Exploring Auditory Encoding in Misophonia using Frequency Following Response

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CERTIFICATE

This is to certify that this dissertation entitled **"Exploring Auditory Encoding in Misophonia using Frequency Following Response"** is a bonafide work submitted as part of fulfilment of the degree of Master of Science (Audiology) of the student with Registration Number **P01II24S023012**. 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.

Mysuru
June, 2026

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CERTIFICATE

This is to certify that this dissertation entitled **"Exploring Auditory Encoding in Misophonia using Frequency Following Response"** 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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Mysuru
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DECLARATION

This is to certify that this dissertation entitled **"Exploring Auditory Encoding in Misophonia using Frequency Following Response"** 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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DEDICATED TO APPA, AMMA,

THATHA, PAATI,

ANANTHA AND THE ALMIGHTY…

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ABSTRACT

Misophonia is a condition characterized by intense negative emotional and physiological reactions to specific auditory trigger sounds, despite normal peripheral hearing sensitivity. Although previous research has demonstrated altered cortical processing in individuals with misophonia, limited evidence exists regarding auditory encoding at the brainstem level. The present study aimed to investigate auditory encoding in individuals with misophonia using Frequency Following Responses (FFR). Thirty normal-hearing individuals participated in the study, including 15 individuals with misophonia and 15 individuals without misophonia serving as controls. FFRs were recorded using a 170 ms /ba/ stimulus presented at 70 dB SPL. Temporal envelope and temporal fine structure components were extracted and analyzed using customized MATLAB scripts. Statistical analyses revealed no significant differences between individuals with and without misophonia for any of the FFR parameters. Additionally, no significant correlation was observed between misophonia severity scores and FFR measures. The findings suggest that auditory brainstem encoding of acoustic information remains largely preserved in individuals with mild-to-moderate misophonia. These results are consistent with contemporary models that propose misophonia is driven more by abnormal auditory-limbic interactions at higher cortical levels than by deficits in early auditory sensory encoding. The study adds to the understanding of the neuropathophysiology of misophonia and underscores the need for future research using ecologically valid auditory paradigms and multimodal neurophysiological approaches.

Keywords: misophonia, frequency following responses, electrophysiology, auditory encoding, brainstem

CHAPTER 1

INTRODUCTION

Misophonia is a strong dislike or aversion to certain sounds that produces adverse emotional reactions (Jastreboff & Jastreboff, 2001). It reflects abnormally strong reactions of the autonomic and limbic systems due to enhanced connections between the auditory and limbic systems (Jastreboff & Jastreboff, 2001). It is a condition wherein an abnormally strong reaction occurs to a sound with a specific pattern and/or meaning to an individual. The stimuli eliciting such aversive reactions are referred to as "triggers," which often include repeated, human-generated sounds such as chewing, breathing, or similar actions, with the aversive impact occurring regardless of their loudness level (Jastreboff & Jastreboff, 2014).

A defining characteristic of misophonia is heightened sensory sensitivity, particularly to repetitive, human-made sounds (Rinaldi et al., 2023). Individuals with misophonia tend to exhibit heightened sympathetic nervous system arousal and emotional distress when exposed to specific, pattern-based auditory triggers. These trigger sounds evoke an unpleasant, strong emotion associated with negative physiological and behavioural responses, which are generally considered unexpected for the acoustic stimulus from an individual without misophonia (Swedo et al., 2022). They experience strong negative emotional reactions due to the activation of their autonomic system by specific sound stimuli, termed ‘trigger sounds’ (Edelstein et al., 2013). Several studies have been conducted to investigate the various emotional reactions experienced by individuals with misophonia. They often report anger, disgust, and anxiety. In addition, it was found that depressive symptoms and lowered self-esteem were also observed in individuals with misophonia (Dibb & Golding, 2022). The physiological

reactions accompanying these emotional states may include muscle tension, increased heart rate (Siepsiak & Dragan, 2019), sensations of pressure in the chest, arms, head, or throughout the body, elevated body temperature, physical pain, and shortness of breath (Edelstein et al., 2013b). Some individuals have also reported episodes of verbal or physical aggression in response to misophonic triggers, including outbursts directed toward objects or, in rare cases, toward others. While anxiety was not always explicitly noted, these aggressive reactions were often accompanied by an intense sense of losing self-control (Schröder et al., 2013).

On reviewing the prevalence of this condition, a systematic review found that the global prevalence of misophonia in adolescents and adults ranges from 5% to 34.67% (Gowda & Prabhu, 2024). In a representative sample of the adult population in the US, 78.5% reported sensitivity to sounds associated with misophonia, while 4.6% indicated experiencing clinical levels of misophonia (Dixon et al., 2024). Similarly, a Brazilian study indicated that 4.5% of young adults experienced misophonia. (Sanchez & Silva, 2018). Within the Indian context, 15.85% of college-going students met criteria for moderate to severe misophonia. (Patel et al., 2023), while a higher prevalence of 34.67% was observed in school-going students (Sujeeth et al., 2024). Attempts are ongoing to understand this condition from an audiological, psychological, and neuroscience perspective.

1.1 Need for the study

Over the past decade, numerous research studies have been conducted to understand the mechanisms underlying misophonia. Neuroimaging studies have indicated that misophonia involves aberrant activation of the anterior insular cortex and its heightened connection with emotion-processing regions, resulting in exaggerated physiological responses to trigger sounds. (Kumar et al., 2017). This region additionally

exhibits stronger connections to areas involved in emotion and memory, such as the amygdala, hippocampus, posteromedial cortex, and ventromedial prefrontal cortex, which may account for the heightened emotional responses to everyday sounds. (Kumar et al., 2017). According to a recent review, these alterations cause the brain to place excessive importance, or "salience," on trigger sounds, making them feel overwhelming. (Neacsiu et al., 2022).

Among audiology-based assessments, one of the first was an Electroencephalography (EEG) study, which showed that individuals with misophonia exhibited diminished N1 responses to oddball tones, pointing to atypical early auditory processing. (Schröder et al., 2014). Cortical auditory evoked potential studies have further demonstrated significantly shortened P1 and N1 latencies in this group, suggesting altered auditory processing at higher cortical levels. (Aryal & Prabhu, 2024a). MMN response amplitudes were found to be significantly reduced in individuals with misophonia suggesting abnormal processing of specific auditory stimuli, particularly deviant stimuli (Patro et al., 2025). A recent study compared auditory brainstem responses (ABR) in individuals with and without misophonia. The study reported that absolute latencies of waves III and V were earlier at 11.1/s in individuals with misophonia than in control groups, while amplitudes remained comparable across groups. These findings indicate a possible hyperactivity at the subcortical auditory centers. Moreover, this neural hyperactivity indicates the heightened response to the auditory stimuli in individuals with misophonia. (Karupaiah & Prabhu, 2025a).

Several studies have used Frequency Following Response (FFR) as a tool to investigate auditory processing. The temporal fine structure (TFS) obtained from FFR is found to be abnormal in older adults with hearing loss (Anderson et al., 2013). Pitch discrimination ability is found to deteriorate significantly with age, irrespective of normal

hearing thresholds, and this is reflected in the changes in FFR (Clinard et al., 2010). FFR to speech stimuli are also highly variable in children with dyslexia, indicating auditory processing deficits (Hornickel & Kraus, 2013). Abnormal FFR peaks and delayed onset latencies are also observed in children with learning difficulties, suggesting neural timing deficits at the brainstem level that disrupt auditory processing (King et al., 2002). Such auditory processing deficits have also been suspected in individuals with misophonia. Research findings suggest that individuals with misophonia tend to focus more on their trigger sounds, which may lead to difficulty concentrating on complex sounds and impaired auditory processing (Brout et al., 2018). A recent study investigated the dichotic consonant-vowel test and pitch pattern test, and found significantly poor scores in individuals with misophonia compared to their controls (Karupaiah & Prabhu, 2025b). Thus, individuals with misophonia not only exhibit heightened emotional responses to certain trigger sounds but also experience specific deficits in their auditory processing ability (Karupaiah & Prabhu, 2025b).

This highlights the need for the present study to examine how auditory stimuli are perceived at both the brainstem and cortical levels in individuals with misophonia. The Frequency Following Response (FFR) is a valuable electrophysiological tool that offers insight into the neural representation of complex sounds. (Kraus et al., 2017). It closely follows the periodic fluctuations of the acoustic waveform, and scalp-recorded FFRs to complex sounds reflect phase-locked activity to both the temporal envelope and temporal fine structure (TFS) generated by populations of neurons in the rostral brainstem. (Anderson et al., 2013; Krishnan, 2002). Importantly, recent evidence demonstrates that the FFR is not exclusively subcortical. It is identified that there is a significant contribution from the right auditory cortex, particularly in encoding the fundamental frequency (F0) of speech sounds. (Coffey et al., 2017). This indicates that the FFR has a

composite origin, with both brainstem and cortical sources varying according to the stimulus. This highlights its utility in probing how abnormal auditory processing in misophonia may emerge across different levels of the auditory pathway.

However, there is a lack of research on how people with misophonia perceive and interpret sounds. While existing literature indicates that the auditory pathways to the cochlea are mostly intact, abnormalities appear to occur at central levels of the auditory pathway, such as the brainstem and cortex. Understanding the neural encoding of sound across these levels is essential to explaining the heightened reactivity to everyday auditory stimuli reported in misophonia. Thus, FFR provides a valuable window into these processes, as it encompasses contributions from both the brainstem and the cortex. However, despite its potential, no research has been conducted to explicitly evaluate FFR in individuals with misophonia.

1.2 Aim of the study

The study aims to investigate auditory encoding using frequency-following responses in individuals with misophonia.

1.3 Objectives of the study

- To compare the F0 spectral amplitude and amplitude of harmonics for the envelope FFR between individuals with and without Misophonia
- To compare the amplitude of the first formant (F1) for the fine structure FFR between individuals with and without Misophonia
- To assess the correlation between the scores of the Revised Amsterdam Misophonia Scale (RAMISO-S) and parameters of FFR in individuals with misophonia

1.4 Null hypotheses

- There is no significant difference in the F0 spectral amplitude and amplitude of harmonics for the envelope FFR between individuals with and without Misophonia

- There is no significant difference in the first formant (F1) amplitude for the fine structure FFR between individuals with and without Misophonia
- There is no correlation between the scores of the Revised Amsterdam Misophonia Scale (RAMISO-S) and parameters of FFR in individuals with misophonia

CHAPTER 2

REVIEW OF LITERATURE

2.1 Definition of Misophonia

Misophonia was first described by Jastreboff and Jastreboff (2001) while working with patients with tinnitus. During their clinical observations, they identified a subgroup of individuals who exhibited symptoms that could not be adequately explained by previously recognized auditory disorders (Jastreboff & Jastreboff, 2001). The term misophonia was derived from the Greek words misos meaning hate and phonia referring to sound. It was initially defined as an abnormally strong reaction of the autonomic and limbic nervous systems caused by enhanced functional connections between the auditory and limbic systems (Jastreboff & Jastreboff, 2001).

Misophonia refers to a condition in which individuals exhibit disproportionately strong emotional and physiological reactions to specific sound stimuli that possess a particular pattern or personal meaning. These sounds, commonly referred to as trigger sounds, misophonic stimuli, or misophonic sounds, evoke aversive reactions despite often being relatively low in intensity. Importantly, these stimuli typically possess consistent acoustic patterns rather than high loudness levels (Jastreboff & Jastreboff, 2014). Certain triggering sounds have also been reported to contain prominent high-frequency spectral components (Dacremont, 1995). Initially, misophonia was conceptualized as one component of Decreased Sound Tolerance (DST), along with hyperacusis and phonophobia (Jastreboff & Jastreboff, 2001).

Subsequently, misophonia began to be examined from a psychological perspective and was proposed to be related to the obsessive–compulsive spectrum disorders (OCSD). Researchers observed that individuals with misophonia frequently demonstrate a characteristic symptom pattern in which auditory or visual stimuli

trigger immediate aversive physical reactions accompanied by emotions such as anger, disgust, and impulsive aggression (Schröder et al., 2013). Recently, a consensus definition of misophonia was proposed, which defined misophonia as a 'disorder of decreased tolerance to specific sounds or stimuli associated with such sounds. These stimuli, known as "triggers," are experienced as unpleasant or distressing and tend to evoke strong negative emotional, physiological, and behavioral responses that are not seen in most other people (Swedo et al., 2022).

2.2 Prevalence of Misophonia

Recent research suggests that misophonia is increasingly being recognized as a clinically relevant condition. A systematic review reported that the global prevalence of misophonia ranges from 5% to 34.67% in the general population (Gowda & Prabhu, 2024). At the same time, prevalence estimates vary considerably across countries and populations. In a representative study from the United States, about 78.5% of adults reported sensitivity to sounds commonly associated with misophonia, while 4.6% met the criteria for clinically significant misophonia (Dixon et al., 2024). Similarly, a population-based study from Germany found that 33.3% of participants were sensitive to at least one misophonic sound, and 12.1% showed clinically significant misophonia symptoms (Pfeiffer et al., 2025). In Brazil, a study among young adults reported a prevalence of approximately 4.5% (De Almeida Silva et al., 2024).

In the Indian context, several studies have also examined the prevalence of misophonia. Among college students, 15.85% were found to have moderate-to-severe misophonia (Patel et al., 2023). A higher prevalence of 34.67% was reported among school-going children (Sujeeth et al., 2024). In addition, clinically significant misophonia symptoms were identified in 17.63% of university students (Yadav et al., 2024).

Researchers have also explored the relationship between misophonia and other clinical conditions. Evidence suggests that the frequency and severity of misophonia symptoms tend to be higher in individuals with tinnitus, hyperacusis, anxiety, or depressive symptoms (Aazh et al., 2022; Cusack et al., 2018). Earlier studies also suggested that the emotional responses triggered by specific sounds may be similar in individuals with misophonia and hyperacusis (Jastreboff & Jastreboff, 2014). In addition, some authors have proposed that misophonia may lie within the obsessive–compulsive spectrum because of the obsessive thoughts, emotional reactivity, and impulsive behavioral responses associated with trigger sounds (Schröder et al., 2013).

2.3 Pathophysiology of Misophonia

The underlying pathophysiological mechanisms of misophonia remain incompletely understood. Current evidence suggests that the condition may involve abnormal neural connectivity between the auditory system and the limbic and autonomic nervous systems. Such atypical connectivity is believed to contribute to heightened emotional and physiological responses to specific auditory stimuli.

Neurobiological models propose that misophonia may arise from altered sensory processing combined with increased emotional salience of particular sounds. Functional neuroimaging studies have reported increased activation in brain regions associated with emotional regulation and salience detection when individuals with misophonia are exposed to trigger sounds. These findings suggest that the disorder may involve abnormal integration between auditory-perception and emotional-processing networks.

2.3.1 Altered brain functions in misophonia

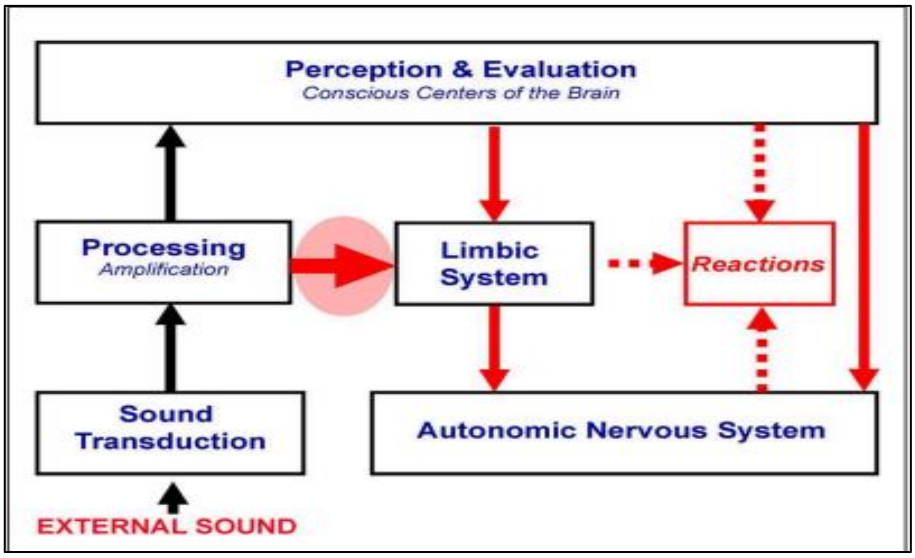
The advent of technologies such as fMRI and BOLD has enabled researchers to study changes in brain activity in individuals with misophonia. An fMRI study demonstrated that trigger sounds evoke heightened blood-oxygen-level-dependent (BOLD) activity in the anterior insular cortex (AIC) of individuals with misophonia. (Kumar et al., 2017b). The AIC functions as a central node within the salience network, playing a crucial role in interoceptive awareness and emotional processing. Furthermore, trigger sounds were found to produce aberrant functional connectivity between the AIC and key emotion-related regions, including the ventromedial prefrontal cortex (vmPFC), posteromedial cortex (PMC), hippocampus, and amygdala, suggesting disrupted emotional regulation mechanisms in misophonia (Kumar et al., 2017b).

2.3.2 Neurophysiological model of misophonia

According to Jastreboff and Jastreboff in 2023, misophonia results from enhanced functional communication among the auditory, limbic, and autonomic nervous systems. The misophonic trigger sounds are viewed as complex conditioned stimuli. It is hypothesized that the defining feature of misophonic triggers is the meaning they are attributed and their association with an individual's previous experiences, such as particular people, places, or situations. This model attempts to explain why only specific sounds act as triggers in misophonia and why the same sounds can be more distressing when produced by certain individuals compared to others (P. J. Jastreboff & Jastreboff, 2023).

Figure 2.1

The neurophysiological model of misophonia (Adapted from Jastreboff & Jastreboff, 2023)



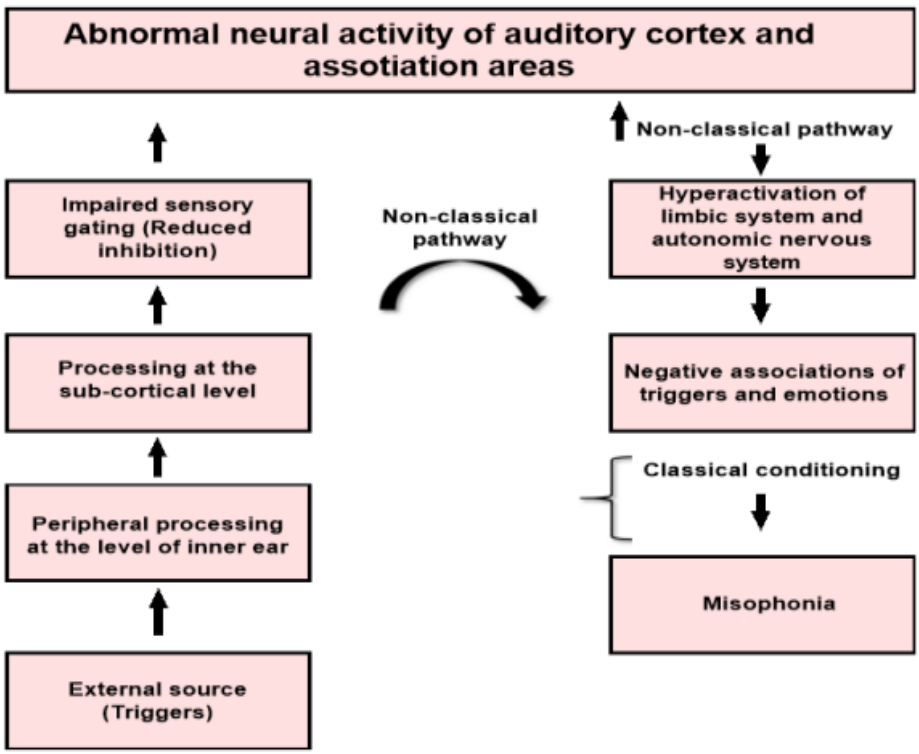
2.3.3 Neuroaudiological model of misophonia

The neuroaudiological model conceptualizes misophonia as a neurophysiological condition situated at the interface of audiology, neurology, and psychiatry. As described by Aryal and Prabhu (2023c), this model explains misophonia from an auditory-system perspective by integrating peripheral auditory processing, central auditory pathways, and emotional regulatory mechanisms. An external auditory trigger is initially processed normally at the peripheral level and then transmitted to subcortical auditory centers, where it undergoes further processing through both classical and non-classical auditory pathways. However, individuals with misophonia appear to have impaired sensory gating, resulting in reduced inhibitory control over incoming auditory input. Consequently, trigger sounds receive heightened neural processing within higher auditory cortical and association areas. The auditory cortex also interacts with the limbic system and autonomic nervous system, which contribute to emotional and

physiological responses such as anger, anxiety, and increased heart rate. With repeated exposure, the trigger sound may become classically conditioned to elicit negative emotional responses (Aryal & Prabhu, 2023c).

Figure 2.2

The neuroaudiological model of misophonia (Adapted from Aryal & Prabhu, 2023c)



2.3.4 Neurocognitive model of misophonia

The neurocognitive model emphasizes the involvement of higher-order cognitive networks in individuals with misophonia (Savard & Coffey, 2025). It highlights abnormal integration between auditory processing and emotional regulatory systems. A key structure implicated in this model is the anterior insular cortex (AIC). Neuroimaging studies have shown exaggerated AIC activation when individuals with misophonia are exposed to trigger sounds. The AIC is an important node in the salience

network and plays a central role in identifying behaviourally relevant stimuli and directing attentional resources. Hyperactivation of this region may lead trigger sounds to be perceived as highly salient and emotionally significant, which in turn contributes to intensified emotional responses. Functional connectivity studies further demonstrate abnormal communication between the AIC and several emotion-related brain regions, including the ventromedial prefrontal cortex (vmPFC), posteromedial cortex (PMC), amygdala, and hippocampus. These regions are involved in emotional evaluation, memory processing, and regulation of affective responses. Dysfunctional connectivity within this network may impair emotional regulation and amplify negative emotional reactions to specific sounds.

The model also demonstrated that misophonic responses negatively affected cognitive control. Individuals with misophonia showed greater cognitive interference during a Stroop task while simultaneously being exposed to trigger sounds, when compared to their normal counterparts. (Daniels et al., 2020). One other study also reported poor performance of individuals with misophonia in the dichotic sentence test while trigger sounds were present. (Silva & Sanchez, 2019).

Thus, the neurocognitive model proposes that misophonia arises from abnormal interactions among auditory perception, attention networks, emotional processing systems, and associative learning mechanisms in the brain.

2.3.5 Genetics and misophonia

A few studies have investigated the genetic etiology of misophonia. Evidence for a possible genetic contribution to misophonia was reported in a familial study by Sanchez & Silva (2018). The study described a family in which 15 members across three generations exhibited symptoms of misophonia and experienced symptom

onset during childhood or adolescence. The authors proposed a possible autosomal dominant pattern of inheritance. (Sanchez & Silva, 2018).

One other study examined the behavioural and physiological characteristics of individuals with misophonia and reported that several participants indicated that close family members exhibited similar symptoms, suggesting a potential hereditary component. (Edelstein et al., 2013a). A recent investigation using a genome-wide association study examined the genetic etiology of rage related misophonia symptoms. (Smit et al., 2023). The study identified a significant genetic locus (rs2937573) near the TENM2 gene, which is involved in neuronal development. The findings also showed that misophonia symptoms have measurable SNP-based heritability (approximately 8.5%), indicating that genetic factors contribute to individual differences in misophonic traits. Additionally, genetic correlations were observed between misophonia symptoms and psychiatric conditions such as major depressive disorder, generalized anxiety disorder, and post-traumatic stress disorder, suggesting that misophonia may share genetic pathways with certain psychiatric and personality traits. (Smit et al., 2023).

2.4 Assessment of Misophonia

The consensus definition states that neither misophonia's presence nor its severity appears to be related to the patient's pure-tone sensitivity. However, it is unclear what effect hearing loss may have on this perception. (Aazh et al., 2022). The main approach to diagnosing misophonia currently involves obtaining a detailed case history and administering standardized questionnaires to assess its severity. To date, no specific protocol has been established for the assessment of misophonia. Various audiological tests are being investigated to determine their potential in diagnosing misophonia.

In recent research, 59.32% of audiologists recommended the use of Puretone audiometry and Loudness Discomfort Level (LDL) for the assessment of misophonia

(Aryal & Prabhu, 2023b). Studies report normal hearing thresholds at standard audiometric frequencies and in extended high-frequency audiometry (Aryal & Prabhu, 2023b). However, LDL levels were lower in the misophonia group than in the non-misophonia group (Aryal & Prabhu, 2024b; Jastreboff & Jastreboff, 2015). Another study also observed lower LDL levels in individuals with frequent misophonia symptoms compared with those with no or less frequent symptoms (Aazh et al., 2022). Despite ongoing research, audiological findings do not provide a valid measure for diagnosing misophonia. Extended high-frequency audiometry was also conducted, and individuals with misophonia exhibited significantly enhanced auditory thresholds compared to their controls, indicating increased auditory sensitivity (Chalotra et al., 2025).

Physiological assessments examining the peripheral auditory pathways were also studied in misophonia. A study investigating distortion-product otoacoustic emission (DPOAE) input–output characteristics reported that post hoc analysis showed statistically significant differences between non-misophonics and moderate/severe misophonics at all input levels. (Suraj Urs et al., 2025). This finding suggests that peripheral cochlear mechanics may also differ in individuals with moderate-to-severe misophonia, despite normal hearing sensitivity.

Apart from routine audiological evaluation, some studies have also investigated central auditory processing in individuals with misophonia, yielding mixed results. Temporal processing abilities, as assessed by the duration pattern test, pitch pattern test, and gap detection test, were unaffected in individuals with misophonia. (Ila et al., 2023). However, dichotic listening tasks and speech in noise tasks were found to be poorer in individuals with misophonia (Kim et al., 2023;Silva & Sanchez, 2019). Another study also observed significantly poor scores on the Dichotic CV test and Pitch pattern test. (Karupaiah & Prabhu, 2025b).

Misophonia is frequently assessed using questionnaires. Case history is the primary tool for understanding the onset and nature of the problem, the type of triggering sounds, and the response elicited. Followed by a detailed case history, the major diagnostic criteria of Misophonia are assessed. Schröder et al. (2013) diagnostic criterion is a frequently adopted criterion in the assessment of misophonia. The suggested criteria state that a diagnosis of misophonia should be made when all of the following conditions are met:

- The anticipation or presence of a misophonic trigger stimulus provokes an aversive physical reaction that initiates with a feel of disgust/irritation that instantaneously evolves into anger.
- Anger reaction towards specific sounds causes a sense of losing self-control with occasional instances of aggressive outbursts.
- Individual him/herself realizes the anger or disgust felt is excessive and unreasonable with disproportionate reactions to the circumstances.
- Individual makes efforts to avoid the misophonic situation, and if not avoidable the individual tends to tolerate/endure the misophonic sound with intense feel of anger, disgust and discomfort.
- The reactions towards misophonic triggers causes distress and impacts the life style of the individual.
- Individuals' reactions towards misophonic triggers should not be triggered by any other disorder.

Several questionnaires are available to assess the severity of misophonia. These include the Amsterdam Misophonia Scale (Schröder et al., 2013; Naylor et al., 2021), MisoQuest (Siepsiak et al., 2020), the Misophonia Response Scale (Dibb et al., 2021),

the Core Discriminant Sounds of Misophonia (Enzler et al., 2021), the Duke Misophonia Questionnaire (Rosenthal et al., 2021) and the Misophonia Questionnaire (MQ) (Wu et al., 2014).

2.4.1 Electrophysiological Assessment of Misophonia

Recent research has increasingly focused on identifying objective neurophysiological markers to better understand the neural mechanisms underlying misophonia. Electrophysiological techniques, particularly electroencephalography (EEG)-based auditory evoked potentials, provide valuable insight into the temporal dynamics of auditory processing across different levels of the auditory pathway. Early EEG studies found reduced N1 amplitudes to oddball tones in individuals with misophonia, suggesting atypical cortical auditory processing at an early stage (Schröder et al., 2014). This implies that neural encoding of auditory stimuli may already be altered during the initial phases of cortical sound processing.

Later studies of cortical auditory evoked potentials (CAEPs) reported significantly shorter P1 and N1 latencies in individuals with misophonia compared to control groups (Aryal & Prabhu, 2024a). Shorter latencies may reflect increased cortical excitability or greater neural synchrony, indicating enhanced responsiveness of auditory cortical neurons to sound. Additional evidence for altered central auditory processing has come from mismatch negativity (MMN) paradigms, which assess automatic auditory discrimination. Reduced MMN amplitudes have been reported in individuals with misophonia, suggesting impaired neural detection of deviant auditory stimuli (Patro et al., 2025). These findings point to possible deficits in pre-attentive auditory discrimination.

Beyond cortical responses, studies of brainstem-level auditory processing have also shown differences in individuals with misophonia. Using auditory brainstem response (ABR), researchers reported earlier absolute latencies for waves III and V at a stimulation rate of 11.1/s, while response amplitudes remained comparable to those of control participants (Karupaiah & Prabhu, 2025a). These findings suggest possible subcortical auditory hyper-responsivity, indicating that atypical auditory processing may occur at multiple levels of the auditory pathway. Taken together, the available evidence suggests altered neural processing of auditory stimuli at both brainstem and cortical levels in individuals with misophonia. However, despite these emerging findings, the neurophysiological mechanisms underlying intermediate stages of auditory processing remain poorly understood.

One electrophysiological measure that may help clarify these mechanisms is the Frequency Following Response (FFR), which reflects phase-locked neural activity in rostral brainstem and midbrain structures. FFR provides information regarding the neural representation of complex acoustic features such as pitch, temporal periodicity, and spectral encoding. Given that misophonia is often triggered by specific acoustic patterns rather than by sound intensity alone, investigating neural encoding using FFR may offer important insights into how trigger sounds are represented in the auditory system.

CHAPTER 3

METHODS

3.1 Study Design and ethical guidelines

A convenience sampling procedure was used to recruit the participants for the study. A standard group comparison method (Orlikoff et al., 2014) involving two groups (with and without misophonia) was carried out. The study is quasi-experimental descriptive research where the FFR parameters are compared between individuals with misophonia and those without misophonia.

3.2 Participants

A total of 30 participants were divided into two groups, each comprising 15 individuals: those with misophonia (Group I) and those without misophonia (Group II). The basic demographic and audiological findings of the participants in the two groups are given in Table 3.1.

Table 3.1

Demographic and basic audiological findings of the participants

	Individuals with misophonia (Group I)	**Individuals without misophonia (Group II)**
Mean age of participants (in years)	23.93 ± 3.03	22.87 ± 2.09
No. of participants	15	15
No. of ears	30	30
Average PTA (dB HL)	10.52 ± 3.42	10.75 ± 2.82
Average SIS scores	100 % ± 0	100 % ± 0
Average UCL (dB HL)	85.83	91.66

Tympanogram type	'A' Type	'A' Type
Average ipsilateral acoustic reflex thresholds (dB HL)	88.72 ± 0.84	90.25 ± 0.74
Average contralateral acoustic reflex thresholds (dB HL)	99.36 ± 0.73	99.43 ± 0.92

3.3 Participant selection criteria

3.3.1. Individuals with misophonia

To fulfil the objectives of the study, 15 participants with misophonia, aged between 18 and 35 years, were selected for the study (Group I). The selection of participants with misophonia was based on the inclusion criteria proposed by Schröder et al. (2013) and assessed using the MisoQuest questionnaire (Siepsiak et al., 2020). The severity of misophonia was measured using the Revised Amsterdam Misophonia Scale (RAMISO-S) (Jager et al., 2020). Only participants who met the inclusion criteria were included in the study.

All the participants with pure tone thresholds within the normal limits (15 dB HL) and having 'A' type tympanogram with present acoustic reflexes were included in the study. Individuals with hearing loss, tinnitus, hyperacusis, migraine, or any ear-related pathology were excluded.

In addition, tinnitus, hyperacusis, and migraine were screened using screening checklists, which are the Tinnitus Handicap Inventory (THI) (Newman et al., 1996), Modified Khalfa Hyperacusis Questionnaire (Khalfa et al., 2002), and International Classification of Headache Disorders diagnostic checklist - 3 (ICHD 3, 2018) for Migraine, respectively.

3.3.2. Individuals without misophonia

A group of 15 normal-hearing individuals, age-matched to the former group, experiencing no misophonic symptoms, and meeting the inclusion and exclusion criteria for pure-tone hearing sensitivity, middle ear functioning, and ear-related pathology similar to those of the former group, were considered for Group II.

3.4 Procedure

Informed consent was obtained from all the participants included 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 severity of misophonia. Phase II involved routine audiological evaluation, and Phase III involved FFR testing.

3.4.1 Recruiting participants and Assessment of misophonia severity

The participants in the study were recruited using a one-to-one survey. A questionnaire containing details such as consent for participation, demographic and contact details, MisoQuest questionnaire (Siepsiak et al., 2020), Tinnitus Handicap Inventory (THI) (Newman et al., 1996), Modified Khalfa Hyperacusis Questionnaire (Khalfa et al., 2002), and International Classification of Headache Disorders diagnostic checklist - 3 (ICHD 3, 2018) for Migraine was distributed. The questionnaires used in the study are shown in Appendix 1-4. The presence of misophonia was confirmed using the MisoQuest questionnaire. It is a 14-item questionnaire which uses a five-point likert scale where '1' stands for 'I definitely do not agree' and '5' stands for 'I definitely agree'. A cut-off score of 61 indicated individuals with misophonia.

The assessment of the severity of misophonia was done for individuals who fit the diagnostic criteria provided by Schröder et al. (2013) using the Revised Amsterdam Misophonia Scale (RAMISO-S) (Jager et al., 2020). The scale consists of 10 questions

with a rating from 0-4. The scores are classified as follows: 0-10 indicates no/subclinical misophonia, 11-20 indicates mild misophonia, 21-30 indicates moderate misophonia, and 31-40 indicates severe misophonia. Only individuals with scores above 11 were considered to have clinical misophonia and included in the study. Participants who completed the 12th standard and had sufficient English language proficiency to understand the questionnaire were alone included in the study.

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 carried out as a routine procedure to visually assess the integrity of the external auditory canal and tympanic membrane. Behavioral pure tone thresholds were determined 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 250 to 8 kHz were determined using TDH 39 supra-aural headphones (Telephonics, Farmingdale, NY, USA), and for bone conduction from 250 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 frequencies 500 Hz, 1 kHz, 2 kHz and 4 kHz, were carried out using a calibrated Grason-Stadler Incorporation Tympstar Pro immittance audiometer (Grason Stadler, Inc, MN, USA) to ensure normal middle ear functioning.

3.4.3 Frequency Following Response (FFR) Recording

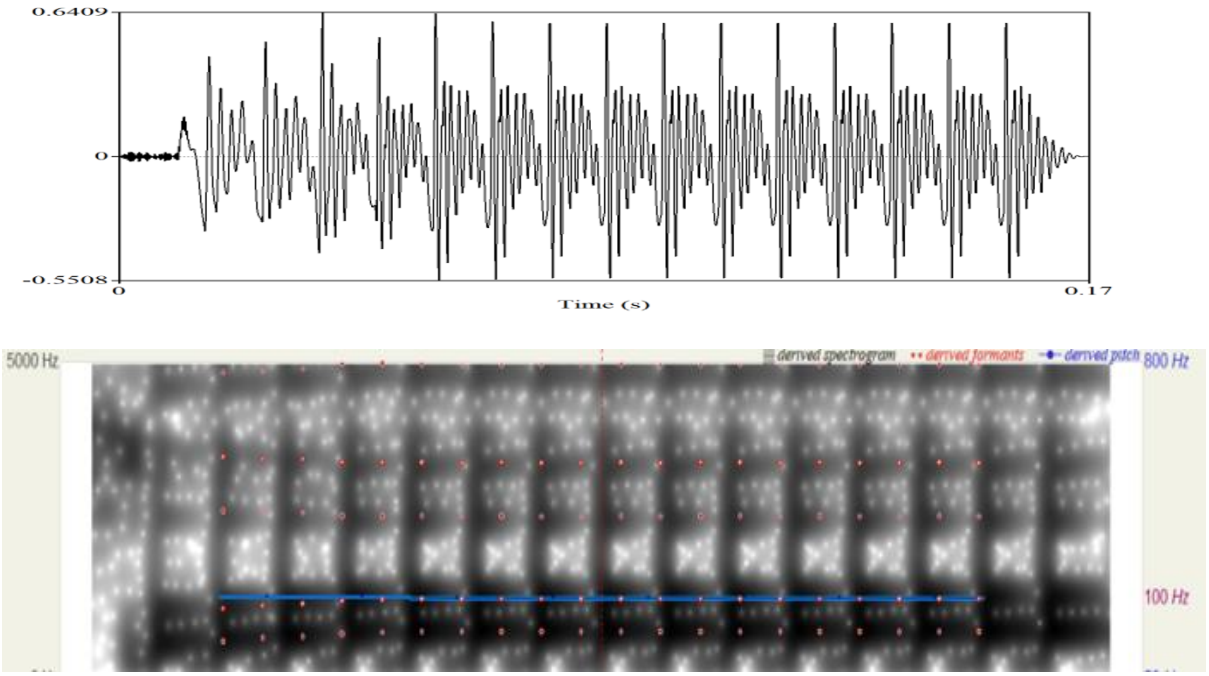
FFR was obtained using a calibrated four-channel auditory evoked potential system, Intelligent Hearing Systems (IHS) – SmartEP (Intelligent Hearing System Corp., Miami, FL).

Stimuli characteristics

FFRs were recorded using 170 ms /ba/ stimuli at an intensity of 70 dB SPL, with a repetition rate of 4.1 Hz, delivered binaurally using Insert earphones ER-3. Alternating polarity was used. The total number of averaged sweeps was taken as 1024. The waveform and spectrogram of the /ba/ stimulus used are given in Figure 3.1.

Figure 3.1

Waveform and spectrogram of 170ms /ba/ stimulus.



3.4.4 FFR measurement

Before commencing the test procedure, clear instructions were provided to the participants, outlining the steps of the procedure. During the recording, participants

were made to sit comfortably on a reclining chair in a sound-treated room. They were instructed to sleep and to minimize movements throughout the session. FFR was recorded using two channel electrode placements following the 10-20 classification (Jasper,1958) with active electrode at Cz, reference electrodes at left and right mastoids (linked) and C_7. The ground electrode was placed at Fpz. All the electrode impedances were kept below 5 kΩ.

Stimulus and acquisition parameters are given below in Table 3.2.

Table 3.2

Stimulus and acquisition parameters for the recording of FFR

Stimulus Parameters	
Type of Stimuli	/ba/
Duration	170
Transducer	ER-3 shielded Insert earphones
Repetition rate	4.1/sec
Intensity	70 dB SPL
Polarity	Alternating
Sweeps	1024
Mode	Binaural
Acquisition Parameters	
Recording time window	200 ms
Gain	1,00,000
Transducer	ER-3 Insert earphones
Filter	50-5000 Hz

Electrode montage	Inverting electrodes – Linked mastoid, C7
	Non-inverting electrode – Vertex (Cz)
	Ground – Lower forehead (Fpz)
Electrode impedance	< 5 kΩ (Absolute) and < 2 kΩ (Inter electrode)
No. of channels	2

3.4.5 FFR analysis

The FFR analysis was performed using customized scripts in MATLAB. The recordings were subjected to offline filtering with a high-pass filter of 75Hz and a low-pass filter of 2500Hz. Envelope (FFR_{ENV}) and temporal fine structure (FFR_{TFS}) components were extracted by addition and subtraction of the response in rarefaction and condensation polarities, obtained by splitting the active recording. Frequency-specific phase locked activity in the responses was obtained from the response spectrum, computed using Fast Fourier transforms (FFT). The parameters such as the F0 spectral amplitude and amplitude of harmonics were extracted from FFR_{ENV}. The amplitude of first formant frequency (F1) was extracted from FFR_{TFS}. These FFR parameters were compared between the two groups. The FFR parameters were also correlated with RAMISO-S scores.

3.5. Statistical analysis

The recorded data were tabulated in Statistical Package for the Social Sciences 26.0 (IBM Corp.; Armonk, NY, USA) and subjected to statistical analysis. The Shapiro-Wilk test was used to determine whether the data are normally distributed or not. The data were not normally distributed; hence, a non-parametric test, the Mann-Whitney U test, was administered. Spearmann's correlation was carried out to assess the

correlation between the scores of the Revised Amsterdam Misophonia Scale (RAMISO-S) and parameters of FFR in individuals with misophonia.

CHAPTER 4

RESULTS

This study examined the differences in the parameters of FFR between normal hearing individuals with and without misophonia. The study also investigated the correlation between the severity of misophonia and the FFR parameters.

4.1 F0 spectral amplitude and amplitude of harmonics

Descriptive statistics were used to summarize and describe all the parameters of FFR, such as mean, median, standard deviation and interquartile range, as shown in Table 4.1.

Table 4.1

Descriptive Statistics of FFR parameters for control and misophonia group

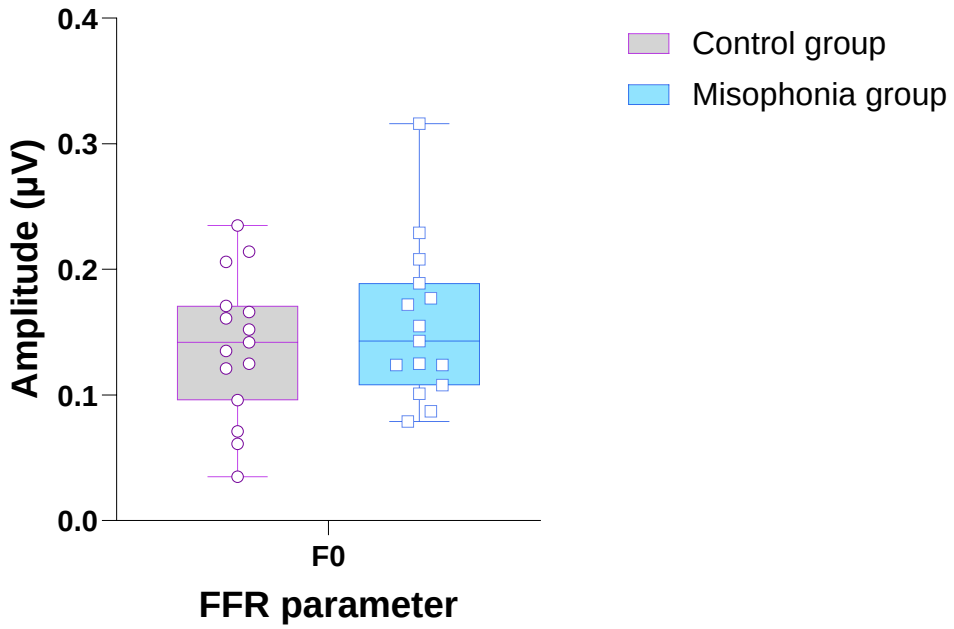
	Control group				**Misophonia group**			
	Mean	**Median**	**SD**	**IQR**	**Mean**	**Median**	**SD**	**IQR**
F0 amplitude	0.139	0.142	0.057	0.075	0.155	0.143	0.062	0.081
H2 amplitude	0.042	0.039	0.021	0.028	0.057	0.044	0.038	0.036
H3 amplitude	0.029	0.022	0.015	0.022	0.028	0.028	0.007	0.008
H4 amplitude	0.022	0.019	0.014	0.016	0.02	0.012	0.014	0.023
F1 amplitude	0.007	0.006	0.003	0.005	0.006	0.005	0.004	0.003

Shapiro-Wilk test was employed to check whether the data was normally distributed. The F0 amplitude parameter was found to be normally distributed ($p > 0.05$), and an appropriate parametric test was selected for further statistical analysis. All other parameters, such as the amplitude of harmonics, were not normally distributed ($p < 0.05$), hence an appropriate non-parametric test was selected for further statistical analysis. An

independent-samples t-test was performed to assess differences in F0 between the control and misophonia groups. The results showed no statistically significant difference between the groups [$t(28) = 0.751$, $p > 0.05$]. Box plot depicting the median, quartiles, minimum, maximum and individual data points for F0 amplitude is represented in Figure 4.1.

Figure 4.1

Comparison of mean and standard deviation for F0 amplitude across both groups



Mann-Whitney U tests were used to assess differences in harmonic amplitude between the control and experimental groups. The results revealed no statistically significant difference between the groups. The results of the inferential statistics are shown in Table 4.2. Box plot depicting the median, quartiles, minimum, maximum and individual data points for the amplitude of harmonics is represented in Figure 4.2.

Table 4.2

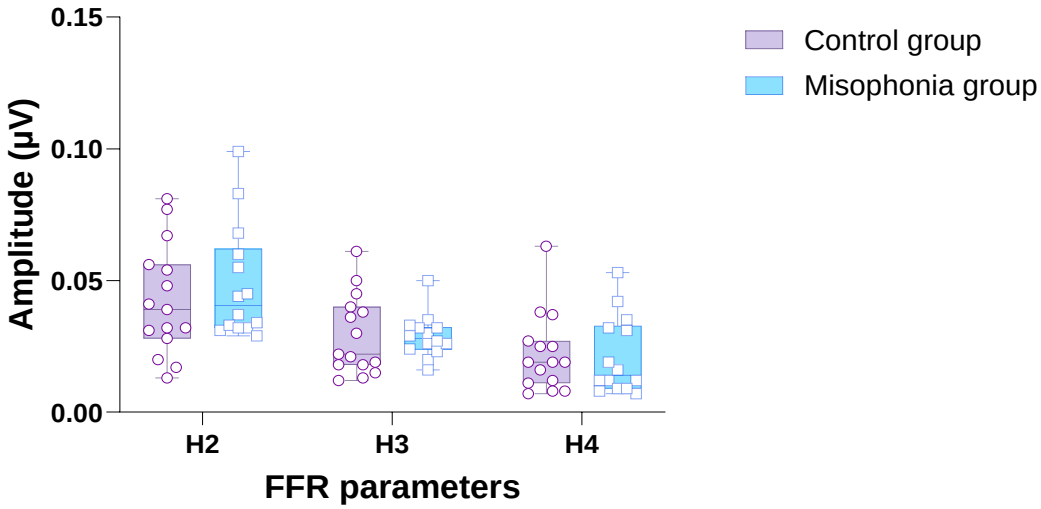
Mann Whitney U test Results for the Harmonics amplitude

	H2 amplitude	H3 amplitude	H4 amplitude
U	84.5	102.0	102.0
Z	1.16	0.44	0.44
p	0.24	0.66	0.66

Note: U- U value, Z – z score, *p* – *p* value/ Asymptotic significance

Figure 4.2

Comparison of median and standard deviation for amplitude of harmonics across both groups



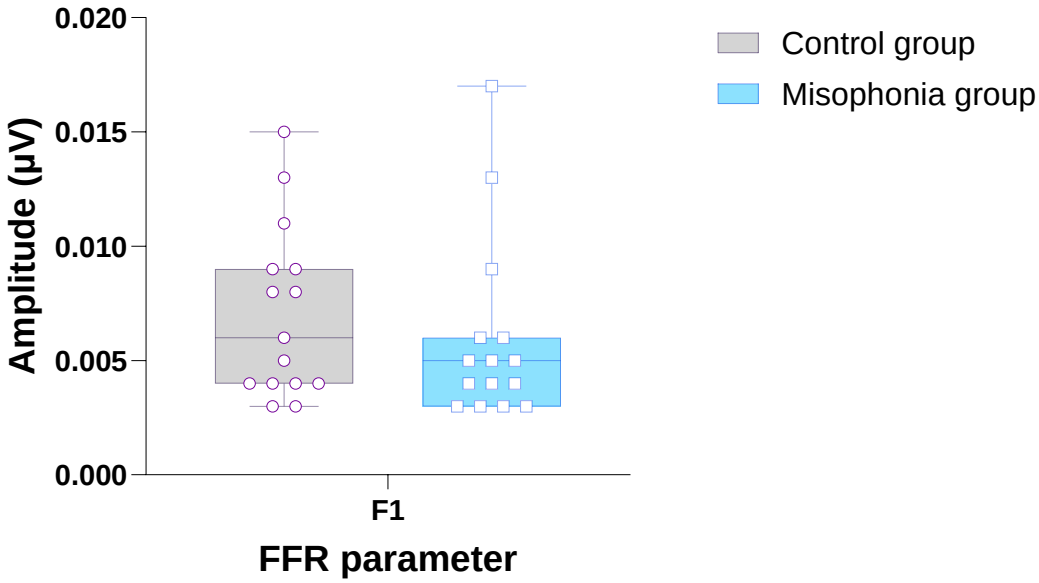
4.2 F1 spectral amplitude

The descriptive statistics of F1 amplitude across both the groups is given in Table 4.1. Shapiro Wilk test was administered to check for normality, and the data was found to be not normally distributed ($p<0.05$). An appropriate non-parametric test was selected for statistical analysis. Hence, Mann-Whitney U test was administered to check for significant difference between the control and misophonia groups. The result showed no

statistically significant difference [Z = 0.94, p = 0.34] between the two groups. Box plot depicting the median, quartiles, minimum, maximum and individual data points for F1 amplitude is represented in Figure 4.3.

Figure 4.3

Comparison of median and standard deviation for F1 amplitude across both groups



The grand average time waveforms and spectra of the envelope and fine structure across both groups are shown in Figures 4.4 and 4.5 respectively. The graphs clearly show that the responses are similar between the two groups.

Figure 4.4

Grand average time-domain waveform and spectrum of the envelope for the control and misophonia groups

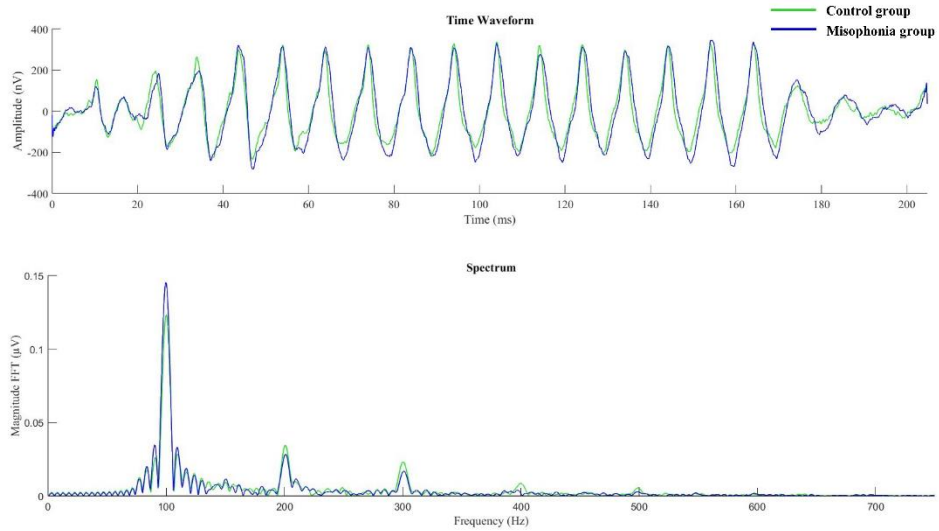
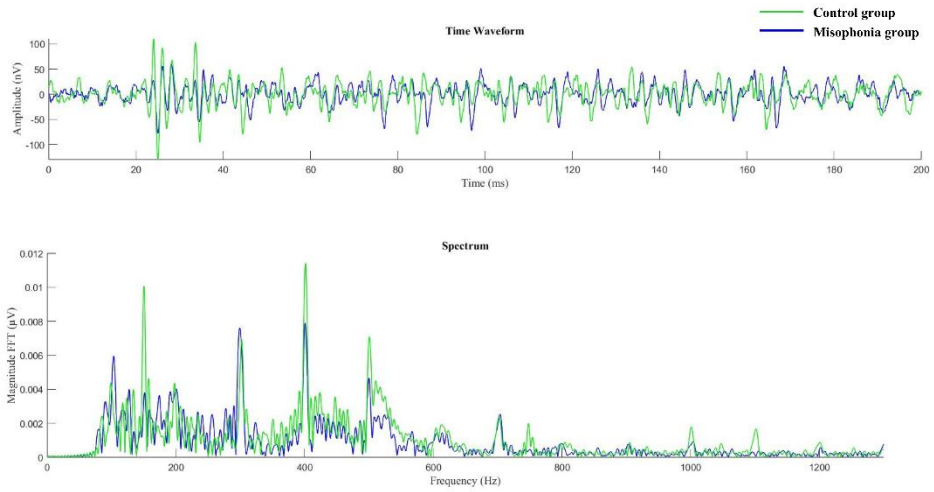


Figure 4.5

Grand average time-domain waveform and spectrum of the fine structure for the control and misophonia groups



4.3 Correlation between severity and FFR parameters in individuals with misophonia

The study also attempted to examine the correlation between the parameters of FFR in individuals with misophonia and their severity of misophonia. Spearman's rank correlation test was administered, and it showed no significant correlation for all the parameters with the severity of misophonia. The results of the correlation test are shown in Table 4.3.

Table 4.3

Correlation between severity of misophonia and FFR parameters using Spearman's Rank correlation test

FFR parameters	*Rho* ρ	Sig. (2-tailed)
F0 amplitude	0.23	0.41
H2 amplitude	0.01	0.96
H3 amplitude	0.46	0.08
H4 amplitude	0.06	0.84
F1 amplitude	0.33	0.22

Overall, the test results showed no statistically significant difference in FFR parameter values between the two groups. Also, there is no correlation between the severity of misophonia and the parameters of FFR.

CHAPTER 5

DISCUSSION

The present study investigated auditory encoding in individuals with misophonia using Frequency Following Responses (FFR). The results revealed no statistically significant differences between individuals with misophonia and controls in the spectral amplitudes of the fundamental frequency (F0), harmonic components (H2, H3, H4) of the envelope FFR, and the first formant frequency (F1) associated with fine-structure encoding. Furthermore, no significant correlations were observed between misophonia severity, as determined by the Revised Amsterdam Misophonia Scale (AMISOS-R), and any of the FFR parameters. These findings suggest that the neural encoding of complex auditory stimuli at the subcortical (brainstem) level remains largely preserved in individuals with mild-to-moderate misophonia.

The present findings contribute to the growing body of literature suggesting that misophonia may not primarily involve abnormalities in peripheral hearing sensitivity or early-stage subcortical auditory encoding, but rather dysfunctions in higher-order auditory-emotional integration networks (Aryal & Prabhu, 2023c; Jastreboff & Jastreboff, 2023; Melanthiou et al., 2026; Patro et al., 2025). FFR is considered a reliable electrophysiological measure of temporal and spectral sound encoding, reflecting phase-locked neural activity predominantly generated within the rostral brainstem, particularly the inferior colliculus (Anderson et al., 2013; Kraus et al., 2017). The absence of significant group differences in FFR measures indicates that the fidelity of the temporal envelope and fine-structure representation is not substantially altered in individuals with misophonia.

These findings are consistent with previous electrophysiological studies investigating auditory pathway integrity in misophonia. Aryal & Prabhu (2023a)

recorded auditory brainstem responses and reported no significant differences in absolute, interpeak latencies, and amplitudes of Waves I, III, and V between individuals with misophonia and controls, suggesting that the auditory pathway up to the brainstem is functionally unaltered. In a more recent investigation, Laag et al. (2025) also examined auditory brainstem responses in indivduals with misophonia and similarly found no significant group differences in the absolute latencies, interpeak latencies, or amplitude of Waves I, III and V. Furthermore, Karupaiah and Prabhu (2025b) observed no significant differences in Masking Level Difference (MLD) at 500 Hz, further supporting the notion of intact binaural interaction mechanisms at lower levels of the auditory brainstem. Since both ABR and MLD are measures of early auditory processing, the current FFR findings further strengthen the notion that basic auditory processing mechanisms remain largely unaffected in misophonia.

Nevertheless, the present findings should be interpreted in the context of contrasting evidence reported in the literature. Karupaiah and Prabhu (2025a) reported significantly shorter latencies for ABR waves III and V in individuals with misophonia, suggesting increased neural synchrony or possible subcortical hyperactivity. Notably, their study included participants with moderate-to-severe misophonia, whereas the current study predominantly included individuals with mild-to-moderate misophonia. The differences in participant characteristics may partly explain the inconsistency in findings. Methodological factors, such as the specificity of trigger profiles, the type of stimuli used, and the recording parameters, may also have contributed to the variation across studies. It is possible that neural alterations are more pronounced in individuals with severe misophonia or in those who show stronger emotional reactivity to trigger sounds (Karupaiah & Prabhu, 2025a). Accordingly, although the present findings indicate preserved auditory encoding at the brainstem level, they do not rule out subtle subcortical

changes that may become apparent under more complex experimental conditions or in individuals with greater symptom severity.

Growing evidence suggests that the neuropathophysiology of misophonia is more strongly related to abnormalities in cortical and limbic networks than to deficits in early auditory processing at the brainstem level (Aryal & Prabhu, 2023a; Jastreboff & Jastreboff, 2023). Aryal and Prabhu (2024a) reported significantly shorter P1 and N1 latencies in Auditory Late Latency Responses (ALLR), indicating atypical cortical auditory processing and altered neural responsiveness to sound. Likewise, Karupaiah and Prabhu (2025b) found poorer performance on the Dichotic Consonant-Vowel (DCV) and Pitch Pattern Sequence (PPS) tests in individuals with misophonia, suggesting reduced efficiency in higher-order auditory functions such as binaural integration, auditory discrimination, and temporal sequencing. Together, these findings suggest that although auditory signals may be encoded relatively normally at subcortical levels, abnormalities may emerge during cortical processing and perceptual interpretation.

This interpretation is supported by neuroimaging studies showing abnormal activation and connectivity within auditory-emotional networks in individuals with misophonia (Kumar et al., 2017; Neacsiu et al., 2022). Functional magnetic resonance imaging (fMRI) studies have consistently shown hyperactivation of the anterior insular cortex in response to trigger sounds (Kumar et al., 2017). The anterior insula, which is a key part of the salience network, plays an important role in emotional awareness, autonomic regulation, and the assignment of affective significance to sensory input. Altered functional connectivity involving the anterior insula, amygdala, hippocampus, anterior cingulate cortex, and ventromedial prefrontal cortex has also been reported, suggesting stronger coupling between auditory and limbic systems in individuals with misophonia (Kumar et al., 2017; Schröder et al., 2015, 2019; Neacsiu et al., 2022). Kumar

et al. (2021) further demonstrated enhanced connectivity between auditory, visual, and premotor regions during exposure to trigger sounds, even though no resting-state differences were observed in auditory cortex activity between the misophonia and control groups. These neural changes may help explain why sounds that are usually harmless can evoke unusually intense emotional, autonomic, and behavioral responses in individuals with misophonia.

The absence of significant correlations between AMISOS-R scores and FFR parameters further suggests that misophonia severity is not directly associated with abnormalities in early auditory encoding at the brainstem level. If misophonia mainly resulted from deficits in subcortical sound representation, greater symptom severity would be expected to correspond with progressively altered FFR measures. However, the lack of such associations suggests that the emotional and autonomic distress associated with misophonia is more likely driven by atypical cognitive-affective and auditory-limbic processing rather than by impaired acoustic encoding.

This interpretation is consistent with the neurophysiological model proposed by Jastreboff and Jastreboff (2001, 2014, 2023), which describes misophonia as a conditioned reflex disorder involving maladaptive interactions between the auditory system and limbic-autonomic networks. Within this framework, specific trigger sounds gain excessive emotional salience through associative learning, leading to heightened emotional and physiological responses despite otherwise normal auditory perception. Supporting this view, Edelstein et al. (2013) reported increased autonomic responses, including elevated skin conductance, in individuals with misophonia during exposure to trigger sounds, suggesting abnormal emotional arousal rather than impaired sensory detection. Similarly, Swedo et al. (2022) emphasized that misophonia is characterized by reduced tolerance to specific sounds, along with exaggerated emotional and

physiological reactions, rather than abnormalities in peripheral auditory sensitivity. Taken together, these findings support contemporary models that view misophonia as a disorder of abnormal salience attribution and affective regulation rather than a primary auditory deficit (Neacsiu et al., 2022; Savard & Coffey, 2025).

5.1 Limitations of the Study

- Despite its contributions, the present study has several limitations. First, the sample size was small, with only 15 individuals with misophonia included in the study. Although the use of stringent inclusion criteria strengthened internal validity, the limited sample may have reduced the statistical power needed to detect subtle group differences. In addition, all participants had mild-to-moderate symptom severity, so the findings may not generalize to individuals with more severe misophonia.
- Second, speech stimuli presented in quiet were used to elicit FFRs. Although such stimuli are commonly used in auditory neuroscience because they offer reliability and experimental control, they may not fully capture the complexity and emotional salience of real-world trigger sounds experienced by individuals with misophonia.
- Third, although FFR is a useful measure of neural encoding of acoustic signals, it cannot by itself distinguish the relative contributions of cortical and subcortical generators with complete precision. Recent evidence suggests that FFRs may reflect contributions from both brainstem and cortical sources (Coffey et al., 2017, 2019).

CHAPTER 6

SUMMARY AND CONCLUSIONS

The present study investigated auditory encoding in individuals with misophonia using Frequency Following Responses (FFR). Thirty normal hearing individuals were considered for this study. Out of which, 15 had misophonia identified based on inclusion criteria proposed by Schröder et al. (2013), and were considered as the experimental group and remaining 15 without misophonia were the control group. Severity of misophonia was assessed using the Revised Amsterdam Misophonia Scale (RAMISO-S). In addition, tinnitus, hyperacusis, and migraine were screened and eliminated using screening checklists, which are the Tinnitus Handicap Inventory (THI), Modified Khalfa Hyperacusis Questionnaire, and International Classification of Headache Disorders diagnostic checklist - 3 (ICHD 3, 2018) for Migraine, respectively. Routine audiological assessment was carried out involving Puretone audiometry, speech audiometry, immittance audiometry and reflexometry to rule out any ear-related pathology.

FFR was recorded using 170 ms /ba/ stimulus at an intensity of 70 dB SPL. Envelope (FFR_{ENV}) and temporal fine structure (FFR_{TFS}) components were extracted, and responses were computed using Fast Fourier transforms. The recordings were analysed using customized scripts in MATLAB. The results revealed no significant differences between individuals with and without misophonia in measures of temporal envelope and fine-structure encoding. Furthermore, no significant relationships were observed between misophonia severity and FFR parameters. These findings suggest that auditory brainstem encoding of acoustic information under quiet listening conditions remains largely preserved in individuals with mild-to-moderate misophonia. They also add to the growing view that misophonia is unlikely to arise from deficits in peripheral hearing sensitivity or early auditory processing. Instead, the condition appears to involve

atypical interactions among auditory, emotional, and salience-processing networks at higher cortical and limbic levels. By separating preserved sensory encoding from abnormal affective processing, the present study offers a more refined understanding of the neuropathophysiology of misophonia. This may help guide the development of targeted interventions that place greater emphasis on emotional regulation, neural connectivity, and maladaptive sound-emotion associations than on auditory mechanisms alone.

6.1 Implications of the Study

- The findings suggest that auditory brainstem encoding remains largely intact in individuals with mild-to-moderate misophonia.
- The results support contemporary neurophysiological models that view misophonia as a disorder of altered salience attribution and affective regulation rather than a primary auditory deficit.
- The study highlights the importance of extending clinical assessment beyond routine audiological evaluation.
- The study contributes to a better understanding of the neuropathophysiology of misophonia and may help inform targeted, evidence-based intervention strategies.

6.2 Future Directions

- Future studies should include larger and more diverse samples that represent the full range of symptom severity.
- Ecologically valid paradigms using individualized trigger sounds, competing background noise, degraded speech, or emotionally salient listening environments may provide deeper insight. Examining auditory encoding under these more demanding conditions may reveal subtle neural differences that are not apparent in quiet laboratory settings.

- Multimodal neurophysiological and neuroimaging approaches, such as high-density electroencephalography (EEG) and functional magnetic resonance imaging (fMRI), may offer a more comprehensive understanding of auditory-limbic interactions in individuals with misophonia.
- Longitudinal studies examining neural plasticity before and after therapeutic intervention may also improve understanding of the mechanisms underlying symptom change.

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