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A symptom network model of misophonia: From heightened sensory sensitivity to clinical comorbidity

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Abstract

Objectives: Misophonia—an unusually strong intolerance of certain sounds—can cause significant distress and disruption to those who have it but is an enigma in terms of our scientific understanding. A key challenge for explaining misophonia is that, as with other disorders, it is likely to emerge from an interaction of traits that also occur in the general population (e.g., sensory sensitivity and anxiety) and that are transdiagnostic in nature (i.e., shared with other disorders).

Methods: In this preregistered study with a large sample of participants ($N = 1430$), we performed a cluster analysis (based on responses to questions relating to misophonia) and identified two misophonia subgroups differing in severity, as well as a third group without misophonia. A subset of this sample ($N = 419$) then completed a battery of measures designed to assess sensory sensitivity and clinical comorbidities.

Results: Clinical symptoms were limited to the most severe group of misophonics (including autistic traits, migraine with visual aura, anxiety sensitivity, obsessive-compulsive traits). Both the moderate and severe groups showed elevated attention-to-detail and hypersensitivity (across multiple senses). A novel symptom network model of the

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data shows the presence of a central hub linking misophonia to sensory sensitivity which, in turn, connects to other symptoms in the network (relating to autism, anxiety, etc.).

Conclusion: The core features of misophonia are sensory-attentional in nature with severity linked strongly to comorbidities.

KEYWORDS

anxiety, comorbidity, interoception, misophonia, network model, sensory sensitivity

1 | INTRODUCTION

Misophonia is a disorder of decreased tolerance to specific sounds, referred to as triggers (Jastreboff & Jastreboff, 2001, 2002; Swedo et al., 2022). Trigger sounds can be idiosyncratic but are often human-made noises that originate from the mouth (lip-smacking, crunching, chewing, breathing). Triggers can also include repetitive noises (e.g., clicking and tapping) or sounds made by animals or inanimate objects (e.g., clocks, fans, bass from speakers etc.). While most people perceive such triggers as moderately irritating or might even fail to notice them, the emotional reactions experienced by misophonics include anger, disgust, anxiety, and feelings of powerlessness, as well as physiological responses such as increased heart rate, sweating and a strong wish to escape consistent with the fight-or-flight response (Dozier & Morrison, 2017; Edelstein et al., 2013; Rouw & Erfanian, 2018; Schröder et al., 2013, 2019). Although little is known about its epidemiology, misophonia is thought to be relatively common. Jakubovski et al. (2022) found that 5% of a German representative sample reported what they described as clinically significant misophonia. Misophonia is often comorbid with other clinical disorders and symptoms such as generalized anxiety (Wu et al., 2014), autism (Cassielo-Robbins et al., 2021; Rinaldi, Simner, et al., 2022) and obsessive-compulsive personality traits (Brout et al., 2018; Cavanna & Seri, 2015; Schröder et al., 2013). These profiling studies represent important first steps in characterising misophonia but what is lacking is a mechanistic understanding of how and why these features co-occur.

Current approaches to understanding mental disorders typically regard them as emerging from multiple simpler interacting elements that are, to some degree, prevalent within the general population and shared by other disorders (i.e., transdiagnostic). For example, Insel et al. (2010) proposed a new framework for research on mental disorders, which treats any contributing factor as interacting elements they term “domains,” in an approach the authors call Research Domain Criteria, RDoC. These domains consist of systems such as learning, arousal, and so on, with the ultimate goal of having a diagnosis and treatment based on an understanding of these mechanisms (Insel et al., 2010). A related mathematical modeling approach regards mental disorders as a network of symptoms (Fried et al., 2017), and symptoms themselves potentially explicable as a network of interacting simpler elements (like the domains in the RDoC approach). When several symptoms interact with each other they would appear, in a network model, as an inter-connected hub. These hubs may approximate traditional multi-symptom diagnostic categories. But note that they emerge in a data-driven way from sets of partial correlations (e.g., worry symptoms correlating with negative affect symptoms) independently of clinical judgments. Crucially, comorbidities arise because some symptoms/domains are shared between hubs (Fried et al., 2017). In this scenario, comorbidities are a natural product of how disorders arise, rather than a failure to develop sufficiently precise clinical classifications to avoid this situation in the first place (Borsboom, 2017). Network approaches naturally lend themselves to “spectrum”

views where each disorder can vary in terms of both severity and symptom presentation. This creates complex issues about how to diagnose and treat misophonia (Schröder et al., 2013) and, from a scientific perspective, how to identify relevant elements and understand their interactions. Network models are informative in this regard because it is possible to quantify whether symptoms are central (influence many other symptoms) or peripheral (having little influence) by, for example, summing the partial correlations emanating from each. The network approach has been applied to disorders such as post-traumatic stress disorder (Armour et al., 2017), bipolar disorder (Pfennig et al., 2016; Ritter et al., 2015) and psychosis (Isvoranu et al., 2016). Here we apply it to the first time to misophonia.

In terms of the simpler domains relevant to misophonia, there are several candidates which we explain in turn below: interoception (and/or physiological reactivity), sensory sensitivity (Wu et al., 2014), and social-emotional cognition (e.g., Cusack et al., 2018; Eijsker et al., 2019; Jager et al., 2020). Interoception refers to our ability to detect and interpret signals from the body (heart, viscera, etc.) and can itself be divided into further domains (e.g., interoceptive accuracy v. interoceptive awareness, Garfinkel et al., 2015). These processes may be linked to misophonia because auditory triggers elicit strong bodily reactions (Dozier, Lopez, et al., 2017; Edelstein et al., 2013) and neural activity in regions linked to interoception (Kumar et al., 2017). Sensory sensitivity is our responsiveness to the exteroceptive world (e.g., our responses to lights, sounds, tastes, odors etc.), and can be classified as hyper-sensitivity (over responsiveness, e.g., finding lights too bright) or hypo-sensitivity (under responsiveness, e.g., failing to notice smells, sounds etc.). Misophonia may be similar to a family of potentially related conditions that share this trait of sensitivity, including hyperacusis (Fackrell et al., 2017), autism (Ward et al., 2017), and migraine (Hibbard & O'Hare, 2015). There is some evidence that people with misophonia experience discomfort in response to a broad range of sounds not limited to their own triggers (Hansen et al., 2021) and this sensitivity even extends into heightened responses in the nonauditory domain (e.g., sensitivity to smells, tastes etc.; Rinaldi, Simner, et al., 2022; Wu et al., 2014). Finally, social skills may be implicated at the core of misophonia given that misophonia is characterized by interpersonal difficulties (e.g., annoyance with others), and changes in social behavior (e.g., avoiding family meals; Rinaldi et al., 2021) and since most misophonic triggers are human-made sounds (Edelstein et al., 2013).

In this study we test the hypothesis that misophonia co-occurs with a number of symptoms and traits, specifically: autistic traits (measured via the Autism Spectrum Quotient [AQ]; Baron-Cohen et al., 2006), anxiety (measured by the Anxiety-Sensitivity Index [ASI-3]; Taylor et al., 2007), migraines (measured via the Penn Online Evaluation of Migraines [POEM]; Kaiser et al., 2019), obsessive-compulsive traits (measured via the Obsessive-Compulsive Inventory [OCI]; Foa et al., 1998), as well as other hearing-related conditions (e.g., tinnitus, hyperacusis; Aazh et al., 2022). We also hypothesize that misophonia is directly related to heightened sensory sensitivity, as measured by the self-report Glasgow Sensory Questionnaire (GSQ) (Robertson & Simmons, 2013) which asks about different sensory modalities, and a task-based measure of "visual stress" termed the Pattern Glare test (Braithwaite et al., 2013). Here, striped patterns of different spatial frequencies are presented on a screen and participants experience mid- and high-spatial frequencies as particularly aversive, generating visual distortions (i.e., bending of lines and jittering) and somatic discomfort (i.e., headache and dizziness). Interoceptive sensibility can be regarded as a particular form of (embodied) sensory sensitivity and the multidimensional assessment of interoceptive awareness (MAIA) (Mehling et al., 2012) asks how much people notice, rely on, trust (etc.) their body sensations.

In terms of measuring and diagnosing misophonia itself, there are a variety of measures that have recently been developed that have good convergent validity with each other. Here we use the Sussex Misophonia Scale (SMS; Rinaldi et al., 2021; Rinaldi, Smees, et al., 2022) which asks about common misophonia triggers and contains a set of Likert questions which, when responses are summed together, offers excellent discriminative validity (Rinaldi et al., 2021). It has been independently validated against the most widely used misophonia measure to date (the Misophonia Questionnaire [MQ]; Wu et al., 2014) and has both convergent validity and divergent validity (Rinaldi et al., 2021). Another reason for choosing this measure is that it consists of five factors. This enabled us to explore possible heterogeneity amongst

misophonics (see also Norris et al., 2022). For example, some misophonics may be misophonic via high scores on Factors 1 and 2 and others via Factors 3–5. To anticipate our results, we will present a cluster analysis where we find that the misophonia subgroups are not defined according to individual factors, they are defined in terms of misophonia severity (i.e., high, medium, and low across all factors). We further demonstrate that severity is a strong predictor of co-morbidity of clinical symptoms. Finally, having five factors relating to misophonia enables these to be modeled separately within a symptom network model leading to a better understanding of how different facets of misophonia are linked to sensory sensitivities and clinical symptoms.

2 | METHOD

The method and analysis plan were fully preregistered (<https://osf.io/27fgv/>) and any deviations to the plan are explicitly noted. The anonymised data (raw and processed) are available at the same OSF location, alongside analysis scripts.

2.1 | Participants

Participants were recruited into a two-part online study from a variety of sources likely to favor either people with misophonia or non-misophonics. In the former case, a very large group of self-reported misophonics joined our participant pool following a BBC radio interview where we discussed misophonia and invited misophonics to contact us (May 18, 2021); we received $N = 1158$ responses after exclusions. In addition, a group of people were recruited via the student body, via acquaintances, and via an online participation panel (hosted by <https://www.prolific.co>); together these comprised $N = 272$ after exclusions. Assignment into groups was based on a cluster analysis of the factors scores from a validated measure of misophonia (SMS; Rinaldi et al., 2021) rather than the initial recruitment stream. Hence, the details of the extracted groups are reported in the Results (and their demographic details summarized in Table 1). To anticipate these results, we found that participants fell into three groups: non-misophonics, moderate misophonics, and severe misophonics (see Results and Table 1).

The inclusion criteria were being aged 18–70 years with self-reported normal or corrected-to-normal vision and hearing. In Part 2, participants with epilepsy were excluded (a normal criteria for the Pattern Glare test; Braithwaite et al., 2013; Evans & Stevenson, 2008). Additionally, participants were excluded after data collection if their responses were substantially incomplete (to be included participants needed to complete all of the questionnaires except the pattern glare task) or unfeasibly fast (<25 min across all measures, or <8 min on the Phenomenological Responses to Sounds task). Duplicate attempts at completing the task battery from the same participant were excluded (with the last full attempt retained). Details about excluded participants from Part 2 can be found in the Supporting Information Material.

Our final participants represented a combined sample of $N = 1430$, all of whom completed Part 1 of the study (misophonia screening and hearing profile), and a subgroup ($N = 419$) completed Part 2 (clinical comorbidities and sensory sensitivity). There was a small amount of missing data from the included participants ($N = 26$ for the pattern glare, $N = 1$ for the auditory sensory sensitivity task). For Part 2, an a priori power analysis, conducted in G*Power, based on $N = 201$ in two groups showed that this sample size would be sensitive to detect small effect sizes (Cohen's $d = 0.3$ or Pearson's $r = 0.15$ and above) at a power of 85%, $\alpha = 0.05$. Comparing sub-groups of $N = 105$ would be sufficient to detect medium effect sizes size (Cohen's $d = 0.5$ or equivalent) with a power of 95%, $\alpha = 0.05$.

This study was approved by The Cross-Schools Science and Technology Research Governance and Ethics Committee of the University of Sussex, UK.

TABLE 1 Demographic details of participants who completed the first and second parts of the study (the latter is a subsample of the former).

| Part 1: Participant characteristics following cluster analysis | | | |
|--|---------------------------------|------------------------------------|----------------------------------|
| | Non-misophonic (Cluster 1) | Moderate misophonia (Cluster 2) | Severe misophonia (Cluster 3) |
| N = 1430 | 367 | 643 | 420 |
| Age in years | 38.38 (17.38) | 47.17 (13.74) | 43.28 (14.14) |
| Gender (F: M: Non-bin) | 260: 103: 3 | 514: 126: 1 | 344: 65: 9 |
| Sussex Misophonia Scale, SMS | | | |
| N triggers (/48) | 10.27 (7.70) | 20.85 (9.42) | 27.50 (10.35) |
| Total score | 17.65 (13.85) | 65.18 (13.39) | 102.17 (14.92) |
| Total score > 50.5 | 0% | 83.8% | 100% |
| F1: Feelings of isolation | 7.63 (7.90) Z = -1.32 (0.49) | 30.93 (8.57) Z = 0.11 (0.53) | 45.30 (7.79) Z = 0.99 (0.48) |
| F2: Life consequences | 0.89 (1.45) Z = -0.95 (0.26) | 5.19 (3.45) Z = -0.17 (0.63) | 12.20 (4.58) Z = 1.10 (0.83) |
| F3: Inter- social Reactivity | 1.12 (1.55) Z = -1.12 (0.33) | 6.51 (3.21) Z = -0.02 (0.68) | 10.93 (3.73) Z = 0.95 (0.79) |
| F4: Avoidance Repulsion | 7.10 (4.93) Z = -1.33 (0.64) | 18.62 (3.87) Z = -0.18 (0.51) | 24.07 (3.74) Z = 0.89 (0.49) |
| F5: Pain | 0.90 (1.77) Z = -0.87 (0.39) | 3.93 (3.24) Z = -0.20 (0.72) | 9.67 (3.59) Z = 1.07 (0.79) |
| Part 2: Assessment of clinical comorbidities and sensory sensitivity | | | |
| | Nonmisophonic (Cluster 1) | Moderate misophonia (Cluster 2) | Severe misophonia (Cluster 3) |
| N = 419 | 196 | 113 | 110 |
| Age in years | 33.08 (14.29) | 42.09 (14.70) | 40.92 (13.95) |
| Gender (F: M: Non-bin) | 140: 55: 1 | 86: 25: 2 | 90: 17: 3 |

Note: Division into three groupings was made via a cluster analysis on the five factors (F1–F5) of the Sussex Misophonia Scale (SMS) on the data collected in Part 1 (N = 1430). Figures in parentheses show 1 S.D.

2.2 | Materials and procedure

Part 1 comprised the Hearing Questionnaire and SMS. All other measures were taken in Part 2. All research was conducted online with the tasks hosted on our own website (<https://www.misophonia-hub.org/>). The median time to complete Part 1 of the study was 7.77 min and for Part 2 it was 46.38 min. All questionnaire items required a forced response, whereas responses for the Phenomenological Responses to Sounds (considered elsewhere) and Pattern Glare task descriptors used a Visual Analog Scale (with an initial default of 0). Participants completing Part 2 were required to use a computer screen (a necessity for the Pattern Glare task) and ideally headphones (for the

Phenomenological Responses to Sounds task), or a computer speaker. A summary of the list of materials and procedure is given below in the order in which they are completed by participants.

2.2.1 | Hearing questionnaire

The hearing questionnaire describes distinct experiences of sound intolerance and hearing problems. Participants are asked "Do any of these apply to you?" and given descriptions of migraine phonophobia, phonophobia, misophonia, hyperacusis, tinnitus, and hearing difficulties. These were adapted from several sources (Dawes et al., 2020; Fackrell et al., 2017; Swedo et al., 2022) in consultation with a Clinical Audiologist. The descriptions are listed below.

"Migraine phonophobia: A temporary increase in sound intolerance that is related to a migraine attack and fades away after the event."

"Phonophobia: When some or all sounds consistently cause extreme fear or anxiety which does not bother other people in the same way."

"Misophonia: When sounds (e.g., crunching) consistently cause extreme emotions like anger or disgust which does not bother other people in the same way."

"Hyperacusis: When everyday sounds feel overwhelming, loud, intense, or painful that do not bother other people in the same way."

"Tinnitus: Experiencing persistent or intermittent noises (such as ringing or buzzing) in your head or in one or both ears that lasts for more than 5 min at a time (even if you haven't been exposed to loud noises or music)."

"Hearing difficulties: Do you find it very difficult to follow a conversation if there is background noise (such as TV, radio, children playing)?"

Participants answered these questions on a 4-point scale ("This definitely does NOT apply to me," "I don't think this applies to me", "This may apply to me," "This definitely DOES apply to me"). Additionally, we asked whether participants had sought any help (e.g., from a doctor, audiologist, and psychiatrist) for one or more of these problems with options Yes/No.

2.2.2 | SMS

The SMS is a two-part questionnaire with Part 1 eliciting information about specific triggers for misophonia (e.g., chewing sounds) and Part 2 comprising 39 Likert-type questions about the associated experiences of people with misophonia (e.g., feelings, behaviors and life consequences) (Rinaldi et al., 2021). Hence, Part 1 contains 48 potential triggers for misophonia (e.g., crunching) grouped into eight categories (e.g., "I hate... the sound of people eating") followed by the specific related triggers (e.g., "crunchy foods [e.g., apples]", "crispy snacks," "chewing" etc.). The questionnaire items in Part 2 (e.g., "I avoid going to work because of difficulties with sounds") are rated on a scale from 0 to 4 (Never, Hardly Ever, Sometimes, Often, Always), with the total score ranging from 0 to 156. The items in part 2 fall into five factors as follows: feelings/isolation (16 items), life consequences (work/friends) (6 items), inter-social reactivity (5 items), avoidance/repulsion (8 items), and pain (4 items). Rinaldi et al. (2021) show that a cut-off value of 50.5 on the SMS has "excellent" discriminative ability between misophonics and non-misophonics (AUC = 0.91). The Cronbach's alpha reliability of SMS in our sample was 0.98.

2.2.3 | Phenomenological responses to sounds

This task is written up as a separate study (Andermane et al., 2023), with the detailed methodology including stimulus creation described in the pre-registration (<https://osf.io/27fgv/>). This task is an extension of prior research in which misophonics are presented with a range of sounds and asked to rate them for discomfort (Edelstein et al., 2013; Hansen et al., 2021). The sound clips lasted 15 s each and belonged to four different categories, with 8 sounds per category (human oral-nasal, other human, nonhuman, and scrambled sounds). The key innovation in our task was to obtain a multivariate profile of responses across 17 descriptors (e.g., pain, rage, disgust and soothing) when listening to 32 sound clips.

2.2.4 | AQ

The AQ is 50-item questionnaire comprising five subscales: attention switching, attention to detail, communication, imagination, and social skill. The scores on each subscale can range from 1 to 10 (Baron-Cohen et al., 2001). Example items include "I would rather go to a library than a party" and "I enjoy doing things spontaneously" (reverse coded). Responses are given on a 4-point scale (Definitely disagree, Slightly disagree, Slightly agree, and Definitely agree) and recoded as 1 or 0 depending on whether the trait resembles an autistic behavior or not. The Cronbach's alpha reliability of the AQ in our sample was 0.85.

2.2.5 | The GSQ

The GSQ is a 42-item questionnaire with the total score ranging from 0 to 168 (Robertson & Simmons, 2013). Items covering seven sense domains (*visual, auditory, gustatory, olfactory, tactile, vestibular, proprioception*) with half of all items measuring *Hyper-sensitivity* (e.g., "Do you hate the feeling or texture of certain foods in your mouth?") and half measuring *hypo-sensitivity* (e.g., "Do you enjoy wearing very strong perfumes/after-shaves?"). Responses are given on a 5-point scale coded from 0 to 4 (Never, Rarely, Sometimes, Often, and Always). The Cronbach's alpha reliability of the GSQ in our sample was 0.90.

2.2.6 | The ASI-3

The ASI-3 includes 18 questions that are divided in three subscales: Cognitive, Physical, and Social (Taylor et al., 2007). Example items include "I worry that other people will notice my anxiety" (Social) and "When I have trouble thinking clearly, I worry that there is something wrong with me." (Cognitive). Responses are given on a 5-point scale from 0 to 4 (Very little, A little, Some, Much, Very much). The scores on each subscale can range from 0 to 24 and the total score from 0 to 72. The Cronbach's alpha reliability of the ASI-3 total in our sample was 0.93. The ASI was chosen over alternative measures (e.g., STAI State-Trait Anxiety Index, Spielberger et al., 1983) because the division into three factors fitted well with the symptom-level approach of this study but with the caveat that this may be capturing anxiety-related processes rather than longer-term history of clinical anxiety.

2.2.7 | The MAIA

The MAIA is a 32-item questionnaire that assesses interoceptive abilities or bodily awareness relating to distinct aspects of embodied experience (Mehling et al., 2012). It consists of eight subscales: Noticing (4 items), Not

Distracting (3 items), Not Worrying (2 items), Attention Regulation (7 items), Emotional Awareness (5 items), Self-Regulation (4 items), Body Listening (3 items), and Trusting (3 items). Examples include items such as “I listen to my body to inform me about what to do” (Body Listening) and “I notice when I am uncomfortable in my body” (Noticing). The items are rated on a scale from 0 to 5 (from Never to Always) and the subscale scores are calculated by computing the average score in each. Five items were reverse-scored. The Cronbach's alpha reliability of the total MAIA score in our sample was 0.92.

2.2.8 | OCI

The OCI (Foa et al., 1998) is a 42-item questionnaire assessing the experiences that characterize the obsessive-compulsive disorder (e.g., “Unpleasant thoughts come into my mind against my will and I cannot get rid of them” or “I check things more often than necessary”). The items are rated on a 5-point scale rated from 0 to 4 (Not at all, A little, Moderately, A lot, Extremely) and the total score ranges from 0 to 168. The 42 items form seven subscales, which are based on symptom categories that are commonly found in obsessive-compulsive disorder: Checking (9 items), Washing (8 items), Obsessing (8 items), Mental Neutralizing (6 items), Ordering (5 items), Hoarding (3 items), and Doubting (3 items). The Cronbach's α reliability of the OCI in our sample was 0.96.

2.2.9 | Migraine and headache (POEM)

The POEM covers the experience of migraines and headaches (Kaiser et al., 2019). The questionnaire has a branching logic, such that some questions are conditional on particular responses to other questions. The questions pertain to headache history, including headache duration, frequency and severity, as well as asking about particular headache types, headache triggers, and whether the headaches are accompanied by other symptoms (e.g., sensitivity to light and sound, visual experiences or aura). Based on their responses, participants are classified into one of seven categories: headache-free (HA-free), mild non-migrainous headache (MNMH), headache not otherwise specified (NOS), migraine without aura (MwoA), migraine with visual aura, and migraine with other aura, and migraine with both visual and other aura (the latter three belonging to the broader migraine with aura (MWA) category). The POEM is moderately sensitive (i.e., 42%–83% sensitivity) but is highly specific (i.e., 84%–100%) in identifying subjects as HA-free (those that are headache free or with mild MNMH), as well as those with migraines without aura, or migraines with aura.

2.2.10 | Visual sensitivity: Pattern glare task

This task of visual sensitivity was based on previous studies on visual stress (Evans & Stevenson, 2008; Fong et al., 2019). Participants are asked to view black and white parallel lines of various spatial frequencies (low, middle, and high) for 12 s, and then to rate the discomfort and reactions experienced when viewing these gratings (on 19 descriptors, see below). The stimuli were created by Braithwaite et al. (2013) and consisted of black and white alternating horizontal stripes presented in an oval window around a small fixation point and against a mid-gray (RGB 128,128,128) background. The low-, mid-, and high-spatial frequency stimuli comprised of 4.5, 31.5, and 130 cycles (i.e., stripes) and were always presented centrally at their actual resolution of 652 × 500 pixels. When viewed at an appropriate distance this corresponds to around 0.4, 3.0, and 12.4 cycles per degree (1° being around 40 pixels). Given that the blind spot has a known eccentricity of 12–15°, one can use this to calibrate a viewing distance by making two red dots located at 480 and 600 pixels (12–15° × 40 pixels) “disappear”—a form of “virtual chinrest” (Li et al., 2020). Participants were asked to close their left eye and stare at a central fixation cross. There were two red

dots, positioned to the right of fixation, and participants were instructed to move their head backwards and forwards until both dots disappeared. At this point, participants were instructed to open both eyes and remain at this approximate distance to the screen.

Our stimuli are known to trigger “glare” at medium and high (but not low) spatial frequency. This glare takes a number of different forms from person to person (e.g., lines appear to shimmer, or bend, or blur etc.; see below), and we provided 19 such descriptors which participants rated using a visual analog scale with slider values ranging from 0 to 100. An exploratory factor analysis (EFA) based on a separate normative sample (see the preregistered analyses on OSF: <https://osf.io/qpv7d>) revealed the presence of two factors that we refer to as Visual Distortions (comprising the descriptors: Illusory stripes, Bending of lines, Faint lines, Shadowy shapes, Shimmering, and Flickering) and Somatic Discomfort (comprising the descriptors: Headache, Unease, Nausea, Lightheadedness, Dizziness, Visual pain, Physical eye strain, Jitter, and Zooming). Factor scores were derived by averaging the responses across the relevant items for each spatial frequency (noting that two descriptors were not retained after factor analyses: “Colors” and “Blur”).

2.3 | Analysis plan

2.3.1 | Cluster analysis based on the SMS

A cluster analysis is a data-driven approach to identifying groups of participants (Jain, 2010). This approach is considered appropriate given that misophonia may well show sub-classes (e.g., differing by severity or symptom profile) even though there is currently no commonly agreed taxonomy for identifying them. We therefore ran an analysis that would allow us to stratify our sample into sub-groups, beyond a simple dichotomy of presence/absence of misophonia. For this analysis, the dependent variables were the scores of the five factors of the SMS (i.e., feelings/isolation, life consequences, intersocial reactivity, avoidance/repulsion, and pain), here converted to z-scores to ensure they are on the same scale (given the differing numbers of items within each factor). In theory, this type of approach could identify a subgroup of misophonics who are high on, for example, factors 1, 2 and 3 versus another subgroup high on 4 and 5. A two-step cluster analysis was performed (Zhang et al., 1996). The first step involves hierarchical clustering, using Ward’s method (Ward, 1963), and serves the purpose of identifying a plausible number of clusters and generating an initial set of cluster centroids (i.e. group means across the five factors). The second step involves a nonhierarchical k-means analysis with 50 iterations, which generates a final set of centroids and classifies each participant in one of the groups. Extracting three or more groups offers a way of exploring stratification beyond a simple dichotomy of presence/absence of misophonia and allows us to explore how participants might differ in their expression of misophonia.

2.3.2 | Inferential statistics

The questionnaires with continuous dependent variables (AQ, GSQ, OCI, MAIA, and ASI-3) were analyzed as mixed linear models (t tests, analysis of variance [ANOVA]) with participant group as a between-subject independent variable. Following the cluster analysis, the participants were grouped in three groups: nonmisophonic, moderate misophonia, and severe misophonia. Subscale was an additional independent variable (e.g., the AQ comprises five subscales). When appropriate, we applied post hoc one-way ANOVAs on each variable and Tukey HSD or Dunnett’s T3 comparisons to identify group differences depending, respectively, on whether equal variances are assumed or not (as indicated by the Levene’s test). Categorical dependent variables (e.g., for headache data in the POEM) are analyzed via chi-square against group membership. To account for multiple comparisons, we employed false discovery rate (FDR, Benjamini & Hochberg, 1995) on the set of *p* values for the group differences.

Effect sizes (Cohen’s *d*) and Bayes factors are calculated for each measure separately. Bayes factors are computed using the Dienes (2014) calculator implemented in R. The observed data for calculating Bayes factors is

the difference in means between the groups and the standard error (SE) of the difference. The prior was modeled using the room-to-move heuristic (Dienes, 2019). This heuristic makes the assumption that small group differences are more probable than larger differences and, moreover, that the ceiling of any such difference is the ceiling of the scale itself. Specifically, the distance between the non-misophonic mean and the scale ceiling is operationalized as two standard deviations of the half-normal.

2.3.3 | Symptom network modeling

We included 31 nodes in the model, one for each of the following symptoms (e.g., scale factors): SMS ($N = 5$), AQ ($N = 5$), GSQ ($N = 1$, i.e., taking the overall score), OCI ($N = 7$), MAIA ($N = 8$), ASI-3 ($N = 3$), and visual pattern glare responses to medium spatial frequency stimuli ($N = 2$). As these scales are continuous data, a Gaussian Graphical Model was used. This was conducted in R using the qgraph package with the code and data made public. The model also contains “edges,” which are the connections between nodes (symptoms), and are partial correlations estimated by pairwise random Markov fields (Epskamp et al., 2012). For example, an edge between the symptoms of anxiety sensitivity and sensory sensitivity would represent the connection between these symptoms in terms of their partial correlations in our data. Model selection was based on EBIC (Extended Bayesian Information Criteria) and the default gamma parameter of 0.5 (which controls how sparse the model is so not all nodes are fully connected). The graphical LASSO (least absolute shrinkage and selection operator) is applied for regularization, that is, to reduce over-fitting, using the R glasso package. This shrinks all coefficients in a systematic way and sets small ones to zero (Friedman et al., 2008).

After the network has been estimated, the robustness and stability was explored following the guidelines of Epskamp et al. (2018). This was conducted in R using the bootnet package with $N = 1000$ bootstrapped resamples taken. This ultimately replaces a single weight estimate for each edge with a distribution of weights for each edge from which other metrics can be derived: that is, each edge is a distribution of partial correlations and not a single partial correlation. The extent to which a node is central and can be calculated by summing the edges (partial correlations) emanating out of each node, and it is possible to observe how stable these measures are to changes in the sample. The CS-coefficient (Centrality Stability) was computed with the corStability function. This measures whether the order of the node centrality (ranked from most to least central) is stable after observing only portions of the bootstrapped data. If the network properties change substantially when dropping only a small number of participants, then the model is unstable. This is based on a correlation between the original and reduced datasets of at least $r = 0.7$ (the size of the reduced data set to achieve this correlation is estimated). Secondly, the function bootInclude returns a probability (0–1) that an edge will be present (i.e., weighted above zero) or absent in the bootstrapped models. A stable network would have edges that are consistently present and consistently absent.

3 | RESULTS

3.1 | Cluster analysis of SMS

Recall that participants were assigned into three groups (non-misophonic, moderate misophonia, and severe misophonia) based on their factor scores on the SMS (rather than the recruitment stream) which were entered into a cluster analysis. Visual inspection of the dendrogram (see Supporting Information Results) suggested the extraction of a small number of groups (2 or 3). However, if we limit our clustering to a two-group solution, this adds little more value than the original diagnostic cut-offs established via ROC curves (Rinaldi et al., 2021). A three-group solution to the cluster analysis generated groups who were low, medium, and high across all five factors of the SMS. These became our three groups, respectively, and the outcome of the cluster analysis can be seen in Table 1 (where z-scores are more informative because they act as a common scale) with the full sample ($N = 1430$). Specifically, the two groups scoring highest tended to come

from the self-referred misophonic stream¹, and generally met the diagnostic threshold for misophonia (a score > 50.5 established by using a ROC analysis of the total SMS score; Rinaldi et al., 2021). Indeed, 84% in the moderate misophonia group and 100% of the severe misophonia group crossed this diagnostic threshold, while 0% of the non-misophonic group crossed it. The top two groups (i.e., moderate/severe misophonia) also tended to self-report misophonia in the hearing questionnaire (91% in the moderate group and 98% in severe group, compared to 0% in the non-misophonia group). The SMS total scores have also previously contrasted with a commonly used measure of misophonia severity, the MQ (Wu et al., 2014), where an SMS total score > 50.5 corresponds to a moderate score of ≥ 7 on the MQ (Rinaldi et al., 2021), with severe scores on the MQ (≥ 10) linked to total SMS scores > 80 (see figure in Supporting Information: Results). As such, our designation of "Moderate" and "Severe" is in line with the nomenclature of other misophonia studies. The three groups did not differ significantly according to gender but the misophonic groups tended to be older ($F(2, 418) = 16.309, p < 0.001$), which likely reflects the different recruitment sources (many non-misophonics were recruited from a University sample). The Supporting Information Material shows a repeat of our analyses with closer demographically-matched samples (albeit a smaller N) and demonstrates that the key findings are not confounded by this.

In summary, a cluster analysis of misophonia scores (from the SMS questionnaire) points toward three groups that show a quantitative overall difference of misophonia in terms of symptom severity rather than a different profile across the five factors. Put differently, we do not see double dissociations among the z scores (e.g., one group high on factors 1, 2, 3, and another group high on factors 4 and 5) or even simple dissociations (e.g., one group high on four factors, and another high on all five factors). Further explorations show that the same trend (i.e., grouping based on severity) is found when extracting four groups (see Supporting Information Materials). The same trend is also found if factors that are highly correlated ($r > 0.7$) are combined together before the analysis (as specified in our preregistration). Ultimately, the utility of any stratification in misophonia can be judged by its ability to predict other measures and real-life behavior. The results reported below show exactly this: the three-group solution (i.e., two misophonic groups differing in symptom severity plus nonmisophonics) does indeed provide important new information about clinical comorbidities.

3.2 | Hearing questionnaire

The three groups differed significantly in their ratings for all the hearing-related conditions ($p < 0.05$), as shown in Figure 1, with full details of the one-way ANOVAs and post hoc t -tests reported in Supporting Information Materials. Most group differences were between the non-misophonics and the other groups, but differences amongst misophonics (moderate vs. severe) were found for hyperacusis ($p = 0.005$) and phonophobia ($p < 0.001$). Professional help-seeking was generally low for all conditions, although the groups also differed significantly here too across the board ($p < 0.05$). The sizeable difference between help-seeking in severe and moderate misophonia groups (22% vs. 6%) provides further external validity to the SMS and the notion of real-word differences between misophonic groups beyond a single diagnostic cut-off.

3.3 | Clinical comorbidities

Part 2 of the study assessed comorbidities and sensory sensitivity. The comorbidities that we considered are anxiety sensitivity (measured by the ASI-3), autism spectrum traits (measured by the AQ), obsessive-compulsive tendencies (measured by the OCI), and migraines/headaches (measured by the POEM). The results are summarized in Figure 2 (overall scores) and Figure 3 shows the effect sizes broken down by subscale (with means and SDs for subscale scores in the Supporting Information Materials).

For the AQ, there were significant main effects of group ($F(2, 416) = 10.69, p < 0.001, \eta^2 = 0.05$), subscale ($F(4, 1664) = 188.54, p < 0.001, \eta^2 = 0.31$), and a significant group X subscale interaction ($F(8, 1664) = 2.569,$

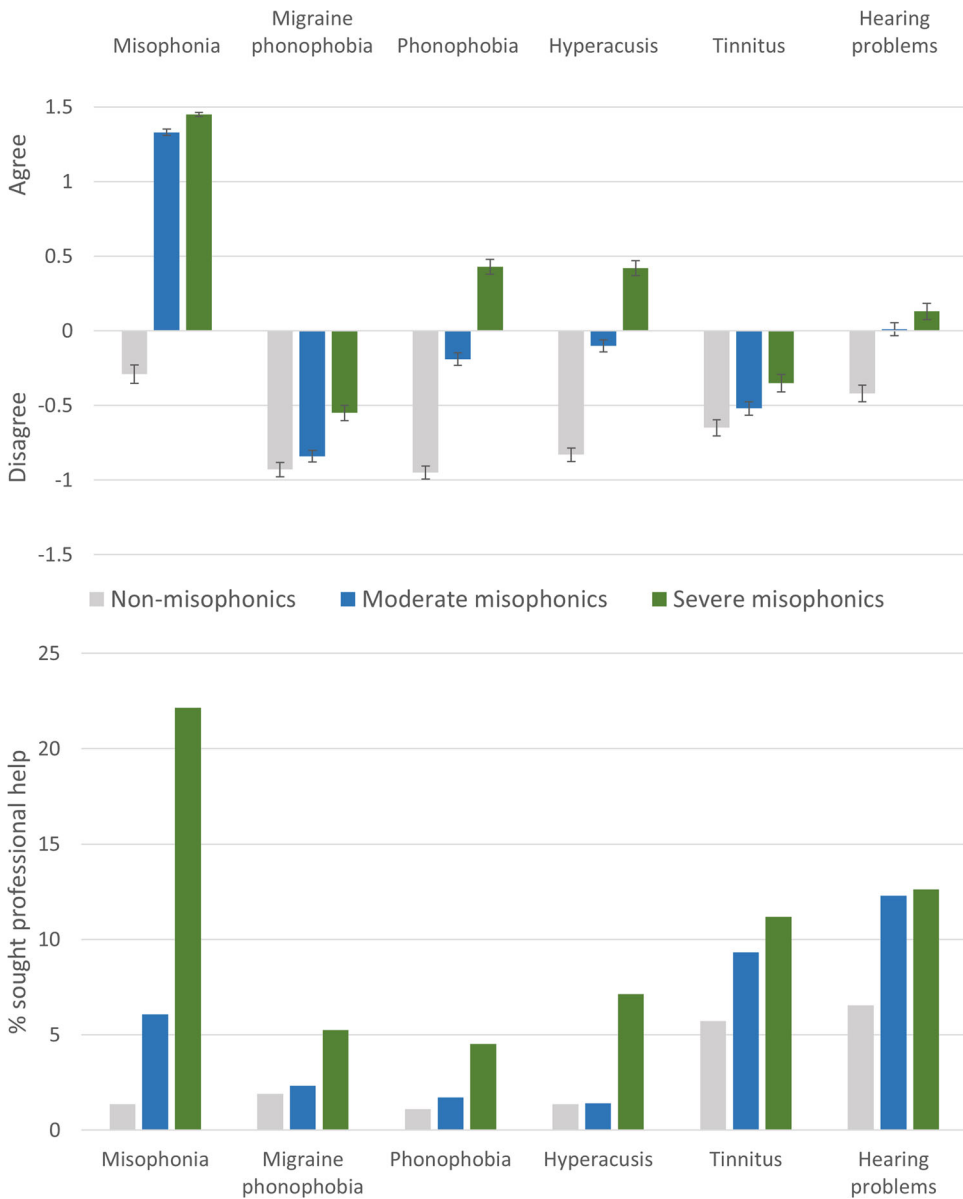


FIGURE 1 Hearing profile as a function of misophonia severity: (top) mean agreement scores relating to whether they felt this symptom applied to them (using a 4-point scale where 0 is neutral, error bars denote ± 1 SEM), (bottom) percentage of participants in each group who have sought professional help for each symptom.

$p < 0.001$, $\eta^2 = 0.01$). Post-hoc analyses showed significant group effects for social skills ($F(2, 416) = 3.76$, $p = 0.024$, $\eta^2 = 0.02$), attention to detail ($F(2, 416) = 17.10$, $p < 0.001$, $\eta^2 = 0.08$), and communication ($F(2, 416) = 7.46$, $p < 0.001$, $\eta^2 = 0.04$). Neither attention switching nor imagination were linked to significant group differences ($F(2, 416) = 2.91$, $p = 0.056$, $\eta^2 = 0.01$, and $F(2, 416) = 2.76$, $p = 0.065$, $\eta^2 = 0.01$, respectively). Post hoc pairwise comparisons showed that all three groups differed from each other on attention-to-detail (severe vs. moderate: $p = 0.034$; severe vs. non-misophonics: $p < 0.001$, moderate v. non-misophonics: $p = 0.008$) but the differences on social and communication subscales were driven by severe misophonics (social: severe vs. non-misophonics $p = 0.025$; communication: severe vs. non-misophonics $p < 0.001$, severe vs. moderate $p = 0.016$).

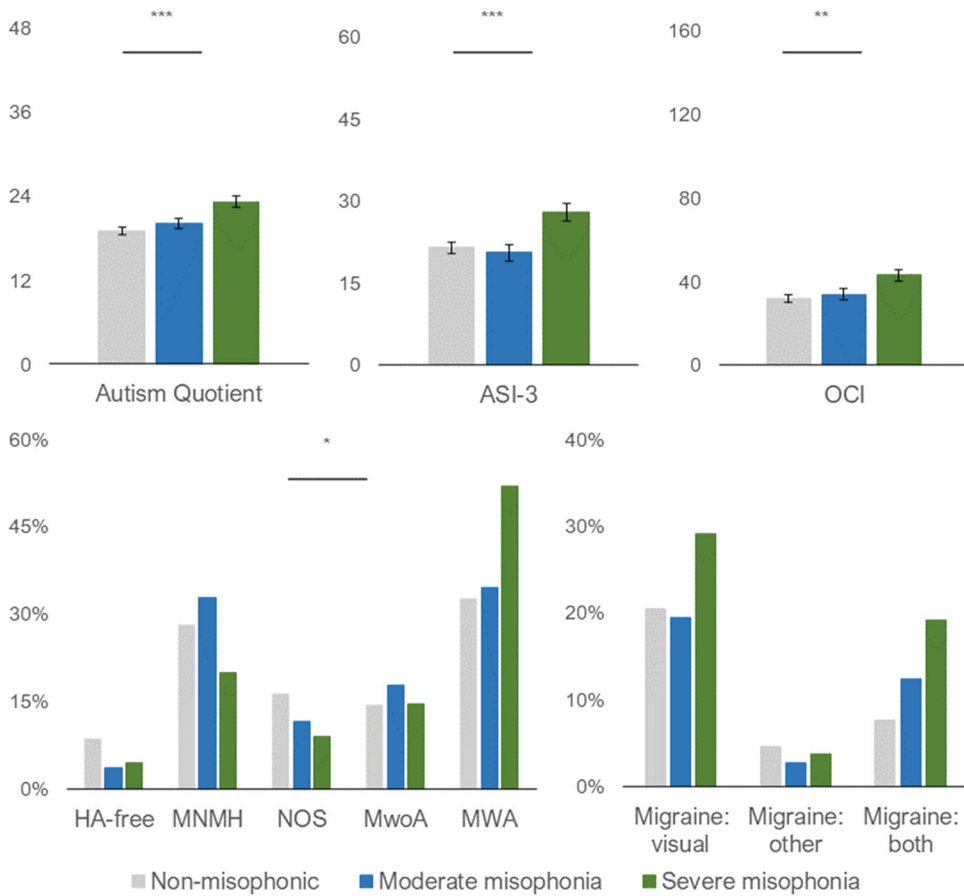


FIGURE 2 Group differences in clinical traits measured by the total Autism Spectrum Quotient (AQ) scores, total Anxiety Sensitivity Index (ASI-3) scores, and total Obsessive-Compulsive Inventory (OCI) scores. Mean \pm 1 SEM. The lower panels show the POEM headache categories: headache free (HA-free), mild nonmigrainous headache (MNMH), headache not otherwise specified (NOS), migraine without aura (MwoA), and migraine with aura (MWA). The MWA category is broken down into three specific aura symptoms (final panel).

For the ASI-3, there were significant main effects of group ($F(2, 416) = 8.06, p < 0.001, \eta^2 = 0.04$) and subscale ($F(2, 832) = 73.66, p < 0.001, \eta^2 = 0.15$), but no significant group \times subscale interaction ($F(4, 832) = 1.97, p = 0.10, \eta^2 = 0.01$). Post-hoc tests, combining subscale scores, showed that severe misophonics differed significantly from moderates and non-misophonics (both $p = 0.001$), with the latter groups not differing significantly ($p = 0.867$).

For the OCI, there were significant main effects of group ($F(2, 416) = 6.84, p = 0.001, \eta^2 = 0.03$) and subscale ($F(6, 2496) = 117.38, p < 0.001, \eta^2 = 0.22$), and a significant group \times subscale interaction ($F(12, 2496) = 2.09, p = 0.015, \eta^2 = 0.01$). Post hoc analyses showed significant group differences in five subscales (washing, checking, obsessing, ordering, doubting) which, in all cases, were due to significant differences between severe misophonics and non-misophonics ($p = 0.004, 0.025, 0.045, <0.001$, and 0.002 respectively), with no significant differences between moderate misophonics and non-misophonics. The difference between severe and moderate groups was significant for checking and ordering ($p = 0.026$ and 0.031 , respectively).

The POEM classifies individuals according to how they experience headaches in distinct classifications (see above). One category, nonvisual aura, was rare in isolation (generating expected counts < 5 in the χ^2) so these cases were excluded from the analysis. Among the remaining categories, the association between participant group and headache category was

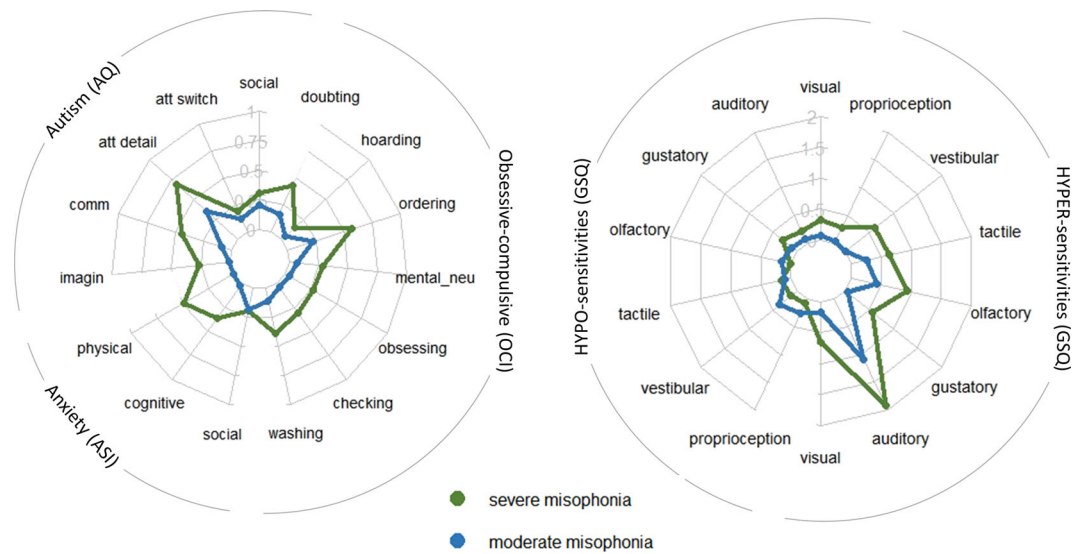


FIGURE 3 Effect sizes (Cohen's *d*) for the misophonic groups, relative to nonmisophonics, on individual subscales of the clinical questionnaires (left) and the Glasgow Sensory Questionnaire (right).

significant, $\chi^2 (10, N = 403) = 21.54, p = 0.018$. The severe misophonic group were more likely than the other two participant groups to experience migraines with aura (and less likely to have mild MNMH). Pairwise, severe misophonics differed from non-misophonics ($\chi^2 (5, N = 293) = 16.22, p = 0.006$) but moderate misophonics did not differ significantly from either the severe or non-misophonic groups ($\chi^2 (5, N = 297) = 6.67, p = 0.246$ and $\chi^2 (5, N = 216) = 7.94, p = 0.160$).

In summary, our research confirms findings of comorbidities between misophonia and autistic traits (Rinaldi, Simner, et al., 2022), anxiety sensitivity (Cusack et al., 2018), and obsessive-compulsive tendencies (Norris et al., 2022). We show, for the first time, a link between misophonia and MWA. The key novel contribution of our research is to show that clinical comorbidities are linked to the severity of misophonia rather than to misophonia status per se. We find only one trait that shows a significant difference linked to both groups of misophonia (moderate and severe) and this is unusual in that it relates to a cognitive ability rather than a problem/deficit (attention-to-detail from the AQ).

3.4 | Sensory sensitivity

Our measures of sensory sensitivity consisted of questionnaires examining hyper- and hypo-sensitivity in seven different senses (the GSQ), as well as interoceptive awareness/sensibility (the MAIA), together with a test of visual stress (the Pattern Glare task).

For the GSQ scores, a one-way ANOVA revealed a statistically significant difference between groups, $F(2, 416) = 24.16, p < 0.001, \eta^2 = 0.10$, driven by differences between severe misophonics. The other two groups did not differ. For exploratory analyses (not preregistered), the GSQ can also be broken down by sensory modality and hyper-versus hypo-sensitivity (over and under-responsiveness to incoming sensory information, respectively). This is summarized, as Cohen's *d* effect sizes, in Figure 4. For GSQ hyper-sensitivities, there were significant main effects of group, $F(2, 416) = 49.97, p < 0.001, \eta^2 = 0.07$, modality ($F(6, 2496) = 374, p < 0.001, \eta^2 = 0.30$), and a significant group X modality interaction ($F(12, 2496) = 16.40, p < 0.001, \eta^2 = 0.03$). Post hoc analyses showed significant group differences in most Hypersensitivity subscales (Visual, Auditory, Gustatory, Olfactory, Tactile, and Vestibular) which were due to significant differences between participants with severe misophonia and non-misophonics across all modalities (*p* values of $<0.001, <0.001, <0.001, 0.001, 0.019$, and <0.001 , respectively). Participants with moderate misophonia were also significantly

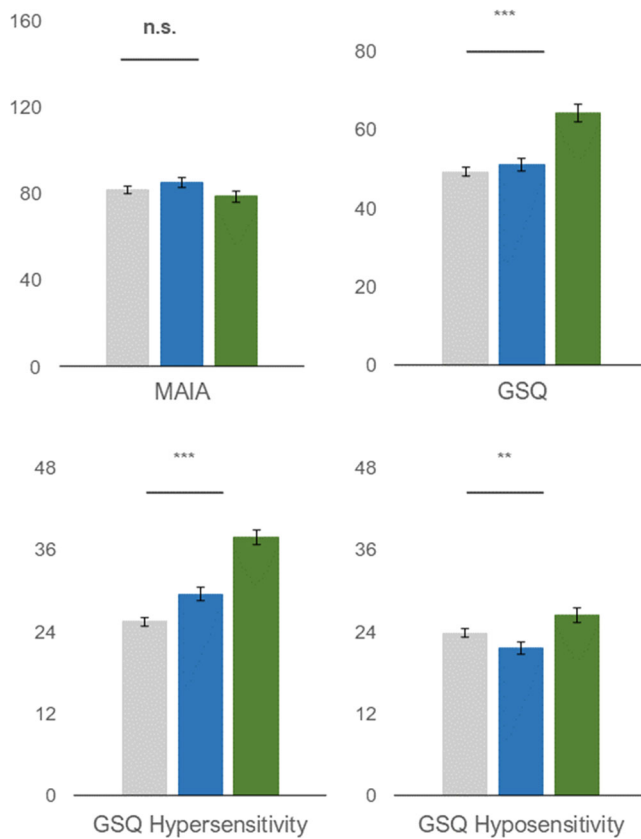


FIGURE 4 Sensory sensitivity questionnaires (mean \pm 1 SEM) that focus on exteroceptive senses (GSQ, Glasgow Sensory Questionnaire) or interoception (MAIA, Multidimensional Assessment of Interoceptive Awareness).

different from non-misophonics in terms of Auditory and Olfactory hypersensitivity ($p < 0.001$ and 0.002 respectively; individual auditory hyper-sensitivity questions are analyzed in the Supporting Information Results). Additionally, participants with moderate misophonia were significantly different from the severe group across all modalities. The groups also differed significantly on hyposensitivity ($F(2, 416) = 6.52, p = 0.002, \eta^2 = 0.03$), with marginally higher scores for severe misophonics relative to non-misophonics ($p = 0.073$) and significantly higher scores for severe misophonics relative to moderate misophonics ($p = 0.001$).

For the MAIA, there was no significant main effect of group ($F(2, 416) = 2.21, p = 0.111, \eta^2 = 0.01$) and no significant group \times subscale interaction ($F(14, 2912) = 1.536, p = 0.090, \eta^2 = 0.01$). The effect of subscale was significant ($F(7, 2912) = 67.48, p < 0.001, \eta^2 = 0.14$). This lack of difference in the interoceptive domain stands in contrast to the differences observed for exteroceptive senses. Post hoc group comparisons of groups are not reported due to lack of significant group effects. The Supporting Information Material reports individual subscales for completeness and alternative ways of grouping them.

Recall that the Pattern Glare task consists of three visual stimuli varying in spatial frequency (low, mid, and high), with a series of 17 descriptions (e.g., shimmer) broken into two different factors (somatic discomfort, visual distortions). These acted as repeated measures in a $3 \times 2 \times 3$ mixed ANOVA, with participant group as a between-subjects factor with three levels (non-misophonics, moderate misophonia, and severe misophonia), crossed with descriptor factor (somatic and visual), and spatial frequency of the grating (low, middle, and high spatial frequency). In previous literature, low spatial frequencies of visual gratings are not linked to visually induced stress and act here as a “sanity check” (e.g., to ensure we do not have

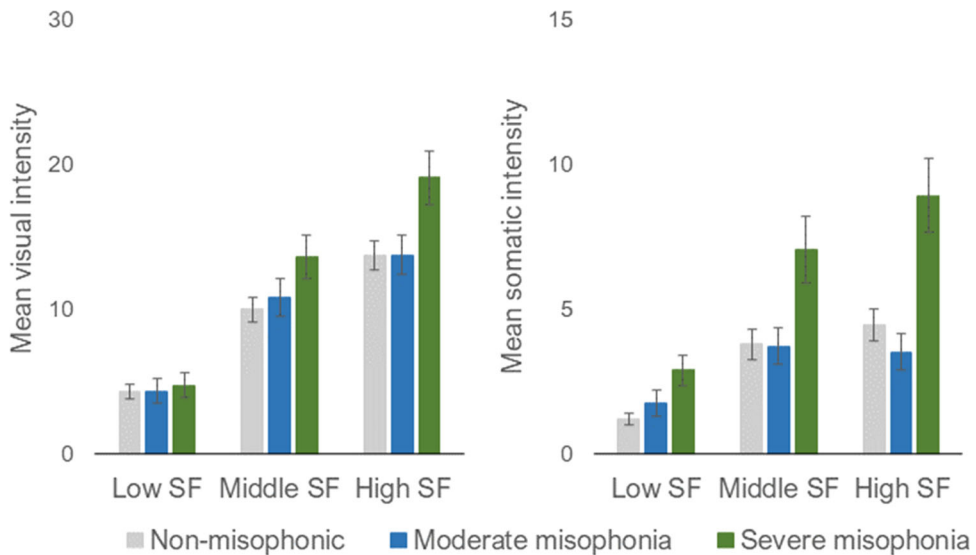


FIGURE 5 The average ratings for visual and somatic descriptors (left and right panels, respectively) for the three spatial frequencies of gratings (low, middle, and high). Group mean \pm 1 SEM.

biased or false responding). Figure 5 shows that the severe misophonic group gave higher ratings than other groups for both the mid- and high-spatial frequency conditions, which is confirmed by the analysis. There was a main effect of participant group ($F(2, 388) = 7.07, p < 0.001, \eta^2 = 0.02$), a main effect of spatial frequency ($F(2, 388) = 138.77, p < 0.001, \eta^2 = 0.07$), and a main effect of descriptor factor ($F(1, 388) = 204.27, p < 0.001, \eta^2 = 0.07$). There was also a group \times spatial frequency interaction ($F(4, 776) = 4.49, p = 0.001, \eta^2 = 0.004$), such that the groups differed in terms of their ratings in the different spatial frequencies; and a significant interaction between spatial frequency and descriptor factor ($F(4, 776) = 67.38, p < 0.001, \eta^2 = 0.02$), such that the different spatial frequencies were rated differently depending on which descriptor factor they belonged to. There was no significant three-way interaction. Post hoc analyses of the group \times spatial frequency interaction were performed (collapsing the visual and somatic factors by averaging the two scores). The three participant groups differed significantly at high ($F(2, 389) = 8.90, p < 0.001, \eta^2 = 0.04$) and middle ($F(2, 389) = 4.83, p = 0.008, \eta^2 = 0.02$) spatial frequencies, but not low spatial frequency ($F(2, 389) = 1.26, p = 0.286, \eta^2 = 0.04$). The group differences were driven by the severe misophonia group in these two spatial frequency conditions.

3.5 | Bayes factors and multiple comparisons

Considering the $N = 7$ main effects of group (AQ, ASI, OCI, POEM, GSQ, MAIA, and Pattern Glare) all except the MAIA were significant, and these six significant results survive correction for multiple comparisons using FDR.

Bayes Factors were computed at the level of scale and subscale ($N = 36$ variables) and reported in full in the Supporting Information Material. Comparing the moderate misophonia group against non-misophonics there were two sensitive ($BF > 3$) rejections of the null for AQ attention-to-detail and GSQ hypersensitivity (with almost all other results being sensitive null results of $BF < 1/3$). Comparing the severe misophonia group against non-misophonics there were thirteen sensitive ($BF > 3$) rejections of the null for AQ total score, AQ attention-to-detail, AQ communication, GSQ total score, GSQ hyper-sensitivity, GSQ hypo-sensitivity, ASI total score, ASI physical concerns, ASI cognitive concerns, OCI total score, OCI washing, OCI ordering, OCI doubting, and Pattern Glare somatic symptoms from high spatial frequency visual stimuli (other results were either insensitive or sensitive nulls).

3.6 | Symptom network modeling

Figure 6 shows the modeled symptom network based on partial correlations using the pre-registered dependent variables. For this approach, we no longer make use of three different groups but instead directly model the SMS factor scores as continuous variables (noting that our previous three groups are ranked by severity using the same factors, so is conceptually similar). In interpreting the model, we advise the reader that any nonzero weighted association can be interpreted as making a significant contribution to the model. The thickness of the lines indicates the strength of the association measured as partial correlations (these numeric values are provided in Supporting Information Results along with exploratory, not preregistered, networks that yield similar results). The spatial separation of the symptoms, for visualization purposes, is also based on the strength of association ignoring whether the partial correlation is positive or negative (termed a spring layout; Kamada & Kawai, 1989). Symptoms known to belong to the same construct (e.g., different subscales of the AQ) tend to associate together and this is to be expected if the construct is meaningful (a ‘clique’ in network terms occurs when a set of nodes is fully interconnected as happens here for SMS with other constructs being near-cliques). There are notable exceptions. The AQ Attention-to-detail (but not other AQ subscales) has significant partial correlations to the Ordering and Hoarding subscales of the OCI which results in it being placed closer to the OCI than to other AQ subscales in the diagram. A similar mismatched pattern emerges for the Not-Distracting subscale of the MAIA (e.g., “I try to ignore pain”), which negatively connects with other constructs such as GSQ and OCI-Doubting and is perhaps conceptually more related to attention than interoception.

There are several measures of centrality which give an indication of the relative importance of a symptom in the network (see Figure 7). First, degree centrality sums all the connection strengths emanating from each symptom (ignoring sign). This means that symptoms with a higher degree centrality either have more links than others, or a

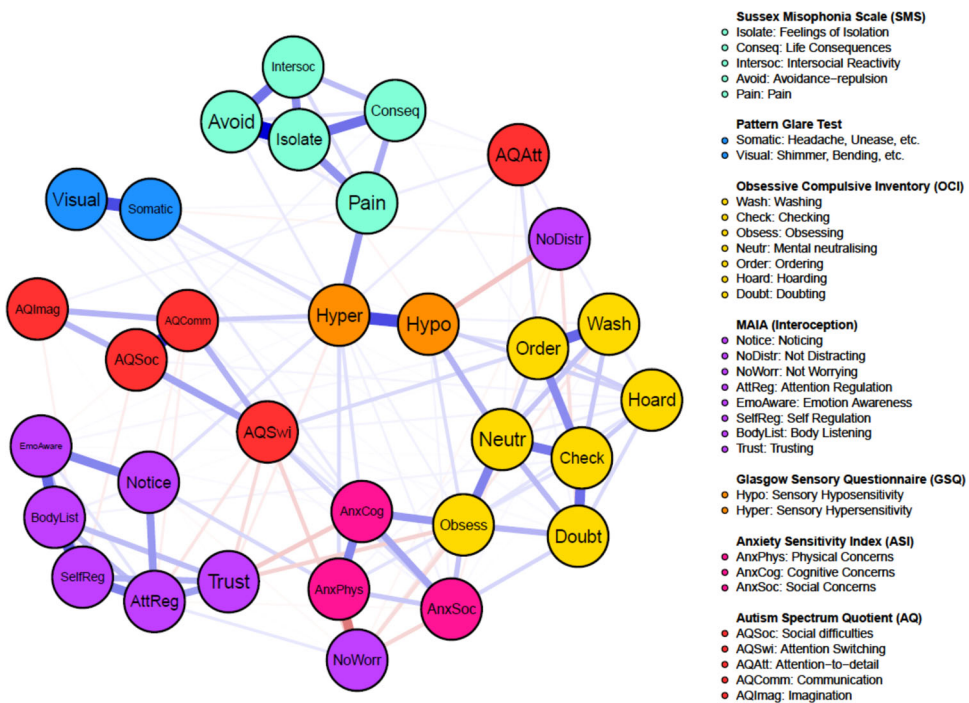


FIGURE 6 A symptom network model showing the relationship between misophonia (in turquoise) and other symptoms/behaviors. The strength of the lines indicates degree of association with blue depicting positive associations and red depicting negative associations. Comorbidities can be understood, in this approach, through mutual influence of associated symptoms (which spread throughout the network).

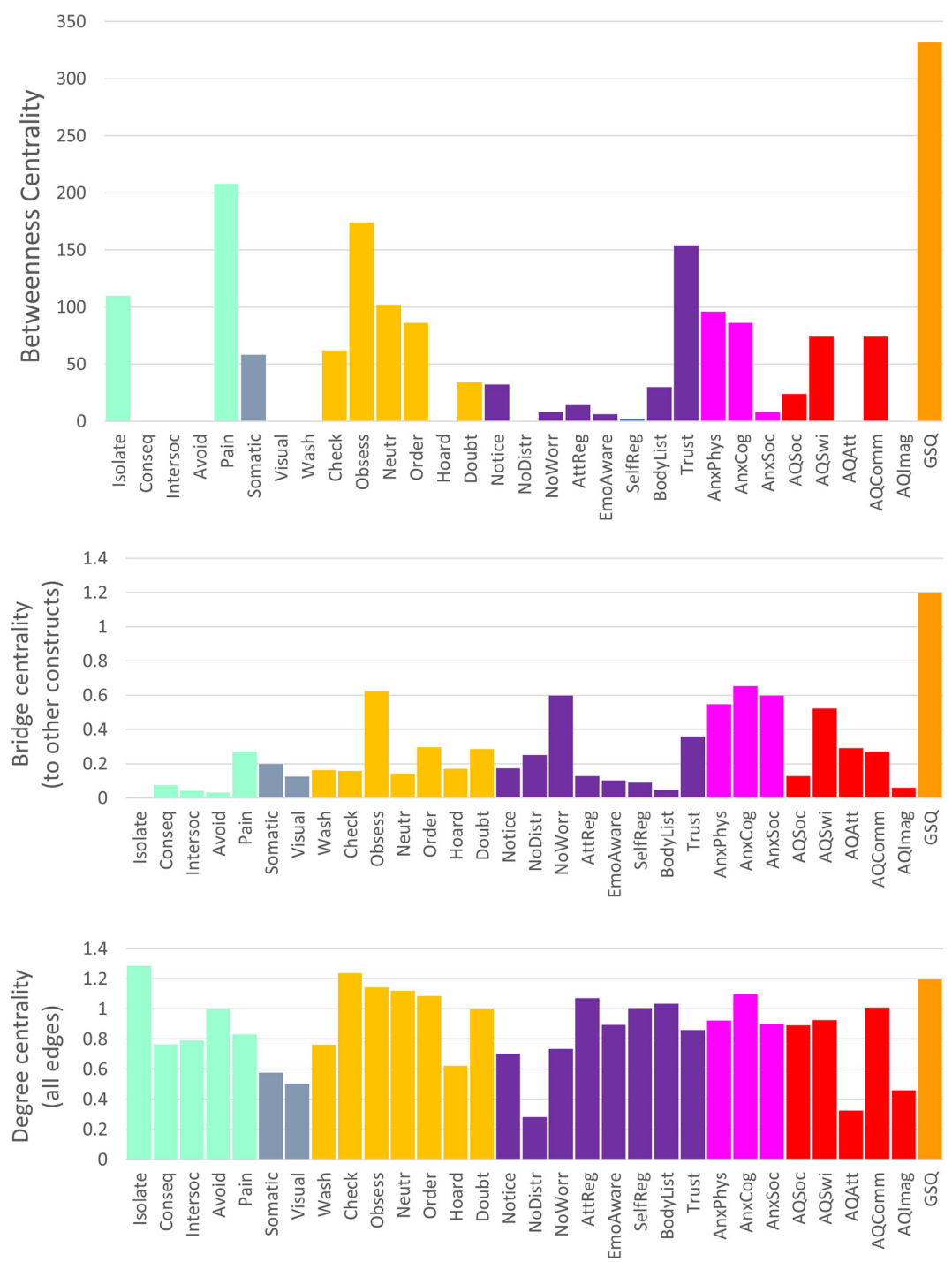


FIGURE 7 Different measure of centrality. Top: Betweenness centrality measures the extent to which a node serves as a bridge from one part of the network to another (unweighted shorted paths). Middle: Most nodes increase their degree centrality by forming clique-like arrangements with similar subscales, and the middle panel strips this away to show summed “bridge” weights to different measures. Bottom: Degree centrality measure the sum of weights emanating from each symptom.

smaller number of very strong links. Second, bridge centrality is mathematically equivalent to degree centrality except that it selects only those weights between (not within) different measures (Jones et al., 2021): in Figure 6 we can think of it in terms of summing only the connections between different colored circles. Third, betweenness centrality is related to how many shortest paths between all pairs pass through a given symptom: an analogy is that the shortest path between two cities in the United Kingdom, in terms of rail connections, is often via London (i.e., London has very high betweenness centrality). To calculate betweenness centrality, every pair in the network is considered and one counts how many times a node can interrupt the shortest path between the two nodes of the pair. The GSQ (sensory sensitivity) occupies a key position in the symptom network on all counts (see below), and this is maintained even if sensory sensitivity is split into separate hyper- and hypo-sensitivity variables (see Supporting Information Results). Within the five SMS subscales, the one with the highest bridge and betweenness centrality is Pain. Summing connections between the SMS and the different AQ subscales we find the strongest link to attention-to-detail. The overall results of the network simulation support the conclusion that comorbidities linked to misophonia occur primarily via sensory-attentional symptoms that are themselves transdiagnostic (i.e., linked to constructs such as autism, OCD, and anxiety). This finding is not trivial insofar as there were other plausible outcomes that could have emerged. For example, social-based symptoms (the social subscales of the AQ, SMS, MAIA, etc.) could intuitively have formed a central clique but did not.

The network model was explored following a bootstrap analysis, in which participants are randomly sampled with replacement. The bootstrapped CS-coefficient was 0.75 where a value greater than 0.5 is considered good and a value less than 0.25 is poor. This means that up to 75% of cases could be dropped and one would still obtain a correlation of 0.7 (the recommended value of Epskamp et al., 2018) between the original and reduced datasets (in terms of degree centrality estimates). This is akin to a post-hoc power analysis because it determines whether the actual sample size is adequate to achieve network stability.

Further, post hoc, explorations of the network were conducted and summarized here (see Supporting Information Materials for full details). The GSQ continues to occupy a central position even after all auditory items are removed. In this revised model, the strongest connections from the SMS are still to the GSQ with AQ attention-to-detail as a second bridge symptom (both emanating from the SMS Pain factor). We also reran an exploratory network analysis dividing our sample into misophonics and non-misophonics (taking the groups derived from the earlier cluster analysis) and show that the overall network structure is similar. One potentially important difference is that the bridge symptom linking misophonia (the SMS subscales) to the remaining network shifts from the Avoidance subscale (considering non-misophonics alone) to Pain (misophonics alone, and in the combined sample). This suggests a transition from aversion to sensitization along the misophonia spectrum.

4 | DISCUSSION

In the present study we aimed to characterize the perceptual, affective, and cognitive profile of individuals who experience misophonia, a type of aversive emotional and physiological reaction to classes of everyday sounds (e.g., human-made or repetitive). We also set out to explore whether misophonia can be categorized into distinct subgroups, and if so, whether groups of misophonics tend to differ quantitatively or qualitatively in the severity of their experience, as well as their sensory sensitivities and the tendency for clinical traits. The associations between our variables were modeled using a symptom network approach to identify how these are related.

We used a validated measure, the SMS (Rinaldi et al., 2021), to identify symptoms and traits of misophonia. For the present research this measure has the advantage of having five factors, which can capture heterogeneity in symptoms. Importantly, its questions were written to be understandable to people without misophonia and can therefore also be modeled as a continuous variable (spectrum). Our cluster analysis revealed that our sample of participants can be categorized into three groups, based on their standardized scores on the five factors of the SMS. The groups were distinguished by the severity of misophonia: the upper two groups largely self-identified as having

misophonia and would exceed the threshold value for diagnosing misophonia from the SMS (i.e., when a single score is calculated across all factors). The third group would fall below the threshold of SMS and comprise participants who were recruited as controls and who do not self-identify as misophonic. Whereas other studies have noted the continuous nature of misophonia symptoms using other measures (Edelstein et al., 2013; Wu et al., 2014), here we have shown that three distinct groupings fall out from a cluster analysis. Crucially, these three groups also show differences in their sensory sensitivity profile and clinical traits.

The group we classed as moderate misophonia are similar to the non-misophonic group on most measures (i.e., anxiety, migraine, pattern glare, obsessive-compulsive traits, and most AQ subscales) but are nevertheless different in some important ways (i.e., AQ attention to detail, GSQ hyper-sensitivity in auditory and olfactory modalities, and hearing comorbidities such as hyperacusis). In effect, there are multiple transitions in symptom profile that coincide with increasing misophonia severity. The moderate group, while still being “misophonic” (i.e., experiencing its negative impact in important ways), will not necessarily all have *clinical needs*, in the sense that they can likely function in life without external intervention. The fact that the moderate group often lack comorbidities (sensitive null results) suggests that these comorbidities are not a primary cause of misophonia, although they may play a role in the transition to a more severe profile. Those in the severe group are likely to be at a higher clinical symptom level, both in terms of their misophonia itself but also from their wider clinical profile. A recent study also applied clustering methods in misophonia and, like our study, identified three groups differing in severity (including one without misophonia) but, unlike our study, found more clinical comorbidities in the moderate group (Norris et al., 2022). There are multiple differences between the studies that may have affected the results. In our study, the clustering was based on the misophonia profile alone (with other measures acting as independent tests of the clusters) whereas Norris et al. (2022) derived their clusters from a mix of misophonia and clinical measures. Our study used purposive sampling (explicitly targeting misophonics of different severity levels) whereas Norris et al. (2022) used opportunity sampling (from the general population) such that people with clinically significant misophonia were a minority in all of their clusters (0.8%, 18.6%, and 33.3%). Thus, it is hard to be certain of the extent to which their cluster differences are directly attributable to misophonia.

Our symptom network model showed that some symptoms/traits are closely connected to misophonia while others are more distant. In terms of our groups, we can think of this in terms of severe misophonia having a wider reach within the network, and moderate misophonia having a more local symptom profile. The key statistical innovation of the symptom network is its use of partial correlations over regular correlations. Thus, in a three-symptom network $A \leftrightarrow B \leftrightarrow C$, the correlation between A and C would be close to 1 and the partial correlation would be close to 0. Having a sparser network model is also achieved through a process called regularization (where weak connections are forced to zero) with the degree of regularization itself based on the goodness of fit of model to data (using EBIC, Extended Bayesian Inference Criteria) (Epskamp et al., 2018). Sparser models, fit to a statistically optimal criteria, help us to understand the relationship between symptoms. But it is not a causal model. In particular, we cannot conclude that misophonia causes the comorbidities or vice versa, but we can conclude that closer and stronger connections have greater mutual influence over each other. Specifically, the main bridge between misophonia and other clinical symptoms appears to be via sensory sensitivity. This includes auditory sensory sensitivity but is not limited to it, and it is important to note that the auditory questions in the GSQ hypersensitivity subscale are not necessarily asking about misophonia itself (e.g., “Do you react very strongly when you hear an unexpected sound?”, “Do you dislike loud noises?”). As a limitation of our network model, it is conceivable that sensory sensitivity ceases to be central when other important, but presently omitted, symptoms are added (Neal et al., 2022). However, we made reasonable efforts to include a set of symptoms based on our current knowledge of the field.

Increased sensory sensitivity may lead to the experience of pain and affective (emotional) dysregulation, corresponding to anxiety and other clinical traits. However, the exact mechanism is yet to be fully explained. Other researchers have proposed that in misophonia there is an increased sensory (auditory cortex) functional connectivity to anterior insula, which represents interoceptive maps and moment-to-moment general emotional wellbeing of the individual (Kumar et al., 2017; McGeoch & Rouw, 2020). This increased connectivity may be the

neural mechanism that is formed when associations between auditory input and negative valence emotions are learned (McGeoch & Rouw, 2020). In our research individual differences linked to interoceptive awareness (as measured by the self-report MAIA, Mehling et al., 2012) were not strongly related to misophonia. It is possible that alternative measures of interoception (e.g., Garfinkel et al., 2015; Garfinkel et al., 2016; Miller et al., 1981) will be more strongly related to misophonia. It is also possible that it is the production of autonomic responses to sounds that is atypical in misophonia, rather than the ability to sense them (via interoception).

Previous observations in misophonia have noted the presence of visual triggers (e.g., repetitive actions) but it is unclear whether this can be related to visual sensitivity per se or the fact that these actions imply a sound (Edelstein et al., 2013). Our research provides the most compelling evidence, to date, that misophonia is linked to visual sensitivity, at least in the more severe cases. This is manifested in terms of reports of migraine (specifically for MWA) and in terms of both somatic reactions and visual distortions to aversive striped patterns. These two observations—migraine and pattern glare—are known to be linked (see Marcus & Soso, 1989; Wilkins et al., 1984) and have a shared putative neural mechanism of increased (visual) cortical excitability (Hibbard & O'Hare, 2015; Huang et al., 2003). That is, it need not be the case that misophonia causes migraine, or vice versa, but that both are different clinical outcomes of a common mechanism (increased cortical excitability). A stronger influence of this mechanism may give rise to stronger symptom severity and increased chances of co-morbidity as multiple brain systems become affected.

To summarize, severe misophonia differs from moderate misophonia not only in terms of quantitative differences in symptom severity (i.e., SMS factor scores) but also qualitatively in the perceptual and clinical traits of these groups. Specifically, severe misophonics differ from moderates in the breadth of sounds that are reported as triggers, in a broader profile of sensory sensitivities (i.e., visual, tactile), elevated clinical traits (autism spectrum, anxiety sensitivity, and obsessive-compulsive traits), a greater tendency to have migraines and experience other hearing related issues (e.g., hyperacusis). Notably, moderate misophonics did not differ from controls in terms of clinical traits. From this comparison of misophonia subgroups, it appears that elevated sensory sensitivity and associated pain-like experiences in response to sensory stimuli may be the primary cause for the aversive effects of misophonia on mental health and life consequences of the severe group. This is in line with our network analyses, showing that sensory sensitivity is the central trait, connecting to the misophonic experiences (especially the SMS factor of pain in misophonia), and the other sensory and clinical traits.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available in Open Science Framework at <https://doi.org/10.17605/OSF.IO/27FGV>.

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PEER REVIEW

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ENDNOTE

- ¹ Of the $N = 1158$ recruited as self-reported misophonics following the radio interview $N = 1031$ (89%) fell into the two misophonic groups, and of the $N = 272$ recruited as control participants from normative samples $N = 240$ (88%) were classed as non-misophonic; $\chi^2(1, N = 1430) = 689.31, p < 0.001$.

REFERENCES

- Aazh, H., Erfanian, M., Danesh, A. A., & Moore, B. C. J. (2022). Audiological and other factors predicting the presence of misophonia symptoms among a clinical population seeking help for tinnitus and/or hyperacusis. *Frontiers in Neuroscience*, 16, 900065. <https://doi.org/10.3389/fnins.2022.900065>
- Andermane, N., Bauer, M., Sohoglu, E., Simner, J., & Ward, J. (2023). A phenomenological cartography of misophonia and other forms of sound intolerance. *iScience*, 26(4), 106299. <https://doi.org/10.1016/j.isci.2023.106299>
- Armour, C., Fried, E. I., & Olff, M. (2017). *PTSD symptomics: network analyses in the field of psychotraumatology* (8, p. 1398003). Taylor & Francis.
- Baron-Cohen, S., Hoekstra, R. A., Knickmeyer, R., & Wheelwright, S. (2006). The autism-spectrum quotient (AQ)—adolescent version. *Journal of Autism and Developmental Disorders*, 36(3), 343–350.
- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders*, 31(1), 5–17.
- Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal statistical society: Series B (Methodological)*, 57(1), 289–300.
- Borsboom, D. (2017). A network theory of mental disorders. *World Psychiatry: Official Journal of the World Psychiatric Association (WPA)*, 16(1), 5–13.
- Braithwaite, J. J., Brogna, E., Bagshaw, A. P., & Wilkins, A. J. (2013). Evidence for elevated cortical hyperexcitability and its association with out-of-body experiences in the non-clinical population: New findings from a pattern-glare task. *Cortex*, 49(3), 793–805.
- Brout, J. J., Edelstein, M., Erfanian, M., Mannino, M., Miller, L. J., Rouw, R., Kumar, S., & Rosenthal, M. Z. (2018). Investigating misophonia: A review of the empirical literature, clinical implications, and a research agenda. *Frontiers in Neuroscience*, 12, 36.
- Cassello-Robbins, C., Anand, D., McMahon, K., Brout, J., Kelley, L., & Rosenthal, M. Z. (2021). A preliminary investigation of the association between misophonia and symptoms of psychopathology and personality disorders. *Frontiers in Psychology*, 11, 519681.
- Cavanna, A. E., & Seri, S. (2015). Misophonia: Current perspectives. *Neuropsychiatric disease and treatment*, 11, 2117–2123. <https://doi.org/10.2147/ndt.S81438>
- Cusack, S. E., Cash, T. V., & Vrana, S. R. (2018). An examination of the relationship between misophonia, anxiety sensitivity, and obsessive-compulsive symptoms. *Journal of Obsessive-Compulsive and Related Disorders*, 18, 67–72.
- Dawes, P., Newall, J., Stockdale, D., & Baguley, D. M. (2020). Natural history of tinnitus in adults: A cross-sectional and longitudinal analysis. *BMJ Open*, 10(12), e041290.
- Dienes, Z. (2014). Using Bayes to get the most out of non-significant results. *Frontiers in Psychology*, 5, 781.
- Dienes, Z. (2019). How do I know what my theory predicts? *Advances in Methods and Practices in Psychological Science*, 2(4), 364–377.
- Dozier, T. H., Lopez, M., & Pearson, C. (2017). Proposed diagnostic criteria for misophonia: A multisensory conditioned aversive reflex disorder. *Frontiers in Psychology*, 8, 1975.
- Dozier, T. H., & Morrison, K. L. (2017). Phenomenology of misophonia: Initial physical and emotional responses. *The American Journal of Psychology*, 130(4), 431–438. <https://doi.org/10.5406/amerjpsyc.130.4.0431>
- Edelstein, M., Brang, D., Rouw, R., & Ramachandran, V. S. (2013). Misophonia: Physiological investigations and case descriptions. *Frontiers in Human Neuroscience*, 7, 296.
- Eijsker, N., Schröder, A., Smit, D. J. A., Van Wingen, G., & Denys, D. (2019). Neural basis of response bias on the stop signal task in misophonia. *Frontiers in Psychiatry*, 10, 765.
- Epskamp, S., Borsboom, D., & Fried, E. I. (2018). Estimating psychological networks and their accuracy: A tutorial paper. *Behavior Research Methods*, 50(1), 195–212.
- Epskamp, S., Cramer, A. O. J., Waldorp, L. J., Schmittmann, V. D., & Borsboom, D. (2012). qgraph: Network visualizations of relationships in psychometric data. *Journal of Statistical Software*, 48, 1–18.
- Evans, B. J. W., & Stevenson, S. J. (2008). The pattern glare test: A review and determination of normative values. *Ophthalmic and Physiological Optics*, 28(4), 295–309.
- Fackrell, K., Potgieter, I., Shekhawat, G. S., Baguley, D. M., Sereda, M., & Hoare, D. J. (2017). Clinical interventions for hyperacusis in adults: A scoping review to assess the current position and determine priorities for research. *BioMed Research International*, 2017, 1–22.

- Foa, E. B., Kozak, M. J., Salkovskis, P. M., Coles, M. E., & Amir, N. (1998). The validation of a new obsessive-compulsive disorder scale: The Obsessive-Compulsive inventory. *Psychological Assessment*, 10(3), 206–214.
- Fong, C. Y., Takahashi, C., & Braithwaite, J. J. (2019). Evidence for distinct clusters of diverse anomalous experiences and their selective association with signs of elevated cortical hyperexcitability. *Consciousness and Cognition*, 71, 1–17.
- Fried, E. I., van Borkulo, C. D., Cramer, A. O. J., Boschloo, L., Schoevers, R. A., & Borsboom, D. (2017). Mental disorders as networks of problems: A review of recent insights. *Social Psychiatry and Psychiatric Epidemiology*, 52(1), 1–10.
- Friedman, J., Hastie, T., & Tibshirani, R. (2008). Sparse inverse covariance estimation with the graphical lasso. *Biostatistics*, 9(3), 432–441.
- Garfinkel, S. N., Seth, A. K., Barrett, A. B., Suzuki, K., & Critchley, H. D. (2015). Knowing your own heart: Distinguishing interoceptive accuracy from interoceptive awareness. *Biological Psychology*, 104, 65–74.
- Garfinkel, S. N., Tiley, C., O'Keeffe, S., Harrison, N. A., Seth, A. K., & Critchley, H. D. (2016). Discrepancies between dimensions of interoception in autism: Implications for emotion and anxiety. *Biological Psychology*, 114, 117–126.
- Hansen, H. A., Leber, A. B., & Saygin, Z. M. (2021). What sound sources trigger misophonia? Not just chewing and breathing. *Journal of Clinical Psychology*, 77(11), 2609–2625.
- Hibbard, P. B., & O'Hare, L. (2015). Uncomfortable images produce non-sparse responses in a model of primary visual cortex. *Royal Society Open Science*, 2(2), 140535.
- Huang, J., Cooper, T. G., Satana, B., Kaufman, D. I., & Cao, Y. (2003). Visual distortion provoked by a stimulus in migraine associated with hyperneuronal activity. *Headache: The Journal of Head and Face Pain*, 43(6), 664–671.
- Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., Sanislow, C., & Wang, P. (2010). *Research domain criteria (RDoC): Toward a new classification framework for research on mental disorders* (167, pp. 748–751). Am Psychiatric Assoc.
- Isvoranu, A.-M., Borsboom, D., van Os, J., & Guloksuz, S. (2016). A network approach to environmental impact in psychotic disorder: Brief theoretical framework. *Schizophrenia Bulletin*, 42(4), 870–873.
- Jager, I., de Koning, P., Bost, T., Denys, D., & Vulink, N. (2020). Misophonia: Phenomenology, comorbidity and demographics in a large sample. *PLoS One*, 15(4), e0231390.
- Jain, A. K. (2010). Data clustering: 50 years beyond K-means. *Pattern Recognition Letters*, 31, 651–666.
- Jakubovski, E., Müller, A., Kley, H., de Zwaan, M., & Müller-Vahl, K. (2022). Prevalence and clinical correlates of misophonia symptoms in the general population of Germany. *Frontiers in Psychiatry*, 13, 1012424. <https://doi.org/10.3389/fpsy.2022.1012424>
- Jastreboff, M. M., & Jastreboff, P. J. (2001). Components of decreased sound tolerance: Hyperacusis, misophonia, phonophobia. *ITHS News Lett*, 2(5–7), 1–5.
- Jastreboff, M. M., & Jastreboff, P. J. (2002). Decreased sound tolerance and tinnitus retraining therapy (TRT). *Australian and New Zealand Journal of Audiology*, 24(2), 74–84.
- Jones, P. J., Ma, R., & McNally, R. J. (2021). Bridge centrality: A network approach to understanding comorbidity. *Multivariate Behavioral Research*, 56(2), 353–367.
- Kaiser, E. A., Igdalova, A., Aguirre, G. K., & Cucchiara, B. (2019). A web-based, branching logic questionnaire for the automated classification of migraine. *Cephalalgia*, 39(10), 1257–1266.
- Kamada, T., & Kawai, S. (1989). An algorithm for drawing general undirected graphs. *Information Processing Letters*, 31(1), 7–15.
- Kumar, S., Tansley-Hancock, O., Sedley, W., Winston, J. S., Callaghan, M. F., Allen, M., Cope, T. E., Gander, P. E., Bamiou, D.-E., & Griffiths, T. D. (2017). The brain basis for misophonia. *Current Biology*, 27(4), 527–533.
- Li, Q., Joo, S. J., Yeatman, J. D., & Reinecke, K. (2020). Controlling for Participants' viewing distance in large-scale, psychophysical online experiments using a virtual Chinrest. *Scientific Reports*, 10, 904. <https://doi.org/10.1038/s41598-019-57204-1>
- Marcus, D. A., & Soso, M. J. (1989). Migraine and stripe-induced visual discomfort. *Archives of Neurology*, 46(10), 1129–1132.
- McGeoch, P. D., & Rouw, R. (2020). How everyday sounds can trigger strong emotions: ASMR, misophonia and the feeling of wellbeing. *BioEssays*, 42(12), 2000099.
- Mehling, W. E., Price, C., Daubenmier, J. J., Acree, M., Bartmess, E., & Stewart, A. (2012). The multidimensional assessment of interoceptive awareness (MAIA). *PLoS One*, 7(11), e48230.
- Miller, L. C., Murphy, R., & Buss, A. H. (1981). Consciousness of body: Private and public. *Journal of Personality and Social Psychology*, 41(2), 397–406.
- Neal, Z. P., Forbes, M. K., Neal, J. W., Brusco, M. J., Krueger, R., Markon, K., Steinley, D., Wasserman, S., & Wright, A. G. C. (2022). Critiques of network analysis of multivariate data in psychological science. *Nature Reviews Methods Primers*, 2, 90.
- Norris, J. E., Kimball, S. H., Nemri, D. C., & Ethridge, L. E. (2022). Towards a multidimensional understanding of misophonia using cluster-based phenotyping. *Frontiers in Neuroscience*, 16, 8325616.

- Pfennig, A., Ritter, P. S., Höfler, M., Lieb, R., Bauer, M., Wittchen, H. U., & Beesdo-Baum, K. (2016). Symptom characteristics of depressive episodes prior to the onset of mania or hypomania. *Acta Psychiatrica Scandinavica*, 133(3), 196–204.
- Rinaldi, L., Simner, J., Koursarou, S., & Ward, J. (2022). Autistic traits, emotion regulation, and sensory sensitivities in children and adults with misophonia. *Journal of Autism and Developmental Disorders*, 53, 1162–1174.
- Rinaldi, L. J., Smees, R., Ward, J., & Simner, J. (2022). Poorer Well-Being in children with misophonia: evidence from the sussex misophonia scale for adolescents. *Frontiers in Psychology*, 13, 808379.
- Rinaldi, L. J., Ward, J., & Simner, J. (2021). A Factor Structure within Misophonia: The Sussex Misophonia Scale for researchers and clinicians. <https://psyarxiv.com/5eb39>
- Ritter, P. S., Höfler, M., Wittchen, H.-U., Lieb, R., Bauer, M., Pfennig, A., & Beesdo-Baum, K. (2015). Disturbed sleep as risk factor for the subsequent onset of bipolar disorder—data from a 10-year prospective-longitudinal study among adolescents and young adults. *Journal of Psychiatric Research*, 68, 76–82.
- Robertson, A. E., & Simmons, D. R. (2013). The relationship between sensory sensitivity and autistic traits in the general population. *Journal of Autism and Developmental Disorders*, 43(4), 775–784.
- Rouw, R., & Erfanian, M. (2018). A large-scale study of misophonia. *Journal of Clinical Psychology*, 74(3), 453–479. <https://doi.org/10.1002/jclp.22500>
- Schröder, A., Vulink, N., & Denys, D. (2013). Misophonia: Diagnostic criteria for a new psychiatric disorder. *PLoS One*, 8(1), e54706.
- Schröder, A., van Wingen, G., Eijker, N., San Giorgi, R., Vulink, N. C., Turbyne, C., & Denys, D. (2019). Misophonia is associated with altered brain activity in the auditory cortex and salience network. *Scientific Reports*, 9(1), 7542.
- Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-Trait Anxiety Inventory*. Consulting Psychologists Press.
- Swedo, S. E., Baguley, D. M., Denys, D., Dixon, L. J., Erfanian, M., Fioretti, A., Jastreboff, P. J., Kumar, S., Rosenthal, M. Z., Rouw, R., Schiller, D., Simner, J., Storch, E. A., Taylor, S., Werff, K. R. V., Altimus, C. M., & Raver, S. M. (2022). Consensus definition of misophonia: A delphi study. *Frontiers in Neuroscience*, 16, 841816. <https://doi.org/10.3389/fnins.2022.841816>
- Taylor, S., Zvolensky, M. J., Cox, B. J., Deacon, B., Heimberg, R. G., Ledley, D. R., Abramowitz, J. S., Holaway, R. M., Sandin, B., Stewart, S. H., Coles, M., Eng, W., Daly, E. S., Arrindell, W. A., Bouvard, M., & Cardenas, S. J. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the anxiety sensitivity Index-3. *Psychological Assessment*, 19(2), 176–188.
- Ward, J., Hoadley, C., Hughes, J. E. A., Smith, P., Allison, C., Baron-Cohen, S., & Simner, J. (2017). Atypical sensory sensitivity as a shared feature between synaesthesia and autism. *Scientific Reports*, 7(1), 41155.
- Ward, Jr., J. H. (1963). Hierarchical grouping to optimize an objective function. *Journal of the American Statistical Association*, 58(301), 236–244.
- Wilkins, A., Nimmo-Smith, I., Tait, A., Mcmanus, C., Sala, S. D., Tilley, A., Arnold, K., Barrie, M., & Scott, S. (1984). A neurological basis for visual discomfort. *Brain*, 107(4), 989–1017.
- Wu, M. S., Lewin, A. B., Murphy, T. K., & Storch, E. A. (2014). Misophonia: Incidence, phenomenology, and clinical correlates in an undergraduate student sample. *Journal of Clinical Psychology*, 70(10), 994–1007. <https://doi.org/10.1002/jclp.22098>
- Zhang, T., Ramakrishnan, R., & Livny, M. (1996). BIRCH: An efficient data clustering method for very large databases. *ACM sigmod record*, 25(2), 103–114.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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