limited the papers to those published within the past 10 years. Finally, 585 articles were found describing the influence of smoking on the auditory system. After a careful study of all the papers, as the results of many of them were coherent, the final version of our review was based on 30 publications listed in the bibliography, giving priority to research that covered the biggest samples and answered the research criteria the most adequately. The total sample included in the selected studies reached over 300,000 individuals.

## RESULTS

In-depth analysis of selected studies shows the strong coincidence of smoking and hearing impairment. The harmful effect of smoking on hearing organs was a subject of numerous studies both on animal and human subjects.

Animal studies conducted in laboratory conditions proved a damaging influence of smoking on outer hair cells of cochlea.

Cigarette smoke was identified as a cause of disorganization of stereocilia and damages to internal structure of cells. The most evident changes were usually found in basal turn of cochlea, whilst no significant changes were found in cochlear nerve. The research on animal subjects also showed that the level of damage is clearly dose-dependent.

Numerous studies in humans confirmed similar effects of smoking on their hearing. Studies were conducted on different level of human development as well as with coexistence of smoking with other factors that may affect the quality of hearing.

Research conducted on neonates showed lower presence of transiently evoked otoacoustic emissions (TEOAEs) in those, who were exposed to smoking during pregnancy. Similar findings were obtained in smoking adults. Lower TEOAEs responses were a result of damage of hair cells.

Studies performed in smoking exposed children on further development stages showed the decrease of TEOAEs levels. Apart from this finding, lower signal-to-noise ratios in children with exposure were evident. The same effect was also found in smoking adults.

According to other studies, in adolescents exposed to smoking prenatally, increased pure tone audiometry (PTA) thresholds were present and the changes showed statistical importance versus control group. The same effect of increased PTA and speech audiometry (SA) thresholds were found in adolescents brought up in smoking environment. Hearing impairment in such cases was sensorineural and involved mainly speech frequencies.

High frequency sensorineural hearing loss was also described in many studies of adult smokers, both males and females. Many research results also show a dose dependency meaning that the risk of sensorineural hearing loss increases with a number of pack-years.

Studies were also performed to analyse the influence of smoking on hearing impairment in subjects with co-existing conditions, including exposure to noise, central adiposity, poor glycaemic control and cardiovascular diseases. All of them were found to increase the risk of hearing loss in smokers. Summary of the results is presented in Table 1.

## DISCUSSION

The influence of smoking on the auditory system is an area of concern for many researchers in the world. This was a topic of numerous studies conducted by experimental biologists, histologists, physiologists, clinical neurologists, and specialists in otorhinolaryngology.

***Table 1.*** *Summary of audiometric changes observed in passive and active smokers vs. non-smokers*

| Summary | |
|---|---|
| TEOAE's | Emissions are lowered or responses are absent |
| SNR | Lower ratio, increase of noise component |
| PTA | Elevated hearing thresholds mainly at high frequencies, strong positive correlation with pack-years of smoking |
| SA | Speech discrimination threshold shift, deteriorated speech understanding |

TEOAE's – transiently evoked otoacoustic emissions; SNR – signal to noise ratio; PTA – pure tone audiometry; SA – speech audiometry

In their studies using guinea pigs, Abdel-Hafez et al. (9) examined the influence of nicotine on the structure of the cochlea. They used 15 male guinea pigs and exposed them to nicotine in different dosages (3 mg/kg and 6 mg/kg). Following this, they studied the structure of the cochlea using light microscopy, transmission electron microscopy, and scanning electron microscopy. The findings showed different levels of damage to the cochlea related mainly to the outer hair cells. The structure of the cells was seriously disorganized and damaged with a distorted shape of the cells, alteration of internal cell structures, disorganization of stereocilia, and expansion of supporting cells. Interestingly, the damages were prevalently present in the basal turn of the cochlea. The nicotine dosage increase resulted in more damage and caused a protrusion of the apical poles of hair cells. Therefore, the nicotine was proven to cause damage to the cochlea, with the range of damage showing a dose increase sensitivity.

Quite similar evidence was provided by Zhao et al. (10), who conducted research in rats exposed to nicotine. The examination of the cochlea proved their susceptibility to nicotine-induced toxicity presented as damage to outer hair cells of basic turn. The cells had damaged stereocilia that were swelled, and the endoplasmic reticula were degranulated. Compared to the changes in the cochlea, the study did not show changes in the structure of the spiral ganglia or auditory nerve fibres, whose morphology was basically unchanged.

The association of smoking with hearing impairment in humans was examined by different groups of scientists. Some of the researchers examined the effect of nicotine consumption during pregnancy on the hearing of neonates. Durante et al. (11) concentrated on otoacoustic emissions in neonates exposed to smoking during pregnancy to examine whether toxic substances in cigarette smoke may be harmful to the cochlea. Their measurements were conducted on a group of 105 neonates with appropriate hearing screening after birth, 47 in the study group with nicotine exposure and 58 neonates who were not exposed. They showed significantly lower responses in transiently evoked otoacoustic emissions and signal-to-noise ratios. The differences between the groups were of statistical significance. Interestingly, the same effect was not present when assessed by distortion products otoacoustic emissions (DPOAEs), which brought to the conclusion that TEOAEs seem to be a better indicator of cochlear malfunction potentially caused by smoking. To prevent the bias caused by other reasons, the study group was recruited in a way that excluded other hearing impairment risk factors (low birth weight, gestational age, Apgar, etc.) to ensure homogeneity of the group.

Jedrzejczak et al. (12) concentrated their research on the analysis of otoacoustic emissions (OAEs) in smokers and non-smokers. They focused on spontaneous otoacoustic emissions (SOAEs). These are the sounds spontaneously emitted by the cochlea. It is still not clear why they are present in some people, and not present in others; however, SOAEs are usually associated with good hearing. The study was conducted on a group of 48 young adults with average smoking history of 1.8 years. They showed a general decrease in OAE levels in smokers compared to non-smokers; however, ears with SOAEs did not present a statistical difference in OAE levels between the groups. Alternatively, in ears without SOAEs, smaller evoked

OAE were registered in smokers than in non-smokers, even though the hearing thresholds of the PTA were normal. Thus, the conclusion was that ears without SOAEs tend to be more sensitive to damage caused by smoking.

Another study conducted by Durante et al. (13) examined the effect of tobacco smoke exposure in childhood on cochlear physiology. In this study, the study group was composed of 145 students aged 8 to 10 years old. The group was divided into two subgroups, those with exposure to tobacco smoke and those without exposure. To double-check the effect of smoke, the level of cotinine, the main metabolite of nicotine, was measured in each participant's urine. The exposed group presented increased cotinine levels (over 5.0 ng/mL), lower response levels in TE-OAE, and lower signal-to-noise ratios compared to those without exposure. The results of these studies showed that exposure to smoke in prenatal life and in childhood (passive smoking) may lead to damage of the cochlea, resulting in hearing impairment. The strong dependency between chronic smoking and cochlea function was also described by Prabhu et al. (14), who tested ultra-high frequency sensitivity in smokers and non-smokers. They measured ultra-high frequency OAEs, which appeared to be significantly elevated in smokers. Coherent observations were also made by other authors (15–17).

Ultrastructural changes in hair cells as a result of exposure to smoking may lead to hearing impairment. Numerous studies were conducted worldwide that prove the association between smoking (passive and active) and hearing impairment. The study was performed by Weitzman et al. (18) as a cross-sectional data analysis of 964 adolescents aged 12–15 years to evaluate whether their prenatal exposure to tobacco smoke is associated with hearing loss. Adolescents exposed to tobacco smoke prenatally presented elevated hearing thresholds in PTA, mainly at 2 kHz and 6 kHz frequencies. In the same study, the risk of unilateral low-frequency sensorineural hearing loss was found to be 3 times higher in exposed adolescents compared to those without exposure. The researchers concluded that prenatal exposure to parental smoking may be harmful to a child's auditory system.

Talaat et al. (19) examined 411 children aged 5–11 years divided into three groups: no exposure, mild exposure and heavy exposure to smoke in their home environment. Every child was subjected to pure tone audiometry and speech audiometry. The prevalence of hearing impairment was 3.8%, 4.5% and 12%, respectively. Analysing the results, researchers found a significant difference in hearing thresholds between the heavy exposure group and two others. In the affected group, children had sensorineural hearing loss up to 25 dB. Even though this is a minimal level of hypoacusis, the correlation between passive smoking in childhood and sensorineural hearing loss was found in the study. Wilunda et al. (20) made similar observations.

The research done by Sumit et al. (21) in Bangladesh included 184 subjects (90 smokers and 94 non-smokers) and showed statistically higher hearing thresholds in smokers compared to non-smokers. The main differences were found in frequencies of 4, 8 and 12 kHz, whilst no difference was found for 1 kHz. As far as the duration of smoking was concerned, higher auditory thresholds were present in those who smoked for more than 5 years versus those who smoked for a shorter time. Therefore, the researchers concluded that smoking cigarettes negatively affects hearing, mainly at higher frequencies. Similar findings were described by Kumar et al. (22) based on their study in a group of 148 individuals (108 smokers and 40 non-smokers). The smoking history of the smokers ranged from 20 to 60 years. Increased hearing thresholds were found in smokers, prevailingly in the form of mild (26–40 dB) and, in some cases, severe hypoacusis. Most of the examined smokers (almost 57%) had hearing impairment, which was mainly of the sensorineural type (77.5%). These observations are pretty much in line with the results achieved by Wang et al. (23) in their cross-sectional study of a population of 4,685 individuals in China. They found a significant association between smoking and sensorineural hearing loss in speech frequencies and high frequencies. The prevalence was in middle-aged males showing much higher hearing thresholds. Apart from this, they also found a dose-dependent relationship between the intensity of smoking and hypoacusis. In older males, speech frequency sensorineural hearing loss was found mostly in past smokers versus never-smoking peers. Therefore, the study showed a relationship between smoking and hearing loss that varied according to age and gender. Demir et al. (24) conducted a clinical study to evaluate whether the effect of smoking on human hearing showed gender differences. They examined a sample of 236 individuals (90 non-smokers, 42 females and 48 males; and 146 smokers, 72 females and 74 males). They proved that both female and male smokers, who smoked intensively (more than 20 pack-years), had significantly higher hearing thresholds, mainly at 4 kHz and 6 kHz. In the range of 11–20 pack-years, more females presented with a 6 kHz threshold increase. The final conclusion was that tobacco smoking in males and females leads to high-frequency hearing loss, which is evidently dose-dependent.

Syed et al. (25) proved the influence of smoking on sensorineural hearing loss in middle-aged men. They conducted a cross-sectional study of 500 hundred Pakistani male smokers aged 21 to 50 years old and a control group of 500 non-smoking peers. The history of smoking was also considered. As a result, smokers had statistically higher hearing thresholds than non-smokers. The level of hearing loss was also positively correlated with the smoking history and greater in those smoking for over 10 years. Moderate or severe hearing impairment was found in association with smoking in a prospective study that was done in a group of 85,505 women by Lin et al. (26). The study group was divided into subgroups of current smokers, past smokers, and non-smokers. The researchers found a much higher risk of hearing impairment in the group of current smokers increasing with a higher number of pack-years smoking history. Simultaneously, the risk diminished with a longer time since smoking cessation; however, the tendency was higher as the time since quitting reached more than 10 years. Similar results were obtained also by other authors (27–33). The study of Mehrparvar et al. (34), who conducted a study of 224 workers exposed to noise, showed that distortion product otoacoustic emissions in subjects were significantly higher at frequencies over 1 kHz. This led to the conclusion that smoking can aggravate the effect of noise on cochlear function, thus may lead to more severe hypoacusis. A strong dependency between increasing pack-years of smoking and noise-induced hearing loss was also described by other authors (35, 36). The hearing loss in smokers exposed to noise was proved to appear faster and usually was deeper. Therefore, both smoking and noise separately are risks of hearing loss; however, the coincidence of both factors increases the risk significantly.

Other risk factors, including central adiposity, poor glycaemic control and cardiovascular disease were found to increase the risk of hearing loss in smokers. This was described in the research conducted by Criucshanks et al. (37) and Lohi et al. (38), and continues to be a subject of interest for other researchers worldwide.

## CONCLUSIONS

The review of the literature and the results of multicentre studies leads to the conclusion that smoking cigarettes, both passive and active, is strongly associated with hearing loss. The mechanisms of the damage caused by smoke to the auditory system still need deeper studies; however, the ultrastructural changes proved so far include damage to outer hair cells, probably by disturbing protective antioxidant mechanisms, with the structure of spiral ganglia and auditory nerve fibres unchanged.

The damage to the auditory system caused by smoking leads to hearing impairment of the sensorineural type, mostly covering speech and high-frequency ranges. The association of smoking and hearing loss is dose-dependent, increasing with the number of pack-years of smoking. Smoking cessation diminishes the risk of sensorineural hearing loss. Smoking can aggravate the negative effect of noise on the auditory system.

Based on the conclusions gathered from the review, the protective protocols for smokers should mainly include actions to cease smoking, or if this is not possible, search for alternatives that could eliminate the intake of the most harmful chemical substances present in cigarette smoke as a result of the combustion process.

Additionally, a crucial role of primary prevention has to be stressed. Educational systems and healthcare policies should be first concentrated on increasing awareness of health benefits from not taking up smoking. Stronger awareness should be also built amongst smoking parents on how their smoking may affect minors.

**Conflicts of Interest**
None declared

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