A social cognition perspective on misophonia

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The current dominant perspective on misophonia considers this disorder to be related to emotional sound processing, in which a "trigger sound" initiates a strong reaction in the perceiver. However, a sound processing model cannot explain all aspects of our growing understanding of the phenomenology and empirical findings in misophonia, such as the importance of context, coping strategies used, and lack of trigger-specific auditory cortical activation. In this article, we argue that a complete model of misophonia must include the context within which a sound is perceived and the emotional reaction triggered. We review the current behavioural and neuroimaging literature, which lends support to this idea. Based on the current evidence, we propose that misophonia should be understood within the broader context of social perception and cognition, and not restricted within the narrow domain of being a disorder of auditory processing. We discuss the evidence in support of this hypothesis, as well as the implications for potential treatment approaches.

Introduction

Misophonia is a disorder characterised by an intense emotional reaction and often physical discomfort to everyday sounds, such as other people eating, drinking, nasal and throat sounds (Edelstein et al., 2013; Schroder et al., 2013; Wu et al., 2014). Although the condition was named and referred to in the early 2000s (Jastreboff, 2000; Jastreboff and Jastreboff, 2001), scientific research into the condition has only begun in recent years. Based on the limited evidence available, the prevalence of clinically-relevant misophonia is suggested to be anywhere from 5 to 20% (Wu et al., 2014; Zhou et al., 2017; Kılıç et al., 2021; Vitoratou et al., 2023), indicating its wide prevalence where further research is necessary. Misophonia can lead to severe social isolation and withdrawal from familial activities (Hadjipavlou et al., 2008), and is highly distressing to those that experience it on a frequent basis, even leading to suicidal thoughts in up to ~20% of sufferers (Rouw and Erfanian, 2018).

Current model of misophonia

At present, neurophysiological or psychological models of misophonia are in their infancy, partly due to a lack of research in the area until recent years. Thus far, the dominant current model, put forward by Jastreboff and Jastreboff (2003, 2023), considers sounds as the main driving factor of misophonic distress and reaction. The model proposes that the negative emotional reaction in misophonia is caused specifically by the trigger sounds, after being processed in auditory brain regions, which in turn causes aberrant activity in brain regions involved in emotion processing. Importantly, this model makes a distinction between hyperacusis and misophonia: while the negative reaction in hyperacusis (increased sense of loudness discomfort) depends on the acoustic features such as spectral energy of the sound, misophonia is affected by the acoustic pattern and meaning, not on the intensity of sound. It was, therefore, suggested that the negative reaction in misophonia was not primarily due to abnormal processing in the auditory pathways, as is the case for hyperacusis, but related to aberrant processing in the 'limbic' system which processes emotion-related information of the sound. Although in its latest version (Jastreboff and Jastreboff, 2023) the model mentions that the emotional reaction in misophonia depends on the context – for example, a sound from a particular person in a situation or place is a trigger but not from other people/places/situations – sound remains the most important component in driving the emotional reaction. Indeed, even the origin of the word misophonia is derived from the Greek words for "hate" and "sound", although it is worth acknowledging that a literal interpretation of this definition as a basis for misophonia should be avoided (Jastreboff and Jastreboff, 2023).

Limitations of a sound-centric model

1. A sound-centric model ignores the source of sound

Although sound is its primary focus, a key issue with a sound-centric model is that it does not give due consideration to the source that generates the sound. There are a number of studies which show that the source of sound is important for setting the context within which sound is perceived. For example, Reuter et al. (2011) presented the sound of chalk on a chalkboard, which is typically perceived as aversive by most (Kumar et al., 2008), and asked a group of participants to rate the aversiveness of the sounds. Half of these participants were told the true source of sounds and the other half were made to believe that the sound was part of contemporary music. It was found that the same sound was rated less aversive and registered a lower skin conductance response by the 'musical-source' group of participants compared to 'chalkboard-source' group, indicating that knowledge of the source of sounds shapes the perception and physiological response to aversive sounds. In another similar experiment, Samermit et al. (2019) asked participants to rate discomfort and unpleasantness of typical aversive sounds, such as a knife grating on glass, when these were presented alone or presented in association with a silent video. The silent video could either be the original source of the sound or a video

source with positive attributes (e.g., silent video of bird chirping presented with a sound of knife grating on glass). Importantly, the video source with positive attributes was temporally-synchronised with the sound, so that the sound was more likely to be attributed to the visual source. The results showed that sounds that were synchronised to videos with positive attributes were rated less unpleasant, caused less discomfort, and reduced the intensity of bodily sensations, indicating again that the perceived source of a sound can modulate the reaction to that sound. A similar finding was also demonstrated by Cox et al. (2008) using static images, highlighting that biasing towards a particular expected context, without the temporal dynamics that videos include, can influence the perceived sound.

In relation to misophonia, recent work shows that the source of a sound plays a crucial role in determining the reaction triggered by the sound. Edelstein et al. (2020) asked misophonia subjects to rate human eating/chewing sounds, which are typical trigger sounds in misophonia. In addition to the rating, subjects were also asked to guess the source of the sound. Interestingly, misophonia subjects rated the correctly-identified sounds as more aversive compared to incorrectly-identified sounds (e.g., human eating sounds misidentified as non-eating sounds). Furthermore, non-eating sounds incorrectly identified as human eating sounds were rated as more aversive compared to correctly identified non-eating sounds. These findings of sound source identification playing a role in modulating the trigger reaction in misophonia have been further confirmed by more recent studies (Heller and Smith, 2022; Savard et al., 2022; Siepsiak et al., 2023). The importance of social context has also been highlighted in the recent consensus definition of misophonia (Swedo et al., 2022). All of these studies highlight an important implication for understanding misophonia: the perceived source of a sound is important for whether and to what degree a particular sound is experienced as a trigger sound, even in cases of incorrectly identified sound sources, emphasising that the sounds per se are not the driving factor in misophonia.

2. Trigger sounds activate brain areas which process social emotions and interpersonal stimuli

Within a sound-centric model, activation of 'limbic' areas of the brain by trigger sounds is highlighted as being one of the most important factors in the trigger reaction in misophonia. Although the term 'limbic', in general, has been criticised (Kotter and Stephan, 1997; Pessoa, 2008) for being vague and often contradictory in its description of the emotional system, it typically includes brain structures like the amygdala, hippocampus, thalamus, hypothalamus, basal ganglia, and cingulate gyrus. Of the few neuroimaging studies of misophonia that exist, there is only limited evidence for the involvement of commonly-defined 'limbic' structures, such as increased activation to trigger sounds in the anterior cingulate cortex in a single study (Schroder et al., 2019) and structural abnormalities in the amygdala in misophonia participants (Eijsker et al., 2021), although the Schröder et al study did not find any differences in amygdala activation between misophonia participants and controls.

Contrastingly, a brain structure that has been found to be consistently involved in misophonia studies is the anterior insula. In the very first fMRI study of misophonia, Kumar et al. (Kumar et al., 2017) showed stronger activation of anterior insula in misophonia subjects specifically for trigger sounds. Furthermore, the anterior insula had stronger functional connectivity to areas of the default mode network, including ventromedial prefrontal cortex (vmPFC) and posterior cingulate. Using audiovisual stimuli, the findings of anterior insula by Kumar et al. (2017) have been replicated in two other fMRI studies (Schroder et al., 2019; Cerliani and Rouw, 2020, preprint). Although the precise role of anterior insula in misophonia remains unknown, there is now a large body of evidence which links anterior insula to social processing in general and processing of social emotions in particular (for a review, see Lamm and Singer, 2010). Social emotions refer to processes that are generated because of interactions with other people. For example, anterior insula is activated not only by the experience of self-emotions but also by observing the emotional expressions of others (Wicker et al., 2003; Bastiaansen et al., 2009). In addition to processing of social emotions, anterior insula is also involved in pain experienced after social rejection (Eisenberger, 2015), social compliance (Bellucci et al., 2018), and recognition of social cues (Rogers-Carter and Christianson, 2019). Taken together, these results suggest that a comprehensive theoretical model of misophonia should account for the activation of brain structures implicated in social processing.

3. Hyperactivation of motor cortex to trigger sounds is not explained within a sound-centric model

In addition to the insula, as highlighted in the previous section, another brain region that has been consistently shown to have stronger activation to trigger sounds in misophonia subjects is the motor cortex. For example, Kumar et al. (2021) showed stronger activation of ventral premotor cortex (vPMC) in misophonia subjects specifically in response to trigger sounds, while another imaging study in preprint (Cerliani and Rouw, 2020) also found involvement of vPMC using audiovisual trigger stimuli. One might argue that the activation of motor cortex in misophonia is due to the motor component of the emotional response, for example, stronger facial muscle activation due to feelings of anger/disgust, in response to trigger sounds in misophonia subjects. However, stronger connectivity of the motor cortex to both visual and auditory cortex in the resting state, i.e., when no explicit stimuli were presented, do not support this argument (Kumar et al., 2021). Using a small sample of subjects, Hansen et al. (2022) also replicated this finding. Thus, involvement of motor cortex in mediating responses to trigger sounds in misophonia runs counter to the prediction of a sound-centric model, which posits processing in auditory and limbic areas as the basis of misophonia.

4. Lack of differential activation in auditory cortex to trigger sounds is hard to explain within a sound-centric model

If we are to place misophonia within a model that considers the sound itself as the main driving factor, then one would expect that there would be some involvement of

the auditory cortex. Contrasting with this idea, in two fMRI studies of misophonia subjects, Kumar et al. (2017; 2021) demonstrated that there were no differences in auditory cortex activation to trigger sounds between misophonia sufferers and controls. Similarly, a recent study utilising auditory brainstem responses to examine the subcortical auditory pathway found no differences between misophonia participants and controls (Aryal and Prabhu, 2023). It should be noted that the results of Kumar et al. (2017; 2021) seemingly contrast with Schroder et al. (2019), wherein activity in the posterior portion of right superior temporal cortex was increased in misophonia subjects. However, in the Schroder et al. study activity in this area did not correlate with the severity of misophonia symptoms as may be expected, although this itself could be a consequence of the low number of trials in the study. More importantly, in Schroder et al. (2019) this region did not correspond to primary auditory cortex but a higher-order area, specifically posterior superior temporal gyrus/sulcus, which are known to be multi-modal areas activated by moving visual stimuli and important for social perception (Pitcher and Ungerleider, 2021). Thus, the lack of differential activation indicates that trigger sounds are not processed differently in core auditory cortex in misophonia subjects when compared to controls.

5. A high prevalence of mimicry in misophonia

Potentially the strongest behavioural evidence to move beyond a purely sound-centric interpretation of misophonia comes from anecdotal reports of a high incidence of mimicry in misophonia sufferers. Even in some of the earliest published case studies of misophonia, mimicry was mentioned as an important feature of the condition (Hadjipavlou et al., 2008). Early self-report evidence suggested an incidence of mimicry in approximately 55% of misophonia sufferers, although the sample size was small (n = 11; Edelstein et al., 2013). Recent evidence from a large-scale online study (n = 676) found a similar incidence of mimicry in those with misophonia (>45%; Ash et al., 2023). Moreover, in this study, the tendency to mimic increased with increasing misophonia severity, with the severity of misophonia predicting the likelihood of mimicry. Compared to environmental sounds and other human-produced sounds, orofacial sounds (eating and chewing) were more likely to result in mimicking behaviour, and mimicry provided some relief from distress to individuals with misophonia. Why do misophonia subjects mimic in response to trigger sounds, and why does this act often provide relief when used as a coping strategy? These questions are hard to explain within a sound-centric model of misophonia, arguing for a revision of the theoretical and conceptual model of misophonia.

Misophonia: From sound perception to action perception

Using an integrated framework, where both trigger sounds and their social source are considered together, a different picture of misophonia emerges. Since the most common trigger sounds, for example someone eating/chewing, are associated with an action (e.g., orofacial movement) of another person, it could be the case that misophonic distress is due to the perceived action of others and not due to the sound per se, which is a by-product of that action. There is now overwhelming evidence that the psychological/neural processes involved in social signals are different compared to non-social signals (for reviews see Adolphs, 2010; Molapour et al., 2021). In social cognition and neuroscience, it is well known that the mere observation or hearing the sounds of actions of other leads to 'mirroring' or 'mimicking' of the same actions by the perceiver, without any intention or awareness to do so (Chartrand and van Baaren, 2009; Heyes, 2011; Chartrand and Lakin, 2013). In this scenario, mimicry is commonly understood within the framework of a 'perception-action' link, which posits that perceiving the action of others automatically activates representations of that action in the perceiver which, in turn, executes movements that are congruent to the perceived actions. With respect to the underlying brain function, the perception-action link is suggested to be instantiated as communication between sensory areas and the motor areas of the brain. Thus, with emphasis on the actions of the trigger-person, could it be that the perception-action link is relatively stronger in misophonia which is activated by the sight or sounds of action? This would appear to be supported by the results of Kumar et al (2021) showing stronger resting state connectivity of both auditory and visual cortex to motor cortex.

The phenomenon of imitation and mimicry has been commonly studied within a social perception/cognition framework. Thus, given the high incidence of mimicry in misophonia highlighted above, when considering misophonia from a social cognitive perspective it is useful to examine the role of imitation and mimicry within the wider social cognition literature. Non-conscious mimicry can often occur in a social setting, involving matching of others' behaviours, such as particular mannerisms or gestures of those with whom the mimicker is interacting. This type of mimicry is termed the "chameleon effect", and has been shown to potentially increase liking between individuals and facilitate smoother interactions (Chartrand and Bargh, 1999), although the specific effects of mimicry can depend on the social context in which it takes place e.g. Stel et al., 2010), or even the specific orientation of the mimicked actions (Casasanto et al., 2020). The chameleon effect has been shown to be stronger in people that show a higher disposition of empathy (Chartrand and Bargh, 1999). The main types of empathy can be broadly defined as cognitive, affective, and motor (Blair, 2005), which may have distinct neural correlates (Blair, 2005; Moore et al., 2015). The specific role of empathy within misophonia is yet to be explored. Research examining this may elucidate whether mimicry potentially reflects other heightened cognitive processes in these individuals, or alternatively if it is a byproduct of the underlying neural mechanisms of misophonia, or an effective coping strategy to attempt to inhibit activity in insular cortex. Interestingly, the anterior insula, which is particularly hyperactive in misophonia, has been long implicated as a neurobiological marker of empathy (e.g. Singer et al., 2004; Jabbi et al., 2007; Saarela et al., 2007). Therefore, another possibility of hyperactivation in the insula is that it reflects a greater degree of a specific type of empathy in individuals with misophonia.

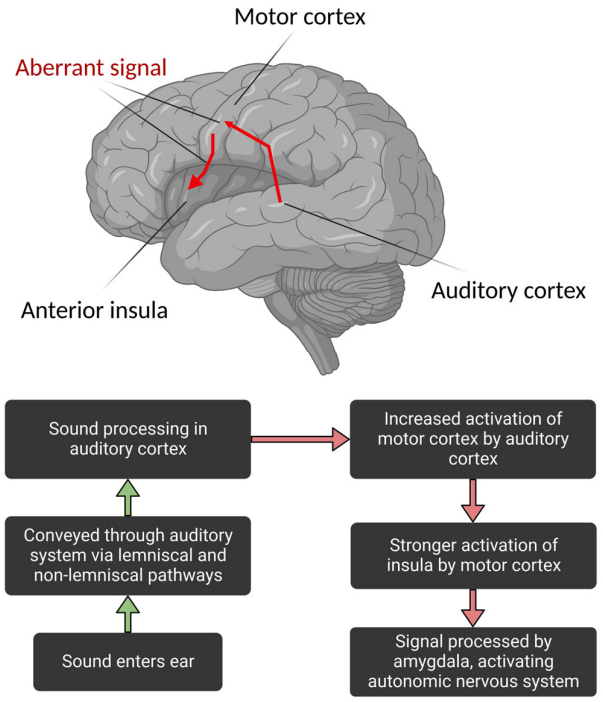

Figure 1: Simplified outline of proposed model. Simplified outline of proposed model. Sounds travel from the ear through lemniscal and non-lemniscal pathways auditory pathways, to be processed by higher level auditory cortex. Additionally, this signal is transmitted to motor regions to form a motor representation, which in the case of trigger sounds in misophonia is conveyed by an aberrant signal to the insula, causing hyperactivation of this region. The insula in turn communicates this to the amygdala, causing an emotional response and heightened autonomic processes (e.g., increased heart rate). Red arrows indicate part of pathway along which current evidence points to an aberrant signal being conveyed. Importantly, although we have implied unidirectionality in this model, there are likely bidirectional processes (such as those posited by predictive coding frameworks), that are as yet unknown and need further studies to explore.

As yet, a role for differing degrees of a particular subtype of empathy in misophonia has not been tested, though it is worth noting that the model we are positing here considers the concept of motor empathy to be a crucial one, rather than necessarily the cognitive or affective construct of empathy, at least based on current evidence.

There is already neuroimaging evidence highlighting regions that may underlie the importance of mirroring and context in the role of misophonia. As mentioned earlier, early auditory areas such as primary auditory cortex do not show a difference between misophonia sufferers and controls (Kumar et al., 2017; Schroder et al., 2019; Kumar et al., 2021). The earliest level in the hierarchy that appears to show any difference in misophonia sufferers is in the posterior superior temporal region (Schroder et al., 2019). Importantly, this area is also commonly implicated in imitation of others, through a proposed perception-action link mechanism (Molenberghs et al., 2010). When combined with evidence of the high incidence of mimicry in misophonia, it is suggestive of the possibility that "hyper-mirroring" may underlie the presence of the condition, as proposed by Kumar et al. (2021).

On the basis of the evidence presented above, we outline a simplified neural model for a social perspective of misophonia in Figure 1. From this perspective, auditory signals are conveyed through the lemniscal auditory pathways to motor regions as well as higher levels of the auditory system, such as superior temporal sulcus, transforming the stimulus into a motor representation. Indeed, increased connectivity is evident between the auditory system and motor regions in misophonia subjects (Kumar et al., 2021). Depending on the context of the auditory stimulus, this aberrant signal is then communicated to the insula, as evidenced by increased functional connectivity between motor regions and insula (Kumar et al., 2021), causing increased activation in this region (Kumar et al., 2017). Relevant to this, neuroimaging data show that anterior insula is engaged when countering a mirrored action, i.e. either deliberately or unintentionally performing an opposite imitative movement (Campbell et al., 2018), suggestive of an error signal being conveyed to this region. The insula then in turn may communicate this signal to the amygdala, resulting in an emotional response, as well as areas involved in autonomic reactions, such as periaqueductal gray, causing previously observed changes in physiology (e.g., heart rate, galvanic skin response; Kumar et al., 2017; Grossini et al., 2022). This neurobiology of misophonia involving a perception-action network highlights a potential explanation for the high incidence of mimicry, which may involve either unconscious or deliberate imitation, in an attempt to provide relief to the person experiencing misophonia and thus inhibit a hyperactive insula.

A potential therapeutic implication of a model incorporating the social cognitive aspects of misophonia would be to consider whether the underlying neurobiological or physiological responses to trigger sounds are changed by an altered context of the stimulus, in a similar manner to the premise of Samermit et al. (2019). That is, if a person with misophonia experiences a trigger sound re-framed in a different context, does this diminish the severity of their response? In the context of a social cognitive model of misophonia, one may assume that training subjects to re-frame the context of the stimulus could provide relief and act as a treatment strategy. Relevant to this, in a study of non-misophonia participants, galvanic skin responses – an indicator of stress – caused by 'nails screeching' on a blackboard were diminished and auditory stimuli were rated less unpleasant if subjects were under the false impression that the stimulus was from a piece of contemporary music (Reuter and Oehler, 2011). Additionally, any therapies based around a model that places a greater emphasis on the context of the stimulus, rather than the stimulus per se, would likely be generalisable to the visual analog of misophonia, i.e. misokinesia, of which to-date - to our knowledge - only two dedicated studies exist (Jaswal et al., 2021; Webb, 2022). Indeed, preliminary evidence suggesting some efficacy from cognitive behavioural therapy for misophonia is based on the premise that context is a critical component of the aversive response (Bernstein et al., 2013; Roushani and Mehrabizadeh Honarmand, 2021).

There are clearly a number of as yet untested hypotheses laid out here. The precise neural mechanisms of action perception in general is still under considerable debate, although the role of mirror neurons in underlying

imitation – defined as neurons that fire both when observing and executing an action – is supported even by the most prominent critics (Heyes, 2011; Hickok, 2014). Further exploration of the role of the perception-action system in mimicry in people with and without misophonia will help to inform the underlying neural basis of mimicry, both as it relates to this condition and also in a broader social cognitive framework. Furthermore, it is likely that further refinements of this model – or indeed any other models - will need to attempt to account for heterogeneity of trigger types in the condition. As is evident with other conditions, such as tinnitus, a homogenous approach that ignores the non-uniformity of a particular condition can often result in overlooking certain treatment strategies for particular subtypes, due to these same strategies not working for other subtypes of the particular condition (Cederroth et al., 2019; Hansen et al., 2022). With such a strategy, treatments can then be developed to attempt to alleviate misophonia on an individualised basis and hopefully minimise variation in treatment response.

In summary, how does considering misophonia within a social perception/cognition framework overcome the limitations of a sound-centric model? One, shifting the focus from sounds to the underlying action of the trigger person emphasis the importance of social context in misophonia. Two, the action perception framework also explains why brain structures such as anterior insula, which processes social signals, are activated in misophonia. Third, since mere observation of action of others is known to activate motor cortex (e.g. Buccino et al., 2001), the action perception framework accounts for the involvement of motor cortex in misophonia. Fourth, this model does not posit involvement of the auditory system beyond normal processes. Lastly, since the phenomenon of mimicry is common within social contexts, the high prevalence of mimicry in misophonia can be understood within this framework.

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Author contributions

J.I.B, P.E.G. and S.K. conceptualized the paper, J.I.B. and S.K. wrote the initial draft. J.I.B., P.E.G and S.K. all edited the final version.

Competing interest statement

No competing interests declared.