**Research Article**

# Auditory Efferent Pathway Functioning in Individuals with Misophonia

Sajana Aryal[1*], Prashanth Prabhu[2]

[1.] Department of Speech, Language, and Hearing Sciences, University of Texas at Austin, Austin, Texas, United States of America

[2.] Department of Audiology, All India Institute of Speech and Hearing, Mysuru, India

**ORCID ID:**
Sajana Aryal: 0000-0002-3589-5938
Prashanth Prabhu: 0000-0002-9878-4733

**Citation:** Aryal S, Prabhu P. Auditory Efferent Pathway Functioning in Individuals with Misophonia. Aud Vestib Res. 2024;33(4):?-?.

**Article info:**
Received: 08 Jan 2024
Revised: 02 Mar 2024
Accepted: 10 Mar 2024

[*] **Corresponding Author:** Department of Speech, Language, and Hearing Sciences, University of Texas at Austin, Austin, Texas, United States of America. E-mail: sajanaaryal5566@gmail.com

**Short running title:** Auditory Efferent Pathway Functioning in…

**Highlights:**

- Misophonia is a little-understood disorder from an audiological perspective
- The study shed light on the underlying auditory mechanisms involved in misophonia
- The outcomes provide crucial insights for the investigation of misophonia

## ABSTRACT

**Background and Aim:** Misophonia, characterized by a decreased tolerance for specific auditory stimuli, has been insufficiently explored within audiology. Limited research has been conducted, and the auditory mechanisms involved in this disorder remain to be explored. Hence, our study aimed to investigate the auditory efferent systems in individuals with misophonia. By focusing on this specific aspect, we aim to contribute to a better understanding of misophonia and shed light on the underlying auditory mechanisms involved in the condition.

**Methods:** A cross-sectional research was performed with students from Mysore University to investigate misophonia. The severity of misophonia was evaluated using the revised Amsterdam misophonia scale. The participants were divided into two groups based on their misophonia severity: mild (n=15) and moderate-severe (n=15). All participants underwent transient evoked otoacoustic emissions with contralateral suppression to assess the auditory function. The overall amplitude and frequency-specific amplitudes were analyzed and compared across the various groups.

**Results:** The analysis of variance results revealed no significant differences between the groups in global amplitude suppression and suppression of all frequencies. These findings imply that the medial-olivocochlear bundle efferent pathway is intact among individuals with misophonia.

**Conclusion:** Our findings have concluded that the medial olivocochlear bundle appears intact among individuals with misophonia ($p>0.05$). However, it is essential to note that the generalizability of these findings may be limited due to the relatively small sample size used in our study. Therefore, further research involving a more extensive and diverse population is needed to validate and generalize these conclusions.

**Keywords:** Misophonia; efferent pathway; contralateral suppression; neurophysiology; audiology

## Introduction

Misophonia is a disorder characterized by a decreased tolerance for particular auditory stimuli [1]. These stimuli, referred to as triggers, elicit diverse emotional and physiological responses in affected individuals, such as anxiety, rage, irritability, and disgust. Misophonia is a little-known disorder with a high prevalence rate. Literature has shown that the prevalence rate of misophonia ranges from 3.5% [2] to 23.28% [3] to 49.1% [4]. The significant disparity in prevalence rates could be attributed to the diverse methodology and samples used in the research.

Misophonia can occur as an isolated disorder or associated with other auditory and psychiatric disorders [5]. The comorbidity with auditory disorders includes hyperacusis and phonophobia. Hyperacusis is the physical experience of discomfort or pain in response to sounds. The sounds are perceived as too loud, even though they would typically be considered tolerable by most individuals [6]. Phonophobia, conversely, is characterized by fear of specific sounds known as triggers [7].

Recently, there has been an increasing interest in the scientific literature to comprehend the neuroanatomy and neurophysiology underlying misophonia. Researchers are actively exploring the neural mechanisms and processes associated with this disorder [8-11]. There has been debate in the literature to categorize misophonia as an auditory, psychiatric, or neurological disorder [12]. In individuals suffering from misophonia, the neuroanatomical pattern analyzed using functional Magnetic Resonance Imaging (fMRI) revealed activation of auditory-insula-limbic regions [11]. Several researchers have also reported similar findings in the literature [13, 14].

Currently, the assessment and management of misophonia are not known clearly. In addition, there is no medical treatment for it, even though different drugs have been trialed across the literature [15]. From the audiological perspective, very little research has been done [16]. Neurophysiological findings have shown hyperactivation of the ascending central auditory pathway structures, with no changes in the efferent pathway [16]. However, there is a lack of studies done from an audiological perspective to understand the functioning of the efferent pathway. Hence, we aim to evaluate the functioning of the efferent pathway in individuals suffering from misophonia disorder through the electroacoustic test.

The efferent pathway is part of the central auditory pathway, starting from the auditory cortex and ending in the inner ear [17]. The part of the efferent auditory pathway, the Olivocochlear Bundle (OCB), located within the brainstem has two parts: the medial olivocochlear bundle and the lateral olivocochlear bundle. The medial olivocochlear bundle has thick and myelinated nerve fibers that terminate at the base of the Outer Hair Cells (OHCs), predominantly on the contralateral side [17]. The Medial Olivocochlear bundle (MOC) fibers establish synaptic connections with the outer hair cells, and their activation leads to the inhibition of the basilar membrane response to low-frequency sound [18]. Otoacoustic Emissions (OAE) reflect the modulation in the gain of the cochlear amplifier, which is facilitated by the efferent pathway.

Eliciting MOC activity with the elicitor sound ipsilateral or contralateral to the OAE test ear is one way to monitor the MOC effect. Contralateral suppression, on the other hand, has been frequently adopted due to additional issues created by ipsilateral suppression, such as cochlear masking [17]. Contralateral suppression of the Transient Evoked Otoacousic Emissions (TEOAEs) refers to the reduction in amplitude observed when simultaneous noise is presented to the ear opposite to the one being tested. This phenomenon manifests as a decrease in the amplitude of TEOAEs during the presence of contralateral noise.

Our study evaluates the MOC activity by administering contralateral suppression of the TEOAEs. TEOAEs have been chosen as a reliable test as their sensitivity is high for the presence of hearing loss [17, 19]. Misophonia can manifest as an independent disorder or coexist with other auditory conditions, including tinnitus, hyperacusis, and phonophobia. Several studies have demonstrated abnormal activation patterns within the efferent auditory pathway in individuals with tinnitus and hyperacusis [6, 20]. However, few studies have shown no problem with the efferent pathway in these populations [21]. Hence, to understand the association of misophonia with tinnitus and hyperacusis, we aim to evaluate efferent pathway functioning in individuals with misophonia disorder using contralateral suppression of TEOAE.

## Methods

Before their participation, all individuals received comprehensive information regarding the study procedures. Written informed consent was obtained from each participant, ensuring their voluntary agreement to participate in the study. The Revised Amsterdam Misophonia Scale (Revised A-MISO-S) was utilized to survey the

prevalence of misophonia among students from Mysore University [22], which was initially developed by Schroder et al. [23].
An experimental study was conducted to investigate individuals who experienced clinically significant misophonia with a healthy control group for comparison. Based on the survey results, 40 individuals exhibiting misophonia symptoms were invited to participate in the study. The average age of the participants was 25 years (±7.8 years). Among the individuals in the misophonia group, 36 (90%) out of the 40 were female, while 4 (10%) were male. All participants were literate and came from diverse educational backgrounds, including audiology, business, medicine, and speech-language pathology. All subjects had been experiencing misophonia for a minimum of three years and had no hearing loss. For the control group, fifteen individuals with no misophonia or other ear/health-related symptoms were recruited ($x\square$=24±6years). To enable effective comparison, the gender distribution in the control group was carefully matched with that of the misophonia group. This matching process ensured a balanced representation of genders in both groups, minimizing potential gender-related confounding factors during the analysis and interpretation of the results. Overall, the study included a total of 55 participants.

**Misophonia severity evaluation**
The participants were invited to the study after confirming that the patient had misophonia symptoms through the analysis of the responses to the Amersdam misophonia questionnaires and the misophonia assessment questionnaires received via the survey. As the second step of the study, the revised version of the Amsterdam Misophonia Scale was readministered to the participants. This step involved assessing and quantifying the severity of misophonia symptoms using the updated scale [22]. Revised A-MISO-S was chosen as this is the most widely used questionnaire developed to assess misophonia.
The questionnaire used in the study consisted of ten questions, with scores ranging from 0 to 40. The scale aimed to assess various aspects related to misophonia, including the amount of time an individual spends preoccupied with misophonic sounds, the interference of misophonic sounds with social functioning, the level of anger triggered by sounds, resistance to impulsive reactions, control over thoughts and anger, and the amount of time spent avoiding misophonic situations. For scoring purposes, the categorization of misophonia symptoms was as follows: a score ranging from 0 to 10 indicated subclinical misophonia symptoms, a score between 11 and 20 indicated mild misophonia, a score between 21 and 30 indicated moderate to severe misophonia, and a score between 31 and 40 indicated severe to extreme misophonia. This scoring system was used to classify the severity of misophonia symptoms based on the participant's responses to the revised version of the Amsterdam misophonia scale.
Out of the initial 40 participants invited to the study, ten individuals who exhibited subclinical symptoms with a score lower than ten on the Revised Amsterdam misophonia scale were excluded from further analysis. Consequently, a total of 30 participants were selected for the misophonia group (mild misophonia group=15, moderate-severe group=15) with comparison to control group (n=15).

**Tinnitus and hyperacusis evaluation**
Misophonia can occur as a co-morbid disorder with other auditory disorders, such as tinnitus and hyperacusis. The questions were asked regarding the presence of tinnitus subjectively for all the participants. A tinnitus handicap inventory (Kannada version) was administered among all the participants to rule out the severity of tinnitus [24]. A score of less than 10 on tinnitus handicap inventory is considered no tinnitus handicap. Similarly, a Loudness Discomfort Level (LDL) test was done starting from 70 dB HL and increasing on the ascending run until the subject felt uncomfortable with the sound to evaluate the presence of hyperacusis among all the participants included in the study [25]. A loudness discomfort level greater than 90 dB HL is a normal loudness tolerance ability.

**Audiological evaluation**
In the audiological evaluation sequence, several tests were conducted by an experienced audiologist with at least six years of work experience. The evaluation began with obtaining a detailed case history of the participants, focusing on the ear and health-related information. Afterward, an otoscopic examination was conducted to evaluate the state of the outer and middle ear. To ensure accurate results in the subsequent tests, pure tone audiometry, tympanometry, and reflexometry were performed on all participants before administering TEOAEs. Pure tone audiometry was conducted using the Grason-Stadler (GSI) Audio Star Pro in a sound-treated room, adhering to the guidelines set by the American National Standard Institute (ANSI) [26]. Before conducting the tests, the audiometer was calibrated subjectively to ensure accurate measurements. Supraural TDH-50 headphones were employed to assess the Air Conduction (AC) thresholds spanning from 0.25 kHz to 8 kHz,

while the B-71 bone vibrator was utilized to measure the bone conduction thresholds ranging from 0.25 kHz to 4 kHz. The hearing thresholds were determined by averaging the results obtained at four frequencies: 500 Hz, 1 kHz, 2 kHz, and 4 kHz. Normal hearing was defined as an average AC value of 15 dB HL or lower with no air-bone gap [27].

**Transient evoked otoacousic emissions and contralateral suppression paradigm**

TEOAEs with contralateral suppression were done in the sound-treated room using the otodynamics Echo port ILOV6 equipment following the ANSI guidelines (Frank., 1997). The continuous contralateral noise paradigm was employed to measure TEOAEs and evaluate the contralateral suppression effect of TEOAEs. This method involves presenting a click stimulus to the ear being tested using probe one while simultaneously introducing broadband noise as the suppressor to the contralateral ear through probe 2. Continuous contralateral noise suppression is a widely utilized technique for studying the impact of contralateral noise on TEOAE responses, allowing researchers to assess the level of suppression in the efferent auditory pathway.

TEOAEs were measured using the two conditions. In the first condition, TEOAEs were recorded with the MASKER OFF condition, in which the suppressor was not presented to the contralateral ear. In the second condition, TEOAEs were recorded with MASKER ON condition in which a suppressor was delivered to the contralateral ear. The recording was done three times in all conditions to obtain reliable TEOAEs with better stimulus stability and response reproducibility [28]. Minimum stimulus stability of 70% and reproducibility of 80% were set as the criteria for accepting the TEOAEs response [29]. The intensity of the click stimulus used was 80 dB SPL in the nonlinear mode. During the test, participants were asked to relax and sit comfortably on the reclining chair.

Similarly, the broadband white noise was used as the suppressing stimulus in the contralateral ear, and the suppressor level was 50 dB SPL. The suppressor level was not set too high to prevent the activation of the middle ear reflex and prevent cross-hearing to the contralateral ear [29]. Few participants reported annoyance with the suppressor and click stimulus during the recording. They were given a gap between the tests to make them comfortable during the testing.

All the participants were asked to sit on a comfortable chair inside the sound booth for the TEOAE measurements. Participants were given careful instructions about the test procedure and asked to remain silent during the process. Before recording each participant, calibration was done using the ILO probe-fit paradigm. The recording was done by inserting probe 1 in the right ear canal to deliver stimulus and probe 2 in the left ear canal to provide broadband white noise suppressor stimulus and vice versa. The proper ear tip was chosen depending on the ear canal size of each participant. The recording was done in all the participants with and without suppressing stimulus.

The global amplitude was calculated for each participant. Frequency-specific amplitude value was obtained for the 1000 Hz, 1414 Hz, 2000 Hz, 2828 Hz, and 4000 Hz frequencies. The total suppression value of the global amplitude and the amplitude of each frequency were calculated by subtracting TEOAE responses with the masker on condition from the masker off condition. All parameters' suppression values were analyzed among the control, mild, and moderate-severe groups.

**Statistical analyses**

The data collected for the study were analyzed using the IBM SPSS program, version 25.0. Firstly, the Shapiro-Wilk test assessed whether the data followed a normal distribution. Since the data exhibited a normal distribution, a parametric one-way analysis of variance test was utilized to investigate significant differences between the misophonia and control groups. The dependent variables in the study consisted of the global amplitude and the amplitude at each frequency, while the severity of misophonia was treated as the independent variable. The criterion for determining statistical significance was set at a p-value less than 0.05, with a 95% confidence interval. The study aimed to identify significant group differences by employing these statistical procedures.

**Results**

**Misophonia severity**

According to the study findings, ten individuals were categorized as having moderate to severe misophonia, as evidenced by scores ranging from 21 to 30 on the RAMISO-S. Furthermore, 5 participants were identified as having severe to extreme misophonia, scoring between 31 and 40 on the scale. Conversely, 15 participants exhibited mild misophonia, scoring between 11 and 20. In the control group, all 15 participants scored zero on the updated RAMISO-S scale, indicating the absence of misophonia symptoms. All the participants included in

the misophonia and control group did not have comorbidity with other auditory and psychiatric disorders. All the participants had loudness discomfort levels (>90 dB HL), indicating the absence of hyperacusis. Likewise, all the participants included in both the control and experimental groups had a tinnitus handicap score of less than 10 (mean score=4.5), indicating no tinnitus handicap.

Due to insufficient sample size to create three distinct groups, the misophonia participants were categorized into two groups. The first group, comprising 15 participants, included individuals with mild misophonia. The mean score for this group was 15.93, with a standard deviation of 2.89. The second group consisted of individuals with moderate to severe misophonia, also comprising 15 participants. The mean score for this group was 25.86, with a standard deviation of 4.98.

Furthermore, it was observed that all participants had experienced misophonia for a minimum duration of 3 years. The range of experience varied from 3 to 8 years, with a mean duration of 4.93 years and a standard deviation of 1.52 years. Most participants (86.67%) reported a gradual onset of their misophonia symptoms. Various sounds were identified as triggers by a significant number of participants, with scratching being the most commonly reported trigger (66.67%), followed by loud sounds (50%) and chewing (46.67%). Table 1 provides further details on the misophonia characteristics of all participants included in the study

**Audiological evaluation**

Upon examination, it was found that all subjects' external and middle ear appearances were normal, and they reported normal hearing. All the participants included in the study had the presence of an 'A' type tympanogram and the presence of acoustic reflexes within the normal range. The statistical analysis indicated that there were no significant differences in AC thresholds between the study group and the control group for the right ear ($F_{(2.42)}$=0.587, p=0.561) and the left ear ($F_{(2.42)}$=2.540, p=0.091). These findings suggest no significant differences in the AC thresholds between the groups being compared.

In terms of bone conduction testing also, statistical analysis did not reveal any significant differences between the study and control groups for bone conduction thresholds in both the right ear ($F_{(2.42)}$=0.678, p=0.66) and the left ear ($F_{(2.42)}$=1.540, p=0.08). These results suggest no significant differences in bone conduction thresholds between the groups being compared. For more detailed information about the audiological findings of each participant, refer to Table 2.

**Transient evoked otoacoustic emissions with contralateral suppression findings**

The result of the TEOAE's with contralateral suppression was analyzed to determine the functioning of the MOC bundle efferent pathway in individuals with misophonia. The mean value of stimulus stability was 98%, and reproducibility was 96%. The suppression value was analyzed in terms of global amplitude suppression and amplitude suppression of each frequency for the right and left ear in each groups.

**Global amplitude suppression**

Upon analyzing the results, it was observed that both the control and experimental groups exhibited contralateral signal suppression. When exposed to noise, there was a decrease in the overall amplitude of the response for both groups. For the control group, the mean value of global amplitude suppression was 0.87 dB (SD=0.29) for the right and 1.25 dB (SD=0.49) for the left ear. The mild group was 1.36 dB (SD=0.41) for the right and 0.88dB (SD=0.30) for the left ear. The moderate to severe group had 1.25 dB (SD=1.23) for the right and 1.43 dB (SD=1.32) for the left ear.

The one-way ANOVA results revealed no significant differences in global amplitude suppression between groups. For the right ear, the ANOVA result was $F_{(2,42)}$=1.12, with a p-value of 0.39. Similarly, for the left ear, the ANOVA result was $F_{(2,42)}$=1.69, with a p-value of 0.28. These findings are summarized in Table 3.

**Amplitude suppression of each frequency**

Amplitude suppression of each frequency, including 1000 Hz, 1414 Hz, 2000 Hz, 2828 Hz, and 4000 Hz, was analyzed separately for both ears. The result analysis showed the presence of contralateral suppression for all the frequencies analyzed for all the groups.

Table 3 illustrates the standard deviation and mean of each frequency amplitude suppression. The one-way ANOVA revealed no significant differences in amplitude suppression across groups at all frequencies, including 1000 Hz, 1414 Hz, 2000 Hz, 2828 Hz, and 4000 Hz, with a p-value larger than 0.05. Table 3 provides the detailed mean and standard deviation values of the amplitude suppression for each frequency and the ANOVA findings for the left and right ear.

## Discussion

Misophonia, a disorder that has received limited attention in audiology, was the focus of our study. We employed TEOAEs with contralateral suppression to investigate the functioning of the efferent pathway in individuals with misophonia. An absence of the suppression effect or an increase in TEOAE response amplitude, when suppressors were present, indicates abnormal efferent pathway functioning. Our findings demonstrated that persons with misophonia, like the control group, have a suppressing effect. There was no significant difference in suppression value between all test frequencies between groups. This finding implies that misophonia patients' medial olivocochlear bundle efferent path functions normally. Our study replicates findings reported by Suraj et al. [30] reported normal efferent pathway functioning among individuals with misophonia. However, they assessed the linear and non-linear processes of the cochlea using both TEOAEs and DPOAEs.

Misophonia can manifest independently or in conjunction with other auditory disorders, including tinnitus, hyperacusis, and phonophobia. Several studies have evaluated the functioning of the medial olivocochlear bundle efferent pathway in tinnitus. Most of the studies done across the literature have reported abnormal functioning of the efferent pathway in individuals with tinnitus [31-33]. Similarly, studies on hyperacusis have also reported abnormal functioning of the auditory efferent pathway in individuals with misophonia [32]. The differences in the findings of misophonia compared to tinnitus and hyperacusis suggest that the pathophysiological mechanism behind these auditory disorders is different, and we need to diagnose these disorders differently.

Several studies in the existing literature have extensively employed fMRI to examine the functioning of the auditory nervous and limbic systems concerning misophonia. These investigations have consistently revealed abnormal activation patterns in ascending auditory cortical regions. Specifically, fMRI studies have demonstrated atypical activity in the central auditory nervous system and limbic system, suggesting their involvement in the processing and perception of misophonic triggers. However, as per our knowledge none of the fMRI studies has shown abnormal processing of the efferent pathway.These findings provide valuable insights into the neurobiological basis of misophonia and shed light on the underlying mechanisms associated with the condition [34-36]. According to our findings, individuals with misophonia exhibit normal processing of descending medial olivocochlear bundle pathways. The efferent auditory pathways are responsible for modulating and regulating the transmission of information from the brain to the auditory periphery. In misophonia, our research suggests that medial olivocochlear bundle pathways function within the expected range and do not show any significant abnormalities or disruptions. The findings of our study using electroacoustic measures align with the findings using neurophysiological measures.

There werte some limitations of the study, the sample size in our study was limited, which may have introduced bias and affected the generalizability of the findings. Therefore, future studies should aim to include larger sample sizes in each group to ensure more reliable and representative results.

## Conclusion

Misophonia has received limited attention in audiological research. In our study, we sought to assess the function of the efferent pathways by administering Transient Evoked Otoacousic Emissions (TEOAEs) with contralateral suppression to individuals with misophonia. Our findings suggest that the medial olivocochlear bundle efferent pathway, which regulates auditory responses, functions normally in misophonia patients. However, further research is necessary to validate these results with a larger sample size and diverse population.

The outcomes of our study serve as a foundational step for audiologists and researchers working in the field of misophonia, providing a starting point to investigate the neurophysiology of the disorder from an audiological perspective. Furthermore, the rostral part of the auditory efferent pathway could not be evaluated using TEOAEs. Additional research to evaluate rostral parts of efferent systems using electrophysiological measures will contribute to a more comprehensive understanding of misophonia and its underlying mechanisms.

## Data availability statement

The datasets utilized in this study can be obtained by making a reasonable request to the corresponding author.

## Ethical Considerations

## Compliance with ethical guidelines

The research protocol underwent evaluation by the institutional review board. It received ethical approval with SH/ERB/2022-24/37.

## Funding

The study was funded by So Quiet Misophonia Advocacy Non-profit Organization

## Authors' contributions

SA: Concept development, study design, stimulus preparation, analysis of the results, interpretation, and writing the manuscript; PP: Concept development, study design, stimulus preparation, and writing the manuscript.

## Conflict of interest

There is no conflict of interest to disclose.

## Acknowledgments

The authors acknowledge the director of All India Institute of Speech and Hearing for allowing to carry out this study. The authors acknowledge the participants for their cooperation.

**References**

1. Swedo SE, Baguley DM, Denys D, Dixon LJ, Erfanian M, Fioretti A, et al. Consensus Definition of Misophonia: A Delphi Study. Front Neurosci. 2022;16:841816. [DOI:10.3389/fnins.2022.841816]
2. Jastreboff PJ, Jastreboff MM. Decreased sound tolerance: hyperacusis, misophonia, diplacousis, and polyacousis. Handb Clin Neurol. 2015;129:375-87. [DOI:10.1016/B978-0-444-62630-1.00021-4]
3. Aryal S, Prabhu P. Misophonia: Prevalence, impact and co-morbidity among Mysore University students in India - A survey. Neuroscience Research Notes. 2022;5(4):161. [DOI:10.31117/neuroscirn.v5i4.161]
4. Naylor J, Caimino C, Scutt P, Hoare DJ, Baguley DM. The Prevalence and Severity of Misophonia in a UK Undergraduate Medical Student Population and Validation of the Amsterdam Misophonia Scale. Psychiatr Q. 2021;92(2):609-19. [DOI:10.1007/s11126-020-09825-3]
5. Aryal S, Prabhu P. Awareness and perspectives of audiologists on assessment and management of misophonia in India. J Otol. 2023;18(2):104-10. [DOI:10.1016/j.joto.2023.02.003]
6. Baguley DM. Hyperacusis. J R Soc Med. 2003;96(12):582-5. [DOI:10.1177/014107680309601203]
7. Henry JA, Theodoroff SM, Edmonds C, Martinez I, Myers PJ, Zaugg TL, et al. Sound Tolerance Conditions (Hyperacusis, Misophonia, Noise Sensitivity, and Phonophobia): Definitions and Clinical Management. Am J Audiol. 2022;31(3):513-27. [DOI:10.1044/2022_AJA-22-00035]
8. Aryal S, Bhattarai B, Prabhu P. Development and standardization of Morningness- Eveningness questionnaire (MEQ) in the Nepali language. Biol Rhythm Res. 2022;53(11):1692-701. [DOI:10.1080/09291016.2021.2010968]
9. Dozier TH, Morrison KL. Phenomenology of Misophonia: Initial Physical and Emotional Responses. The American Journal of Psychology. 2017;130(4):431-8. [DOI:10.5406/amerjpsyc.130.4.0431]
10. Aryal S, Prabhu P. Auditory brainstem functioning in individuals with misophonia. J Otol. 2023;18(3):139-45. [DOI:10.1016/j.joto.2023.05.006]
11. Grossini E, Stecco A, Gramaglia C, De Zanet D, Cantello R, Gori B, et al. Misophonia: Analysis of the neuroanatomic patterns at the basis of psychiatric symptoms and changes of the orthosympathetic/ parasympathetic balance. Front Neurosci. 2022;16:827998. [DOI:10.3389/fnins.2022.827998]
12. Danesh A, Aazh H. Misophonia: A Neurologic, Psychologic, and Audiologic Complex. Hear J. 2020;73(3):20,22,23. [DOI:10.1097/01.HJ.0000657984.74790.d5]
13. Schröder A, van Diepen R, Mazaheri A, Petropoulos-Petalas D, Soto de Amesti V, Vulink N, et al. Diminished n1 auditory evoked potentials to oddball stimuli in misophonia patients. Front Behav Neurosci. 2014;8:123. [DOI:10.3389/fnbeh.2014.00123]
14. Schröder A, San Giorgi R, Van Wingen G, Vulink N, Denys D. P.1.i.015 Impulsive aggression in misophonia: results from a functional magnetic resonance imaging study. European Neuropsychopharmacology. 2015;25(Suppl 2):S307-8. [DOI: 10.1016/S0924-977X(15)30374-6]
15. Webb J. β-Blockers for the Treatment of Misophonia and Misokinesia. Clin Neuropharmacol. 2022;45(1):13-4. [DOI:10.1097/WNF.0000000000000492]
16. Aryal S, Prabhu P. Understanding misophonia from an audiological perspective: a systematic review. Eur Arch Otorhinolaryngol. 2023;280(4):1529-45. [DOI:10.1007/s00405-022-07774-0]
17. Guinan JJ Jr. Olivocochlear efferents: anatomy, physiology, function, and the measurement of efferent effects in humans. Ear Hear. 2006;27(6):589-607. [DOI:10.1097/01.aud.0000240507.83072.e7]
18. Ciuman RR. The efferent system or olivocochlear function bundle - fine regulator and protector of hearing perception. Int J Biomed Sci. 2010;6(4):276-88.
19. Probst R, Harris FP. A comparison of transiently evoked and distortion-product otoacoustic emissions in humans. Prog Brain Res. 1993;97:91-9. [DOI:10.1016/s0079-6123(08)62266-9]
20. Sturm JJ, Weisz CJ. Hyperactivity in the medial olivocochlear efferent system is a common feature of tinnitus and hyperacusis in humans. J Neurophysiol. 2015;114(5):2551-4. [DOI:10.1152/jn.00948.2014]
21. Tayade A, Tucker D. Evaluation of Medial Olivocochlear Neural Efferent Pathway in Tinnitus Perception in Normal-hearing Individuals. Int Tinnitus J. 2022;26(1):20-6. [DOI:10.5935/0946-5448.20220004]
22. Jager I, de Koning P, Bost T, Denys D, Vulink N. Misophonia: Phenomenology, comorbidity and demographics in a large sample. PLoS One. 2020;15(4):e0231390. [DOI:10.1371/journal.pone.0231390]
23. Schröder A, Vulink N, Denys D. Misophonia: diagnostic criteria for a new psychiatric disorder. PLoS One. 2013;8(1):e54706. [DOI:10.1371/journal.pone.0054706]
24. Zacharia T, Naik PV, Sada S, Kuniyil JG, Dwarakanath VM. Development and standardization of tinnitus handicap inventory in Kannada. Int Tinnitus J. 2012;17(2):117-23. [DOI:10.5935/0946-5448.20120022]
25. Siepsiak M, Rosenthal MZ, Raj-Koziak D, Dragan W. Psychiatric and audiologic features of misophonia: Use of a clinical control group with auditory over-responsivity. J Psychosom Res. 2022;156:110777. [DOI:10.1016/j.jpsychores.2022.110777]
26. Frank T. ANSI Update: Specification of Audiometers. Am J Audiol. 1997;6(3):29-32. [DOI:10.1044/1059-0889.0603.29]
27. Davis H, Kranz Fw. The international standard reference zero for pure-tone audiometers and its relation to the evaluation on impairment of hearing. J Speech Hear Res. 1964;7:7-16. [DOI:10.1044/jshr.0701.07]
28. Berlin CI, Hood LJ, Wen H, Szabo P, Cecola RP, Rigby P, et al. Contralateral suppression of non-linear click-evoked otoacoustic emissions. Hear Res. 1993;71(1-2):1-11. [DOI:10.1016/0378-5955(93)90015-s]

29. De Ceulaer G, Yperman M, Daemers K, Van Driessche K, Somers T, Offeciers FE, et al. Contralateral suppression of transient evoked otoacoustic emissions: normative data for a clinical test set-up. Otol Neurotol. 2001;22(3):350-5. [DOI:10.1097/00129492-200105000-00013]
30. Suraj U, Nisha KV, Prabhu P. Normal linear and non-linear cochlear mechanisms and efferent system functioning in individuals with misophonia. Eur Arch Otorhinolaryngol. 2023. [DOI:10.1007/s00405-023-08273-6]
31. Attias J, Bresloff I, Furman V. The influence of the efferent auditory system on otoacoustic emissions in noise induced tinnitus: clinical relevance. Acta Otolaryngol. 1996;116(4):534-9. [DOI:10.3109/00016489609137885]
32. Attias J, Zwecker-Lazar I, Nageris B, Keren O, Groswasser Z. Dysfunction of the auditory efferent system in patients with traumatic brain injuries with tinnitus and hyperacusis. J Basic Clin Physiol Pharmacol. 2005;16(2-3):117-26. [DOI:10.1515/jbcpp.2005.16.2-3.117]
33. Riga M, Papadas T, Werner JA, Dalchow CV. A clinical study of the efferent auditory system in patients with normal hearing who have acute tinnitus. Otol Neurotol. 2007;28(2):185-90. [DOI:10.1097/MAO.0b013e31802e2a14]
34. Kumar S, Dheerendra P, Erfanian M, Benzaquén E, Sedley W, Gander PE, et al. The Motor Basis for Misophonia. J Neurosci. 2021;41(26):5762-70. [DOI:10.1523/JNEUROSCI.0261-21.2021]
35. Kumar S, Tansley-Hancock O, Sedley W, Winston JS, Callaghan MF, Allen M, et al. The Brain Basis for Misophonia. Curr Biol. 2017;27(4):527-33. [DOI:10.1016/j.cub.2016.12.048]
36. Schröder A, van Wingen G, Eijsker N, San Giorgi R, Vulink NC, Turbyne C, Denys D. Misophonia is associated with altered brain activity in the auditory cortex and salience network. Sci Rep. 2019;9(1):7542. [DOI:10.1038/s41598-019-44084-8]

Table 1. Misophonia characteristics of all the participants included in the study (n=30)

| | Mild group | | | | Moderate-severe group | | | |
|---|---|---|---|---|---|---|---|---|
| | Revised amsterdam misophonia scale | | Misophonia characteristics | | Revised amsterdam misophonia scale | | Misophonia characteristics | |
| **SN** | **Score** | **Severity** | **Onset** | **Duration (years)** | **Score** | **Severity** | **Onset** | **Duration (years)** |
| **1** | 12 | Mild | Sudden | 3 | 21 | Moderate-severe | Gradual | 4 |
| **2** | 13 | Mild | Gradual | 4 | 24 | Moderate-severe | Gradual | 3 |
| **3** | 17 | Mild | Gradual | 6 | 23 | Moderate-severe | Gradual | 5 |
| **4** | 19 | Mild | Gradual | 7 | 24 | Moderate-severe | Gradual | 4 |
| **5** | 19 | Mild | Gradual | 6 | 32 | Severe-extreme | Gradual | 8 |
| **6** | 15 | Mild | Gradual | 4 | 31 | Severe-extreme | Gradual | 6 |
| **7** | 19 | Mild | Sudden | 3 | 35 | Severe-extreme | Gradual | 6 |
| **8** | 17 | Mild | Gradual | 6 | 33 | Severe-extreme | Gradual | 3 |
| **9** | 18 | Mild | Gradual | 6 | 31 | Severe-extreme | Gradual | 4 |
| **10** | 11 | Mild | Gradual | 8 | 22 | Moderate-severe | Gradual | 6 |
| **11** | 12 | Mild | Gradual | 5 | 22 | Moderate-severe | Gradual | 7 |
| **12** | 16 | Mild | Gradual | 4 | 21 | Moderate-severe | Gradual | 6 |
| **13** | 14 | Mild | Gradual | 5 | 22 | Moderate-severe | Gradual | 4 |
| **14** | 19 | Mild | Sudden | 3 | 22 | Moderate-severe | Sudden | 3 |
| **15** | 18 | Mild | Gradual | 4 | 25 | Moderate-severe | Gradual | 4 |

SN; serial number

Table 2. Pure tone average of all the participants included in the study (n=45)

| | Control group | | Mild group | | Moderate-severe group | |
|---|---|---|---|---|---|---|
| **SN** | **Right ear (dB HL)** | **Left ear (dB HL)** | **Right ear (dB HL)** | **Left ear (dB HL)** | **Right ear (dB HL)** | **Left ear (dB HL)** |
| **1** | 0 | 0 | 8.75 | 7.5 | 18.75 | 13.75 |
| **2** | 10 | 6.5 | 8.75 | 2.5 | 10 | 11.25 |
| **3** | 6.67 | 6.5 | 6.25 | 8.75 | 15 | 10 |
| **4** | 6.25 | 10 | 0 | 3.75 | 2.5 | 12.5 |
| **5** | 2.5 | 1.25 | 0 | 1.25 | 11.25 | 6.25 |
| **6** | 1.25 | 3.25 | 10 | 10 | 2.5 | 6.25 |
| **7** | 5 | 7.5 | 11.25 | 11.25 | 15 | 8.75 |
| **8** | 7.5 | 6 | 1.25 | 5 | 1.25 | 2.5 |
| **9** | 10 | 6 | 13.75 | 11.25 | 8.75 | 10 |
| **10** | 3.75 | 0 | 7.5 | 1.25 | -1.225 | 5 |
| **11** | 5 | 6.67 | 3.75 | 8.75 | 2.5 | 2.5 |
| **12** | 8.75 | 10 | 5 | 3.75 | 6.25 | 7.5 |
| **13** | 8.75 | 5 | 30 | 15 | 18.75 | 11.25 |
| **14** | 7.5 | 5 | 10 | 1.25 | 11.25 | 6.25 |
| **15** | 7.5 | 0 | 0 | 0 | 2.5 | 2.5 |
| **Mean(SD)** | 6.03(SD=3.07) | 4.91(SD=3.35) | 7.75 (SD=7.57) | 6.08(SD=4.60) | 8.33(SD= 6.59) | 7.75(SD=3.69) |

SN; serial number

Table 3. Result of a one-way analysis of variance to examine group differences in the mean amplitude suppression values across various frequencies for both the right and left ears, (n=45)

| | Mean(SD) amplitude suppression | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Global | | 1 kHz | | 1.414 kHz | | 2 kHz | | 2.828 kHz | | 4 kHz | |
| **Group** | **Right ear** | **Left ear** | **Right ear** | **Left ear** | **Right ear** | **Left ear** | **Right ear** | **Left ear** | **Right ear** | **Left ear** | **Right ear** | **Left ear** |
| **Control (n=15)** | 0.87(0.29) | 1.25 (0.49) | .89(0.35) | 2.77(0.39) | 1.04(0.20) | 1.34(0.16) | 1.09(0.22) | 1.08(0.79) | .86(0.36) | 1.02(0.47) | 0.48(0.29) | 0.82(0.66) |
| **Mild (n=15)** | 1.36(0.41) | 0.88(0.30) | 1.79(0.44) | 0.67(0.16) | 1.76(0.50) | 1.04(0.25) | 0.83(0.34) | 0.55(0.17) | 0.97(0.47) | 0.54(0.48) | 1.47(0.29) | 0.60(0.43) |
| **Moderate-severe (n=15)** | 1.25(0.23) | 1.43(0.32) | 1.27(0.15) | 1.75(0.39) | 1.03(0.21) | 1.29(0.51) | 1.07(0.12) | 0.78(0.29) | 1.59(0.49) | 0.95(0.19) | 1.29(0.16) | 0.57(0.24) |
| **$F_{(2,42)}$** | 1.12 | 1.69 | 0.89 | 3.79 | 0.91 | 0.45 | .36 | 1.99 | 2.32 | 1.18 | 1.15 | 1.01 |
| **p** | 0.391 | 0.282 | 0.421 | 0.054 | 0.415 | 0.643 | 0.704 | 0.156 | 0.112 | 0.173 | 0.336 | 0.374 |