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Toward cognitive models of misophonia

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ABSTRACT

Misophonia is a disorder in which specific common sounds such as another person breathing or chewing, or the ticking of a clock, cause an atypical negative emotional response. Affected individuals may experience anger, irritability, annoyance, disgust, and anxiety, as well as physiological autonomic responses, and may find everyday environments and contexts to be unbearable in which their ‘misophonic stimuli’ (often called ‘trigger sounds’) are present. Misophonia is gradually being recognized as a genuine problem that causes significant distress and has negative consequences for individuals and their families. It has only recently come under scientific scrutiny, as researchers and clinicians are establishing its prevalence, distinguishing it from other disorders of sensory sensitivity such as hyperacusis, establishing its neurobiological bases, and evaluating the effectiveness of potential treatments. While ideas abound as to the mechanisms involved in misophonia, few have coalesced into models. The aim of the present work is to summarize and extend recent thinking on the mechanistic basis of misophonia, with a focus on moving towards neurologically-informed cognitive models that can a) account for extant findings, and b) generate testable predictions. We hope this work will facilitate future refinements in our understanding of misophonia, and ultimately inform treatments.

1. Introduction

Misophonia is a disorder of decreased sound tolerance (Swedo et al., 2022) involving hypersensitivity to specific sounds (e.g., eating, sniffing, or clicking; Brout et al., 2018; Jastreboff and Jastreboff, 2014; Schröder et al., 2013). The sounds are typically referred to as ‘misophonic sounds/stimuli’ (or ‘trigger sounds’), and generate a ‘misophonic response’ characterized by a strong negative emotional state and heightened physiological responsivity (Brout et al., 2018). People with misophonia may experience anger, irritability, annoyance, disgust, and anxiety, as well as physiological autonomic responses. As affected individuals often find everyday environments and contexts in which triggers are present to be unbearable, misophonia can cause significant distress and has negative consequences for individuals and their families. It has only recently come under scientific scrutiny, as researchers and clinicians are establishing its prevalence, distinguishing it from other disorders of sensory sensitivity such as hyperacusis, establishing its neurobiological bases, and evaluating the effectiveness of potential treatments.

The emerging picture is that misophonia is a complex neurophysiological condition (Brout et al., 2018; Cavanna and Seri, 2015) with an auditory focus that is distinct from related sensory sensitivity problems and causes significant negative consequences for a surprisingly large proportion of the general population. While we have made considerable progress in documenting and characterizing misophonia, the theoretical framework surrounding misophonia’s neurobiological and cognitive basis is not yet well-developed. In effect, this field lacks models. Models can accelerate progress

across basic and clinical research by encouraging scientists to make assumptions explicit, which facilitates their rigorous examination and development into better, more useful models (Blohm et al., 2020). The overall aim of the present work is to summarize and extend recent thinking concerning misophonia, with a focus on neurologically-informed cognitive models that are parsimonious, can account for extant findings, and generate testable predictions that will facilitate further refinements in our understanding of misophonia.

Particularly as misophonia is a newly-defined issue (Swedo et al., 2022) and its phenomenology is complex, our models must be well-grounded in clinical observations and individuals’ experiences. We focus on models which address ‘what’ and ‘why’ questions concerning specific differences in perception and cognition in people with misophonia as compared with normative controls. For the most part, we avoid including etiological factors, as there is little work available to date that bears on development of misophonia (but see Mednicoff et al., 2022 for an overview of that which exists, and Palumbo et al., 2018 for a summary of how associative and non-associative learning principles may be relevant for the development, maintenance, and perhaps treatment of misophonia). Additionally, creating models of a single state, such as having a disorder in adulthood, seems like a logical precursor to models of dynamic states, such as the process of developing a disorder. We also do not attempt to generate computational models, which use mathematical approaches to simulate neurocognitive processes at various levels of organization from single membrane channels to distributed whole-brain networks. These types of models may eventually prove to be useful if not essential in understanding misophonia, but typically presuppose that their creators have clearly isolated the phenomenon of interest (Blohm et al., 2020); this condition is not yet met in misophonia

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research. Thus, we suggest that models should for the time being be conceptual in nature, describing how differences in particular cognitive functions may be implicated in contributing to misophonic reactions. Finally, we propose that models should be situated spatially within the brain. Taking advantage of newly available neuroimaging findings will allow us to connect misophonia with a wider body of existing knowledge concerning brain structure and function.

In this work, we first briefly summarize what is known about misophonia to date, with the goals of familiarizing researchers who are unacquainted with the disorder with its characteristics, and referring readers who wish to have more in-depth information to some of the recent publications that have comprehensively reviewed specific aspects of misophonia. An overview of factors described in these sections which are potentially relevant for developing neurocognitive models of misophonia is found in Table 1. In subsequent sections, we review current knowledge on the neural basis of misophonia. We then describe two published models of misophonia, discussing their explanatory power concerning existing results and contrasting their respective predictions. Finally, we reflect on how models of misophonia can be improved and developed. We hope this work will facilitate future refinements in our understanding of misophonia, and ultimately inform treatments.

2. Overview of misophonia

2.1. Recent progress on misophonia

As recently as a decade ago, misophonia was a foreign term even to most auditory researchers and clinicians, and research on misophonia was nearly non-existent. In the interim, a considerable amount of empirical work has taken place, and over 30 reviews published between 2014 and 2024 have indexed the progress made in this emerging field. While some of these reviews provide general overviews of misophonia's core characteristics and clinical presentations (e.g., Bruxner, 2016; Cavanna and Seri, 2015; Siepsiak and Dragan, 2019), others have taken a more specialized approach, focusing on clinical implications and treatment approaches (Brout et al., 2018; Erfanian et al., 2019b; Mattson et al., 2023), associated comorbidities (Rinaldi et al., 2023; Williams et al., 2021), as well as neurobiological mechanisms (Neacsu et al., 2022) and underlying frameworks (Berger et al., 2024; Palumbo et al., 2018). Reviews have also explored misophonia alongside other auditory disorders, mostly related to treatment approaches (Henry et al., 2022; Jastreboff and Jastreboff, 2014, 2015) and assessment tools (Kula et al., 2022). More recent systematic reviews have identified trends in the literature, revealing patterns in prevalence and variability in diagnostic approaches (Aryal and Prabhu, 2023; Ferrer-Torres and Giménez-Llort, 2022; Gowda and Prabhu, 2024). Notably, a committee of experts has recently established a consensus definition of misophonia, recognizing it as a disorder and defining its main features in terms of misophonic stimuli, responses, and associated functional impairments (see Swedo et al., 2022).

2.2. Prevalence & comorbidity

A fundamental question in misophonia research concerns establishing who is affected by it. Multiple studies across different populations and cultures (Jager et al., 2020; Naylor et al., 2021; Rouw and Erfanian, 2018; Siepsiak et al., 2020b; Vitoratou et al., 2023; Wu et al., 2014; Zhou et al., 2017) estimate that between 5% and 20% of people experience clinically significant symptoms of misophonia. These estimates are derived using different diagnostic questionnaires and tools, which leads to some variability in reported prevalence rates (see Gowda and Prabhu, 2024 for a review of prevalence studies). In addition, some research suggests a higher prevalence of misophonia in females, though the samples on which these observations were based primarily consisted of female university students, with 67–84% female; Brennan et al., 2024; Kılıç et al., 2021; Wu et al., 2014; Zhou et al., 2017, and large-scale studies with more balanced samples show no such difference (Savard et al., 2022; Vitoratou et al., 2023). Several studies have also suggested that younger individuals are more likely to experience misophonia (Kılıç et al., 2021; Vitoratou et al., 2023), though this observation could be influenced by the predominantly younger samples in these studies, and potentially that older people might have developed coping strategies and adjusted their lives so as to reduce exposure to misophonic sounds and thus report lower severity. Across the literature, misophonia is increasingly viewed as existing along a spectrum, where a large portion of the population experiences subclinical symptoms. Notable examples include Naylor et al. (2021) who found mild, moderate and severe misophonia symptoms in 37%, 12%, 0.3% of the sample, respectively; Zhou et al. (2017) who observed that 68% of people reported experiencing such sub-clinical misophonia symptoms; and Savard et al. (2022) who found that misophonia symptoms are normally distributed, suggesting that mild symptoms occur in many individuals who may not experience significant distress. These observations regarding the distribution of misophonia overall support the notion that those who experience more severe symptoms represent the tail end of a broader spectrum of sound sensitivity (Savard et al., 2022).

Comorbidity is common among individuals with misophonia, with various studies identifying associations with different mental health conditions. Anxiety disorders are reported, including generalized anxiety, posttraumatic stress-disorder and obsessive-compulsive disorder (all between 1–3% in Jager et al., 2020 but up to 13% in Rouw and Erfanian, 2018); see Wu et al. (2014) and Cusack et al. (2018) for investigations of the relationship between misophonia and obsessive-compulsive symptoms as well as anxiety symptoms and anxiety sensitivity. Additionally, obsessive-compulsive personality disorder (OCPD; personality disorder characterized by perfectionism and rigid control; American-Psychiatric-Association, 2013) is prevalent in people with misophonia, with studies indicating that self-reports of OCPD in misophonic samples range from 8 (Rouw and Erfanian, 2018) to 26% (Jager et al., 2020), while studies using professional mental health assessments

show comorbidity rates with OCPD in up to 52% of people Schröder et al. (2013). Comorbidity with autism spectrum conditions (2% of misophonics in Jager et al., 2020; 4% in Claiborn et al., 2020; see Williams et al., 2021 for a review of misophonia in autism) and autistic traits (see Rinaldi et al., 2023) has also been observed, as well as with mood disorders (Erfanian et al., 2019a; Schröder et al., 2013; 10% of the sample in Jager et al., 2020). It is worth considering how misophonia may contribute to some of the comorbid diagnoses. For instance, an adolescent unable to avoid misophonic triggers in a classroom might experience heightened anxiety or inattention than they would have if they did not experience misophonic reactions to classroom sounds (or could control their acoustic environment).

Misophonia can also coexist with other auditory disorders relating to sound hypersensitivity (e.g., tinnitus, hyperacusis, noise sensitivity, and phonophobia; Jastreboff and Jastreboff, 2014), though research indicates distinctions between the disorders (see (Henry et al., 2022) for a review). In brief, hyperacusis is characterized by discomfort or pain in response to loud sounds; noise sensitivity refers to increased reactivity to sounds that may include general discomfort due to a perceived noisy environment, regardless of its loudness; and tinnitus involves distress from internally perceived sounds (Cavanna and Seri, 2015); these conditions contrast with the misophonic response being tied to specific, idiosyncratic misophonic sounds in the environment that are problematic even at low intensities (Henry et al., 2022), and are independent of the sound's physical properties (e.g., sound level, frequency, harshness, decibel level; Jastreboff and Jastreboff, 2002).

While comorbidity is common, misophonia often exists independently of other disorders (Schröder et al., 2013). In a recent study in which 575 individuals with misophonia completed clinical interviews, about 72% of the sample had misophonia without a comorbid psychiatric disorder, and 59% had misophonia without a comorbid personality disorder. In addition, around 94% of the sample did not report other hearing problems or disorders (Jager et al., 2020). Although the mechanisms by which conditions overlap could be very informative, differential diagnosis, characterizing the sample population and for some investigations, isolating the monodagnostic or primary misophonia population, will likely prove critical to understand aspects of neurobiology and cognition that are unique to misophonia.

These observations concerning prevalence and comorbidity have a few implications for developing models of misophonia. 1) Misophonia seems to lie on a continuum in the general population; we therefore might expect the underlying neuroanatomy and cognitive characteristics to vary in a continuous rather than categorical fashion with respect to healthy controls. 2) Although males with misophonia seem to be harder to recruit into studies, the disorder itself does not appear to be strongly tied to sex-related genetic or neurological differences. 3) The considerable comorbidity with other disorders suggests that some of the empirical results which we might incorporate into models could be explained

by their differential inclusion into misophonic and control groups (for a more detailed discussion of the similarities and differences between misophonia and these disorders, see the review by Ferrer-Torres and Giménez-Llort, 2022), such that tools for quantifying and documenting misophonia, as well as comorbid conditions, become critical components in the study of misophonia.

2.3. Measuring misophonia

There are now numerous questionnaires available for assessing misophonia, reflecting a growing understanding of the disorder. Earlier measures include the Misophonia Questionnaire (MQ; Wu et al., 2014) and the Amsterdam Misophonia Scale (A-MISO-S; Schröder et al., 2013), which have been used extensively in research. The psychometric properties, strengths, and limitations of some of these early misophonia scales (including the MisoQuest; Sieppiak et al., 2020a) were evaluated in Kula et al. (2022). More psychometrically-validated scales have recently become available such as the Selective Sound Sensitivity Syndrome Scale (S-Five; Vitoratou et al., 2021), the Duke Misophonia Questionnaire (DMQ; Rosenthal et al., 2021), the Duke-Vanderbilt Misophonia Screening Questionnaire (DVMSQ; Williams et al., 2022), the Berlin Misophonia Questionnaire Revised (BMQ-R; Remmert et al., 2022), the Sussex Misophonia Scale (Rinaldi et al., 2021; Simner et al., 2024), and Misophonia Impact Questionnaire (MIQ; Aazh et al., 2023). While results from these scales are generally correlated (e.g., see Rosenthal et al., 2021 for correlations of the DMQ with other scales), they are not entirely equivalent. For example, the S-Five is a quick measure used to capture subfactors of misophonia experiences, the MQ focuses more on trigger frequency and emotional responses, and the DMQ is a longer, more comprehensive measure which includes many individual subscales to be used independently or together. Each measure may exhibit some relative bias reflecting the researchers' focus and understanding of misophonia (noting that most of the scales were developed prior to the consensus definition; Swedo et al., 2022). For example, the MisoQuest assesses only anger reactions and overlooks other emotions common in misophonic responses, such as disgust, while the A-MISO-S is based on earlier diagnostic criteria and adapted from measures used in Obsessive-Compulsive Disorder research, which may not fully capture the emotional and behavioural patterns specific to misophonia. The variety of scales available, the range of symptoms they target, and the fact that they may not agree (e.g., in Hansen et al., 2024) can complicate cross-study comparisons and the interpretation of findings.

As regards separating primary misophonia from more complex cases, it is necessary to have short, efficient measures of the main comorbid conditions. Unfortunately, there is currently no consensus on the best battery of questionnaires to achieve this differentiation. Rinaldi et al. (2021) point out that most misophonia assessments lack divergent validity, making it difficult to separate misophonia from other disorders that also primarily involve hypersensitivity

sound and can present similarly, notably hyperacusis. Questionnaires used to assess hyperacusis do not differentiate it from misophonia, and to date only Enzler et al. (2021)'s online psychoacoustic test has been proposed as a short and reliable tool for separating primary misophonia from hyperacusis. As it stands, misophonia scales primarily assess the severity of misophonia itself, without providing clear differentiation from other psychiatric or developmental conditions that may overlap in symptoms. Alternatives include a complete clinical interview (e.g., in Guetta et al., 2024; Jager et al., 2020), or relying on potential study participants reporting on concurrent diagnoses. Noting that as with misophonia, the characteristics of many disorders also exist on continua, it may be preferable for some applications to measure subclinical tendencies rather than rely on categorical distinctions.

As the field develops, the relative merits and biases of misophonia scales, scale cut-off points for determining different levels of disorder, and the best means of differentiating primary misophonia from more complex cases within study constraints will likely become more standardized. For instance, Möllmann et al. (2023) identified cutoffs for various symptom measures that distinguish between functional impairment and non-impairing sound sensitivity. For the present purpose of model development, it is important to be aware that inconsistencies in measuring misophonia and separating experimental participants into groups exist, and to consider how scales and study design choices might have affected the empirical results that models attempt to account for.

3. Potential insights into the mechanisms of misophonia

3.1. Brief overview of misophonia phenomenology

Misophonia literature shows that triggers encompass a wide range of stimuli, primarily auditory, including orofacial sounds, human-made noises, and sometimes visual stimuli. The most common trigger sounds are orofacial in nature (i.e., produced by the mouth, nose, throat), though individuals report distress from a variety of other sounds that are not either non-orofacial or not produced by humans, including barking dogs, clinking glasses, clocks ticking, feet shuffling, doors slamming, and refrigerators humming (Enzler et al., 2021; Ferreira et al., 2013; Hadjipavlou et al., 2008; Hansen et al., 2021, 2022; Jager et al., 2020; Jastreboff and Jastreboff, 2014; Johnson et al., 2013; Neal and Cavanna, 2013; Vitoratou et al., 2021; Webber et al., 2014). Many people with misophonia report that while misophonic reactions to real-life experiences are most problematic, responses can be triggered by sounds from television or videos (Rouw and Erfanian, 2018).

The acoustic patterns of misophonic sounds and the resulting emotional responses vary significantly among individuals, suggesting that personal history, learning, and context play crucial roles in shaping aversive reactions (Brout et al., 2018; Jastreboff and Jastreboff, 2001; Palumbo et al.,

2018). Even imagining a trigger sound can provoke the same aversive reaction as hearing the actual sound (Ferrer-Torres and Giménez-Llort, 2021), further suggesting that history with the sound is more important than acoustic properties. Furthermore, results from several studies show that people can experience misophonic responses to visual stimuli that are associated with triggering sounds (Edelstein et al., 2013; Schröder et al., 2013; Webber and Storch, 2015; Wu et al., 2014). Misophonia-like sensitivity to visual triggers has been referred to as 'misokinesia' in the literature (a reference to visual triggers often stemming from the repetitive movements of others). The current consensus definition of misophonia does specify that misophonic triggers include sounds associated with oral functions, non-oral/nasal sounds produced by people or objects, as well as *visual triggers* (Swedo et al., 2022). However, it is not yet clear how misokinesia and misophonia are interrelated; a subset of people with misophonia report both sensitivities, yet misokinesia can apparently exist independently of any triggering auditory stimuli (Jaswal et al., 2021). One possible account for similarities between the conditions is that both are produced by structural hyperconnectivity to the motor system (Kumar et al., 2021). Supporting the idea of hyperconnectivity in misophonia, researchers have also noted similarities between misophonia and synesthesia, a perceptual phenomenon in which stimulation of one sensory or cognitive pathway leads to involuntary experiences in a second sensory or cognitive pathway that is thought to result from hyperconnectivity of brain regions (including auditory cortex and insula) that lead to cross-activation (see Edelstein et al., 2013; McGeoch and Rouw, 2020; Palumbo et al., 2018).

Emotional reactions to trigger sounds commonly include anger, irritability, annoyance, disgust, and anxiety (Rouw and Erfanian, 2018; Siepsiak and Dragan, 2019) and are accompanied by increased autonomic nervous system activity. Sympathetic nervous system arousal produces increases in muscle tension, skin conductance, and heart rate (Brout et al., 2018; Edelstein et al., 2013; Ferrer-Torres and Giménez-Llort, 2021; Jastreboff and Jastreboff, 2014; Siepsiak and Dragan, 2019). The intensity of misophonic responses varies depending on the sound source, often becoming more pronounced when the trigger is produced by family members or acquaintances (Edelstein et al., 2013; Guetta et al., 2022; Taylor, 2017). The misophonic response often includes hyperfixation on the triggering sound (Rouw and Erfanian, 2018), as attention becomes intensely focused on the stimulus, making it difficult to ignore or disengage from the source of distress. Misophonic reactions can lead to feelings of being overwhelmed and escape behaviour such as removing oneself from the environment where misophonic sounds are heard (Brout et al., 2018; Edelstein et al., 2013; Kumar et al., 2014). People with misophonia may also report a desire to harm those producing the sounds (Bernstein et al., 2013; Edelstein et al., 2013), which can manifest as verbal or physical aggression toward people or objects (Bernstein et al., 2013; Edelstein et al., 2013; Schröder et al., 2013; Swedo et al., 2022). They typically recognize their reactions

Models of misophonia

as unwanted, uncontrolled, sometimes excessive, and unacceptable (Schröder et al., 2013; Schwartz et al., 2011), yet still experience strong emotional responses and often feel offended by those emitting the misophonic stimulus (Taylor, 2017).

Interestingly, people with misophonia typically do not experience the same intense reactions to self-generated sounds as they do to sounds produced by others or by animals/machines (Swedo et al., 2022). In Edelstein et al. (2013), 10 of 11 participants reported that self-produced triggers do not evoke as strong of an aversive response as sounds produced by other sources, and even noted that eating foods in synchrony with other people reduced their distress. Early case studies documented this tendency in people with misophonia to mimic misophonic sounds or actions (e.g., Edelstein et al., 2013; Hadjipavlou et al., 2008), and a larger, more recent study revealed that nearly 47% of participants engaged in mimicry, with its prevalence increasing with misophonia severity (Ash et al., 2024). Many participants described mimicry as deliberate and controllable, with 68% reporting that it provided relief, potentially acting as a coping mechanism by ‘cancelling out’ intrusive stimuli. Many individuals also report relief upon localizing the source of a trigger sound (Rouw and Erfanian, 2018), suggesting that being able to pinpoint the origin of a sound may offer psychological relief, possibly by giving a sense of control or the ability to anticipate when the triggering stimulus will end.

People with misophonia often avoid contexts in which triggers are likely to be encountered, including social gatherings, classrooms, and family meals (Schröder et al., 2013), as well as individuals who are known to produce misophonic sounds (Rouw and Erfanian, 2018). Avoidance can result in impairments in social interaction, and social isolation, negatively affecting well-being, education, and interpersonal relationships (Brout et al., 2018; Jager et al., 2020; Neal and Cavanna, 2013; Rouw and Erfanian, 2018; Swedo et al., 2022; Wu et al., 2014; Zhou et al., 2017). The distress provoked by misophonia can also contribute to behavioural health problems (Brout et al., 2018), emotional hyper-reactivity (Jastreboff and Jastreboff, 2014), and difficulties in regulating emotions (Cassiello-Robbins et al., 2020; Guetta et al., 2022).

In general, misophonia can have serious consequences for quality of life (Guetta et al., 2022; Sieksiak and Dragan, 2019). A large-scale study by Rouw and Erfanian (2018), which explored the impact of misophonia on daily life, offers some interesting additional observations. Thirty-six percent of participants reported that alcohol helped alleviate their misophonia symptoms, with some indicating that they used alcohol specifically for this purpose. In contrast, most participants stated that caffeine did not seem to influence their symptoms, and the majority did not use nicotine or other substances. Among those who did report symptom relief from other chemicals, marijuana or cannabis was most commonly mentioned. Many study participants noted worsened symptoms when fatigued. Alarmingly, over 20% of the

misophonia sample reported suicidal thoughts (Rouw and Erfanian, 2018), underscoring the most severe consequences of misophonia and motivating the search for solutions.

From these observations of how misophonia is manifested and experienced by people with the condition, several themes of relevance to the production of misophonia models emerge. In general, the heterogeneity of individuals' misophonic sounds and their acoustic properties does not suggest that a low-level, perceptual mechanism is to blame. That a misophonia stimulus' origin and meaning is relevant to the strength of the reaction it produces further emphasizes the role of higher-level cognitive processes (possibly involving the cortical ventral processing stream; Rauschecker, 2018; an idea that will be further discussed in Section 5). However, the mirroring, mimicry, and blocking observations suggest a strong motor and proprioceptive component (more likely to be computations taking place within the dorsal stream). The fact that people with misophonia are generally aware that their reactions are considered excessive and undesirable, yet nonetheless react, suggests strong and possibly pre-attentive connections to the limbic system. Finally, we note a great deal of inter-individual variability in misophonic stimuli and reactions, suggesting that the disorder may have subtypes or vary along several scales, perhaps each with its own neurophysiological correlates.

3.2. Auditory and cognitively-based coping mechanisms

In the absence of a clear understanding of the causes of misophonia or of effective treatments, it may be informative for developing cognitive models to consider what coping mechanisms sufferers of misophonia use spontaneously. In the context of developing the Duke Misophonia Questionnaire (DMQ), Rosenthal et al. (2021) interviewed 100 misophonia sufferers. After psychometric refinement, the DMQ contains Coping-Before, Coping-During, and Coping-After subscales. Coping-Before concerns planning and avoidance behaviours (e.g., ‘I avoided certain people, places, or things so I would not have to hear sounds I dislike’), whilst Coping-After mostly focuses on mood regulation following (e.g., ‘I did something to comfort myself (e.g., exercised, went somewhere calming, pet animals)'). Of particular interest for our purposes is the 10 item Coping-During subscale, which has items clustered into three domains: ‘Masking’ (‘I increased the background noise to cover up the bothersome sound (e.g., turned on TV, rolled down car window)'), ‘Distraction’ (e.g. ‘I focused my attention on an activity (e.g., watched TV or videos)'), and ‘Cognitive Techniques’ (e.g., ‘I changed my way of thinking about the sound’). Amongst community support groups and videographic testimonials of lived experience (e.g., ‘Breaking the Sound Barrier - Teens With Misophonia’ by Black, 2024), anecdotes concerning using noise-cancelling headphones or ear plugs are common. Strategies such as listening to white noise or music are common in environments where trigger sounds are present, but where avoidance behaviours would lead to negative consequences (e.g., in classrooms, workplaces, or family dining

situations). Individuals also report making their own sounds in mimicry as a masker, intentionally regulating their mood through breathing techniques, removing themselves from the environment, and cultivating understanding and empathy so as to reduce the emotional impact of the interpretation that a loved one is making the problematic sound out of malicious intent.

The themes that emerge from documenting coping behaviours hint at mechanisms that are blocked or reactions that are mitigated through these behaviours. Adjusting the audibility or salience of troublesome sounds appears to be a means of reducing the sounds' propensity to capture and monopolize attention. It is possible that some relief can be achieved via occupying brain networks that are involved in misophonia with other incompatible activities. For example, listening to music might reduce higher-level auditory processing of misophonic stimuli (i.e., beyond the primary auditory cortex and planum temporale, Puschmann et al., 2024), or producing motor activity oneself might engage motor networks so as to reduce communication between auditory and motor networks that may be overactive during misophonic responses (Kumar et al., 2021). That reactions can sometimes be modulated by voluntarily attending to other sound sources such as music implicates attentional networks, as does intentionally modulating the semantic interpretation of sounds or regulating mood directly. This group of coping strategies uses executive functions, a set of cognitive processes that support goal-directed behaviour (i.e., attentional control, cognitive inhibition, inhibitory control, and cognitive flexibility). In misophonia, executive functions themselves could be abnormal leading to poorer control, or instead they could function normally yet be constantly taxed by the additional workload associated with mitigating misophonia reactions. The latter idea would be congruent with reports that individuals with misophonia are more affected by misophonic sounds when in fatigued states (i.e., when available cognitive resources are depleted). A parallel may be found in pain research. People suffering from chronic pain are thought to use a greater proportion of their cognitive resources for attentional control and self-regulation in everyday life than healthy controls, leaving fewer resources available for other tasks and leading to apparent cognitive and self-regulatory deficits (Phelps et al., 2021; Solberg Nes et al., 2009). It may be relevant for considering the role of executive networks in models of misophonia that the challenges of coping with misophonia might be the cause of observed group differences in executive function, rather than group differences in executive function being responsible for misophonia.

3.3. Behavioural and cognitive observations

The role of context and sound meaning in misophonia

Recent research shows that both the actual context of a sound and the way it is cognitively processed can alter the intensity of the misophonic reaction. Edelstein et al. (2020) (*preprint*) demonstrated that individuals rated human-made sounds as less aversive when they believed the sounds were

of non-human origin. Similar findings were reported by Heller and Smith (2022), who found that misidentifying chewing sounds as cereal being stirred reduced their perceived unpleasantness, Samernit et al. (2022), who demonstrated that pairing incongruent visual stimuli with misophonic sounds, such as the sound of chewing paired with a video of stepping on snow, reduced the aversive response, as well as Sieksiak et al. (2023) who found that participants rated mouth smacking sounds paired with incongruent videos less negatively than when paired with congruent videos. Savard et al. (2022) also showed that when misophonic sounds were identified, they elicited stronger feelings of anger, disgust, and anxiety, particularly among individuals with severe misophonia symptoms. Therapeutic interventions have explored this avenue by encouraging patients to reframe or modify their perception of trigger sounds, such as imagining that the sound comes from a benign source like a motor, or a gorilla (e.g., Frank and McKay, 2019).

Altogether, evidence indicates that the cognitive context of sound processing significantly influences emotional reactivity. Emotional responses are not solely driven by acoustic properties; rather, the brain's interpretation and identification of sounds within specific contexts play a crucial role. These findings further suggest that misophonia is closely linked to higher-level cognitive processes (and likely has a strong learned component (Palumbo et al., 2018)), rather than being predominantly sensory in nature. They also suggest involvement of the ventral auditory processing stream (described in Section 5) where perceptual decisions are made concerning source identification and recognition, in a process called 'auditory-scene analysis'. In earlier, upstream stages, neurons encode the auditory properties of a stimulus, whereas later stages encode sensory evidence that leads to perceptual decisions (Cohen et al., 2016; Rauschecker, 2018). The ventral pathway, illustrated in Figure 2, may therefore be relevant for models of misophonia as a locus of action of abnormal or voluntary attentional processes, since attention can affect the degree of enhancement and extent of processing that neural representations of incoming auditory information receives (Puschmann et al., 2024).

Cognitive differences in the absence of misophonic sounds

Differences in cognitive processes in people with misophonia as compared with healthy controls have been studied in the presence and in the absence of trigger sounds. Deviations in cognitive processes in the absence of trigger sounds are generally assumed to reflect stable differences in the auditory system that affect all auditory processing, whereas those observed during or following symptom provocation are understood to incorporate differences that may be specific to the response to trigger sounds, including attentional and emotional aspects.

In Eijsker et al. (2019), individuals with misophonia showed slower reaction times and more errors in a visual response inhibition task compared to control groups in the

Models of misophonia

absence of misophonic sounds, suggesting that misophonia may be linked to impaired response inhibition. Further evidence of altered cognitive and sensory processing in misophonia is provided by electroencephalography (EEG) studies, such as Schröder et al. (2014), who found a diminished N1 component in response to deviant auditory tones in misophonia patients, compared to healthy controls, in the absence of trigger sounds. The authors interpreted this result as a deficit in early sensory processing, suggesting that the reduced N1 amplitude reflects a general impairment in the misophonic brain's ability to detect and prioritize novel auditory stimuli. They argue that this deficit may underlie the heightened sensitivity to certain sounds seen in misophonia, as the brain is less effective at distinguishing between relevant and irrelevant sensory information, even when no explicit trigger is present.

Interestingly, some studies have found *enhancements* in some cognitive processes in people with misophonia. For instance, in a study by Simner et al. (2021), participants with misophonia showed greater accuracy than a control group in identifying target shapes embedded within complex visual figures, without increased response times, which suggests better attention control. In addition, Murphy et al. (2024) explored cognitive performance in children and adolescents with misophonia and found superior signal detection abilities compared to those with anxiety disorders, indicating heightened perceptual alertness. These results could be interpreted in a neuroplastic framework. Engaging in challenging cognitive processes, for example those involved in musicianship and multilingualism, appears to cause improvements both to domain-specific and sometimes domain-general cognitive functions that support task performance (Bialystok and Craik, 2022; Roman-Caballero et al., 2018; Román-Caballero et al., 2021). Learning to function with misophonia might be thought of as a kind of long-term naturalistic training paradigm in which processes such as attentional control that are continually challenged become strengthened, potentially leading to some benefits on other tasks. While quite preliminary, this work offers hope that the effects of misophonia might be partly mitigated over time as cognitive skills are strengthened, and that training-based approaches could further support these compensatory processes. As regards models, these observations may inform dynamic models that consider misophonia's etiology and evolution, and the roles of neuroplasticity in both.

Cognitive differences in the presence of misophonic sounds

Overall, studies which presented misophonic trigger sounds during cognitive tasks demonstrate that misophonic responses negatively affect cognitive control. For instance, in Daniels et al. (2020), participants showed greater cognitive interference during a visual Stroop task when exposed to trigger sounds, and participants with misophonia in Silva and Sanchez (2019) performed worse than those without misophonia in identifying dichotically presented sentences in the presence of distracting sounds, particularly when

the distraction consisted of chewing sounds. Seaborne and Fiorella (2018) asked participants to read a text passage on which they would receive a comprehension test, while in the presence of a confederate (i.e., member of the research team acting as a participant) who either audibly chewed gum (sound group) or read quietly (control group). Although the two groups did not differ overall on their comprehension test scores, a moderation analysis revealed that students with higher misophonia sensitivity performed worse than those with lower sensitivity when exposed to background chewing sounds, while in the absence of these sounds, higher sensitivity students outperformed their less sensitive peers. This finding suggests that the presence of trigger sounds may hinder attentional focus for individuals with misophonia, yet these same individuals could experience a cognitive advantage in quieter environments, potentially due to an increased ability to concentrate when distractions are minimized.

These observations are supported by Frank et al. (2020) and Zumbrunn et al. (2023) (*preprint*) who found, when assessing attention with a visual Attention Network Task (Posner et al., 1990), that different attention networks (see Section 6.2) showed different patterns of performance during exposure to trigger sounds. Participants with misophonia had poorer alerting attention when exposed to trigger sounds, which indicates difficulties in maintaining a state of readiness to respond to stimuli. Interestingly, despite apparent impairments in the alerting network, participants with misophonia showed better orienting attention (i.e., the process of selecting and prioritizing sensory inputs) than control participants in Zumbrunn et al. (2023) when responding to aversive sounds, suggesting heightened attentional responsiveness to threatening stimuli.

Taken together, the results concerning behavioural and cognitive differences between people with and without misophonia are illustrative of the disorder's complex nature: it is not a disorder affecting only the peripheral sensory systems, but rather is a phenomenon deeply intertwined with cognitive processes, including perceptual decision-making, attentional control, response inhibition, and perceptual alertness. We thus expect comprehensive models of misophonia to include multiple cognitive functions and widespread brain networks.

3.4. Structural neuroanatomy of misophonia

One means of gaining insights into why populations differ is by contrasting their neuroanatomy with those of normative controls. By identifying brain regions and structures that vary in morphology and other biophysical characteristics, we gain insights into potential neural factors that contribute to differences in cognition, behaviour, and subjective experience.

Most approaches to studying group differences in neuroanatomy non-invasively involve magnetic resonance imaging (MRI). From standard T1-weighted anatomical images, the shape, size, and properties of brain tissue within brain structures can be derived, using techniques such as

Models of misophonia

Table 1

Summary of factors potentially relevant for the development of neurocognitive models of misophonia (citations are given in text; note that the row organization is intended solely to enhance readability of the point-form list and does not carry any inherent meaning.)

Prevalence & Demographics	Comorbidity	Trigger stimuli	Responses	Impact	Common coping mechanisms	Factors that may impact symptoms
Misophonia exists on the tail end of a continuous distribution	Misophonia without comorbid disorders: -no psychiatric disorder: 72% -no personality disorder: 59%	Primarily auditory stimuli, including: -orofacial (mouth/nose/throat) -human-produced non orofacial (e.g., door slamming, pen clicking) -non-human produced (e.g., barking dogs, refrigerator humming)	Emotional responses: anger, irritability, annoyance, disgust, and anxiety	Impaired attentional control, especially during symptom provocation (e.g., greater cognitive interference during tasks)	Avoiding problem stimuli: -Avoiding certain situations or people -Seeking a trigger-free environment following a reaction	Substance use: -Caffeine does not seem to affect symptoms -Some people report alcohol helping alleviate symptoms -Other chemicals are sometimes used to alleviate symptoms
Prevalence estimates of clinically significant (moderate to severe) misophonia symptoms: between 5% and 20%	Misophonia without other auditory disorders or sensitivities: 94% of cases	Visual stimuli (misokinesia): -Repetitive Movements (e.g., tapping fingers, leg shaking) -Stimuli associated with auditory triggers (e.g., sight of someone eating)	Increased autonomic nervous system activity: muscle tension, sweating, increased heart rate, increased body temperature, sensation of pressure in chest/arms/head	Social isolation due to avoidance of gatherings, classrooms, and meals	Decreasing salience/audibility: -Noise-cancelling headphones or earplugs -Listening to music or white noise -Increasing background noise	Fatigue may worsen symptoms
Prevalence is similar between males and females (in large balanced samples)	Common comorbidities (from large-sample studies): -OCPD -Anxiety disorders -Autism spectrum conditions -Mood disorders	Can be triggered by stimuli from media in addition to real-life interactions	Reactions more pronounced when trigger stimuli are produced by family members or acquaintances	Negatively affects well-being, education, and interpersonal relationships	Directing attention away: -Attending to another sound source -Engaging in pleasurable activities	Severity of reactions often depends on social contexts where triggers are likely to be encountered (e.g., gatherings, classrooms, family meals)
Symptoms often begin in childhood or adolescence, with many individuals reporting the emergence of sensitivity around this time	Common comorbidities (auditory disorders): -Tinnitus -Hyperacusis -Phonophobia	Generally not self-produced stimuli	Feelings of being overwhelmed leads to: -Escape behaviour (e.g., leaving the environment) -Aggressive outbursts (e.g., verbal or physical aggression)	Some individuals experience thoughts of suicide due to distress	Altering interpretation: -Change way of thinking about the stimuli -Cultivating understanding and empathy to avoid assigning blame	Severity of reactions often depends on the person producing the trigger sound
		Meaning of the triggering stimuli can alter emotional responses (e.g., identifying sounds incorrectly reduces the misophonic response)	Difficulty focusing, hyper-fixation on sounds	Development of mental health problems, increased emotional reactivity, and difficulties regulating emotions	Taking steps to reduce anxiety and enhance emotional regulation: -Mindfulness -Breathing techniques	
		Simply imagining trigger stimuli can evoke similar emotional responses	Experience of relief when identifying the source of the trigger stimuli		Mimicry: engaging in similar actions to "cancel out" intrusive stimuli	
			Recognize reactions as unwanted, uncontrolled, and sometimes excessive			

voxel-based morphometry or cortical thickness analyses. Using specialized pulse sequences and processing pipelines, diffusion-weighted brain images capture complementary information concerning the freedom of movement of water molecules in the brain, which is affected by differences in

white matter properties (see O'Donnell and Pasternak, 2015 for an introduction), and has also proven valuable to index tissue changes and differences in grey matter in clinical contexts (e.g., in Alzheimer's Disease; see Silva-Rudberg and Mecca, 2024). Diffusion-weighted data can be used in

Models of misophonia

a volumetric fashion, i.e., studying the intensity of signals in whole-brain or volume-based regions of interest probabilistically encompassing white matter tracts. Anatomical templates can be used to reduce the analysis to a white matter ‘skeleton’, or white matter tracts can be explored and dissected using tractography approaches (Johansen-Berg and Rushworth, 2009). These tools reveal relatively static differences between people with misophonia and controls in specific brain regions and in the network of white matter tracts that allow for inter-region communication.

To date, there have been few studies comparing structural neuroanatomy in people with and without misophonia, especially using whole-brain approaches. Results and implications from those which were published prior to 2022 were reviewed in Neacsu et al. (2022). Here follows an abbreviated summary, which we represent schematically in Figure 1A. In brief, the first reported finding of a structural abnormality in people with misophonia ($N = 20$) as compared with controls ($N = 22$) comes from Kumar et al. (2017), who found higher myelination in the ventromedial pre-frontal cortex whole-brain analysis (using a magnetization transfer saturation index, which reflects myelination in grey matter), noting that statistical analyses were restricted to regions found via functional connectivity analysis to be connected to the anterior insular cortex. Eijsker et al. (2021b) compared patients with misophonia ($N = 24$) with controls ($N = 25$) in a whole-brain analysis and found larger grey matter volume in the right amygdala, which they interpreted as being linked to the increased emotional reactivity to misophonia sounds observed in people with the disorder. Their related work in the same sample showed greater white matter volumes in the left inferior fronto-occipital fasciculus, the body of the corpus callosum connecting bilateral superior frontal gyri, and the anterior thalamic radiation, as well as lower averaged radial and mean diffusivities in widespread white matter areas in patients as compared to controls (Eijsker et al., 2021a). The authors speculate that biological alterations may be related to differences in social-emotional processing. In an investigation of structural anomalies associated with sound sensitivities ($N = 80$), Kliuchko et al. (2018) found correlations between a metric of noise sensitivity and enlarged grey matter volumes in bilateral hippocampus and temporal pole, and in right insula. This larger study is well-powered and likely includes people with misophonia, but the study design did not differentiate misophonia from other noise sensitivities (for which reason the results are not included on the summary diagram).

The results of structural abnormalities in misophonia demonstrate that there are indeed observable neurophysiological differences in this population to which MRI measures are sensitive. Widespread differences in white matter tissue properties, particularly in frontal regions, agree with the suggestion that transfer of information between regions involved in higher-level cognitive functions may be relevant to the disorder, as well as the involvement of limbic structures such as amygdala in the emotional experiences associated with misophonic reactions.

While these results could contribute to spatially-informed brain-wide models, there are several important caveats to doing so at the present time. Most importantly, there are few studies that take a whole-brain approach, and those which exist differ considerably in their approaches. It is currently difficult to combine results across studies, and impossible to assess replicability. Additionally, the structural brain differences which have been identified have generally not yet been directly associated with specific aspects of cognitive or behavioural measures or symptom severity, limiting their interpretability.

Even in fields with an extensive history and corpus of published anatomical studies, the clarity, quality, and consistency of aggregate results can be hampered by methodological issues including small sample size, characterization of groups, and whether confounding factors (e.g., medication use, comorbidities) have been statistically accounted for (Brambilla et al., 2003). Because misophonia can be confused with other sound-sensitivity disorders (Henry et al., 2022), and is frequently comorbid with other conditions (Jager et al., 2020), a thorough investigation of how structural neuroanatomy in primary misophonia differs from both healthy controls and comorbid disorders is needed (see Table 2 in Neacsu et al., 2022 for a summary of brain region alterations relevant to misophonia in common comorbid disorders). Thus we encourage caution in interpreting these findings and including them in models, but suggest that future models will benefit from data derived from structural investigations in well-controlled, large samples, appropriate controls, whole brain/discovery based analyses in parallel with the use of ROI-based analyses for focused research questions, and open science (e.g., through data-sharing).

3.5. Functional neurophysiology of misophonia

Considerably more work is available on functional as compared with structural neuroanatomy in misophonia. Blood oxygen level-dependent (BOLD) fMRI is the most commonly used technique for investigating brain-behaviour relationships in a spatially-resolved fashion. It indexes transient hemodynamic changes that are associated with neural activity in grey matter. In resting state fMRI studies, participants are asked to lie passively in the scanner and allow their minds to wander, usually for about 10 minutes. The recordings are subsequently used to compute patterns of connectivity between brain regions and within networks of regions, many of which have established functions (e.g., sensory networks; see Lee et al., 2013 for a review). The strength of connectivity patterns can differ between groups and be correlated with external variables.

In task-based fMRI studies, brain activity is evoked by presenting stimuli or asking participants to perform cognitive tasks, and contrasts are made between conditions to isolate specific functions of research interest. As compared with resting state analyses, task-based studies allow for higher precision and greater explanatory power concerning cognitive processes, though studies must be designed for specific research questions (and often have smaller samples). Both

Models of misophonia

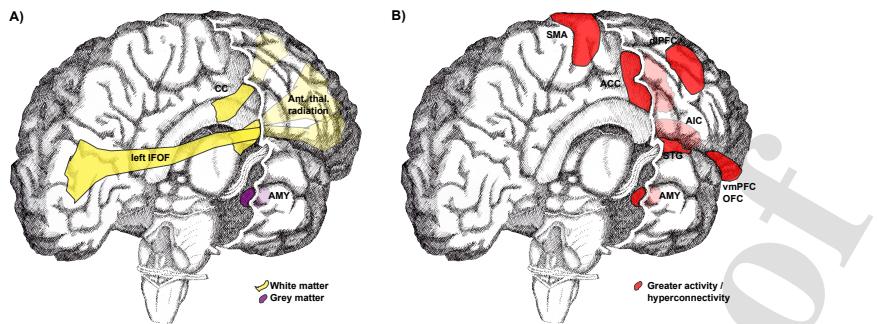


Figure 1: Brain regions and tracts implicated in misophonia. A) Structural and B) functional differences between people with misophonia and controls from whole-brain neuroimaging analyses. IFOF = inferior fronto-occipital fasciculus, CC = corpus callosum, ATR = anterior thalamic thalamic radiation, AMY = amygdala, SMA = supplementary motor area, AIC = anterior insular cortex, STG = superior temporal gyrus, ACC = anterior cingulate cortex, vmPFC = ventromedial prefrontal cortex, OFC = orbitofrontal cortex. Note that right hemisphere homologues are shown in due to the orientation of the brain image, hemisphere is labelled if not observed bilaterally. Drawing by E.B.J. Coffey used with permission.

contrast-based analyses and functional connectivity analyses (i.e., correlation of activity between different regions) can be used on data collected during task performance, to identify brain regions that are more active in one condition than another, and to investigate communication between them. As with structural neuroimaging results, results from functional neuroimaging studies could contribute spatial information to development of models of misophonia, sometimes with added specificity concerning links between brain functions and regions.

Neacsu et al. (2022) have recently reviewed the fMRI results associated with misophonia (vs. controls), which we summarize in Figure 1B (please see also Table 1 in Neacsu et al., 2022). Most results as regards lateralization and connectivity under specific conditions have either not yet been directly investigated or replicated, for which reason we aim for a high-level view of involved structures. In brief, differences in activation of the amygdala and connectivity between amygdala and other brain regions have been found in people with misophonia when they listen to trigger stimuli, and under resting state conditions (though Schröder et al., 2019 suggested that observed amygdala activity may relate more to raised anxiety levels than to the misophonic response per se). Other limbic regions implicated in processing and regulating emotions and behaviour, including the amygdala, have been found in group comparisons. These regions also include the anterior cingulate cortex, which is involved in emotion, action, and memory, and the orbitofrontal cortex, to which it is connected and from which it receives information about reward and non-reward outcomes (see Rolls, 2019 for a review). The anterior cingulate cortex also shows hyperconnectivity to frontal, temporal and occipital regions at rest, hyperactivation during exposure to trigger sounds, and hyperconnectivity with other structures including the amygdala and hippocampus, in people with misophonia. Individuals with misophonia also show greater activity in the anterior insular cortex when listening to sounds, a region

which is thought to play a role in subjective emotional experience by integrating bottom-up interoceptive signals with top-down predictions so as to generate a current awareness state, as well as provide descending predictions to visceral systems that provide a point of reference for autonomic reflexes (Gu et al., 2013). In addition, the ventromedial prefrontal cortex activation, which is involved in evaluating risk and regulating emotions; and dorsolateral prefrontal cortex, which has a range of higher-level cognitive functions including working memory, planning, decision making, processing threat-induced anxiety, social perspective taking, theory of mind, and deductive reasoning, show irregularities in connectivity in people with misophonia. Connectivity differences have also been found between superior temporal gyrus, which is involved in identifying and interpreting sound sources and auditory attention, and both limbic and motor system structures. Finally, a recent study additionally reported activity within bilateral dorsal cerebellar regions in response to trigger sound (Grossini et al., 2022). Dorsal cerebellar regions are proposed to play critical roles in social and emotional processing and learning, via reciprocal connections to cortical networks for sensorimotor integration, understanding mental and emotional states of others, and a limbic network supporting core emotional (dis)pleasure and arousal processes (see Van Overwalle (2024)).

fMRI-based approaches have several limitations as we consider their contributions to our models. Notably for resting-state designs, participants' cognitive state and activities may vary. For example, participants frequently fall asleep during resting state scans (Soon et al., 2021; Tagliazucchi and Laufs, 2014). Sleep is a unique physiological state and vigilance levels and autonomic physiology both affect brain hemodynamics, sometimes in regionally-specific ways, showing that the physiological constituents of fMRI signals can vary considerably and confound research questions (Gold et al., 2024). Vigilance and autonomic factors may also impact results from task-based results in unanticipated ways. For example, subjects with misophonia

Models of misophonia

who have signed up for a study in which they expect to hear their trigger sounds may be in a different state of arousal than those without such expectations. This hypothetical heightened vigilance state difference might affect how much sleep data are included in a group contrast, directly affect how well a participant is able to concentrate on a cognitive task, and affect at the physiological level properties of the hemodynamic response.

Despite such potential confounds, our incomplete view of neuroanatomical differences in misophonia, and limitations in our knowledge concerning the specificity of current results to the disorder, the regions involved and their known function contribute ideas as to which systems and networks should be included in models of misophonia. In the next section, we review the few existing models of misophonia and then propose additional networks that could be included in future models.

4. Existing models of misophonia

While many papers on the neurocognitive basis of misophonia include mechanistic speculations, few have elaborated these ideas into models. Exceptions include Jastreboff and Jastreboff (2014, 2023), who focus on connectivity differences between auditory and limbic systems; Aryal and Prabhu (2023), who put forth a neuroaudiological model of misophonia; and Kumar et al. (2021, 2017), who proposed a model that focused on interactions between auditory and motor system, which was more recently updated with a social cognition focus (Berger et al., 2024). We briefly describe here the main ideas of these models (see original works for details).

Jastreboff and Jastreboff (2023)'s model emphasizes the differences between misophonia and hyperacusis, with a focus on developing mechanistically-informed treatments for these disorders. They propose that in hyperacusis, pre-attentive steps in the auditory pathway are subject to abnormally high gain, resulting in over amplification of neuronal activity. The over-amplified signal then causes exaggerated responses of the limbic system via direct connections, and indirectly via brain regions responsible for perception and evaluation of sounds. In contrast, the auditory system is proposed to function normally in misophonia, but to stimulate the limbic and autonomic nervous system regions through abnormal connectivity. These abnormal connections are driven by a process called 'complex conditioned stimuli' in which other elements of a scene such as personal relationships and controllability account for the over representation of orofacially-generated sounds and greater reactions to sounds produced by family members. This model is somewhat situated in the brain, accounts for many extant findings, and leads to testable predictions. For example, we might predict that misokinesia (visually-generated) would be independent of misophonia, or that the severity of misophonia would strongly correlate with measures of connectivity between auditory and limbic systems.

Aryal and Prabhu (2023) centres on the role of auditory processing in misophonia, whilst acknowledging that it is

most likely a neurophysiological disorder that borders audiology, neurology, and psychiatry. Their neuroaudiological model proposes that auditory processing from the auditory periphery through to the subcortical areas is normal; however, that (non-classical) pathways from subcortical structures to the limbic system and autonomic nervous system are stronger or hyperactive in people with misophonia, leading to the disorder's emotional and physical responses. Aryal and Prabhu (2023) also hypothesize that impaired sensory gating (reduced inhibition) leads to abnormal neural activity of auditory cortex and association areas, which then increases negative association of triggers and emotions via processes of classical conditioning.

This model has many similarities with that of Jastreboff and Jastreboff (2023), and is also loosely spatially situated in the brain, in the sense that subcortical and cortical processes and their interaction are indicated. It would account for several of the main findings related to auditory processing in people with misophonia, and partly addresses etiology by invoking processes of classical conditioning. However, it is not explicit about where, when and how neuroplastic changes might take place, and which of the group differences (reduced inhibition or abnormal connectivity between subcortical auditory structures and limbic system) might be the primary neurophysiological cause of misophonia. A mechanism that explains both the stronger subcortical connection to the limbic system and impaired sensory gating, and whether these factors might lead to different subtypes of misophonia, is not explained. This model does not address the full range of observations summarized in Table 1; for example, why general group differences in response inhibition or subcortical pathways to the limbic system should be linked to specific types of triggers (e.g., predominantly orofacially-generated sounds). The authors acknowledge the preliminary nature of the model and emphasize the need for more work in larger samples, and particularly of the need to consider comorbidity, potential sub-types, and the severity of misophonia.

Berger et al. (2024)'s model of misophonia centers around social cognition, positing that misophonic distress arises from the interplay between auditory perception and motor representation (the 'perception-action' link). The authors argue that in misophonia, the brain generates motor representations of sounds, a process that typically occurs during normal sound processing. Heightened connectivity between auditory and motor regions in people with misophonia, influenced by the context of the auditory stimulus, leads to an aberrant signal possibly in the form of an error signal being forwarded to the insula, a brain region known to be involved in 'motor empathy', which is defined as the tendency to automatically mimic the motor response of others (amongst other functions). From there, abnormal information is passed to the amygdala and autonomic nervous system, where it generates abnormally high emotional and physiological responses. Notably, the model allows for the bidirectional nature of these processes; a feed-forward pathway from auditory system to limbic system generates

Models of misophonia

a response, but is also influenced by predictive coding mechanisms that can lead to anticipatory responses and feedback loops that act to further intensify the emotional impact of trigger sounds.

The motor-related basis of Berger et al. (2024)'s model allows for an explanation for observed involvement of the motor system including reported mimicry behaviours, and can be extended to further predictions such as that misophonia sufferers would exhibit stronger auditory-motor imagery, as do professional musicians. It is more explicit than previous models as regards brain structures, e.g., emphasizing motor cortex involvement and hypothesizing roles for anterior insular cortex and amygdala.

The three models presented are generally not mutually exclusive, though they emphasize different aspects of cognition, neuropathophysiology, and etiology; and they differ in the degree to which neural structures are delineated. While parsimonious models are desirable, each of these does not account for the full range of phenomenological, behavioural and cognitive observations (Table 1). In the next section, we introduce some neural networks and processes which are plausibly involved in misophonia from structural and functional observations (Figure 1), and which may contribute to a more comprehensive view of the disorder.

5. Situating misophonia within the broader auditory network

While existing models of misophonia emphasize normality of lower-level auditory processing - correctly in our view - it is unclear where the auditory system is thought to *stop* in each of these models. For example, Berger et al. (2024) argue that misophonia is not primarily an auditory disorder, partly because the first clear abnormality has been linked to the posterior superior temporal gyrus/sulcus, which processes both auditory and visual information. We instead support the view that brain acts as an integrated system, well beyond the primary auditory cortex, to process sound. Increasingly complex and abstract sound representations are produced at progressively higher levels, combined with information from other sensory modalities, used by and influenced by a variety of cognitive systems. Higher-order processes play a key role in propagating auditory representations through the system, even at early stages (e.g., Puschmann et al., 2024), via descending projections.

This view has relevance for how we conceptualize misophonia and which other areas of work may have relevance to understand it. For example, a great deal is known concerning how auditory perception gives rise to emotional reactions in the context of musical pleasure; the neural structures involved extend well beyond primary auditory cortex and invoke predictive processes (see Zatorre (2024) for an in-depth treatise on this topic). Of particular interest is that auditory processing branches into two streams after exiting the primary auditory cortex (in superior temporal gyrus), illustrated in Figure 2. The dorsal stream deals with temporal dependencies and predictions, and sensorimotor integration.

The ventral stream is specialized in holding sounds in mind, extracting structural regularities, organizing sounds into categories, and assigning them meaning. Both streams could be involved in misophonia. The motor involvement emphasized by Kumar et al. (2021) would be in-line with dorsal stream processing, whereas trigger sound identification (e.g., Heller and Smith, 2022; Samermit et al., 2022; Savard et al., 2022) would most congruent with ventral stream functions. Consideration of dorsal and ventral processing would lead to models that predict partly dissociable subtypes of misophonia, perhaps accounting for some of the variability observed in the disorder.

Sensory predictions in the auditory system are computed at the cortical level (for both streams), and bidirectional connectivity to subcortical structures is involved in generating and assigning emotional responses so as to guide behaviour, which also strongly influences processes of learning and neuroplasticity (Zatorre, 2024). As the lack of capacity to experience musical pleasure can be tied to reduced connectivity between the auditory cortex and the subcortical reward network (i.e., 'musical anhedonia', see Martínez-Molina et al. (2016)), misophonia may also be related to abnormalities in the wider auditory system. In fact, some evidence suggests that the frequency with which individuals experience emotional responses to sound covaries across both negative (i.e., misophonic triggers) and positive (i.e., musical) emotional contexts (Mednicoff et al., 2024).

For the purposes of generating mechanistic models of misophonia, we suggest that several ideas should be kept in mind. First, that the auditory system neither starts nor ends at the primary auditory cortex. Its function and dysfunction is largely defined via interactions with other cognitive systems and structures, which may treat other types of information. Second, the absence of observed abnormality in one experimental paradigm and measurement method does not preclude the existence of abnormality. Finally, when differences in neural signals are observed during early processing stages, they may reflect top-down modulation from later stages or group differences in vigilance states, rather than alterations in lower-level processes. The importance of higher-level cognition is suggested by observed patterns in misophonic responses and coping mechanisms (Table 1) and is congruent with the considerable frontal lobe involvement (Figure 1B); however, the roles of higher-level networks are not explicit in extant models. In the next section, we summarize several networks that could be involved in misophonia, for consideration in future models.

6. Potential involvement of higher-level networks

6.1. Salience, default mode, & central executive networks

The salience network (SN), described by Seeley et al. (2007), plays a critical role in detecting and filtering significant stimuli based on cognitive, homeostatic, or emotional factors (Craig, 2002, 2009; Goulden et al., 2014). Within

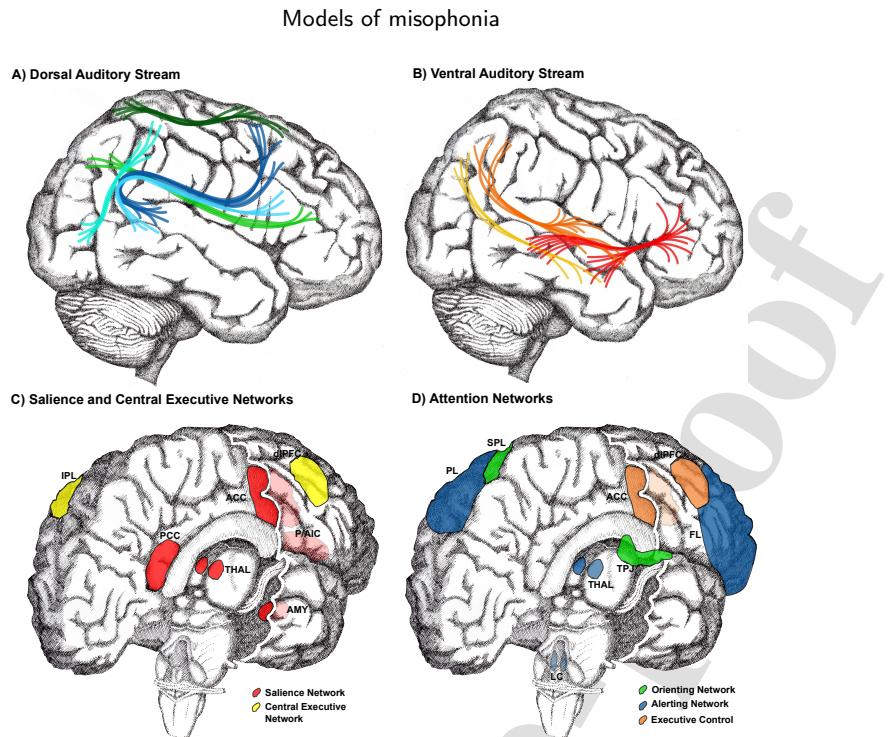


Figure 2: Networks and pathways of potential interest for misophonia models. A) Dorsal and B) ventral auditory processing streams, showing simplified schematic depictions of anatomical connectivity, based on diffusion MRI. The dorsal stream is specialized in temporal dependencies and sensorimotor integration, whereas the ventral stream is involved in holding sounds in mind, extracting regularities, categorizing sound, and assigning meaning. Both streams are likely to be involved in misophonia, as well as their differing connectivity with subcortical structures, possibly explaining variability in phenotype. Colours represent different fibre tracts. C) Illustrates the neuroanatomy of the salience and central executive networks (which are anticorrelated with the default mode network, not shown). D) Illustrates prominent regions involved in alerting, orienting and executive control aspects of attention. IPL = inferior parietal lobule, THAL = thalamus, P/AIC = posterior / anterior insular cortex, SPL = superior parietal lobule, TPJ = tempoparietal junction, FL = frontal lobe, PL = parietal lobe, LC = locus coeruleus. Drawings by E.B.J. Coffey with pathway overlay as in Zatorre (2024), used with permission.

the SN, the anterior insula facilitates bottom-up detection of salient events and manages transitions between networks essential for attention and working memory. It monitors both internal and external environments, regulating autonomic responses with the posterior insula (Seeley et al., 2007). Strong connectivity with the ACC enables quick reactions to salient stimuli (Menon and Uddin, 2010), while the ACC integrates cognitive and emotional information, guiding behaviour and supporting decision-making via the prefrontal cortex (Goulden et al., 2014). The SN, particularly the right AI, coordinates transitions between networks, activating the Central Executive Network (CEN) while deactivating the Default Mode Network (DMN) to engage different cognitive processes (Sridharan et al., 2008). The main areas implicated in SN and CEN are illustrated in Figure 2C.

The CEN, which comprises the dorsolateral prefrontal cortex and parietal regions, is crucial for higher cognitive functions like attention and decision-making, balancing demands from the dorsal attention system and the hippocampal-cortical memory system (Seeley et al., 2007; Vincent et al., 2008). In contrast, the DMN—active during rest and introspection—supports self-referential thought and memory retrieval, deactivating during goal-directed tasks to

maintain a baseline state of awareness that facilitates the processing of stimuli (Raichle et al., 2001). The SN and CEN are correlated, and both are anti-correlated to the default mode network (Raichle et al., 2001). The balance between the SN and DMN is critical for cognitive control; failures to deactivate the DMN during external attention tasks are linked to attention lapses (Leech and Sharp, 2014; Weissman et al., 2006). A stronger anticorrelation between SN and DMN activity correlates with better cognitive control (Kelly et al., 2008). Thus, the balance between these networks is crucial for modulating attentional focus.

In misophonia, heightened SN activation—especially in the AI—may lead to exaggerated responses to trigger sounds, affecting emotional and attentional processes, and increased coupling between the AI and DMN may impair DMN deactivation, allowing memories and contextual associations to intensify the misophonic response (Kumar et al., 2017). Studies suggest hyperactivation of the AI in misophonia, linking the SN's role in detecting significant stimuli to these intensified responses. Additionally, the triple network model posits that aberrant interactions within the SN, CEN, and DMN contribute to neuropsychiatric disorders (see Schimmelpfennig et al., 2023 for a review),

emphasizing the implications of altered salience processing in conditions like misophonia (Menon, 2011).

6.2. Attention networks

The Attention Network Theory (ANT; Posner et al., 1990) identifies three interacting attention networks: alerting, orienting, and executive control, the main components of which are illustrated in Figure 2D. These networks engage specific brain regions and contribute to different attentional processes, with research on misophonia highlighting impairments and enhancements in each (see section Cognitive differences in the presence of misophonic sounds). Networks are introduced below, but see Posner and Rothbart (2007) and Fernandez-Duque and Posner (2001) for more details.

The alerting network, linked to the norepinephrine system, activates the thalamus, frontal cortex, and parietal cortex to increase sensitivity to stimuli (Fan et al., 2005). It increases activation in the right frontal-parietal system, even in the absence of stimuli, across sensory modalities, including auditory attention (Belin et al., 1998). The orienting network is responsible for selecting and aligning attention with sensory inputs, both automatically and voluntarily (Spence and Driver, 1994). Key regions in this network include the superior parietal lobe (for orienting to a cued location) and the temporal parietal junction (for orienting to uncued, unattended targets) (Corbetta and Shulman, 2002). The executive control network monitors and resolves conflicts among thoughts and responses, mainly involving the ACC, which shows enhanced connectivity with the attended sensory modality (Crottaz-Herbette and Menon, 2006). For reviews on the role of executive attention in different cognitive tasks, see Bush et al., 2000; Carter et al., 1999; Posner and Rothbart, 1998). Other key areas include the dorsolateral prefrontal cortex, and portions of the basal ganglia. Notably, ventral areas of the ACC are closely involved with emotional regulation, while dorsal areas are more involved in purely cognitive tasks (Bush et al., 1998).

Considering the activity in these networks when developing models of misophonia presents a valuable opportunity for researchers to elucidate the cognitive mechanisms underlying this disorder. The observed behavioural impairments in the alerting network indicate difficulties in maintaining focus and readiness, which may contribute to heightened emotional responses to aversive stimuli. At the same time, the enhancements noted in the orienting network suggest a selective attentional bias toward threatening sounds.

7. Challenges and next steps

Models are only as good as the data upon which they are based. Recent reviews of the neurobiology of misophonia note the lack of consensus on assessing and defining misophonia, as well as limited quantity and specificity of available data (Berger et al., 2024; Jastreboff and Jastreboff, 2023; Neacsu et al., 2022). Inconsistency in the use of misophonia questionnaires and in inclusion of comorbid conditions complicates the interpretation of findings, their aggregation, and model development. Therefore, future research

Outstanding Questions

- Does misophonia affect multiple networks through widespread abnormalities, or via a single process with widespread connections?
- Are there misophonia subtypes (e.g., related to dorsal (motor) and ventral (semantic) cortical streams)?
- Are neurophysiological abnormalities in misophonia common to misokinesia (or other related disorders), or specific to the auditory modality?
- What is the neurophysiological relationship between positive and negative emotional experiences generated through sound (e.g., musical reward, misophonic distress)?
- If there are cognitive advantages to misophonia (under quiet conditions), are they the result of neuroplastic compensatory processes or inherent to abnormalities that have both negative and positive consequences?
- In which misophonia-relevant networks can neuroplastic processes be stimulated (i.e., through coping mechanisms, training, or brain stimulation)?
- What will we be able to learn about the mechanisms of misophonia from the success or failure of treatments that target different aspects?

should continue to investigate how misophonia measures relate and which best capture the misophonic experience, and care should be taken to control for comorbidities and examine their interactions with misophonia, to clarify its unique neurobiological and behavioural profile. In addition to addressing these limitations, we propose that misophonia models would be significantly advanced by research addressing the outstanding questions listed in Box 1.

8. Conclusions

The present work summarizes the growing body of observations concerning the phenotype of misophonia, with a view to building cognitive and neurophysiologically-informed models of the disorder. In considering the few available models, we can conclude that while they each account for some of the observations (and could be used to generate some testable predictions), misophonia is a complex disorder with considerable phenotypical variability which likely cannot be explained by abnormality within a single cognitive network. While neurocognitive models that are as parsimonious as possible are desirable (as with other sorts of models of cognition; Cassimatis et al., 2008), models which do not account for the complexity and variability of the disorder will have limited explanatory value. We therefore propose consideration of the auditory system as a unified whole including processes downstream of the

Models of misophonia

primary auditory cortex, and inclusion of additional higher-level networks. We hope this work will facilitate refinements in our understanding of misophonia by contributing to neurologically-informed cognitive models and that will allow for their evolution, ultimately informing better treatments.

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10. CRediT author statement

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Models of misophonia

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Toward cognitive models of misophonia

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1. Recent work has revealed misophonia as a complex and heterogeneous disorder
2. Cognitive and neurobiologically-informed models facilitate theoretical advancements
3. Existing models require extension to account for observed phenotypes