AUDITORY SENSORY GATING IN MISOPHONIA: A NEUROPHYSIOLICAL STUDY

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This Dissertation is submitted as part of fulfilment for the Degree of Master of Science in Audiology

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June, 2026

CERTIFICATE

This is to certify that this dissertation entitled **"Auditory Sensory gating in misophonia: A neurophysiological study"** is a bonafide work submitted as part of fulfilment of the degree of Master of Science (Audiology) of the student with Registration Number **P01II24S023021**. This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award of any other Diploma or Degree.

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DECLARATION

This is to certify that this dissertation entitled "'**Auditory sensory gating in misophonia: A neurophysiological study** " is the result of my own study under the guidance of Dr. Prashanth Prabhu., Assistant Professor of Audiology, All India Institute of Speech and Hearing, Mysuru and has not been submitted earlier to any other university for the award of any other Diploma or Degree.

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CERTIFICATE

This is to certify that this dissertation entitled "'**Auditory sensory gating in misophonia: A neurophysiological study** " has been prepared under my supervision and guidance. It is also being certified that this dissertation has not been submitted earlier to any other university for the award of any other Diploma or Degree.

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ACKNOWLEDGMENT

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Abstract

Strong emotional and physical responses to particular repeating noises are the hallmark of misophonia. There is still little objective neurophysiological evidence to support the theory that impaired auditory processing is the underlying mechanism. The purpose of this study was to assess auditory sensory gating in misophonia sufferers and investigate its connection to the intensity of symptoms. The objectives of this study are to evaluate auditory sensory gating in individuals with and without misophonia by comparing amplitude and latency differences of P1, N1, and P2 components between conditioning (S1) and test (S2) stimuli. It also aims to calculate gating ratios for these components and examine correlations between Revised Amsterdam Misophonia Scale (RAMISO-S) scores and sensory gating measures, thereby identifying potential neurophysiological markers of misophonia. The study adopts a group comparison design including 16 individuals with misophonia and 16 controls aged 18–35. Participants will undergo routine audiological evaluation, misophonia severity assessment using RAMISO-S, and exclusion of tinnitus or hyperacusis. Auditory sensory gating will be measured using a conditioning-testing paradigm with cortical evoked potentials (P1, N1, P2) recorded and analyzed for amplitude, latency, and gating ratios. Individuals with misophonia demonstrated significantly reduced or absent auditory sensory gating, particularly reflected in diminished suppression of the P1, N1 and P2 component to test auditory stimuli. Gating ratios were abnormal indicating an impaired ability to filter out repetitive sounds, and higher misophonia severity correlated with weaker gating. These results suggest that people with misophonia have an early inhibitory processing deficit specific to repetitive auditory information, marking altered neurophysiological auditory functioning in this population. The study concludes that evaluating auditory sensory gating in misophonia may reveal distinct neurophysiological abnormalities, offering potential objective markers for diagnosis. Findings can enhance understanding of altered auditory processing in misophonia and provide a foundation for future research into its mechanisms and clinical management

Keywords: Misophonia, Auditory Sensory Gating, Cortical Auditory Evoked Potentials, P1, N1, P2, RAMISO-S, Inhibitory Processing, Auditory Processing, Neurophysiology.

CHAPTER 1

INTRODUCTION

Misophonia is a disorder where a person experiences a negative emotional or physiological reaction to certain sounds. (Jastreboff & Jastreboff, 2001). The person's reaction to sound depends upon the physical characteristics of sound and the environment in which it is presented. Physiological and emotional responses include accelerated heart rate, sweating, anxiety, rage, irritability, and disgust (Jastreboff & Jastreboff, 2001). A recently identified disorder called misophonia is typified by a dislike of common human noises. (Schröder et al., 2014). The responses can be intense and distressing. Physiological responses can include increased heart rate and sweating, while emotional reactions often include anxiety, rage, and feelings of disgust. The responses affect the daily living and social interaction (Jastreboff & Jastreboff, 2001).

Although it is a newly defined clinical condition, it is characterized by a dislike of human sound. Schröder et al. (2014) emphasize that the condition lies just beyond annoyance and reflects sensitivity to specific auditory triggers. Triggering factors for misophonia vary from person to person. Some individuals have difficulty with soft sounds, such as chewing, but do not have a problem with loud music. For some individuals, misophonia occurs in certain environments. However, it may not be present in other environments (e.g., annoyance with chewing sounds only at home, not in public places). A systematic evaluation was carried out by Gowda and Prabhu (2024) to determine the prevalence of misophonia in adults and adolescents worldwide. Their primary objective was to raise awareness of the condition and provide a consolidated reference point for researchers by synthesizing data across various countries. In line with these findings, a recent cross-sectional study by Küçükkelepçe et al. (2026) examined misophonia among high school students in Turkey. The study's goal was to ascertain the prevalence of

misophonia among high school students in Turkey. The results showed that there was a notably high prevalence of clinically significant misophonia among high school students in Turkey (49.7%). The higher misophonia scores were essentially linked to high exam stress, poor parent-child relationships, and poor general health. A further study was conducted to determine the frequency of misophonia among Saudi Arabian medical students and to identify related issues including depression and obsessive-compulsive disorder. (Almadani et al., 2024). The result of the study indicated prevalence of misophonia was 42.32% which was based on the AMISO scoring, in which 71.16% had subclinical misophonia and 28.84% had clinical misophonia which was ranging from mild to moderate and with respect to depression and the OCD finding 33.15% participants showed depression and 31% of the students likely showed OCD, Therefore, conducting further research on misophonia is important to improve understanding of its prevalence, underlying mechanisms, and associated psychological comorbidities, which will help in early identification, proper diagnosis, and development of effective management strategies to enhance quality of life. A similar study was conducted on the prevalence and characteristics of misophonia in Ankara, Turkey. A population-based study by Kılıç et al. (2021) aims to check the prevalence of misophonia in relation to the clinical and demographic variables in a large population sample. The sample comprised 541 participants aged 15 or older, selected randomly from Ankara city center, Turkey. All participants were assessed using a semi-structured interview, and the study found a prevalence of 12.8% for misophonia.

Patel et al. (2023) Conducted the prevalence study in the collage going student in India, the study basically explore the prevalence across the different age groups, location, socio-economic status and communities, they basically recruited 328 undergraduate student all over India, it was conducted with the help of online survey through the google forms, wherein the participants were asked to fill the self-rated Misophonia Questionnaire

and Amsterdam Misophonia Scale. According to the study's findings, 15.85% of Indian college students had moderate to severe misophonia. As a result, the study found that misophonia is very common in India. However, studies were available from only seven nations: China, Germany, India, Iran, Turkey, the United States, and the United Kingdom. Reported prevalence rates of clinically significant misophonia ranged from 5% to 34.67%. The authors highlighted the urgent need for increased awareness, the development of standardized diagnostic tools, and further research to enhance understanding and clinical management of misophonia. Notably, the review found no significant gender differences in misophonia prevalence. The relatively high prevalence observed in India underscores the need for further research and public education initiatives to support timely diagnosis and effective intervention. The authors urged future research for increased awareness for better understand the assessment of misophonia; no significant gender differences were observed. The finding of misophonia suggests an underlying abnormality in auditory processing and sensory regulation mechanisms. One such mechanism is sensory gating, the brain inherent ability to selectively process needed stimuli while suppressing irrelevant or redundant sensory input. This inhibitory mechanism is essential for accurate perception and efficient cognitive functioning (Bruner, 1957). In healthy individuals, sensory gating helps in basically filtering out the task-irrelevant information and selecting the task-relevant information for processing, and this phenomenon can be assessed by the multiple testing paradigms (Gjini et al., 2010; Hu et al., 2012; Mayer et al., 2009; Todorovic et al., 2011; Zouridakis & Boutros, 1992).

1.1 NEED FOR THE STUDY

Many studies investigated the individuals with sensory gating having

tinnitus, hearing loss, traumatic brain injury, bipolar disorder, and schizophrenia, among others (Arciniegas et al., 2000; Campbell et al., 2018; Campbell et al., 2020a; Lijffijt et al., 2009; Nagamoto et al., 1989). Notably, the magnitude of sensory gating correlates with symptom severity in these pathologies (Campbell et al., 2018; Smith et al., 2013). For instance, Campbell, Nielsen, LaBrec, and Bean (2020b) reported reduced gating of the P2 component of CAEP in individuals with deficits in speech perception in noise.

A similar reduction in P2 sensory gating responses was also observed in individuals with hearing impairment (Campbell et al., 2020a).

Due to absence for definitive neurophysiological marker for misophonia, Schröder et al. (2014) investigated whether the disorder involves deficits in early auditory processing. Using an oddball paradigm with pure tones, event-related potentials (ERPs) were recorded from 20 individuals with misophonia and 14 controls. While no significant differences observed for P1 or P2 components or responses to standard tones, misophonia participants exhibited significantly reduced N1 amplitudes to deviant tones. This attenuation of N1 suggests an underlying neurobiological dysfunction and may reflect impaired early Misophonia sufferers' auditory processing. From an audiological perspective, Auditory late latency responses (ALLRs) in misophonia were compared to normals by Aryal and Prabhu (2024). The misophonia population exhibits significantly earlier latencies of the P1 and N1 components, according to the results., particularly among individuals with moderate to severe symptoms. These results suggest altered cortical auditory processing in misophonia, emphasizing the potential of ALLR as an objective tool for evaluating the condition.

Similarly, Karupaiah et al. (2025) employed multichannel Auditory Late Latency Responses (ALLR) to examine auditory cortex processing in people with misophonia. To identify underlying neurophysiological differences independent of emotional reactions, the study used non-trigger auditory stimuli to check P1, N1, and P2 latencies, amplitudes, and scalp topographies in persons with misophonia and healthy controls. The findings demonstrated that people with misophonia had lower N1 amplitude throughout different sites of electrode and significantly earlier latencies for P1, N1, and P2, suggesting modified early auditory cortex processing. A neurophysiological basis for misophonia was supported by scalp topography, which showed aberrant centro-parietal activity for misophonia populaions in contrast to fronto-central activation in controls. Patro et al. (2025) conducted a study using an oddball paradigm to check neurophysiological, perceptual, cognitive correlates in controls and misophonia . While P1 and P2 amplitudes, as well as behavioral performance, were similar between the two groups, individuals with misophonia showed significantly less N1 and N2 amplitudes in response to deviant tones. These findings suggest that although higher-order attentional and cognitive control mechanisms may remain intact, misophonia is associated with a selective dysfunction in early auditory cortical processing. This is consistent with deficits in mechanisms related to mismatch negativity (MMN), further supporting the of altered gating in the pathophysiology of misophonia.

According to Lodol (2024), SGI-B scores, or self-reported sensory gating issues were significantly correlated with misophonia severity. This suggests that individuals exhibiting heightened sound sensitivity may also experience greater difficulty in filtering out irrelevant sensory information. Objective electrophysiological measures of sensory gating showed abnormal sensory gating and were associated with misophonia severity. The study concluded that individuals with misophonia may exhibit abnormalities in sensory gating, highlighting the need for further research that employs standardized and detailed

measurement of auditory sensory gating in this population. Such investigations could help identify objective diagnostic criteria for misophonia and shed light on the neurophysiological mechanisms that underlie the condition. Further exploration of sensory gating is therefore warranted, as it may also advance our understanding of the sensory processing characteristics associated with misophonia.

Strong negative reactions to specific trigger sounds are a defining feature of misophonia, yet the underlying brain mechanisms responsible for these reactions remain poorly understood. The brain's ability to filter out irrelevant auditory information, commonly referred to as sensory gating, may be a key factor in explaining the heightened sound sensitivity seen in individuals with misophonia. Most existing assessments, however, rely heavily on subjective self-report measures, which offer limited insight into the neurophysiological basis of the condition.

Further research is needed to clarify the neurophysiological mechanisms of sensory gating in individuals with misophonia. According to the neuroaudiological model, abnormal connectivity between auditory pathways and the limbic and autonomic systems gives rise to intense emotional reactions to specific trigger sounds. Impaired sensory gating, reflecting a reduced capacity to inhibit repetitive or non-salient auditory input, may represent a central deficit within this framework. Examining sensory gating therefore offers an objective means of evaluating this hypothesis and determining whether early auditory inhibitory processing is compromised. Identifying abnormalities in sensory gating may help distinguish misophonia from other sound intolerance disorders, improve diagnostic precision, and ultimately support the development of targeted, evidence-based treatment approaches.

1.2 AIM OF THE STUDY

The purpose of the study is to compare the auditory sensory gating abilities of people with and without misophonia..

1.3 OBJECTIVES OF THE STUDY

- To contrast the amplitude difference of P1, N1, P2 of the LLR between conditioning (S1) and test stimuli (S2) in People with and without misophonia
- To compare latency difference of P1, N1, P2 of the LLR between S1 and S2 in people with and without misophonia
- To compare gating ratio of amplitude and latency for P1, N1, P2 of S1 and S2 in individuals with and without Misophonia
- To assess the relationship between Revised Amsterdam Misophonia Scale (RAMISO S) and the gating difference and gating ratio (amplitude and latency) of the LLR's P1, N1, P2 components in people with and without misophonia

CHAPTER 2

REVIEW OF LITERATURE

2.1 Definition of misophonia

Misophonia was first described as an unusually intense distaste or loathing of particular sound stimuli with unique patterns or meanings, resulting from aberrant limbic and autonomic nervous system activation in people with normal hearing. The physical characteristics of auditory stimuli, such as intensity or pitch, appeared to be less relevant in individuals with misophonia, as misophonia sufferers did not show a relationship between the intensity of the trigger stimulus and the reflexive responses (Jastreboff & Jastreboff, 2002, 2014, 2015). Misophonia could be interpreted by laypeople as a severe hatred of specific sounds, such as chewing. Misophonia is usually present with issues in the specific sensory input, which leads to a strong emotional response to specific sounds (Swedo et al., 2022)

2.2 Prevalence of misophonia

The MQ Severity Scale criterion for clinical misophonia symptoms was met or exceeded by 5% of the sample, according to the data (5.9% based on AMISOS-R). Misophonia is a prevalent condition that requires independent audiological examination. The prevalence of misophBetween December 2020 and March 2021, a comprehensive prevalence investigation was conducted in Germany. The Amsterdam Misophonia Questionnaire (AMISOS-R) and the Misophonia Questionnaire (MQ) were used to assess a total sample size of 2,519 individuals. According to the findings, 5% of the sample had clinical misophonia symptoms that were equal to or higher than the MQ Severity Scale criteria (5.9% baA comprehensive analysis of the prevalence of misophonia in adults and adolescents was conducted. According to studies, the prevalence of the condition is

influenced by age, gender, and socioeconomic level. In order to obtain thorough data, the study intends to examine twelve English-language papers that are accessible online. According to the study's findings, the stated worldwide prevalence varies from 5% to 34.67%, depending on the population and the assessment tools used. (Gowda et al, 2024)This study attempts to determine the incidence of misophonia among college-bound students in India; a related study looked at the prevalence of college-going students in India. 328 undergraduate students from various language and cultural backgrounds were questioned throughout India. Participants were requested to complete the Misophonia This study attempts to determine the incidence of misophonia among Indian college-bound students; a related study look Participants were requested to fill out the Misophonia Questionnaire and the Self-Rating Amsterdam Misophonia Scale as part of the Google Forms survey. According to the findings, the prevalence of moderate to severe misophonia was almost 15.85 percent. In India, misophonia was very common and there was no gender domination (Patel et al., 2023). A study was based on the prevalence and relationship with the trigger sound mimicry in misophonia, The main goal of study was to comprehend the severity in misophonia and the tendency to mimic as the function of the misophonia, here a set of 676 participant was taken in which 505 was female and 166 were males and aged between 18-81 years (Ash et al. (2024). The results showed that the prevalence of mimicry was roughly 46.7%. The materials were essentially misophonia sets of questionnaires generated using Google forms, and an online link to these was made available on internet websites/social media or by email.

2.3 Etiology and pathophysiology of misophonia.

Actual cause of misophonia still didn’t clearly understand, and research on this condition remains in its initial stages. Although several theories have been proposed, no definitive explanation has been confirmed so far. Early descriptions linked misophonia to the

neurophysiological model of tinnitus, proposing that it may result from enhanced interactions among the auditory, limbic, and autonomic nervous systems (Jastreboff, 1990; Jastreboff & Hazell, 2004; Jastreboff & Jastreboff, 2015). Existing literature suggests that researchers have attempted to explain the etiology and underlying mechanisms of misophonia through various perspectives, including conditioned reflex responses, abnormal brain activity in response to trigger sounds as demonstrated by imaging studies and event-related auditory evoked potentials, and theoretical models describing its pathophysiological processes.

2.3.1 Genetic links to misophonia

According to Smit et al. (2023), they conducted a study based on the age-aspect misophonia symptoms and genetic connection to personality, mental illnesses, and audiological characteristics. In this current study, which focused on investigating genetic cuase of misophonia and understanding the genetic factors, they basically used genetic data from 23andMe (a company that provides genetic testing). They checked the genetics of misophonia-related symptoms against 43 other traits, grouped them into Audiological, psychiatric, personality, and neurological and socioeconomic traits, and conducted gene-based analysis to identify which genes are linked to misophonia symptoms. Performed the functional annotation to check how the genes affect the biological pathway. Clustering analysis was performed to group related traits based on their genetic relationships. The result showed several genes showed association with the misophonia TENM2, TMEM256, NEGR1, TFB1M, these are some of the genes which are linked to brain function, hearing processes and the neural signalling, and concluded that misophonia is not purely hearing disorder and it has some genetic links with the personality trait and the emotional/psychiatric condition, suggesting misophonia may involve emotional regulation and personality-related brain pathways, not just auditory

sensitivity.

2.3.2 amygdala's function in misophonia

Eijsker et al., (2021) conducted a study investigate whether people with misophonia shows any structural and functional brain differences compared to healthy individuals, the objectives includes to check comparision in the grey matter volume in the brain using he MRI, to investigate the resting state of functional connectivity between the brain region, they had taken 24 misophonia patients and 25 normals for the study and were matched with respect to age, sex , and educational level, The brain imaging was done basically using the structural MRI and the Resting state fMRI , 49 people were taken for the current investigation. The participants have been age, sex and educationally matched, During the experiment the participants have went Structural MRI to assess brain anatomy which helps to measure the grey matter volume content in the brain , and they assessed the resting state fMRI scan which helps in understanding the functional connectivity of the brain, basically how the brain communicate with each other, Therefore by comparing the imaging data of the control and the misophonia ,the study aimed to identify the potential biomarker as the neural correlates of the misophonia. Overall, this research contributes to the understanding of the neurobiological basis of misophonia by using advanced imaging techniques to examine both brain structure and functional communication patterns in individuals with the condition .the analysis techniques were voxel-based morphometry basically help in the identifying the differences in the grey matter volume across the brain region and used the Seed-based functional connectivity analysis to Examines connectivity of specific brain regions (here, the amygdala),and Functional Connectomics to examine connectivity across the entire brain network, The result of the study concluded misophonia patient had larger grey matter volume in the right amygdala. The amygdala is a region involved in emotional processing and threat

detection. The study concluded that misophonia patients show both structural and functional abnormalities in the brain, particularly the amygdala and the cerebellum.

2.3.3 Role of the cingulate, insular and temporal cortices in misophonia

Some studies suggest that misophonia may originate from atypical neural anatomy and abnormal physiological connectivity between different brain regions. Schröder et al. (2019) conducted an fMRI study involving 21 individuals with misophonia and 23 individuals without the condition, so goal was to identify the brain region responsible for the emotional response, they also administered Questionnaire to assess the psychological and emotional characteristic which included the Amsterdam Misophonia scale, Symptoms checklist, Hamilton Anxiety Rating scale, Hamilton depression rating scale, Buss-perry Aggression Questionnaire, these all the questionnaire assessed mainly the anger, disgust, anxiety, sadness, and happiness using a visual analogue scale after viewing the stimulus. During the fMRI scan the participants were asked to watch three types of the video which included misophonia clips(includes the eating sound, heavy breathing, typing)and aversive clip (which included violent or disgusting scenes from the movie) and the neutral clips, data analysis was done on the focused brain region which included auditory processing, emotional processing, salience network activity. The right insula, right anterior cingulate cortex, and right superior temporal cortex were the three main brain regions that showed greater activation during misophonic triggers compared to neutral stimuli, according to the results of neuroimaging analysis. Misophonia is therefore associated with aberrant activation in the salience network and auditory cortex, showing changed brain processing of trigger noises. Blood oxygen level–dependent (BOLD) imaging was performed by Kumar et al. (2017) on 20 people with misophonia and 22 control subjects without misophonia. Their findings showed that people with misophonia demonstrated significantly heightened activation of the anterior insular cortex

when exposed to trigger sounds. The primary objective of the study was to explore the neural mechanisms underlying misophonia by examining how the brain responds to trigger sounds in affected individuals. The researchers aimed to determine whether there are differences in brain activity and physiological responses between individuals with misophonia and healthy controls. Participants were presented with three categories of sounds: trigger sounds (such as eating, chewing, and breathing), unpleasant sounds (including a baby crying and screaming), and neutral sounds. Brain activity was assessed using functional MRI, while structural MRI was used to evaluate brain anatomy and tissue characteristics. Physiological responses were measured through heart rate monitoring and galvanic skin response (GSR) recordings to assess autonomic arousal. Results revealed strong activation of the anterior insular cortex in people with misophonia. Additionally, abnormal functional connectivity was observed between the anterior insula and regions such as the ventromedial prefrontal cortex, posteromedial cortex, hippocampus, and amygdala, which are associated with emotional processing and regulation. Increased heart rate was also observed, suggesting heightened autonomic nervous system activation in individuals with misophonia.

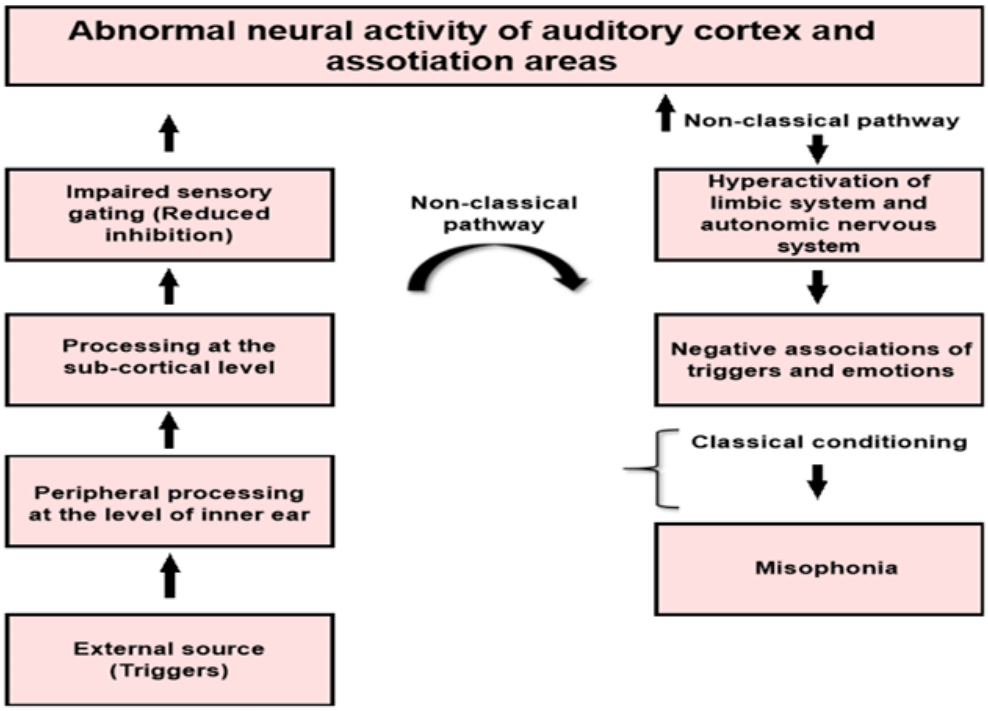
2.3.4 Models to explain the Pathophysiology of misophonia

In their thorough examination, Aryal & Prabhu (2023b) put up the neuro-audiological hypothesis of misophonia, which highlights abnormal brain activity in the auditory cortex and related areas. Furthermore, the model suggests that non-classical audiological pathways, such as hyperactivation of the autonomic nervous system and limbic system, are involved in misophonia. Through classical conditioning, these pathways cause negative emotions to be associated with trigger sounds (Figure 2.1). There are parallels between the neuro-audiological model and physiological models of hyperacusis and tinnitus. It is possible to hypothesize cochlear or efferent-based activity

parallels between tinnitus and misophonia.

Figure 2.1

Neuro-audiological model in misophonia

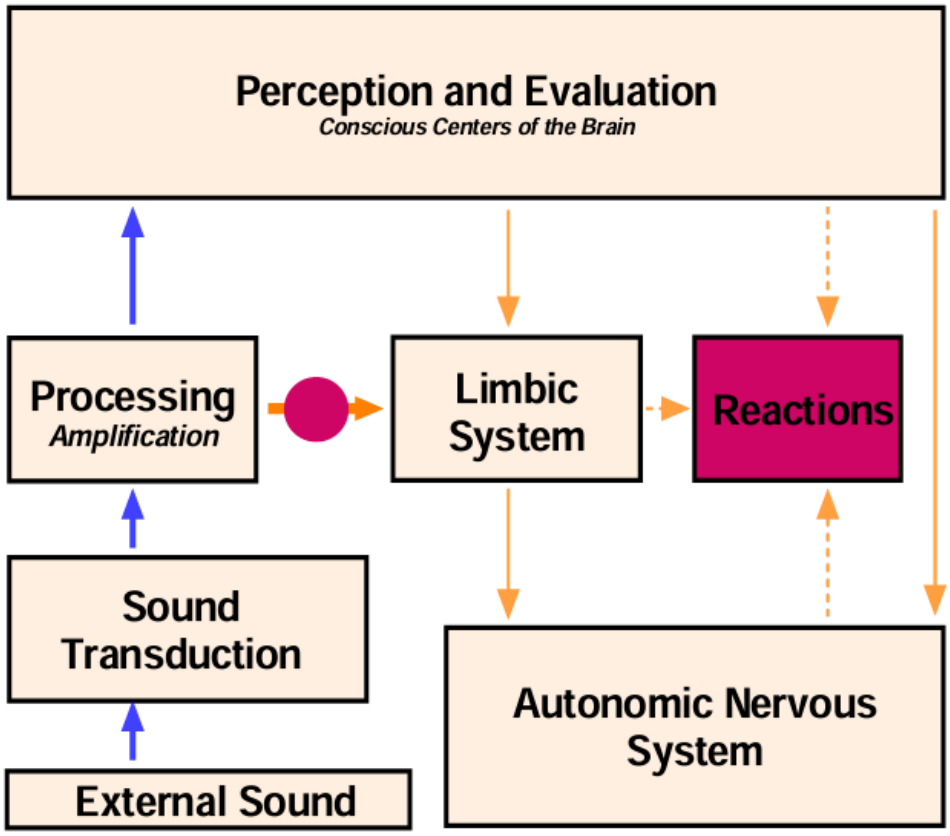


A neurophysiological explanation was proposed by Jastreboff & Jastreboff (2023) to explain misophonia. The paradigm states that responses to trigger sounds are shaped by interactions between the limbic, autonomic, subconscious, cognitive, and auditory nerve systems, with the auditory system having a small effect. The paradigm emphasizes how the auditory, limbic, and autonomic nerve systems interact with the conscious, cognitive, and subconscious minds to condition the irritation responses brought on by particular stimuli. as shown in Figure 2.2. Trigger noises increase brain activity and elicit powerful emotional responses because they acquire meaning from prior experiences. This explains why some low-intensity noises are more upsetting, especially when they come from close family members. According to the paradigm, the goal of treatment should be to disrupt

the conditioned reflex that links auditory information to cortical-limbic reactions. The concept of complex conditioned stimuli explains why specific sounds are more likely to serve as misophonic triggers, even when their intensities are lower or equal to that of other non-trigger sounds. Additionally, it sheds light on why trigger sounds produced by close family members can evoke a more pronounced distressing reaction compared to those from other individuals.

Figure 2.2

Neurophysiological model in Misophonia



Note: The thick orange arrow in the red circle represents the functional connection between the auditory, limbic and autonomic nervous systems, which is theorized to be the cause of misophonia, with normal cortical functioning and the subconscious portion of the auditory system.

2.4 Diagnosis of misophonia

According to Enzler et al. (2021), in the early years of misophonia research, assessing misophonia was very difficult. In the early stages, there was no universally accepted definition or diagnostic guideline for misophonia. Different researchers used different criteria to identify the condition. There was Absences of the objective clinical test misophonia did not have psychoacoustic or physiological diagnostic tests. Diagnosis mainly relied in the Clinical interviews and the self-report questionnaires there was limited awareness among the clinicians, there was poor understanding of the neurobiological mechanism. and the overall impact of the condition on relationships and functioning at home, school, college, and the workplace (Duddy & Oeding, 2014). Dozier et al., (2017) suggested an updated version of the diagnostic standards put forth by Schröder et al. (2013). The revised criteria indicate diagnosis of misophonia when, the anticipation or presence of misophonic triggers, which may be auditory, visual, or other sensory stimuli, can produce an intense emotional reaction such as irritation or disgust that may escalate into anger. To evaluate these symptoms, various misophonia assessment questionnaires have been developed, and the commonly used tools are presented in Table 2.1.

Table 2.1

Severity assessment of the Misophonia using various questionnaires

Sl. No	Questionnaires
1	Amsterdam Misophonia Scale (Schröder et al., 2013)
2	Amsterdam Misophonia Scale – Revised (A-MISO-S-R) (Naylor et al., 2021)
3	Misophonia Questionnaire (Wu et al., 2014)
4	Misophonia Assessment Questionnaire (Johnson et al., 2013)
5	Duke Misophonia Questionnaire (DMQ) (Rosenthal et al., 2021)
6	Selective Sound Sensitivity Syndrome Scale (S-Five) (Vitoratou et al., 2021)
7	Misophonia Response Scale (MRS) (Dibb et al., 2021)
8	MisoQuest (Siepsiak et al., 2020)
9	Berlin Misophonia Questionnaire – Revised (BMQ-R) (Remmert et al., 2022)
10	Sussex Misophonia Scale for Adolescents (SMS-A) (Rinaldi et al., 2022)

CHAPTER 3

METHODS

3.1 Study Design and Ethical Guidelines

Convenience sampling was used to select participants from the university campus and the All India Institute of Speech and Hearing (AIISH), Mysore's Audiology Outpatient Department. The AIISH Institutional Ethics Committee accepted the study methodology, and all procedures were carried out in compliance with the Declaration of Helsinki's guiding principles. People with misophonia and a control group without misophonia were included in a typical two-group comparison design (Orlikoff et al., 2014). Prior to enrollment, each subject provided written informed permission.

3.2 Participants

The study recruited 30 participants aged 18–35 years, comprising 15 individuals with misophonia (Group I) and age- and gender-matched individuals without misophonia (Group II). The basic audiological and demographic characteristics of the people listed in Table 3.1

Table 3.1

Participants' fundamental audiological results and demographics

	Misophonia sufferers (Group I)	**Controls (Group II)**
Mean age of participants (in years)	24.4+_2.92	22.92+_2.16
No. of participants	15	15
No. of ears	30	30
Tympanogram type	A type	A type
Average PTA	10.75	10.52
Ipsilateral reflex thresholds	Present	Present
Average UCL	91.66%	85.83%
Average SIS	100%	100%
Contralateral reflex thresholds	Present	Present

3.3 Selection Criteria

3.3.1 Individuals with Misophonia

To meet the study objectives, 30 participants aged 18–35 years with misophonia were recruited as Group I. Participants were selected based on the inclusion criteria proposed by Schröder et al. (2013). The severity of misophonia was assessed using the Revised Amsterdam Misophonia Scale (RAMISO-S) (Jager et al., 2020). Only those who met the inclusion criteria were included in the study.

3.3.2 Individuals Without Misophonia

Group II consisted of 30 age-matched individuals with normal hearing, no misophonic symptoms, no otological complaints, and hearing sensitivity within normal limits (15 dB HL).

3.3.3 Participant Inclusion Criteria

- All participants had to meet the following criteria for inclusion in the study:
- Hearing sensitivity had to be within normal limits at octave frequencies from 250 Hz to 8000 Hz for air conduction and from 250 Hz to 4000 Hz for bone conduction, with thresholds of 15 dB HL or better (Clark, 1981). This ensured that any group differences were not due to hearing loss.
- Normal middle ear function was required, as shown by an "A" type tympanogram and normal ipsilateral and contralateral acoustic reflex thresholds at 500 Hz and 1 kHz in both ears, using a 226 Hz probe tone at 85 dB SPL (Clark et al., 2007; Schairer et al., 2013). This ensured that middle ear status did not affect the results.
- Participants had to have completed the 12th standard and have sufficient English language ability to understand the questionnaires. This helped ensure accurate responses.

3.3.4 Exclusion Criteria for Individuals With and Without Misophonia

- Individuals with a history of hearing symptoms, vestibular symptoms, migraine, or middle ear disorders were excluded. This information was obtained through a detailed case history.
- Participants with tinnitus and hyperacusis were excluded. Tinnitus was screened using the Tinnitus Handicap Inventory (THI), a 25-item questionnaire that assesses the functional, emotional, and catastrophic effects of tinnitus (Newman et al.,

1996).

- Individuals with scores above the cut-off for tinnitus were excluded. Hyperacusis was screened using the Modified Khalfa Hyperacusis Questionnaire (MKHQ) (Khalfa et al., 2002). Participants who scored above the cut-off on the migraine screening questionnaire, based on the International Classification of Headache Disorders, 3rd edition (ICHD-3, 2018), were also excluded. These screening measures helped ensure that the study findings could be attributed to misophonia rather than to tinnitus, hyperacusis, or migraine.

3.4 Procedure

Informed consent was obtained via Google Forms from all participants in the study. Institutional ethical guidelines for bio-behavioral research (Venkatesan & Basavaraj, 2009) Studies were followed. The study involved three phases of data collection. Phase I involved recruiting participants and assessing their misophonia severity; Phase II involved routine audiological evaluation; and Phase III involved sensory gating.

Phase I

Routine audiological evaluations were carried out to select participants for the study. In addition, the RAMISO-S was administered to determine the severity of misophonia.

3.4.1 Recruiting participants and Assessment of misophonia severity

Routine audiological evaluations were carried out to select participants for the study. In addition, the RAMISO-S was administered to determine the severity of misophonia. Phase I included the collection of a comprehensive case history, quantification of hearing sensitivity, and analysis of middle ear and cochlear functioning

Google Forms, email, and social media sites including Facebook, Instagram, and WhatsApp were used to find study participants. A survey was given out that included information on demographics, contact information, and agreement to participate. The

Revised Amsterdam Misophonia Scale (RAMISO-S) was used to gauge the degree of misophonia (Jager et al., 2020). RAMISO-S scores fell into four categories: 0–10 (subclinical/no misophonia), 11–20 (mild), 21–30 (moderate), and 31–40 (severe). Additional demographic and clinical information, such as age, sex, and the presence of comorbid conditions (e.g., anxiety or depression), was collected to ensure comparability between groups and to allow for covariate adjustments in data analysis. Participants underwent a structured clinical interview conducted by a trained researcher to confirm the presence of these diagnostic criteria and to ensure they met all necessary inclusion requirements. Those scoring below ten were classified as individuals without misophonia, whereas those obtaining a score of 11 or higher were categorized as individuals with misophonia. Only participants with a definite misophonia diagnosis (those who have symptoms as listed in the diagnostic criteria) were considered in Group I.

3.4.2 Routine audiological evaluation

All audiological testing was conducted for both ears of each participant in an electrically and acoustically insulated chamber with noise levels that are within the permissible limits of ANSI standards (ANSI S3.1-1991, R2018). Otoscopic examination was performed as a routine procedure to visually assess the integrity of the external auditory canal and the tympanic membrane. Behavioral pure tone audiometry for both ears was administered using the modified version of the Hughson-Westlake procedure. (Carhart & Jerger, 1959), using a calibrated 2 channel Grason-Stadler Incorporation Audio Star Pro audiometer (Grason Stadler, Inc, MN, USA).

For air conduction, pure tone thresholds of 0.5 to 8 kHz were established using TDH 39 supra-aural headphones (Telephonics, Farmingdale, NY, USA), and for bone conduction from 0.5 to 4 kHz using a Radioear B-81 bone vibrator (RadioEar, Middelfart, Denmark). Evaluation of immittance with a 226 Hz probe tone and testing acoustic reflex thresholds at 500Hz and 1kHz, were carried out using a calibrated Grason-Stadler Incorporation

Tympstar Pro immittance audiometer (Grason Stadler, Inc, MN, USA) was done to ensure normal middle ear functioning.

Phase II

Sensory Gating

The following stimuli and procedures were used to assess sensory gating in all participants.

Stimuli

A conditioning-testing paradigm was used to gauge the participants' capacity for sensory gating. The stimuli were two identical 250 Hz tone bursts produced in Adobe Audition version 3.0 with a 50 ms plateau and 10 ms rise-fall durations, spaced 500 ms apart. S1 was the first stimulus in the pair, and S2 was the second. Two successive stimulus pairs were separated by seven seconds. The stimuli are represented graphically in Figure 1.

Figure 3.1 Illustration of the Sensory Gating Experiment's Stimulus Paradigm

Note. 500 ms inter-stimulus interval and a 7 s inter-pair interval between two identical stimulus pairings.

Recording of Auditory Evoked Potentials

The SmartEP device's Continuous Acquisition Module (Intelligent Hearing System Corp., Miami, FL) was used to display the calibrated stimuli. The participants were given detailed instructions describing the actions involved before the test procedure started. Participants were situated in a sound-treated room in comfortable reclining chairs for the recording. Throughout the session, they were told to limit eye movements and stay as motionless as possible

A 1–100 Hz band-pass filter was used to record the raw EEG, which was then transformed

into a digital signal with a 1000 Hz sampling frequency. A total of 150 stimulus pairs had artifact-free responses recorded. For the duration of the recording, the participant's preferred closed-captioned film was played. Electrically insulated ER-3A insert earphones were used to deliver stimuli to the participants' right ear at 70 dB nHL.

The ground electrode was positioned on the non-test-ear mastoid, the inverting electrode on the test-ear mastoid, and the non-inverting electrode at Cz. A gain of 50,000 μV was used. Additionally, electrodes positioned above and below the right eye as well as at the outer canthi of both eyes were used in two bipolar ocular channels to capture both horizontal and vertical eye movements. Every electrode impedance was kept below 5 kΩ. Based on previously published research, the stimulus and acquisition parameters were modified (Campbell et al., 2018; Gafoor & Kumar, 2022). The stimulus and acquisition parameters for sensory gating Illustrated in Table 3.2.

Table 3.2 Stimulus and acquisition parameters for sensory gating

Stimulus Parameters	
Type of stimulus	Pair of identical 250 Hz tone bursts
Duration	10 ms – 50 ms – 10 ms
Inter-stimulus interval	500 ms
Inter-pair interval	7 s
Transducers	Insert earphones
Repetition rate	1.1/sec
Intensity	70 dB nHL
Sweeps	150
Mode	Monoaural
Acquisition parameters	
Time window recording	Continuous recording
Gain	500000
Filter setting	1-100 Hz
montage	Inverting electrode – test ear mastoid Non-inverting electrode – vertex (Cz) Ground – nontest ear mastoid 2 bipolar electrode pairs for VEOG & HEOG
impedance	< 5 kΩ (Absolute) < 2 kΩ (Inter
No. of channels	2

Note. , HEOG – horizontal electrooculogram. VEOG – vertical electrooculogram

3.4 Offline Processing and analyses

The continuous EEG will be epoched for 570 ms and filtered (to 1–30 Hz). We will extract the average event-related potentials for S1 and S2 separately. For both S1 and S2, the cortical evoked response peaks P1, N1, and P2 will be located, and their amplitudes and latencies will be recorded. The sensory gating ratio will then be calculated by dividing this (S2/S1) and multiplying the result by 100.The absolute differences in amplitudes and latencies between S1 and S2 for all peaks, and the sensory gating ratios, were compared between the two groups. An attempt was also made to correlate these parameters with RAMISO-S scores**.**

3.5. Statistical analyses

Statistical analysis was performed on the recorded data using (SPSS) the Statistical Package for the Social Sciences version 26.0 (IBM Corp.; Armonk, NY, USA). To ascertain if the data were normally distributed, the Shapiro-Wilk test was employed. Because the data was not regularly distributed, suitable parametric and non-parametric tests (such as the independent t-test) were used, respectively. The gating difference/ratio for amplitude and latency of P1, N1, and P2 did not correlate with AMISO-S scores, according to the results of the Pearson's rank correlation test**.** and the independent t-test showed significantly lesser amplitude difference and lower gating ratio for individuals with misophonia for P1, N1, and P2, showing poor sensory gating. Similarly, independent t-test also showed significantly lesser latency difference and lower gating ratio for individuals with misophonia for P1, showing poor sensory gating.

CHAPTER 4

RESULTS

The gating ratio and disparities between normal persons and those with and without misophonia were investigated in this study. The study also investigated the correlation between the AMISO scores and the gating differences of amplitude and latency and the Gating ratio of Amplitude and latency in the individuals with misophonia

4.1 Gating differences of Amplitude

The primary characteristics of the dataset were summarized and described using descriptive statistics, such as mean, median, standard deviation, and range, as Illustrated in Table 4.1

Table 4.1

Descriptive statistics of gating differences of amplitude for controls and misophonia

	Gating Differences of amplitude	Mean	Median	Standard deviation	Minimum	Maximum
P1	Control	1.5	1.5	0.4	1.0	2.3
	Misophonia	0.8	0.9	0.3	0.3	1.7
N1	Control	2.6	2.4	1.6	5.7	1.2
	Misophonia	0.7	1.1	0.8	1.4	1.7
P2	Control	3.0	2.3	1.3	1.7	5.8
	Misophonia	1.1	1.3	0.6	0.18	2.0

To determine data was regularly distributed, the test used was Shapiro- wilk test . The appropriate parametric test was performed for additional statistical analysis when it was determined that the data were normally distributed ($p>0.05$). To determine whether there were any significant changes between the experimental and control groups, an independent t-test was used. The result showed that the gating differences were significantly lesser ($p<0.05$) in individuals with misophonia compared to the control group. The median, minimum, maximum and inidividual data points for both the groups

is shown in figure 4.1.

Gating differences for Amplitude for the induvial with and without misophonia

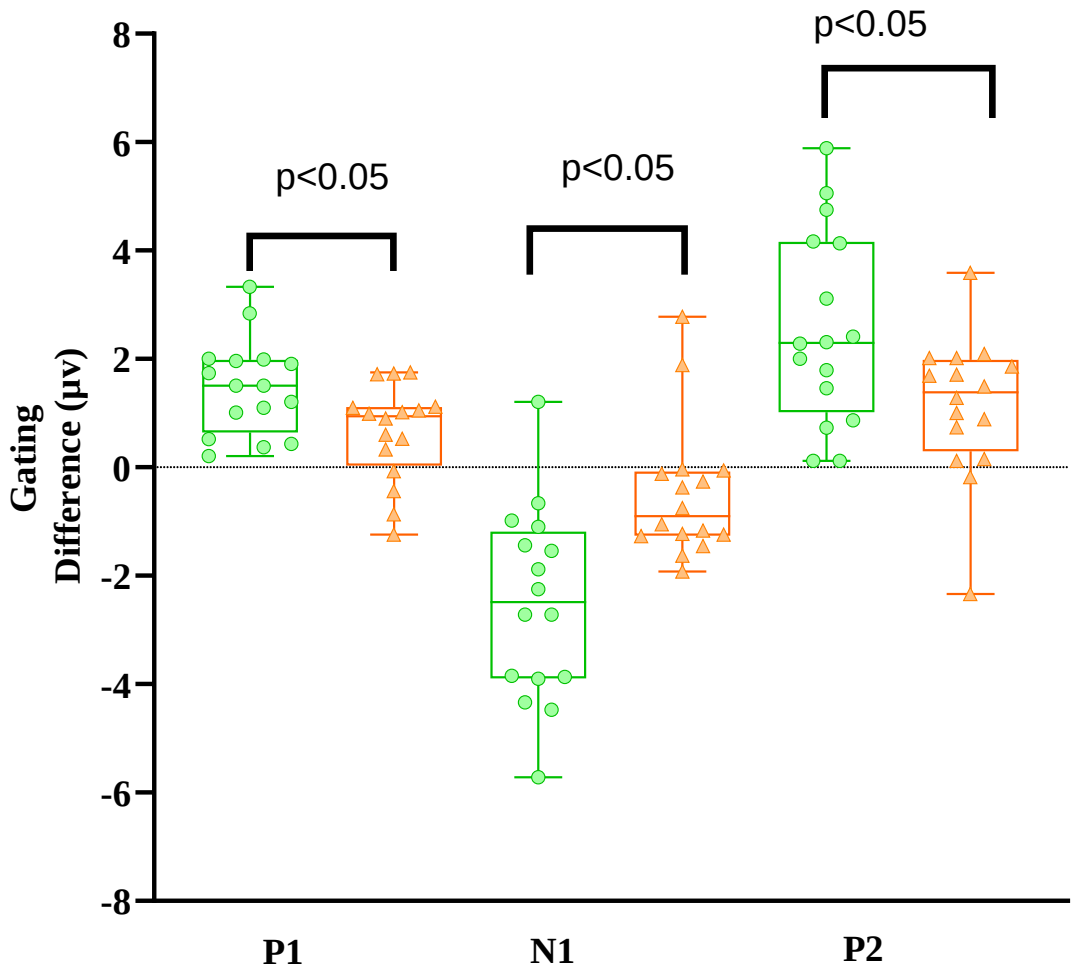


Figure 4.1. *The median, minimum, maximum and individual data points for amplitude differences for both the groups*

4.2 Gating Differences of latency

The primary data, which included the mean, standard deviation, median, minimum, and maximum as indicated in Table 4.2, were summarized using descriptive statistics.

Table 4.2

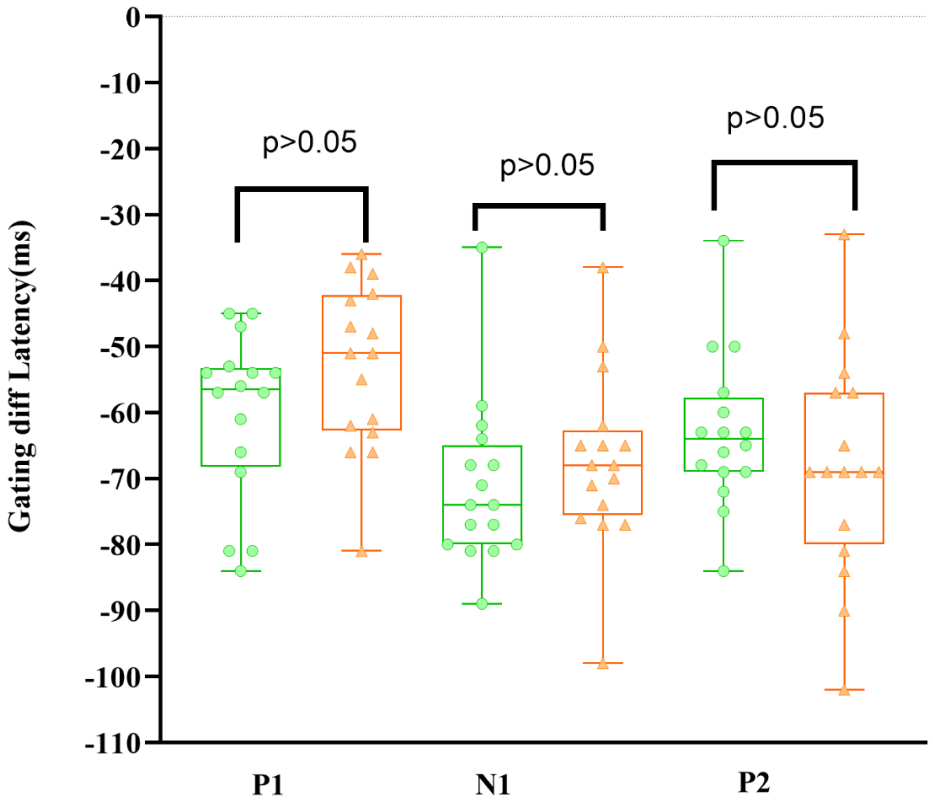
Descriptive statistics of gating differences in latencies for controls and misophonia

	Gating Differences in Latency	**Mean**	**Median**	**Standard Deviation**	**Minimum**	**Maximum**
P1	Control	6	56	12	84	45
	Misophonia	53	51	12	81	36
N1	Control	71	74	12	89	35
	Misophonia	67	68	13	98	38
P2	Control	65	64	25	153	34
	Misophonia	68	69	16	102	33

Shapiro wilk carried to check whether the data was normally distributed. The data was lies to be normally distributed ($p>0.05$) and following this appropriate parametric test was carried out for further statistical analysis. The Independent t- test was done on both control and misophonia group the result of the study indicated [$p>0.005$] which indicates no statistical differences obtained between the control and experimental group in the gating differences of latency. The Gating Differences of the latency for both the groups is shown in the figure. The median, minimum, maximum and individual data points for both the groups is shown in figure 4.2.

Figure 4.2

The latency disparities between the two groups' median, minimum, maximum, and individual data points



4.3 Gating ratio of Amplitude, P1, N1, P2

Descriptive statistics were used to summarize and describe the main feature of the dataset, such as mean, median, standard deviation, and range as shown in Table 4.3.

Table 4.3

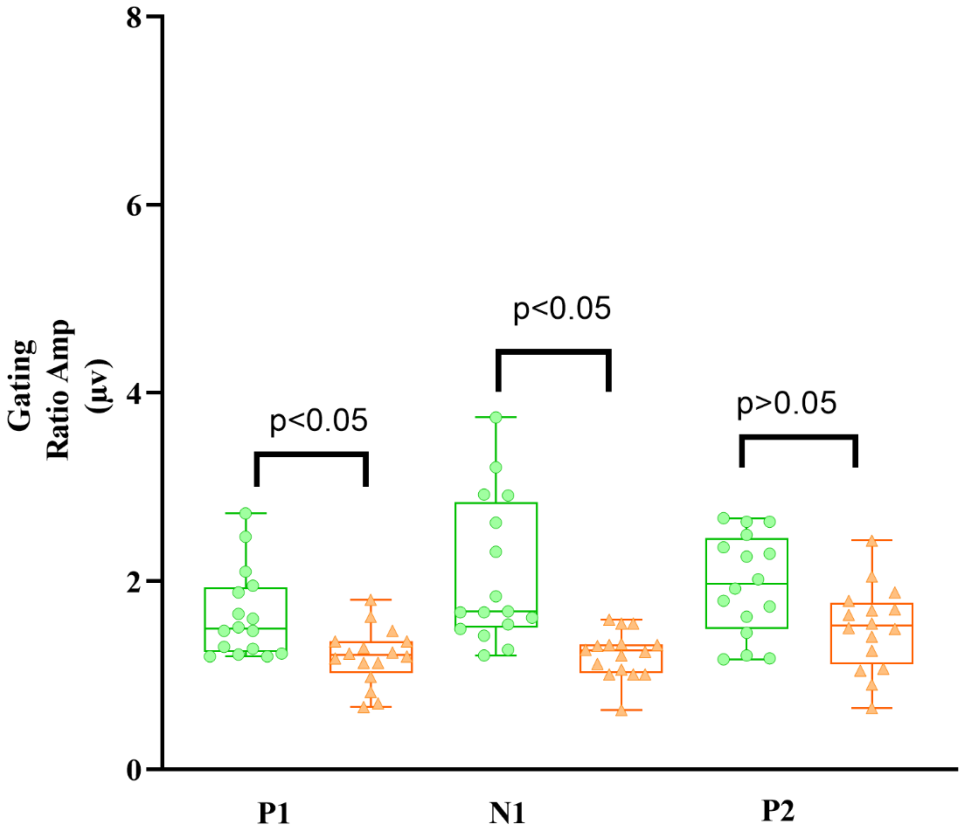
Descriptive statistics in gating ration of amplitude for controls and misophonia

	Gating Ratio of Amplitude	Mean	Median	Standard Deviation	Minimum	Maximum
P1	Control	1.6	1.4	0.4	1.2	2.7
	Misophonia	1.1	1.2	0.3	0.6	1.8
N1	Control	2.0	1.6	0.7	1.2	3.7
	Misophonia	1.2	1.2	0.2	0.6	1.5
P2	Control	1.9	1.9	0.5	1.1	2.6
	Misophonia	1.5	1.5	0.4	0.6	2.4

To ascertain if the data was normally distributed, the Shapiro-Wilk test was employed. An independent t-test was employed to search for significant differences between the groups. It was discovered that the P1, N1 gating ratio of amplitude was statistically significant ($p<0.05$). The gating ratio of P1, N1, and P2 amplitude for both control and misophonia is shown in the picture. The median, minimum, maximum, and individual data points for each group are shown in Figure 4.3.

Figure 4.3

The median, minimum, maximum and individual data points for gating ratio of amplitudes for both the groups



4.4 Gating Ratio of the Latency

Descriptive Statistics were used to summarize the main feature of the data set which include the Mean, Median, Standard Deviations, Minimum, maximum as shown in the Table 4.4.

Table 4.4

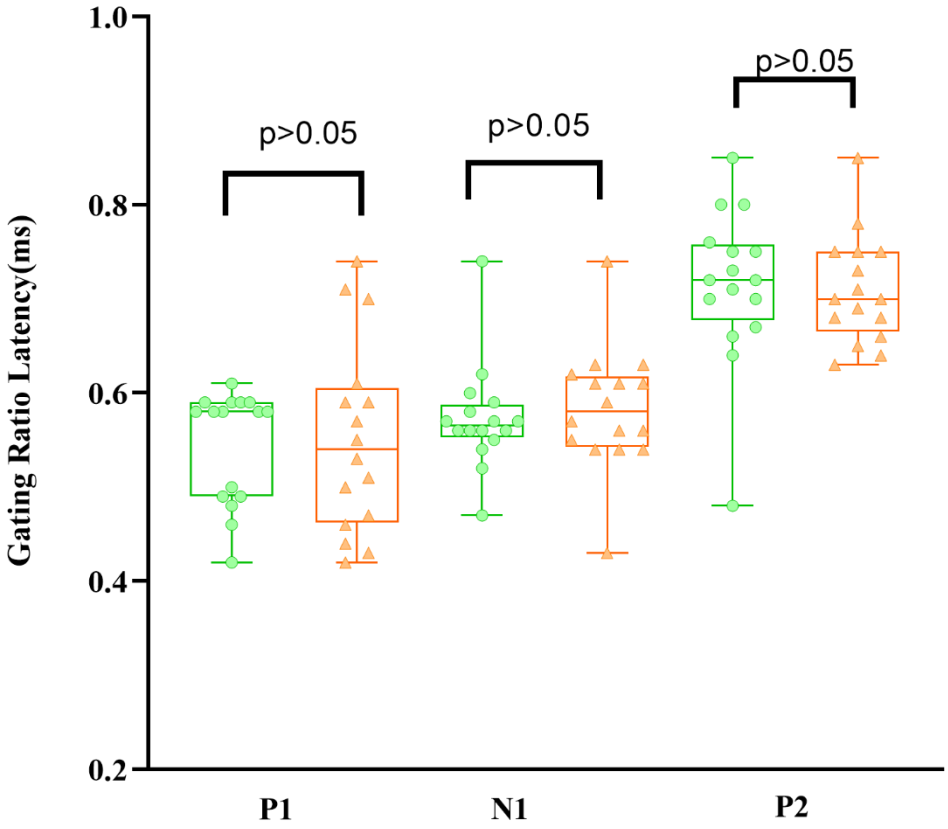
Descriptive Statistics on Gating ratio of Latency for both Control and misophonia.

	Gating ratio of Latency	**Mean**	**Median**	**Standard Deviation**	**Minimum**	**Maximum**
P1	Control	0.5	0.5	0.05	0.4	0.6
	Misophonia	0.5	0.5	0.1	0.4	0.7
N1	Control	0.5	0.5	0.5	0.4	0.7
	Misophonia	0.5	0.5	0.06	0.4	0.7
P2	Control	0.7	0.7	0.08	0.4	0.8
	Misophonia	0.7	0.6	0.05	0.6	0.8

The Shapiro-Wilk test was used to determine whether the data was normal. Additional statistical analysis was performed because the data had a normal distribution. There was no statistically significant difference between the control and misophonia groups, according to the results of the independent t-test, which showed [$p>0.05$]. Figure 4.4 displays the latency gating ratio as well as the median, minimum, maximum, and individual data points for both groups.

Figure 4.4

The median, minimum, maximum and individual data points for gating ratio of latencies for both the groups



4.5 Correlation of severity and sensory gating parameters

The results reveal a substantial association between the severity of misophonia using RAMISO scores and sensory gating characteristics, as determined by Spearman's rank correlation. The correlation analysis revealed a strong relationship between RAMISO scores and amplitude gating ratios and amplitude difference for P1 and N1 amplitude parameters. The findings demonstrated that the amplitude difference and gating ratio for people with misophonia decreased with increasing severity. This indicates unequivocally that people with higher degrees of misophonia are more impacted by sensory gating. Table 4.5 displays the correlation results.

Table 4.5

Correlation between severity and Gating Differences of Amplitude, Latency and Gating ratio of Amplitude and Latency

RAMISO	Rho ρ	Sig. (2-tailed)
Gating Diff Amp P1	-0.58	0.002
Gating Diff Amp N1	-0.61	0.001
Gating Diff Amp P2	+0.21	0.24
Gating Diff Lat P1	-0.4	0.44
Gating Diff Lat N1	-0.046	0.80
Gating Diff Lat p2	-0.26	0.14
Gating ratio Amp P1	-0.52	0.002
Gating Ratio Amp N1	-0.54	0.002
Gating Ratio Amp P2	0.35	0.051
Gating Ratio Lat P1	-0.049	0.79
Gating Ratio Lat N1	-0.24	0.16
Gating Ratio Lat P2	-0.12	0.49

CHAPTER 5

DISCUSSION

The present study examined sensory gating in individuals with misophonia using the Late Latency Response (LLR), with particular attention to gating differences and gating ratios for both latency and amplitude in the misophonia group compared with typically hearing controls. The results showed clear evidence of impaired sensory gating in the misophonia group, especially in the gating difference and gating ratio of amplitude. This finding suggests that the auditory cortex in individuals with misophonia may not adequately suppress or filter repetitive sounds. In practical terms, a trigger sound presented a second time appears to be processed with almost the same cortical strength as the first presentation. In healthy listeners, repeated sounds are usually processed with reduced neural activity, but this reduction appears to be weaker in misophonia. The present findings are consistent with those of Lodol et al. (2023), supporting the view that reduced cortical filtering may be an important feature of misophonia.

The statistical analysis showed significant group differences in both the gating ratio and gating differences of amplitude, whereas the latency measures did not show significant differences. This amplitude-specific result is important because it suggests a problem in the strength of cortical responses rather than in response timing alone. The fact that the brain continues to respond strongly to repeated auditory input, instead of showing normal habituation, may help explain why trigger sounds remain highly unpleasant in misophonia even after repeated exposure.

5.1 Peripheral vs. Central Origin

An important issue in interpreting these findings is the site of dysfunction in misophonia. Increasing evidence suggests that peripheral hearing loss is not a causal factor. Aazh (2020) carried out a detailed audiological evaluation of individuals with misophonia and concluded that peripheral hearing function is largely intact in this population. This supports the view that misophonia is more likely related to central auditory processing differences than to cochlear or peripheral damage. The present study

is consistent with this view because it focused on cortical electrophysiological measures.

5.2 Cortical Auditory Processing Abnormalities

Several recent studies have reported abnormalities in cortical auditory processing in misophonia, which provide a broader context for the present findings. Karupaiah et al. (2025) studied cortical auditory processing using a multichannel auditory late latency response paradigm in 30 participants, including 15 individuals with misophonia and 15 controls. They found earlier latencies and reduced N1 amplitudes across all recording sites, including Fz, Cz, and Pz, in the misophonia group. The authors interpreted this as evidence of heightened cortical excitability and suggested that reduced N1 amplitude may serve as a potential neurobiological marker of misophonia.

Similarly, Patro et al. (2025) examined neurophysiological, perceptual, and cognitive mechanisms in misophonia using a broader set of electrophysiological and cognitive tests. They reported significantly smaller mean peak amplitudes of the N1 and N2 components in response to oddball tones in individuals with misophonia compared with controls. This points to abnormalities in early auditory processing beyond simple behavioral sensitivity. Together, these studies support the view that the cortex may be a key site of dysfunction in misophonia, making the study of cortical inhibitory mechanisms such as sensory gating both relevant and timely.

5.3 Sensory Gating: LLR vs. P50

It is important to acknowledge that not all electrophysiological paradigms have demonstrated gating impairment in misophonia. Du et al. (2026) examined sensory gating using the P50 suppression paradigm and found that individuals with misophonia showed overall normal P50 suppression. Moreover, misophonia severity did not correlate with the degree of P50 suppression, suggesting that early pre-attentive gating which operates automatically at the subcortical and early cortical levels may be largely intact in this population. However, this finding does not necessarily contradict the present results; rather, it highlights a critical distinction between early and late cortical gating mechanisms. The P50 component reflects pre-attentive, early-stage sensory filtering that

occurs relatively automatically and without the need for conscious attention. In contrast, LLR-based sensory gating as measured in the current study captures later cortical processing stages that involve attentional modulation, higher auditory cortex engagement, and top-down regulatory filtering. It is therefore plausible that misophonia selectively disrupts later, attention-dependent cortical gating while leaving early, automatic gating largely unaffected. This distinction not only reconciles the apparently conflicting findings in the literature but also suggests that the neural disruption in misophonia occurs at a stage where sounds are being evaluated for relevance and emotional significance — precisely the stage at which trigger sounds would acquire their heightened aversive meaning.

5.4 Sample Characteristics and Future Directions

The present study evaluated 32 participants,16 individuals with misophonia of mild-to-moderate severity and 16 typically hearing controls and the findings indicate that even at mild-to-moderate levels of symptom severity, measurable impairment in cortical sensory gating is present. This is a clinically important observation, as it suggests that LLR-based sensory gating measures may be sensitive enough to detect neural abnormalities before misophonia reaches severe levels.

That said, several avenues remain open for future investigation. First, studies with larger sample sizes are needed to increase statistical power and improve the generalizability of findings. Second, evaluating sensory gating across the full spectrum of misophonia severity including severe and profound cases would allow for a clearer characterization of the dose-response relationship between symptom severity and gating impairment. Third, integrating advanced electrophysiological measures with neuroimaging techniques would provide a more comprehensive picture of the neural networks involved.

CHAPTER 6

SUMMARY AND CONCLUSIONS

Misophonia is disorder where the everyday sounds affect the quality of life in the induvial with misophonia. There have been studies reported the sensory gating has been affected in the induvial with Tinnitus. Since then, tinnitus, hyperacusis, misophonia have the similar pathophysiology, so abnormal sensory gating can be expected in misophonia. This study aimed to find the same. For the study, thirty-two people were enlisted. 16 of them were misophonia and 16 were control. The AMISO-R administered to determine the severity of the misophonia, and other Questionnaire like Tinnitus handicap inventory, Khalfa Questionnaire for hyperacusis was used to rule the conditions like tinnitus and hyperacusis respectively.

The individuals with the history of migraine, diabetes, tinnitus, hyperacusis, anxiety are eliminated from the study. The sensory gating was chceked using the LLR sensory gating paradigm, in which the cortical evoked potentials was recorded with the a pair of Click-stimulus which was presented, the gating was evaluated by comparing the amplitude suppression of P1-N1-P2 complex with S1, S2 stimulus

Result revealed there was statistically significant differences between the individuals with Controls and misophonia in the sensory gating test. The differences was seen in the gating differences and gating ratio of the amplitude, and no differences was observed in the gating ratio and gating differences of the latency, This shows Deficit in the Cortical inhibitory mechanism, which intern shown the impaired gating in the sensory gating test

6.1 Implications of the study

- This study provided a between insight on the cortical inhibitory mechanism skills in misophonia.
- This study provided a between insight on LLR sensory gating measures (S2/S1 amplitude ratio) can serve as a potential objective biomarker
- This study provided a between insight on Objective evidence for cortical inhibitory dysfunction

6.2 Future Directions

- A larger Sample size can be considered for the future studies
- The study can be performed with the Various severity in the individuals with misophonia
- The Sensory gating test can be performed to assess the Useful for monitoring treatment outcomes like tracking the Improvement after CBT, Auditory training programs etc
- Advanced electrophysiological studies can be used to understand the underlining physiology
- Explore the neural generator and cortical regions
- To validate LLR sensory gating as an objective diagnostic and monitoring tool, and to better understand the cortical inhibitory dysfunction underlying misophonia.

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