Could Transcranial Magnetic Stimulation Modulate the Neural Networks Underlying Misophonia?

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1. Introduction

Misophonia is a neurological disorder in which patients are troubled by specific sounds or visual signals and experience unusually strong negative reactions, leading to emotional, physiological, and behavioral symptoms. This definition was first put forth as a consensus definition by Swedo et al. (2020) employing a modified Delphi method to conduct the research. The Delphi method consists of organizing a 15-expert panel and voting four times on characterizing statements taken from 68 research articles on misophonia, and in each round, at least 80% of the panel experts were in consensus. While undeniably drawn from expert opinions, this definition of misophonia is based on opinionated experiences rather than experimental evidence, and the panel unfortunately failed to offer a detailed diagnostic criterion.

The two features of misophonia are that it is triggered by specific sounds rather than by loudness alone, and the response varies depending on the context and perceived sound sources. Woolley et al. (2025) compared 60 treatment-seeking adult patients with 60 matched controls group volunteers using audiometric and otoacoustic emission measurements and found no evidence of a peripheral hearing loss. Though without organic changes or lesions, misophonia can significantly disrupt patients' daily lives, as according to the 1,881 respondents of a study by Möllmann et al. (2023), 839 of them experienced functional impairment, and the more severe the symptoms, the greater the impairment.

When it comes to treatment, cognitive behavioral therapy (Jager et al., 2020) is widely considered a reliable treatment that can help patients adjust their thoughts and feelings about a particular sound. However, the actual effect of the above modifications on the basic neural process is unknown, and few studies in this field have examined whether treatments produce meaningful changes in neurological outcomes. This paper sets to investigate whether an intervention that directly alters cortical activity can serve as an effective adjunct therapy.

2. Misophonia as a neurobehavioral disorder

2.1 Establishing the baselines: The anomaly lies in the assessment, not the detection.

If misophonia were a hearing sensitivity disorder, this abnormality should appear during testing. However, two independent studies suggest it does not. In a 2022 experiment, Savard et al. (2022) presented 300 adults with neutral, unpleasant and trigger sounds in noisy, multi-person environments at five different signal-to-noise ratios and asked them to identify the sounds. Trigger sounds had a recognition threshold 2.3 dB and 9.9 dB lower than unpleasant sounds and neutral sounds, respectively. The results showed that the thresholds in groups displaying the most and least severe misophonia symptoms (n = 66 and 68, respectively) followed the

same pattern and were not statistically different ($F(1, 132) = 0.58$, $p = 0.884$). It was only after the sounds were adjusted to become distinguishable did differences emerge, with the most severe group showing a negative rating increase, particularly for trigger sounds ($d = 0.64$) and not for neutral sounds ($p = 0.760$).

Earlier research by Kumar et al. (2021) found the same results, during which sound-triggered functional magnetic resonance imaging (fMRI) were used to examine the volunteers' auditory cortex and found no statistically significant difference in their response to trigger sounds. Taken together, these research results clearly showed that misophonia is not due to an oversensitivity to sound overall, as the symptomatic responses only occur after the sound has been recognized, evaluated and assigned an emotional connotation.

2.2 Trigger sounds can produce measurable autonomic nervous system responses

Based on the results of previous electrodermal conductance studies (Edelstein et al., 2013), Grossini et al. (2022) measured heart rate variability and electrodermal conductance in 11 patients with misophonia and 44 healthy controls. The misophonia group had a higher power of low-frequency heart rate variability at rest ($p = 0.012$) and a lower parasympathetic index (0.451 vs. 1.347, $p = 0.001$). More significantly, the groups responded differently to the trigger sounds: those with misophonia showed sympathetic activation, and the sympathetic index in healthy controls decreased by 83.2 ± 31%. However, the groups were of different ages, and after controlling for age, the frequency-domain differences were no longer statistically significant; nevertheless, a general trend persisted.

Evidence for autonomic nervous system involvement is not consistent. Siepsiak et al. (2023) had 61 subjects and 45 controls, and only one significant difference in skin conductance was found by a non-parametric test. They also proposed that the changes in heart rate observed were more likely due to attention than emotions. Therefore, based on the above results, trigger sounds can cause physiological changes in people with misophonia. However, the evidence is still limited and may be influenced by the method of measurement or other factors, such as age and attention.

2.3 The response depends on the perceived source of the sound

People with misophonia are more likely to experience a stronger sense of dominance from mouth sounds linked to an inconsistent source. Siepsiak's team during their research presented the same 8-second recording both alone and with other consistent or inconsistent videos. The results showed that the same sound was rated as less negative and less arousing when it matched the video. However, these

effects were observed in the self-report data but not in skin conductance measurements. Another research by Berger et al. (2024) reviewed related studies and found that beliefs about the origin of a sound can affect both sound-aversion ratings and skin conductance responses. Mahzouni et al. (2024) have also found that linking trigger sounds with harmless visual cues reduced their impact.

In short, the above results indicate that a misophonic reaction is not solely due to the acoustic characteristics of a sound but also how that sound as well as its sources are perceived. Therefore, this essay argues that misophonia is an abnormal evaluation of sound rather than a simple hearing disorder caused by physical lesions or changes, and further research on the neurological mechanism is needed.

3. The neurological mechanisms of misophonia

Based on the above evidence, the neurological causes of misophonia are likely not based on a single neural pathway but rather a multi-layered process: the triggering sound is first encoded, identified and attributed to a particular source, then given excessive salience, integrated with interoceptive and emotional signals by the brain, and eventually converted into arousal and motor readiness.

3.1 Auditory cortex and superior temporal gyrus cortex

Schröder et al. (2019) compared 21 patients with misophonia and 23 healthy controls, and found that when exposed to saliva, the patients with misophonia showed increased activation in the right insula, anterior cingulate cortex and superior temporal gyrus. However, this increased activation was not induced by misophonia only, as other unpleasant sounds also triggered the same response. Kumar et al. (2021) found no significant difference in the auditory cortex's response between groups; Therefore, the auditory cortex may be responsible for processing the trigger, but it is unlikely to be the single origin of the abnormal responses, and later stages of sound assessment, such as salience and emotional processing, may be more appropriate targets of transcranial magnetic stimulation (TMS) tests.

3.2 Anterior Insula and Saliency Network

The anterior insula is a part of the brain that is closely related to misophonia. Kumar et al. (2017) found that the anterior insula exhibited a stronger response to triggered sounds as symptom severity increased. They have also reported enhanced connectivity between the anterior insula and the ventromedial prefrontal cortex, as well as the posteromedial area, hippocampus and amygdala. Hansen et al. (2026) also carried out a recent study. Resting-state functional magnetic resonance imaging (fMRI) data from 162 adults were analyzed, and a model trained on an independent

sample of 777 adults was used to assess the degree of misophonia. They found increased connectivity among the anterior insula, temporal plane, operculum, precentral gyrus and supplementary motor area with increasing severity of misophonia. Two results are relatively significant. First, this effect is anatomically specific: it was found only in the salience/ventral attentional insula among the six insular subregions and could not be explained by anxiety, depression or autism traits. Secondly, the relationship is spectral rather than categorical. When the misophonia and control groups were divided, no significant clusters of brain regions were found, suggesting that neural patterns change continuously with the degree of symptoms rather than forming two distinct groups in the population.

In short, the above results indicate that the anterior insula may be involved in enhancing the salience of trigger sounds and connecting them with emotional and physical responses. More importantly, this connection involves multiple brain regions and not just abnormal activities in the insula, and thus a network-based diagnostic explanation for misophonia is potentially more productive.

3.3 Anterior cingulate cortex (ACC)

The anterior cingulate cortex (ACC) is also activated by misophonia in people. Schröder et al. (2019) have discovered that after being exposed to a trigger sound, patients with misophonia show increased ACC activity. This activity may be related to regulating emotional responses or to social discomfort caused by others' sounds (Neacsiu et al., 2022). Jain et al. (2026) also found that connectivity between the ACC and the frontal pole was negatively correlated with the severity of misophonia in all subjects (rho = −0.385, adjusted p = 0.0023). In other words, stronger connections among these areas were associated with lower misophonia scores. However, this correlation may be due to differences in the subject groups. Based on the above results, the ACC may be responsible for regulating emotional and social responses to a trigger sound rather than detecting the sound itself. However, the current evidence is mainly correlational; therefore, the precise reason why this factor causes or regulates misophonia responses has not been identified.

3.4 Sensorimotor dysfunction and unresolved controversies

Kumar et al. (2021) believe that misophonia may be an "over-mirroring" of oral and facial movements that produce sound. In a sample of 33 people, increased resting connectivity was found in the auditory cortex, visual cortex and ventral premotor cortex, as well as enhanced responses of the orofacial motor cortex to trigger sounds. Hansen et al. (2022) also found a small change. In 19 adults with mild, non-clinical misophonia, motor localization tests showed that misophonia involves a larger sensorimotor system and is not limited to responses in the oral and facial areas. The problem of whether the sensorimotor involvement is an “over-mirroring”

of the sound producer's oral or facial movement has not been solved, but both studies indicate that the sensorimotor system is involved in triggering misophonia. Hansen et al. (2026) have found that misophonia is related to the precentral gyrus and the supplementary motor area, which is consistent with the above results. Therefore, the above results suggest that the response to misophonia may involve both the motor system and auditory-emotional processing. However, it is still unknown whether this is a particular reflection of the movements that produce sound or a general sensorimotor response.

3.5 Networks rather than individual brain regions

The first type of evidence indicates that misophonia may be better explained by differences in brain networks rather than by abnormalities in a particular area of the brain. Jain et al. (2026) compared the task-based functional magnetic resonance imaging (fMRI) responses to standardized emotional sounds in people with misophonia, hyperacusis, both conditions, and healthy controls (N = 92). They identified a whole-brain activation cluster in the left visual association cortex ($F(3,87) = 11.39$, $pFWE = 0.0038$, $k = 962$). Misophonia was associated with increased visual activity and reduced connectivity between the visual and salience networks, and hyperacusis was linked to reduced salience-prefrontal connectivity. Both conditions also had stronger prefrontal-motor connections. Based on the above results, the two conditions share some neural features but have different connectivity patterns. However, the study used generally unpleasant sounds instead of personalized misophonia triggers, and thus the direct applicability of the results to misophonia is limited.

In short, the evidence this essay presented throughout Sections 3.1-3.4 have unanimously pointed toward a network-based explanation of misophonia, because the condition involves three different neurological mechanisms: the abnormal connections between the auditory and sensorimotor systems, the salience and interoceptive network centered on the anterior insula, and the regulating prefrontal cortex areas. A network-based explanation is thus more suited for the TMS hypothesis to help build an effective multi-area diagnosis.

4. Current treatment methods and their limitations

Jager et al. (2020) conducted the first randomized controlled trial of a treatment for misophonia. Out of the 71 recruited patients, 54 were randomly assigned to one of the three-month weekly group cognitive behavioral therapy (CBT) arms or a waitlist control group, and 46 patients (85%) completed the study. It had a strong effect. The Amsterdam Misophonia Scale–Revised scores in the CBT group decreased by 9.7 points (95% CI -12.0 to -7.4; $d = 1.97$) compared with only a 0.8-point reduction in the control group. In addition, 37% of the CBT group reported that their symptoms

had improved much or very much (Clinical Global Impression–Improvement scale) while 0% of the control group did so ($p < .001$). General psychological symptoms also improved (SCL-90-R, $d = 0.86$), and this treatment effect maintained at the 12-month follow-up.

However, this study also has some deficiencies. Firstly, the control group was placed on a waitlist instead of receiving active treatment or a placebo and consequently the expectation and treatment-contact effects had not been fully controlled. Secondly, only 37% of the patients were classified as much or very much improved in the intention-to-treat analysis and lastly, although disability in social and family life improved, there was no significant quality-of-life improvement as measured by EQ5-D and WHOQoL-BREF, the self-reported outcome instruments designed to evaluate broad domains such as mobility, self-care, psychological well-being, and social functioning.

Nonetheless, CBT helps adjust a patient's cognitive interpretation of a trigger sound, thereby reducing emotional arousal. As the evidence in Section 2 showed, misophonia has to do with how patients perceive the sound, which can be alleviated by CBT treatment. What awaits further research, though, is whether CBT treatment could induce significant changes in the neurological responses mentioned in Section 3, as no such results have been measured by previous treatment studies.

In short, the above evidence shows that CBT can alleviate symptoms of misophonia, but we do not know how it affects the neural networks of patients diagnosed with misophonia. This essay proposes looking into TMS as a potential adjunct treatment and its validity in modulating cortical activities.

5. TMS and its potential application to misophonia

5.1 What is TMS?

TMS is the treatment approach that employs magnetic pulses to non-invasively induce electric currents in the cortex and modify the brain’s neuronal excitability (Sack et al., 2023). This essay suggests TMS as an applicable treatment to misophonia as the evidence presented in Section 3 suggests misophonia is likely caused by abnormal connections between brain networks.

It is necessary to keep in mind that TMS-generated magnetic fields are local and near the cortex surface, about 2 cm beneath the scalp (Neacsiu et al., 2022), and may be unable to directly stimulate deeper regions associated with misophonia such as the anterior insula. Therefore, this essay will now explore if surface cortical stimulations can indirectly change the neuronal network that causes misophonia.

5.2 Why TMS could work for misophonia

There are several reasons why it may be so. First, the effect area of TMS is not limited to the region directly under the stimulation coil. Neacsiu et al. (2022) pointed out that prefrontal stimulation can alter activity and connections in deeper areas, such as the insula, anterior cingulate cortex and amygdala.

Oathes et al. (2023) are typical of such a network effect. Among the 36 depressed patients, based on its functional connectivity with the subgenual anterior cingulate cortex, an accessible cortical target was selected. Combined TMS and fMRI showed that stimulation of this cortical target produced a response in the deeper cingulate area. Although the depression symptoms decreased by 34% after three days of stimulation, as there was no sham control group, it could not be confirmed that the improvement was due to TMS. However, this study has also found that stimulating an accessible cortical node can influence a linked deep-brain region.

He et al. (2023) has provided evidence that is more directly related to the proposed mechanism. In 117 adults receiving either active or sham 10 Hz stimulation of the ventrolateral prefrontal cortex, active stimulation increased activity in the prefrontal cortex and decreased activity in the amygdala and insula. Therefore, the analysis suggested that the impact on the insula occurred indirectly via the ventromedial prefrontal cortex.

The anterior insula is one of the areas that have been repeatedly linked to misophonia in the above studies. If an accessible cortical region connected to the anterior insula can be identified, TMS may thus be able to modulate the misophonia-related network without directly stimulating the insula.

5.3 Proposed hypothesis

Based on the above evidence, this essay proposes that TMS could reduce misophonic responses by activating an accessible cortical area that is functionally connected to the anterior insula and the broader salience network. The hypothesis follows from the neural model that misophonia is caused by altered connections among the auditory and sensorimotor systems, the anterior insula-centered salience network, and prefrontal regulatory areas. Therefore, a treatment that modifies the network connections may be more suitable than one targeting a single brain area.

The targets for different people may also vary. Lynch et al. (2022) have developed a way that integrates individual brain-network mapping with electric-field modelling for optimization of TMS coil placement. On the other hand, Balderston et al. (2024) was also able to determine that the modelled electric field direction predicted changes in functional connectivity after stimulation. Based on these results, functional connectivity can be leveraged to find a suitable and accessible target for each patient. Another important factor is the person's mental state during stimulations. He

et al. (2023) found that the decrease in insula and amygdala activity occurred during cognitive reappraisal, not in the baseline condition.

Taken together, this essay proposes that TMS can be administered while the patient is exposed to a trigger sound and actively reappraises it. This may activate the relevant network during TMS application and thus alter the neural connections involved in the misophonic response. Instead of replacing CBT, TMS complements psychotherapy and works to alter brain function while cognitive therapies gradually adjust a patient's experience of a trigger.

6. Limitations of the hypothesis

Despite the promising potential, there is no scientific research substantively proving that TMS can treat misophonia. This is the most significant deficiency of the hypothesis. Most of the evidence for TMS comes from studies of depression, OCD, and other psychiatric disorders.

Moreover, the function of the anterior insula remains unknown to neurologists. Neuroimaging studies have thus far shown that activity and connectivity in the anterior insula are associated with misophonia and symptom severity, but these studies have not identified a causal relationship

There are also problems with connectivity-based targeting. Elbau et al. (2023) analyzed data from 295 depressed patients and found only a weak relationship between connectivity in the stimulated cortex and treatment results ($r = -0.16$), with about 3% of the variation explained. Indeed, clinical practitioners should be cautious as it's still too early to conclude that individual functional connectivity predicts an effective TMS target. The problem may be even more severe in misophonia, and many neuroimaging studies have relatively small sample sizes.

TMS is not always more effective in combination with trigger exposure or cognitive reappraisal. Lantrip et al. (2022) reported a trial combining exposure with active deep TMS failed to outperform sham stimulation, and the trial was discontinued due to poor efficacy. Therefore, the activation of a suitable psychological state during stimulation is not sufficient for TMS to work.

7. Conclusion

Based on the current evidence, misophonia is better understood as a disorder of abnormal evaluation and networked responses to specific sounds. The anterior insula and salience network are relatively more prominent; however, auditory, sensorimotor, anterior cingulate and prefrontal systems may also be involved.

TMS as a treatment approach shows promise and can be employed to ascertain whether the neural networks are involved in producing a misophonic response. As

the anterior insula is too deep for direct targeting by conventional TMS, I propose stimulating an accessible cortical area that is functionally connected to the anterior insula and the salience network.

However, this is still a hypothesis and lacks the support of clinical data. At present, there is no direct clinical evidence that TMS reduces the symptoms of misophonia, and the proposed mechanism is based mainly on correlational neuroimaging data and evidence based on treatment of other diseases. Existing evidence provides a rational scientific basis for further research on whether network-targeted TMS can reduce misophonic responses in controlled studies.

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