



UvA-DARE (Digital Academic Repository)

How everyday sounds can trigger strong emotions: ASMR, misophonia and the feeling of wellbeing

McGeoch, P.D.; Rouw, R.

DOI

[10.1002/bies.202000099](https://doi.org/10.1002/bies.202000099)

Publication date

2020

Document Version

Final published version

Published in

BioEssays

License

Article 25fa Dutch Copyright Act

[Link to publication](#)

Citation for published version (APA):

McGeoch, P. D., & Rouw, R. (2020). How everyday sounds can trigger strong emotions: ASMR, misophonia and the feeling of wellbeing. *BioEssays*, 42(12), [2000099]. <https://doi.org/10.1002/bies.202000099>

General rights

It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

Disclaimer/Complaints regulations

If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: <https://uba.uva.nl/en/contact>, or a letter to: Library of the University of Amsterdam, Secretariat, Singel 425, 1012 WP Amsterdam, The Netherlands. You will be contacted as soon as possible.

UvA-DARE is a service provided by the library of the University of Amsterdam (<https://dare.uva.nl>)

PROBLEMS & PARADIGMS

Prospects & Overviews

How everyday sounds can trigger strong emotions: ASMR, misophonia and the feeling of wellbeing

Paul D. McGeoch^{1,2}  | Romke Rouw³¹ Center for Brain and Cognition, University of California, San Diego, California, USA² School of Medicine, Dentistry and Biomedical Sciences, Queen's University, Belfast, United Kingdom³ Brain and Cognition, Dept of Psychology, University of Amsterdam, Amsterdam, Netherlands

Correspondence

Paul D. McGeoch, Center for Brain and Cognition, University of California, San Diego, California, USA.
Email: pdmcgeoch@ucsd.edu

Abstract

We propose that synesthetic cross-activation between the primary auditory cortex and the anatomically adjacent insula may help explain two puzzling conditions—autonomous sensory meridian response (ASMR) and misophonia—in which quotidian sounds involuntarily trigger strong emotional responses. In ASMR the sounds engender relaxation, while in misophonia they trigger an aversive response. The insula both plays an important role in autonomic nervous system control and integrates multiple interoceptive maps representing the physiological state of the body to substantiate a dynamic representation of emotional wellbeing. We propose that in ASMR cross-activation of the map for affective (sensual) touch leads to an increase in subjective wellbeing and parasympathetic activity. Conversely, in misophonia the effect of the cross-activation is to decrease emotional wellbeing and increase sympathetic activity. Our hypothesis also illuminates the connection between hearing and wellbeing more broadly and helps explain why so many people experience decreased wellbeing from modern urban soundscapes.

KEYWORDS

ASMR, emotion, insula, misophonia, sound, synesthesia, wellbeing

INTRODUCTION

Recent reports have highlighted two anomalous conditions—autonomous sensory meridian response (ASMR) and misophonia—in which certain everyday sounds involuntarily generate strong emotional reactions.^[1–7] Notably, these reactions are not readily explained by any physical properties of the sounds themselves. We propose that in individuals with these conditions there is atypical “synesthetic” cross-activation of the insula by the auditory cortex.

In synesthesia, individuals experience “crossed” sensations, in which an inducer sensation (e.g., seeing a letter “R”) automatically and consistently evokes another, seemingly unrelated, concurrent sensation (e.g., a light-blue color).^[8,9] Synesthesia is different from “normal” cross-modal associations because these are typically based on factual relationships and are usually experienced by everyone in a similar way.^[10] Thus, most people feel fear on hearing a dog bark as a barking dog could indeed be dangerous.

Conversely, synesthetic experiences are specific and subjective, and each individual synesthete has his own inducer-to-concurrent set.^[11] In comparison to controls, synesthesia has been found to be correlated with functional and structural brain differences, including increased connectivity between inducer and concurrent brain areas.^[9] Synesthesia, thus, helps explain how a particular stimulus might evoke a response that is consistent and automatic, yet is not obviously explained by the physical properties of the stimulus itself.

We also argue that an understanding of ASMR and misophonia affords the opportunity to better understand the neurological basis of the underappreciated, but close, relationship between hearing, autonomic nervous system (ANS) control and the feeling of wellbeing. That this relationship exists should not actually be surprising since the primary auditory cortex (A1) lies in the transverse temporal gyri of Heschl and as shown in Figure 1 these gyri are anatomically juxtaposed to the insula, which is a key brain area in homeostasis—that is, the maintenance of a stable internal physiological milieu. As such, the insula is

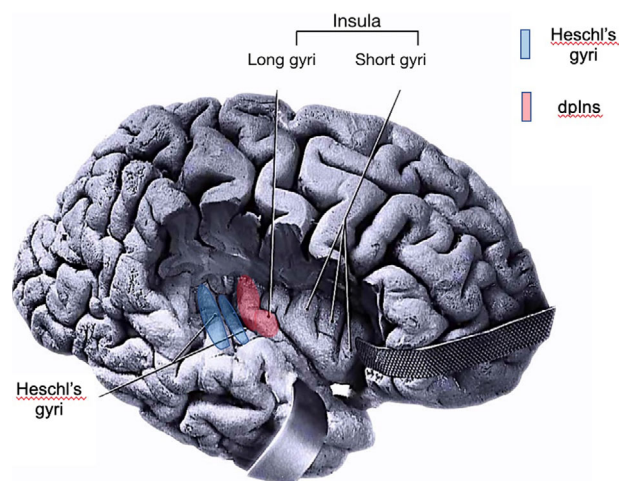


FIGURE 1 Lateral view of the right cerebral hemisphere. The frontal and parietal opercula are removed, and the temporal operculum is retracted to expose the insula and adjacent auditory cortex on the transverse temporal (Heschl's) gyri

involved in interoception (i.e., monitoring the physiological condition of the body), autonomic control and the dynamic representation of emotional states.^[12]

THE FEELING OF WELLBEING

The role of autonomic balance in wellbeing

The concept of wellbeing has been linked by psychologists to a wide gamut of different phenomena, such as “positive emotion, engagement, relationships, meaning, and accomplishment.”^[13] However, on a physiological level, there is emerging evidence that balance in the activity of the two sides of the ANS—sympathovagal balance—plays a key role emotional wellbeing.^[12,14] “Vagal” here refers to the vagus nerve, which is the tenth cranial nerve and a major component of the parasympathetic nervous system. Specifically, it is proposed that states of chronic sympathetic hyperactivity and parasympathetic hypoactivity are associated with reduced emotional wellbeing and vice versa.

The default neurological response to novel situations is sympathetic activation, which has obvious homeostatic advantages, as this side of the ANS mobilizes energy to survive threats and challenges.^[12,14–16] Conversely, during sympathetic activation the parasympathetic rest and digest functions are suppressed.^[12,14–16] Sympathetic activation, thus, utilizes energy, while parasympathetic activation conserves it.^[12,14–16] However, states of chronic sympathetic hyperactivity, with corresponding parasympathetic hypoactivity, can develop and lead to unsustainable energy demands.^[14]

Indeed, such states, if persistent, are correlated with an increased risk of cardiovascular disease, anxiety, post-traumatic stress disorder, diabetes mellitus, osteoporosis, Alzheimer's dementia, various cancers and generally increased frailty, disability, premature aging and death.^[14,17] Thus, a mechanism that evolved to maintain homeostasis

can end up jeopardizing it. Conversely, a direct link between increased vagal activity and improvements in affect can be found in the clinical literature, as vagus nerve stimulation improves mood in some patients with treatment resistant depression.^[18]

Although a detailed consideration is beyond the scope of this paper, there are techniques that can be used to measure sympathovagal balance. These include analyzing baroreflex sensitivity and heart rate variability (HRV).^[12,14,19,20,21] Baroreflex sensitivity involves measuring the blood pressure and assessing its relationship to heart rate (HR).^[21] HRV is easier to assess and refers to variations in the beat-to-beat interval of the heart.^[20] Increased sympathetic activity reduces the variability of this interval, while parasympathetic activity increases it, and a spectral analysis of HRV allows ANS balance to be assessed.^[20] Importantly, assessments of HRV that indicate increased parasympathetic activity have been correlated with improvements in subjective ratings of wellbeing.^[22,23] ANS balance has also been implicated in alleviating stress and pain, supporting healthy behavior, decreasing the chance of physical and psychological disease, and positively affecting social relationships.^[12,14]

The “global emotional moment” emerges in the anterior insula

The question, thus, arises as to why there is this apparent link between emotional wellbeing and sympathovagal balance. The valence model of emotion, in which negative emotions are lateralized to the right hemisphere and positive ones to the left, offers a potential explanatory route.^[24] This is because forebrain control of the ANS also appears to be lateralized.^[16,25,26] Specifically, there is evidence, for instance from stimulation during awake brain surgery, implicating the left insula in parasympathetic and the right in sympathetic control.^[27] Moreover, this split in autonomic function seems to occur across all vertebrates, which implies that it arose during the Cambrian explosion over 500 million years ago.^[28]

When considered from an evolutionary perspective, the advantage of emotions is to generate behavior that maintains homeostasis. It seems plausible that an ancient lateralization in forebrain control of the ANS, with its central role in homeostasis, would be co-opted and expanded by natural selection to also represent concordant emotional valencies.^[16,26] Indeed, Craig has argued that to optimize energy efficiency, emotions evolved “based on the coordinated opponency of the autonomic system—that activity in the right side of the forebrain is associated with energy expenditure, sympathetic activity, arousal, withdrawal (aversive) behaviour and individual-oriented (survival) emotions, and activity in the left side is associated with energy nourishment, parasympathetic activity, relaxation, approach (appetitive) behaviour and group-oriented (affiliative) emotions.”^[15] Supporting the view that the left hemisphere is associated with positive, affiliative type emotions, and the right with negative, challenging emotions is evidence, from a variety of sources including stimulation during awake surgery, and functional imaging studies, including two meta-analyses.^[16,25,29,30]

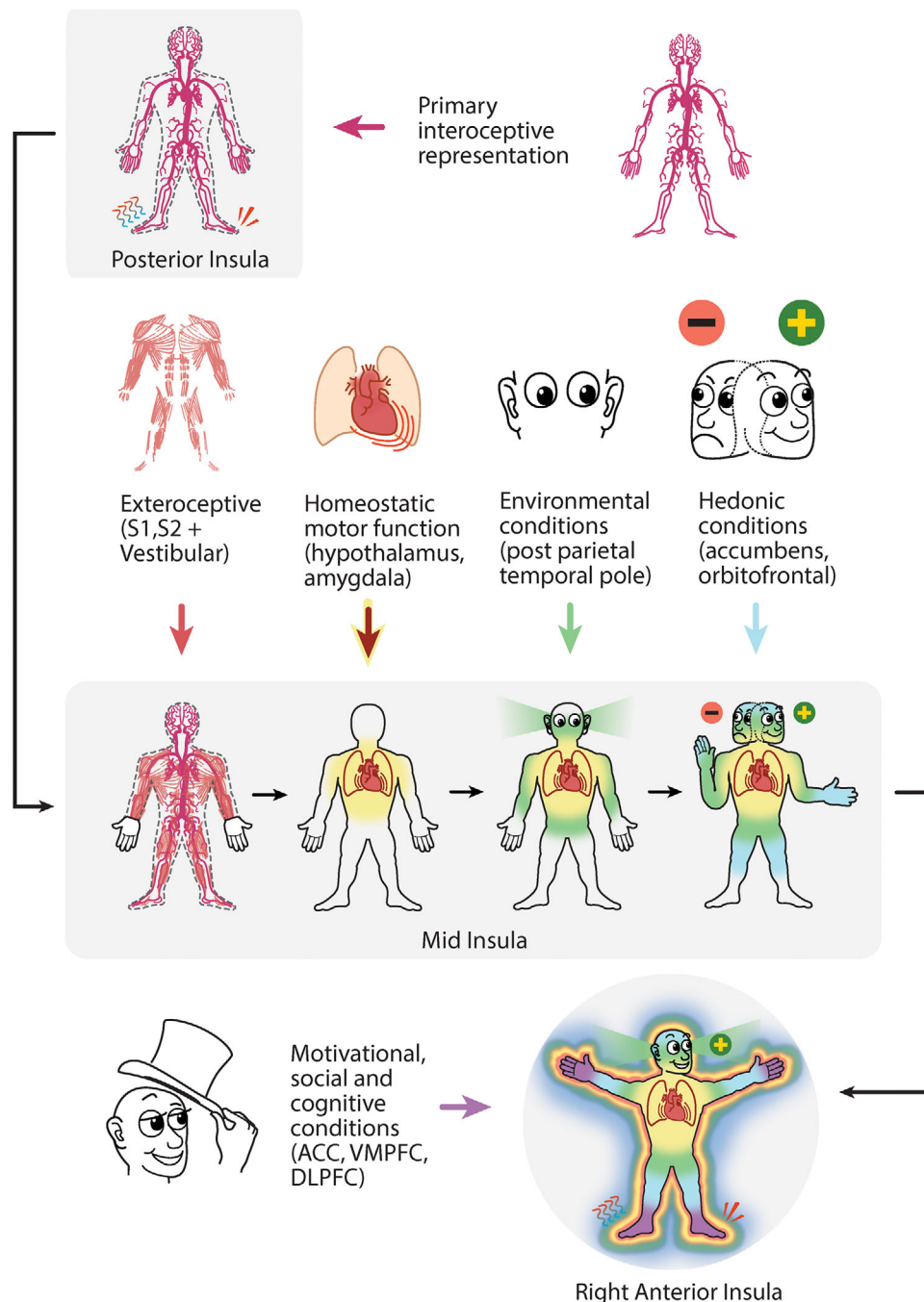


FIGURE 2 The posterior-to-mid-to-anterior integration in the homeostatic model. The integration of salience in the middle insula is built upon the interoceptive representation in the posterior insula, as detailed in the text. It culminates in the anterior insula in the complete representation of all ongoing feelings, indicated here by a glowing person. Craig calls this construct the global emotional moment, and it represents the sentient self. It is continuously changing. Used with permission from Barrow Neurological Institute^[16]

The dorsal posterior insula (dPIs) contains multiple maps constituting a constantly updated representation of the body's physiological state.^[16,25] According to a model proposed by Craig, these interoceptive maps in the dPIs are re-represented, and sequentially integrated with inputs from other brain areas, in the mid-insula and ultimately in the anterior insular cortices (AIC) to construct a dynamic representation of the current state of emotional wellbeing.^[16,25] This is termed the "global emotional moment" by Craig.^[16,25] This

is illustrated in Figure 2. Recent studies provide support for this model by showing both the extensive number of brain areas that project to the insula, and the posterior-to-anterior transition of insular functions.^[31–33]

In this model the global emotional moment is constructed from the underlying physiological condition of the body and lateralized according to its valency. Indeed, pleasant music, happy voices, maternal affection and seeing a smile all do lateralize to the left AIC.^[16,25] While

conversely, various types of pain, self-recognition and subjective cooling activate the right AIC.^[16,25] There is also evidence of opponent inhibition between the two AICs, suggesting how positive emotions (represented in the left AIC) can counteract negative ones (represented in the right AIC) and vice versa.^[16,25,34] This could explain why increasing parasympathetic activity (i.e., left insular activity) can reduce negative emotions and enhance emotional wellbeing. It should also be noted though that in many conditions (e.g., time perception and decision making) both AICs are jointly active.^[16,25]

Nonetheless, it would be inaccurate to portray the valence model of emotion as universally accepted. We consider the evidence supporting it to be robust and, we believe, of particular relevance to better understanding ASMR and misophonia, which is why we have expanded upon it here. However, there are several competing models of emotional processing.^[24] Also, whilst we judged it most relevant to focus our above discussion on the insula, other structures, such as the amygdala and anterior cingulate, are also implicated in the valence model.^[12,14,16]

Summary of the link between the ANS and wellbeing

The cortical map for hearing is anatomically adjacent to the insula, which is a homeostatic brain area involved in ANS control and the integration of physiological inputs to create the “global emotional moment.”^[12,16,25] The balance between the two sides of the ANS—sympathovagal balance—is a physiological marker of emotional wellbeing,^[12] with chronic ANS imbalance (specifically sympathetic hyperactivity and parasympathetic hypoactivity) implicated in reduced wellbeing and a variety of mental and physical ailments.^[12,14] Conversely, ANS balance is implicated in increased emotional wellbeing and the degree of balance can be measured using techniques such as HRV.

ASMR AND MISOPHONIA

What is known about ASMR?

In ASMR certain “trigger” stimuli evoke feelings of calm and relaxation, together with a pleasant tingling sensation that typically starts in the scalp, head and shoulders and spreads down the spine and into the limbs.^[1–5] Although there are many ASMR-inducing videos on the internet (some with millions of views), there is limited scientific understanding of the phenomenon.^[1–5] Two surveys found that among the most common triggers were whispering, “crisp sounds”, people speaking softly, and soft touching of hair or face.^[1,4] The cardinal feature of ASMR videos is an auditory stimulus, often recorded in stereo. Most people with ASMR date first experiencing it to childhood and claim it can relieve pain, depression, anxiety, stress and insomnia.^[1,5]

ASMR triggers produce both a fall in HR and a rise in skin conductance response (SCR).^[4] While a fall in HR suggests a parasympathetic swing, a rise in SCR is associated with sympathetic activation, which is

a contradiction that is discussed further in Section 4.2. At the time of writing, there have been four functional magnetic resonance imaging (fMRI) studies on ASMR. Two of these were resting state studies looking at functional connectivity. In the first, Smith et al. found a reduction in the connectivity of the default mode network (DMN).^[3] The DMN is implicated in internally directed thoughts and mind-wandering. They also found increased connectivity between frontal, occipital and temporal cortices, which they suggested reflects “a blending of multiple resting-state networks.”^[3] (Intriguingly, this overall fMRI pattern is similar to that observed during a psychedelic experience).^[35,36] In a second study, this same group subsequently examined several different brain networks and, as well as confirming their earlier observation, found that “ASMR [is] associated with reduced functional connectivity in the salience and visual networks.”^[37] The salience network includes the AIC, anterior cingulate and inferior frontal gyrus.^[37]

Smith et al. also carried out an fMRI study while playing ASMR tingle triggering videos to susceptible volunteers and controls. They report increased activation of the right cingulate gyrus, right paracentral lobule and both thalami in the ASMR group.^[38] Conversely, Lochte et al. found that the experience of relaxation in ASMR was correlated with bilateral medial prefrontal activation, while the tingling also bilaterally activated the insulae, nuclei accumbens and supplementary motor areas, and they linked ASMR to networks involved in reward, arousal and empathy.^[39]

What is known about misophonia?

In misophonia a seemingly innocuous sound elicits a strongly negative emotion, such as anger, anxiety, discomfort, or disgust,^[6,7,40–48] with an accompanying sympathetic (i.e., fight or flight) response.^[42,44,46] Typical triggers are manmade sounds, such as another person eating, breathing, and throat, nose or hand sounds. Other triggers include pen clicking, repetitive tapping and low-frequency sounds.^[40,41,44,45] Self-reported measurements in a large student population showed a prevalence of nearly 20%, with “clinically significant” impairment in 6%.^[41,42] Severe misophonia induces distress and may even cause suicidal ideation.^[49] Misophonic triggers increase both HR and SCR, indicating a sympathetic shift.^[45,46]

Kumar et al. showed increased activity in both AICs to misophonic trigger sounds.^[46] This increase was significantly greater than either misophonic subjects or controls produced to generally unpleasant sounds, and was correlated with the subjects’ degree of distress.^[46] They found increased myelination in the ventromedial prefrontal cortices of misophonic subjects, including the anterior cingulate cortex (ACC), and corresponding increased functional connectivity to both AICs. The authors moreover noted greater interoceptive awareness in misophonia, which, as mentioned, localizes to the insula.^[46] In another study Schröder et al. found activation in the right insula, right ACC and right temporal superior temporal cortex in response to misophonic triggers.^[50]

While different proposals have been advanced to explain misophonia, there is currently no generally agreed upon theory. Some authors

have implicated attention and learning in its etiology. In short, that misophonia is a conditioned physical and emotional response that develops through associative learning.^[7,40,47] Conversely, given the bilateral AIC activation they observed in response to misophonic triggers, Kumar and colleagues proposed that misophonia is related to the salience network and that the Bayesian inference model of interoception may play a role in attributing salience to certain sounds.^[35] The Bayesian inference model is a method of statistically modeling how the brain works, in which (top-down) probabilistic predictions are constrained by (bottom-up) sensory inputs.

Summary of studies on ASMR and misophonia

In summary, ASMR triggers produce positive emotions associated with an increase in wellbeing, and misophonic triggers do the opposite. In ASMR there is physiological evidence of both sympathetic and parasympathetic activity, whereas in misophonia the pattern is more clearly that of a sympathetic shift. In terms of brain imaging, studies have implicated atypical connectivity in brain networks involving higher-order processes, including emotion, externally versus internally directed attention and the salience of stimuli. However, these findings do not explain a cardinal characteristic of ASMR and misophonia; why the triggers are mainly sounds. Although the insula has been implicated directly in both phenomena, so has the functional connectivity between the AIC and the ACC.^[39,46,50] In ASMR this connectivity appears to be reduced,^[37] while in misophonia it is increased.^[46]

A NEW PARADIGM

Synesthetic Cross-Activation between auditory cortex and insula

The insula and cingulate cortex seem both to be involved in ASMR and misophonia. As discussed, the insula is a key homeostatic site and so is the ACC, which plays an important role in motivating behavior. Indeed, many of the same inputs regarding the physiological state of the body that project to the dplns also project to the ACC.^[12,16,25] If the insula is viewed as homeostatic sensory cortex then the ACC can be viewed as homeostatic motor cortex.^[12,16,25]

A connection between ASMR and misophonia has been previously been noted,^[51] with Barratt and Davis questioning if they were “two ends of the same spectrum of synaesthesia-like emotional responses.”^[1] We concur with this view and hypothesize here that ASMR and misophonia involve a synesthetic crossing-over of activation from A1 in Heschl's gyri into the neighboring insula. Note, although not previously defined in these terms, this proposal does, as it relates to misophonia, seem a logical extension of the work by Kumar's group.^[46,52] However, ASMR in particular has not been considered in this way before.

We will first consider in turn how this specific proposal applies to each of ASMR and misophonia before discussing its broader implications for hearing and wellbeing. One of the interoceptive maps in dplns is for affective touch and receives input from a distinct class of unmyelinated tactile afferents, which are activated by slow stroking, brushing or caressing as might occur during close contact and emotional bonding between lovers or close relatives.^[16,25,53,54] The resulting pleasant, tingling sensation has obvious parallels with ASMR. We propose that ASMR occurs due to cross-activation between A1 and this map for affective touch in the dplns. This auditory input into the interoceptive representation is then re-represented into the global emotional moment in the AIC to generate the sense of wellbeing and euphoria described in ASMR.^[1,25] This is in contrast to previous proposals that ASMR is a form of auditory-somatosensory synesthesia (meaning that A1 activates the primary somatosensory cortex, which anatomically is more distant, in the postcentral gyrus of the parietal lobe).^[1,3] This would not explain the emotional wellbeing so characteristic of ASMR.

As regards misophonia, the insula integrates multiple homeostatic inputs to produce emotions with both positive and negative valencies.^[16,25] Extrapolating from Kumar's findings,^[46] we contend that in misophonia the auditory stimulus could spread via synesthetic cross-activation from A1 into the insula, but this time to elicit a visceral, negative effect upon the global emotional moment in the AIC.^[16,25] However, which specific map in the insula might be cross-activated by misophonic triggers remains unclear.

Implications for the link between hearing and wellbeing

Can an auditory-insular synesthesia hypothesis for ASMR and misophonia be used to better understand the connection between hearing and wellbeing more broadly? The hypothesis suggests that, when considering wellbeing, it is not only a sound's objective, physical properties that are important but also its subjective impact upon the global emotional moment. As discussed in Section 2.1, sympathovagal balance is a useful physiological metric of wellbeing and a parasympathetic shift in sympathovagal balance decreases negative emotional states, such as anger, anxiety and depression, and increases positive ones, such as self-esteem—i.e., it increases wellbeing.^[12,16,25] Indeed, practices associated with increased wellbeing, such as yoga, binaural beats, breathing techniques, biofeedback and meditation all cause a parasympathetic shift.^[55–60] While previous studies have examined physiological parameters in ASMR and misophonia (see Section 3),^[4,45,46] there are no studies specifically examining sympathovagal balance.

It is known that ASMR triggers cause HR to fall and SCR to rise,^[4] suggesting both sympathetic and parasympathetic involvement. Indeed, as noted earlier in Section 2.2, there are conditions in which both AICs are jointly active.^[16,25] This is a problem that is eminently open to empirical assessment, for instance using HRV. We suspect that the overall shift in ASMR will prove to be towards the parasympathetic nervous system, since although both sides of the ANS innervate the

heart, the eccrine sweat glands that determine SCR only receive sympathetic input.^[61] The decrease in HR, thus, suggests that the overall effect is an increase in vagal tone. Moreover, ASMR is strongly associated with flow state experiences and such states have, outside the context of ASMR, been correlated with increased parasympathetic activity.^[1,55]

EVALUATING THE MODEL

Neurological and behavioral evidence

The model is parsimonious, as a single mechanism—activity in A1 synesthetically influencing the insula's evaluation of the global emotional moment and thus, state of wellbeing—is proposed to underlie both ASMR and misophonia. There are several distinct lines of evidence to support this view. First, fMRI evidence has suggested a critical role for the insula in misophonia and possibly also ASMR.^[39,46,50] In misophonia, the right preponderant activation described by Schröder et al. is compatible with the reported negative, challenging emotions.^[50] Conversely, the report by Kumar et al. of bilateral AIC activity may additionally reflect misophonic disgust,^[46] which, unlike most negative emotions, is left lateralized (perhaps because of parasympathetic control of vomiting).^[16]

Second, the gyri where A1 lies run transversely towards the dplns, (Figure 1) and synesthetic cross-activations are more common between anatomically adjacent cortical areas.^[8] In fact, recent tractography studies found structural connections between posterior insular seed regions and ipsilateral Heschl's gyri.^[31,32] Such a pattern of connectivity helps explain why some individuals might develop unusual trigger sounds (see Section 5.3). Tractography has also suggested direct projections from the insula to the cingulate cortex, which, as discussed in Sections 3 and 4.1, is an area implicated by imaging studies of ASMR and misophonia.^[32,38,46] Third, the posterior-to-anterior axis in insular function has been shown specifically for auditory stimuli. The posterior insula represents A1 activity and the anterior insula the emotional valency of the sound.^[62]

Fourth, there is evidence that ASMR, misophonia, and synesthesia co-occur more often than would be expected by chance alone.^[49,51] Fifth, subjects with misophonia have shown increased interoception and the insula is implicated in this.^[46,48] Interoceptive awareness is yet to be formally tested in ASMR. Sixth, there is a remarkable similarity between ASMR and what is felt during physical grooming and affiliative behaviors, which suggests involvement of the map for affective touch in the dplns.^[40]

Relationship to existing hypotheses

This specific hypothesis, of local A1-to-insular synesthetic cross-activation, has not been put forward before in either the ASMR or misophonia literature. Existing physiological explanations for ASMR are lacking. However, as discussed in Section 3.2, there are explanatory

hypotheses for misophonia. We consider the two main ones here and how they might relate to our model.

First, the hypothesis that misophonia is a conditioned response that arises from associative learning.^[7,40,47] This hypothesis implicates higher order functions, such as learning, memory and attention, and brain networks, in particular the salience network. However, it does not explain how or why this conditioning might occur. As the A1-to-insular cross-activation model offers a low-level neurological mechanism it has the potential to better explain the root cause of misophonia. Learning, memory and salience networks, even if not in a primary causative role, could explain downstream strengthening of the response over time.

The second hypothesis for misophonia implicates the Bayesian inference model of interoception in abnormally evaluating interoceptive inputs to the insula. Kumar relates the bilateral AIC activation to the salience network abnormally processing auditory inputs. However, this explanation neither explains why misophonic triggers are auditory, rather than another sensory modality, nor why they are often specific, innocuous sounds. Bayesian models of the brain do not in themselves inform on the details of underlying neuroanatomy or physiology. Conversely, our model could extend these ideas, as A1-to-insular synesthesia would explain why such altered predictive processing mostly affect the perception of auditory stimuli in misophonia. The proposed hypothesis, thus, has the potential to complement and extend rather than contradict previously formulated explanations.

Indeed, both ASMR and misophonia seem complex and multifaceted conditions. The often highly specific nature of trigger sounds points at low-level, local mechanisms being involved. While, conversely, complex behavioral and emotional changes suggest that high-level, global networks also play a role.^[46] Indeed, although auditory triggers are characteristic, non-auditory triggers can occur in ASMR and sometimes misophonia too. Notably though, when these non-auditory triggers do occur, they often have similar properties to the auditory triggers. They might, thus, include close personal attention and repetitive, slow manmade movements (e.g., wiggling of a foot).^[1,49] The involvement of high-level, global networks may well play a role in explaining them. Thus, it seems plausible that neither exclusively local nor exclusively global processes explain ASMR and misophonia, but rather an interaction of both.

Explaining the individual nature of trigger sounds

In ASMR and misophonia, it is the interpretation of the sound by the individual that seems to drive the subjective emotional response, rather than any specific physical property of the stimulus itself. The auditory-insular synesthesia model provides a neuroanatomical basis for why, in certain individuals, particular sounds subjectively evoke distinct emotions. We suggest that at an individual level, environmental influences act in conjunction with a genetic or developmental susceptibility to shape particular triggers and predict that functional connectivity between Heschl's gyri and insular cortex plays a crucial role in this shaping.

Why might such cross-activation exist between hearing and interoception? Again there are insights from synesthesia, where there is evidence that a genetic factor, for example in cortical pruning or axonogenesis,^[63] causes the brains of individuals with synesthesia to be structurally different from controls.^[9] Synesthesia also suggests a possible role for learning in shaping the particular inducer-to-concurrent pairings in susceptible individuals. Hyperconnectivity between brain areas leads, over time, to an increase in the strength of these synesthetic concurrents.^[64,65]

Thus, it seems plausible that in ASMR and misophonia a genetic predisposition might interact with environmental factors to shape responses that become more consistent, automatic and stronger over time. Moreover, the experienced emotion, in predisposed individuals, might be part of an iterative process with the response strengthening every time the trigger occurs.^[40,47] This would especially be true of individuals with ASMR who deliberately, and repeatedly, trigger the phenomenon due to its positive effect on wellbeing. Similarly, subjects with misophonia report that repeated exposure to the same trigger sound intensifies their emotional response.^[49]

In short, although the sensitivity to particular sounds in ASMR and misophonia, and their ability to trigger specific emotions is seemingly irrational, the auditory-insular synesthesia model can account for it. A dynamic process of altered neurological activation in susceptible individuals could, through a positive feedback process, lead to particular auditory stimuli becoming triggers for strong emotional responses.

TESTING THE HYPOTHESIS

There are several empirical approaches that could be used to assess the role of A1-to-insular cross-activation in ASMR and misophonia and tease it apart from existing hypotheses. First, although a number of fMRI studies have been carried out on both conditions, there have been no studies using magnetoencephalography (MEG). Modern MEG techniques offer both good spatial and excellent temporal resolution,^[66] to the extent that it should have the potential to show activation spread from A1 to the adjacent dplns and then to the AIC.

Second, typical measurement settings and analyses in diffusion MRI studies are only reasonably accurate when imaging longer tracts—i.e., long association and commissural fibers. However, some studies have succeeded in finding connectivity differences in short association fibers (sometimes termed subcortical u-fibers) between adjacent gyri.^[67] Thus, a study specifically designed to adequately resolve white matter connectivity at such small spatial scale, particularly in the complex mid-temporal region could reveal connectivity differences between A1 and the insula.^[68]

Third, as has been done in misophonia, interoceptive awareness, which is localized to the insula, should be assessed in ASMR, for instance by use of the body consciousness questionnaire.^[46,48] We predict it would be heightened. Fourth, as discussed in Section 2.1, ANS balance can be measured using HRV and baroreflex sensitivity.^[12,14,19–21] We predict that these metrics would confirm a

parasympathetic shift in ASMR and a sympathetic shift in misophonia during exposure to trigger sounds.

FUTURE PERSPECTIVES

ASMR and misophonia are not the only phenomena in which hearing impacts wellbeing. In frisson and indeed music this can be positive, while conversely, in hyperacusis, tinnitus and auditory sensitivity it can be strongly negative (see Box 1).^[69–71] A disproportionate emotional response to everyday sounds is a feature of a variety of clinical, developmental and psychological disorders, including depression, posttraumatic stress disorder, autism and burnout.^[69–71] Anything that illuminates the neurological basis of how hearing modulates emotion could be relevant in better understanding all of these conditions. Nonetheless, from a public health perspective it is clear that the most pressing issue is that of urban noise pollution.

The World is urbanizing rapidly. In 1950 about 30% of Earth's population lived in urban areas but according to the United Nations by 2018 this percentage was 55% and rising fast [<https://population.un.org/wup/Publications/>]. Living in an urban environment has been associated with an increased risk of experiencing several types of psychiatric disease, including mood and anxiety disorders, psychosis, and schizophrenia.^[72] This urgently calls for a better understanding of the particular challenges to mental and physical health that urban lifestyles pose.

One clear characteristic of urban environments is the intensity of sensory information that city dwellers are exposed to on a daily basis. In particular, urban auditory overstimulation is a pressing and persistent concern. The World Health Organization's (WHO) Regional Office for Europe reported in 2011 that “environmental noise, also known as noise pollution, is among the most frequent sources of complaint regarding environmental issues” and went on to state that in “comparison to other pollutants, the control of environmental noise has been hampered by insufficient knowledge of its effects on humans and of exposure–response relationships” [= 1 (Accessed 4/30/20)]. Examples of urban auditory overstimulation include heavy traffic, noise from numerous neighbors in close proximity, and regular use of mass transit networks.^[73] In a large-scale study in Amsterdam, 41% of respondents indicated serious issues with noise pollution [(Accessed 4/30/20)].

The adverse physical and mental consequences of urban noise pollution include: hearing impairment, increased cardiovascular disease, stress, disturbed sleep and concentration, decreased school performance and a general decrease in quality of life.^[74] Research on so-called “soundscapes” has found that natural sounds are typically perceived as pleasant, but unnatural, technological noises as unpleasant.^[75] Stress recovery (as assessed with SCR) was faster for exposure to natural as compared to urban (traffic) sounds.^[76] Thus, it is not just the loudness or omnipresence but also the very nature of urban sounds that can be harmful to health. The same sound when presented with a natural image (e.g., a beach) produced a different pattern of A1 activity than when presented with an unnatural one (e.g., a freeway).^[77] We suggest that measuring sympathovagal balance could

BOX 1: Related Phenomena

There are a number of phenomena, from different fields, in which sounds inexplicably affect wellbeing, without a clear explanation based on the physical properties of the sounds. We argue that ASMR and misophonia are not mere anomalies, but reveal the neurological basis of the normal, close relationship between hearing, central control of the ANS and emotion.

- **Frisson.** A short-lived pleasurable response to a particular passage of music (typically one that violates musical expectation).^[78] Frisson is described as an involuntary, pleasant, tingling and shivering sensation down the back of the spine. As in ASMR, frisson provokes “chills” and piloerection, has measurable effects on ANS function, and increases wellbeing.^[5,77] ASMR and frisson activate similar brain areas.^[17]
- **Hyperacusis.** Hypersensitivity to usually innocuous sounds with an aversive emotional response.^[70] The sounds are described as painful, or too loud, causing discomfort and an involuntary decrease in wellbeing.
- **Tinnitus.** Continuous perception of noise (such as ringing, clicking or buzzing) without an external source. Tinnitus often originates from some type of damage to the auditory system (e.g., hearing loss). It can be extremely debilitating and is associated with depression and decreased wellbeing.^[69,70]
- **Auditory sensitivity in other disorders.** Hyperacusis and tinnitus may occur in isolation, but are common comorbidities in many other disorders, particularly depression, posttraumatic stress disorder, anxiety and other stress-related disorders.^[70–72] Moreover, there is a physiological mechanism self-strengthening the stress response to sounds: sound overload can evoke psychological and endocrine stress responses, which can in turn increase the risk of developing sound sensitivity, including tinnitus and hyperacusis.^[70–72]

be a valuable adjunct to public health reviews of urban soundscapes, as this would allow an assessment of the soundscape's impact on emotional wellbeing. HRV in particular, as it is easy to measure, seems a prime candidate for this purpose.

In summary, modern urban soundscapes seem to be associated with a range of negative sequelae but our understanding of exactly what is going on is limited. However, ASMR and misophonia act as paradigms for understanding the auditory-insular mechanisms connecting hearing and wellbeing, which is of direct relevance to the health of all city dwellers. Indeed, a better understanding of the link between hearing and wellbeing would have consequences for both individual health and society at large.

CONCLUSIONS AND PROSPECTS

Our hypothesis provides an explanation for the automatic, strong emotional responses to everyday sounds that occur in ASMR and misophonia. It proposes that they occur due to synesthetic cross-activation of the insula by activity in adjacent auditory cortex. This then modulates the global emotional moment in the AIC. In misophonia, wellbeing decreases and sympathetic activity increases. ASMR appears to be the converse; emotional wellbeing increases along with, we suspect, an overall increase in in parasympathetic tone. As described above, this hypothesis is derived from several converging lines of evidence and testable.

Although ASMR and misophonia might seem like curiosities, of limited interest, by placing their genesis in the insula, with its role in homeostasis, ANS control and representation of the global emotional moment, this hypothesis has the potential to help better understand a feature of all human brains—the link between hearing and emotional wellbeing. A better understanding of the neurological basis for this link has clear public health relevance. Indeed, the burden of mental and physical ill-health due to urban auditory environments is an increasingly urgent issue, which has been termed an “underestimated threat” by the WHO [<http://www.euro.who.int/en/health-topics/environment-and-health/noise/data-and-statistics> (Accessed 4/30/20)].

Our hypothesis suggests that when evaluating the impact of soundscapes on wellbeing, it is vital to assess their subjective impact on the global emotional moment, as measured by sympathovagal balance. While parasympathetic re-equilibration leads to improvement in the feeling of wellbeing and the homeostatic integrity of the entire organism, chronic sympathetic hyperactivity leads to a reduction in wellbeing and is associated with an increased risk of a host of diseases, as well as premature aging and death.^[12,14,17] Thus, in terms of human health, a better understanding of the physiological impact of how sounds are perceived could literally save lives.

ACKNOWLEDGMENTS

The authors thank Stuart Anstis, Richard Ridderinkhof, Karen Dobkins, Bill Rosar, Jason McKeown and V.S. Ramachandran for their comments and discussion.

CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

None

ORCID

Paul D. McGeoch  <https://orcid.org/0000-0002-8880-3654>

REFERENCES

1. Barratt, E. L., & Davis, N. J. (2015). Autonomous sensory meridian response (ASMR): A flow like mental state. *PeerJ*, 3, e851. <https://doi.org/10.7717/peerj.851>.

2. Barratt, E. L., Spence, C., & Davis, N. J. (2017). Sensory determinants of the autonomous sensory meridian response (ASMR): Understanding the triggers. *PeerJ*, 5, e3846. <https://doi.org/10.7717/peerj.3846>.
3. Smith, S. D., Fredborg, B. K., & Kornelsen, J. (2017). An examination of the default mode network in individuals with autonomous sensory meridian response (ASMR). *Soc. Neurosci.* 12, 361–365.
4. Poerio, G. L., Blakey, E., Hostler, T. J., & Veltri, T. (2018). More than a feeling: Autonomous sensory meridian response (ASMR) is characterized by reliable changes in affect and physiology. *Plos One*, 13(6), e0196645. <https://doi.org/10.1371/journal.pone.0196645>.
5. Kovacevich, A., & Huron, D. (2019). Two studies of autonomous sensory meridian response (ASMR): The relationship between ASMR and music-induced frisson. *Emp. Music. Rev.*, 13, 39–63.
6. Cavanna, A. E., & Seri, S. (2015). Misophonia: Current perspectives. *Neuropsychiatr. Dis. Treat.* 11, 2117–2123.
7. Brout, J. J., Edelstein, M., Erfanian, M., Mannino, M., Miller, L. J., Rouw, R., ... Rosenthal, M. Z. (2018). Investigating misophonia: A review of the empirical literature, clinical implications, and a research agenda. *Front. Neurosci.* 12, 36. <https://doi.org/10.3389/fnins.2018.00036>
8. Ramachandran, V. S., & Hubbard, E. M. (2001). Psychophysical investigations into the neural basis of synaesthesia. *Proc Biol Sci.* 268, 979–983.
9. Rouw, R., & Scholte, H. S., (2007). Increased structural connectivity in grapheme-color synesthesia. *Nat. Neurosci.* 10, 792–797.
10. Deroy, O., & Spence, C. (2013). Why we are not all synesthetes (not even weakly so). *Psychon Bull Rev*, 20, 643–664.
11. Palmeri, T. J., Blake, R., Marois, R., & Flanery, M. A., Whetsell, W. (2002). The perceptual reality of synesthetic colors. *Proc. Natl. Acad. Sci. U. S. A.* 99, 4127–4131.
12. Strigo, I. A., & Craig, A. D. (2016). Interoception, homeostatic emotions and sympathovagal balance. *Philos Trans R Soc Lond B Biol Sci*, 371, 20160010.
13. Forgeard, M. J. C., Jayawickreme, E., Kern, M. L., & Seligman, M. E. P. (2011). Doing the right thing: Measuring wellbeing for public policy. *Int J Well-being*, 1, 79–106.
14. Thayer, J. F., & Brosschot, J. F. (2005). Psychosomatics and psychopathology: Looking up and down from the brain. *Psychoneuroendocrino*, 30, 1050–1058.
15. Amat, J., Baratta, M. V., Paul, E., Bland, S. T., Watkins, L. R., & Maier, S. F. (2005). Medial prefrontal cortex determines how stressor controllability affects behavior and dorsal raphe nucleus. *Nat. Rev. Neurosci.* 8, 365–371.
16. Craig, A. D. (2014). *How Do You Feel? An interoceptive moment with your neurobiological self*. Princeton: Princeton University Press.
17. Jung, W., Jang, K. I., & Lee, S. H. (2019). Heart and brain interaction of psychiatric illness: A review focused on heart rate variability, cognitive function, and quantitative electroencephalography. *Clin Psychopharmacol Neurosci*, 17, 459–474.
18. Conway, C. R., & Xiong, W. (2018). The mechanism of action of vagus nerve stimulation in treatment-resistant depression: Current conceptualizations. *Psychiatr Clin North Am*, 41, 395–407.
19. Goldberger, J. J. Sympathovagal balance: How should we measure it? *Am. J. Physiol.* 276, H1273–H1280.
20. Montano, N., Porta, A., Cogliati, C., Costantino, G., Tobaldini, E., Casali, K., & Iellamo, F. (2009). Heart rate variability explored in the frequency domain: A tool to investigate the link between heart and behavior. *Neurosci. Biobehav. Rev.* 33, 71–80.
21. Swenne, C. A. (2013). Baroreflex sensitivity: Mechanisms and measurement. *Neth Heart J*, 21, 58–60.
22. Geisler, F. C. M., Vennwald, N., Kubiak, T., & Weber, H. (2010). The impact of heart rate variability on subjective well-being is mediated by emotion regulation. *Pers. Individ. Differ.* 49, 723–728.
23. Trimmel, M. (2015). Relationship of heart rate variability (HRV) parameters including pNNxx with the subjective experience of stress, depression, well-being, and every-day trait moods (TRIM-T): A pilot study. *Ergon. Open J.*, 8, 32–37.
24. Demaree, H. A., Everhart, D. E., Youngstrom, E. A., & Harrison, D. W. (2005). Brain lateralization of emotional processing: Historical roots and a future incorporating dominance. *Behav Cogn Neurosci Rev*, 4, 3–20.
25. Craig, A. D. (2009). How do you feel – now? The anterior insula and human awareness. *Nat. Rev. Neurosci.* 10, 59–70.
26. Craig, A. D. (2005). Forebrain emotional asymmetry: A neuroanatomical basis? *Trends in Cognitive Sciences*, 9, 566–571.
27. Oppenheimer, S. M., Gelb, A., Girvin, J. P., & Hachinski, V. C. (1992). Cardiovascular effects of human insular cortex stimulation. *Neurology*, 42, 1727–1732.
28. MacNeilage, P. F., Rogers, L. J., & Vallortigara, G. (2009). Origins of the left and right brain. *Sci. Am.* 301, 60–67.
29. Stevens, J. S., & Hamann, S. (2012). Sex differences in brain activation to emotional stimuli: A meta-analysis of neuroimaging studies. *Neuropsychologia*, 50, 1578–1593.
30. Duerden, E. G., Arsalidou, M., Lee, M., & Taylor, M. J. (2013). Lateralization of affective processing in the insula. *Neuroimage*, 78, 159–175.
31. Cloutman, L. L., Binney, R. J., Drakesmith, M., Parker, G. J. M., & Lambon Ralph, M. A. (2012). The variation of function across the human insula mirrors its patterns of structural connectivity: Evidence from in vivo probabilistic tractography. *Neuroimage*, 59, 3514–3521.
32. Ghaziri, J., Tucholka, A., Girard, G., Houde, J.-C., Boucher, O., Gilbert, G., ... Nguyen, D. K. (2017). The corticocortical structural connectivity of the human insula. *Cereb. Cortex*, 27, 1216–1228.
33. Cerliani, L., Thomas, R. J., Jbabdi, S., Siero, J. C. W., Nanetti, L., Crippa, A., ... Keysers, C. (2012). Probabilistic tractography recovers a rostro-caudal trajectory of connectivity variability in the human insular cortex. *Hum. Brain Mapp*, 33, 2005–2034.
34. Zautra, A. J., Fasman, R., Davis, M. C., & Arthur, D. (2010). The effects of slow breathing on affective responses to pain stimuli: An experimental study. *Pain*, 149, 12–18.
35. Carhart-Harris, R. L., Leech, R., Hellyer, P. J., Shanahan, M., Feilding, A., Tagliazucchi, E., ... Nutt, D. (2014). The entropic brain: A theory of conscious states informed by neuroimaging research with psychedelic drugs. *Front. Hum. Neurosci.* 8, 20. .
36. Müller, F., Dolder, P. C., Schmidt, A., Liechti, M. E., & Borgwardt, S. (2018). Altered network hub connectivity after acute LSD administration. *NeuroImage Clin.* 18, 694–701.
37. Smith, S. D., Fredborg, B. K., & Kornelsen, J. (2019). Atypical functional connectivity associated with autonomous sensory meridian response: An examination of five resting-state networks. *Brain Connect*, 9, 508–518.
38. Smith, S. D., Fredborg, B. K., & Kornelsen, J. (2019). A functional magnetic resonance imaging investigation of the autonomous sensory meridian response. *PeerJ*, 7, e7122 <https://doi.org/10.7717/peerj.7122>.
39. Lochte, B. C., Guillory, S. A., Richard, C. A. H., & Kelley, W. M. (2018). An fMRI investigation of the neural correlates underlying the autonomous sensory meridian response (ASMR). *Bioimpacts*, 8, 295–304.
40. Jastreboff, M. M., & Jastreboff, P. J. (2002). Decreased sound tolerance and tinnitus retraining therapy (TRT). *Aust. N. Z. J. Audiol.*, 24, 74–84.
41. Wu, M. S., Lewin, A. B., Murphy, T. K., & Storch, E. A. (2014). Misophonia: Incidence, phenomenology, and clinical correlates in an undergraduate student sample. *J. Clin. Psychol.* 70, 994–1007.
42. Zhou, X., Wu, M. S., & Storch, E. A. (2017). Misophonia symptoms among Chinese university students: Incidence, associated impairment, and clinical correlates. *J. Obsess-Compuls. Rel.*, 14, 7–12.
43. Potgieter, I., MacDonald, C., Partridge, L., Cima, R., Sheldrake, J., & Hoare, D. J. (2019). Misophonia: A scoping review of research. *J. Clin. Psychol.* 75, 1203–1218.

44. Schröder, A., Vulink, N., & Denys, D. (2013). Misophonia: Diagnostic criteria for a new psychiatric disorder. *Plos One*, 8, e54706. <https://doi.org/10.1371/journal.pone.0054706>.
45. Edelstein, M., Brang, D., Rouw, R., & Ramachandran, V. S. (2013). Misophonia: Physiological investigations and case descriptions. *Front. Hum. Neurosci.* 7, 296. DOI: 10.3389/fnhum.2013.00296.
46. Kumar, S., Tansley-Hancock, O., Sedley, W., Gander, P. E., Bamiou, D. E., & Griffiths, T. D. (2017). The brain basis for misophonia. *Curr. Biol.* 27, 527–533.
47. Dozier, T. H., & Morrison, K. L. (2017). Phenomenology of misophonia: Initial physical and emotional responses. *Am. J. Psychol.* 130, 431–438.
48. McKay, D., Kim, S. K., Mancusi, L., Storch, E. A., & Spankovich, C. (2018). Profile analysis of psychological symptoms associated with misophonia: A community sample. *Behav. Ther.* 49, 286–294.
49. Rouw, R., & Erfanian, M. (2018). A large-scale study of misophonia. *J. Clin. Psychol.* 74, 453–479.
50. Schröder, A., van Wingen, G., Eijsker, 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. *Sci. Rep.* 9, 7542. <https://doi.org/10.1038/s41598-019-44084-8>.
51. McErlean, A. B. J., & Banissy, M. J. (2018). Increased misophonia in self-reported autonomous sensory meridian response. *PeerJ*, 6, e5351. <https://doi.org/10.7717/peerj.5351>
52. Palumbo, D. B., Alsalman, O., De Ridder, D., Song, J. J., & Vanneste, S. (2018). Misophonia and potential underlying mechanisms: A perspective. *Front. Psychol.* 9, 953.
53. Bjornsdotter, M., Löken, L., Olausson, H., Vallbo, A., & Wessberg, J. (2009). Somatotopic organization of gentle touch processing in the posterior insular cortex. *J. Neurosci.* 29, 9314–9320.
54. McGlone, F., Wessberg, J., & Olausson, H. (2014). Discriminative and affective touch: sensing and feeling. *Neuron*, 82, 737–755.
55. Peifer, C., Schulz, A., Schächinger, H., Baumann, N., & Antoni, C. H. (2014). The relation of flow-experience and physiological arousal under stress- can u shape it? *J. Exp. Soc. Psychol.* 53, 62–69.
56. Satyapriya, M., Nagendra, H. R., Nagarathna, R., & Padmalatha, V. (2009). Effect of integrated yoga on stress and heart rate variability in pregnant women. *Int. J. Gyn. Obst.*, 104, 218–222.
57. Gantt, M. A., Dadds, S., Burns, D. S., Glaser, D., & Moore, A. D. (2017). The effect of binaural beat technology on the cardiovascular stress response in military service members with post deployment stress. *J. Nurs. Scholarsh.* 49, 411–420.
58. Brown, R. P., & Gerberg, P. L. (2009). Yoga breathing, meditation and longevity. *Ann. N. Y. Acad. Sci.* 1172, 54–62.
59. Deschodt-Arsac, V., Lalanne, R., Spiluttini, B., Bertin, C., & Arsac, L. M. (2018). Effects of heart rate variability biofeedback training in athletes exposed to stress of university examinations. *Plos One*, 13, e0201388.
60. Krygier, J. R., Heathers, J. A., Shahrestani, S., Abbott, M., Gross, J. J., & Kemp, A. H. (2013). Mindfulness meditation, well-being, and heart rate variability: A preliminary investigation into the impact of Vipassana meditation. *Int. J. Psychophysiol.* 89, 305–313.
61. Critchley, H. D. (2002). Electrodermal responses: What happens in the brain. *Neuroscientist*, 8, 132–142.
62. Zhang, Y., Zhou, W., Wang, S., Zhou, Q., Wang, H., Zhang, B., ... Wang, X., (2019). The roles of subdivisions of human insula in emotion perception and auditory processing. *Cereb. Cortex*, 29, 517–528.
63. Tilot, A. K., Kucera, K. S., Vino, A., Asher, J. E., Baron-Cohen, S., & Fisher, S. E. (2018). Rare variants in axonogenesis genes connect three families with sound-color synesthesia. *Proc. Natl. Acad. Sci. U. S. A.* 115, 3168–3173.
64. Root, N. B., Rouw, R., Asano, M., Kim, C. Y., Melero, H., Yokosawa, K., & Ramachandran, V. S. (2018). Why is the synesthete's "A" red? Using a five-language dataset to disentangle the effects of shape, sound, semantics, and ordinality on inducer-concurrent relationships in grapheme-color synesthesia. *Cortex*, 99, 375–389.
65. Simner, J., & Bain, A. E. (2013). A longitudinal study of grapheme-color synesthesia in childhood: 6/7 years to 10/11 years. *Front. Hum. Neurosci.* 7, 603.
66. Huang, M. X., Dale, A. M., Song, T., Halgren, E., Harrington, D. L., Podgorny, I., ... Lee, R. R. (2006). Vector-based spatial-temporal minimum L1-norm solution for MEG. *Neuroimage*, 31, 1025–1037.
67. Attar, F. M., Kirilina, E., Haenelt, D., Pine, K. J., Trampel, R., Edwards, L. J., & Weiskopf, N. (2020). Mapping short association fibers in the early cortical visual processing stream using in vivo diffusion tractography. *Cereb. Cortex*, 30, 4496–4514.
68. Baker, C. M., Burks, J. D., Briggs, R. G., Conner, A. K., Glenn, C. A., Robbins, J. M., ... Sughrue, M. E. (2018). A connectomic atlas of the human cerebrum - chapter 5: The insula and opercular cortex. *Oper Neurosurg*, 15, S175–S244.
69. Kraus, K. S., & Canlon, B. (2012). Neuronal connectivity and interactions between the auditory and limbic systems. Effects of noise and tinnitus. *Hearing Res*, 288, 34–46.
70. Canlon, B., Theorell, T., & Hasson, D. (2013). Associations between stress and hearing problems in humans. *Hearing Res*, 295, 9–15.
71. Hasson, D., Theorell, T., Bergquist, J., & Canlon, B. (2013). Acute stress induces hyperacusis in women with high levels of emotional exhaustion. *Plos One*, 8, e52945.
72. Gruebner, O., Rapp, M. A., Adli, M., Kluge, U., Galea, S., & Heinz, A. (2017). Cities and mental health. *Dtsch. Ärztebl. Int.*, 114, 121–127.
73. Stansfeld, S. A., Berglund, B., Clark, C., Lopez-Barrio, I., Fischer, P., Öhrström, E., ... Berry, B. F. (2005). Aircraft and road traffic noise and children's cognition and health: A cross-national study. *Lancet*, 365, 1942–1949.
74. Goines, L., & Hagler, L. (2007). Noise pollution: A modern plague. *South Med J*, 100, 287–294.
75. Lavandier, C., & Defréville, B. (2006). The contribution of sound source characteristics in the assessment of urban soundscapes. *Acta Acust. United Ac*, 92, 912–921.
76. Alvarsson, J. J., Wiens, S., & Nilsson, M. E. (2010). Stress recovery during exposure to nature sound and environmental noise. *Int. J. Environ. Res. Public Health*, 7, 1036–1046.
77. Hunter, M. D., Eickhoff, S. B., Pheasant, R. J., Douglas, M. J., Watts, G. R., Farrow, T. F., ... Woodruff, P. W. R. (2010). The state of tranquility: Subjective perception is shaped by contextual modulation of auditory connectivity. *Neuroimage*, 53, 611–618.
78. Harrison, L., & Loui, P. (2014). Thrills, chills, frissons, and skin orgasms: Toward an integrative model of transcendent psychophysiological experiences in music. *Front. Psychol.* 5, 790.

How to cite this article: McGeoch, P. D., & Rouw, R. (2020). How everyday sounds can trigger strong emotions: ASMR, misophonia and the feeling of wellbeing. *BioEssays*, 42, e2000099. <https://doi.org/10.1002/bies.202000099>